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Evaluation of infection type and inheritance of resistance to powdery mildew in two crosses in barley

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Abstract

In order to evaluate the gene number, gene effect and heritability to powdery mildew in barley Two resistant cultivars were crossed with a susceptible cultivar. In a field study, the parents (P_1, P_2) and the generations (F_1, F_2 and F_3) of two crosses were evaluated in a randomized complete block design with three replications. The infection type of flag leaf and the whole plant was assessed in booting stage using Saari, E.E., and Prescott method**.** The Scaling test indicated that the effects of additive, dominant and epistatic, and mainly additive × additive effect has an important role in controlling to resistance to powdery mildew in barley. In the cross Hebe × Arigashar, using χ^2 test for segregating F_2 generation , it was determined that duplicate dominant epistasis shows 15:1 ratio. Also in the cross Igri × Arigashar, using χ^2 test the F_2 generation, it was determined that the distribution of F_2 generation of threefold dominant epistatic shows 35:1 ratio. general heritability of infection type in two crosses were estimated respectively 68% and 88%. Depending on traits and crosses, the gene number ranged from 1-2 and 3-6.

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Introduction

Powdery mildew is one of the most important diseases in barley; however, its different stages of virulence are still unknown (Brown, 1991). It is caused by the fungus *Blumeria graminis f. sp. hordei DC. f. sp. hordei* Em. Marchal (Bgh) which infects leaves and the atmosphere and makes a lot of economic damage in north America, north and central Europe, where barley is produced (Antonio, 2005). Powdery mildew is especially harmful in barley that produced for malting. This fungus has a short generation which is less than 8 days (Jenkyn,1978) and produces spores that are spread by the wind. Reciprocal effects between pathogenicity and resistance conforms the gene for gene system (Jorgenson, 1988). Since Biffen started to study genetically resistance to powdery mildew, more than 100 resistant genes to powdery mildew have been identified. In Europe, barley reformers usually use resistant genes like $Mla_6, Mla_7, Mla_9, Mla_{12}, Mla_{13}$ which belong to Mla locus. They also use resistant alleles, $Ml_{ra}, Ml_k, Ml_{la}, Ml_g, Ml_h$, which originated from the local races of barley in West Asia, northern Ethiopia, North Africa and Morocco. However, all these genes were gradually overcome by new virulent strains within 4-5 years (Bayles, 1990). In Iran, this disease was first obsereved in barley by Esfandiari in 1326 and then in wheat by Manouchehri in 1343. Powdery Mildew is prevalent in almost all areas in Iran and it was observed in Azerbaijan, coasts of the Caspian Sea and Central Provinces, Fars, Khuzestan and Esfahan.

The damage has been estimated 15 to 25% in regions such as Gorgan, Moghan, and Sari, where the disease is severe , and 7 to 10% in other regions like Khorasan, Fars and Khuzestan (Patpour, 1998). Today, the disease has spread throughout the world. It causes the maximum damage in cold and humid climates, though it also occurs in semiarid areas. Two main methods have been proposed to control powdery mildew which include selecting varieties with greater resistance to disease, and using pesticides. The problem of this method is that it is often observed that pathogens are able to escape the resistant cultivars directly due to high compatibility rate, short generation and high sexual recombination throughout the year. Another important reason for the rapid spread of the disease is the natural spread of pathogens. Compatible pathotype have recently proved to be able to be transported rapidly by the wind and scattered everywhere. The most reliable way to control this disease is using resistant cultivars. Considering the fact that barley landraces are one of the major sources of resistance genes pool for preparing new commercial varieties (Behrav & Levy, 1988), it is necessary to use the local barley in Iran which is one of the main areas of barley varieties in order to identify disease resitance sourses in breeding programs (Harlen,1979). Few details are available about the mode of quantitative inheritance of resistance to powdery mildew in maturity in barley. The aim of this study was to investigate the determination of quantitative inheritance of resistance to powdery mildew in adualt stage. The results will help the researcher in fulfilling breeding programs for disease resistance.

Materials and methods

Plant materials and experiment design

Two resistant cultivars (Hebe, Igri) were crossed with a susceptible cultivar (Arigashar) to powdery. The Parents (p_1 و p_2) and the generations F_1, F_2 and F_3 of the two crosses of Hebe × Arigashar and Igri × Arigashar, were seeded in a randomized complete block design with three replicates on 1-metre lines with 30cm between lines and 10cm between plants in Karaj Research Station of Cereal Research Centre. Each replication consisted of parents and F_1s in one row , and F_2 and F_3 generations in 7 and 64 rows respectively. In order to have a uniform disease spread, the susceptible cultivar, Afzal was planted between each 20 rows and also around the experimental field. Critical observation to fight weeds and also Irrigation were done during the season. Saari and Prescott (1975) 0-9 scale was used in order to record the infection type of the Flag leaf and whole

plant, based on the disease progression on the surface of the flag leafs and its spread from the lower leaves into clusters, where 0 and 9 were completely resistant and fully susceptible respectively.

Data analysis methods

Means generations analysis were used to estimate the gene number, gene action and heritability in both crosses and also for traits of infection type of flag leaf and whole plant. In order to determine the types of interactions of genes in F_2 generation plants, the phenotypic classification which contains all F_2 plants was performed by χ^2 s test. To determine the degree of genetic dominance, a method by Mather & Jinks (1982) was used for pollinated plants. For estimating the average degree of dominance, variance components i.e., D (additive) and H (dominance deviations) were used and the average degree of dominance was calculated using the formula $\sqrt{\frac{H}{D}}$ (Ahmadi, 1992).

Gene effects were estimated using the genetic analysis of mean generation analysis based on a model from Mather & Jinks (1982). In this model, the overall mean of each trait is as follows:

$$Y = m + a[d] + \beta[h] + a^2[i] + 2a\beta[j] + \beta^2[l]$$

Components of the formula include: $\overline{Y}$ generation mean, m: mean of all generations, $[d]$ total additive effects, $[h]$ total dominance effects, $[i]$: the total additive interaction effects, $[l]$ total dominance interaction effects , $[j]$ total additive and dominance effects, and a ، β ، a^2 ، $2a\beta$ and β^2 , the coefficients of each of the different genetic parameters by the weighted least squares method. First, in case of significant, χ^2 was calculated for the simple additive - dominance model in goodness of fit tests for each of the characters with the lowest χ^2 . At the end, two, three, four, or five parameter model fitted for each trait was given (Naghavi, 2001; Mather, 1982).

Using the chi square test with four, three, two and one degree of freedom (scaling test), all models were compared by goodness of fit test (Mather & Jinks, 1982; Ghannadha, 1999). The following formulas were used to calculate genetic variance components.

$$V_{F_2} = \frac{1}{3}D + \frac{1}{4}H + E_1,$$

$$V_{\overline{F_3}} = \frac{1}{2}D + \frac{1}{16}H + E_2$$

$$W_{F_2/F_3} = \frac{1}{2}D + \frac{1}{8}H,$$

$$\overline{V}_{F_3} = \frac{1}{4}D + \frac{1}{8}H + E_1$$

In the above formulas, by creating the four normal equations using weighted least squares method of the opposite of variance and multiplying matrices,s the values E_2, E_1, H, D were calculated using Mini Pro tabs. The number of genes was calculated using Cockerham's model (Cockerham, 1988) by the following formula.

GNF$_1$ $$n = \frac{\left(\overline{p_1} - \overline{p_2}\right)^2}{\left[8\left(\hat{\sigma}_{F_2}{}^2 - \hat{\sigma}_{F_1}{}^2\right)\right]}$$

GNF$_2$

$$n = \frac{\left(\overline{p_1} - \overline{p_2}\right)}{\left(8\left[\hat{\sigma}_{F_2}{}^2 - \left(0/5\hat{\sigma}_{F_1}{}^2 + 0/25\hat{\sigma}_{P_1}{}^2 + 0/25\hat{\sigma}_{P_2}{}^2\right)\right]\right)}$$

Estimated heritability for different traits using population variance was calculated by the following formula (Burnette & Whithe, 1985; Van ginkel & Schareh, 1987).

$$h_{bs}{}^2 = \frac{\left(\hat{\sigma}_{F_2}{}^2 - \hat{\sigma}_e{}^2\right)}{\hat{\sigma}_{F_2}{}^2}$$

Environmental variance (non-inherited), based on the mean of three generations without segregating P_1, P_2 and F_1 were calculated with different methods

(kearsey and Pooni, 1996). As a result,the various formulas are obtained for estismating heritability.

$$HF_1) \quad \hat{\sigma}_e^{\,2} = \frac{\left(\hat{\sigma}_{p_1}^{\,2} + \hat{\sigma}_{p_2}^{\,2}\right)}{2}$$

$$HF_2) \quad \hat{\sigma}_e^{\,2} = \sqrt{\hat{\sigma}_{p_1}^{\,2} + \hat{\sigma}_{p_2}^{\,2}}$$

$$HF_3) \quad \hat{\sigma}_e^{\,2} = \hat{\sigma}_{F_1}^{\,2}$$

$$HF_4) \quad \hat{\sigma}_e^{\,2} = \sqrt[3]{\hat{\sigma}_{p_1}^{\,2} \times \hat{\sigma}_{p_2}^{\,2} \times \hat{\sigma}_{F_1}^{\,2}}$$

$$HF_5) \quad \hat{\sigma}_e^{\,2} = \frac{\left(\hat{\sigma}_{p_1}^{\,2} + \hat{\sigma}_{p_2}^{\,2} + \hat{\sigma}_{F_1}^{\,2}\right)}{3}$$

$$HF_6) \quad \hat{\sigma}_e^{\,2} = \frac{\left(\hat{\sigma}_{p_1}^{\,2} + \hat{\sigma}_{p_2}^{\,2} + \hat{\sigma}_{F_1}^{\,2}\right)}{4}$$

Results and discussion

Identification of gene action to powdery mildew

The results of the weighted variance analysis was significant for traits including infection type of flag leaf, infection type of total plant in two crosses. The significant differences between generations indicates the possibility of the genetic analysis of their inheritance. In table 1, the mean of measured traits for two crosses in different generations indicates that the susceptible parent (Arigashar) has greater infection type than the two resistant parents (Hebe and Igri).

The observed continuous variation in F_2 and F_3 could be due to genetic effects or genotype–environment interaction. But continuous variation does not necessarily imply polygenetic inheritance (Thompson, 1975). Continuous variation may even be controlled monogenically which is subject to large environmental effects (Hoff and Mc Donald, 1980).

The estimated five fold genetic effects indicated that the four parameters model containing m ،$[d]$ ، $[h]$ ،$[i]$ is appropriate for inflection type of flag leaf of two crosses and also for the infection type of whole plant in cross Igri × Arigashar, but the best model for infection type of whole plant in cross Hebe × Arigashar is the four parameters model containing m ،$[d]$ ،$[h]$ ،$[i]$ ،$[l]$. The mean (m), additive effect $[d]$, and additive × additive effects were significant by the *t*-test at the 1% level, While the dominance effects were significant at the 5% level, much smaller than additive effects(Table 2). Therefore it could be concluded that additive, additive × additive, and dominance effects have a major role in controlling these traits. The additive effect was significant at 1% level; however, the value is negative and the negative value $[d]$ depends on the fact that which parent is p_1 and which parent is p_2. The additive × additive effect is positive. Opposite signs of $[i]$and$[d]$ indicates that there is opposional nature in two genes. Since the additive - dominance model was not suitable for the infection type of flag leaf, and $[i]$ interaction was significant at 1% level, it can be concluded that the epistatic effects are important in the mode of inheritance of these traits. So by observing the epistasis , it is reasonable to assume that more than one gene controls the trait. The $[h]$ negative sign indicates that the relative dominance is to reduce the size of the trait i.e. to the resistant parent with lower infection type. The mean (m), additive effect $[d]$, dominance effect $[h]$, additive × additive effects $[i]$, and dominance × dominance effects $[l]$ were significant for the infection type of whole plant in cross Hebe × Arigashar indicating that both the additive and non-additive components are involved in controlling the inheritance of this trait. The dominance effects and dominance × dominance interactions for the infection type of whole plant is greater than $[d]$ additive effects. Thus, the dominant effects have a vital role in the inheritance of this trait in the studied generations. And selection cannot be fixed under conditions of selfing. Other researchers have also reported the mode of genetic resistance to powdery mildew in barley varieties as dominance type (Kasha, 1996; Pickering, 1998). The variance of F_2 plants, mean of F_3 progeny, covariance of F_2 and F_3 plants, average variance of F_3 progeny, variance

of non-segregating generations (E_2), and average variance of segregating generations for the infection type is shown in table 3. In most cases, additive variance was less than dominance variance which indicates that the selection method is not a stable method and hybridization would be more effective than the selection method for producing higher resistance i.e.lower infection type. For selecting resistant plants, selection should be done in the first generation and this is consistent with results obtained by other researchers (Naghavi, 2001; Fazeli, 2008).

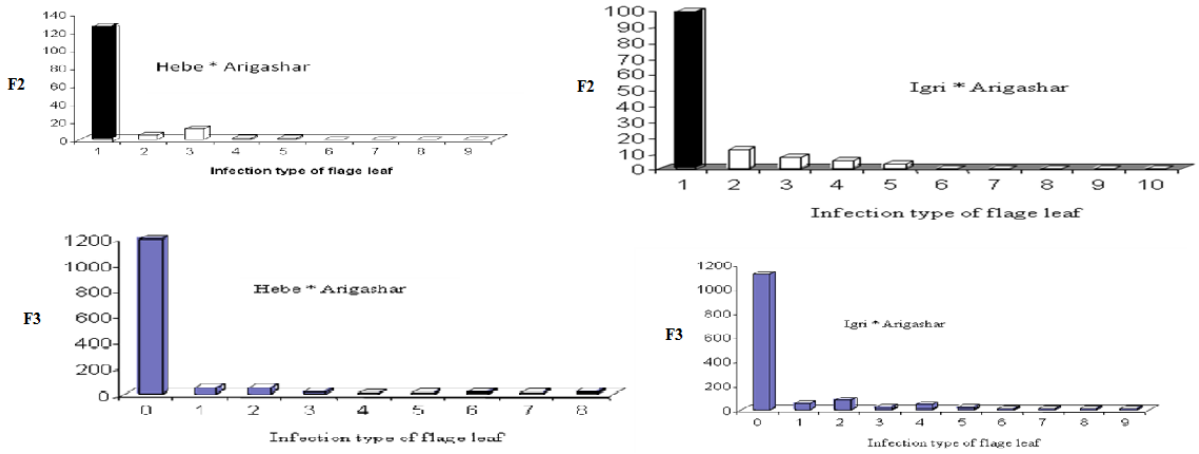

Fig. 1. The F_2 and F_3 frequency infection types of flag leaf in two crosses

IT_1 = Infection Type of Flag leaf

IT_2 = Infection Type of Total plant

Table 1. Mean and standard deviation of infection type of flag leaf and total plant traits in different generations of two crosses.

Generation	Hebe × Arigashar		Igri × Arigashar	
	IT_1	IT_2	IT_1	IT_2
	IT_1	IT_2	IT_1	IT_2
P_1	0.33±0.57	1.33±0.57	0.33±.57	1±1
P_2	4±1	7.66±0.57	2.8±1.48	7.33±1.15
F_1	0.66±0.57	2.67±0.57	0.66±0.58	2.66±1.52
F_2	0.39±0.88	2.06±1.36	0.27±0.75	3.63±2.26
F_3	0.42±0.82	3.79±1.73	0.33±0.63	3.34±1.94

IT_1 = Infection type of flag leaf

IT_2 =Infection type of total plant

Gene number and heritability

Different formulas were used to estimate the heritability of infection type of flag leaf in two crosses. The mean of heritability for two crosses were estimated 0.68 and 0.88 respectively (Table 5) indicating that the relatively high heritability value for cross Igri × Arigashar is due to the poor effect of environment on the examined trait. Low levels of environmental variance compared with the additive and dominance variances confirms this point.

Table 2. Estimate of genetic components of means for different traits in two crosses.

	Hebe × Arigashar		Igri × Arigashar	
Component	IT_1	IT_2	IT_1	IT_2
m	**0.417±0.19	**6.8±0.43	**0.31±0.29	**3.307±0.29
$[d]$	**-1.83±0.33	**-3.16±0.47	**-2.5±1.105	**-3.17±0.44
$[h]$	*-0.06±0.31	**14.8±1.71	*0.027±0.266	*0.38±0.77
$[i]$	**1.75±0.53	*-2.3±0.64	**2.51±1.11	*0.86±0.53
$[j]$	-	-	-	-
$[l]$	-	**10.7±1.48	-	-
χ^2	0.00658^{ns}	0.0007^{ns}	1.70978^{ns}	2.1010^{ns}

* = Significant at 5% Level
Ns= Not Significant
m= mean generation, $[d]$= Additive effect, $[h]$= Dominance effect, $[i]$= Additive × Additive effect, $[j]$= Additive × Dominance effect, $[l]$= Dominance × Dominance effect.

Table 3. Measured parameters in F_2 and F_3 generations

Statistic	Hebe×Arigashar		Igri × Arigashar	
	IT_1	IT_2	IT_1	IT_2
Vf_2	0.7835	1.8519	0.5692	5.1107
$V\overline{f_3}$	0.6658	3.0026	0.4015	3.8003
$Wf_{2/}f_3$	0.7428	3.0788	0.3675	3.656
$\overline{V}f_3$	0.0035	0.016	0.00219	0.02
E_1	0.5554	7.666	4.998	1.5553
E_2	4.592	4.148	7.814	10.7778

Vf_2 = F_2 Variance, $V\overline{f_3}$ = F_3 mean variance, $Wf_{2/}f_3$ = F_2 and F_3 Covariance,
$\overline{V}f_3$ = F_3 variance mean, E_1 = Non- segregant generation variance, E_2 = segregant generation variance

Table 4. The component of variance and estimated of different traits for infection type of flage leaf and total plant in two crosses.

	Hebe×Arigashar		Igri × Arigashar	
Component	IT_1	IT_2	IT_1	IT_2
D	-38.106	9.34	-6.81525	-26.271
H	125.409	-34.6359	4.48569	89.6559
E_1	-5.702	5.1724	2.999	-2.4109
E_2	8.237	2.3227	5.671	11.0551

Table 5. Estimated of general heritability by different formula for flag leaf infection type in two crosses

	treat	HF_1	HF_2	HF_3	HF_4	HF_5	HF_6	$\overline{HF}$
Hebe× rigashar	IT_1	0.55	0.79	0.82	0.64	0.60	0.68	0.68
Igri × Arigashar	IT_1	0.77	0.74	0.54	0.69	0.65	0.79	0.88

Table 6. Estimated number of gene and degree of dominance for different traits in two crosses

	Hebe×Arigashar		Igri × Arigashar	
Component	IT_1	IT_2	IT_1	IT_2
GNF_1	3.73	3.3	2.04	1.8
GNF_2	5.92	3.95	2.1	1.489
$\sqrt{\frac{H}{D}}$	-1.81	1.925	1.81-	1.847

Table 7. Reaction of infection type flag leaf in F_2 generation

Phenotype	scale	)o (	)E (	χ^2
Resistance	0-2	118	118.125	0.0156
Susceptable	3-8	8	7.875	0.0156
126=n				χ^2 =0.03

Table 8. Reaction of infection type flag leaf in F_2 generation

Phenotype	scale	)O (	)E (	χ^2
Resistance	0-2	143	137.8125	0.1952
Susceptable	3-8	4	9.1875	0.9289
147=n				χ^2 =3.124

The number of genes in two crosses was estimated by Cockerham methods (Cockerham, 1988) (Table 6). To calculate the number of genes, each formula is based on assumptions and its is not possible to expect that all of these assumptions be correct in estimating segregating genes. However, the estimated number of effective genes for resistance to powdery mildew in Igri × Arigashar cross for infection type of flag leaf and the whole plant between one or two genes is in consistence with the results obtained by other researchers (Kasha et al, 1996; Gawande, 2003; Antonin, 2005; Naghavi, 2001; Fazeli, 2008). But for both infection types of flag leaf and whole plant in Hebe × Arigashar cross, more genes were estimated (Table 6). Degree of dominance for infection type of flag leaf and whole plant was estimated greater than 1. And being greater than 1 indicates the importance of dominance component in controlling this trait (Table 4). In order to determine epistatic type in F_2 plants for infection type of flag leaf (IT), all plants were divided into two groups of susceptible and resistant plants. According to Saari and Prescott (1975), plants with zero to two IT were considered resistant and other plants were considered sensitive. The results showed that in cross Hebe × Arigashar , using the χ^2 test for segregating generation, it was determined that the dominant epistatic double ratio is 15:1, and the presence of two recessive genes causes sensitivity; otherwise resistance occurs. In cross Igri ×

Arigashar , using the χ^2 test in F_2 generation, it was determined that the distribution of F_2 generation with threefold dominant epistatic ratio is 35:1 and the presence of two or three recessive genes causes susceptibility(Table 7). There were more plants which had resistant infection types (0-2) in F_2 generation in cross Igri × Arigashar, and this is due to the presence of at least one resistant gene (Fig. 1).

Therefore, using progeny of Hebe × Arigashar cross in which had lower infection type and used Cockerham methods in which resistance is controlled by 3 to 6 genes and it also, it is concluded that by hybridization methods, cultivars with greater resistance would be achieved in breeding programs.

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