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Genetic analysis of resistance to stripe rust in some Iranian bread wheat cultivars and elite lines

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ABSTRACT

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Stripe rustis the most important disease of wheat in many wheat growing areas in Iran. Good knowledge of thegenetic basis of resistance to stripe rust in commercial bread wheat cultivars and selected elite lines is an important objective in wheat breeding programs. This study aimed to identify resistance genes and modes of inheritance of stripe rust resistance in recently released Iranian commercial wheat cultivars (Aflak, Parsi, Sivand, Uroum, and Pishgam) and elite bread wheat lines (M-84-14 and M-83-6). Crosses were made between these cultivars and elite lines with Avocet S and the F1, F2, and F3 generations were developed. Two F3-derived families (one adult plant stage and one seedling stage), as well as parents and controls, were grown under field and greenhouse conditions and inoculated with stripe rust pathotypes 134E158 A+, 166E150 A+, and 6E150 A+, *Yr27*. The adult plant responses of parental cultivars Aflak, Uroum, Parsi, Pishgam, Sivand, and elite lines M-84-14 and M-83-6 to stripe rust in the field were 40MR, 10R, 50M, 10R, 50MS, respectively, in 2011, and 60MR, 5R, 40MR, 30MR, and 40M, respectively, in 2013. Cultivars and elite lines were resistant to stripe rust at the seedling stage test. Avocet S was susceptible at both adult plant and seedling stages. In addition to the seedling resistance responses of the parents, frequencies of F3 lines for each of the crosses in both adult plant and seedling stages conformed well with those expected for segregating for a trait at two loci, indicating that all five cultivars and two elite lines carry two dominant seedling resistance genes that have so far been effective for controlling stripe rust in Iran.

Keywords:bread wheat, genetic analysis, pathotype, resistance gene, stripe rust.

INTRODUCTION

Stripe rust, caused by *Puccinia striiformis* Westend f. sp. *tritici* (*Pst*) Eriks., is one of the most economically important diseases of wheat worldwide (Saari and Prescott, 1985; McIntosh *et al*., 1995; Chen, 2005; Wellings, 2011), including for many wheat growing areas of Iran (Torabi *et al*., 1995; Afshari *et al*., 2014). In recent decades, a wide range of serious stripe rust epidemics have occurred in major wheat producing countries (including Iran), causing significant yield losses (Saari and Prescott, 1985; McIntosh *et al*., 1995; Zakeri, 2002; Singh *et al*., 2004; Wan *et al*., 2004; Chen, 2005; Wellings, 2011).

The most severe epidemics in Iran occurred in 1993 and 1995 and caused losses of over 30% on extensively grown wheat cultivars such as Falat (Seri 82) and Ghods (Torabi *et al*., 1995). The latest intermediate epidemics in Iran occurred in 2010 and to a lesser extent in 2013 (Afshari *et al*., 2014).

Past experiences have shown that stripe rust disease is highly specific and adapts very effectively due to host pressure, resulting in the emergence of new virulence under rapid evolutionary mechanisms accompanied by conducive conditions (McIntosh *et al*., 1995; Sharma-Poudya *et al*., 2013). An important factor in the recurring epidemics in Iran was the occurrence of virulence for *Yr9*, a resistance gene present in the CIMMYT-derived germplasm widely cultivated in Iran (Torabi *et al*., 1995), and recently for *Yr27*, a resistance gene in the most important commercial cultivars Chamran and Shiroudi in the Fars province of Iran (Afshari *et al*., 2003, 2014).

Genetic resistance is the most economic and environmentally friendly control measure to protect crops from damage due to biotic factors such as stripe rust (Johnson, 1988; McIntosh *et al*., 1995; Chen, 2005). Resistance to stripe rust can be divided in two broad categories: seedling (all stages) or

overall resistance, and adult plant resistance. Resistance that is expressed at the seedling stage usually remains effective to a virulent pathotypes of the pathogen for the entire growth cycle of the plant (McIntosh, 1988; Roelfs *et al*., 1992; McIntosh *et al*., 1995). Many pathologists have suggested that seedling resistance is synonymous with the terms vertical, race-specific, non-durable, and major gene resistance (Johnson, 1988; McIntosh, 1988; McIntosh *et al*., 1995; McIntosh and Brown, 1997). However, plants that are susceptible at the seedling stage may develop adult plant resistance during post-seedling growth stages (McIntosh, 1988; Roelfs *et al*., 1992; McIntosh *et al*., 1995).

Around 40 resistance genes have been formally identified and designated to wheat stripe rust, most of which are seedling resistance genes (McIntosh *et al*., 1995, 2010; Chen, 2005; Chen *et al*., 2013; Sharma-Poudyal*et al*., 2013). Many of the seedling resistance genes and some adult plant resistance genes have become ineffective due to the presence of corresponding virulence in the pathogen in various geographic areas of the world (McIntosh *et al*., 1995; Chen, 2005; Wellings, 2011; Sharma-Poudyal *et al*., 2013).

A good knowledge of thegenetic basis of resistance to stripe rust in commercial wheat cultivars and in selected elite lines is an important objective of wheat breeding programs (McIntosh and Brown, 1997). Inheritance studies have usually shown that resistance to rust diseases is simply inherited (McIntosh, 1988; Knott, 1989; Roelfs *et al*., 1992). In terms of its genetic basis, resistance can be single or major gene (i.e. controlled by a single Mendelian gene), oligogene (i.e. controlled by a few genes with large effects), or polygene (multi or minor gene), where resistance is controlled by a larger number of genes, each with a small effect (Johnson, 1988; McIntosh, 1988; McIntosh *et al*., 1995).

Deployment of race-specific resistance genes in commercial cultivars usually risksthe possibility that new virulent races can emerge and break the resistance of the cultivars within a few years (Johnson and Bimb, 1996; Singh *et al*., 2004). For example, more than 80% of wheat cultivars released in the late 1980s had *Yr9*, which became susceptible to a stripe rust race virulent on *Yr9* a few years later (Johnson, 1988; Johnson and Bimb, 1996; Mclntosh and Brown, 1997; Wan *et al*., 2004).

Gene *Yr27* has also been widely deployed in many wheat cultivars grown in India, Iran, Turkey, Syria, Pakistan and some other countries (Singh *et al*., 2004; Afshari *et al*., 2014). The emergence of new races virulent to *Yr27* caused intermediate to severe stripe rust epidemics in south and central Asian countries between 2002 and 2009 (Afshari *et al*., 2003, 2014; Singh *et al*., 2004). Highly effective sources of seedling resistance genes should therefore be deployed with care, and should preferably be combined with effective adult plant resistance in the same genetic backgrounds (McIntosh, 1988). Various studies of the inheritance of adult plant resistance to stripe rust in wheat have shown that highly effective sources comprise two or more resistance genes that usually behave in an additive modeof gene action (Johnson, 1988; Singh and Rajaram, 1994; Bariana and McIntosh, 1995; McIntosh and Brown, 1997).

Formal genetic analyses may be conducted on new sources of resistance by crossing them with susceptible lines to identify the number of genes controlling the resistance and to determine the respective modes of inheritance (Knott, 1989; Roelfs *et al*., 1992). The genetic bases of adult plant and seedling resistance have been studied in some commercial bread wheat cultivars from various wheat breeding institutes. Singh and Rajaram (1994) reported that the moderate adult plant resistance of Penjamo 62, Lerma Rojo 64, Nacozari 76, Tesia 79, and Wheaton were under monogenic control, and attributed this to *Yr18*, whereas Pavon 76 carried two unknown partially effective additive genes that were different from *Yr18* and remained effective in Mexico and several other countries.

Singh and Rajaram (1994) also identified that the resistance of Tonichi 81 was based on additive gene interaction involving *Yr18* and two additional partially effective genes. A study by Afshari (2006) in Iran showed that cultivar Chamran conferred one seedling resistance gene (*Yr27*) whereas cultivars Marvdashat, Shiraz, Shiroudi, and Pishtaz each carried two resistance genes, one of which operated at the seedling stage and the second at the adult plant stage. Zakeri *et al*. (2014) reported that Iranian bread wheat cultivars Niknejad and Bahar each possessed two resistance genes to stripe rust pathotype 166E138 A+, one of which in both cultivars was effective at the seedling stage. The second gene in Bahar conferred adult plant resistance but further work is needed to elucidate the role of the second gene in Niknejad. Darab-2 carried one adult plant resistance gene and Star lacked any resistance genes.

It should be noted that some of the Iranian bread wheat cultivars (Chamran, Shirodi, Shiraz, Bahar, Darab-2, and Star) have become susceptible to virulent races of stripe rust in Iran. Among the Iranian commercial wheat cultivars and elite lines evaluated in this study, Uroum and Pishgam are grown in the Cold Agro-ecological Zone; Parsi,

Sivand, and elite lines M-84-14 and M-83-6 in the Temperate Agro-ecological Zone; and Aflak in the Southern Warm and Dry Agro-ecological Zone of Iran. All five cultivars have good agronomic characteristics, including high yield potential, good adaptation, tolerance to lodging and wind burn, good bread quality, and resistance to stripe rust. The two elite lines M-84-14 and M-83-6 have not been released as new cultivars because of their inferior agronomic characteristics compared to the check cultivars. This study aimed to determine the genetic basis of resistance to stripe rust in five Iranian bread wheat cultivars and two elite breeding lines.

MATERIALS AND METHODS

Plant materials

Plant materials included fiveIranian commercial bread wheat cultivars (Aflak, Uroum, Parsi, Pishgam, and Sivand) and two elite lines (M-84-14 and M-83-6) (Table 1). In addition, the standard stripe rust differential sets comprised of world, European, and Australian sets (Johnson *et al*., 1972; Wellings and McIntosh, 1990), near-isogenic lines (Wellings and McIntosh, 1990; Wellings *et al*., 2009), and Avocet S and Bolani cultivars as susceptible parent and susceptible check, respectively.

Population development

The Iranian cultivars and elite lines were crossed with the susceptible parent Avocet S to produce F1, F2, and F3 generations in successive years. Two F3-derived families from each cross, one for resistance at the adult plant stage and one at the seedling stage, were considered for genetic analysis.

Table 1. Cross name and response of Iranian bread wheat cultivars and elite lines to *Pst*pathotypes at adult plant and seedling stages.

			Response to *Pst* pathotypes		
			Adult plant stage		Seedling stage
Cultivar/line	Year released	Cross name	2011 134E158 A+ , 166E150 A+ and 6E150 A+, *Yr27*	2013 6E174 A+, *Yr27*	2011 6E150 A+, *Yr27*
Aflak	2010	H0160/5/Tob/Cno/23854/3/Nai60//Tit/ Son64/4/LR/Son64	40MR	60MR	0
Uroum	2010	Alvand//NS732/Her	10R	5R	0
Parsi	2009	Dove"s"/Buc"s"//2*Darab 1	50M	40MR	0;
Pishgam	2008	Bkt/90-Zhong 87	10R	30MR	0
Sivand	2009	Kauz"s"/Azadi	50MS	40M	0
M-84-14	NR	Ww33/ Vee"s"// Niknejad	60MS	50MS	2
M-83-6	NR	GkZmbor/Attila	20M	30MR	2
Avocet S	1988	WW119/WW15/Egret[w]	100S	100S	8
Bolani	-	-	100S	100S	8

NR = not released; R = resistant; MR = moderately resistant; M= intermediate (between MR and MS); MS = moderately susceptible; S = susceptible; 0 = not fleck (immune); and 0; = immune and flecking.
[w]Wellings *et al*., 1988

Adult plant tests

The seeds of F3 populations, as well as the parents, differential genotypes, near-isogenic lines, and susceptible check Avocet S, were grown 5-7 cm apart on long rows in the field and inoculated three times with a mixture of *Pst* pathotypes134E158 A+, 166E150 A+, and 6E150 A+, *Yr27* from late tillering to flag leaf appearance. The responses of at least 79-104 F3 lines (each included at least 50 individual plants per line) as well as the parental cultivars, elite lines, and differential genotypes to the *Pst* pathotypes were recorded three times at the adult plant stage in 2011 using a Modified Cobb's scale (Peterson *et al*., 1948) and Roelfs *et al*. (1992) method. Severity and infection type were scored when the flag leaf reaction of the susceptible control (Bolani) reached 100S. The response of the parental cultivars, elite lines, and differential genotypes to *Pst* pathotype 6E174 A+, *Yr27* was also recorded in 2013 using the same methods.

Seedling tests

An experiment was also conducted for seedling stage studies under controlled greenhouse conditions. For this, 50 seeds of each of the F3 lines, as well as the parents and susceptible control were grown in two pots of 9 cm diameter and maintained at 15-20 C° for 12 days. The two leaves seedlings were then inoculated with *Pst*pathotype 6E150 A+, *Yr27* using a mixture ratio of one g pathogen urediospores and five g of talcum powder. Seedlings were then put in the cold dark room at 10 ºC and high humidity (9%5) for 24 h and transferred to a room under crystal covers at 17± 2 ºC with 16 h light at 16000 luxes and eight h darkness for 7-14 days.

The responses of at least 95-127 F3 lines were recorded after the leaves of the susceptible control (Bolani) were fully infected by the disease using the 0-9 scale following McNeal *et al*. (1971).

Statistical analyses

Based on the responses at adult plant and seedling stages to *Pst*pathotypes, F3 lines were categorized to three homozygous resistant, segregating, and homozygous susceptible classes. A chi-squared test was conduted on F3 data for genetic analyses following Little and Hills (1978).

$$¡ 2(n-1)\text{d.f.} = \sum \frac{(O-E)2}{E}$$

RESULTS

Responses of parental cultivars and elite lines

Different levels of resistance to *Pst* pathotypes were observed among the parental cultivars and elite lines at the adult plant stage in the field in 2011 and 2013 (Table 1). The responses of the parental cultivars (Aflak, Uroum, Parsi, Pishgam, and Sivand) to stripe rust at the adult plant stage in the field were 40MR, 10R, 50M, 10R, 50MS, respectively, in 2011, and 60MR, 5R, 40MR, 30MR, and 40M, respectively, in 2013. The two elite lines M-84-14 and M-83-6 exhibited reactions of 60MS and 20M (2011), and 50MS and 30MR (2013), respectively, at the adult plant stage in the field (Table 1). Susceptible parent Avocet S displayed a response of 100S at the adult plant stage in the field in both years. All cultivars were highly resistant to stripe rust at the seedling stage, though the two elite lines displayed a moderate level of resistance (Table 1). Avocet S was also susceptible to stripe rust at the seedling stage. The cross names of the parental cultivars and elite lines differed from each other, indicating that different parental genotypes were used in their pedigrees (Table 1).

Response of differential hosts

The response of the standard differential sets and near-isogenic lines to *Pst*pathotypes134E158 A+, 166E150 A+ and 6E150 A+, *Yr27* at the adult plant stage are presented in Table 2. Among the differential sets and near-isogenic lines, virulence was present for *Yr2*, *Yr6*, *Yr7*, *Yr9*, *Yr17*, *Yr27*, *YrA*, and *YrSU* in both 2011 and 2013, with the exception of additional virulence observed on *Yr25* in 2013. The remaining differential hosts and near-isogenic lines, particularly those with more than one resistance gene, were immune to the identified pathotypes at the adult plant stage.

Table 2. Adult plant response of standard differentials and near-isogenic lines of wheat to *Pst*pathotypes134E158 A+, 166E150 A+, 6E150 A+, *Yr27* and 6E174 A+, *Yr27*in 2011 and 2013 in the Zarghan trap nursery of Fars province, Iran.

No.	Host genotype	Yr gene (s)	Response to *Pst*pathotypes		No.	Host genotype	Yr gene (s)	Response to *Pst*pathotypes	
			2011	2013				2011	2013
1	Chinese 166	*Yr1*	70MS	90MS	24	Trident	*Yr17*	40MS	40MS
2	Lee	*Yr7*	70S	90S	25	Jupateco 73 R	*Yr18*	30MS	60MS
3	HeinesKolben	*Yr2*	70S	100S	26	Jupateco 73 S	-	70S	90S
4	Vilmorin 23	*Yr3*	40M	10R	27	Hugenoot	*Yr25*	10MR	80MS
5	Moro	*Yr10*	5R	20MR	28	Selkirk	*Yr27*	70S	100S
6	StrubsDikkoff	*YrSD*	5R	5R	29	*Yr1*/6*Avocet S	*Yr1*	40MS	30MS
7	Suwon92/Omar	*YrSU*	80S	70S	30	*Yr6*/6*Avocet S	*Yr6*	90S	100S
8	Clement	*Yr2,Yr9,*+	0	10R	31	*Yr5*/6*Avocet S	*Yr5*	20R	0
9	Hybrid 46	*Yr4*	10R	10R	32	*Yr7*/6*Avocet S	*Yr7*	100S	100S
10	Reichersberg 42	Yr7, +	5R	10R	33	*Yr8*/6*Avocet S	*Yr8*	60MR	5MR
11	HeinesPeko	*Yr2, Yr6,*+	20MR	10MR	34	*Yr9*/6*Avocet S	*Yr9*	100S	100S
12	NordDesprez	*YrND*	10MR	10R	35	*Yr10*/6*Avocet S	*Yr10*	0	0
13	Compair	*Yr8*	10R	30MR	36	*Yr15*/6*Avocet S	*Yr15*	0	0
14	Carstens V	*YrCV*	0	5R	37	*Yr17*/6*Avocet S	*Yr17*	90S	100S
15	Spalding Prolific	*YrSP*	5R	5R	38	*Yr18*/6*Avocet S	*Yr18*	70MS	80MS
16	HeinesVII	*Yr2,*+	5R	10R	39	*Yr24*/6*Avocet S	*Yr24*	20MR	60MR
17	Federation*4/Kavkaz	*Yr9*	70S	100S	40	*Yr26*/6*Avocet S	*Yr26*	80S	70MS
18	Federation	-	80S	100S	41	*Yr27*/6*Avocet S	*Yr27*	90S	100S
19	Anza	*YrA, Y r18*	50MS	60MS	42	*Yr32*/6*Avocet S	*Yr32*	5R	20MR
20	Avocet R	*YrA*	100S	100S	43	*YrSP*/6*Avocet S	*YrSP*	0	0
21	Avocet S	-	100S	100S	44	Bolani	-	100S	100S
22	Kalyansona	*Yr2*	70S	60S	45	Hugenoot	*Yr25*	10MR	80MS
23	*Triticum spelta* Var. alba	*Yr5*	0	10R					

indicates no stripe rust resistance gene(s)

F3 adult plant responses

Adult plant responses of the F3 lines to the *Pst*pathotypes are presented in Table 3. Based on field observations, the distribution of F3 lines in the first populations derived from the crosses of all five Iranian bread wheat cultivars and the two elite lines with Avocet S to *Pst*pathotypes conformed well at the adult plant stage with the predicted ratio of 7 homozygous resistant: 8 segregating: 1 homozygous susceptible, indicating segregation at two loci that was consistent with the result expected for two independent dominant genes. F3 lines that

had no susceptible plants were considered as homozygous resistant lines whereas F3 lines with no resistant plants were considered as homozygous susceptible lines. When both resistant and susceptible plants were observed, F3 lines were considered as segregating.

The phenotypic responses of the F3 homozygous resistant lines to stripe rust in the field were either similar to those of their resistant parents (when the effects of the two resistance genes from each of the above cultivars or lines were expressedin the F3 lines) or relatively higher than those of their resistant parents (when the effect of one of the resistance genes was expressed in the F3 lines). In the F3 segregating lines, the responses of resistant plants to stripe rust were either similar to those of their resistant parents (when the lines segregated for both resistance genes) or relatively higher than those of their resistant parents (when the lines segregated for one of the resistance genes).

Table 3. F3 adult plant responses to *Pst*pathotypes 134E158 A+, 166E150 A+ and 6E150 A+, *Yr27* among crosses of Iranian bread wheat cultivars and elite lines with Avocet S.

		Frequencies of F3 lines					
Cross name	Family	Homozygous resistant	Segregating	Homozygous susceptible	Total lines	Chi-squared χ^2	P value[ns]
Aflak/Avocet S	1	40	53	7	100	χ^2 7:8:1= 0.59	P 2 d.f.> 0.50
Uroum/Avocet S	1	39	57	8	104	χ^2 7:8:1= 1.75	P 2 d.f.> 0.250
Parsi/Avocet S	1	36	40	6	82	χ^2 7:8:1= 0.174	P 2 d.f.> 0.750
Pishgam /Avocet S	1	46	48	9	103	χ^2 7:8:1= 1.28	P 2 d.f.> 0.50
Sivand /Avocet S	1	39	57	6	102	χ^2 7:8:1= 1.43	P 2 d.f.> 0.250
M-84-14/Avocet S	1	48	46	5	99	χ^2 7:8:1= 0.98	P 2 d.f.> 0.50
M-83-6/ Avocet S	1	32	42	5	79	χ^2 7:8:1= 0.35	P 2 d.f.> 0.750

ns = not significant

F3 seedling responses

At seedling stage, the frequencies of F3 lines in segregation classes for the second population derived from the crosses of the above cultivars and elite lines with Avocet S were also consistent with the expected ratio of 7 homozygous resistant: 8 segregating: 1 homozygous susceptible, assuming segregation at two loci that corresponded to those ratios expected for two independent dominant genes (Table 4).

Table 4. F3 seedling responses to *Pst*pathotype 6E150 A+, *Yr27* among crosses of Iranian cultivars and elite lines with Avocet S.

		Frequencies of F3 lines					
Cross name	Family	Homozygous resistant	Segregating	Homozygous susceptible	Total lines	Chi-squared χ^2	P value[ns]
Aflak/Avocet S	2	44	46	5	95	χ^2 7:8:1= 0.338	P 2 d.f.> 0.750
Uroum/Avocet S	2	58	62	6	126	χ^2 7:8:1= 0.612	P 2 d.f.> 0.500
Parsi/Avocet S	2	60	64	3	127	χ^2 7:8:1= 3.429	P 2 d.f.> 0.100
Pishgam /Avocet S	2	51	60	8	119	χ^2 7:8:1= 0.068	P 2 d.f.> 0.950
Sivand /Avocet S	2	55	65	4	124	χ^2 7:8:1= 1.970	P 2 d.f.> 0.250
M-84-14/Avocet S	2	55	65	4	124	χ^2 7:8:1= 1.970	P 2 d.f.> 0.250
M-83-6/ Avocet S	2	42	46	7	95	χ^2 7:8:1= 0.242	P 2 d.f.> 0.750

ns = not significant

DISCUSSION

Based on the Chi-square analysis of data obtained from the F3 lines, and also the response of parental bread wheat cultivars and elite lines at both adult plant and seedling stages to *Pst*pathotypes used in this study, it was concluded that all five Iranian wheat cultivars and the two elite lines each carry two independent dominant seedling resistance genes.

There was little variation associated with the resistance of the parental cultivars and elite lines to stripe rust at the adult plant stage in the field during 2011 and 2013. Cultivar Uroum displayed a very resistant response at the adult plant stage, but the other four cultivars and elite lineshad intermediate levels of resistance. However, these cultivars showed high resistance and low infection types in their seedling stage test, which can be attributed to the effect of the other two extra *Pst*pathotypes (134E158 A+ and166E150 A+ in 2011 and pathotype6E174 A+, *Yr27* in 2013) that were used in the adult plant tests in the field (but were not used in the seedling tests).

Reduction or changes in the resistance of some of the differential hosts can be attributed to changes in stripe rust races, weather conditions, and other environmental factors. Given the virulence and avirulence formula for *Yr* gene(s) in differential hosts and near-isogenic lines, it can be concluded that the two seedling resistance genes in each of this study's five Iranian bread wheat cultivars and two elite lines might be among the formally or informally characterised stripe rust seedling resistance genes that have been previously identified (McIntosh *et al*., 1995, 2010; Chen, 2005; Chen

et al., 2013; Sharma-Poudyal *et al*., 2013). Thus either there is no virulence for them in Iran or they may be new sources of resistance to stripe rust. Further studies are needed to determine their nature and detailed characteristics of the resistances associated with these genes to stripe rust in Iran.

Pedigree analysis showed that cultivars Darab-1 and Alvand (each one parent of Parsi and Urom, respectively) are both susceptible to stripe rust. Darab-1 has the pedigree "Rsh*Irni49(60-61)*C271-Pk868, and Alvand has the pedigree CF1770/1-27-6275. These cultivars were released in 1980 and 1995, respectively, and became susceptible to stripe rust several years after their release (Torabi *et al*., 1995; Afshari *et al*., 2012, 2014). Thus the stripe rust resistance of Parsi and Urom can be attributed to the resistance of their second parents, Dove"s"/Buc"s" and NS732/Her, respectively. Both parents of Sivand (Kauz"s" and Azadi) are susceptible to stripe rust, based on their own resistance alone. The additive interactions of the resistance of these two cultivars have probably resulted in a moderately susceptible response of Sivand to stripe rust.

There is no record of susceptibility of the parents of Aflak and Pishgam to stripe rust in recent years in Iran. It appears that the elite line M-84-14 did not inherit all the resistance associated with Niknejad, because its level of resistance is lower. According to Zakeri *et al.* (2014), Niknejad carries two resistance genes and has exhibited a long lasting and higher level of resistance (10R) since it was released in 1995. It is possible that the plants with two resistance genes from Niknejad were not selected through selection processes of the elite line M-84-14. Results also showed that the elite line M-83-6 inherited its resistance from the GkZmbor parent, not from Attila.

Recent observations in farmers' fields in different parts of Fars province, Iran, have shown that the level of resistance of cultivars Uroum, Aflak, Parsi, Sivand, and Pishgam is commercially acceptable for protecting the crop from yield losses caused by intermediate epidemics of stripe rust. Despite their current effectiveness, the level of resistance of these cultivars would not be guaranteed under high disease pressure and severe epidemics in some areas of Iran where conducive conditions and failure of agronomic principles predispose rapid disease development and severity.

Past experiences in different areas around the world have shown that resistance based on one or two seedling resistance genes is usually short lived due to mutation and variability in the stripe rust pathogen populations (Rajaram *et al*., 1988; McIntosh and Brown, 1997). As a result, new emerging races are able to infect previously resistant cultivars, rendering them susceptible (Chen, 2005; Wellings, 2011; Sharma-Poudyal *et al*., 2013).

The results emphasize that the identification of resistance genes is important for better understanding the race changes that can affect the resistance of commercial bread wheat cultivars, and for making informed decisions in deploying effective stripe rust resistance genes in bread wheat breeding programs. In addition to avoiding over-dependence on monogenic inherited resistances in areas where stripe rust is a major problem, it is important to continue conducting genetic analyses to establish diversity and genetic relationships among resistant cultivars, and to progressively introduce new sources of resistance into elite germplasm (Rajaram *et al*., 1988; Singh *et al*., 2004; Chen, 2005). In this way, a cultivar that carries several effective resistance genes is more likely to remain resistant when exposed to a changing pathogen population (Rajaram *et al*., 1988; Wellings, 2011).

The use of combinations of race-specific resistance genes, particularly those for which matching virulence is rare, may give rise to more satisfactory control of stripe rustin regions where the climate is less favourable for the pathogen. However, effective integrated disease management, continuous disease monitoring systems, annual rust surveillance, genetic diversity, and proper agronomic practices are also essential (Johnson, 1988; McIntosh and Brown, 1997).

More emphasis should be given to deploy adult plant stripe rust resistance sources in bread wheat breeding programs. Adult plant resistance to rust diseases is commonly controlled by combinations of genes with additive effects (McIntosh, 1988; Rajaram *et al*., 1988; Singh and Rajaram, 1994). The current policy of CIMMYT bread wheat breeders is to select wheat varieties with moderate levels of adult plant resistance in order to avoid the hypersensitive responses associated with single major genes and their lower durability (Rajaram *et al*., 1988; McIntosh and Brown, 1997). As these resistances are based on gene combinations, they probably confer low coefficients of infection over wide areas of cultivation and are likely to be more durable (Johnson, 1988; McIntosh, 1988; Singh and Rajaram, 1994).

CONCLUSIONS

Results of this study indicated that the Iranian bread wheat cultivars Aflak, Uroum, Parsi, Pishgam, and Sivand each carry two independent dominant seedling resistance genes to stripe rust. While their

resistance levels are currently acceptable for protecting crop yields from the threatof stripe rust in Iran, intermediate to severe epidemics tend to occur every few years in Iran and there is the possibility for disease variability. Thus it is important to integrate proper agronomic practices, implement correct and timely use of effective fungicides, maintain genetic diversity, and monitor new aggressive virulences to increase the effectiveness and longevity of these cultivars and mitigate likely potential epidemics.

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REFRENCES

Afshari, F. 2006. Inheritance of resistance to stripe rust (*Puccinia striiformis* f. sp. *tritici*) in some cultivars and promising lines of wheat. Seed and Plant 22: 489-501.

Afshari, F., M. Torabi, and A. Malihipour. 2003. Appearance of a new race of *Puccinia striiformis* f. sp. *tritici* in Iran. Seed and Plant 19: 543-546 (In Persian).

Afshari, F., A. Zakeri, M. A. Dehghan, M. Chaichi, R. Hoshiar, S. A. Saffavi, M. Hassanpour Hosni, M. Dalvand, S. Ebrahimnejad, M. Atta Hossaini, K. Shabazi, G. Ahmadi, and A. Nabati. 2012. Virulence survey of *Puccinia striiformis*, the causal agent of wheat yellow rust by trap nursery during 2010-2012. Final report of research project.Seed and Plant Improvement Institute (In Persian).

Afshari, F., K. Nazari, M. Aghaei, G. Najafian, M. Esmaeilzadeh, A. Yazdansepas, M. Khodarahmei, A. Amini, R. Roohparvar, M. Hashamei, and A. Malihipour. 2014. The wheat stripe rust pathogen in Iran and achievement of resistant wheat cultivars over the last 10 years.p. 5. *In* Proceedings of the Second International Wheat Stripe Rust Symposium, Izmir, Turkey.

Bariana, H. S., and R. A. McIntosh. 1995. Genetics of adult plant stripe rust resistance in four Australian wheats and the French cultivar 'Hybride-de-Bersee'. Plant Breed. 114: 485-491.

Chen, X. M. 2005. Epidemiology and control of stripe rust on wheat. Can. J. Plant Pathol. 27: 314-337.

Chen, S. S., G.Y. Chen, C. Yang, Y. M. Wei, W. X. Wu, Y. J. He, Y. X. Liu, W. Li, Z. E. Pu, X. J. Lan, and Y. Zheng, 2013. Identification and mapping of a stripe rust resistance gene in spring wheat germplasm HRMSN-81 from CIMMYT. Crop and Pasture Sci. 64: 1-8.

Johnson, R. 1988. Durable resistance to yellow (stripe) rust in wheat and its implications in plant breeding. Pp.. 63-75. *In* N. W. Simmonds and S. Rajaram (eds.). Breeding Strategies for Resistance to the Rusts of Wheat. CIMMYT, Mexico. D.F.

Johnson, R., and H. P. Bimb. 1996. Genetics of resistance to yellow rust in CIMMYT wheats. Pp. 195-197. In: G. H. J. Kema, R. E. Niks and R. A. Daamen (eds.). Proceedings of the Ninth European and Mediterranean Cereal Rusts and Powdery Mildews Conference. Lunteren, The Netherlands.

Johnson, R., R. W. Stubbs, E. Fuchs, and N. H. Chamberlain. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. Trans. British Mycol. Society. 58: 475-480.

Knott, D. R. 1989. The wheat rusts: breeding for resistance. monograph on theoretical and applied genetics 12. springer Verlag, Berlin. 201 pp.

Little, T. M., and F. Jackson Hills. 1978. Agricultural experimentation: Design and analysis. John Wiley and Sons. 354 pp.

McIntosh, R. A. 1988. Genetic strategies for disease control. Pp. 39-44. *In*: T. E. Miller and R. M. D. Koebner (eds.). Proceedings of the Seventh International Wheat Genetics Symposium.Vol. 1. Institute of Plant Science Research, Cambridge, U.K.

Mclntosh, R. A., and G. N. Brown. 1997. Anticipatory breeding for resistance to rust diseases in wheat. Ann. Rev. Phytopathol. 35: 311-326.

Mclntosh, R. A., C. R. Wellings, and R. F. Park. 1995. wheat rusts: an atlas of resistance genes. csiro publications, Victoria, Australia. 200 pp.

McIntosh, R. A., J. Dubcovsky, W. J. Rogers, C. Morris, R. Appels, and X. C. Xia. 2010. Catalogue of gene symbols for wheat: 2010 supplement. Ann. Wheat Newsletter 56: 273-282.

McNeal, F. H., C. F. Konzak, E. P. Smith, W. S. Tale, and T. S. Russell. 1971. A uniform system for recording and processing cereal research data. United States Department of Agriculture, Agricultural Research Services Bulletin 34-121. 42 pp.

Peterson, R. F., A. B. Campbell, and A. E. Hannah. 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. Can. J. Res. Section C 26: 496-500.

Rajaram, S., R. P. Singh, and E. Torres. 1988. Current CIMMYT approaches in breeding wheat for rust resistance. Pp. 101-118. *In*: N. W. Simmonds and S. Rajaram (eds.). Breeding Strategies for Resistance to the Rusts of Wheat. CIMMYT, Mexico. D.F.

Roelfs, A. P., R. P. Singh, and E. E. Saari. 1992. Rust disease of wheat: concepts and methods of disease management. CIMMYT, Mexico. 81 pp.

Saari, E. E., and J. M. Prescott. 1985. World distribution in relation to economic losses. Pp. 259-298.*In:* A. P. Roelfs and W. R. Bushnell (eds.). The Cereal Rusts:

Volume 2. Diseases, Distribution, Epidemiology and Control. Academic Press: New York, London, Orlando, U.S.A. 592 pp.

Sharma-Poudyal, D., X. M. Chen, A. M. Wan, G. M. Zhan, Z. S. Kang, S. Q. Cao, S. L. Jin, A. Morgounov, B. Akin, Z. Mert, S. J. A. Shah, H. Bux, M. Ashraf, R. C. Sharma, R. Madariaga, K. D. Puri, C. R. Wellings, K. Q. Xi, R. Wanyera, K. Manninger, M. I. Ganzález, M. Koyda, S. Sanin, and L. J. Patzek. 2013. Virulence characterization of international collections of the wheat stripe rust pathogen, *Puccinia striiformis* f. sp. *tritici*. Plant Dis. 97: 379-386.

Singh, R. P., and S. Rajaram. 1994. Genetics of adult-plant resistance to stripe rust in ten bread wheats. Euphytica 72: 1-7.

Singh, R. P., H. M. William, J. Huerta-Espino, and G. Rosewarne. 2004. Wheat rust in Asia: meeting the challenges with old and new technologies. New directions for a diverse planet.Pp. 1-13. *In:* Proceedings of the 4th International Crop Science Congress, Brisbane, Australia.

Torabi, M., V. Mardoukhi, K. Nazari, F. Afshari, A. R. Forootan, M. A. Ramai, H. Golzar, and A. S. Kashani. 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. Cereal Rust and Powdery Mildews Bulletin 23: 9-22.

Wan, A. M., Z. H. Zhao, X. M. Chen, Z. H. He, S. L. Jin, Q. Z. Jia, G. Yao, J. X. Yang, B. T. Li, G. B. Wang, Y. Q. Bi, and Z. Y. Yuan. 2004. Wheat stripe rust epidemic and virulence of *Puccinia striiformis* f. sp. *tritici* in China in 2002. Plant Dis. 88: 896-904.

Wellings, C. R. 2011. Global status of stripe rust: A review of historical and current threats. Euphytica 179: 129-141.

Wellings, C. R., and R. A. McIntosh. 1990. *Puccinia striiformis* f. sp. *tritici*in Australasia: Pathogenic changes during the first 10 years. Plant Pathol. 39: 316-325.

Wellings, C. R., R. P. Singh, A. Yahyaoui, K. Nazari, and R. A. Mclntosh. 2009. The development and application of near-isogenic lines for monitoring cereal rust pathogens. p. 77-87. *In* R. A. McIntosh (ed.). "Borlaug Global Rust Initiative, Technical Workshop". Cd. Obregon, Sonora, Mexico.

Zakeri, A. 2002. Genetic analyses of rust resistance in wheat. Ph.D Thesis, University of Sydney, Plant Breeding Institute, Cobbitty, Australia. 220 pp.

Zakeri, A., F. Afshari, M. Yassaie, A. R. Nikzad, and S. Rejaei. 2014. Inheritance of resistance to stripe rust in several commercial cultivars and selected elite genotypes of wheat from Fars province. Iran. J. Plant Pathol. 50 (2): 163-174.