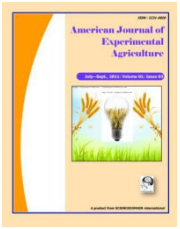

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Virulence of *Puccinia striiformis* f. sp. *tritici* in Khuzestan Province of Iran

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Research Article

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ABSTRACT

Yellow rust caused by *Puccinia striiformis* f. sp. *tritici* is undoubtedly, the most important fungal disease of wheat especially in Central and Western Asia causes significant annual yield losses. To investigate the population structure of the pathogen, isolates were collected from four regions and tested on 26 differential genotypes with known resistance genes in greenhouse and field conditions on the territory of Khuzestan province in Iran during 2009 - 2010. According to the results of race determination, races 4E14, 4E15, 6E128, 6E148, 6E142, 6E130, 6E158, 134E4 and 166E232 were common in all locations during the course of this study. Isolates with virulence on lines with yellow rust resistance gene Yr2, Yr6, Yr7, Yr7+, Yr6+, Yr9, Yr17, Yr18, Yr25 and YrA represented the most frequent phenotypes. Virulence to Yr1, Yr3V, Yr4, Yr5, Yr10 and YrSU was not found in any of the tested isolates. At the adult plant stage, virulence on wheat genotypes Heines Kolben, Kalyansona, Lee, Avocet R, Federation* 4/Kavkaz, TP1295 and Nord desprez was common during the period of investigation. The frequency of virulence factors in the yellow rust population on the differential genotypes were above 91% for the resistance genes Yr2,Yr6, Yr7, Yr2, Yr6+, Yr18, YrA, Yr2, Yr17, Yr25 and YrA however, virulence frequencies for Yr7+ and Yr8 were less than 10%. Frequency of virulence factors was low for Yr 2+, Yr7+, Yr8, Yr3N and YrSd.

Keywords: Triticum aestivum; resistance genes; Puccinia striiformis;

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1. INTRODUCTION

With more than 7000 species, rust fungi (Basidiomycota, Uredinales) are the largest group of obligate plant pathogens known to date (Aime, 2006). Among them, there are disease agents that severely affect field crops, vegetables, ornamentals, fruit, and forest trees (Agrios, 1997). Wheat rusts, for example, have influenced the course of early civilization by destroying a major source of food (Wiese, 1987) and are still most destructive pathogens in all provinces of Iran where, in 1993, have caused more than 1 million tons yield loss (Okhovat, 1999). Stripe rust is a micro cyclic rust disease causing important economic losses on some important members of Graminae family (Kavak, 2009). Stripe rust was dominant disease in Central Asian countries in the late 1990s and early 2000s, accounting for yield losses of 20-40% in 1999 and 2000 (Morgounov et al., 2004). During the last decades, several yellow rust epidemics in most of the wheat-growing areas of Iran caused over 30% crop loss and estimated grain losses were 1.5 and 1.5 million ton in 1993 and 1995, respectively (Torabi et al., 1995). Stripe rust can cause 100% yield loss if infection occurs very early and the disease continues to develop during the growing season provided the cultivars are susceptible (Afzal et al., 2007). In Iran, epidemics of cereal rusts occur every 3 or 4 years and frequent stripe rust epidemics have been reported in Iran (Esfandiari, 1947; Khazra et al., 1974; Bamdadian, 1984). The annual yield losses due to wheat yellow rust have been estimated up to 8- 75% (Elahinia, 2000).

Bamdadian (1984) estimated that overall losses in these years may be as high as 30 to 40%. In 1994, an estimated 15% (1.5 million tonnes) of the nation's wheat yield loss in Iran was caused by stripe rust (Torabi et al., 1995), particularly where the susceptible variety Very was grown over large areas. Wan et al., (2004) reported that, in the 2001–2002 growing season, a widespread stripe rust epidemic affected about 6.6 million hectares of wheat in China. Johnson et al., (1972) suggested a differential set and a new system for pathotype nomenclature based on the use of binary codes. Pathogenic changes have been a significant factor in the recurrent *P. striiformis* epidemics in the Middle East. In Iran, yellow rust has been observed annually since 1984 and the disease has spread to all wheat-growing areas. Under irrigated conditions and in the high rainfall areas in northern Iran, the severity of infection reached up to 80% on susceptible cultivars (Elahinia, 1989). The use of genetic resistance in wheat is the most economic way of controlling the disease (Röbbelen and Sharp, 1978; Line and Chen, 1995; Elyasi-Gomari and Lesovaya, 2009). In general, there are two mechanisms of resistance to stripe rust: seedling resistance, which can be expressed in all stages of plant development, and adult plant resistance, which expresses in adult stages. Several researchers have reported rust resistance in different wheat genotypes in Iran (Torabi et al., 1995; Khalilzadeh, 2008). However, their studies were based only on vertical resistance. Little research has been reported on screening of wheat lines for slow rusting. Seven pathotypes, *viz.* 6E0, 20E148, 38E134, 166E150, 6E20, 134E150 and 230E150 were reported in Iran between 1996 and 1997 (Torabi et al., 1998). Elahinia (2000) reported that the races identified in 1999, such as 230E150 and 230E134, have wider spectra of virulence on resistant genotypes than races collected in 1994. Afshari et al. (2006) reported seven pathotypes including five pathotypes of yellow rust, 14E176A+, 134E142A+, 6E210A+, 4E128A- and 64E146A+ in Iran between 2003 and 2004 and virulence wasn't detected for plants with genes Yr1, Yr4, Yr5 and Yr10 but virulence on plants with genes Yr2, Yr6, Yr7, Yr9, Yr22, Yr23 and YrA was common until 2005. The objectives of this study were to (i) Identify the frequencies of wheat strip rust races Khuzestan Province of Iran in 2009 and 2010 and (ii) determine the virulence factors of the wheat yellow rust pathogen.

2. MATERIALS AND METHODS

For determination of the virulence spectra of the isolates, differential host genotypes with known seedling resistance genes were used. These include entries from the European and world sets. A set of the world and European wheat yellow rust differentials as proposed by Johnson et al. (Johnson et al., 1972) was used for this study (Table 1).

Table 1. Infection types of *Puccinia striiformis* f. sp. *tritici* isolates on wheat

Description of infection	Index value
Immune/no symptom	0
Hypersensitive flecks without uredia	1
Necrotic/Chlorotic flecks without uredia	2
Necrotic/Chlorotic stripes without uredia	3
Necrotic/Chlorotic stripes with trace uredia	4
Necrotic/Chlorotic stripes with light uredia	5
Necrotic/Chlorotic stripes with intermediate uredia	6
Necrotic/Chlorotic stripes with moderate uredia	7
Chlorotic stripes with abundant uredia	8
Abundant uredia without necrosis/chlorosis	9

Ten additional cultivars–lines, Anza, Avocet 'R', Avocet 'S', Kalyansona, Federation 4/Kavkaz, Triticum Spelta Album, TP 981, TP 1295, Trident, and Bolani, were added, making a total of 26 cultivars–lines (Table 2). Under field conditions, spreader rows served as a source of inoculum. Primary infection by airborne rust spores developed rapidly on the mixture of susceptible cultivars, and then subsequent spread of urediniospores occurred naturally on the surrounding plots of the differential cultivars. The trap nursery was evaluated at all testing sites for two years. Severity of infection (0 to 100%) and reaction type (R, S) as designated by Peterson et al. (1948) were assessed at heading stage (Zadoks et al*.,* 1974). The frequency of infection of each genotype was calculated as the relative percent frequency of infection of susceptible lines over 2 years at all the testing sites in Khuzestan province of Iran.

Collection of stripe rust samples was carried out in the 2009 and 2010 growing season Khuzestan province of Iran. Uredospores from a single pustule were isolated and propagated on the susceptible cultivar Bolani for each collection. For inoculation, uredospores were mixed with talcum powder in the ratio 1:3, and sprayed on to seedlings using a fine mist atomizer. The objective of using a mixture of talcum powder and the uredospores was to help settling spores in a uniform manner on seedling leaves. After each inoculation, the spraying equipment was thoroughly washed in water and put in an oven with 60°C for 12 hours to avoid contamination when consecutive inoculations with different pathotypes were carried out. *P. striiformis*, inoculation rooms consisted of a trolley with a base tray containing 2 cm of tap water. After inoculation, seedlings were placed on the trolleys and covered with plastic hoods. Trolleys were placed in an incubation room at 10°C where the differential temperatures between the water and room temperature resulted in dew formation. Following incubation, plants were moved to greenhouse chambers capable of being set to a range of temperatures. The temperatures used 18°C with 16h/8h day/night. Virulence surveys of cereal rust fungi have traditionally used differential host genotypes that express resistance in the primary leaves of seedling plants (Kolmar, 1997).

Table 2. Host differential genotypes for *Puccinia striiformis*

Wheat differential genotypes–cultivars	Resistance genes
World differential set	
Chinese 166	Yr1
Lee	Yr7
Heines Kolben	Yr2,Yr6
Vilmorin 23	Yr3V
Moro	Yr10
Strubes Dickopf	YrSd
Suwon 92×Omar	YrSu
Clement	Yr2, Yr9+
European differential set	
Hybrid 46	Yr4
Reichersberg 42	Yr7+
Heines Peko	Yr2+, Yr6
Nord Desprez	Yr3N
Compair	Yr8
Carstens V	Yr32+
Spaldings prolific	YrSp
Heines VII	Yr2+
Supplemental differential set	
Anza	Yr18, YrA
Federation*4/Kavkaz	Yr9
Avocet ' R '	YrA
Avocet ' S '	-
Kalyansona	Yr2
Triticum Spelta Album	Yr5
Bolani	-
TP 1295	Yr25
Trident	Yr17
TP 981	-

The infection types (IT) of *P. striiformis* on the wheat genotypes were scored using a 0 to 9 scale 15-18 days after inoculation as described by MacNeal et al., (1971) and Stubbs (1988). They were classified into resistant (0-5) and susceptible (6-9) types. Infection types (ITs) 7 to 9 were regarded as virulent (susceptible) and less than seven was avirulent.. For *P. striiformis* the pathotype nomenclature of Johnson et al. (1972) was used. Uredospores of the pathogen were stored in aluminum foil packets placed in liquid nitrogen (-196°C) for further investigations.

3. RESULTS AND DISCUSSION

For a period of two consecutive years, leaf samples were collected from durum and bread wheat cultivars, as well as from differential cultivars grown under field conditions in Khuzestan province of Iran. Single-uredinial isolates of *P. striiformis* were characterized for physiologic races. The results obtained from this study showed that there were identified 27 physiologic races during the course of this study (Table 3).

Table 3. Yellow rust races identified in Khuzestan Province of Iran from 2009 to 2010

Physiologic race	Khuzestan Province							
	Ahvaz	Dezful	Shushtar	Ize	MasjdSleman	Abadan	Mahshahr	Behbehan
4E15	+	+	+	+	+	+		
4E14	+	+	+		+	+	+	
6E0	+	+	+	+	+	+	+	+
6E2	+		+					+
6E4			+				+	
6E6	+	+						
6E22	+							+
6E44		+	+					
6E78			+		+			
6E128	+	+	+	+				+
6E130	+	+		+	+			+
6E134	+	+	+					+
6E138			+					+
6E142	+	+			+		+	+
6E148	+	+	+		+		+	+
6E150	+	+	+		+		+	+
6E158	+	+	+		+			
6E174		+	+		+			
38E66	+		+		+			+
64E216			+					+
70E0			+					+
70E132			+					+
124E160	+	+	+					
132E48	+	+						
134E150	+	+	+					+
166E134			+					+
166E232	+	+			+			+

** Presence of physiologic race*

The composition of physiologic races found in 2009 and 2010 differed greatly on the world differential set but not as much on the European differential set. All *P. Striiformis* physiologic races identified in this study differed in their occurrence during a two years period. On the world differential set, the differential genes, *YrSU* and *Yr9+* in Suwon92 × Omar and Clement, respectively, allowed clear discrimination among the races in Khuzestan province of Iran. Virulence for these genes occurred in 2010 but not in 2009 (Table 3).

In the case of the European differential genotypes, virulence for the resistance genes *Yr7+, Yr6+, Yr2+*, and *Yr8,* and avirulence for *Yr4+, YrCV*, and *YrSP,* were observed in the 2009 and 2010 *P. striiformis* populations. The world differential did not allow distinction between four races 4E14, 4E15,6E0, 6E128 in 2009 and two races 6E44 and 6E148 in 2010, whereas European differentials showed differential reactions on *Yr7+, Yr6*+, and *Yr8* resistance genes (Table 4) for these races. Thus, the use of both differentials allowed for better discrimination between yellow rust races. Four distinct physiologic races were identified in 2010 compared with seven races in 2009. Races identified in 2010, such as 4E15, 6E44 and 70E32, have wider spectra of virulence for yellow rust resistance genes than the races identified in 2009, such as 4E14, 4E15, and 6E0. In 2010, virulence for the *YrSu* resistance gene was the major virulence change recorded in Khuzestan compared with the results from 2009. Physiologic race 6E6 was first observed in the region in 1972 (Bamdadian, 1972). Significant changes in race composition were identified using the world and European differential sets (Table 4).

In this study, 6E6 have virulence on 10 known genes in the host plants and this race is virulent for *Yr2, Yr6, Yr7, Yr2, Yr6+, 7+, 17, 18* and *Yr*A. 6E6 and is present in many wheat cultivars that are still cultivated over relatively large areas in Iran. Race 6E148 and 6E158 were the second most frequent race in Iran and was found for five locations in years, whereas 6E142, 6E130 and 6E128 were identified for four locations, respectively (Table 3). Races 6E0 and 4E15 are among the most virulent races identified and could be among the *P. striiformis* races that contributed most to yellow rust epidemics in this region. This race has a broad virulence spectrum and was recovered during four consecutive years (1994 to 1997) in Iran (Nazari, 1998; Torabi et al., 1995). In Iran, only three physiologic races 6E0, 6E2, 6E4 occurred for four consecutive years in the early 1990's and races 6E134 and 6E150 were also found only once in 1997 and 1996, respectively (Nazari, 1998).

Among the races that occurred only once during the period of the study, races 4E14, 4E15 6E22, 6E128, 6E130, 6E148 6E134, 6E142, 6E158, 134E150, and 166E232 were common in all locations and observed during the course of the study could be attributed to changes in bread wheat varieties being cultivated over large areas, to the extension of resistant durum wheat varieties to irrigated areas of Iran, to monoculture of limited numbers of bread wheat varieties, or simply to limited sampling locations. According to the results, virulence on plants with gene/s *Yr2, Yr6, Yr7, Yr9, Yr3N, Yr2, Yr6+, Yr2, Yr9+* and *YrA* was detected (Table 4). The majority of isolates with high frequency showed virulence on plants with *Yr2, Yr6, Yr7, Yr9, Yr25* and *YrA* genes. No virulence was detected on plants with *Yr1, Yr3V, Yr4, Yr5, Yr10* and *YrSU* genes.

The presence of virulence on plants with the genes *Yr3N, Yr8, Yr32+, YrSD* and *YrSP* in the seedling test and absent in the four regions at the adult plant stage, could be due to the low frequency of virulence for those genes under field conditions. Special leaf collections were made from selected differential genotypes associated with resistance genes, some of which are being exploited in many wheat varieties, and from land race bread wheat cultivars.

Table 4. Reaction of host differential genotypes to Khuzestan yellow rust physiologic races in 2009 and 2010

Differential genotype	Yr gene*	Physiologic races** and reaction* of differential sets										
		2009				2010						
		4E14	4E15	6E0	134E150	6E6	6E148	6E158	6E130	70E0	70E132	166E232
World differential set												
Chinese 166	*Yr1*	1	0	0	0	0	1	1	1	0	0	1
Lee	*Yr7*	8	6	6	0	7	7	7	8	8	9	0
Heines Kolben	*Yr2, Yr6*	8	7	9	8	7	8	8	8	7	8	8
Vilmorin 23	*Yr3V*	2	3	2	2	2	2	0	1	2	3	1
Moro	*Yr10*	2	3	0	4	3	2	0	2	0	3	1
Strubs Dikkopf	*YrSd*	2	3	1	3	3	7	1	0	6	7	1
Suwon 92m/Omar	*YrSu*	2	2	1	3	3	3	0	0	1	6	6
Clement	*Yr2, Yr9+*	3	3	2	2	2	2	0	1	2	2	0
European differentials set												
Hybrid 46	*Yr4*	2	1	0	1	1	0	1	1	1	0	0
Reichersberg 42	*Yr7+*	8	1	1	1	8	7	1	8	7	8	6
Heines Peko	*Yr2, Yr6+*	8	6	1	7	8	8	2	0	7	7	2
Nord Desprez	*Yr3N*	2	7	0	4	7	6	4	2	2	4	2
Compair	*Yr8*	3	3	0	8	7	8	2	6	2	1	1
Carstens V	*Yr32+*	2	7	2	2	2	2	1	1	2	3	0
Spalding prolific	*YrSp*	1	3	4	0	1	0	2	1	0	0	0
Heines VII	*Yr2+*	7	1	8	7	6	8	2	1	0	9	8
Supplemental differential cultivars												
Federation*4/Kavkaz	*Yr9*	7	8	8	8	9	8	7	7	7	9	9
Anza	*Yr18, YrA*	8	8	8	9	9	9	9	7	8	9	8
Avocet ' R '	*YrA*	8	9	8	9	8	7	9	6	8	9	6
Kalyansona	*Yr2*	9	6	7	9	7	8	8	9	9	7	6
T. spelta. var. album	*Yr5*	0	1	1	2	1	1	1	1	0	1	1
TP1295	*Yr25*	9	8	6	7	7	8	8	8	6	7	7
Federation	*Yr17*	9	8	8	7	7	7	8	9	9	7	7

**Yr = yellow rust resistance genes; **Physiologic races identified in Khuzestan province; ***Disease reaction scale 0 to 9.*

The selected lines were exposed in trap nurseries to natural yellow rust infection in Ahvaz, Dezful, Shoushtar, Abadan, Masjedsoleyman, Eze, Mahshar and Behbehan during the same period of the study. One single isolate was tested from each sample and analyzed for race identification. According to the results, virulence on Heines Kolben (with genes *Yr2* and *Yr6*), Kalyansona (*Yr2*), Lee (*Yr7*), Avocet R (*YrA*), Federation* 4/Kavkaz (*Yr9*), TP1295 (*Yr25*) and Nord despres (Yr3N) was common during the period of investigation (Table 6).

Virulence for *Yr1* which is common in Central Asia and China (Anmin et al., 2004), and in most of the Middle East and in north-western Europe were absent in Iran (Afshari et al., 2004; Hovmoller et al., 2007). No virulence was observed on plants with *Yr1, Yr3V, Yr4, Yr5, Yr10, Yr25, Yr32+, YrSP* and *YrSD genes* in the trap nurseries. The virulence frequency for the *Yr2, Yr6* genes were between 8-16 with a moderately susceptible reaction in Heines peko when for *Yr2* in Heines Kolben was calculated to be 100 (Table 5). The frequency of virulence factors to *Yr* resistance genes (Table 6) was determined based on infection of resistant cultivars under field conditions in Iran. The frequency of virulence factors in the yellow rust population on the differential genotypes tested in the trap nurseries were above 91% for the resistance genes *Yr2,Yr6, Yr7, Yr2, Yr6+, Yr18,YrA, Yr2*, *Yr17*, *Yr25* and *YrA* however, virulence frequencies for *Yr7+* and *Yr8* were less than 10%. frequency of virulence factors to all other lines were between 25 and 70% (Table 6).

Rabaninasab et al., (2008) noted that virulence wasn't detected for plants with genes Yr1, Yr4, Yr5 and Yr10 and virulence on plants with genes *Yr2, Yr6, Yr7, Yr9, Yr22, Yr23* and *YrA* was common in Iran. For instance, virulence to *Yr2, Yr6, Yr7* and *Yr9* occur in most wheat producing areas of the world (Chen et al. 2002). In Pakestan virulence has been reported to Yr genes *Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr17, YrCv, YrSu* and *YrSd* (Sumaira, 2009). *Yr1, Yr5, YrCv, Yr7, Yr17* and *YrSp* stripe rust resistance genes were found effective against all isolates collected across the major wheat growing regions of Iran. *Yr4* showed high resistance against all or most of the yellow rust isolates. The findings were in agreement with the work of Chen (2005), Afsharri (2008) and Chunmei et al. (2008) who reported that virulences to *Yr4* gene rarely occur in most wheat producing areas of the world as a rare phenomenon. Of this virulence, pathotypes possessing the combination of virulence for plants with *Yr7* and *Yr9* were particularly implicated in the epidemics on Falat cultivar in 2003 in Iran, because this combination overcame the resistance of Seri 82 and the many derivatives of that which were widely grown in West Asia and North Africa (WANA), (Elahinia, 2005). Bread wheat cultivars Seri 82, Falat (in Iran), Mexipac (in Syria) and Gereck (in Turkey) were resistant to the stripe rust populations when initially released. Within a few years of release the corresponding stripe rust virulence genes increased and the resistance genes such as *Yr9* associated with the above cultivars, became ineffective (Torabi et al., 1995; Yahyaoui et al., 2004). The composition of *P. Striiformis* populations could change over time and this can be an important consideration for breeding programs.

The most recently deployed resistance genes *Yr18* and *Yr27* in several bread wheat cultivars cultivated in CWANA are becoming ineffective against stripe rust pathotypes (Singh et al., 2004). Diverse virulence phenotypes of *P. striiformis* exist under natural field conditions, and some virulence types could eventually develop and spread over larger areas in the region. Surveys of pathogen populations and the genetic characterization of virulence genes continue to provide valuable information used to design breeding strategies and prioritize which pathogen species and physiologic races to target. *P. striiformis* populations in Iran are genetically diverse, and differences in virulence have been identified. In this study, we showed that composition of *P. striiformis* populations changed over time, and this can be an important consideration for wheat breeding programs in the region.

Table 5. Virulence frequencies of *Puccinia striiformis* f. sp. *tritici* isolates collected in 2009 and 2010 from Khuzestan provinces of Iran

Yr gene	Virulence frequency (%)							
	Ahvaz		Dezful		Izeh		Behbahan	
	2009	2010	2009	2010	2009	2010	2009	2010
Yr1	0	0	0	0	0	0	0	0
Yr7	60	70	60	80	80	100	80	100
Yr2, Yr6	100	100	100	100	100	100	100	100
Yr3V	0	0	0	0	0	0	0	0
Yr10	4	4	8	1	0	0	0	0
YrSd	0	0	2	2	2	1	0	0
YrSu	24	48	8	20	0	0	8	20
Yr2, Yr9+	0	0	0	8	1	0	0	0
Yr4	0	0	0	1	1	1	0	2
Yr7+	0	0	0	0	0	0	0	0
Yr2, Yr6+	12	14	8	20	6	16	12	16
Yr3N	70	64	64	64	45	8	8	64
Yr8, Yr18	0	1	2	0	0	0	0	12
Yr32+	0	0	0	2	2	1	0	0
YrSp	0	0	0	1	0	0	0	0
Yr2+	1	0	4	8	8	8	12	20
Yr9	60	64	70	80	24	24	40	50
Yr18, YrA	24	8	24	24	12	8	8	16
YrA	100	100	100	100	80	100	100	100
Yr2	90	40	40	90	80	90	60	80
Yr5	0	0	0	0	0	0	0	0
Yr25	90	90	80	90	90	100	100	100
Yr17	0	0	0	0	0	0	0	0

Table 6. Frequencies of virulence factors of wheat strip rust pathogen in Khuzestan province of Iran

Differential genotype	*Yr gene*	% of Frequencies of virulence factors
Chinese 166	*Yr1*	0
Lee	*Yr7*	91.1
Heines Kolben	*Yr2, Yr6*	100
Vilmorin 23	*Yr3V*	0
Moro	*Yr10*	0
Strubs Dikkopf	*YrSd*	25
Suwon 92m×Omar	*YrSu*	0
Clement	*Yr2, Yr9+*	42
Hybrid 46	*Yr4*	0
Reichersberg 42	*Yr7+*	8
Heines Peko	*Yr2, Yr6+*	71
Nord Desprez	*Yr3N*	37
Compair	*Yr8*	8
Carstens V	*Yr32+*	57
Spalding prolific	*YrSp*	0
Heines VII	*Yr2+*	48
Federation*4/Kavkaz	*Yr9*	97
Anza	*Yr18, YrA*	100
Avocet ' R '	*YrA*	100
Kalyansona	*Yr2*	100
T. spelta. var. album	*Yr5*	0
TP1295	*Yr25*	82
Federation	*Yr17*	85

4. CONCLUSION

Despite frequent stripe rust epidemics in Khuzestan Province of Iran, there have been not enough studies on the genetic basis of resistant wheat cultivars and the structures of pathogen population. In the present study genetic structure of wheat cultivars and *P. striiformis* population that naturally occur in the major wheat growing regions of Iran were analyzed and results were discussed in the context of wheat breeding to control stripe rust in the country.

Virulence and diversity of the Iranian stripe rust population were studied using differential genotypes with known *Yr* genes. There were marked differences in the composition of individual sub-populations in the four tested regions of Iran. Out of the 27 physiological races, only nine of them were common in all regions. This regional variation of the pathotypes might indicate that wheat cultivars could have been developed and released for production on a regional basis. Virulent pathotypes have been already observed to most of the *Yr* genes used in Iranian wheat cultivars. Thus, searching for new effective sources of

stripe rust resistance genes is necessary to cope with changes in the host-pathogen interaction.

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