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Genetic analysis of Fusarium head blight resistance in bread wheat

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Abstract Seven spring wheat varieties were crossed in a half diallel mating system to assess the genetic parameters of some traits of resistance to Fusarium head blight (FHB) including disease incidence (DIC), disease severity (DSV), Fusarium damaged kernels (FDK), disease index (DI) and incidence severity kernels (ISK). Differences were found to be significant ($p<0.01$) for all the characters. The significance of additive components (D) and dominant components (H_1, H_2) demonstrated the importance of both additive and dominance effects for all traits. The greater value of D over H_1 and H_2 demonstrated the additive nature of genes for all traits, which suggested the utilization of pedigree and full/sib selection for improvement of these parameters. All traits exhibited high narrow and broad sense heritability. Graphical representation demonstrated in DIC recessive alleles and in DSV, FDK, DI and ISK dominant alleles led to decreasing level of traits and increasing resistance to FHB.

Keywords Fusarium head blight · Wheat · Genetic analysis · Diallel cross

Introduction

Fusarium head blight (FHB) or scab of wheat has caused serious epidemics in many wheat growing areas worldwide (Bai and Shaner 1994; McMullen et al. 1997). Although several Fusarium species can cause FHB, *Fusarium graminearum* Schwabe (telomorph *Gibberella zeae* (Schw.) Petch) is the most important pathogen worldwide (Bateman 2005). The main causative agents of FHB in Iran are *F. graminearum* and *F. culmorum* (Zamanizadeh and Khorsandi 1995). FHB is an important disease of wheat in areas of Iran such as Mazandaran, Gorgan, Gonbad and Moghan regions (Moosawi-Jorf et al. 2007). When warm and wet weather coincides with wheat anthesis and early grain-filling, severe infection can dramatically reduce grain yield and quality (Bai et al. 2001).

Grains infected by *Fusarium graminearum* are often shriveled, with significantly lower kernel weight, and can be easily blown away with the chaff during threshing (Bai and Shaner 2004). Additional losses come from contamination of grains with mycotoxins produced by *F. graminearum* (Bernardo et al. 2007). Deoxynivalenol is a major toxin produced by the fungus during infection and is harmful to animal and human health (Steiner et al. 2008). To protect consumers from mycotoxicosis many countries, including the European Union Member States have established maximum allowed levels for the most prevalent Fusarium mycotoxins in cereal and cereal products (Van Egmond 2004; Anonymous 2005). For example, the EU regulation allows a maximum DON content in unprocessed bread wheat of 1.25 ppm, in bread and bakeries of 0.5 ppm and in baby food of 0.2 ppm (Anonymous 2005).

Several methods such as crop rotation and chemical and biological agents have been used to control FHB. Utilization of wheat cultivars with improved Fusarium resistance in combination with appropriate crop management practices is economic and effective ways to control

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FHB (Mardi et al. 2004). The resistance of wheat to FHB is a complex phenomenon. The forms, types or components of physiological resistance (Mesterhazy 1995, 2001) are: (i) resistance to initial infection (Schroeder and Christensen 1963); (ii) resistance to spreading (Schroeder and Christensen 1963); (iii) resistance to kernel infection (Mesterhazy 1995; Mesterhazy et al. 1999); (iv) tolerance to infection (Mesterhazy 1995; Mesterhazy et al. 1999) and (v) resistance to DON accumulation (Miller et al. 1985).

Breeding for resistance to FHB has received increasing attention in China since the 1980s (Wu et al. 1984), and in Europe and North America since the 1990s (Miedaner 1997; Rudd et al. 2001). So far significant progress in wheat research has been achieved and some resistant varieties have been released (Bai and Shaner 2004; McKendry et al. 2004; Mergoum et al. 2005).

Previous studies suggested that FHB resistance in wheat is inherited predominantly as a quantitative trait in an additive-dominance model (Bai et al. 2000; Snijders 1990b; Jiang and Ward 2006). Multiple loci or genes were involved in the resistance, and each had low expressivity or low contribution to heritability and was sensitive to genetic background (Gervais et al. 2003; Shen et al. 2003; Somers et al. 2003; Klahr et al. 2004; Mardi et al. 2005).

Heritability estimates for FHB resistance are sparse and contradictory, depending on the genetic materials and methods used. Snijders (1990b) reported broad sense heritability of FHB resistance in F2 single-plant populations from 0.05 to 0.89. Heritability estimates by Saur and Trottet (1992) and Singh et al. (1995) were in the range of 0.66 to 0.93 but were derived from single environments. Malla et al. (2009) reported narrow sense heritability of FHB resistance from 0.4 to 0.64.

Bai et al. (1989) using a 3×3 half diallel cross of three resistant and three susceptible genotypes, concluded that variation among the six parents was conferred by three gene-loci and several minor modifying genes. In addition, it was determined that inheritance of FHB resistance is a partially or fully dominant trait. Lin et al. (1992), also using half diallel crosses, found that the inheritance of scab resistance is governed by dominant genes, which act in an additive manner.

Therefore, the objective of the present research was finding the action of resistance genes and evaluating heredity of resistance and other genetic components in several wheat genotypes exhibiting various levels of FHB resistance.

Material and method

Seven spring wheat genotypes with different levels of FHB resistance were used (Table 1). F1 crosses were obtained by hand emasculation and pollination in the field at the Agricultural Research Center of Gorgan, Golestan in 2008. Twenty-eight genotypes including parents and F1 were included in the test. The wheat lines and crosses were evaluated in the experimental field at Gorgan Agricultural Research Center in 2009 using a randomized complete-block design with three replications, each plot consisting of two rows (1 m length) with 15 plants sown by hand.

Inoculation and disease assessment

To prepare inoculum, a fungal isolate was collected from a field trap nursery and cultured on potato dextrose agar medium. About 5 g of powdered straw was added to 125 ml of distilled water in 250 mL flasks. Mixtures were autoclaved at 121°C and 1 atmosphere for 30 min twice in 48 h. Then, each flask was inoculated with an agar plug from a clean *F. graminearum* isolate under laminar flow hood. The flasks were swirled gently at 120 rpm at 25°C for 96 h. The number of conidiospores per mL was determined using a hemacytometer and adjusted to the desired spore concentration (10^5 conidia/mL) with distilled water. At the beginning of anthesis each plot was inoculated with the conidial suspension by spraying onto each plot using a manual atomizer and Inoculum was applied until run off. Inoculation carried out every other day for 5 times after 4 p.m. Inoculated plots were misted using a mist irrigation system for 30 min after each inoculation to favor development of the disease.

Table 1 Genotype, origin, pedigree and reaction to Fusarium head blight (FHB) of wheat genotypes used as parent for diallel crossing scheme

Genotype	Origin	Pedigree	FHB-reaction	Type of resistance
Frontana	Brazil	Fronteira/Mentana	Moderately resistant	I
Sumai3	China	Funo/Taiwanxiaomai	Moderately resistant	II
Wangshuibai	China	Chinese landrace	Moderately resistant	II
Morvarid	Mexico	Milan/Shanghai	Moderately resistant	I,II
Tajan	Iran	Bow "S"/Nkt"S"	Moderately susceptible	II
Falat	Iran	Kvz/Buho"s"//Kal/Bb = seri82	susceptible	null
Golestan	Iran	Unknown	susceptible	null

Disease assessment

Most studies indicate that visual assessment of FHB disease symptoms gives a good indication of FHB-associated yield loss (Arseniuk et al. 1993; Doohan et al. 1999; Mentewab et al. 2000). Other researchers have found strong relationships between visual FHB score and the fungal DNA content of grain (Doohan et al. 1999) or mycotoxin content of grain (Mesterhazy 2002). In this study, our observation was on the basis of visual assessment. Disease incidence (DIC) (type I resistance) and disease severity (DSV) (type II resistance) were recorded 21 days after the first conidial suspension application in the field and Fusarium damaged kernels (FDK) (type III resistance) recorded after harvesting spikes when mature. Disease rating for each entry was averaged across 30 heads. Disease incidence was measured as the percentage of number of spikes infected across total spikes. Disease severity was measured as the percentage of infected spikelet (s) within the spike. The field disease severity was recorded based on 0–5 scale (0 = no disease, 1 = to 20%, 2 = to 40%, 3 = to 60%, 4 = to 80% and 5 = more than 80% disease severity) (Wan et al. 1997). Fusarium damaged kernels (FDK) was measured as percentage of infected kernels within the spike. The Disease index (Browne 2007) and Incidence- severity- kernels (ISK) index (Gilbert and Woods 2006) were calculated according to the following formulas:

$$DI = [(incidence \times severity)/100]$$

$$ISK = (0.3{*}incidence) + (0.4{*}severity) + (0.4{*}FDK)$$

Statistical and genetic analyses

Homogeneity of variance was tested by Bartlett's test by D2 (Dick 1988) statistical package, which showed all traits were homogeneous (Bartlett 1937). Analysis of variance for each genotype was calculated using the general linear model (GLM) procedure of the SAS/STAT software (SAS Institue Inc. 2002 & 2003). Diallel analysis based on Jinks and Hayman (1953) method was used to estimate genetic components. Diallel analysis was done using the D2 statistical package (Dick 1988).

Results and discussion

The populations originating from seven parent diallel crosses were evaluated in the field in 2009. Uniform infection with FHB depends on a number of factors, apart from resistance, such as time, type and amount of infection and in environmental variation (Parry et al. 1995). Since FHB resistance is non-specific and horizontal (Van Eeuwijk et al. 1995), the inoculation was carried out using a highly aggressive *Fusarium* isolate at anthesis, which is the most susceptible developmental stage for Fusarium ear infection (Pough et al. 1933). In order to account for ear to ear variation in flowering time within each plot, repeated inoculations were applied. Optimal humidity was provided using a mist-irrigation system.

Mean squares from the analysis of variance for the characters under study are presented in Table 2. Presence of significant genotypic differences for all the characters allowed proceeding with further analysis (Mather and Jinks 1982).

The diallel technique developed by Jinks and Hayman (1953) and modified by Mather and Jinks (1982) was used in this experiment. Assumptions of the additive-dominance model such as multiple allelism and independent action and distribution of non-allelic genes were tested by subjecting the data against two adequacy tests. The first adequacy test was joint regression analysis, in which the regression coefficient (b) must deviate significantly from zero but not from unity, if all the assumptions underlying the genetic model were met. The second adequacy test was analysis of variance of (Wr+Vr) and (Wr-Vr) values. In this test the mean squares for (Wr+Vr) should be significantly different between the arrays while the mean squares for (Wr-Vr) should be non-significant (Mather and Jinks 1982; Singh and Chaudhary 1985).

For all traits, the regression coefficient test indicated that b differed significantly from zero but not from unity (Table 5) and according to second test, Wr-Vr was non-significant, indicated existing of the additive-dominance model for these characters (Tables 3 and 4). Previous studies suggested that FHB resistance in wheat is inherited predominantly as a quantitative trait in an additive-

Table 2 Analysis of variance (mean squares values) for different traits in wheat

S.O.V	Df	DIC	DSV	FDK	DI	ISK
Genotypes	27	1815.94**	821.72**	4.97**	302.61**	627.48**
Replications	2	473.36ns	208.22*	0.64ns	5.39ns	144.43**
Error	54	111.93	56.1	0.28	31.09	28.29

* Significant at the 0.05 level of probability, ** significant at the 0.01 level of probability and ns = non-significant

Table 3 Analysis of variance for Wr-Vr in studied characters

S.O.V	Df	DIC	DSV	FDK	DI	ISK
Block	2	32713.21^{ns}	2593.45^{ns}	0.66^{*}	0.46^{*}	240.34^{ns}
Parent	6	9606.61^{ns}	1536.66^{ns}	0.19^{ns}	0.15^{ns}	359.96^{ns}
Error	12	9910.61	1204.49	0.07	0.07	221.72

* = significant at the 0.05 level of probability, n.s = not significant at the 0.05 level of probability

dominance model (Bai et al. 2000; Snijders 1990b; Jiang and Ward 2006).

Among the genetic components of variation (D, F, H1, H2, h^2), the statistic D was an estimate of additive effects; H1 and H2 estimated variation due to dominance effects of genes; and F provided an estimate of the relative frequency of dominant to recessive alleles in the parental lines and the variation in dominance over loci. The statistic h^2 provided direction of dominance i.e. a positive sign shows increasing dominance of a gene at most loci and a negative sign shows decreasing dominance of a gene. These components were used to compute further information as $(H1/D)^{0.5}$, mean degree of dominance; H2/4H1, proportion of genes with positive and negative effects in the parents and $[(4DH1)^{0.5} + F]/[(4DH1)^{0.5} F]$ provides the proportion of dominant and recessive genes in the parents.

The estimates of genetic components of variation (Table 5) revealed significant values of both D and H components suggesting that all traits were under the control of both additive and dominance gene effects. In all traits, D value was greater than H value thus indicating that additive gene effects were more important than non-additive gene effects. According to the comparisons, for all 3 types of studied resistance (I, II, III) additive gene effect was more important than non additive effects. Also, Bai and Shaner (1994) and Devkota (2002) indicated that inheritance of resistance to FHB was under the control of additive and non-additive genes. Several studies indicated that additive gene effects were more important than non-additive gene effects (Snijders 1990a, b; Buerstmayr et al. 1999; Bai et al. 2000; Oettler et al. 2004; Mardi et al. 2004). Unequal values of H1 and H2 for all of them indicated the presence of positive and negative alleles in unequal frequencies (Table 5). This was supported by $H_2/4H_1$ ratio which indicated the presence of positive and negative alleles in unequal frequencies (Table 5). It was suggested that where the genes are equally distributed among parents, this value is equal to 0.25 (Singh and Chaudhry 1985). The F value was negative for all traits and revealed the excess of recessive alleles present in genetic material and this was finally sustained by the value of $[(4DH_1)^{0.5} + F]/[(4DH_1)^{0.5}- F]$ which was less than unity. The lack of significance for component h^2 for DIC and FDK traits illustrated that dominance was not unidirectional but DSV, DI and ISK traits displayed significant h^2 values resulting in unidirectional type of dominance and suggesting that heterosis breeding could be rewarding for these traits. The average degree of dominance $(H1/D)^{0.5}$ for all traits was less than 1 indicating partial dominance with additive gene effect. The positive intercept of Wr/Vr regression line (Figs. 1a, 2a, 3a, 4a and 5a) for all traits also indicated additive gene action with partial dominance.

Broad sense heritability (H^2) measures the fraction of phenotypic variance attributable to genetic differences among individuals in a population. Narrow sense heritability measures the extent of correspondence between breeding values and phenotypic values and expresses the magnitude of genotypic variance in the population, which is mainly responsible for changing the genetic composition of the population via selection (Falconer 1989; Dabholkar

Table 4 Analysis of variance for Wr + Vr in studied characters

S.O.V	Df	DIC	DSV	FDK	DI	ISK
Block	2	133177^{ns}	49874^{ns}	2.2^{ns}	3.58^{ns}	17649^{ns}
Parent	6	142128^{ns}	173894.3^{*}	6.95^{*}	6.83^{ns}	22565.83^{*}
error	12	83303.34	17419.29	0.72	2.45	6702.13

* = significant at the 0.05 level of probability, n.s = not significant at the 0.05 level of probability

Table 5 Estimates of genetic components for variation of five studied characters

Genetic component	DIC	DSV	FDK	DI	ISK
B-1	1.55^{ns}	0.96^{ns}	1.64^{ns}	0.73^{ns}	0.95^{ns}
D±S.E$_{(D)}$	$836.64\pm52.28^{*}$	$464.83\pm17.94^{*}$	$2.47\pm0.21^{*}$	$5.85\pm0.14^{*}$	$328.32\pm8.81^{*}$
H_1±S.E$_{(H1)}$	$302.53\pm125.87^{*}$	$177.04\pm43.19^{*}$	$1.04\pm0.51^{*}$	$1.06\pm0.33^{*}$	$54.94\pm21.22^{*}$
H_2±S.E$_{(H2)}$	$259.39\pm110.91^{*}$	$171.11\pm38.06^{*}$	$1.07\pm0.45^{*}$	$1.14\pm0.29^{*}$	$59.14\pm18.69^{*}$
F±S.E$_{(F)}$	-321.52 ± 125.43^{ns}	-13.87 ± 43.04^{ns}	-0.6 ± 0.51^{ns}	-1.4 ± 0.33^{ns}	-91.24 ± 21.15^{ns}
h^2	-20.72 ± 74.49^{ns}	$100.65\pm25.56^{*}$	0.31 ± 0.3^{ns}	$0.43\pm0.19^{*}$	$29.42\pm12.56^{*}$
E	$111.92\pm18.49^{*}$	$56.1\pm6.34^{*}$	$0.29\pm0.8^{*}$	$0.56\pm0.05^{*}$	$28.29\pm3.12^{*}$
$(H_1/D)^{0.5}$	0.6	0.62	0.65	0.42	0.41
$H_2/4H_1$	0.21	0.24	0.25	0.26	0.26
$\frac{(4DH_1)^{0.5}+F}{(4DH_1)^{0.5}-F}$	0.52	0.95	0.68	0.56	0.49
$h^2_{n.s}$	0.77	0.71	0.73	0.81	0.83
$h^2_{b.s}$	0.86	0.84	0.86	0.87	0.89

* = significant at the 0.05 level of probability, ** = significant at the 0.01 level of probability, n.s = not significant at the 0.05 level of probability, D = additive effect, H_1 and H_2 = dominance effect, F = determines frequencies of dominant to recessive alleles in parents, h^2 = determines the overall dominance effect due to heterozygous loci, $h_{N.S}{}^2$ = narrow sense heritability, $h_{B.S}{}^2$ = broad sense heritability

1992). Estimates of narrow and broad sense heritability $\left(h^2_{n.s}, h^2_{b.s}\right)$ showed high heritability for all traits. So, all three types of studied resistance (I, II and III) illustrated high heritability. High estimates of heritability in the narrow sense represented fixable and additively heritable variation, which indicated that selection response should be rapid for these characters. Heritability estimates for FHB have varied with methods of calculation. Buerstmayr et al. (2000) reported high broad-sense heritability (H>0.75) in two winter wheat populations. Snijders (1990b, c) estimated broad-sense and realized heritabilities ranging from 0.05 to 0.89 (mean 0.39) in F2 populations, and 0.00 to 0.96 (mean 0.23) in F populations. Singh et al. (1995) observed moderate to high narrow sense heritability (0.66 to 0.93). Malla et al. (2009) reported narrow sense heritability of FHB resistance from 0.4 to 0.64.

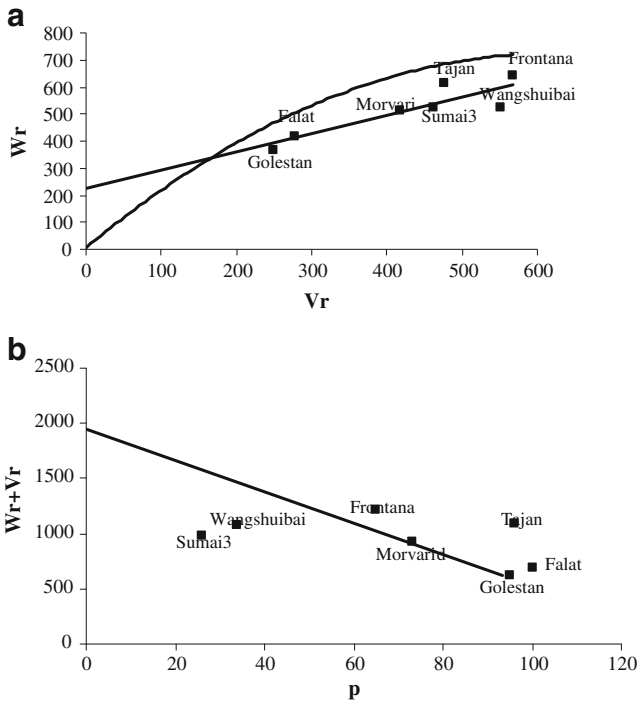

Fig. 1 Wr/Vr graph (**a**) and Wr+Vr/P graph (**b**) for DIC trait

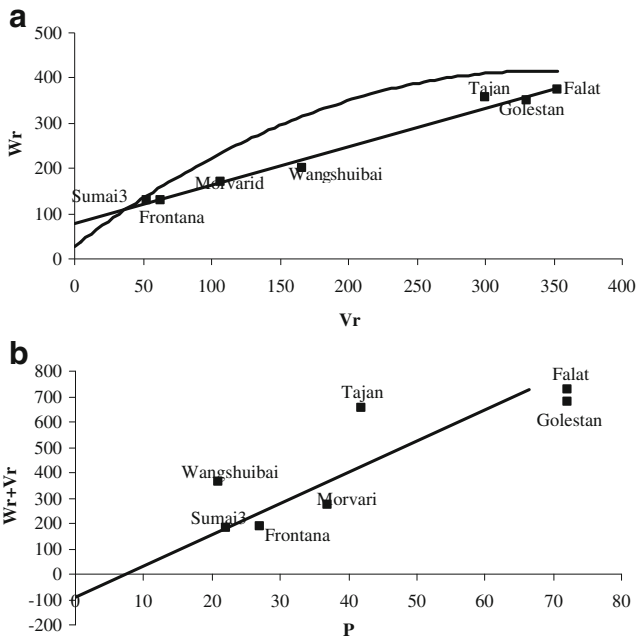

Fig. 2 Wr/Vr graph (**a**) and Wr+Vr/P graph (**b**) for DSV trait

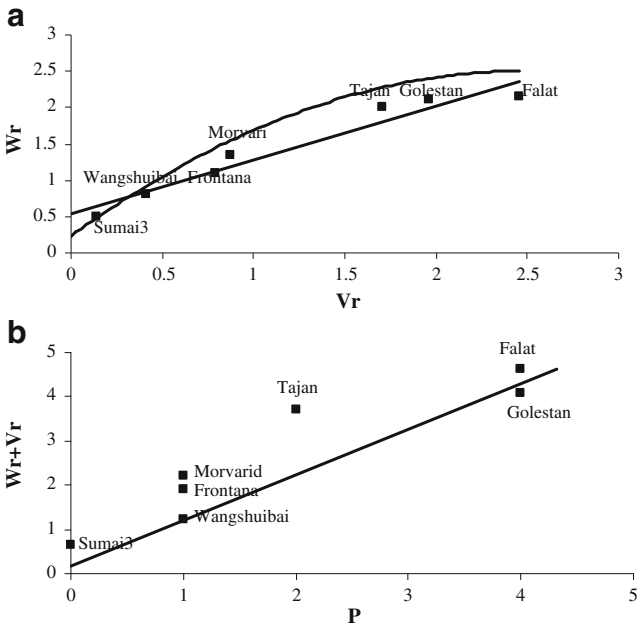

Fig. 3 Wr/Vr graph (**a**) and Wr+Vr/P graph (**b**) for FDK trait

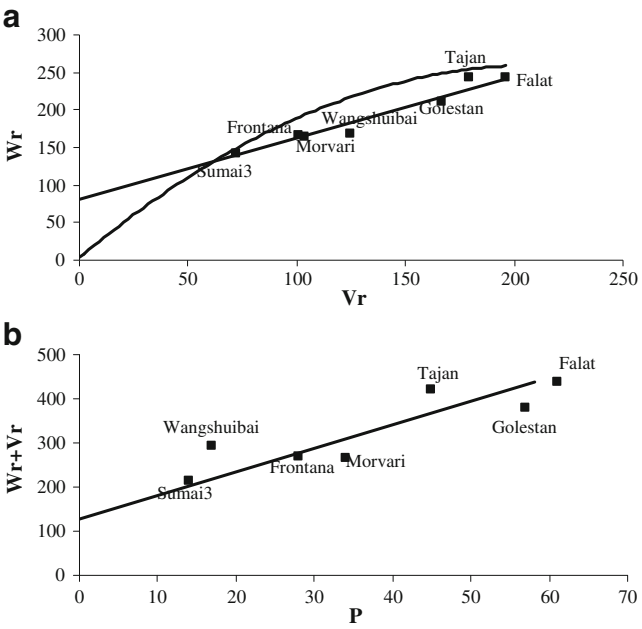

Fig. 5 Wr/Vr graph (**a**) and Wr+Vr/P graph (**b**) for ISK trait

Placement of array points displayed (Fig. 1a) that Golestan had the maximum dominant genes for DIC being nearest to the origin, whereas Frontana had the least dominant genes being farthest from the origin. For DSV, Sumai3 and Falat possessed the maximum dominant and recessive genes, respectively (Fig. 2a). For FDK, Sumai3 had the maximum dominant genes and Falat had the most recessive genes (Fig. 3a). Sumai3 genotype possessed maximum dominant genes for DI, whereas, Tajan had the most recessive genes for this trait (Fig. 4a). For ISK, Sumai3 had the maximum dominant genes and Falat had the most recessive genes for this trait (Fig. 5a). To find out the correlated response of dominant genes with phenotype of the common parent, Wr+Vr values of the arrays were plotted against the parental values (Figs. 1b, 2b, 3b, 4b and 5b). For all traits except DIC, the graph presented that parents with highest symptom levels had greater Wr+Vr values and parents with lowest symptom levels had smaller Wr+Vr values. Thus, it was clear that greater level of DSV, FDK, DI and ISK resulted from more recessive genes. Dominant genes decreased the symptoms and recessive genes increased them. For DIC a negative correlation clearly depicted that the parents with higher DIC levels had smaller values of Wr+Vr and thus, had lesser number of dominant alleles. So, recessive alleles have decreased DIC level and have increased resistance.

The results revealed that there was significant genotypic variation among genotypes which made the diallel analysis practical. The data of all the characters were fully adequate for genetic interpretation. For all traits, the additive and dominance gene effects are significant. The Wr/Vr graph also shows additive gene control for these traits. The high narrow-sense heritability in our study further highlighted the importance of additive gene action and indicated that progress in disease resistance could be made from selection using the current set of parental lines or crosses among them.

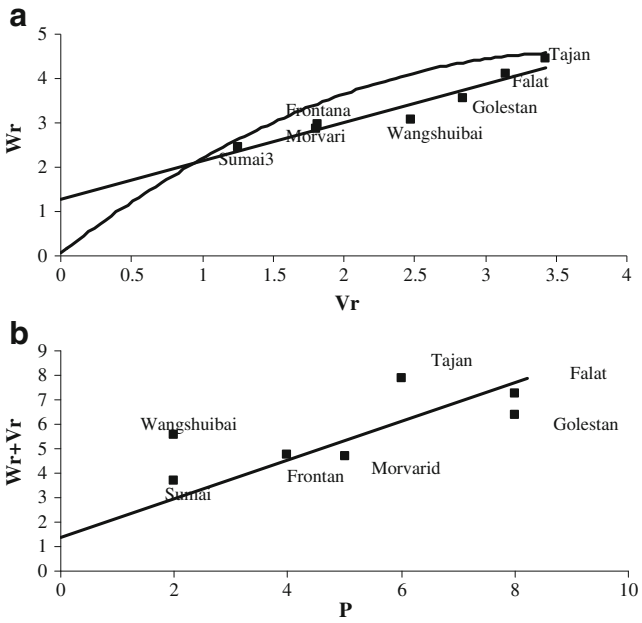

Fig. 4 Wr/Vr graph (**a**) and Wr+Vr/P graph (**b**) for DI trait

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