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Prevalent Pathotypes of *Puccinia striiformis* f.sp. *tritici* in Iran

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ABSTRACT

The stripe (Yellow) rust in wheat is one of the most important plant diseases in Iran. Since 1993 several epidemics have occurred in Iran causing the breakdown of widely utilized sources of resistance in wheat cultivars. Twenty-seven pathotypes were identified during 2003 and 2004 in greenhouse tests. Pathotypes 6E6A+, 6E22A+, 6E130A+, 6E134A+, 6E142A+, 6E158A+, 134E130A+ and 134E142A+ were more common during the course of this study. Virulence on plant/s with gene/s *Yr2, Yr6, Yr7, Yr8, Yr9, Yr24, Yr25, YrSD, YrSP, Yr3N, Yr2+, Yr6+, Yr9+, Yr7+, Yr32+* and *YrA* was detected under greenhouse conditions. The majority of isolates with a high frequency (more than 88%) showed virulence on plant/s with *Yr2, Yr6, Yr7, Yr9, YrA* and *Yr24* genes. No virulence was detected on plant/s with *Yr1, Yr3V, Yr4, Yr5, Yr10* and *YrSU* genes. In a greenhouse test, frequency of virulence to wheat genotypes with the *Yr32+, YrSP* and *YrSD* gene was less than 7%; frequency of virulence to all other wheat genotypes was between 19 and 100%. During three years of field study, virulence on wheat genotypes Heines Kolben (with genes *Yr2* and *Yr6*), Kalyansona (*Yr2*), Lee (*Yr7*), Avocet R (*YrA*), Federation*4/Kavkaz (*Yr9*) and TP1295 (*Yr25*) was common. No virulence was observed on plants with *Yr1, Yr3V, Yr3N, Yr4, Yr5, Yr8, Yr10, Yr18, Yr24, Yr32+, YrSP, YrSD* and *YrSU* genes in the trap nurseries. The coefficient of infection (C. I.) of the adult plant resistance gene, *Yr18*, was between 16-64 with moderate susceptibility and is going to be used in the breeding program in combination with other resistant sources.

Keywords: Pathotypes, *Puccinia striiformis*, Resistance genes, Wheat.

INTRODUCTION

Wherever wheat is grown, one or more of the rust diseases is capable of causing significant losses. Stripe rust in wheat, caused by *Puccinia striiformis* f.sp. *tritici* (*Pst*), is an important disease, particularly under cool conditions. *P. srtiifomis,* which has a macrocyclic life cycle and unknown alternate host, is an important disease in the cooler wheat growing areas of Iran. Where they occur the alternate hosts may be important in disease epidemiology, in providing the primary inoculum to initiate early rust development and as a source of new pathotypes generated by sexual reproduction.

Macer (1972) noted that stripe rust was important in cooler parts of Yugoslavia, Egypt, Turkey and Iran. He also suggested some adaptation of the pathogen to higher temperatures within the investigation areas. Mundy (1973) reported the breakdown of adult plant resistance to stripe rust in the winter wheat cultivar Joss Cambier in the U. K. resulting in a yield loss of 34 percent. Stripe rust in wheat was first detected in Australia in 1979 (O'Brien *et al.*,1980). Initial losses of 50-60% were reported in susceptible cultivars in Australia by McIntosh (1979). The single original pathotype was identified in Australia as being similar to one present in Europe, suggesting that it had

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been man-borne perhaps on clothing or other personal goods (Wellings *et al.,* 1987). The annual value of control of losses due to stripe rust in Australia was estimated by Brennan and Murray (1988) to be 139 million Australian dollars. 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).

Stripe rust uredospores can be wind-borne in a viable state for more than 800 Km, (Zadoks, 1961). In 1980, the pathotype first found in Australia appeared in New Zealand presumably having been air-borne from Australia, a distance of approximately 2,000 Km, (Beresford, 1982). McIntosh (1992) noted the possible effects of common evolutionary forces, *viz.* migration, mutation, asexual recombination, selection and chance in influencing gene frequencies and determining evolutionary pathways in cereal rust pathogens.

For convenience, resistance to rust diseases can be divided into two categories; firstly the early growth stage (seedling) resistance and, secondly, the later growth stage or adult plant resistance (APR) (Knott, 1989). Wellings (1986) reported that, due to the variability of the pathogen, some cultivars resistant to stripe rust in one region were susceptible in another region.

Johnson *et al.* (1972) suggested a differential set and a new system for pathotype nomenclature based on the use of binary codes. The differential sets comprised a "world set" of seven genotypes previously known to distinguish variation in response over a wide geographical area. A second set of eight "European" differentials was considered suitable for the European regions. These differentials and the nomenclature system were widely adopted, not only in Europe but also elsewhere, including in Australia and Iran.

Seven pathotypes, *viz.* 6E0, 20E148, 38E134, 166E150, 6E20, 134E150 and 230E150 were reported in Syria and Lebenon between 1993 and 1994 (Yahyaoui *et al.*, 2001). In Iran, 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 2001 (Torabi *et al.*, 2001). This study was carried out to determine virulence factors and prevalent of stripe rust pathotypes in greenhouse and field conditions.

MATERIALS AND METHODS

In this study, stripe rust populations in Iran were characterized for their virulence using two methods. In the first method the reaction of seedlings of differential genotypes was evaluated in greenhouse for two years and in second method adult plant reaction in the field condition tested for three years.

Sample Collection and Maintenance of Pathogen

A differential set of wheat stripe rust as proposed by Johnson *et al.* (1972) was used in this study. Ten supplementary lines and cultivars were also used as differentials (Table 1). A collection of stripe rust infected leave was obtained from commercial wheat cultivars from different parts of Iran. Forty-three collections were purified and propagated on the wheat seedlings of susceptible wheat cultivar Bolani. Uredospores from a single pustule were isolated and propagated on the susceptible cultivar Bolani for each collection. Uredospores of the pathogen were stored in aluminium foil packets placed in liquid nitrogen (-196°C) for further investigations.

Inoculation

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

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Table 1. Differential wheats used to detect pathotypes of *Puccinia striiformis* f. sp. *tritici*.

Differential [a]	Resistance gene	Decanary value
World differential set		
Chinese 166	*Yr1*	1
Lee	*Yr7*	2
Heines Kolben	*Yr2,Yr6*	4
Vilmorin 23	*Yr3V*	8
Moro	*Yr10*	16
Strubes Dickkopf	*YrSD*	32
Suwon 92/OMAR	*YrSU*	64
Clement	*Yr2, Yr9+*	128
European differential set		
Hybrid 46	*Yr4*	1
Reichersberg 42	*Yr7+*	2
Heines Peko	*Yr2,Yr6+*	4
Nord Desprez	*Yr3N*	8
Compare	*Yr8*	16
Carstens V	*Yr32+*	32
Spaldings prolific	*YrSP*	64
Heines VII	Yr2+	128
Supplemental differential set		
Federation*4/Kavkaz	*Yr9*	
Anza	*YrA*	
Avocet 'R'	*YrA*	
Avocet 'S'		
Kalyansona	*Yr2*	
Triticum Spelta Album	*Yr5*	
TP 981		
TP 1295	*Yr25*	
Meering+*Yr24*	*Yr24*	
Bolani (Susceptible check)		

[a] The differential sets were obtained from the Plant Breeding Institute, University of Sydney.

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.

Infection Assessment at the Seedling Stage

Infection types were assessed on a 0-9 scale 16 and 18 days after inoculation using a scale similar to that described by McNeal *et al.* (1971), with modifications made by Wellings (1986). Infection types (ITs) 7 to 9 were regarded as virulent (susceptible) and less than seven was avirulent. Pathotypes avirulent and virulent on selection Avocet R were described as A- and A+, respectively (Wellings and McIntosh, 1990). For *Pst* the pathotype nomenclature of Johnson *et al.* (1972) was used. The symbols plus ("+") and minus ("-") were used to denote a greater or lesser development of symptoms,

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Table 2: Reaction of wheat stripe rust differential sets to stripe rust and pathotype identification in Iran.[a]

			World differential set								European differential set								Supplemental differential set									
			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
Ent. No.	Location	Pathotype	Chinese 166	Lee	Heines Kolben	Vilmorin 23	Moro	Strubes Dickkope	Suwon 92/Omar	Clement	Hybrid 46	Reichersberg 42	Heines Peko	Nord Desprez	Compare	Carstens V	Spaldings Prolific	Heines VII	Fed.*4/Kavkaz	Anza	Avocet 'R'	AVOCET 'S'	Kalyansona	T.Spelta Var. Album	TP 981	TP 1295	Meering + Yr24	Bolani
1	Gonbad	4E0A+	0	0	7	0	0	0	0;	0;	0;	0;	0;	0;	0	0	;C	0	9	8	8	8	8	0	0	0	7	7
2	Joyem Lar	4E4A+	0	0	8	0	0	0	0	0	0	0	7	0	0	2+CN	0	0	8	8	8	8	8	0	0	0	7	7
3	Maneh.1	4E8A+	0	CN	8	3	0	0	0	1C	0	;C	0	8	0	5	2CN	0	9	9	9	9	9	0	9	9	9	9
4	Sari	6E0A+	0	7	7	;CCN	0	3+CN	0	0	0	5+C	4+CN	4+CN	0	0;CN	0	0	9	8	8	7	7	0	9	7	7	9
5	Islam Abad.1	6E2A+	0	8	8	;CN	0	6+CN	0	4+CN	0	9	5+CN	3+CN	0	4+CN	0	5+CN	9	9	9	9	9	0	9	9	6C	9
6	Garakhil.1	6E4A+	0	7	7	0	0	0	0	0	0	0	7	0	0	0	0	0	7	7	7	7	7	0	7	7	7	7
7	Garakhil.2	6E6A+	0	8	8	4CN	0	4CN	4+CN	4CN	0	8	8	0	0	3+CN	0	5C	8	8	8	8	8	0	0	7	8	7
8	Malayer	6E6A+	0	7	7	;CN	0	3C	0	0	0	7	7	;CN	0	;1C	0	0	7	7	7	7	7	0	0	0	7	7
9	Araghimahaleh	6E6A+	0	7	7	0	0	0	0	0	0	7+	7	0	0	0	0	0	8	7	7	7	7	0	0	0	0	8
10	Darab.1	6E6A+	0	8	8	0	;0	0	0	5C	;0	8	8	0	0	3CN	0	5+C	8	8	9	8	8	0	8	7	8	8
11	Hamadan	6E22A+	0	8	8	3+CN	0	4+CN	;0	;1+CN	0C	9	9	4+CN	7	0C	0C	4+CN	9	9	9	9	7	0	7	7	7	9
12	Zargan	6E22A+	0	7	7	4+CN	;CN	4+CN	4+CN	4CNN	;0	8	8	4+CN	7	4+CN	0	6C	8	8	8	8	8	0	8	8	8	8
13	Darab.2	6E44A+	;0	8	8	;	0	0	0	4CN	0	6+CN	7	7	0	7	0	6C	9	8	9	9	9	0	9	9	9	9
14	Gorgan	6E78A+	;	7	7	4	0	0	4+	0;	0	7	8	8	0	4	7	3+CN	0;	7	7	7	9	0;	6+	7	7	7
15	Islam Abad.2	6E128A+	;0	7	7	0	0	0	0	;1+	0	;1+	;0	0	0	0	0	7	7	7	7	7	7	0	0	0	8	8
16	Gazvin.1	6E130A+	0	8	8	4CN	0	0	0	0	;CN	8	6C	0	0	;CN	0	8	8	8	8	8	8	0	8	8	8	8
17	Gazvin.2	6E130A+	0	8	8	4CN	0	0	0	0	;CN	8	6C	0	0	;CN	0	8	8	7	8	8	8	0	8	8	8	8
18	Ahvza.1	6E134A+	;0	8	8	3+CN	0	5+CN	0	0	0	9	8	5+CN	4+CN	2+CN	0	7	9	9	9	9	9	0	9	9	7	9
19	Dezful.1	6E134A+	0	7	7	0;CN	;1+C	5+C	0;	;3+CN	;0	8	7	4+CN	;3+CN	;0	0	7	8	8	8	8	8	0	8	8	8	9

Table 2: (Continue)

			World differential set								European differential set								Supplemental differential set									
Ent. No.	Location	Pathotype	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
20	Bojnord.1	6E134A+	0	8	8	4+CN	;0	5CN	3+C	4CN	;CN	8	7	5CN	2+CN	5CN	0	7	8	8	8	8	8	0	7	8	8	9
21	Dezful.2	6E134A+	0	7	7	;0CN	;1+C	5+C	;0	3+CN	;0	8	7	4+CN	3+CN	;0	0	7	8	8	8	8	8	0	8	8	8	9
22	Ahvza.2	6E134A+	;0	8	8	3+CN	0	5+CN	0	0	0	9	7+	5+CN	4+CN	2+CN	0	7	9	9	9	9	9	0	9	9	7	9
23	Mogan.1	6E138A+	0	8	8	3+CN	0	;CN	0	4+CN	;CN	9	6+C	7	0	3+CN	0	7	8	8	8	8	8	0	7	8	7	9
24	Karaj	6E142A+	0	8	8	3+CN	0	;CN	0	4+CN	;CN	9	7	7	0	3+CN	0	7	8	8	8	8	8	0	6C	8	7	9
25	Ahvza.3	6E142A+	0	7	7	0	0	0	0	0	0	8	8	8	6C	;2CN	0	8	8	8	9	9	7	0	0	7	9	9
26	Ahvza.4	6E142A+	0	7	7	;2-cn	0	0	0	0	0	8	7+	7+	0C	0;C	0	7	8	8	8	8	8	;0	7	7	7	7
27	Lar.1	6E142A+	0	7	7	3+CN	0	1+CN	0	6C	0	7	7	7	0	0	0	7	8	7	7	7	7	0	7	7	7	7
28	Mogan.2	6E142A+	0	7	7	0	0	0	0	0	0	7	8	7	5CN	0	0	7	8	7	7	8	7	0	7	8	7	8
29	Bye Kola	6E148A+	0	7	7	4	0	0	4	;C	;0	;01+	7	0	7	;3+	0	7	8	7	7	7	7	0	7	7	8	8
30	Mogan.3	6E150A+	;0	8	8	;CN	0;	;CN	0	;CN	0;	8	8	;2+CN	7+	;CN	;	8	9	7	8	8	8	0;	8	8	6CN	9
31	Mogan.4	6E158A+	0	7	7	2+CN	0	5+C	0	4+CN	;CN	7	7	7	7	3+CN	0;	7	8	8	8	8	8	0	8	8	8	9
32	Mogan.5	6E158A+	0	7	7	2+CN	0	5+C	0	4+CN	;CN	7	7	7	7	3+CN	0;	7	8	8	8	8	8	0	8	8	8	9
33	Garakhil.3	6E174A+	0	7	7	5CN	0	0	0	5+	0	8	8	7	6+C	7	0	7	8	7	7	8	8	0	8	8	8	8
34	Bojnord.2	38E66A+	0	7	7	3CN	;C	7	;C	;C	;C	7	3+CN	0	0;C	2CN	7	2+CN	7	7	7	7	7	0	7	0	0	7
35	Bojnord.3	134E4A+	0	7	7	;C	0	;C	0	7	0	0;	7	0	0	0	0	3+	9	9	9	9	9	0	9	9	9	9
36	Mashhad	134E6A+	0	7	7	;	0;C	0C	0	7	0	7	7	0	0	0	0	0	7	9	9	9	9	0	9	9	0	8
37	Yazd.1	134E130A+	;0	8	8	3+CN	0;	0;	0;	8	0	8	5+CC	4+CC	5+CC	;CC	0;	7	9	8	7	7	7	;0	8	8	8	8
38	Yazd.2	134E130A+	0	7	7	0	0	3+CN	0	7	0	7	0	0	3+	0;	0;	7	8	8	8	8	7	0	7	8	7	8
39	Yazd.3	134E142A+	0	7	7	0	0	0	0	7	0	7	7	7	0	0	0	7	7	7	7	7	7	0	7	7	7	7
40	Torogh	134E142A+	0	7	7	3+CN	0;	4+CN	0	8	0	8	8	7	5+C	2+CN	0	7	8	8	8	7	8	0	4+C	7	7	7
41	Maneh.2	134E150A+	0	8	7	0	;1	2+	0	7	0	7	7	0	7	1CN	0	7	8	8	8	8	8	0	8	8	7	8
42	Gachsaran	166E30A+	0	7	8	2+C	0	8	1+CN	7	0	8	8	7	7	3+CN	0	;C	8	8	9	9	9	0	9	9	9	9
43	Lar.2	166E134A+	0	7	8	4+CN	0	8	0	7	;CN	8	8	6CN	;CN	2+CN	0	8	8	8	8	9	9	0	9	9	9	9

[a] ;= Necrotic flecks (very resistant), 0= No visible uredia (Immune), C= Chlorotic, N= Necrotic, 1 to 6= Resistant to moderately resistant, 7 to 9= Moderately susceptible to susceptible.

respectively, relative to the infection type scale, whereas the symbols "C" and "N" emphasized more than normal levels of chlorosis and necrosis, respectively.

Infection Assessment at the Adult Plant Stage

Field evaluations were performed as a trap nursery at four sites in Iran: Ardebil (Northwest), Karaj (North), Mashhad (Northeast) and Zargan (Center) during a three-year cropping season in 2001-2, 2002-3 and 2003-4. These sites represent the major wheat growing areas of Iran. Each differential line/cultivar was planted in two-meter rows and 30 cm apart. Due to a lower chance of stripe rust development yearly, the plants at Karaj were inoculated at tillering and flag leaf growth stages with urediniospores collected and increased from an infected field, which were determined to contain pathotype 134E134A+ and, for the other three sites, nurseries were naturally infected with stripe rust. In the flag leaf stage when the infection and severity of infection on a susceptible control was high, field assessments were done on disease severity according to the modified Cobb scale by Peterson *et al.* (1948) and on disease reaction based on the Roelfs (1978) method. The coefficient of infection (C. I.) was obtained by multiplying the severity by a constant for host responses.

RESULTS AND DISCUSSION

Stripe rust is the most serious disease of wheat in Central and Western Asia and North Africa (CWANA), including Iran. The development of a resistant cultivar is the most effective, safe and economic method of control. However, stripe rust pathogenic variation remains the underlying cause of this elusive rust resistance. Genetic variation in the stripe rust pathogen is continuously evolving in CWANA by using trap nursery stripe rust network.

In greenhouse tests of the 43 collections in Iran during 2003-2004, 27 pathotypes were identified (Table 2). The stripe rust population in the region consists of a number of pathotypes that differ in their pathogenicity toward the host plant. Some pathotypes such as 4E0A+ (Gonbad) and 4E4A+ (Joyem Lar) can attack 5 and 6 resistance genes of the host plant, respectively (Table 3). Other pathotypes such as 166E134A+ (Lar.2) and 166E30A+ (Gachsaran) have virulence on 12 and 13 known genes in the host plants, respectively (Table 2 and Table 3). According to the results, virulence on plants with gene/s *Yr2, Yr6, Yr7, Yr8, Yr9, Yr24, Yr25, YrSD, YrSP, Yr3N, Yr2+, Yr6+, Yr9+, Yr7+, Yr32+* and *YrA* was detected. The majority of isolates with high frequency (more than 88%) showed virulence on plants with *Yr2, Yr6, Yr7, Yr9, YrA* and *Yr24* genes (Figure 1). No virulence was detected on plants with *Yr1, Yr3V, Yr4, Yr5, Yr10* and *YrSU* genes. Torabi *et al.* (2001) 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. 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 1993 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), (Torabi *et al.,* 1995). Hakim *et al.* (2002) reported that the Iranian stripe (yellow) rust pathotypes do not differ in their pathogenicity from those found in Syria and Lebanon. Yahyaoui *et al.* (2001) reported seven pathotypes including 6E0, 20E148, 38E134, 166E150, 6E20, 134E150 and 230E150 in Syria and Lebanon between 1993 and 1994. Pathotype 134E150 has been detected in Iran too. Otherwise the pattern of virulence factors of Syrian and Lebanese pathotypes with virulence on plants with the genes *Yr2, Yr6, Yr7, Yr9* and *YrA* is almost similar to the Iranian pathotypes. In addition, more diverse pathotypes could be identified which include compatibility with *Yr1, Yr3V, Yr5, Yr10* and *YrSU*

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Table 3. Stripe rust pathotypes and their virulence factors detected in Iran.

No	Pathotype	Virulence factors on World and European wheat differential sets	Virulence factors on supplemental set	No. of *vir.* factors
1	4E0A+	*Yr2, Yr6*	YrA; Yr2; Yr9; Yr24	5
2	4E4A+	*Yr2; Yr6; Yr2, Yr6+*	*YrA; Yr2; Yr9; Yr24*	6
3	4E8A+	*Yr2; Yr6, Yr3N*	*YrA; Yr2; Yr9; Yr24; Yr25*	7
4	6E0A+	*Yr2; Yr6, Yr7*	*YrA; Yr2; Yr9; Yr24; Yr25*	7
5	6E2A+	*Yr2; Yr6; Yr7, Yr7+*	*YrA; Yr2; Yr9; Yr25*	6 (7)[a]
6	6E4A+	*Yr2; Yr6; Yr7; Yr2, Yr6+*	*YrA; Yr2; Yr9; Yr24; Yr25*	7 (8)
7	6E6A+	*Yr2; Yr6; Yr7; Yr7+; Yr2, Yr6+*	*YrA; Yr2; Yr9; Yr25; Yr24*[b]	6-7(8-9)
8	6E22A+	*Yr2; Yr6; Yr7; Yr7+; Yr2; Yr6+, Yr8*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (10)
9	6E44A+	*Yr2; Yr6; Yr7; Yr7+; Yr2, Yr6+; Yr3N, Yr32+*	*YrA; Yr2; Yr9; Yr24; Yr25*	9 (12)
10	6E78A+	*Yr2; Yr6; Yr7; Yr7+; Yr2, Yr6+; Yr3N, YrSP*	*YrA; Yr2; Yr24; Yr25*	8 (10)
11	6E128A+	*Yr2; Yr6; Yr7, Yr2+*	*YrA; Yr2; Yr9; Yr24*	6 (7)
12	6E130A+	*Yr2; Yr6; Yr7; Yr7+, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	7 (9)
13	6E134A+	*Yr2; Yr6; Yr7; Yr7+; Yr2, Yr6+, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	7 (10)
14	6E138A+	*Yr2; Yr6; Yr7; Yr7+; Yr3N, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (10)
15	6E142A+	*Yr2; Yr6; Yr7; Yr7+; Yr2, Yr6+; Yr3N, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (11)
16	6E148A+	*Yr2; Yr6; Yr7; Yr2, Yr6+; Yr8, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (10)
17	6E150A+	*Yr2, Yr6; Yr7; Yr7+; Yr2, Yr6+; Yr8; Yr2+*	*YrA; Yr2; Yr9; Yr25*	7 (10)
18	6E158A+	*Yr2, Yr6; Yr7; Yr7+; Yr2, Yr6+; Yr3N; Yr8; Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	9 (12)
19	6E174A+	*Yr2; Yr6; Yr7; Yr7+; Yr2; Yr6+; Yr3N; Yr32+, Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	9 (13)
20	38E66A+	*Yr2; Yr6; Yr7; YrSD; Yr7+, YrSP*	*YrA; Yr2; Yr9*	7 (8)
21	134E4A+	*Yr2; Yr6; Yr7; Yr2, Yr9+; Yr2, Yr6+*	*YrA; Yr2; Yr9; Yr24; Yr25*	7 (9)
22	134E6A+	*Yr2, Yr6; Yr7; Yr2, Yr9+; Yr7+; Yr2, Yr6+*	*YrA; Yr2; Yr9; Yr25*	6 (9)
23	134E130A+	*Yr2, Yr6; Yr7; Yr2, Yr9+; Yr7+; Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	7 (10)
24	134E142A+	*Yr2, Yr6; Yr7; Yr2, Yr9+; Yr7+; Yr2, Yr6+; Yr3N; Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (12)
25	134E150A+	*Yr2, Yr6; Yr7; Yr2, Yr9+; Yr7+; Yr2, Yr6+; Yr8; Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (12)
26	166E30A+	*Yr2, Yr6; Yr7; YrSD; Yr2, Yr9+; Yr7+; Yr2, Yr6+; Yr3N; Yr8*	*YrA; Yr2; Yr9; Yr24; Yr25*	10 (13)
27	166E134A+	*Yr2, Yr6; Yr7; YrSD; Yr2, Yr9+; Yr7+; Yr2, Yr6+; Yr2+*	*YrA; Yr2; Yr9; Yr24; Yr25*	8 (12)

[a] Including the additional factors, [b] Present in three out of four collections pathotype 6E6A+.

genes that have not been deployed in Iran. In the greenhouse population, the frequency of virulence to wheat genotypes with the *Yr32+, YrSP* and *YrSD* genes was less than 7%, while virulence to the other wheat genotypes was between 19 and 100% (Figure 1).

The composition of *Pst* 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 prevalent stripe rust pathotypes (Singh *et al*., 2004). Bread wheat cultivars Seri 82, Falat (in Iran), Mexipac (in Syria) and Gereck (in Turkey) were resistant to the prevalent 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).

Therefore, the monitoring of stripe rust pathotypes and their changes over time can be an important consideration for breeding programs in Iran. Thus the pathogen population should be monitored regularly to determine whether new virulence pathotypes have been introduced and developed in the different parts of Iran as well as obtaining up-to-date information through the CWANA stripe rust network.

During this study in the field, stripe rust was developed in the four nurseries. The results are presented in Table 4. According to the results, virulence on Heines Kolben (with genes *Yr2* and *Yr6*), Kalyansona (*Yr2*), Lee (*Yr7*), Avocet R (*YrA*), Federation*4/Kavkaz (*Yr9*) and TP1295 (*Yr25*) was common during the period of investigation (Table 4). Virulence for *Yr1* which is common in Central Asia and China (Anmin *et*

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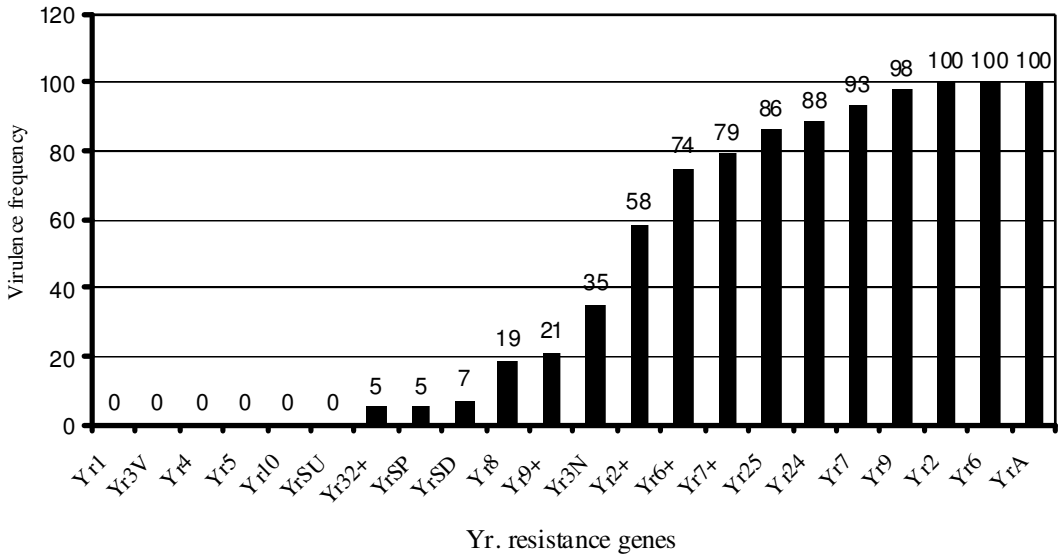

Figure 1. Virulence frequency of wheat stripe rust for resistance genes in seedling test.

al., 2004), and in most of the Middle East was absent in Iran (Afshari *et al.,* 2004). No virulence was observed on plants with *Yr1, Yr3V, Yr3N, Yr4, Yr5, Yr8, Yr10, Yr18, Yr24, Yr32+, YrSP, YrSD* and *YrSU genes* in the trap nurseries. The presence of virulence on plants with the genes *Yr3N, Yr8, Yr32+, YrSD* and *YrSP* in the seedling test (Figure 1) 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. The coefficient of infection (C. I.) for the *Yr18* gene was between 16-64 with a moderately susceptible reaction in Anza when the C. I. for *YrA* in Avocet R was calculated to be 100 (Table 4). Ma and Singh (1996) noted that *Yr18* might not provide adequate protection when deployed alone in a susceptible background. According to them, the preferred option for achieving durable stripe rust control is to have combinations of adult plant resistance genes giving extended protection approaching the levels of the most effective seedling resistance genes. The *Yr18* gene still remains resistant in Iran as an adult plant resistance gene and is going to be used in the breeding program in combination with other resistance sources to obtain an acceptable level of resistance in the new released cultivars. Virulence for *Yr27* (Selkirk gene) wasn't reported from the trap nurseries, but virulence for this gene has appeared in farmers fields and is confirmed by a seedling test in the greenhouse (Afshari, 2004). Moreover, the population of this pathotype is still limited and isn't common at the moment. However, using those resistance genes in combination with resistant adult plant genes could be a useful method to control yellow rust in Iran. Data from virulence surveys of pathogen populations have provided valuable information in the last 10 years and have frequently been used in our breeding programs.

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Table 4. Coefficiant of infection of wheat stripe rust differential set to stripe rust pathogen in four major wheat growing regions of Iran.

			Ardabil			Karaj			Zargan			Mashhad		
Entry	Name/Pedigree	*Yr gene/s*	2001-02	2002-03	2003-04	2001-02	2002-03	2003-04	2001-02	2002-03	2003-04	2001-02	2002-03	2003-04
1	Chinese 166	*Yr1*	0	0	0	0	0	0	0	0	12	8	4	4
2	Lee	*Yr7*	**90** [b]	**70**	**60**	**80**	**100**	**90**	**90**	**50**	8	**80**	8	8
3	Heines Kolben	*Yr2,Yr6*	**90**	40	48	**90**	**80**	**90**	**100**	32	24	**90**	16	8
4	Vilmorin 23	*Yr3V*	0	0	0	1	0	1	0	0	0	0	0	4
5	Moro	*Yr10*	16	20	16	2	0	1	0	0	4	0	0	1
6	Strubs Dikkopf	*YrSD*	0	0	0	2	2	1	0	0	0	0	0	1
7	Suwon 92/Omar	*YrSU*	56	56	40	1	0	1	0	56	0	0	8	0
8	Clement	*Yr2,Yr9+*	0	0	0	16	1	8	0	0	0	8	0	1
9	Hybrid 46	*Yr4*	0	0	0	1	0	1	0	0	0	0	0	1
10	Reichersberg 42	*Yr7+*	0	0	0	2	0	1	4	32	0	0	0	1
11	Heines Peko	*Yr2,Yr6+*	4	2	0	48	8	4	4	24	0	16	0	1
12	Nord Desprez	*Yr3N*	0	0	0	1	0	1	0	0	0	0	0	1
13	Compair	*Yr8,Yr18* [a]	4	2	2	0	0	1	0	12	0	0	0	1
14	Carstens V	*Yr32+*	0	0	0	1	0	1	0	0	0	0	0	1
15	Spalding Prolific	*YrSP*	0	0	0	1	0	0	0	0	0	0	0	0
16	Heines VII	*Yr2+*	4	2	0	12	1	8	0	0	0	24	0	1
17	Federation *4/Kavkaz	*Yr9*	**80**	40	64	**100**	**80**	**90**	**100**	64	**60**	**60**	0	24
18	Anza	*YrA, Yr18* [a]	32	16	32	64	64	48	40	64	24	48	0	4
19	Avocet 'R'	*YrA*	**100**	**100**	**100**	**100**	**80**	**100**	**100**	**100**	**100**	**100**	40	**80**
20	Avocet 'S'		**100**	**100**	**100**	**90**	**90**	**100**	**100**	**100**	**80**	**100**	40	**80**
21	Kalyansona	*Yr2*	32	32	16	**100**	**80**	**90**	56	**100**	32	**50**	0	2
22	*Triticum spelta* var. *album*	*Yr5*	0	0	0	0	0	0	0	0	0	0	0	0
23	TP981		24	48	16	64	**70**	**90**	**90**	16	40	10	0	2
24	TP1295	*Yr25*	**90**	**64**	**60**	**90**	**90**	**100**	**100**	**100**	**80**	**80**	0	**80**
25	Meering + Yr24	*Yr24*	0	0	0	4	4	6	6	0	8	0	0	2
26	Jupateco '73R'	*Yr18+* [a]	4	16	24	56	30	40	24	16	0	12	0	1
27	Jupateco '73S'		56	56	48	**80**	**100**	**100**	**100**	**60**	12	**60**	8	8
28	Federation		**90**	**100**	**100**	**100**	**80**	**100**	**100**	**60**	40	**80**	**60**	**80**
29	Bolani (Susceptible check)		**100S**	**100**	**90**	**100**	**100**	**90**	**100**	**100**	**60**	**90**	**100**	**80**

[a] Adult plant resistance gene; [b] Bold numbers indicated of susceptible infection type (S).

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پاتوتایپ‌های شایع عامل بیماری زنگ زرد گندم (*Puccinia striiformis* f.sp. *tritici*) در ایران

ف. افشاری

چکیده

بیماری زنگ زرد (نواری) گندم یکی از مهمترین بیماری‌های این محصول در ایران میباشد. از سال ۱۳۷۲ تا کنون چندین اپیدمی وسیع در ایران رخ داده است که سبب شکسته شدن منابع مقاومت در ارقام تجاری شده است. در طی سال‌های ۲۰۰۳ و۲۰۰۴ میلادی تعداد ۲۷ پاتوتایپ در نمونه های جمع آوری شده از کشور تعیین گردیدند. پاتوتایپ های 6E22A+, 6E130A+, 6E134A+ 6E142A+, 6E6A+, 6E158A+, 134E130A+, و 134E142A+ دارای بیشترین فراوانی بودند. بیماریزایی برای گیاهان با ژن‌های *Yr2, Yr6, Yr7, Yr8, Yr9, Yr24, Yr25, YrSD, YrSP, Yr3N, Yr2+, Yr6+, Yr9+, Yr7+*, *Yr32+* و *YrA* در شرایط کنترل شده گلخانه مشخص گردیدند. اغلب جدایه ها با بیش از ۸۸ درصد فراوانی دارای قدرت بیماریزایی بر روی گیاهان با ژن‌های *Yr2, Yr6, Yr7, Yr9, YrA* و *Yr24* بودند. بیماریزایی برای گیاهان با ژن‌های *Yr1, Yr3V, Yr4, Yr5, Yr10* و *YrSU* مشاهده نشد. در شرایط گلخانه بیماریزایی برای گیاهان با ژن‌های *YrSP, Yr32+* و *YrSU* کمتر از ۷ درصد تعیین گردید. بیماریزایی برای سایر گیاهان با ترکیب ژنی مورد مطالعه بین ۱۰۰-۱۹ درصد متغیر بود. در طی سه سال مطالعه در شرایط مزرعه، بیماریزایی برای منابع Heines Kolben (with *Yr2* and *Yr6*), Kalyansona (*Yr2*),

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Lee (*Yr7*), Avocet R (*YrA*), Federation*4/Kavkaz (*Yr9*), و TP1295 (*Yr25*) مشاهده شد. گیاهان با ژن های *Yr1, Yr3V, Yr3N, Yr4, Yr5, Yr8, Yr10, Yr18, Yr24, Yr32+, YrSP, YrSD* و *YrSU* در شرایط مزرعه دارای مقاومت بودند. ضریب آلودگی برای ژن *Yr18* به عنوان یکی از منابع مقاومت پایدار بین ۶۴-۱۶ با واکنش نیمه حساسیت بوده که میتواند با ترکیب سایر ژن های مسئول مقاومت که در این تحقیق معرفی شدند در برنامه‌های اصلاحی مورد استفاده قرار گیرد.

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