Research Paper / 22-35

Efficiency of resistance genes in wheat to powdery mildew in some centers of disease incidence in Iran

Mehdi Zahravi[1*], Mohammad Ali Dehghan[2], Hosein Ali Fallahi[3], Hosein Azimi[4]

[1]Seed and Plant Improvement Institute, Agricultural Research, Education and Extension Organization (AREEO), P. O. Box: 31359-33151, Karaj, Iran.
[2]Agriculture and Natural Resources Research Center of Golestan, Agricultural Research, Education and Extension Organization (AREEO), Golestan, Iran.
[3]Agriculture and Natural Resources Research Center of Mazandaran, Agricultural Research, Education and Extension Organization (AREEO), Mazandaran, Iran.
[4]Iranian Research Institute of Plant Protection, Agricultural Research, Education and Extension Organization (AREEO), Tehran, Iran.
**Corresponding author, Email: m.zahravi@areeo.ac.ir. Tel: +98-026-32701260.*

Received: 19 Mar 2019; Accepted: 13 Jul 2019.
DOI: 10.30479/ijgpb.2019.10100.1229

Abstract

Wheat powdery mildew caused by fungal pathogen *Blumeria graminis* f.sp. *tritici* is a destructive wheat disease, occurring in regions with cool and humid climates. In order to investigate the changes in pathogenicity of the disease and to identify the effective genetic source of resistance, a total of 29 wheat genotypes with different resistance major genes (*Pm*) were studied in two hotspots of Gorgan and Sari under natural incidence of the disease over 2015-2018. Resistance reaction of the genotypes was evaluated by disease development, disease severity and coefficient of infection. The results indicated that mean of coefficient of infection for Gorgan was larger than that for Sari in all three years of the study. The reaction of some genotypes showed apparent differences between the two regions during the years of study. The highest genetic distance was observed between Ralle (*Pm3d*) and Holger (*Pm6*). NK-747 (*Pm6*) and Maris Huntsman (*Pm2+Pm6*) as well as Amigo (*Pm17*) and Maris Huntsman (*Pm2+Pm6*) showed the highest genetic similarities to each other. A total of 65.78% of the data variation was justified by principal component analysis on the basis of the coefficient of infection. The genotypes Maris Dove (*Mld+Pm2*), Shamrock (with unknown *Pm*), Broom (*Pm3d*) and Axona (*Pm2+Pm3d+Mld*) expressed effective resistance over the years and locations. The total results of this research indicated variations in the virulence of wheat powdery mildew population during the years of the experiment. This finding emphasizes on the necessity of constant monitoring of the disease in the infected areas. The genes with effective resistance identified in this study could be used in resistance breeding to powdery mildew especially for Gorgan and Sari.

Key words: *Blumeria graminis* f. sp. *tritici*, Differential cultivars, Pathogen population, Resistance, Virulence.

INTRODUCTION

Wheat is one of the oldest cereals in the world with the widest cultivation providing a major part of the calorie intake of humans and livestock (Yu *et al.,* 2015; Geng *et al.,* 2016). Powdery mildew caused by fungal pathogen *Blumeria graminis* f.sp. *tritici*, is a destructive disease of common wheat in many regions of the world which can induce significant yield losses ranging from 13 to 50% and serious quality deterioration (Liu *et al.,* 2017; Janáková *et al.,* 2019). The introduction and intensive production of semi-dwarf cultivars that are genetically uniform, and the increased utilization of nitrogen fertilizers and irrigation has increased the incidence and severity of powdery mildew (Imani *et al.,* 2002; Ma *et*

al., 2015). Albeit, chemical protection is extensively used to control the disease but it can lead to environmental problems as well as acquisition of fungicide tolerance by the pathogen. Therefore, introducing resistant cultivars which confer resistance genes is the most effective, economical, and environmentally safe disease control strategy.

Breeding resistance to wheat powdery mildew requires understanding the virulence structure and genetic variations of the pathogen (Liu *et al.,* 2015). Various studies have been performed worldwide with the objective of finding out the spectra and distribution of virulence of *B. graminis* f.sp. *tritici*. Persaud and Lipps (1995) investigated virulence genes and virulence gene frequencies of *B. graminis* f.sp. *tritici* in 199 isolates collected in Ohio by using 11 powdery mildew resistance genes (*Pm* genes). As the results suggested, more than 90% of the isolates tested gave a compatible reaction with four to eight *Pm* resistance genes, the frequent occurrence of complex races in the pathogen population was concluded. Niewoehner and Leath (1998) characterized a total of 520 isolatesof *B. graminis* f.sp. *tritici* using a differential host series containing15 known resistance genes and observed an increase in virulence frequencies and complexity of isolates. Imani *et al.* (2002) determined virulence frequencies, complexity, and racial composition of the pathogen populations of *B. graminis* using differential series comprised of 13 known genes conferring resistance to powdery mildew and concluded that the random distribution of virulence genes among pathotypes is an indication of sexual reproduction contributing to the variability of the pathogen. In addition, Karimi-Jashni *et al.* (2006) studied 21 different pathotypes of powdery mildew by 17 near-isogenic lines carrying the known *Pm* genes and concluded that pathotypes of Gharakhil in Mazandaran province, contained the highest number of virulence factors among the isolates. This study was the first report on the presence of virulence for *Pm1+Pm2+Pm9* gene combination in Iran. In other study, Monazzah *et al.* (2008) investigated virulence factors and pathotypes of *B. graminis* f.sp. *tritici* in different provinces of Iran by studying 18 isolates of the pathogen. The results led to the identification of 14 pathotypes indicating the presence of high genetic variability in the population of the pathogen. Pathotypes No. 6 and 12 from Varamin with 87.8% pathogenicity, were the most virulent pathotypes. By studying virulence factors of *B. graminis* f.sp. *tritici* using trap nurseries in natural conditions in Gorgan, Mazandaran, Moghan and Varamin regions during 2005-2007, Razavi *et al.* (2010) concluded that a few near isogenic lines/cultivars used in this research had potential to be incorporated into high yielding commercial wheat cultivars in Iran. Study of 1082 isolates of *B. graminis* f. sp. *tritici* obtained from eight major wheat-growing regions in China by using 22 differential lines with known *Pm* genes detected 1028 pathotypes, of which 984 were unique (Zeng *et al.,* 2014) and genetic distance between different populations correlated significantly with geographical distance. Liu *et al.* (2015) investigated 17 pathogenic populations in Sichuan, China and classified 109 isolates into two distinct groups of high virulence and low virulence based on pathogenicity analysis. Zahravi *et al.* (2018) collected isolates of the disease from different regions within Iran and identified ten pathotypes which all had virulence factors for *Pm2*, *Pm3a*, *Pm3c*, P*m3g*, *Pm4a*, *Pm5*, *Pm6* and *Pm8*.

Virulence characterization of the pathogen population will facilitate the identification and utilization of effective resistance genes in cultivar development (Imani *et al.,* 2002). The climate of the two provinces Mazandaran and Golestan is suitable for the development of powdery mildew disease so that the prevalence of this disease has always been reported in these areas. In the recent years, an outbreak of this disease has been observed more frequently in the northern region of the country, including these two regions. Therefore, monitoring the disease is necessary in decision making of variety cultivation in these areas. The present study was conducted to investigate the changes in the pathogen population, and the results can be used for breeding programs to develop resistant varieties with effective *Pm* gene.

MATERIALS AND METHODS

Experimental methodology

Differential set used in this study consisted of 29 wheat genotypes with different major resistance genes (*Pm*) to powdery mildew which were previously received from International Center for Agricultural Research in the Dry Areas (ICARDA) (Table 1). These genotypes were evaluated for resistance to powdery mildew in two disease hotspots of Sari and Gorgan over three successive years from 2015 to 2018. For this purpose, the experiment was carried out at the fields of Gharakhil (Sari) and Iraqi-Mahalleh (Gorgan) research stations under natural disease incidence. The seeds of each genotype were planted in a row with the length of one meter considering a 60 cm distance from the adjacent rows in three replications. The susceptible variety Bolani which is local to Iran, was cultivated between the rows of differential genotypes and around the experimental plot as the disease spreader.

Table 1. Differential set of wheat genotypes with resistance (R) genes.

Code	Differential cultivar	R gene	Code	Differential cultivar	R gene
DV1	Axminster/ 8*Chancellor	*Pm1*	DV16	Normandie	*Pm1+Pm2+Pm9*
DV2	Ulka/8*Chancellor	*Pm2*	DV17	Axona	*Pm2+Pm3d+Mld*
DV3	Asosan/8*Chancellor	*Pm3a*	DV18	Shamrock	Unknown
DV4	Chul/8*Chancellor	*Pm3b*	DV19	Maris Dove	*Mld+Pm2*
DV5	Sonora/8*Chancellor	*Pm3c*	DV20	Sicco	*Pm5a+MlSi2*
DV6	Khapli/8*Chancellor	*Pm4a*	DV21	Armada	*Pm4b*
DV7	Ronos	*Pm4b*	DV22	Chul	*Pm3b*
DV8	Rector	*Pm5*	DV23	Soissons	*Pm3g*
DV9	Nk-747	*Pm6*	DV24	Broom	*Pm3d*
DV10	Disponent	*Pm8*	DV25	Holger	*Pm6*
DV11	Amigo	*Pm17*	DV26	Hope	*Pm5a*
DV12	Maris Huntsman	*Pm2+Pm6*	DV27	Wembley	*Pm12*
DV13	Apollo	*Pm2+Pm4b+Pm8*	DV28	Galahad	*Pm2*
DV14	Ralle	*Pm3d*	DV29	Ambassador	*Pm8*
DV15	Transfed	*Pm 7*			

Data collection and statistical analysis

The response of genotypes to the disease was recorded at the time of maximum infection on Bolani. Resistance reaction was evaluated based on the vertical development of disease throughout the plant based on the scale of 0 to 9 according to Sari and Prescott (1975) and also based on the severity of the disease (as the percentage of infection). The coefficient of infection was calculated through multiplying disease development by disease severity divided by 100 (Stubbs *et al.,* 1986). Descriptive statistics for the components of resistance were calculated. The Shannon index was used to characterize the diversity of reaction to the disease in the resistance components during different years and locations of the study (Shannon, 1948). Wricke (1962) equivalence (w_i^2) was used to determine the most stable responses within different years and locations. Data dimension was reduced using principal component analysis so that the studied genotypes were separated in the biplot of the first two principal components. Proximity matrix was calculated based on Euclidean distances between the genotypes. The genotypes were also grouped by cluster analysis based on the Ward's method. Statistical analyses were performed by R 3.6.0 software. The R package factoextra was used to illustrate the results of principal component analysis.

RESULTS

The results of Shannon index comparison calculated separately for different years and regions showed that in almost all cases (with the exception of the experiment of 2017-2018 in Sari), the disease severity observed in each year and region had a greater value of this index than the disease development (Table 2).

The correlation between the two traits of disease development and disease severity in different regions and years was investigated and the results showed significant correlations in all the cases except in the year 2015-2016 in Gorgan (Table 3). The highest correlation coefficient between these two traits (r=0.975**) was observed in 2016-2017 at Sari experiment.

Descriptive statistics were also calculated for coefficient of infection as the reaction criterion comprising both resistance components of disease severity and disease development. The results (Table 4) showed that the least mean of this trait (0.14) belonged to the experiment carried out in 2017-2018 in Sari and the highest (0.3) to the experiment of 2015-2016 in Gorgan. Mean of this trait for Gorgan was larger than that for Sari in all three years of the study. The experiments carried out in 2016-2017 in Gorgan and 2017-2018 in Sari had a complete range of coefficient of infection.

Relationship between the experiments was also studied by correlation analysis of coefficient of infection (Table 5) and the results showed that depending on the case, there were significant and non-significant correlations between the values of coefficient of infection in different years and locations.While the coefficient of infection in the experiment of Gorgan carried out in 2015-2016 was not significantly correlated with that in other experiments, the experiment of Gorgan carried out in 2016-2017 showed significant correlations with all other experiments for this trait. The highest correlation coefficient (r=0.643**) was observed between Sari 2016-2017 and Gorgan 2016-2017 experiments.

Table 2. Values of Shannon index in the study of resistance reaction of wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

Experiment		Disease development	Disease severity
Location/year	Gorgan (2015-2016)	1.21	1.41
	Sari (2015-2016)	0.99	1.46
	Gorgan (2016-2017)	1.21	1.78
	Sari (2016-2017)	0.74	1.16
	Gorgan (2017-2018)	1.15	1.85
	Sari (2017-2018)	1.48	0.53
Location	Gorgan	1.46	1.97
	Sari	1.50	1.56
Year	2015-2016	1.13	1.61
	2016-2017	1.04	1.56
	2017-2018	1.38	1.64

Table 3. Spearman coefficients of correlation between two resistance components of disease development and disease severity in the study of resistance reaction of wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018 .

Year/Location	Gorgan	Sari
2015-2016	-0.018	0.885**
2016-2017	0.885**	0.975**
2017-2018	0.725**	0.591**

**: Significant at 0.01 probability level.

Table 4. Descriptive statistics of coefficient of infection in the study of resistance reaction of wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

Experiment	Minimum	Maximum	Range	Mean	Std. Deviation
Gorgan (2015-2016)	0.02	1.00	0.98	0.30	0.23
Sari (2015-2016)	0.05	0.67	0.62	0.20	0.17
Gorgan (2016-2017)	0	1.00	1.00	0.24	0.29
Sari (2016-2017)	0	0.67	0.67	0.15	0.23
Gorgan (2017-2018)	0.02	1.00	0.98	0.29	0.28
Sari (2017-2018)	0	1.00	1.00	0.14	0.20

Table 5. Pearson coefficients of correlation between coefficients of infection in experiments of resistance evaluation of wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

Experiment	Sari (2015-2016)	Gorgan (2016-2017)	Sari (2016-2017)	Gorgan (2017-2018)	Sari (2017-2018)
Gorgan (2015-2016)	0.012	-0.024	-0.347	-0.014	-0.126
Sari (2015-2016)		0.532**	0.207	.513**	0.195
Gorgan (2016-2017)			0.643**	0.390*	0.449*
Sari (2016-2017)				0.128	.575**
Gorgan (2017-2018)					0.036

* and **: Significant at confidence levels of 5% and 1%, respectively.

To better illustrate the difference between reactions in the studied genotypes in different regions and years, comparison plot of coefficient of infection are presented separately for different experiments (Figure 1). Accordingly, in 2015-2016, there were apparent differences between the two regions of Sari and Gorgan for reaction of genotypes DV1 (Axminster/8*Chancellor), DV3 (Asosan/8*Chancellor), DV6 (Khapli/8*Chancellor), DV14 (Ralle) and DV16 (Normandi) among them DV3 (Asosan/8*Chancellor), DV6 (Khapli/8*Chancellor), DV14 (Ralle) and DV16 (Normandi) appeared more resistant in Sari and DV1 (Axminster/8*Chancellor) in Gorgan. Furthermore, DV15 (Transfed) appeared with the highest susceptibility in both regions. In the experiment carried out in 2016-2017, the genotypes DV15 (Transfed), DV21 (Armada) and DV26 (Hope) showed the highest differential reactions between the two study sites with DV15 (Transfed) and DV21 (Armada) appearing more resistant in Sari and DV26 (Hope) in Gorgan. The genotypes DV25 (Holger), DV28 (Galahad) and DV29 (Ambassador) also showed the

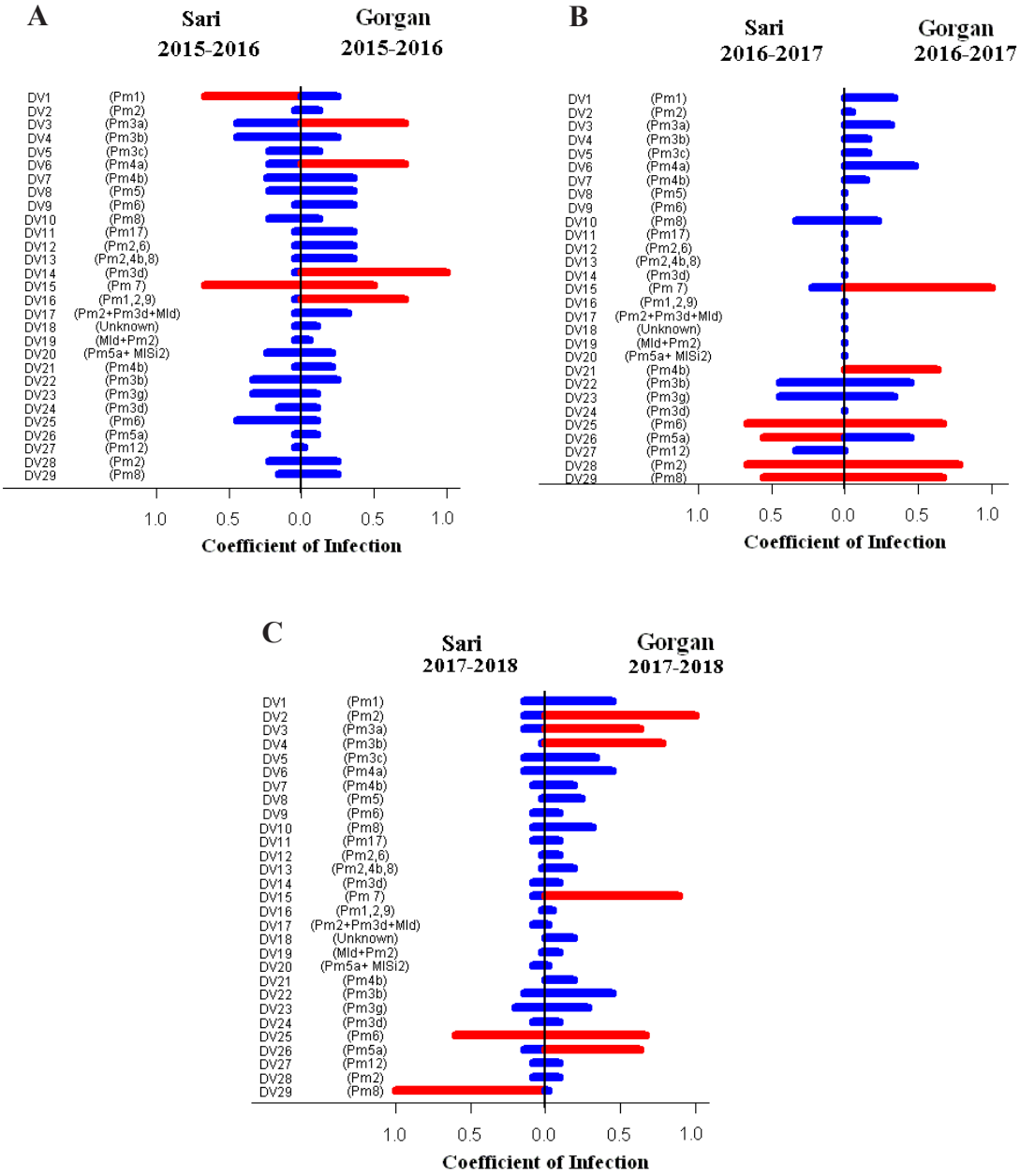

Figure 1. Comparison plot of coefficient of infection expressed by wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years **A:** 2015-2016, **B:** 2016-2017 and **C:** 2017-2018.

highest susceptibility in both experimental sites in this year. In 2017-2018, DV2 (Ulka/8*Chancellor), DV3 (Asosan/8*Chancellor), DV4 (Chul/8*Chancellor), DV15 (Transfed), DV26 (Hope) and DV29 (Ambassador) expressed the highest differential response between two regions, among them DV2 (Ulka/8*Chancellor), DV3 (Asosan/8*Chancellor), DV4 (Chul/8*Chancellor), DV15 (Transfed) and DV26 (Hope) appeared more resistant in Sari and DV29 (Ambassador) in Gorgan. DV25 (Holger) also possessed the highest susceptibility in both locations.

The stability analysis of resistance reaction of the genotypes by Wricke equivalence criterion (Table 6) showed that in the whole experiments, the genotypes Ambassador (*Pm8*), Ralle (*Pm3d*), Ulka/8*Chancellor (*Pm2*) and Galahad (*Pm2*) had the highest and the genotypes Shamrock (unknown *Pm*), Maris Dove (*Mld+Pm2*), Ronos (*Pm4b*), Broom (*Pm3d*) and Sonora/8*Chancellor (*Pm3c*) had the least amount of w_i^2. In the experiments conducted in Gorgan, taking different years into account, the genotypes Ralle (*Pm3d*), Ulka/8*Chancellor (*Pm2*), Galahad (*Pm2*) and Normandi (*Pm1+Pm2+Pm9*) had the highest and the genotypes Maris Dove (*Mld+Pm2*), Broom (*Pm3d*), Wembley (*Pm12*) and Shamrock (unknown *Pm*) had the lowest w_i^2. Based on different years of experiments performed in Sari, the genotypes Ambassador (*Pm8*), Axminster/8*Chancellor (*Pm1*), Galahad (*Pm2*) and Hope (*Pm5a*) had the highest and the genotypes Maris Huntsman (*Pm2+Pm6*), Apollo (*Pm2+Pm4b+Pm8*), Normandie (*Pm1+Pm2+Pm9*), Shamrock (unknown *Pm*), Maris Dove (*Mld+Pm2*) and Armada (*Pm4b*) had zero value (the lowest) for w_i^2. Wricke equivalence was also calculated separately for each year of the experiments. The results showed that in 2015-2016, the genotypes Ralle (*Pm3d*), Normandi (*Pm1+Pm2+Pm9*), Axminster/8*Chancellor (*Pm1*) and Holger (*Pm6*) had the highest and the genotypes Shamrock (unknown *Pm*), Armada (*Pm4b*), Ulka/8*Chancellor (*Pm2*), Ronos (*Pm4b*), Rector (*Pm5*), Hope (*Pm5a*), Galahad

Table 6. Values of Wricke equivalence (w_i^2) for coefficient of infection in experiments of resistance evaluation of wheat differential cultivars to powdery mildew in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

Code	Differential cultivar	Total	Gorgan	Sari	2015-2016	2016-2017	2017-2018
DV1	Axminster/8*Chancellor	0.23	0.02	0.21	0.14	0.03	0.01
DV2	Ulka/8*Chancellor	0.59	0.51	0.02	0.00	0.00	0.24
DV3	Asosan/8*Chancellor	0.22	0.06	0.08	0.01	0.03	0.05
DV4	Chul/8*Chancellor	0.31	0.20	0.09	0.04	0.00	0.17
DV5	Sonora/8*Chancellor	0.04	0.02	0.02	0.02	0.00	0.00
DV6	Khapli/8*Chancellor	0.19	0.04	0.02	0.07	0.08	0.01
DV7	Ronos	0.04	0.02	0.02	0.00	0.00	0.00
DV8	Rector	0.06	0.04	0.02	0.00	0.00	0.00
DV9	Nk-747	0.06	0.05	0.01	0.02	0.00	0.01
DV10	Disponent	0.07	0.02	0.03	0.02	0.02	0.00
DV11	Amigo	0.06	0.05	0.01	0.02	0.00	0.01
DV12	Maris Huntsman	0.05	0.05	0.00	0.02	0.00	0.01
DV13	Apollo	0.05	0.04	0.00	0.02	0.00	0.00
DV14	Ralle	0.63	0.56	0.01	0.36	0.00	0.01
DV15	Transfed	0.48	0.17	0.09	0.01	0.24	0.20
DV16	Normandie	0.30	0.28	0.00	0.15	0.00	0.01
DV17	Axona	0.06	0.05	0.01	0.01	0.00	0.03
DV18	Shamrock	0.01	0.01	0.00	0.00	0.00	0.00
DV19	Maris Dove	0.02	0.00	0.00	0.01	0.00	0.01
DV20	Sicco	0.07	0.02	0.02	0.01	0.00	0.03
DV21	Armada	0.23	0.17	0.00	0.00	0.15	0.00
DV22	Chul	0.08	0.04	0.04	0.02	0.00	0.01
DV23	Soissons	0.13	0.04	0.03	0.05	0.02	0.00
DV24	Broom	0.04	0.00	0.01	0.01	0.00	0.01
DV25	Holger	0.35	0.24	0.04	0.09	0.00	0.01
DV26	Hope	0.32	0.15	0.17	0.00	0.02	0.05
DV27	Wembley	0.15	0.00	0.06	0.01	0.09	0.01
DV28	Galahad	0.52	0.31	0.19	0.00	0.00	0.01
DV29	Ambassador	0.89	0.27	0.41	0.00	0.00	0.66

(*Pm2*) and Ambassador (*Pm8*) the least amount (zero value) of w_i^2. In 2016-2017, the genotypes Transfed (*Pm7*), Armada (*Pm4b*), Wembley (*Pm12*) and Khapli/8*Chancellor (*Pm4a*) showed the highest and the genotypes Shamrock (unknown *Pm*), Ulka/8*Chancellor (*Pm2*), Ronos (*Pm4b*), Rector (*Pm5*), Galahad (*Pm2*), Ambassador (*Pm8*), Maris Dove (*Mld+Pm2*), Axona (*Pm2+Pm3d+Mld*), Broom (*Pm3d*), Sicco (*Pm5a+MlSi2*), Maris Huntsman (*Pm2+Pm6*), Apollo (*Pm2+Pm4b+Pm8*), Nk-747 (*Pm6*), Amigo (*Pm17*), Sonora/8*Chancellor (*Pm3c*), Chul (*Pm3b*), Chul/8*Chancellor (*Pm3b*), Holger (*Pm6*), Normandie (*Pm1+Pm2+Pm9*) and Ralle (*Pm3d*) the lowest (zero value) w_i^2, and in 2017-2018, the genotypes Ambassador (*Pm8*), Ulka/8*Chancellor (*Pm2*), Transfed (*Pm7*) and Chul/8*Chancellor (*Pm3b*) possessed the highest and the genotypes Shamrock (unknown *Pm*), Ronos (*Pm4b*), Rector (*Pm5*), Apollo (*Pm2+Pm4b+Pm8*), Sonora/8*Chancellor (*Pm3c*), Disponent (*Pm8*), Soissons (*Pm3g*) and Armada (*Pm4b*) showed the lowest (zero value) w_i^2 (Figure 2).

The results of principal component analysis on the basis of the coefficient of infection indicated that the two principal components justified 65.78% of the data variation (Table 7). The coefficient of all variables in the first principal component were negative except for the experiment of Gorgan 2015-2016. In the second principal component, the experiment Gorgan 2017-

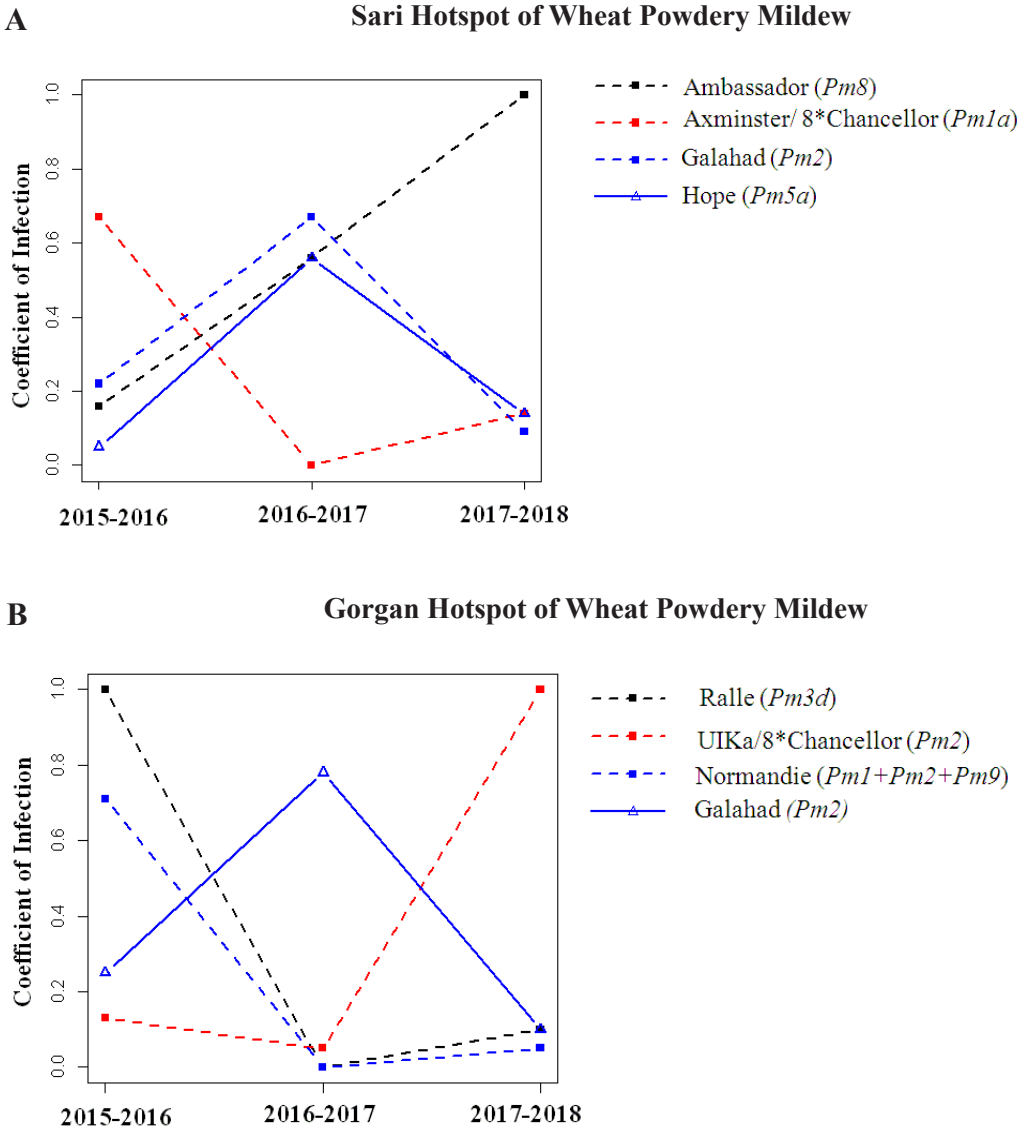

Figure 2. Changes of resistance reaction to powdery mildew in some wheat differential cultivars evaluated in disease hotspots of **A:** Sari and **B:** Gorgan, Iran, during years 2015-2018.

2018 had the highest coefficient regardless of the sign and the experiments carried out in 2016-2017 and 2017-2018 in Sari were the only variables with positive coefficients. In the biplot of the two first principal components, the genotypes DV5 (*Pm3c*), DV9 (*Pm6*), DV11 (*Pm17*), DV12 (*Pm2+Pm6*), DV13 (*Pm2+Pm4b+Pm8*), DV17 (*Pm2+Pm3d+Mld*), DV18 (unknown *Pm*), DV19 (*Mld+Pm2*), DV20 (*Pm5a+MlSi2*), DV21 (*Pm4b*), DV24 (*Pm3d*) and DV27 (*Pm12*) were placed in the upper right quarter, which is related to the positive values for both principal components (Figure 3). None of the vectors of the experiments were placed in this area. The highest and lowest distances from the origin of axes were assigned to DV27 (*Pm12*) and DV5 (*Pm3c*), respectively.

Table 7. Eigen vectors and eigen values of principal component analysis on coefficient of infection measured in resistance evaluation of wheat differential cultivars to powdery mildew in disease hot spots of Gorgan and Sari, Iran, during years 2015-2018.

Experiment	Principal components	
	First	Second
Gorgan (2015-2016)	0.136	-0.466
Sari (2015-2016)	-0.41	-0.468
Gorgan (2016-2017)	-0.544	-0.077
Sari (2016-2017)	-0.485	0.406
Gorgan (2017-2018)	-0.346	-0.507
Sari (2017-2018)	-0.403	0.369
Eigen values	1.600	1.177
Cumulative variance (%)	42.68	65.78

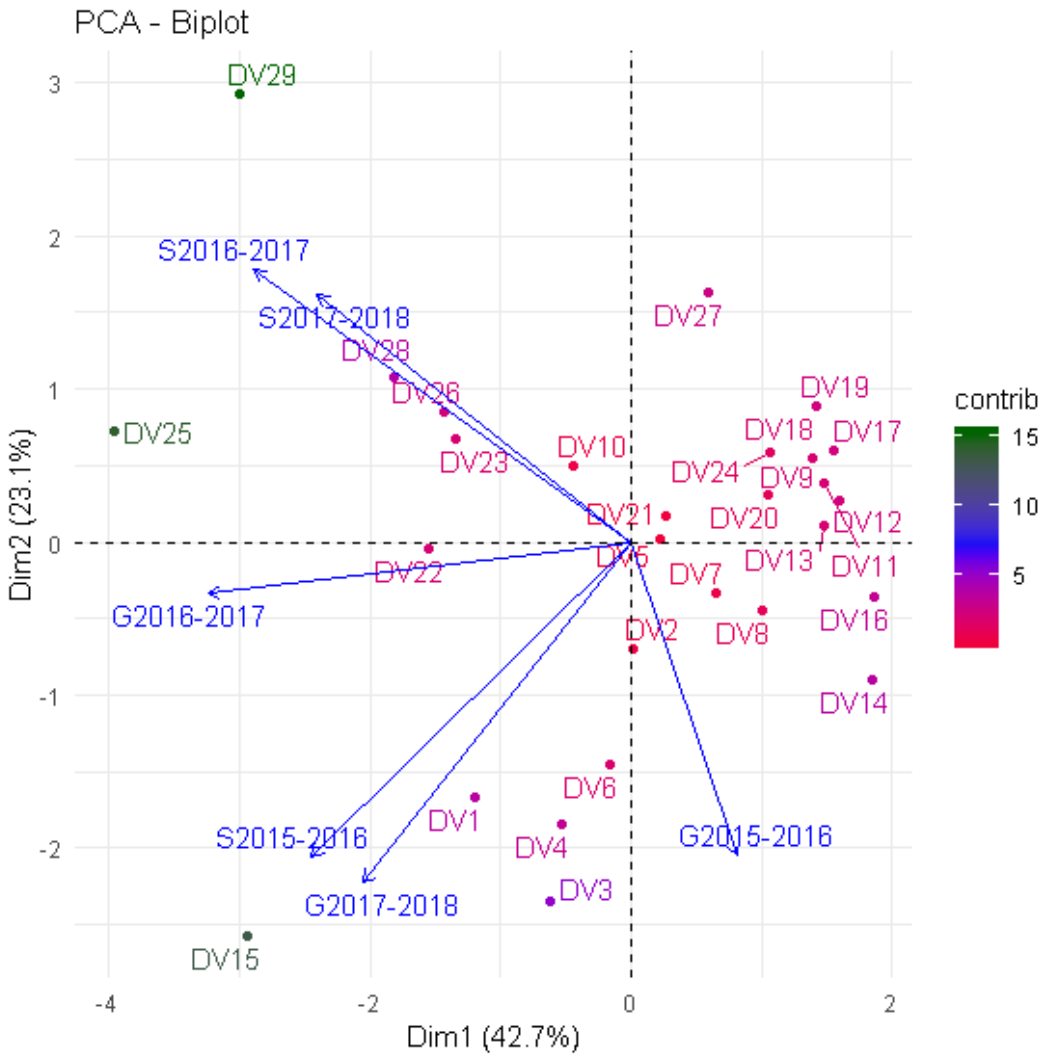

Figure 3. Distribution of wheat powdery mildew differential cultivars in biplot of the first two components of principal component analysis on coefficient of infection in response to the disease evaluated in the hotspots of Gorgan and Sari, Iran, during years 2015-2018.

Among the genotypes of this quarter, DV20 (*Pm5A+MlSi2*) in Sari 2015-2016, DV9 (*Pm6*), DV11 (*Pm17*), DV12 (*Pm2+Pm6*) and DV13 (*Pm2+Pm4b+Pm8*) in Gorgan 2015-2016, DV21 (*Pm4b*) in Gorgan 2016-2017, DV27 (*Pm12*) in Sari 2017-2018 and DV5 (*Pm3c*) in Gorgan 2017-2018 had the highest coefficient of infection. Moreover, DV27 (*Pm12*) in Gorgan 2015-2016 and DV17 (*Pm2+Pm3d+Mld*) and DV20 (*Pm5a+MlSi2*) in Gorgan 2017-2018 had the least coefficient of infection. The highest genetic distance was observed between DV21 (*Pm4b*) and DV27 (*Pm12*). The pairs of DV9 (*Pm6*) and DV11 (*Pm17*), and DV9 (*Pm6*) and DV12 (*Pm2+Pm6*) had the highest genetic similarity.

The genotypes DV10 (*Pm8*), DV23 (*Pm3g*), DV25 (*Pm6*), DV26 (*Pm5a*), DV28 (*Pm2*) and DV29 (*Pm8*) were located in the upper left quarter which is dedicated to negative values of the first component and positive values of the second component. Vectors of the experiments of Sari 2016-2017 and 2017-2018 were also located in this area. DV29 (*Pm8*) and DV10 (Pm8) had the greatest and least distances from the origin, respectively. Among the genotypes of this area, DV25 (*Pm6*) in Sari 2015-2016, DV28 (*Pm2*) and DV29 (*Pm8*) in Gorgan 2015-2016, DV28 (*Pm2*) and DV25 (*Pm6*) in Sari 2016-2017, DV28 (*Pm2*) in Gorgan 2016-2017, DV29 (*Pm8*) in Sari 2017-2018 and DV25 (*Pm6*) in Gorgan 2017-2018 had the highest coefficients of infection. Moreover, DV26 (*Pm5a*) in Sari 2015-2016, DV23 (*Pm3g*), DV25 (*Pm6*) and DV26 (*Pm5a*) in Gorgan 2015-2016, DV10 (*Pm8*) in Sari 2016-2017, DV10 (*Pm8*) in Gorgan 2016-2017, DV10 (*Pm8*) and DV28 (*Pm2*) in Sari 2017-2018 and DV29 (*Pm8*) in Gorgan 2017-2018 had the lowest coefficients of infection DV26 (*Pm5a*) and DV29 (*Pm8*) showed the highest and DV10 (*Pm8*) and DV23 (*Pm3g*) the lowest genetic distance in this area of the biplot.

The genotypes DV1 (*Pm1*), DV3 (*Pm3a*), DV4 (*Pm3b*), DV6 (*Pm4a*), DV15 (*Pm7*) and DV22 (*Pm3b*) were located in the lower left quarter relating to the negative values of both components. The vectors of Gorgan 2016-2017 and 2017-2018 and Sari 2015-2016 experiments were also located in this area. The genotypes DV15 (*Pm7*) and DV22 (*Pm3b*) had the highest and the smallest distances from the origin, respectively, in this area of the biplot. Among the genotypes located in this region, DV1 (*Pm1*) in Sari 2015-2016, DV3 (*Pm3a*) and DV6 (*Pm4a*) in Gorgan 2015-2016, DV22 (*Pm3b*) in Sari 2016-2017, DV15 (*Pm7*) in Gorgan 2016-2017 and DV15 (*Pm7*) in Gorgan 2017-2018 had the highest coefficients of infection. Besides, DV6 (*Pm4A*) in Sari 2015-2016, DV1 (*Pm1*), DV4 (*Pm3B*) and DV22 (*Pm3B*) in Gorgan 2015-2016, DV1 (*Pm1*), DV3 (*Pm3A*), DV4 (*Pm3B*) and DV6 (*Pm4A*) in Sari 2016-2017, DV4 (*Pm3B*) in Gorgan 2016-2017, DV4 (*Pm3B*) in Sari 2017-2018 and DV6 (*Pm4A*), DV22 (*Pm3B*) and DV1 (*Pm1*) in Gorgan 2017-2018 had the lowest coefficients of infection. Based on the genetic distances, the genotypes DV3 (*Pm3A*) and DV6 (*Pm4A*), had the highest and DV4 (*Pm3B*) and DV15 (*Pm7*) had the lowest similarity in this region of the biplot.

The genotypes DV2 (*Pm2*), DV7 (*Pm4b*), DV8 (*Pm5*), DV14 (*Pm3d*) and DV16 (*Pm1+Pm2+Pm9*) were situated in the lower right quarter which is specific to the positive values of the first component and negative values of the second component. The vector of the Gorgan 2015-2016 experiment was also located in this area. Among the genotypes lodged in this region, DV7 (*Pm4B*) in Sari 2015-2016, DV14 (*Pm3d*) in Gorgan 2015-2016, DV7 (*Pm4B*) in Gorgan 2016-2017, DV2 (*Pm2*) in Sari 2017-2018 and DV2 (*Pm2*) in Gorgan 2017-2018 had the highest coefficients of infection. In addition, DV2 (*Pm2*), DV14 (*Pm3D*) and DV16 (*Pm1+Pm2+Pm9*) in Sari 20152016, DV2 (*Pm2*) in Gorgan 2015-2016, DV8 (*Pm5*), DV14 (*Pm3d*) and DV16 (*Pm1+Pm2+Pm9*) in Gorgan 2016-2017, DV8 (*Pm5*) and DV16 (*Pm1+Pm2+Pm9*) in Sari 2017-2018 and DV16 (*Pm1+Pm2+Pm9*) in Gorgan 2017-2018 had the lowest coefficients of infection. Among genotypes of this region, DV14 (*Pm3d*) and DV7 (*Pm4b*) had the highest and the smallest distances from the origin, respectively. DV2 (*Pm2*) and DV14 (*Pm3d*) had the highest and DV7 (*Pm4B*) and DV8 (*Pm5*) had the lowest genetic distance.

The study of the distance matrix between the genotypes showed that the highest genetic distance existed between DV14 (*Pm3d*) and DV25 (*Pm6*), as well as between the DV2 (*Pm2*) and DV29 (*Pm8*). In addition, DV9 (*Pm6*) and DV12 (*Pm2+Pm6*), as well as DV11 (*Pm17*) and DV12 (*Pm2+Pm6*) had the least distances (the highest genetic similarity) (Figure 4).

Dendrogram of cluster analysis divided the studied differential cultivars into two general groups, each group divided into two subgroups. The first group comprised of 15 genotypes, so that the genotypes DV14 (*Pm3d*) and DV16 (*Pm1+Pm2+Pm9*) were sub-grouped and DV5 (*Pm3c*), DV7 (*Pm4b*), DV8 (*Pm5*), DV9 (*Pm6*), DV11 (*Pm17*), DV12 (*Pm2+Pm6*), DV13 (*Pm2+Pm4B+Pm8*), DV17 (*Pm2+Pm3D+Mld*), DV18 (unknown *Pm*), DV19 (*Mld+Pm2*), DV20 (*Pm5a+MlSi2*), DV24 (*Pm3d*)

and DV27 (*Pm12*) were classified into the other subgroup. The second group included 14 genotypes, so that DV1 (*Pm1*), DV2 (*Pm2*), DV3 (*Pm3a*), DV4 (*Pm3b*), DV6 (*Pm4a*) and DV15 (*Pm7*) were sub-grouped and DV10 (*Pm8*), DV21 (*Pm4b*), DV22 (*Pm3b*), DV23 (*Pm3g*), DV25 (*Pm6*), DV26 (*Pm5a*), DV28 (*Pm2*) and DV29 (*Pm8*) cultivars formed the other subgroup. (Figure 5).

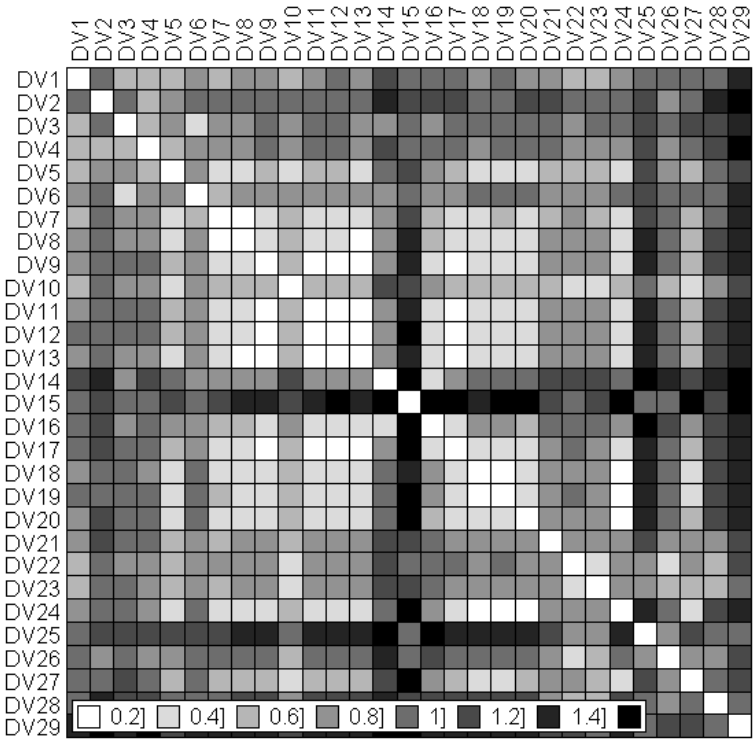

Figure 4. Heat map of genetic distances between differential cultivars for resistance to powdery mildew based on evaluation in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

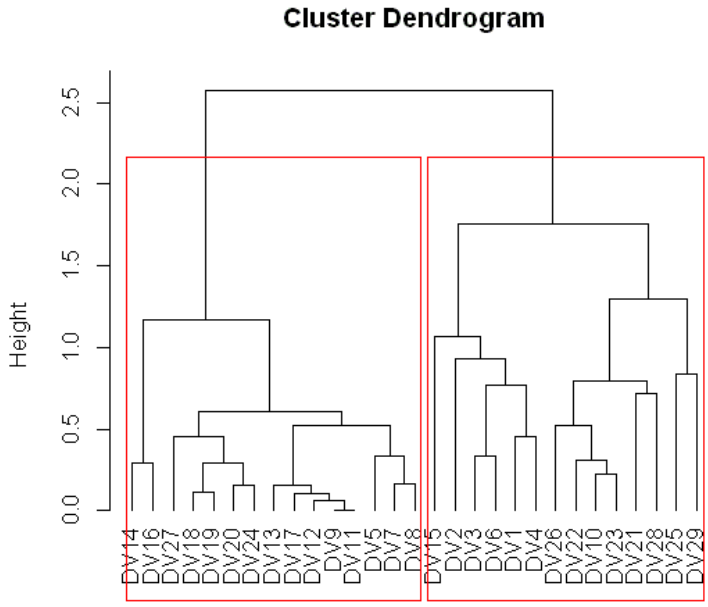

Figure 5. Classification of differential genotypes for resistance to powdery mildew based on evaluation in disease hotspots of Gorgan and Sari, Iran, during years 2015-2018.

DISCUSSION

The overall results of this study indicated virulence changes in the pathogen population during the years of the experiment. This finding has appeared in the various analyses conducted in this study. For example, in the correlation analysis of coefficient of infection in locations and years, some coefficients of correlation were not significant or the calculated coefficients were not large. These results are in line with expectations since shifts in virulence are very common in this pathogen population (Cowger *et al.,* 2018). *B. graminis* f.sp. *tritici* is virtually characterized by high genetic variability (Bougot *et al.,* 2002) which is attributed to mutations, sexual and asexual recombination (Menzies and MacNeill, 1986), high gene flows in fungus populations (Limpert *et al.,* 1999) and host-induced selection (Bougot *et al.,* 2002). These features leads to rapid build-up of virulent isolates, which in turn rapidly overcomes resistance genes widely deployed in wheat cultivars (Abdelrhim *et al.,* 2018; Tan *et al.,* 2018).

Based on correlation coefficients, the highest similarity between the responses of differential cultivars in two regions of the study was observed in 2016-2017. Non-significant correlations between the experiment of Gorgan and other experiments in the year 2015-2016 indicate a significant difference in the virulence pattern of the pathogen in this experiment compared with other experiments. This distinction is well-defined with the opposite direction of vector of this experiment (Gorgan 2015-2016) in relation to vectors of other experiments in the biplot of the first two principal components. Furthermore, the position of the vectors of years and locations in the biplot was consistent with their correlation pattern. In other words, as the Sari 2015-2016 experiment was correlated with the Gorgan 2016-2017 and Gorgan 2017-2018 experiments, the Sari 2015-2016 vector was also situated between the Gorgan 2016-2017 and Gorgan 2017-2018 vectors. In addition, the vector of Sari 2016-2017 experiment, which was found to be correlated with Gorgan 2016-2017 and Sari 2017-2018 experiments, was located between the vectors of these two experiments. The highest number of significant correlations belonged to the coefficients of correlation in Gorgan 2016-2017 experiment with other experiments. Accordingly, the vectors of Gorgan 2017-2018 and Sari 2015-2016 from one side and those of Sari 2016-2017 and Sari 2017-2018 from the other side were placed around the Gorgan 2016-2017 vector.

The lowest value of w_i^2 for Shamrock (unknown *Pm*), Maris Dove (*Mld*+*Pm2*), Ronos (*Pm4b*), Broom (*Pm3d*) and Sonora/8*Chancellor (*Pm3c*) indicated that these genotypes expressed most stable resistance reactions. On the other hand, the genotypes Maris Dove (*Mld*+*Pm2*), Shamrock (unknown *Pm*), Broom (*Pm3d*), Axona (*Pm2*+*Pm3d*+*Mld*) possessed the lowest mean of coefficient of infection. Since Axona (*Pm2*+*Pm3d*+*Mld*) also had a low value of w_i^2, it can be concluded that the genotypes Maris Dove (*Mld*+*Pm2*), Shamrock (unknown *Pm*), Broom (*Pm3d*) and Axona (*Pm2*+*Pm3d*+*Mld*) expressed effective resistance over the years and locations of the study. These results are in concordance with other analyses since the respective genotypes were located in the same region of biplot through PCA or in the same group in the dendrogarm of cluster analysis. Zahravi *et al.* (2018) also observed that Shamrock (unknown *Pm*), Normandie (*Pm1*+*Pm2*+*Pm9*), Axona (*Pm2*+*Pm3d*+*Mld*), Maris Dove (*Mld*+*Pm2*) and Wembley (*Pm12*) were resistant to all ten studied pathotypes collected across Iran. Therefore, the effectiveness of resistance expressed by Maris Dove (*Mld*+*Pm2*), Shamrock (unknown *Pm*) and Axona (*Pm2*+*Pm3d*+*Mld*) observed in the present study is consisted with the report of Zahravi *et al.* (2018).

The interesting point with these results is the alternative presence of the genes *Pm2*, *Pm3d* and *Mld* in these four genotypes. High level of resistance of *Pm3* and *Mld* to some powdery mildew populations like the French ones has been already reported (Bougot *et al.,* 2002). The *Mld* has its origin in tetraploid durum wheat (Wolfe, 1967). This gene is located on chromosome 4B (Alam *et al.,* 2011) and it is probable to be recessive (Johnson *et al.,* 1969). The *Pm2* is known to be located on the short arm of wheat chromosome 5D (McIntosh and Baker, 1970). The *Pm3* is a single, dominant locus (Tommasini *et al.,* 2006) located on the short arm of chromosome 1A (Briggle, 1969) carrying 17 so far identified functional alleles (Yahiaoui *et al.,* 2004, 2006; Srichumpa *et al.,* 2005; Bhullar *et al.,* 2009, 2010; Koller *et al.,* 2018). *Pm3* alleles have been extensively and successfully utilized in breeding programs (Tommasini *et al.,* 2006). Some alleles of *Pm3* have showen effectiveness in western and northern European countries (Bougot *et al.,* 2002). The *Pm3d* is among the rare *Pm* genes not overcome in Europe or the USA (Chen and Chelkowski, 1999; Persaud and Lipps, 1995; Bougot *et al.,* 2002). Therefore, the results of effectiveness of resistance genes obtained in this study, in particular with respect to *Pm3d* and *Mld*, are in accordance with past research studies mentioned above. Among differential cultivars

used in this study, Ralle also carried *Pm3d* in addition to Broom. This cultivar expressed a low mean of coefficient of infection (MCI) with low value of w_i^2, although its MCI and w_i^2 were not among the lowest values. Since a compatible pathogenicity was observed on Ralle in Gorgan 2015-2016 experiment, effective resistance exhibited by Broom may be conferred by other unknown resistance genes so that no virulence was observed for them over the years and locations of the experiment.

On the contrary, Ulka/8*Chancellor (*Pm2*) was among the most variable cultivars for resistance reaction based on w_i^2. Galahad (*Pm2*) also had a high MCI. Among the superior cultivars identified in this study for resistance to powdery mildew, two genotypes (Maris Dove and Axona) both carried *Pm2,* however as it is apparent, the presence of *Pm2* in these cultivars is along with other major resistance genes (accompanying by *Mld* in Maris Dove and by *Pm3d*+*Mld* in Axona). These results indicate that either *Pm2* lacks an effective resistance or its effectiveness depends on epistatic reactions by the other effective resistance genes.

In the present study, Normandi (*Pm1*+*Pm2*+*Pm9*) and Ralle (*Pm3d*), appeared susceptible in Gorgan 2015-2016 experiment, despite their resistant expression in other experiments. Defeat of the resistance of *Pm1*+*Pm2*+*Pm9* was already reported by Karimi-Jashni *et al.* (2006) for the first time. In addition to consistence with Karimi-Jashni *et al.* (2006) observations, this finding suggests that virulence for *Pm1*+*Pm2*+*Pm9* is still prevalent and emphasizes on avoiding the use of this gene combination. These observations also confirm that pathogen population being dynamic towards the creation of new pathotypes, which even neutralize resistance effects of genes in combination, indicating the need to correctly apply resistance genes in the infected areas and, on the other hand, the need to identify the new sources of resistance genes.

Another point is the change of Holger (*Pm6*) response from the resistance range toward susceptibility range over the years of the experiment. Among the genotypes used in the differential cultivars, DV9 (NK-747) also carried *Pm6*. The reaction difference between these two genotypes, despite having the same resistance gene (*Pm6*), could be due to another unknown resistance gene present in the DV9 (NK-747) for which there has not been any virulence over the regions and years of the experiment. The difference in resistance response between Holger and NK-747 was also reported previously by Zahravi *et al.* (2018). Therefore, due to the increased virulence for this gene, it should be used with caution in cultivars cultivated in these areas. This difference in the response pattern was also observed between DV2 (Ulka/8*Chancellor) and DV28 (Galahad), despite having *Pm2* in common, as well as in DV10 (Disponent) and DV29 (Ambassador) genotypes, with common *Pm8* resistance gene, which in turn could be due to another unknown gene in these genotypes. These differences were also previously reported by Zahravi *et al.* (2018).

CONCLUSION

The population of wheat powdery mildew fungi has dynamic characteristics in the infected areas so that there are many variations in virulence/avirulence factors. These changes undermine the effect of resistance genes and emphasize on the importance of continuous monitoring of the population of the pathogen, and on the other hand, necessitate the quest for new resistance genes with high effectiveness. In this study, Maris Dove (*Mld*+*Pm2*), Shamrock (unknown *Pm*), Broom (*Pm3d*) and Axona (*Pm2*+*Pm3d*+*Mld*) were identified as sources of highly effective resistance genes that could be applied in the studied regions. Moreover, it is important to take caution in the use of gene combinations which were previously reported as having effective resistance, since virulence was observed for some of these combinations in the present study.

ACKNOWLEDGMENTS

We gratefully acknowledge the Seed and Plant Improvement Institute (SPII) for providing financial support and facility for this research.

REFERENCES

Abdelrhim A., Abd-Alla H. M., Abdou E. S., Ismail M. E., and Cowger C. (2018). Virulence of Egyptian *Blumeria graminis* f.sp. *tritici* population and response of Egyptian wheat cultivars. *Plant Disease,* 102(2): 391-397.

Alam M. A., Xue F., Wang C., and Ji W. (2011). Powdery mildew resistance genes in wheat: identification and genetic analysis. *Journal of Molecular Biology Research,* 1(1): 20-39.

Bhullar N. K., Street K., Mackay M., Yahiaoui N., and Keller B. (2009). Unlocking wheat genetic resources for the molecular identification of previously undescribed functional alleles at the *Pm3* resistance locus. *Proceedings of the National Academy of Sciences,* 106(23): 9519-9524.

Bhullar N. K., Zhang Z., Wicker T., and Keller B. (2010). Wheat gene bank accessions as a source of new alleles of the powdery mildew resistance gene *Pm3*: a large scale

allele mining project. *BMC Plant Biology,* 10(1): 88-101.
Bougot Y., Lemoine J., Pavoine M. T., Barloy D., and Doussinault G. (2002). Identification of a microsatellite marker associated with *Pm3* resistance alleles to powdery mildew in wheat. *Plant Breeding,* 121(4): 325-329.
Briggle L.W. (1969). Near-Isogenic lines of wheat with genes for resistance to *Erysiphe graminis* f.sp. *tritici*. *Crop Science,* 9(1): 70-72.
Chen Y., and Chelkowski J. (1999). Genes for resistance to wheatpowdery mildew. *Journal of Applied Genetics,* 4(40): 317-334.
Cowger C., Mehra L., Arellano C., Meyers E., and Murphy J. P. (2018). Virulence differences in *Blumeria graminis* f.sp. *tritici* from the central and eastern United States. *Phytopathology,* 108(3): 402-411.
Geng M., Zhang J., Peng F., Liu X., Lv X., Mi Y., Li Y., Li F., Xie C., and Sun Q. (2016). Identification and mapping of *MLIW30*, a novel powdery mildew resistance gene derived from wild emmer wheat. *Molecular Breeding*, 36(9): 130. https://doi.org/10.1007/s11032-016-0553-0.
Imani Y., Ouassou A., and Griffey C. A. (2002).Virulence of *Blumeria graminis* f.sp. *tritici* populations in Morocco. *Plant Disease*, 86(4): 383-388.
Janáková E., Jakobson I., Peusha H., Abrouk M., Škopová M., Šimková H., Šafář J., Vrána J., Doležel J., Järve K., and Valárik M. (2019). Divergence between bread wheat and *Triticumm ilitinae* in the powdery mildew resistance *QPm.tut-4A* locus and its implications for cloning of the resistance gene. *Theoretical and Applied Genetics,* 132(4): 1061-1072.
Johnson R., Wolfe M. S., and Scott P. R. (1969). Diseases of wheat. *Plant Breeding Institute Annual Report,* pp. 116.
Karimi-Jashni M., Torabi M., Roustaie A., Etebarian H., Okhovat S., Razavi M., and Yazdanpanah F. (2006). Pathotypes of *Blumeria graminis* f.sp. *tritici* the causal agent of powdery mildew from some regions of Iran. *Seed and Plant,* 22(2): 257-271.
Koller T., Brunner S., Herren G., Hurni S., and Keller B. (2018). Pyramiding of transgenic *Pm3* alleles in wheat results in improved powdery mildew resistance in the field. *Theoretical and Applied Genetics,* 131(4): 861-871.
Limpert E., Godet F., and Müller K. (1999). Dispersal of cereal mildews across Europe. *Agricultural and Forest Meteorology,* 97(4): 293-308.
Liu N., Bai G., Lin M., Xu X., and Zheng W. (2017). Genome-wide association analysis of powdery mildew resistance in US Winter Wheat. *Scientific Reports,* 7(1): 11743.
Liu N., Liu Z. L., Gong G., Zhang M., Wang X., Zhou Y., Qi X., Chen H., Yang J., Luo P., and Yang, C. (2015). Virulence structure of *Blumeria graminis* f.sp. *tritici* and its genetic diversity by ISSR and SRAP profiling analyses. *PlOS One*, 10(6): e0130881.
Ma P., Xu H., Xu Y., Li L., Qie Y., Luo Q., Zhang X., Li X., Zhou Y., and An D. (2015). Molecular mapping of a new powdery mildew resistance gene *Pm2b* in Chinese breeding line KM2939. *Theoretical and Applied Genetics*, 128(4): 613-622.
McIntosh R. A., and Baker E. P. (1970). Cytogenetic studies in wheat. IV. Chromosome location and linkage studies involving the *Pm2* locus for powdery mildew resistance. *Euphytica,* 19: 71-77.
Menzies J. G., and MacNeill B. H. (1986). Asexual recombination in *Erysiphe graminis* f.sp. *tritici*. *Canadian Journal of Plant Pathology,* 8(4): 400-404.
Monazzah M., Torabi M., Rezaie S., and Razavi M. (2008). Pathotypes of *Blumeria graminis* f.sp. *tritici*, the causal agent of wheat powdery mildew from some regions of Iran. *Seed and Plant,* 24(1): 161-176.
Niewoehner A. S., and Leath S. (1998). Virulence of *Blumeria graminis* f.sp. *tritici* on winter wheat in the eastern United States. *Plant Disease*, 82(1): 64-68.
Persaud R. R., and Lipps P. E. (1995). Virulence genes and virulence gene frequencies of *Blumeria graminis* f.sp. *tritici* in Ohio. *Plant Disease,*79(5): 494-499.
Razavi M., Karimi-Jashni M., Dehghan M. A.,Safavi S. A., and Barari H. (2010). Study on the variability for virulence in *Blumeria graminis* f.sp. *tritici* cause of wheat powdery mildew using trap nursery in Iran. *Applied Entomology and Phytopathology,* 78(1): 97-106.
Saari E. E., and Prescott J. M. (1975). A scale for appraising the foliar intensity of wheat disease. *Plant Disease Reporter,* 59: 377-380.
Shannon, C. E. (1948). A mathematical theory of communication. *Bell System Technical Journal,* 27: 379-423.
Srichumpa P., Brunner S., Keller B., and Yahiaoui N. (2005). Allelic series of four powdery mildew resistance genes at the *Pm3* locus in hexaploid bread wheat. *Plant Physiology,* 139(2): 885-895.
Stubbs R.W., Prescott J. M., Saari E. E., and Dubin H. J. (1986). Cereal disease methodology manual. CIMMYT, Mexico, pp. 46.
Švec M., and Miklovičová M. (1998). Structure of populations of wheat powdery mildew (*Erysiphe graminis* DC f.sp. *tritici* Marchal) in Central Europe in 1993–1996: I. Dynamics of virulence. *European Journal of Plant Pathology,* 104(6): 537-544.
Tan C., Li G., Cowger C., Carver B. F., and Xu X. (2018). Characterization of *Pm59*, a novel powdery mildew resistance gene in Afghanistan wheat landrace PI 181356. *Theoretical and Applied Genetics,* 131(5): 1145-1152.
Tommasini L., Yahiaoui N., Srichumpa P., and Keller B. (2006). Development of functional markers specific for seven *Pm3* resistance alleles and their validation in the bread wheat gene pool. *Theoretical and Applied Genetics,* 114(1): 165-175.
Wolfe M. S. (1967). Physiologic specialization of *Erysiphe graminis* f. sp. *tritici* in the United Kingdom*, 1964-5. Transactions of the British Mycological Society,* 50: 631-640.
Wricke G. (1962). Uber eine methode zur erfassung der okologischen streubreite in feldversuchen. *Zeitschrift Fur Pflanzenzuchtung-Journal of Plant Breeding,* 47: 92-96.
Yahiaoui N., Brunner S., and Keller B. (2006). Rapid generation of new powdery mildew resistance genes after wheat domestication. *The Plant Journal,* 47(1): 85-98.
Yahiaoui N., Srichumpa P., Dudler R., and Keller B. (2004) Genome analysis at different ploidy levels allows cloning of the powderymildew resistant gene *Pm3b* from hexaploid wheat. *The Plant Journal,* 37(4): 528-538.
Yu S., Long H., Deng G., Pan Z., Liang J., and Yu M. (2015). Localization of the powdery mildew

resistance gene *Pm07J126* in wheat (*Triticum aestivum* L.). *Euphytica*, 205(3): 691-698.

Zahravi M., Mansourian M. R. ,Azimi H. , Dehghan M. A. , Allahyari N., and Alitabar, R. (2018). Investigation of resistance of Iranian bread wheat landraces to powdery mildew disease. *Iranian Journal of Field Crop Science,* 49(3): 151-165.

Zeng F. S., Yang L. J., Gong S. J., Shi W. Q., Zhang X. J., Wang H., Xiang L. B., Min-Feng X. U. E., and YU D. Z. (2014). Virulence and diversity of *Blumeria graminis* f.sp. *tritici* populations in China. *Journal of Integrative Agriculture*, 13(11): 2424-2437.