Virulence Characterization of International Collections of the Wheat Stripe Rust Pathogen, *Puccinia striiformis* f. sp. *tritici*

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Abstract

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Wheat stripe rust (yellow rust [Yr]), caused by *Puccinia striiformis* f. sp. *tritici*, is an economically important disease of wheat worldwide. Virulence information on *P. striiformis* f. sp. *tritici* populations is important to implement effective disease control with resistant cultivars. In total, 235 *P. striiformis* f. sp. *tritici* isolates from Algeria, Australia, Canada, Chile, China, Hungary, Kenya, Nepal, Pakistan, Russia, Spain, Turkey, and Uzbekistan were tested on 20 single Yr-gene lines and the 20 wheat genotypes that are used to differentiate *P. striiformis* f. sp. *tritici* races in the United States. The 235 isolates were identified as 129 virulence patterns on the single-gene lines and 169 virulence patterns on the U.S. differentials. Virulences to YrA, Yr2, Yr6, Yr7, Yr8, Yr9, Yr17, Yr25, YrUkn, Yr28, Yr31, YrExp2, Lemhi (Yr21), Paha (YrPa1, YrPa2, YrPa3), Druchamp (Yr3a, YrD, YrDru), Produra (YrPr1, YrPr2), Stephens (Yr3a, YrS, YrSte), Lee (Yr7, Yr22, Yr23), Fielder (Yr6, Yr20), Tyee (YrTye), Tres (YrTr1, YrTr2), Express (YrExp1, YrExp2), Clement (Yr9, YrCle), and Compair (Yr8, Yr19) were detected in all countries. At least 80% of the isolates were virulent on YrA, Yr2, Yr6, Yr7, Yr8, Yr17, YrUkn, Yr31, YrExp2, Yr21, Stephens (Yr3a, YrS, YrSte), Lee (Yr7, Yr22, Yr23), and Fielder (Yr6, Yr20). Virulences to

Yr1, Yr9, Yr25, Yr27, Yr28, Heines VII (Yr2, YrHVII), Paha (YrPa1, YrPa2, YrPa3), Druchamp (Yr3a, YrD, YrDru), Produra (YrPr1, YrPr2), Yamhill (Yr2, Yr4a, YrYam), Tyee (YrTye), Tres (YrTr1, YrTr2), Hyak (Yr17, YrTye), Express (YrExp1, YrExp2), Clement (Yr9, YrCle), and Compair (Yr8, Yr19) were moderately frequent (>20 to <80%). Virulence to Yr10, Yr24, Yr32, YrSP, and Moro (Yr10, YrMor) was low (≤20%). Virulence to Moro was absent in Algeria, Australia, Canada, Kenya, Russia, Spain, Turkey, and China, but 5% of the Chinese isolates were virulent to Yr10. None of the isolates from Algeria, Canada, China, Kenya, Russia, and Spain was virulent to Yr24; none of the isolates from Algeria, Australia, Canada, Nepal, Russia, and Spain was virulent to Yr32; none of the isolates from Australia, Canada, Chile, Hungary, Kenya, Kenya, Nepal, Pakistan, Russia, and Spain was virulent to YrSP; and none of the isolates from any country was virulent to Yr5 and Yr15. Although the frequencies of virulence factors were different, most of the *P. striiformis* f. sp. *tritici* isolates from these countries shared common virulence factors. The virulences and their frequencies and distributions should be useful in breeding stripe-rust-resistant wheat cultivars and understanding the pathogen migration and evolution.

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*The e-Xtra logo stands for “electronic extra” and indicates that three supplementary tables and one supplementary figure are available online.

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Stripe rust (yellow rust), caused by *Puccinia striiformis* Westend. f. sp. *tritici* Erikss., is a worldwide economically important disease of wheat (5,19,36). Frequent and severe disease epidemics are common in major wheat-growing countries (45). Among the countries in the Americas, stripe rust is especially important in the United States (5), Mexico, Chile, and other Andean countries (11). Similarly, stripe rust is a serious problem in Europe, the Middle East (36,45), Central Asia (26), China (4), and the northern Indian subcontinent (34). In Africa, the disease occurs on wheat grown in the highlands of Ethiopia, Kenya, and Uganda (11,37). In recent decades, wheat stripe rust has spread into additional countries and has become important in Australia, New Zealand, and South Africa (45).

Cultivation of resistant cultivars is the most economically effective method for stripe rust management and minimizes environmental impacts by reducing the use of fungicides (5,19). However, within a few years of deploying cultivars with race-specific resistance genes, new virulent races (pathotypes) of the rust pathogen often emerge (20,34,37). As a result, new races can infect previ-

ously resistant cultivars, rendering them susceptible (5,36). Severe stripe rust epidemics are often due to the emergence of new races that cause failure of resistance genes (6). For example, more than 80% of cultivars released in the late 1980s had *Yr9* and, as a result, a new race virulent on *Yr9* was reported in 1985; this race caused epidemics resulting in yield loss of 2.65 million tons in 1990 in China (4,41). Similarly, *Yr27* has been widely used in wheat cultivars grown in India and Pakistan and the emergence of new races virulent to *Yr27* has been reported in South Asia between 2002 and 2004 (34). Serious stripe rust outbreaks in south- and central-Asian countries in 2009 have been attributed to races virulent to *Yr27* (2). These epidemics are further aggravated when new races possess more complex virulence combinations evolved through mutation, recombination, or migration over long distance (34,37,45). Such epidemics result in application of a large amount of fungicides to manage a disease that would otherwise cause substantial yield loss (5,6).

Monitoring *P. striiformis* f. sp. *tritici* races should be a high priority in all epidemic regions in the world, because the rust urediniospores can be wind transported over long distances (34). Frequent monitoring of the virulence spectra of *P. striiformis* f. sp. *tritici* populations and identification of new races are prerequisites for devising effective disease management strategies through resistance breeding and prediction of future races (23). Identification of virulent races is essential for the characterization of non-race-specific resistance (5). Information on virulence is also useful in screening cultivars or breeding lines for the determination of resistance levels and exploitation of new resistance genes. In addition, monitoring the virulence of the stripe rust pathogen population in different regions helps to understand the current genetic variability of host-pathogen interactions.

Virulence of the stripe rust pathogen has been routinely studied at a national scale (4,5,44); however, few studies have compared virulences at an international scale. Stubbs (37) reviewed and narrated the regional distribution of stripe rust virulences in Africa, Asia, Europe, and Americas. Recently, Hvømøller et al. (14) carried out a virulence study of international collections of *P. striiformis* f. sp. *tritici* but isolates were predominantly from Denmark and the Red Sea area. In addition, the study mainly focused on the spread of aggressive strains rather than all virulences. Therefore, the objectives of this study were to identify virulences and races, and determine their frequencies and distributions of *P. striiformis* f.

sp. *tritici* samples collected from 2006 to 2010 in different wheat-growing countries.

Materials and Methods

Stripe rust isolates. In total, 235 *P. striiformis* f. sp. *tritici* isolates were analyzed; 1 from Algeria collected in 2009, 5 from Australia (2006), 5 from Canada (2007), 15 from Chile (2007), 60 from China (2006 and 2007), 4 from Hungary (2009), 4 from Kenya (2006), 21 from Nepal (2007 and 2008), 46 from Pakistan (2006–09), 2 from Russia (2006), 2 from Spain (2008 and 2009), 54 from Turkey (2009 and 2010), and 16 from Uzbekistan (2009 and 2010). Leaf samples bearing stripe rust uredinia were stored at 4°C upon receipt.

Spore production. Urediniospores were revived and increased by placing leaf sections of about 3 cm in length on moist blotting paper and incubating overnight at temperatures gradually cycling from 15 to 4 to 15°C. Fresh urediniospores produced on the leaf surface were inoculated with a clean, fine paint brush on leaves of susceptible winter wheat ‘Nugaines’ grown in plastic pots to the two-leaf stage. Isolates stored as urediniospores in liquid nitrogen were reactivated by submerging spore-containing glass vials or foil bags in a water bath at 50°C for 2 min, and were inoculated on Nugaines seedlings to increase urediniospores (8). Nugaines was grown in plastic pots (7 by 7 by 7 cm) filled with a potting mixture of 24 liters of peat moss, 8 liters of perlite, 12 liters of sand, 12 liters of commercial potting soil mix, 16 liters of vermiculite, and 250 g of 14%-14%-14% (available N-P-K) Osmocote fertilizer (Scotts Miracle-Gro Company). Plants were grown in a rust-free greenhouse prior to inoculation. Inoculated plants were kept in a dew chamber for at least 12 h at 10°C, then transferred into a temperature-controlled growth chamber. The temperature of the growth chamber was programmed between a minimum of 4°C at 2:00 a.m. during the 8-h dark period and a maximum of 20°C at 2:00 p.m. during the 16-h light period. Metal halide lights were used as a supplement to the daylight of a 16-h photoperiod. Inoculated seedlings were isolated from each other with a transparent plastic cylinder surrounding each pot to prevent cross-contamination (7). Urediniospores were collected 16 days after inoculation and spore collection was repeated three to four times until sufficient quantities were harvested (approximately 20 mg). Urediniospores were dried in a desiccator at 4°C for 1 week before being stored at 4°C for a short term (up to 2 months) or in liquid nitrogen

Table 1. Twenty single *Yr*-gene lines of wheat and the set of 20 wheat genotypes for differentiating *Puccinia striiformis* f. *tritici* races in the United States used to test international *P. striiformis* f. sp. *tritici* collections for determination of their virulences and avirulences^a

Number	Single <i>Yr</i> gene lines ^b		U.S. differentials	
	Name	<i>Yr</i> gene	Name	<i>Yr</i> gene
1	AvSYrANIL	<i>YrA</i>	Lemhi	<i>Yr21</i>
2	AvSYr1NIL	<i>Yr1</i>	Chinese 166	<i>Yr1</i>
3	Siete Cerros T66	<i>Yr2</i>	Heines VII	<i>Yr2,YrHVII</i>
4	AvSYr5NIL	<i>Yr5</i>	Moro	<i>Yr10,YrMor</i>
5	AvSYr6NIL	<i>Yr6</i>	Paha	<i>YrPa1,YrPa2,YrPa3</i>
6	AvSYr7NIL	<i>Yr7</i>	Druchamp	<i>Yr3a,YrD,YrDru</i>
7	AvSYr8NIL	<i>Yr8</i>	AvSYr5NIL	<i>Yr5</i>
8	AvSYr9NIL	<i>Yr9</i>	Produra	<i>YrPr1,YrPr2</i>
9	AvSYr10NIL	<i>Yr10</i>	Yamhill	<i>Yr2,Yr4a,YrYam</i>
10	AvSYr15NIL	<i>Yr15</i>	Stephens	<i>Yr3a,YrS,YrSte</i>
11	AvSYr17NIL	<i>Yr17</i>	Lee	<i>Yr7,Yr22,Yr23</i>
12	AvSYr24NIL	<i>Yr24</i>	Fielder	<i>Yr6,Yr20</i>
13	TP981	<i>Yr25</i>	Tyee	<i>YrTye</i>
14	AvSYrUknNIL	<i>YrUkn^c</i>	Tres	<i>YrTr1,YrTr2</i>
15	AvSYr27NIL	<i>Yr27</i>	Hyak	<i>Yr17,YrTye</i>
16	AvSYr28NIL	<i>Yr28</i>	Express	<i>YrExp1,YrExp2</i>
17	AvSYr31NIL	<i>Yr31</i>	AvSYr8NIL	<i>Yr8</i>
18	AvSYr32NIL	<i>Yr32</i>	AvSYr9NIL	<i>Yr9</i>
19	AvSYrSPNIL	<i>YrSP</i>	Clement	<i>Yr9,YrCle</i>
20	AvS/EXP1/1-1 Line 74	<i>YrExp2</i>	Compair	<i>Yr8,Yr19</i>

^a NIL = near isogenic line.

^b *Yr* gene lines are in the ‘Avocet Susceptible’ (AvS) background.

^c *YrUkn* was included in the study initially as *Yr26* but, later, it was found not to be *Yr26* and different from any known *Yr* genes.

for the long term (years). Fresh urediniospores or those stored at 4°C within 2 months were tested on wheat single *Yr*-gene lines and differentials (8).

Determination of virulences and virulence patterns. In all, 20 single *Yr*-gene lines and 20 wheat genotypes used to differentiate races of *P. striiformis* f. sp. *tritici* in the United States (7,8) (Table 1) were used to identify virulences among the *P. striiformis* f. sp. *tritici* isolates. Previously described procedures and conditions (at the low diurnal temperature profile of 4 to 20°C) were followed to grow plants and inoculate wheat differentials (8,20).

Seedlings were evaluated for infection type (IT) 20 to 22 days after inoculation (7). Scoring of ITs from 0 to 9 was done as described by Line and Qayoum (20), where 0 = no visible signs or symptoms; 1 = necrotic or chlorotic flecks, no sporulation; 2 = necrotic or chlorotic blotches, no sporulation; 3 = necrotic or chlorotic blotches, trace sporulation; 4 = necrotic or chlorotic blotches, light sporulation; 5 = necrotic or chlorotic blotches, intermediate sporulation; 6 = necrotic or chlorotic blotches, moderate sporulation; 7 = necrotic or chlorotic blotches, abundant sporulation; 8 = chlorosis behind sporulating area, abundant sporulation; and 9 = no necrosis or chlorosis, abundant sporulation. ITs 0 to 6 were considered avirulent and 7 to 9 virulent (17,40). The virulence test on the two sets of wheat differentials, per isolate, was done once or repeated if the results were inconclusive.

Virulence patterns and frequencies. Virulence and avirulence patterns based on data from the single *Yr*-gene lines and the U.S. differentials were determined separately for all isolates. The virulence or avirulence pattern based on the single *Yr*-gene lines was presented by *Yr* genes whereas the virulence patterns based on the U.S. differentials were presented by the designated differential numbers for each isolate. Isolates from Algeria, Spain, and Russia were included only to calculate the total percentage of virulence factors in the international collection but not used in the country-wise analyses of virulence frequency due to the limited sample numbers. For these analyses, virulence and avirulence data from both differential sets were combined. The virulence and avirulence ITs of each isolate on the differential genotypes were assigned with binary codes of 1 and 0, respectively. Frequencies were calculated for all virulence factors.

Cluster analysis. Virulence relationships among the isolates were analyzed using the NTSYSpc software (version 2.10e; (Applied Biostatistics Inc.) (8,32). A similarity matrix was calculated based on simple matching coefficients. Cluster analysis was performed based on the similarity matrix to generate a dendrogram using the unweighted pair group method with arithmetic mean (UPGMA; 31). A correlation coefficient between cophenetic matrix and similarity matrix was calculated using the Mantel test. Robustness of the dendrogram branches was determined with the Winboot programs (29; <http://archive.irri.org/science/software/winboot.asp>). In addition, a three-dimensional principal coordinate (PC) plot was generated in NTSYSpc by transforming the similarity matrix using the DCENTER module and then using EIGEN module.

Results

Virulences and their frequencies and distributions. ITs of the 235 *P. striiformis* f. sp. *tritici* isolates using the 0-to-9 scale are given in Supplementary Table S1. ITs observed were mostly either low (ITs 0 to 2; 38%) or high (ITs 7 to 9; 58%), with only a very low percentage (4%) showing ITs 3 to 6, which made it relatively easy to assign isolates to virulence patterns. Stripe rust isolates from different countries differed in virulence patterns, frequencies, and distributions, except for Algeria, Russia, and Spain due to a limited number of samples (Table 2). At an international scale, the most frequent virulences (≥80%) were those to resistance genes *YrA* (89%), *Yr2* (92%), *Yr6* (92%), *Yr7* (90%), *Yr8* (80%), *Yr17* (88%), *YrUkn* (89%), *Yr31* (99%), *YrExp2* (89%), and *Yr21* (98%) and U.S. differentials 10 (91%), 11 (84%), and 12 (84%). Moderately frequent virulences (between 20 and 80%) included *Yr1* (49%), *Yr9* (68%), *Yr25* (39%), *Yr27* (69%), and *Yr28* (79%)

and U.S. differentials 3 (56%), 5 (70%), 6 (40%), 9 (31%), 13 (42%), 14 (41%), 15 (31%), 16 (71%), 19 (54%), and 20 (69%). The least frequent virulences (≤20%) were to *Yr10* (9%), *Yr24* (11%), *Yr32* (11%), *YrSP* (9%), and Moro (6%). None of the isolates were virulent to *Yr5* and *Yr15*.

Out of 36 single *Yr* genes or gene combinations of the two sets of differentials, virulences to 25 lines (*YrA*, *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr17*, *Yr25*, *YrUkn*, *Yr28*, *Yr31*, and *YrExp2* and U.S. differentials 1, 5, 6, 8, 10, 11, 12, 13, 14, 16, 19, and 20) were observed in all countries. Isolates from all of these countries were virulent on *Yr21*, except four isolates from Pakistan. More than 50% of isolates from these countries were virulent to *YrA*, *Yr2*, *Yr6*, *Yr17*, *YrUkn*, and *Yr31* and U.S. differentials 1, 10, and 12. All isolates from Canada, Hungary, Nepal, and Uzbekistan were virulent to *YrA*. Similarly, all isolates from Australia, Canada, Chile, Hungary, Kenya, Nepal, and Uzbekistan were virulent to *Yr2* and *Yr31*. All isolates from Turkey were virulent to *Yr31*. All isolates from Canada and Hungary were virulent to both *Yr6* and *Yr7*, whereas all isolates from Uzbekistan were virulent on *Yr6* and from Kenya and Nepal were virulent on *Yr7*. Virulence on Stephens (differential 10) was detected in all isolates from Australia, Canada, Hungary, and Uzbekistan. Isolates were virulent to *Yr27* from all countries except Australia; however, three of five isolates from Australia had an intermediate reaction (IT 6) on the *Yr27* single gene line.

Virulences to *Yr1*, *Yr10*, *Yr24*, *Yr32*, and *YrSP* and U.S. differentials 3, 4, 9, and 15 were present but not in all countries. None of the isolates from Australia, Canada, and Turkey were virulent to *Yr10* and Moro (*Yr10* and *YrMor*). None of the Chinese *P. striiformis* f. sp. *tritici* isolates were virulent on Moro but some (5%) were virulent to *Yr10*. None of the isolates from Canada, China, and Kenya were virulent to *Yr24*; none of the isolates from Australia, Canada, and Nepal were virulent to *Yr32*; and none of the isolates from Australia, Canada, Chile, Hungary, Kenya, Nepal, and Pakistan were virulent to *YrSP*. Only all Chilean and Kenyan isolates were avirulent to Hyak (*Yr17*, *YrTye*).

Isolates from Algeria, Russia, and Spain, not included in the country-wise frequency analysis (Table 2), were virulent to *Yr2*, *Yr6*, *Yr8*, *Yr9*, *Yr17*, and *YrUkn* and U.S. differentials 1 and 10. All of these isolates were avirulent to *Yr5*, *Yr10*, *Yr15*, *Yr24*, and *Yr32* and U.S. differentials 4 and 15. In addition, the isolates from Algeria and Spain were avirulent to *YrSP* and U.S. differentials 6 and 9.

Virulence patterns. More virulence patterns were identified based on the 20 U.S. differentials for most countries than those based on the 20 single *Yr*-gene lines. Of the 235 isolates, the single *Yr*-gene lines identified 129 whereas the set of U.S. wheat differentials identified 169 virulence patterns (Table 3). The virulence and avirulence patterns identified from the 235 isolates based on the single *Yr*-gene lines and the U.S. wheat differentials are listed in Supplementary Tables S2 and S3, respectively. Among the 129 virulence patterns identified with the single *Yr*-gene lines, 101 were detected only with one isolate. The most frequent (13%) pattern was the one virulent to resistance genes *YrA*, *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr17*, *YrUkn*, *Yr27*, *Yr28*, *Yr31*, and *YrExp2* and avirulent to *Yr1*, *Yr5*, *Yr10*, *Yr15*, *Yr24*, *Yr25*, *Yr32*, and *YrSP*. This virulence pattern was detected in isolates from Canada, China, and Turkey.

The highest frequency (93%) of two associated virulence factors was found for *Yr2* and *Yr31*. Similarly, the highest frequencies for three, four, and five virulence factors in combination were 86% (virulences to *Yr2*, *Yr6*, and *Yr31*), 79% (virulences to *Yr2*, *Yr6*, *Yr7*, and *Yr31*), and 74% (virulences to *YrA*, *Yr2*, *Yr6*, *Yr7*, and *Yr31*), respectively. These virulence combinations were present in all countries.

Of the 169 virulence patterns based on the U.S. differentials, 122 patterns each were detected with only one isolate. The most frequent (8%) virulence pattern, which was virulent on U.S. differentials 1 (*Yr21*), 5 (*YrPa1*, *YrPa2*, *YrPa3*), 8 (*YrPr1*, *YrPr2*), 10 (*Yr3a*, *YrS*, *YrSte*), 12 (*Yr6*, *Yr20*), 14 (*YrTr1*, *YrTr2*), 16 (*YrExp1*, *YrExp2*), 17 (*Yr8*), and 20 (*Yr8*, *Yr19*) and avirulent on 2 (*Yr1*), 3 (*Yr2*, *YrHVII*), 4 (*Yr10*, *YrMor*), 6 (*Yr3a*, *YrD*, *YrDru*), 7 (*Yr5*), 9 (*Yr2*, *Yr4a*, *YrYam*), 11 (*Yr7*, *Yr22*, *Yr23*), 13 (*YrTye*), 15

(Yr17, YrTye), 18 (Yr9), and 19 (Yr9, YrCle) was detected in isolates from Chile, China, and Turkey. Twenty of the virulence patterns obtained from the international isolate resembled races identified in the United States. Virulences on U.S. differentials 1 and 10 appeared together at the highest frequency (90%). The highest frequencies for combinations of virulences on three (1, 10, and 11) and four (1, 10, 11, and 12) of the U.S. differentials were 78 and 70%, respectively. These virulence combinations were detected in all of the countries.

Cluster analysis. Cluster analysis was performed on combined virulence and avirulence data obtained from the single Yr-gene lines and the U.S. differentials. A cutoff point of 0.74 similarity coefficient was determined based on mean value of similarity range. In general, isolates from different countries were grouped together in the same groups. All 235 isolates clustered into 11 virulence groups (VGs) (Fig. 1). These 11 VGs were further grouped into two major VGs at a similarity coefficient of 0.48. The first major group consisted of VG 1 to VG 8 and accounted for 97% of the isolates; the second major group consisted of VG 9 to VG 11 and accounted for 3% of the isolates. VGs 1 and 5 were the largest groups and accounted for 81% of isolates. VG 1 had 69 isolates (29%) (all virulent to Yr1, YrA, Yr6, Yr9, Yr21, Yr31, and Stephens

Table 3. Number of *Puccinia striiformis* f. sp. *tritici* virulence patterns based on single Yr-gene lines and U.S. differentials from different countries

Countries	Number of isolates	Number of races	
		Yr-gene	Differentials
Algeria	1	1	1
Australia	5	5	5
Canada	5	4	5
Chile	15	13	14
China	60	39	40
Hungary	4	4	4
Kenya	4	4	4
Nepal	21	15	20
Pakistan	46	28	42
Russia	2	2	2
Spain	2	2	2
Turkey	54	27	45
Uzbekistan	16	9	13
Total ^a	235	129 ^a	169 ^a

^a Total number of virulence patterns was calculated from different patterns from the 235 isolates regardless of the numbers in each country as some patterns shared by different countries.

Table 2. Number and frequency of virulences in *Puccinia striiformis* f. sp. *tritici* isolates collected from different countries on single Yr-gene lines (Yr) and U.S. differentials (Diff)

Number (N) and frequency (%) of virulence in <i>P. striiformis</i> f. sp. <i>tritici</i> isolates from different countries ^a																							
Number	Yr gene	Total		AU		CA		CL		CN		HU		KE		NP		PK		TR		UZ	
		%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	
<i>Yr</i>																							
1	<i>A</i>	89	4	80	5	100	9	60	59	98	4	100	2	50	21	100	32	70	53	98	16	100	
2	<i>I</i>	49	2	40	0	0	4	27	25	42	2	50	3	75	15	71	26	57	21	39	14	88	
3	<i>2</i>	93	5	100	5	100	15	100	54	90	4	100	4	100	21	100	41	89	49	91	16	100	
4	<i>5</i>	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
5	<i>6</i>	92	3	60	5	100	9	60	56	93	4	100	3	75	20	95	44	96	52	96	16	100	
6	<i>7</i>	90	2	40	5	100	12	80	57	95	4	100	4	100	21	100	42	91	51	94	10	63	
7	<i>8</i>	80	1	20	3	60	6	40	53	88	3	75	3	75	18	86	34	74	51	94	10	63	
8	<i>9</i>	68	2	40	3	60	5	33	30	50	3	75	4	100	12	57	39	85	40	74	16	100	
9	<i>10</i>	9	0	0	0	0	5	33	3	5	2	50	1	25	4	19	5	11	0	0	1	6	
10	<i>15</i>	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
11	<i>17</i>	88	5	100	5	100	14	93	41	68	4	100	4	100	18	86	45	98	50	93	15	94	
12	<i>24</i>	11	1	20	0	0	4	27	0	0	1	25	0	0	1	5	13	28	4	7	1	6	
13	<i>25</i>	39	1	20	1	20	6	40	18	30	3	75	2	50	8	38	17	37	22	41	12	75	
14	<i>Ukn</i>	89	3	60	4	80	10	67	49	82	2	50	3	75	21	100	45	98	52	96	16	100	
15	<i>27</i>	69	0	0	3	60	4	27	40	67	3	75	2	50	16	76	38	83	45	83	9	56	
16	<i>28</i>	79	4	80	5	100	13	87	37	62	3	75	1	25	18	86	36	78	50	93	15	94	
17	<i>31</i>	99	5	100	5	100	15	100	59	98	4	100	4	100	21	100	45	98	54	100	16	100	
18	<i>32</i>	11	0	0	0	0	5	33	1	2	2	50	0	0	0	0	13	28	4	7	1	6	
19	<i>SP</i>	10	0	0	0	0	0	0	16	27	0	0	0	0	0	0	0	0	4	7	2	13	
20	<i>Exp2</i>	89	2	40	5	100	12	80	56	93	4	100	4	100	20	95	43	93	49	91	10	63	
<i>Diff</i>																							
1	<i>21</i>	98	5	100	5	100	15	100	60	100	4	100	4	100	21	100	41	89	54	100	16	100	
2	<i>I</i>	49	2	40	0	0	4	27	25	42	2	50	3	75	15	71	26	57	21	39	14	88	
3	<i>2,HVII</i>	56	5	100	3	60	2	13	22	37	4	100	1	25	9	43	33	72	35	65	13	81	
4	<i>10,Mor</i>	7	0	0	0	0	4	27	0	0	2	50	0	0	4	19	5	11	0	0	1	6	
5	<i>Pa1,Pa2,Pa3</i>	70	5	100	1	20	5	33	49	82	3	75	2	50	16	76	31	67	33	61	16	100	
6	<i>3a,D,Dru</i>	40	4	80	1	20	6	40	23	38	2	50	1	25	10	48	17	37	15	28	14	88	
7	<i>5</i>	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
8	<i>Pr1,Pr2</i>	77	3	60	4	80	7	47	53	88	4	100	1	25	12	57	35	76	46	85	14	88	
9	<i>2,4a,Yam</i>	31	0	0	0	0	3	20	20	33	2	50	0	0	5	24	11	24	19	35	11	69	
10	<i>3a,S,Ste</i>	91	5	100	5	100	12	80	54	90	4	100	2	50	20	95	39	85	52	96	16	100	
11	<i>7,22,23</i>	84	2	40	5	100	10	67	52	87	4	100	3	75	18	86	40	87	50	93	10	63	
12	<i>6,20</i>	84	3	60	4	80	8	53	41	68	4	100	3	75	20	95	43	93	52	96	16	100	
13	<i>Tye</i>	42	1	20	2	40	1	7	24	40	3	75	2	50	9	43	22	48	21	39	12	75	
14	<i>Tr1,Tr2</i>	41	1	20	4	80	5	33	30	50	2	50	1	25	6	29	21	46	20	37	4	25	
15	<i>17,Tye</i>	31	1	20	2	40	0	0	20	33	2	50	0	0	2	10	20	43	17	31	8	50	
16	<i>Exp1,Exp2</i>	71	2	40	4	80	7	47	41	68	4	100	4	100	19	90	35	76	40	74	8	50	
17	<i>8</i>	80	1	20	3	60	6	40	53	88	3	75	3	75	18	86	34	74	51	94	10	63	
18	<i>9</i>	68	2	40	3	60	5	33	30	50	3	75	4	100	12	57	39	85	40	74	16	100	
19	<i>9,Cle</i>	54	1	20	3	60	4	27	24	40	3	75	3	75	9	43	25	54	38	70	15	94	
20	<i>8,19</i>	69	1	20	3	60	5	33	49	82	3	75	3	75	15	71	30	65	43	80	7	44	

^a AU = Australia, CA = Canada, CL = Chile, CN = China, HU = Hungary, KE = Kenya, NP = Nepal, PK = Pakistan, TR = Turkey, and UZ = Uzbekistan. Isolates from Algeria, Spain, and Russia were used to calculate the total virulence percentage (%) but country-wise virulence frequency was not calculated for the three countries due to the limited sample numbers.

[Yr3a, YrS, YrSte]) from all countries except Algeria, Canada, Chile, and Spain, with an average similarity of 76% (Supplementary Figure S1). VG 5 was the largest group, with 121 (51%) isolates from all countries except Algeria, having similarity of about 77%. Isolates belonging to VG 5 had different frequencies of virulences on all differentials tested except Yr5 and Yr15. Isolates belonging to VG 2 were virulent to Yr1, Yr2, Yr6, Yr7, Yr8, Yr9, Yr17, Yr25, and Yr31 and U.S. differentials 1, 3, 11, 12, 13, 19, and 20. These isolates were all avirulent to Yr5, Yr10, Yr15, Yr17, Yr24, Yr27, Yr32, and YrSP and U.S. differentials 4, 5, 6, 9, and 15. Isolates on VG 3 were virulent to YrA, Yr1, Yr2, Yr7, Yr9, Yr31, and YrExp2 and U.S. differentials 1, 3, 10, 13, and 20 but avirulent to Yr5, Yr10, Yr15, Yr17, Yr24, Yr26, Yr27, Yr32, and YrSP and U.S. differentials 4, 12, and 16. Similarly, isolates belonging to VG 4 were all virulent to YrA, Yr1, Yr6, Yr7, Yr9, Yr31, and YrExp2 and U.S. differentials 1, 5, 7, 12, 13, and 19 but avirulent to Yr2, Yr5, Yr10, Yr15, Yr24, Yr27, Yr28, Yr32, and YrSP and U.S. differentials 3, 4, 6, 14, 15, 16, and 20. The isolates in VG 6 all had virulences to YrA, Yr2, Yr6, Yr7, Yr8, Yr9, Yr17, Yr31, and

YrExp2 and U.S. differentials 1, 12, 19, and 20. These isolates were avirulent on Yr5, Yr15, and YrSP and U.S. differential 9. VG 7 isolates were all virulent on Yr2, Yr7, Yr17, Yr31, and YrExp2 and U.S. differential 1, 8, 16, and 18 and were all avirulent to Yr1, Yr5, Yr10, Yr15, Yr25, Yr27, and YrSP and U.S. differentials 3, 4, 6, 9, 11, 15, 17, and 20. The isolates in VG 8 were virulent to Yr2, Yr17, Yr28, Yr31, and YrExp2 and U.S. differential 1.

In general, the isolates in VGs 9 to 11 had relatively narrow virulence spectra compared with those of VGs 1 to 8. The isolates in VG 9 were all virulent to Yr2, Yr17, and Yr31 and U.S. differentials 1, 3, 5, 6, and 10. The VG 10 isolates had virulences to Yr2, Yr10, Yr31, and Yr32 and U.S. differentials 1, 5, and 14. All isolates in VG 11 were virulent on Yr2, Yr6, Yr17, YrUkn, and Yr31 and U.S. differentials 1, 3, 12, 13, and 15. Isolates belonging to MG 2 (VG 9-11) were avirulent to Yr5, Yr7, Yr8, Yr9, Yr25, Yr27, and YrSP and U.S. differentials 8, 9, 11, 16, 19, and 20.

Of the 235 isolates, 3 isolates, 1 each from Chile (virulent on YrA, Yr1, Yr2, Yr6, Yr7, Yr8, Yr9, Yr10, Yr17, Yr25, YrUkn, Yr28, Yr31, Yr32, and YrExp2 and U.S. differentials 1, 2, 4, 6, 8, 9, 10,

Fig. 1. Dendrogram based on virulence phenotypes of *Puccinia striiformis* f. sp. *tritici* on 20 single Yr-gene lines and 20 U.S. differentials using the unweighted pair group method with arithmetic mean method. Three out-group isolates were not shown in the figure (see the text for details). Numbers along the nodes are bootstrap values >30%. AU = Australia, CA = Canada, CL = Chile, CN = China, DZ = Algeria, ES = Spain, HU = Hungary, KE = Kenya, NP = Nepal, PK = Pakistan, RU = Russia, TR = Turkey, and UZ = Uzbekistan.

11, 12, 16, 17, 18, and 19), China (virulent on *YrA*, *Yr1*, *Yr2*, *Yr7*, *Yr9*, *Yr17*, *Yr27*, *Yr28*, *Yr31*, *YrSP*, and *YrExp2* and U.S. differentials 1, 2, 5, 6, 8, 10, 11, 13, 15, and 18), and Pakistan (*Yr2*, *Yr6*, *Yr7*, *Yr9*, *Yr17*, *Yr25*, *YrUkn*, *Yr27*, *Yr28*, *Yr31*, and *YrExp2* and U.S. differentials 1, 3, 5, 6, 9, 10, 11, 14, 16, 18, and 19) did not cluster into any group.

The three-dimensional PC analysis showed the pairwise distance between isolates and groupings of isolates based on virulence data (Fig. 2). In general, isolates originating from different countries did not differentiate into unique groups. These results further supported the inclusion of isolates originating from different countries within the same VGs, as revealed by the cluster analysis (Fig. 1). For example, isolates CL07-12 and PK09-30 from Chile and Pakistan, respectively, had similar virulence patterns. However, even some isolates from the same country were found to be distantly related; for example, PK07-9 and PK06-3 from Pakistan (Fig. 2). The first dimension extracted 26% of the total virulence variation, and second and third dimensions accounted for 15 and 7%, respectively, of the total virulence variation.

Discussion

In all, 20 single *Yr*-gene lines and 20 U.S. wheat differentials were used to determine virulences and virulence patterns of 235 *P. striiformis* f. sp. *tritici* isolates collected from 13 countries. The results revealed common and unique virulences and patterns in the *P. striiformis* f. sp. *tritici* populations in these countries. Virulence patterns were generally different among countries; however, most of the virulences were common among isolates from different countries. Because the *P. striiformis* f. sp. *tritici* collections from the 13 countries varied greatly in sample number, the results from virulence analyses were used to make general conclusions about the overall collections; however, country-wise interpretation should be made cautiously where the sample number was limited. Therefore, major emphases have been given to the descriptive analysis rather than inferential statistics.

Comparison of the major virulences in the isolates from different countries was done based on the resistance genes of the single *Yr*-gene lines and U.S. differential genotypes. The *P. striiformis* f. sp. *tritici* isolates in different countries were all virulent to *YrA*, *Yr2*, *Yr6*, *Yr7*, *Yr31*, *Yr21*, and Stephens (*Yr3a*, *YrS*, *YrSte*). Widespread deployment of the same resistance genes over large wheat-growing regions eventually contributes to identical and similar virulence frequencies in the *P. striiformis* f. sp. *tritici* populations. For

example, resistant genes *Yr2*, *Yr6*, and *Yr7* were used extensively in Central Asia, West Asia, and North Africa. This may explain why all the isolates from these regions are virulent on these genes (1). Our finding is consistent with previous report that virulence to *YrA* was common in all wheat-growing continents (25,38). Virulence for *Yr2* was reported in the United States in 1964 (20) and in Turkey in 1967 and, in 1970, it was traced to the Indian subcontinent (33). *Yr2* is very common in both winter and spring wheat, and common in wheat distributed through CIMMYT (25). In addition, *Yr2* avirulence gene in *P. striiformis* f. sp. *tritici* is prone to mutation (35). Therefore, virulence to *Yr2* may have evolved simultaneously or migrated to all countries with isolates tested in the present study. Stubbs (36) also reported widespread presence of *Yr2* on an international scale back to the early 1980s. The high frequency of *Yr6* virulence in all countries may be explained by the presence of *Yr6* in common wheat and durum wheat (10). Similarly, the high frequency of *Yr7* virulence may be due to the worldwide use of *Yr7* through Thatcher (*Yr7*) and its presence in old cultivars or landraces (24). Stubbs (36) and Hovmöller et al. (14) also reported worldwide distribution of *Yr7* virulence. The widespread and high-frequency virulence factors may have been fixed in pathogen populations throughout the world.

The widespread and high frequency of *Yr8* virulence may be due to worldwide use of this gene from *Aegilops comosa* and its common presence in grasses (36). Virulence on *Yr8* was detected in the Middle East in 1973 (13), in England in 1978 (16), in Australia in the 1980s (43), and in Iran during 1997 to 1999 (28). Virulence to *Yr8* was detected in the United States in 2000 (7). The present study revealed that *Yr8* virulence is widespread, and this finding agrees with the results of Hovmöller et al. (14). Similarly, the high frequency of *Yr9* virulence and its presence in all isolates in the present study may have evolved from extensive use of *Yr9*, originating from Petkus rye (*Secale cereale*), in wheat-breeding programs worldwide (31,36). *Yr9* virulence was first identified in the former Soviet Union in 1973 and in The Netherlands in 1974 (39). Until 1996, races virulent to *Yr9* were reported in other regions or countries from East Africa to South Asia (34), in China (42), in South America (Ecuador, Colombia, Bolivia, and Chile) and Mexico (40), and in Australasia (46). *Yr9* virulence is common in most races identified in the United States since 2000 (8). The emergence and spread of *Yr9*-virulent races caused serious stripe rust outbreaks in many major wheat-growing regions (5–8,34).

Fig. 2. Three-dimensional principal coordinate plots of *Puccinia striiformis* f. sp. *tritici* isolates based on virulence and avirulence phenotypes on the single *Yr*-gene lines and U.S. wheat differentials.

Cultivars with *Yr27* were deployed extensively in Syria, Turkey, Iran, India, Pakistan, and other south-Asian countries to address the failure of *Yr9*. The wide cultivation of these cultivars created selection pressure for the pathogen and, thus, virulence for *Yr27* emerged in South Asia between 2002 and 2004 (34). In the present study, the *Yr9* and *Yr27* virulences were common in isolates from Pakistan, Nepal, Turkey, and Uzbekistan. Use of *Yr9* and *Yr27* led to the emergence of new races virulent on both *Yr9* and *Yr27* in the Middle East and South Asia (1). Severe stripe rust epidemics in Central and Western Asia and North Africa during the 2010 wheat season was attributed to the *Yr27* virulence lineage along with the favorable weather conditions (2).

In the present study, *Yr17* virulence was observed in 88% of all isolates and 98% from Pakistan isolates. In contrast, Hovmöller et al. (14) and Bahri et al. (1) reported approximately 3% in international isolates collected from Azerbaijan, Australia, Denmark, Eritrea, France, Iran, Italy, Kazakhstan, Kyrgyzstan, Mexico, Pakistan, South Africa, the United Kingdom, the United States, Uzbekistan, and Yemen and only approximately 4% in *P. striiformis* f. sp. *tritici* collections from Pakistan. The difference in the *Yr17* virulence frequencies might be due to different differential genotypes used in their studies and the present study. In their studies, Hovmöller et al. (14) and Bahri et al. (1) used VPM1 (*Yr17+*). The other *Yr* gene or genes in VPM1 might have masked the virulence to *Yr17* in their isolates. In the U.S. Pacific Northwest, the *Yr17* single-gene line has been widely susceptible, whereas VPM1 is still resistant (X. M. Chen, unpublished data).

The least frequent group of virulences was different among *P. striiformis* f. sp. *tritici* populations in various countries. *Yr10* virulence has been reported in Eastern Europe, the eastern Mediterranean region (including East Africa), and North America (8,14,20,36). The presence of *Yr10* virulence in Southeast Asia, Nepal, and Pakistan differed from the findings of Stubbs (36) and Hovmöller et al. (14). Although virulence to *Yr10* occurs in low frequency (5%) in China, it may have a serious implication because *Yr10* has been reported to be effective (41) and, thus, has been widely used in breeding programs (18).

The *P. striiformis* f. sp. *tritici* populations in different countries shared most of the virulences but their frequencies and virulence spectra were different. These differences may have resulted from selection benefits for certain races with new virulences, higher fitness, or aggressiveness against resistance genes commonly deployed in the regions. Consequently, such differences in selection may determine the frequencies of races in a population (23,34). Recombination shuffles virulence genes and creates more genotypes or races (15,36). Somatic hybridization has shown to result in new races (21,48). Mixed infection of races on same plants under field conditions may facilitate nuclear migration through urediniospore germ tubes and infectious hyphae within plant tissue, which can lead to the development of new races (22). Furthermore, sexual recombination, although not proven under natural conditions for *P. striiformis* f. sp. *tritici*, may play a role in generating new races in areas where the alternate host, barberry, coexists with wheat and the weather conditions are favorable for barberry infection by *P. striiformis* f. sp. *tritici* (15). However, the deployment of the same resistance genes across different continents is more likely to select identical or similar virulences in large regions. The presence of similar virulences in the international collections and in the U.S. populations (9) suggests that the *P. striiformis* f. sp. *tritici* population at the international scale mostly possesses the same genetic background of virulences (36). We are currently characterizing these *P. striiformis* f. sp. *tritici* isolates using molecular markers, which will help us understand genetic structures of the pathogen. In addition, this study will further clarify whether similar races in different countries were due to simultaneous evolution or migration. Migration of *P. striiformis* f. sp. *tritici* has been reported within and among continents (3,30,34,43,47).

Virulence to *Yr5* and *Yr15* were not detected in this study. This result is consistent with previous reports of rare detection of the

virulences throughout the world. *Yr5* was originally identified in hexaploid *Triticum spelta album*. The gene confers resistance to nearly all *P. striiformis* f. sp. *tritici* isolates worldwide. Virulence to *Yr5* has been reported only in India and Australia (27,47) but at a very low frequency. *Yr15* is from *T. turgidum* var. *dicoccoides* (wild emmer) (12). *Yr15* virulence was reported in Afghanistan and Denmark; otherwise, lines with *Yr15* are resistant to diverse *P. striiformis* f. sp. *tritici* isolates from different geographical origins (12,14,25). Thus, *Yr5* and *Yr15* still can be used in wheat-breeding programs. However, use of the race-specific genes alone may not provide long-lasting resistance. Therefore, effective race-specific genes like *Yr5* and *Yr15* and race-non-specific genes should be used in combination to achieve sustainable control of stripe rust.

In addition to the individual virulences and avirulences discussed above, we identified a large number of virulence patterns, similar to races or pathotypes that are usually determined on a relatively small number of selected host genotypes used as differentials (4,8,20,36,47). With the 20 single *Yr*-gene lines, the 235 isolates were differentiated into 129 virulence patterns, while 169 patterns were identified using the 20 U.S. differentials. The U.S. differentials identified more virulence patterns, as expected, because the 20 differential genotypes have at least 34 resistance genes, more than the 20 genes in the single *Yr*-gene lines (Table 1). The relatively large number of virulence patterns identified with both sets of the single *Yr*-gene lines and the U.S. differentials is expected considering the samples came from 13 countries. This might also be attributed to the fact that collectors tended to send diverse samples, which met our major objective of characterizing different virulences and patterns rather than focusing on frequencies. Regarding individual countries, the number of virulence patterns ranged from 1 (Algeria) to 39 (China) based on reactions on the 20 *Yr*-gene lines and 1 to 45 (Turkey) based on the U.S. differentials. The numbers of virulence patterns are not comparable among countries, because the numbers of isolates varied greatly by country. However, the upper range of the virulence pattern numbers is similar to what we have found in the United States in recent years (8). The virulence patterns based on both the *Yr*-gene lines and the U.S. differentials were clustered into 11 VGs. These groups share common virulences and avirulences within groups and have distinctions between groups. The present study is to report the maximum number of virulences and virulence patterns for people to be aware of, which should be more useful for selecting effective resistance genes and gene combinations in developing resistant cultivars. Although the virulence patterns described in the present study are not referred to as races, the virulence data generated using the 20 *Yr*-gene lines, in comparison with the U.S. cultivar differentials, may lead to the establishment of a standardized set of *Yr*-gene differentials. More efforts are being undertaken in this direction.

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