Genetic Control and Heritability of Fusarium Head Blight (FHB) Resistance in Spring Bread Wheat

Asadollah Ahmadikhah¹,* and Leila Nayyeripasand²

¹Department of Biotechnology, Faulty of New Technologies and Energy Engineering, Shahid Beheshti University, Tehran, Iran and ²Department of Biotechnology, International Imam Khomeini University, Qazvin, Iran.

Abstract: Fusarium head blight (FHB) caused by Fusarium graminearum is one of the most important wheat diseases in Iran and worldwide. In this research, we assessed genetic control and the disease resistance heritability using generation mean and generation variance analyses. Five generations of F₁, F₂, F₃, BC₁₁, and BC₁₂ developed from crossing an inbred line (resistant parent) and Falat (susceptible parent) along with two parents were infected by the fungus spores at spike emergence stage at field condition and after adequate incidence of disease, FHB resistance-related traits including percentage of infected florets, percentage of infected kernels (FDK) and disease incidence (DIC) were evaluated. Generation variance analysis showed that narrow-sense heritability of FDK was high (~64%), while that of DIC was very low, indicating the discrete genetic factors of these indices. Mean comparison indicated that means of F₁, F₂, BC₁₁, BC₁₂ and F₃ located between that of two parents and were closer to resistant parent. Joint-scaling test suggested that FHB resistance wasn’t fitted to additive-dominance model. But in contrast, six-parameter model (mean [m], additive [a], dominance [d], additive × additive [aa], additive × dominance [ad], dominance × dominance [dd]) for all FHB resistance indices was best fitted. Therefore, it could be concluded that in the examined cross in addition to additive and dominance effects, epistatic effects also were involved in controlling wheat FHB resistance.

Keywords: Fusarium, Generation mean, Heritability, Resistance, Wheat.

1. INTRODUCTION

Fungi of the group Fusarium spp. are belonged to the most important plant pathogens of many plant species. Normally, F. graminearum, which causes Fusarium head blight (FHB) in wheat and barley is the most devastative species in this group and produces different mycotoxins based upon environmental condition and host diversity [1]. The mycotoxins create toxicosis in human and animals. Moreover, wheat and barley are highly susceptible to infection when the crop is in the flowering to soft dough stages and when weather includes frequent precipitation, high humidity, or heavy dews [2, 3]. In addition to quantitative losses, F. graminearum also causes a reduction in grain quality due to the production of trichothecene mycotoxins [4].

Different methods may be used to reduce the disease undesirable effects including different fungicides, controlling the host weeds etc. However, cultivation of resistant varieties is a better choice to control this disease. Hence, one of important topics in plant pathology and plant breeding is to improve the resistance level, in which using disease-resistant genetic resources and transferring the resistance-related genes via backcrossing method or genetic engineering techniques, the resistance is transferred to susceptible varieties [5, 6]. The wheat FHB resistance genetic resources include Sumai 3 from China, Frontana from Brazil and Nobekabuzu from Japan [7]. Some research projects conducted out to investigate the FHB resistance genetics include: QTL mapping of FHB resistance in a population developed from the cross of Wangshuibai (resistant cv.) and Nanda2419 (susceptible cv.), indicating that using Wangshuibai resistance genes is possible in wheat FHB-resistance improvement programs [8].

Type of gene action play a key role in selection of breeding method of the given trait. Generation mean analysis (GMA) is an efficient method for genetic analysis of quantitative traits [9, 10]. It is a biometric method based on the measurement of given quantitative traits in basic breeding generations including two parents, F₁, backcrosses and segregating generations. Using the method one can estimate both main effects including additive and dominance effects, and digenic interactions, e. g. additive by additive, additive by dominance and dominance by dominance [9]. Estimation of these effects is critical for identifying breeding method (either hybrid production or inbred selection) as well as predicting the superior lines over better parent [10]. Recently we developed an inbred line of wheat (from Milan/Shanghai cross), which showed a considerable resistance against FHB. Study on FHB resistance components in this inbred line may give us an opportunity to use it in breeding programs of FHB resistance in Iran. The objectives of this research
were to assess genetic control and to estimate heritability of FHB-resistance-related traits in spring bread wheat (*T. aestivum*).

### 2. MATERIAL AND METHODS

FHB resistant line (P1; a line derived from Milan/Shanghai cross) was crossed to susceptible parent Falat (P2) in 2010 spring. Falat is a high-yielding and high-quality spring bread wheat, which its resistances to yellow rust and FHB have been broken because of recent emergence of highly pathogenic races of these two fungi, and hence area under its cultivation was considerably reduced in Iran. The F1 hybrid was backcrossed as male parent to resistant parent (BC1.1) and also to susceptible parent Falat (BC1.2) in 2011 spring. For this, several spikes on each of P1 and P2 plants were emasculated and pollinated with the pollen of F1 as male parent. An F2 population was obtained by self-pollination of F1 hybrid in the same year and F3 population was developed by self-pollination of single F2 plants in 2012 spring. In next season (November, 2012) all generations (F1, F2, F3, BC1.1 and BC1.2) along with two parents were sown in a randomized complete block design (RCBD) with three replicates in field experiment, research farm, Faculty of Agriculture, University of Agricultural Sciences and Natural Resources, Goran, Iran. In the next spring (2013) after spike emergence, a solution containing spores of FHB Gorgan isolate (105 spores per cm3) was repeatedly (3 times) sprayed on 18 plants of parents, F1, BC1.1 and BC1.2 (6 plants in each replicate), and on 48 plants of F2 and F3 populations (16 plants in each replicate). Nineteen days after infection (DAI) FHB resistance related traits including number of infected spikes and number of infected florets were measured, and after maturity and harvesting the test plants, number of infected seeds per plant were counted. Then two FHB resistance related indices including percentage of FHB-infected kernels (FDK) and percentage of disease incidence (DIC) (number of infected spikes divided by total number of spikes of all sprayed plants in each replicate) were determined [11].

Gene effects were symbolized as [9]: mean [m], additive [a], dominance [d], additive × additive [aa], additive × dominance [ad], dominance × dominance [dd]. Significance of gene effects was defined using t-student test. Variance components of additive (VA), dominance (VD), additive × dominance (VAD) and environmental (VE) were estimated as Kearsey and Pooni (1996) [9]. Fitness of 3- to 6-parameter models was tested using the analysis of least weighted squares for values of mean, standard error and generations variances [12]. Variance analysis (ANOVA) was conducted using SPSS v. 16 [13] and genetic analyses were conducted using SAS v.9.3.

### 3. RESULTS AND DISCUSSION

Analysis of variance showed that different generations had significant differences in all resistance-related traits including infected florets, FDK and DIC (Table 1), indicating the presence of genetic diversity between genotypes. Thus, it was possible to conduct out generation mean analysis (GMA), because generation effects must be significant to perform GMA [9, 10].

**Table 1: Analysis of Variance on FHB Resistance – Related Traits**

<table>
<thead>
<tr>
<th>Generations</th>
<th>d.f</th>
<th>Infected Florets</th>
<th>FDK</th>
<th>DIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generations</td>
<td>6</td>
<td>695.33**</td>
<td>1747.27**</td>
<td>1483.19**</td>
</tr>
<tr>
<td>Error</td>
<td>12</td>
<td>68.37</td>
<td>41.18</td>
<td>181.45</td>
</tr>
<tr>
<td>CV (%)</td>
<td></td>
<td>22.2</td>
<td>13.7</td>
<td>29.7</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>37.22</td>
<td>46.89</td>
<td>45.39</td>
</tr>
</tbody>
</table>

ns: non-significant; **: significant at 1% level of probability.

Mean comparison of different generations showed that two parents differentially responded to FHB, so that inbred line (P1) had very lower measures of infected florets, FDK and DIC relative to Falat (P2) (Figure 1). In the case infected florets, although means of F1, F2, F3, BC1.1 and BC1.2 located between that of two parents, however hadn`t a significant difference with means of resistant parent (P1) (Figure 1). This indicates that resistance was inherited as a complete or partial dominance trait. In the case of FDK and DIC all means of hybrid generations located between that of parents and not significantly differed from resistant parent, except for the mean of BC1.2, which showed significant difference with that of P1.

**Figure 1: Means of different generations for different FHB resistance-related traits.**
Estimation of variance components (Table 2) showed that in the case of FDK and DIC, there was a high broad-sense heritability (66.8 and 68.4%, respectively), while a high narrow-sense heritability was observed for FDK. Liu et al. (2005) [14] and Jiang and Ward (2006) [15] also reported a high broad-sense heritability, while they reported, respectively, a relatively low and a relatively high narrow-sense heritability for FHB resistance-related traits.

### Table 2: Variance Components and Heritability of the Studied Traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>FDK</th>
<th>DIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenotypic variance</td>
<td>35.51</td>
<td>247.39</td>
</tr>
<tr>
<td>Genetic variance</td>
<td>23.73</td>
<td>169.15</td>
</tr>
<tr>
<td>Environmental variance</td>
<td>11.77</td>
<td>78.24</td>
</tr>
<tr>
<td>Additive variance</td>
<td>22.64</td>
<td>1.71</td>
</tr>
<tr>
<td>Non-additive variance</td>
<td>1.09</td>
<td>167.44</td>
</tr>
<tr>
<td>Broad-sense heritability (%)</td>
<td>66.8</td>
<td>68.4</td>
</tr>
<tr>
<td>Narrow-sense heritability (%)</td>
<td>63.8</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Assessment of joint-scaling test showed that additive-dominance model wasn’t fitted for the studied traits (Table 3), indicating that in all resistance-related traits, epistasis was involved in their control. Testing k-parameter models revealed that for all studied traits no deviance was observed from six-parameter model (Table 4). Therefore, six-parameter model including mean (m), additive component (a), dominance component (d), additive by additive interaction (aa), additive by dominance interaction (ad) and dominance by dominance interaction (dd) was best fitted. This finding indicates that in addition to additive and dominance effects, epistasis also is involved in controlling FHB resistance. Previous research indicates that FHB resistance is quantitatively inherited and while genes with major effects have been identified, none confer complete resistance [4, 16]. Waldron et al. (2008) [17] reported the implication of epistasis effects in controlling FHB resistance. There are many examples of implication of epistasis in controlling different species in different traits: powdery mildew in sesame (Rao et al., 2011) [18], Fusarium root rot in common bean (Mukankusi et al., 2011) [19], common smut in maize (Namayandeh et al., 2011) [20] and six agronomic traits in durum wheat (Bnejdi et al. 2013) [21]. Epistasis complicates the procedure of fixation of desirable genes in the suitable cultivars. Due to quantitative inheritance and difficulties in screening for the presence of resistance genes [22], DNA markers associated with such genes may increase the efficiency of selecting for resistance [23]. Considerable improvements in genetic resistance to FHB have been achieved by conventional selection, due to repeated testing of breeding lines under induced and natural epidemic conditions. DNA-based markers can be applied to augment conventional breeding, especially for traits such as FHB that are difficult or cost intensive to select using conventional methods [16].

### Table 3: Results of Joint-Scaling Test to Test for Fitness of Additive-Dominance Model

<table>
<thead>
<tr>
<th>Trait</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>DIC</td>
<td>-1.00</td>
<td>-44.21</td>
<td>-99.55</td>
</tr>
<tr>
<td>FDK</td>
<td>0.42</td>
<td>-32.16</td>
<td>7.81</td>
</tr>
<tr>
<td>Infected florets</td>
<td>-21.94</td>
<td>-37.09</td>
<td>-17.12</td>
</tr>
</tbody>
</table>

ns: non-significant; * and **: significant at 5% and 1% level of probability, respectively.

### Table 4: Fitting Six-Parameter Model for Prediction of Model Components in FHB Resistance-Related Traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>DIC</th>
<th>FDK</th>
<th>Model Components</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>49.45±16.15</td>
<td>35.63±8.08</td>
<td>m</td>
</tr>
<tr>
<td></td>
<td>-32.35±5.65</td>
<td>-34.90±2.83</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>-44.33±49.29</td>
<td>0.46±24.66</td>
<td>d</td>
</tr>
<tr>
<td></td>
<td>18.75±16.39</td>
<td>20.63±8.20</td>
<td>aa</td>
</tr>
<tr>
<td></td>
<td>43.21±25.29</td>
<td>32.58±12.65</td>
<td>ad</td>
</tr>
<tr>
<td></td>
<td>35.73±37.97</td>
<td>6.48±18.99</td>
<td>dd</td>
</tr>
<tr>
<td>1.71</td>
<td>0.424</td>
<td>χ²</td>
<td></td>
</tr>
</tbody>
</table>

ns: non-significant; * and **: significant at 5% and 1% level of probability, respectively.

**REFERENCES**


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