Estimation of genetic parameters and effective factors in resistance to Septoria Leaf Blotch of wheat under field condition

Forouzan Heydari1, S. Sanaz Ramezanpour2, Hassan Soltanloo2, Mehdi Kalate Arabi3 and Shaban Kia3

1. MSc of Plant Breeding, Department of Plant Breeding and Biotechnology, Gorgan University of Agricultural Sciences and Natural Resources, Gorgan, Iran.
2. Assistant professor of Plant Breeding, Department of Plant Breeding and Biotechnology, Gorgan University of Agricultural Sciences and Natural Resources, Gorgan, Iran
3. Faculty staff of Gorgan Agricultural research centre. Gorgan, Iran

*Corresponding author email: ramezanpours@gau.ac.ir

Abstract: Septoria leaf blotch disease is caused by fungal agent Septoria tritici (Rob) Desm. In order to estimate the genetic mean components and effective factors controlling of resistance against septoria leaf blotch in adult plant stage, six basis generations (P1, P2, F1, F2, BC1, BC2) of cross between a promising line introduced from International Maize and Wheat Improvement Center (CIMMYT) along with two cultivars (Tajan and Moghan3) were planted in an experimental design of complete randomized blocks in the field of agricultural research center of Gorgan, Golestan. The studied traits include disease severity and disease severity area under the disease progress curve (sAUDPC). For both studied traits in crosses of promising line×Tajan and promising line×Moghan3, additive and dominance effects had a significant role in STB resistance. In promising line×Tajan and promising line×Moghan3 crosses, the selected model for disease severity is quite similar to the model for sAUDPC; it may be due to the high correlation between these traits. The additive×additive [i] and dominance×dominance [l] nonallelic interactions had significant effect in studied traits. There was only a significant heterosis for sAUDPC in cross of promising line×Tajan. The minimum number of genes or effective factors controlling in resistance to STB disease was varied between one to two in crosses.

Keywords: AUDPC, heterosis, heterobeltiosis, heritability.

INTRODUCTION

Two separate pathogens cause septoria disease on wheat: Stagonospora nodorum (Berk) cast (syn Septoria nodorum (Break) agent of leaf stagonosora or glume blotch (SNB) and Septoria tritici (Rob) Desm agent of septoria leaf blotch or leaf spot blotch (STB) (Gaurilickienë and Ronis, 2006). Economically, both pathogens have global importance (Cunfer and Ueng, 1999). S. nodorum identified as a pathogenicity which mostly appears in final stage of growth and causes higher levels of blotching in leaf and glume in higher temperatures, while lower temperatures in spring lead to favorable development of induced leaf blotch by S. tritici. The disease agent of septoria leaf blotch has two sexual and asexual in the life cycle. The asexual state is Septoria tritici Rob ex Desm agent which belongs to Deuteromycota kingdom and Sphaeropsidales phylum and produces pycnids with 200–600 µm diameters in epiderm and mesophyll on both surface of a leaf with a stoma in its top. Pycnidiospores are released from pycnidia
when the leaf has been wet for 30 minutes or more. The spores are produced in a thick, sticky matrix containing a high concentration of preserving sugars and proteins. This "preserving medium" named ooze or cirrhus permits the pycnidiospores to remain viable during periods of dry weather (Eyal et al., 1987). Kema et al (2000) through studying on five wheat cultivars in the seedling stages concluded that avirulence in the wheat Septoria tritici leaf blotch fungus, *Mycosphaerella graminicola*, in the isolate, IPO323, is controlled by a single locus. Heritability resistance to the wheat septoria leaf blotch was studied through disease expansion on flag leaf and leaves under the flag leaf and it was mentioned that this resistance is controlled by a single gene (Diaz and Tavella, 1982). In recent years, twelve major genes of *Stb*$_1$ to *Stb*$_{12}$ were identified for the resistance to *M. graminicola* (Chartrain et al., 2005).

Increased in disease severity resulted in linear reductions in test weight ($r = 0.97^\circ$), milling quality ($r = 0.98^\circ$), adjusted flour yield ($r = 0.97^\circ$) and a linear increase in water absorption in the flour ($r = 0.95^\circ$). Increase in the disease severity also resulted in an increase in flour protein and a decrease in baking quality, however, the linear correlation coefficients were not significant (Mckendry et al., 1995). The Generation mean analysis is a genetic project of which disease genotypic value is divided into its composed components, including additive, dominance and epistasis. In a study of eight spring wheat genotypes in a diallel genetic project, Ramezanpour et al (2010) declared that two distinct traits, disease severity and sAUDPC, show a high significant variation due to the general combining ability. Also, the specific combining ability indicated the importance of the additive as well as the non-additive type of gene action in inheritance of the characters. On the other hand, Baker ratio showed the higher importance of additive effects than the non-additive effects of genes for both traits. Some negative and significant heterosis as well as heterobeltiosis effects have emphasized on the existence of dominance gene effects in order to control resistance against STB. Epistasis type and the required amount which is effective in controlling traits, have a great impact on the estimation of validity and planning breeding project. For example, additive$\times$additive interaction may be stabilized in inbreed lines. For a successful breeding project, genetic variety and awareness of gene action is necessary for improving resistance. Otherwise, the selection of breeding methods will not obtain appropriate results. In this study, two cultivars and a promising line from International Maize and Wheat Improvement Center (CIMMYT) were evaluated in a genetic project of generation mean analysis (GMA) and resistance genetic analysis in adult plant.

**MATERIALS AND METHODS**

Three spring bread wheat genotypes were selected based on preliminary field and greenhouse observations of their reaction to *S. tritici* (Table 1). One out of three genotypes was promising line that was introduced from International Maize and Wheat Improvement Center (CIMMYT), while the rest were cultivar. In 2009–2010, genotypes included parents (P$_1$ and P$_2$), F$_1$ and F$_2$ generations and backcrosses (BC$_1$ and BC$_2$) were planted in the field of agricultural research center of Gorgan, Golestan (Araghi Mahaleh) in a randomized completed block design (RCBD) with three replications under mist irrigation. In all crosses, the most resistant genotype was considered as a first parent (P$_1$), while susceptible genotype was considered as a second parent (P$_2$). F$_1$ generation was obtained through hand emasculation and pollination and resulted in the selfing of F$_1$ generation was obtained F$_2$ generation. BC$_1$ has been obtained by F$_1$ generation backcross with a more resistant parent and BC$_2$ resulted from the backcross of F$_1$ generation with a more susceptible parent.

**Table 1. Applied wheat genotypes in crosses**

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Pedigree</th>
<th>Infection response</th>
</tr>
</thead>
<tbody>
<tr>
<td>promising line</td>
<td>BOBWHITE#1/FENGKANG</td>
<td>R</td>
</tr>
<tr>
<td>Moghan3</td>
<td></td>
<td>MS</td>
</tr>
<tr>
<td>Tajan</td>
<td></td>
<td>S</td>
</tr>
</tbody>
</table>

R=Resistant, MS=Moderately Susceptible, S=Susceptible

Direct method of Eyal (1999) was applied for extracting the pathogen. Plants were inoculated at tillering, long stem and appearance flag leaf stages. Disease rating was visually recorded as soon as the first symptoms appeared on the lowest leaves 5 times with 6 interval days using the double-digit scale
(00-99) which developed as a modification of Sarri and Prescott severity scale to assess wheat foliar disease (Eyal et al., 1987; Saari and Prescott, 1975). Disease severity of the last assessment while genotype as susceptible check was severely diseased (90% or more disease severity) and sAUDPC (area under the disease progress curve of disease severity) were used for analysis.

Obtained data were analyzed for below purposes: the generation mean analysis (GMA), area under disease progress curve (AUDPC), effective factors, heritability, genetic advance produced by selection, heterosis, heterobeltiosis, genetic variance components, degree of dominance, dominance deviation and correlation. Generation mean analysis has been accomplished by Minitab software and disease severity percentage was calculated based on the formula 1 (Sharma and Duveiller, 2007).

\[
\% \text{Severity} = \left(\frac{D_i}{9}\right) \times 100
\]

In which \(D_i\) demonstrates vertical disease progress and \(D\) refers to severity measured as diseased leaf area. Disease measuring used for resistance screening which include: identification of a point in growth key stage, calculation of disease development rate and area under disease progress curve. The sAUDPC was calculated using severity percentage estimates based on formula 2 (Irfaq et al., 2009).

\[
\text{AUDPC} = \sum \left[\frac{(X_i + X_{i+1})}{2}\right] t_i
\]

That in which \(X_i\) and \(X_{i+1}\) shows the severity on i and i+1 data, respectively. \(t_i\) = the number of days between i and i+1 data

The minimum numbers of genes or effective factors controlling resistance were estimated by 10 methods for both traits. \(1\ EF\) (Formula 3) proposed by Wright (1968), \(2\ EF\) (Formula 5) proposed by Mather and Jinks (1982) and \(3\ EF\) (Formula 6), \(4\ EF\) (Formula 7) and \(5\ EF\) (Formula 8) proposed by Lande (1981) (quoted from Amanda and Wehner, 2001). There are many hypotheses in estimating effective factors, for example there is no nonallelic interaction, genetic differences have equal effects, similar Alleles have complete linkage in parents and there is no linkage between genes (Taleei, 2000).

\[
\begin{align*}
\sigma^2 = & \frac{(P_2 - P_1)^2}{12} \times 8\left[\sigma^2_{S1} + \sigma^2_{S2} + 2\sigma^2_{S3}\right] \\
\end{align*}
\]

In this formula, \(S^2\) is segregating genetic variance component. Lande (1981) presented four methods to calculate the segregating genetic variance (Formula 10, 11, 12 & 13) and through their replacement in formula 9, \(n_1\), \(n_2\), \(n_3\) and \(n_4\) amounts were evaluated.

\[
\begin{align*}
S^2_{S1} &= S^2_{S2} - S_{S1} \\
S^2_{S2} &= S^2_{S2} - (0.5S^2_{P1} + 0.25S^2_{P2} + 0.25S^2_{P3}) \\
S^2_{S3} &= 2S^2_{S2} - S^2_{Btp} - S^2_{BC2p} \\
S^2_{S4} &= (S^2_{Btp} + S^2_{BC2p}) - (S^2_{P1} + 0.5S^2_{P2} + 0.5S^2_{P3})
\end{align*}
\]

Subsequently, Cockerham (1986) corrected the parental means by their standard errors and combined the formulae for the genetic variance into one by using the least squares to prepare another estimate for gene number (Formula 14) (quoted from Mcpherson et al, 2004).

\[
M = \left[P_2 - P_1\right] \times \left[8\left(S^2_{S1}\right)^{-1}\right]
\]

In which \(N\) is the number of plants, so \(S^2_{S1}\) (Formula 15) can be obtained by following equation.
The increase or decrease percentage of \( F_1 \) hybrids over mid parent as well as better parent was calculated to estimate possible heterosis (Ht) and heterobeltiosis (Hbt) using formula 16 and 17, respectively (Fonseca and Patterson, 1968).

\[
Ht(\%) = \frac{\bar{F}_1 - MP}{MP} \times 100 \quad (16)
\]

\[
Hbt(\%) = \frac{\bar{F}_1 - BP}{BP} \times 100 \quad (17)
\]

In these formula, \( \bar{F}_1 \) is the means of \( F_1 \) hybrid, MP is the mid parent value and BP is the better parent value. The t-test was manifested to determine whether \( F_1 \) hybrid means were statistically different from mid parent (Formula 18) and better parent means (formula 19) as follows (Wynne et al., 1970).

\[
t_i = (\bar{F}_{ij} - MP_{ij}) / \sqrt{3/8(EMS)} \quad (18)
\]

\[
t_i = (\bar{F}_{ij} - BP_{ij}) / \sqrt{1/2(EMS)} \quad (19)
\]

In which \( \bar{F}_{ij} \) is the means of the \( ij^{th} \) \( F_1 \) cross, \( MP_{ij} \) is the mid parents for the \( ij^{th} \) cross, \( BP_{ij} \) is better parent value for the \( ij^{th} \) cross and EMS is error mean square.

RESULTS AND DISCUSSION

Obtained results through variance analysis of studied traits (Table 2) demonstrated the significant difference between generations and the possibility of genetic analysis of these traits. In this study, all of two, three, four, five, and six parametric models were tested for better understanding of traits genetic system controlling. Through the study of all models, the best-fitted obtained model was a model in which Chi square was not significant, its all parameters were significant and their standard error was less (Table 3). In promising line×Tajan cross for both traits, disease severity and sAUDPC, three parametric model was selected as the best fit and its all parameters including: mean, additive effect and additive×additive nonallelic interaction which were significant. The importance of additive effects (additive \([d]\) and additive×additive \([i]\)) is due to their fixability. In those crosses that the total of additive effects (\([d]+[i]\)) is more than the total of dominance effects (\([h]+[l]\)), the selection method will be adequate for the trait improvement in early generations, whereas in those crosses that the total of dominance effects is greater than the total of additive effects, selection should be accomplished in more advanced generations (Mather and Jinks, 1982). In promising line×Tajan cross, selection will be made in early generations because of \([h]+[l]<[d]+[i]\) and the high narrow-sense heritability (Table 4). Here, breeding methods such as pure line selection or backcross method may be used. In promising line×Moghan3 cross, four parametric model was applied for both traits, disease severity and sAUDPC and its all parameters including: mean, additive and dominance effects and dominance×dominance nonallelic interaction were significant. In this cross dominant effects (\([h]+[l]\)) was higher than additive effects (\([d]+[i]\)). Therefore, it is suggested that selection will be postponed to late generations when dominance effects appear. The sign of dominance effect (\(h\)) and dominance×dominance nonallelic interaction (\(l\)) were different and demonstrates some duplicate epistasis. Duplicate epistasis limits variability range and decreases its progress speed via selection, so heterosis breeding may be useful (Iqbal and Nadeem, 2003). In promising line×Tajan cross, the selected model for disease severity is quite similar to the model for sAUDPC, Also in promising line×Moghan3 cross the same process was observed, that it may be due to the high correlation between these traits (Tables 7 and 8). Regarding the importance of this point, it seems essential to note that epistasis effects have acted in the field for increasing their trait appearance (due to their positive sign) so they possess less importance in the resistance against the disease.

<table>
<thead>
<tr>
<th>Cross</th>
<th>Source of variation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Variance analysis for traits in different crosses
Estimations of degree of dominance, broad-sense heritability, narrow-sense heritability, the average of broad-sense heritability, genetic advance produced by selection, heterosis and heterobeltiosis percent are mentioned in Table 4. The broad-sense heritability \( (h^2_{b.s}) \) was estimated by four formula and their average was calculated: \( h^2_{b.s} \) was suggested by Allard (1960), \( h^2_{b.s} \) was suggested by Mahmud and Keramer (1951), \( h^2_{b.s} \) was suggested by Warnner (1952) and \( h^2_{b.s} \) and the narrow-sense heritability \( (h^2_{n.s}) \) were applied by Valizade and Moghadam (2007). Heritability amount depends on all components of variance, so changing in each of the variance components can be affects it. Whereas the gene frequency is different between populations, the estimated heritability of a trait cannot be generalized to other conditions and populations. In promising line×Moghan3 cross for both studied traits, narrow-sense heritability was less than broad-sense heritability, because of the role of dominance effect in controlling the traits. It corresponds with the obtained result by Mohammadi (2010). In studying four spring bread wheat genotypes and one promising line from International Maize and Wheat Improvement Center (CIMMYT), Mohammadi (2010) represented that high difference between broad-sense heritability and narrow-sense heritability can be due to the role of dominance effect in traits control. In promising line×Tajan cross sAUDPC, narrow-sense heritability was high (0.65) which shows that the high proportion of the genetic variance component may be stabilized by selection in segregating generations. Genetic advance was calculated based on this assumption that there is one percent selection for plants with less appearance of studied traits. Genetic advance for sAUDPC in promising line×Tajan cross was 2.5491. Due to the high percentage of heritability and genetic advance, it can be stated that sAUDPC is controlled under the action of additive genes and in return, it enables the breeder to set the basis of selection plan on traits phenotypic appearance.

### Table 3. Estimation of mean genetic components by the use of \( P_1, P_2, F_1, F_2, BC_1 \) and \( BC_2 \) generations for different traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>cross</th>
<th>( m )</th>
<th>( \hat{d} )</th>
<th>( \hat{h} )</th>
<th>( \hat{i} )</th>
<th>( \hat{j} )</th>
<th>( \hat{l} )</th>
<th>( X^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>( P_1 \times P_2 )</td>
<td>0.55±0.017</td>
<td>-0.09±0.01</td>
<td>-</td>
<td>0.131±0.022</td>
<td>-</td>
<td>-</td>
<td>2.26(^{n.s})</td>
</tr>
<tr>
<td>Severity</td>
<td>( P_1 \times P_3 )</td>
<td>0.7±0.004</td>
<td>-0.072±0.004</td>
<td>-0.68±0.073</td>
<td>-</td>
<td>-</td>
<td>0.554±0.095</td>
<td>1.18(^{n.s})</td>
</tr>
<tr>
<td>sAUDPC</td>
<td>( P_1 \times P_2 )</td>
<td>6.22±0.30</td>
<td>-2.49±0.24</td>
<td>-</td>
<td>3.56±0.40</td>
<td>-</td>
<td>-</td>
<td>3.34(^{n.s})</td>
</tr>
<tr>
<td>sAUDPC</td>
<td>( P_1 \times P_3 )</td>
<td>8.81±0.27</td>
<td>-1.39±0.26</td>
<td>-11.53±1.41</td>
<td>-</td>
<td>-</td>
<td>8.89±1.47</td>
<td>0.557(^{n.s})</td>
</tr>
</tbody>
</table>

### Table 4. Estimation of degree of dominance, heritability, genetic advance, heterosis and heterobeltiosis for different traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>cross</th>
<th>( h/d )</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>( \bar{h}^2_{b.s} )</th>
<th>( h^2_{n.s} )</th>
<th>GA</th>
<th>Ht%</th>
<th>Hbt%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>( P_1 \times P_2 )</td>
<td>-1.70</td>
<td>0.67</td>
<td>0.8</td>
<td>0.79</td>
<td>0.44</td>
<td>0.69</td>
<td>0.44</td>
<td>0.15</td>
<td>-20.89(^{n.s})</td>
<td>-9.82(^{n.s})</td>
</tr>
<tr>
<td>Severity</td>
<td>( P_1 \times P_3 )</td>
<td>-1.77</td>
<td>0.43</td>
<td>0.92</td>
<td>0.73</td>
<td>0.97</td>
<td>0.76</td>
<td>0.02</td>
<td>0.003</td>
<td>-18.36(^{n.s})</td>
<td>-8.9 (^{n.s})</td>
</tr>
<tr>
<td></td>
<td>( P_1 \times P_2 )</td>
<td>( P_1 \times P_3 )</td>
<td>( P_2 \times P_3 )</td>
<td>( P_1 \times P_4 )</td>
<td>( P_2 \times P_4 )</td>
<td>( P_3 \times P_4 )</td>
<td>( P_4 \times P_1 )</td>
<td>( P_4 \times P_2 )</td>
<td>( P_4 \times P_3 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>sAUDPC</td>
<td>-1.64</td>
<td>-1.84</td>
<td>-1.67</td>
<td>-0.68</td>
<td>-0.94</td>
<td>-0.83</td>
<td>-0.66</td>
<td>-0.93</td>
<td>-1.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( h_s )</td>
<td>-0.23</td>
<td>-0.21</td>
<td>0.37</td>
<td>0.68</td>
<td>0.72</td>
<td>0.54</td>
<td>0.52</td>
<td>0.81</td>
<td>0.62</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( h_d )</td>
<td>-0.48</td>
<td>-0.24</td>
<td>0.18</td>
<td>0.97</td>
<td>0.87</td>
<td>0.65</td>
<td>0.56</td>
<td>0.71</td>
<td>0.65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( h_b )</td>
<td>0.65</td>
<td>0.48</td>
<td>0.97</td>
<td>0.4</td>
<td>0.02</td>
<td>0.073</td>
<td>0.65</td>
<td>2.55</td>
<td>-21.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.35</td>
<td>0.18</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Degree of dominance (\( \frac{h_d}{d} \)), broad-sense heritability (\( \sigma^2_g + \sigma^2_e \)), genetic advance (\( GA = i \times c \times h_s^2 \times \sigma_{P_2} \)), heterosis (\( H_t \)) and heterobeltiosis (\( H_{bt} \))

\( P_1 \): Promising line, \( P_2 \): Tajan, \( P_3 \): Moghan
Table 5. Estimation of effective factors for different traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>cross</th>
<th>$EF_1$</th>
<th>$EF_2$</th>
<th>$EF_3$</th>
<th>$EF_4$</th>
<th>$EF_5$</th>
<th>$n_1$</th>
<th>$n_2$</th>
<th>$n_3$</th>
<th>$n_4$</th>
<th>$M$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>$P_1 \times P_2$</td>
<td>0.8063</td>
<td>0.6036</td>
<td>0.3294</td>
<td>0.3018</td>
<td>0.3625</td>
<td>0.3692</td>
<td>0.3294</td>
<td>0.3018</td>
<td>0.3625</td>
<td>0.3111</td>
</tr>
<tr>
<td></td>
<td>$P_1 \times P_3$</td>
<td>-</td>
<td>0.1141</td>
<td>2.1978</td>
<td>0.057</td>
<td>0.0602</td>
<td>0.5336</td>
<td>2.1978</td>
<td>0.057</td>
<td>0.0601</td>
<td>0.2828</td>
</tr>
<tr>
<td>sAUDPC</td>
<td>$P_1 \times P_2$</td>
<td>-</td>
<td>1.3772</td>
<td>-</td>
<td>0.6886</td>
<td>0.5643</td>
<td>-</td>
<td>-</td>
<td>0.6886</td>
<td>0.5643</td>
<td>1.8003</td>
</tr>
<tr>
<td></td>
<td>$P_1 \times P_3$</td>
<td>-</td>
<td>0.4408</td>
<td>-</td>
<td>0.2204</td>
<td>0.2414</td>
<td>-</td>
<td>-</td>
<td>0.2204</td>
<td>0.2414</td>
<td>1.5553</td>
</tr>
</tbody>
</table>

$P_1$: Promising line, $P_2$: Tajan, $P_3$: Moghan

Degree of dominance for both traits in the crosses was greater than one ($\frac{h}{d} > 1$) and negative which demonstrates an overdominance for less disease severity on leaf area unit (Table 4). Pseudo overdominance induced by trans-linkage of locus represents complete or incomplete dominance (Jones, 1917).

Heterosis and heterobeltiosis for both traits in the crosses were negative (Table 4) which showed usefulness of hybrid combinations to decrease the traits (increasing resistance). Only in promising line×Tajan cross, heterosis was significant for sAUDPC, which demonstrated that hybrid combinations significantly decrease the trait. Although in promising line×Tajan cross for the total of additive effects was greater than the total of dominance effects sAUDPC but there was a significant heterosis. It can be because of the fact that it is the existence of epistasis effects theory for the justification of heterosis in which significant additive×additive nonallelic interaction was observed for trait control. Such a process was observed for the disease severity but heterosis percent was not significant. In justifying this subject, we can refer to the possibility of heterosis existence increases in overdominant genes action, as it was observed for the incomplete dominant disease severity and overdominant sAUDPC (Table 6).

Table 6. Estimation of variance components for different traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>cross</th>
<th>$E_w$</th>
<th>D</th>
<th>H</th>
<th>F</th>
<th>$\sqrt{H/D}$</th>
<th>F / $\sqrt{HD}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity</td>
<td>$P_1 \times P_2$</td>
<td>0.005</td>
<td>0.023</td>
<td>0.003</td>
<td>-0.01</td>
<td>0.409</td>
<td>-1.134</td>
</tr>
<tr>
<td></td>
<td>$P_1 \times P_3$</td>
<td>0.003</td>
<td>0.091</td>
<td>0.178</td>
<td>0.023</td>
<td>1.395</td>
<td>0.186</td>
</tr>
<tr>
<td>sAUDPC</td>
<td>$P_1 \times P_2$</td>
<td>1.661</td>
<td>9.029</td>
<td>20.047</td>
<td>2.749</td>
<td>1.49</td>
<td>0.204</td>
</tr>
<tr>
<td></td>
<td>$P_1 \times P_3$</td>
<td>1.207</td>
<td>9.52</td>
<td>18.211</td>
<td>-3.235</td>
<td>1.383</td>
<td>-0.245</td>
</tr>
</tbody>
</table>

Environmental variance ($E_w = (\frac{\sigma_p^2 + \sigma_2^2 + 2\sigma_{p2}}{4})$, additive variance ($D = 4(\Sigma F_2 - 2(\Sigma V_{BC1} + V_{BC2})$, dominance variance ($H = 4(V_{BC1} + V_{BC2} - F_2 - V_{BC})$, ($F = V_{BC2} - V_{BC1}$), the mean degree of dominance ($\sqrt{H/D}$) and dominance deviation ($F / \sqrt{HD}$).

$P_1$: Promising line, $P_2$: Tajan, $P_3$: Moghan

For both crosses, estimated effective factors were between one and two (Table 5), which is correspondent with Kema et al (2000) and Diaz and Tavella (1982) results. Here inapplicability of some assumptions leads to inadvisable estimations for traits, these estimations are shown in the table as dashes.

Variance components was shown in Table 6 and included: environmental variance, additive variance, dominance variance, F amount, mean of degree of dominance and dominance deviation. The degree of dominance in promising line×Tajan cross was varied between incomplete dominance and overdominance, while in promising line×Moghan3 cross was shown overdominant. The negative sign of F, demonstrated that dominance genes are mostly located in parents who has less appearance of studied trait and positive sign of F showed that dominant genes are mostly gathered in parents who have more appearance of studied trait. Only in promising line×Tajan cross, dominance deviation was estimated.
greater than one \( \left( \frac{F}{\sqrt{HD}} > 1 \right) \), and it shows that the controlling genes of studied traits are the same in sign and magnitude in different gene loci. If \( \frac{F}{\sqrt{HD}} \) was smaller than one, the sign and magnitude of controlling genes of studied trait would be different or some alleles act for declining the trait. In promising line×Tajan and promising line×Moghan3 crosses, correlation between two traits estimated as 0.965 (Table 7) and 0.969 (Table 8), respectively, which is demonstrative of high correlation and significance between studied traits.

| Table 7. Correlation between traits in promising line×Tajan cross |
|-----------------------|------------------|
| Severity              | sAUDPC           |
| Severity              | 1                |
| sAUDPC                | 0.96             |

**: significant in possible level of 1 percent

| Table 8. Correlation between traits in promising line×Moghan3 cross |
|-----------------------|------------------|
| Severity              | sAUDPC           |
| Severity              | 1                |
| sAUDPC                | 0.96             |

**: significant in possible level of 1 percent

ACKNOWLEDGEMENT

To the memory of my passed away brother Alireza heydari who did not deprived me from any encouragement in my education.

REFERENCES

Lande R. 1981. The minimum number of genes contributing to quantitative variation between and within populations. Genetics 99: 541-553.