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Evaluation of Resistance in Bread Wheat to Against in Iranian Pathotypes of Powdery Mildew Caused by *Blumeria graminis* f.sp.

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Abstract

The resistance of 198 bread wheat genotypes from wheat collection of National Plant Gene Bank of Iran was assessed to powdery mildew. The genotypes were planted in the greenhouse in a randomized complete block design with three replications and separately inoculated by ten pathotypes of powdery mildew. The genotypes were inoculated at seedling stage with the infected leaves. After seven to ten days, each infected genotype was recorded on a scale of 0 to 4. Descriptive statistics that were calculated for the reaction of genotypes showed the lowest average of infection type in genotype 172. The principal component analysis were used to distinguish genotypes. The amounts of variation in main two principal components were justified 81.8%. According to the biplot diagram of the main two components resistant genotypes 142 and 172 were distinct from other genotypes with the attention in Dendrogram of cluster analysis we were located the genotypes in two resistant and susceptible groups. The results showed that the potential of resistance in powdery mildew of bread wheat collection in National Plant Gene Bank of Iran which can be used in breeding programs.

Keywords

Bread Wheat, Powdery Mildew, Pathotype, Genetic Resource

1. Introduction

Powdery mildew caused by *Blumeria graminis* f. sp. *tritici* is one of the most important diseases. In the recent years a lot of damage in humid and wet conditions, were published and it has seem a worldwide distribution (Razavi and Patpur, 2007). Use of chemical pesticides lead to adverse effects on the environment, other methods were needed to combine use of resistant cultivars (Alitabar, R. 2010). Powdery mildew is one of the important diseases of wheat in Iran but precisely the extent of damage is unknown. Manochehri in 1964, Sharif and Ershad in 1966, Mohammadi Dustdar in 1967, Behdad and Daftari 1968 were reported this diseases influence in Iran (Ershad, 2005). In recent years, due to new planting resistance varieties like stripe rust and etc, but we

had some varieties that susceptible to some diseases such as powdery mildew, especially spread has in the provinces of Mazandaran and Golestan has (Behruzin and Forutan, 2002). Damadzadeh and Hasanpur (2001) were reported the Average contamination of this disease in the years 1988-1990 in Esfehan 4/8 percent and they approximately were estimated 85% of the province's wheat fields infected with relatively severe. Pollution tests on susceptible varieties (Sorkhtokhm) and Murku were reported In 1994, in Karaj. Most research about wheat powdery mildew took place in Europe. Powdery mildew is the most important diseases of cereals in the UK and annually was damage, 15 percent of the yield (Reader and Miller, 2001). Also the damage of this disease were reported 45% in New Zealand, and 30% have been reported in India. Leath and Heun in 2000, reported wheat powdery mildew could decrease the amount of product in United States up to 34%. The aim of this research was to identify the resistance cultivar sources to powdery mildew in wheat collection of National Plant Gene Bank of Iran.

2. Material and Method

The resistance of 198 genotypes in *T. aestivum* were assessed for powdery mildew from wheat collection of National Plant Gene Bank of Iran. For this purpose, several isolates of fungus were collected from disease hotspots in Moghan, Kelardasht, Mazandaran (Baykola), Gorgan and Gonbad, in 2011. The isolate were transferred to the greenhouse and propagated and purified pests and plant diseases, in the Medical Plant Research Institute. Thirty differential cultivars were used to identify the virulence factors in the isolates. The genotypes were planted in the greenhouse in a randomized complete design with three replications and separately inoculated by ten pathotypes of powdery mildew. Seedling genotypes in the two-leaf stage were inoculated by a pathogen. The first inoculation was done with water sprayed on the leaves and then inoculated with a spore on friction were performed to transfer the desired genotype. The pots were kept at the temperature of 18 -20 degrees, Seven to ten days after inoculation, the reaction resistance genotypes were evaluated based on a scale of zero to four (Mains and Dietz 1930). Using descriptive statistics were identified to variation between genotypes and pathotypes. The principal component analysis was used to distinguish genotypes. Biplot was drawn using the first two principal components. Grouped genotypes were carried out in response to disease cluster analysis method based on Euclidean distance.

3. Results

Genotypes 135, 158, 177 and 187 regarding the pattern of resistance to pathotypes of the same isogenic numbers 3, 6, 9, 10, 19, 24 and 29 were. So probably, they have genes *Pm3a, Pm4a, Pm6, Pm8 and Pm2*. And also, Genotypes 142 and 172 regarding the pattern of resistance to pathotypes of the same isogenic numbers 16, 17, 18, 20 and 28 were. Which probably, they have genes *Pm1, 2, 9*.

Principal was used components analysis in order to further differentiate from each genotype. Using principal component analysis showed that the two main components would recommend 81.8% of the total variation.

Table 1. *Coefficients of the main components in bread wheat genotypes by ten pathotypes of powdery mildew.*

Components	1	2
Moghan1	.59	.65
Moghan2	.83	-.17
Klar2	.70	.60
Klar3	.86	-.06
Klar4	.83	.28
Bay_D	.83	-.27
Bay_G	.87	-.37
Gorgan_H	.85	-.38
Gorgan_I	.92	-.19
Gonbad	.89	.24

In the first principal component all the coefficients were almost positive sign, numerical value and near together. Greater numeric value indicated infection type of sensitivity. So higher numeric value of this component reflected the greater sensitivity of these components. Less genotype resistant becomes greater resistance genotype. So we absorbed resistance genotypes in the bottom lefts biplot of the main two principal components. According to these genotypes 142, 153, 155, 172, 179 and 180 we were found that be more resistant than other genotypes.

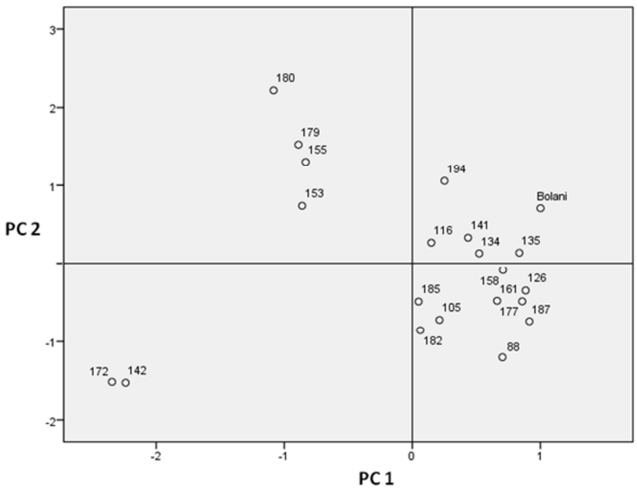

Figure 1. *Biplot two main components in bread wheat genotypes to powdery mildew than ten pathotypes.*

By examining the coefficients of the isolates in the second principal component, isolates Klar2 and Moghan1 had larger coefficients than other isolates. The genotypes that are located in the upper part of the biplot are sensitive to these isolates.

The 142 and 172 genotypes were distinct from each genotype. Cluster analysis is one of the appropriate methods to determine the affinity and proximately isolates so for cutting area we were drown the line within 0-5% of similarity. Finally the Cluster analyses were divided into 4 groups.

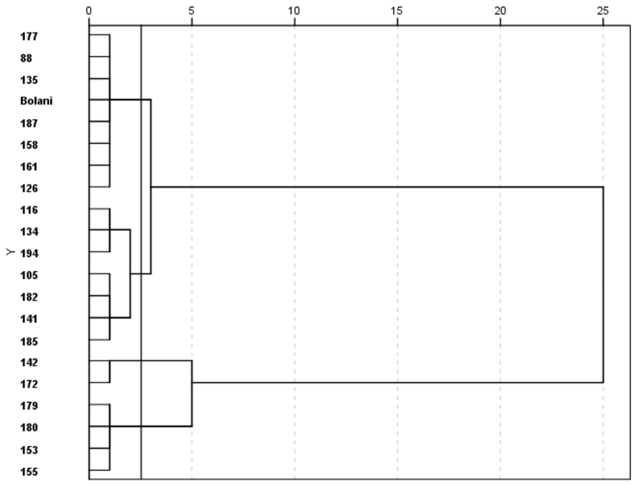

Figure 2. *Dendrogram of cluster analysis in the evaluation of bread wheat genotypes by ten powdery mildew pathotypes.*

Finally, Cluster analysis dendrogram of isolates were drawn based on reaction of each genotype. The genotype groups obtained in the cluster analysis can be seen between the results of two methods of cluster analysis and principal component analysis. The genotypes 142, 153, 155, 172, 179 and 180 have been identified as resistant to powdery mildew in bread wheat genotypes. Descriptive statistics for reaction to isolates of genotypes are presented in Table 2.

172 genotype that had the lowest number (s) (0.7) and is therefore known as the resistant genotype. The genotypes of 142, 153, 155, 179 and 180 were isolated and then tested with the numerical average of less than 2.5 (in tolerance) patients. The highest coefficient of variation was also belong to genotype 180 (83.90%) and in the most diverse types of pollution than other genotypes, indicating that the specific resistance of this genotype. The results showed that the potential of resistance in powdery mildew of bread wheat collection of National Plant Gene Bank in Iran which can be used in breeding programs in the future.

Table 2. *Descriptive statistics related to the evaluation of bread wheat genotypes by ten powdery mildew pathotypes.*

Genotype	N	Range	Minimum	Maximum	Mean	Std. Deviation	cv%
105	10	1.67	2.33	4	3.067	0.654	21.312
116	10	2.33	1.67	4	3.033	0.736	24.270
126	10	1.50	2.50	4	3.650	0.481	13.173
134	10	1.67	2.33	4	3.350	0.441	13.152
135	10	0.67	3.33	4	3.617	0.284	7.848
141	10	1.67	2.33	4	3.267	0.644	19.716
142	10	1.67	0.00	1.67	0.867	0.526	60.678
153	10	3.33	0.00	3.33	1.983	0.951	47.939
155	10	4.00	0.00	4	2.033	1.082	53.233
158	10	1.33	2.67	4	3.467	0.391	11.286
161	10	1.67	2.33	4	3.433	0.545	15.887
172	10	1.33	0.00	1.33	0.700	0.483	69.007
177	10	1.00	3.00	4	3.633	0.367	10.096
179	10	3.00	0.33	3.33	1.967	1.048	53.269
180	10	3.67	0.00	3.67	1.750	1.468	83.902
182	10	2.33	1.67	4	2.900	0.649	22.374
185	10	2.67	1.33	4	2.883	0.737	25.569
187	10	1.33	2.67	4	3.667	0.471	12.856
194	10	2.00	2.00	4	3.100	0.754	24.336
88	10	1.67	2.33	4	3.517	0.640	18.212
Bolani	10	0.33	3.67	4	3.833	0.176	4.583

4. Discussion

Powdery mildew had been a major wheat disease in humid and wet conditions of Iran but also appeared sporadically in Iran. In the present study, we were assessed the resistance of 198 *T.aestivum* genotypes to powdery mildew. Breeding for resistance, as an alternative approach to chemical control of powdery mildew, has been very successful, inexpensive method that no need for training of farmers and special equipment and environmentally safe (Finckh *et al*., 1999). In this study, we could find some genotypes like 135, 158, 177 and 187 probably have *Pm3a, Pm4a, Pm6, Pm8* and *Pm2* genes. The presence of *Pm1a, Pm2, Pm4b, Pm5, Pm6, Pm8* and *Pm9*, alone or in combinations were identified in 12 of the 107 Swedish landraces and cultivars (Hysing *et al*.,

2007). 142 and 172 as examples of resistant genotypes were distinct from other genotypes. Genotypes 142 and 172 were regarding the pattern of resistance to pathotypes of the same isogenic numbers 16, 17, 18, 20 and 28, which probably have *Pm1,2,9* genes. The pathotype identified in 2000 and 2005, the standard varieties contain resistance genes *Pm1,2,9* also found no pathogenicity by Monazah. This indicates that although the population of pathogen virulence factors for individual resistance genes present in this region, the accumulation of several genes in a variety could cause lasting resistance against the disease (Monazzah *et al*., 2009).

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