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Gene Number and Heredity of Barley Powdery Mildew (*Erysiphe graminis* f. sp. *hordei*) Resistance at Adult Plant Stage

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ABSTRACT

Gene number and gene action were estimated for powdery mildew disease reaction in barley. Five generations (P1, P2, F1, F2 & F3) of two crosses, Radical × Afzal and Cwb × Afzal, were grown for recording infection type (IT) and area under disease progress curve (AUDPC). Both additive and dominance gene actions were found to be important in inheritance of powdery mildew resistance including digenic non-allelic interactions. Trigenic interactions and genotype × environment effects, however, were not significantly involved. Resistance to powdery mildew appeared to be controlled by 3 and 2 dominant genes in Radical × Afzal and Cwb × Afzal, respectively based on IT data, while being conditioned by 4 - 12 and 9 - 10 genes based on AUDPC. Broad sense heritabilities were very high in both crosses (71 - 99.8%).

Key Word: Barley; Powdery mildew; Gene number; Gene action; Heritability

INTRODUCTION

Powdery mildew caused by *Erysiphe graminis* f. sp. *hordei* is one of the most devastating diseases of barley (Masterbroek & Balkema-Boomstra, 1995). Development of resistant varieties is the most economical and environmentally safe method by reducing the application of fungicides to combat this disease.

Resistance in seedling is usually race-specific and can be recognized by low infection type (IT) over all growing stages. While in adult plant stage, resistance is race-specific or non-specific and would be evaluated at field by severity, infection type and area under disease progress curve (AUDPC). Studies have shown that AUDPC, which is employed for yield-loss measurement and for field assessment, is a quantitative trait (Chen & Line, 1995; Gawande & Patil, 2003). Chen and line (1995) also indicated that there is a high correlation between IT and disease intensity.

The number of genes controlling a character is of great importance for the study of mechanism of heredity and for plant breeding. The observed and expected Mendelian ratios are compared in order to know how many genes are involved in quantitative traits. It is a hard and almost impossible to determine exactly the number of genes controlling quantitative characters and we estimate only the minimum number of effective factors. An effective factor, according to Mather and Jinks (1977) is a segment of chromosome acting as an inheritable unit and separated from other units by an average recombination frequency of 50%.

A number of approaches have been suggested to estimate the number of effective factors including chromosome assay (Law, 1967), method of moments (Castle, 1921; Lande, 1981; Cockerham, 1986), genotype assay (Jinks & Towy, 1976), inbred back cross technique (Wehrahan & Allard, 1965) and molecular marker-based QTL mapping (Lander & Botstein, 1989; Chantret *et al*., 2000). Chromosome assay, in which the intra-varietal substitution lines are used, is limited to very few species like wheat. One of the earliest, simplest and most known procedures is the method of moments that utilizes information of the phenotypic means and variances of two inbred parental lines and their F1, F2 (B1 & B2 if available). Castle (1921) with his graduate student Wright, were pioneers in employing the square of genotypic range with the estimated genetic variance. This estimation assumes that the trait is controlled by independent non-linked genes with equal additive effects and that all genes with positive influence on the trait are sorted into one parent and those with negative influences into the other (Mather & Jinks, 1977). These assumptions never met completely. The number of genes, therefore, is underestimated. For this reason, geneticists have suggested various modifications in order to reduce the bias (Mather & Jinks, 1977; Lande, 1981; Cockerham, 1986; Zeng *et al*., 1990).

Genotype assay and inbred-back cross technique are less dependent on assumptions but complicated and time consuming on the other hand. QTL mapping, rapidly being developed, involves the search for associations between segregating molecular markers and quantitative character (Lander & Botstein, 1989; Chantret *et al*., 2000).

No breeding method can achieve the desirable goal without precise understanding of gene action involved for resistance. In addition, Heritability is very important for choosing the breeding approach and for estimating selection response. There have been no genetic studies on the number of genes controlling powdery mildew resistance in barley cultivars. The objectives of this research were to estimate the number of genes conditioning resistance to powdery mildew in barley and to study the mode of inheritance.

MATERIAL AND METHODS

Afzal is considered as susceptible line and Radical and Cwb are known as resistant lines on the basis of field

experiments over many years in Seed and Plant Improvement Institute, Karaj, Iran (Naghavi, 2001). Five generations (P1, P2, F1, F2 & F3) of two crosses, Radical × Afzal and Cwb × Afzal, were raised and planted in a randomized complete block design with three replications, each plot consisting of 2 m long rows with 30 cm apart. In each replication parents and F1s seeded in one row but F2s and F3s in 10 and 30 rows, respectively. The experimental field was surrounded by 5 - 6 rows of susceptible line and the same line was interspaced as border rows for every 20 rows in order to have a uniform disease spread. The artificial inoculation was carried out using culture derived from powdery mildew spores collected from Gorgan (Northern Province of Iran) at seedling and booting stage as well. Average disease severity (percentage of leaf area infected) was recorded according to modified Cobb scale (in Peterson *et al*., 1948) three times (i.e., three succeeding weeks). Infection type was recorded using Masterbroek and Balkema (1995) scale. Having been transferred through ln {[x/(100-x)] + 10}, the severity data were used to calculate area under disease progress curve (AUDPC) based on Shanner and Finney (1977) method. Six parameters, viz., *m* (average effect), *d* (additive), *h* (dominance), *i* (additive x additive), *j* (additive x dominance) and *l* (dominance x dominance) were estimated as per Mather and Jinks (1977) model after testing adequacy of additive-dominance (three parameter) model through joint scaling test.

The F3 lines data were grouped into three classes based on their IT: (1) homozygous for resistance, (2) segregating for resistance and (3) homozygous for susceptibility. Chi-square analysis was carried out to test goodness of fit as a check on the hypothetical ratios and to estimate the number of genes influencing IT (Lee & Shanner, 1985). Means and variances of generations and families were used to estimate the number of genes affecting AUDPC. Five Formula (Castle, 1921; Mather & Jinks, 1977; Cockerham, 1986) used for estimating gene number are listed in (Table V). Since each formula has its own restrictions or assumptions, some formula produced numbers, which were not applicable for certain crosses and therefore not given in the table.

Phenotypic variances of parents, F1, F2, F3 based on AUDPC data were used to estimate broad-sense heritability of resistance using two formulas (Table VI).

RESULTS AND DISCUSSION

Analysis of variance for AUDPC in two crosses showed significant difference among generations (Table I). We were therefore, allowed to go ahead to study heredity and to analyze generation means.

As expected, the susceptible parent, Afzal, had higher IT and AUDPC (Table II). Transgressive segregation has frequently been reported for powdery mildew resistance (Falak *et al*., 1999). In this study transgressive segregation was observed for enhanced resistance (i.e., less IT & AUDPC), indicating the contribution of both parents genes to resistance.

Table I. Mean Square of Anova for AUDPC in two crosses

S.O.V	Df	Mean square	
		Radical × Afzal	Cwb × Afzal
Replication	2	0.9	1.2
Generations	4	466.2**	378**
Error	8	1.2	1.67

Table II. Mean and standard deviation of AUDPC and IT in different generations for two crosses

Generation	Radical × Afzal		Cwb × Afzal	
	IT	AUDPC	IT	AUDPC
P1	0.63±0.49	27.63±0.01	0.8± 0.08	27.63±0.09
P2	8.80±0.10	38.26±0.23	8.9±0.12	38.16±0.61
F1	0.66±0.20	27.64±0.04	1.66±0.10	27.67±0.10
F2	3.06±2.78	28.00±1.12	4.76±2.84	28.47±1.24
F3	0.82±1.70	27.67±0.12	2.72±2.88	28.05±1.36

Table III. Estimate of genetic components of means for AUDPC in two crosses

Component	Radical × Afzal	Cwb × Afzal
m	26.98±0.21**	27.08±0.32**
[d]	-5.32±0.03**	-5.26±0.07**
[h]	3.44±1.05**	4.96±1.49**
[i]	5.96±0.21**	5.81±0.33**
[j]	-	-
[l]	-19.16±2.69**	18.34±2.13**
χ^2	0.001^{ns}	0.0003^{ns}

** Significant at 1%
ns Not Significant

Table IV. Distribution and χ^2 tests for F3 in two crosses

Cross	Number of F3 lines			Expected ratio	χ^2
	HR[1]	Seg[2]	HS[3]		
Radical × Afzal	83	5	2	59:4:1	0.081
Cwb × Afzal	56	28	6	10:5:1	0.026

1 Homozygous resistant
2 Segregating for resistance
3 Homozygous susceptible

The results of generation means analysis (Table III) revealed that five parameter model is adequate, the χ^2 being non-significant. It is concluded that trigenic epistasis and genotype × environment interactions are not making a significant contribution to the differences among the generation means. We can, therefore, interpret the resistance to powdery mildew in terms of the additive, dominance and digenic non-allelic interactions including only additive × additive and dominance × dominance effects. The *h* increments of the majority of individual loci must be positive, while the *l* increments of the majority of pairs of loci must be negative. So the non-allelic interaction is mainly of duplicate kind. It means two heterozygous loci together have less effect than the summed effects of two loci separately (Mather & Jinks, 1977).

IT data of F_3 were used to estimate the number of genes controlling the resistance (Table IV), because the groups of resistant, susceptible and segregating lines are better identified in F3 families than on individual plants in the F2 generation, where single plant heterozygotes are

Table V. Estimated number of genes for resistance in two crosses based on AUDPC produced by powdery mildew

Cross	N1	N2	N3
Radical × Afzal	11.38	11.37	3.26
Cwb × Afzal	9.692	9.691	8.9

N1 (Castle and Wright) = $(P_1-P_2)^2/8(V_{F2}-V_E)$
N2 (Cookerham)= $(P_1-P_2)^2 - V_{p1}-V_{p2}/8(V_{F2}-V_E)$
N3 (Mather and Jinks)= $[F_1-(P_1-P_2)/2]^2/H$
Where:
N = Number of genes
$V_E = 1/2V_{P1}+1/4V_{p2}+1/2V_{F1}$

Table VI. Estimated broad-sense heritability (Percent) of resistance in 2 crosses to powdery mildew based on AUDPC using two formula

Cross	H1	H2
Radical × Afzal	98.6	71
Cwb × Afzal	93.5	76

$H1 = V_{F2}-V_E/V_{F2}$
$H2 = V_{2F3}-V_E/V_{2F3}$
Where:
$V_E = 1/2V_{P1}+1/4V_{p2}+1/2V_{F1}$
V_{2F3} = mean variance of F3 families

difficult to distinguish from homozygotes (Fuentes-Davila *et al*., 1995). Radical × Afzal showed a good fit to a ratio of 5 resistant: 4 segregating: 1 susceptible and Cwb × Afzal to that of 10:15:1. Regarding homozygote and segregating resistant lines as a resistant group, 63R:1S and 15R:1S are obtained in Radical × Afzal and Cwb × Afzal, respectively implying that resistance is being controlled by 3 and 2 dominant genes in these two crosses. Chen and Line (1995) based on F2, F3 and F5 ratios estimated 2 - 4, 3 - 4 and 3 - 6 genes, respectively for IT of stripe rust (*Puccinia striformis* f.sp. *tritici*) in wheat.

Based on AUDPC data, the number of genes (effective factors) conditioning resistance was estimated through different formula (Table V). In Radical × Afzal and CWB × Afzal, 4 - 12 and 9 - 10 genes were found, respectively. Qi *et al*. (1998) based on AUDPC data and AFLP markers identified 6 QTLs for partial resistance to leaf rust (*Puccinia hordei*). 2 - 3 genes (Chen & Line, 1995) and 3 QTLs (Milus & Line, 1986) have been reported for high-temperature adult plant (HTAP) resistance to strip rust. Johnson (1978), however, suggested that HTAP is being controlled by many genes.

Broad sense heritabilities (Table VI) were very high in both crosses (71 - 99.8%). As the generation means analysis showed that dominance and non-allelic interactions were highly contributed to the resistance, high heritability did not indicate that resistance to powdery mildew is being heritable.

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