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Generation Mean Analysis of Net Blotch and Scald Diseases on Barley

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Abstract The information on the nature and magnitude of genes controlling the resistance to net blotch and scald in barley is useful in resistance breeding. Thus, field experiment was conducted at Holetta, Ethiopia in 2015 on barley using six basic generations of (P_1, P_2, F_1, F_2, BC_1, and BC_2) which was derived from 'HB42'x'Sabini' parental cross which were evaluated in RCB design with three replications to investigate the gene actions and interactions gene effects involved in controlling resistance to net blotch and scald diseases in barley. The result of scaling tests and generation mean analysis indicated the predominance of non-additive gene effects including epistasis gene effects than additive gene effects for all parameters and this was also confirmed by variance component analysis. Moreover, the digenic epistatic model failed to explain variation in generation means for all parameters may due to the presence of higher-order interaction and linkages. In general, the results suggest greater influence of non-additive genes including epistasis in the control of both disease parameters studied making early selection ineffective. And 'HB42' cultivar contained double resistance to scald and net blotch diseases which needs further study for its exploitation as source of resistance in improving barley yield.

Keywords: *barley disease resistance, gene effects, non-allelic interaction type of gene effects*

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1. Introduction

Barley is among the oldest domesticated cereal crop in Ethiopian, landraces show high diversity and recognized as a center of diversity constituting barley germplasms having global significance because of important traits including disease resistance [1,2]. Currently, the national average productivity of barley is about 2.2 tons ha^{-1} in Ethiopia [3] while the world barley average productivity level was about 3.0 tons ha^{-1} [4] indicating the existence of a large yield gap. Net blotch (*Pyrenophora teres*) and scald (*Rhynchosporium commune)* are among common yield-limiting foliar diseases of barley worldwide including Ethiopia. For instance, in Australia, reports showed that yield losses from net blotch generally range between 10 and 20% whereas grain yield losses of between 12 and 18% where scald symptoms affected between 20-60 % leaf area of the top three leaves [5]. And yield decreases of up to 40% and reduced grain quality has been also reported due to scald [6]. Moreover, in Ethiopia net blotch (*Pyrenophora teres*) and scald (*Rhynchosporium cummune*) diseases are the most widely distributed diseases threatening barley crop in Ethiopia. The yield loss assessment over locations in central high lands of Ethiopia showed mean grain yield loss due to net blotch and scald combined ranged from 14 % to 25 % in 1999 and 2000, respectively while yield losses of 9.8-31.54% resulted from scald in western Ethiopia [7].

Beside this, Ethiopia is one of the major producers of barley in Sub-Saharan Africa and has a growing malt beverage sector. Yet, despite a favorable biophysical environment, conventional varieties tend to be low-yielding and susceptible to pests and disease. In the 2015 year, Ethiopia was forced to import over 63,000 tons of malt, at a cost of 38 million USD [8] and most of malt barley cultivars are introduced from abroad as there has been no malt barley cultivar from Ethiopia landraces that fulfill the malting qualities. However, most of these introduced cultivars were not adaptable to the growing situation in Ethiopia as they are susceptible to scald and net blotch major barley diseases need improvement of its resistance. Many years of research effort and experiences in Ethiopia of a screening of resistant barley plants to major diseases from landraces and introduction of germplasm showed limited success [9] indicating the prescience of few resistant cultivars but not adequately exploited in resistance breeding. Therefore, to utilize the existing resistance sources like 'HB42' cultivar efficiently in

breeding program, investigation on the nature and magnitude of gene actions and interaction of resistance genes is useful.

The improvement in the productivity of a crop involves a multidimensional approach including thorough knowledge of the genetics of the crop under consideration. Productivity is the result of action and interactions of various yield-related traits that have a polygenic inheritance. The knowledge on the nature and magnitude of gene effects controlling inheritance of traits related to crop productivity will in turn become useful in formulating an effective and efficient breeding program [10]. Knowledge of the way genes act and interact will determine which breeding system can optimize gene action more efficiently and will help elucidate the role of breeding systems in the evolution of crops [11]. In this regard, although diallel analysis is an effective and widely used [12] it fails to detect epistasis or non-allelic interaction of genes [13]. Generation means analysis is a useful technique in plant breeding for estimating gene effects (additive and dominance) and their disgenic (additive x additive, additive x dominance, and dominance x dominance) interactions responsible for the inheritance of quantitative traits [14]. Thus the presence or absence of epistasis can be detected through the generation mean analysis using scale test that measures epistasis accurately whether it is complementary (additive x additive) or duplicate (additive x dominance) and (dominance x dominance) at disgenic level [15,16]. And this classic method, which is also complementary to modern breeding, is still useful in the absence of modern tools.

Thus, to increase the yielding potential of barley plants through resistance breeding, the knowledge on the genetic control of disease resistance of parents has major significance in utilizing the resistance sources at hand. 'HB42' cultivar was nationally released food barley which is resistant to scald and net blotch as well as 'Sabini' cultivar (introduced, susceptible to scald and net blotch) was malting barley selected to conduct generation mean analysis. Hence, as a part of an effort to improve the yielding potential of malt barley varieties, a crossing was made between 'HB42' and 'Sabini' cultivars to generate six basic generations (both parents, F_1, F_2, BC_1, and BC_2) to investigate the genetic control of disease resistance for future breeding. Therefore, this study aimed to investigate the gene actions and interactions involved in controlling resistance to both net blotch and scald diseases.

2. Materials and Methods

2.1. Development of Six Basic Generations

The generation means analysis was carried out on six basic generations (P_1, P_2, F_1, F_2, BC_1, and BC_2) derived from a cross between 'HB42' and 'Sabini' cultivars in the main cropping season 2014. All the six basic generations were produced in both 2014/15 main and the small rainy season of 2015 at Holetta Agricultural Research Center. 'HB42' cultivar is mainly characterized by high resistance to net blotch and scald diseases and high yield potential while 'Sabini' cultivar was introduced cultivar from abroad and was highly susceptible to net blotch and scald diseases and low yielding. P_1 ='HB42' parent, P_2 ='Sabini' parent, the F_1 and F_2 are (first and second filial generations of both parental cross), and BC_1 designate first back cross with 'HB42' parent and BC_2, was a backcross with 'Sabini' cultivar. The parents of the cross hybrid were used as the male parent and the F_1 generation as the female parent to obtain the BC_1 (F_1 backcrossed to P_1) and BC_2 (F_1 backcrossed to P_2) generations. F_1 plants of the cross were selfed and backcrossed to the two parents to obtain F_2, BC_1 and BC_2 generations in the following 2015 small rainy season. F_1 and backcrosses were obtained by hand emasculation and pollination in the field.

2.2. Experimental Design and Planting Methods

All the six generations were grown in a randomized complete block design with three replications and barley seeds were sown in variable rows as follows: two rows each for the non-segregating homogenous populations P_1, P_2 and F_1 generations, six rows for F_2 generation and five rows for the BC_1 and BC_2 generations heterogeneous populations, at Holetta Agricultural Research Center, about 30 km west of Addis Ababa, Ethiopia.

2.3. Data Collection

Data were collected on net blotch and scald diseases. The number of plants sampled for data collection was also varied as follows: ten plants for the P_1, P_2 and F_1 generations, 40-127 plants for the F_2 generations for different parameters and 30-70 plants in the BC_1 and BC_2 generations.

2.4. Net Blotch and Scald Assessment

Disease severity for net blotch disease was scored on ten randomly chosen plants for P_1, P_2 and F_1 generations, 40 plants for the F_2 generations, and 30 plants for the BC_1 and BC_2 generations in each plot using modified Saari and Prescott's double-digit scale (D1D2, 00-99) scoring method [17] which was based on the severity scale to assess foliar diseases in cereals. The first digit (D1) indicates the relative height of the disease on the plant and corresponds to the vertical disease progression using the original 0-9 Saari-Prescott scale. While the second digit (D2) refers to severity measured as diseased leaf area. From 21 September 2015 year, disease scoring was repeated five times at seven days intervals starting at growth stages of 53 Zadok's scale [18] as modified by Tottman and Makepeace [19]. For each score, the percentage of disease severity was estimated using the formula of disease severity (%) = (D1/9) × (D2/9) × 100 [20] and area under disease pressure curve (AUDPC) were calculated to estimate the scald severity over time based on the five growth stages' percent disease severity estimation according to Shaner and Finney [21] formula:

$$AUDPC = \sum_{i}^{n-1}\left[(Y_i + Y_{i+1})/2\right](T_{i+1} - T_i)$$

where Y_i=the disease severity on the i^{th} date, $T_{(i+1)}$-$T_{i=}$ time or days between two disease scores, n=number of dates on which the disease was recorded.

2.5. Data Analysis

Data were subjected to analysis of variance according to Steel and Torrie [22] and the generation means analysis was computed using the Statistical Package for Agricultural Research software (SPAR2) [23]. The means and variances were calculated as suggested by Hayman [24]. The scaling tests ('A', 'B' 'C', and D), were applied based on Mather [25] and Hayman and Mather [26] methods to test the adequacy of additive dominance model to detect the presence of non-allelic interactions of gene effects or epistasis. Furthermore, generation means analysis was performed using Hayman [24] and Mather and Jinks [27] method to detect the presence of non-allelic interactions of gene effects. The joint scaling test [27,28] indicated by Chi-square (χ^2) was extended to fit the six parameter model to test the adequacy of the genetic models using $\chi 2$ test. The simple additive dominance model [29] [m], [d], and [h] was applied when χ^2 was non-significant or epistasis was absent. Components within each model were evaluated for significance by t-test. Type of epistasis, predominantly duplicate or complementary, was determined based on the relative signs of dominance [h] and dominance x dominance [l] component effects. When these effects had the same sign, the effects were complementary while different signs indicated duplicate epistasis [14].

Heterosis and percent inbreeding depression were calculated for characters studied according to Mather and Jinks method [27]: percent inbreeding depression (ID) =$[F_1-F_2/F_1)]$x100, mid parent (MP) heterosis= $[F_1$-MP/MP]x100 and heterobeltosis or better parent (BP) heterosis= $[F_1$-BP/BP]x100. Variance components (additive, dominance and environment) were estimated as described by Kearsey and Pooni [14] using the following formulas: Environment variance-V[E] = 1/4 (VP_1 + VP_2 + $2VF_1$), Additive variance - V[d] = ($2VF_2$ –VBC_1 -VBC_2),Dominance variance- V[h] = 4 (VF_2 –1/2$V_{[d]}$ –V[E]), Average degree of dominance- $(H/D)^{1/2}$= $(V[h]/V[d])^{1/}2$,F = (V_{BC1} -V_{BC2}), where V-stands for variance and the subscripts refer to generations. F- is the association between H and D in all loci. Broad (h^2b) and narrow sense (h^2n) heritabilities were estimated using the formula proposed by Burton [30] and Warner [31] as: $h^2b= \delta^2g/\delta^2Ph$, and $h^2n= \delta^2a/\delta^2Ph$. The expected genetic advance (ΔG) from selection was calculated using the formulae proposed by Johnson et al. [32] using selection intensity (k=2.06), narrow sense heritability and F_2 generation variance as: $\Delta G=2.06*h^2n*\delta^2F_2$ and the predicted genetic advance, where the expected genetic gain upon selection was expressed as percentage of F_2 mean. Hence ΔG (%) = [ΔG*square root of F_2 mean *100], where a-additive genetic variance, δ^2g-genetic variance, δ^2Ph –phenotypic variance.

3. Results and Discussion

3.1. Generation Means and Heterosis

Analysis of variance showed significant (Table 1) difference among the means of generations for all characters studied except final percent severity of net blotch indicating high variability among generations and also a possibility to run generation mean analysis. For all net blotch and scald disease parameters studied, P_1 had the lowest mean scores followed by the F_1 hybrids while a large difference between the two parents mean scores for net blotch and scald diseases. BC_1 and BC_2 mean values varied according to the trait studied but it was in the direction of their respective recurrent parents for the studied characters (Table 1). Moreover, mean comparison between F_1 and F_2 for diseases had low and equivalent estimates for initial percent severity, final percent severity for both diseases while F_2 had low AUDPC estimate for net blotch than F_1 but less resistant to scald (Table 1; Figure 1).

Table 1. Analysis of variance, means and standard errors for net blotch and scald Parameters studied in six basic generations of HB42xSabini cross

Source of variation	DF	NBI	NBF	AUDPCNB	SCDI	SCDF	AUDPCSCD
Replication	2	11.2	49.1	13071.7	27.5	131.5	14847.5
Generation	5	90.1**	19.8^{ns}	12910.0*	250.7**	1237.1**	633133.3**
Error	10	6.4	13.5	2251.5	15.9	11.33	5479.6
CV (%)	-	75.7	42.1	34.9	67.4	10.24	15.1
Generation							
P_1		0.0±0.0	6.5± 3.6	77.5±47.2	0.0 ±0	11.9 ± 2.6	88.6±41.8
P_2		14.4±3.3	13.7±0.4	254.6±28.4	23.6 ±5.0	63.5±1.1	1281.6±42.4
F_1		1.2±0.6	7.6±4.1	163.3±65.7	1.1±1.1	27.0±5.3	275.6±90.5
F_2		0.7±0.4	9.0±2.4	114.7±20.9	3.2±1.0	38.8±3.7	499.8±26.6
BC_1		1.1±1.1	7.7±1.5	84.7±14.0	0.0± 0	11.2±2.9	80.5±22.8
BC_2		2.6±1.2	7.9±1.0	122.0±11.1	7.6 ± 3.0	45.0±2.2	721.7±33.4
MP heterosis (%)		-83.3**	-24.7**	-1.64**	-90.7**	-28.4**	-59.8**
BP heterosis (%)		-	16.9**	110.8**	-	126.9**	211**
% ID		41.6**	-18.4**	29.8**	-190	-43.7**	-81.4**

*, ** Significant at probability levels of0.05 and 0.01, respectively, CV (%)- coefficient of variation, DF-degree of freedom, NBI= Initial per cent net blotch severity, NBF= Final per cent net blotch severity, AUDPCNB:=Area under disease pressure curve as per cent of days for net blotch, SCDI= Initial per cent scald severity, SCDF= Final per cent scald severity, AUDPCSCD= Area under disease pressure curve as per cent of days for scald, ID=inbreeding depression, MP=Mid parent, BP=Better parent.

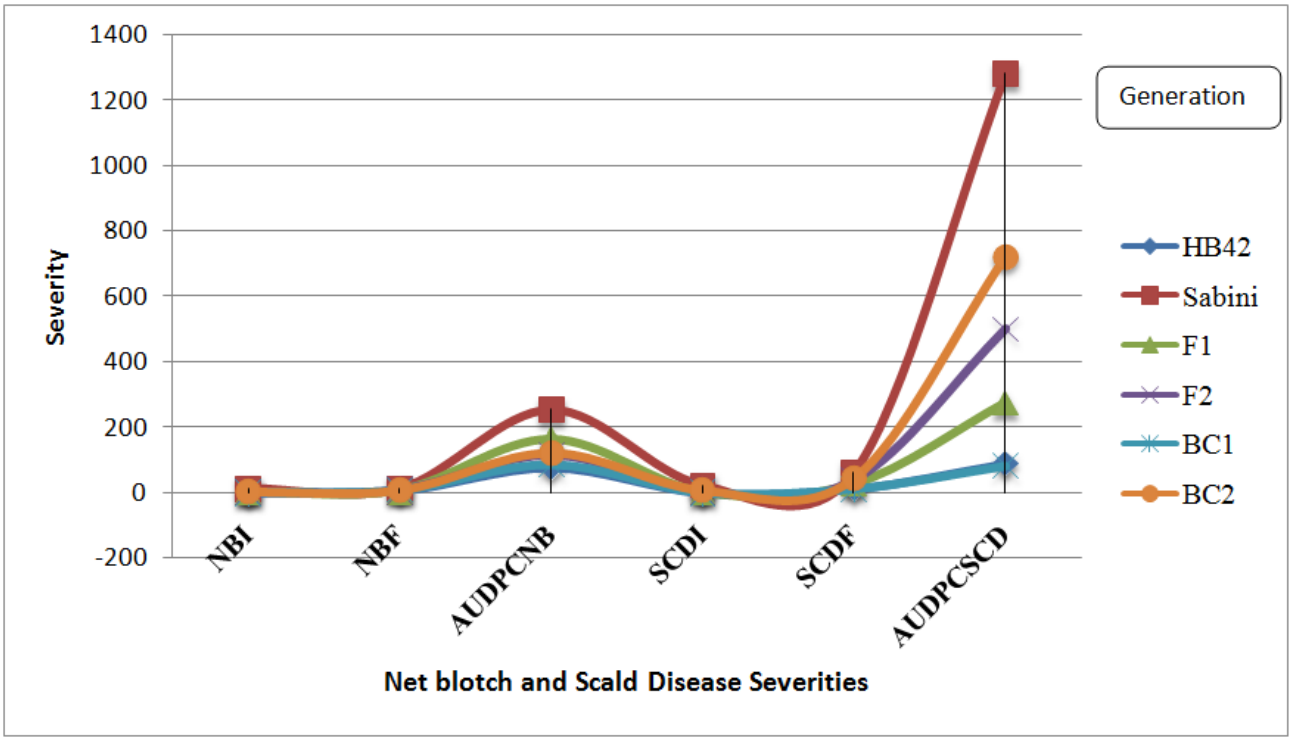

Figure 1. Disease severity of net blotch and scald diseases Score of six basic generationsof HB42 xSabini barley cross (NB: NBI= Initial per cent net blotch severity, AUDPCNB:=Area under disease pressure curve as per cent of days for net blotch, SCDI= Initial per cent scald severity, SCDF= Final per cent scald severity ,AUDPCSCD= Area under disease pressure curve as per cent of days for scald)

To illustrate graphically, the initial percent severity and final severity of net blotch were very low at the bottom followed by AUDPC as per cent of days for net blotch while the initial and final percent severity of scald and AUDPC as per cent of days of for scald was at the top region of the graph (Figure 1). And, it showed that the AUDPC rate for net blotch disease development was lower than the AUDPC rate of scald disease epidemics. It appears that 'HB42' and BC_1 showed low severity symptom or AUDPC followed by F_1 hybrid for both diseases may be due to the presence of double resistance genes found in 'HB42' parent so that it controlled the growth of subsequent pathogen inoculums in successive periods while the 'Sabini' and BC_2 were susceptible. Thus, it needs further investigation if there is any linkage in resistance alleles (linked markers) for both diseases would be useful to utilize the 'HB42' parent for breeding disease resistance.

The F_1 hybrid showed highly significant and negative heterosis for disease severities over mid parent in both diseases showing an increased resistance. The mid parent heterosis (MP) exhibited ranged from -1.64% to -90.7% in the negative direction in which is the desired direction contributing to resistance while better parent heterosis (BP) ranged from 16.9$ to 211% and positive (Table 1) and undesirable. This result was higher than another report [33] on barley which found that heterosis over mid-parent for six parameter model was from 10 to 20.4% and quite similar among the three crosses for heading date, but there was no heterosis over the high parent. While mid parent heterosis for grain yield for the same crosses was from 6 to 39 % for barley.

Maximum reduction in desirable direction due to increased homozygosity in the F_2 has resulted from inbreeding for barley in both diseases except for initial percent severity and AUDPC as per cent of days for net blotch (Table 1). Thus, a significant effect for both heterosis and inbreeding depression appears logical as the expression of heterosis in F_1 was followed by a considerable reduction in the F_2 performances. The reduction in values of non-additive genetic components may have been due to a reduction in intra- and inter-allelic non-additive gene actions including epistasis associated with heterozygosity [34].

3.2. Gene Effects for Net Blotch and Scald Diseases

Scaling tests and generation mean analysis was conducted for characters showing significant variation. Hence, the results of the scaling tests (A, B, C, and D) showed that out of four scaling tests, more than one scales were found significant for all of the characters (Table 2) indicating the inadequacy of additive-dominance model to interpret the gene effects involved. This shows genes are not independently contributing to produce a given amount of trait. However, the six parameter model appears more valid to explain the nature of gene action and interactions. Furthermore, the results of generation mean analysis provides estimates of the main and first-order interaction gene effects. Thus, the main gene effects and interactions gene effects estimate revealed that additive [d], dominance [h], additive x additive [i] and dominance x dominance [l] genic interactions were significant for final percent severity for scald and AUDPC as per cent of days for scald showing the involvement of both fixable and non-fixable gene effects with the magnitude of additive components ([d]+[i]) smaller than non-additive components ([h]+[l]) suggesting the need of delaying selection for these traits. The negative value of dominance

[h] for final percent severity of scald, AUDPC of scald and others in this study revealed that the alleles responsible for less value of the trait were dominant over the alleles controlling high value [35] Gene interactions, where they exist, can either reduce or enhance selection limits, but in general distort predictions of genetic improvement. It is necessary, therefore, to be able to detect gene interactions, estimate their effects, and predict their consequences in a breeding program [14].

All the additive [d] and dominance [h] main gene effects for parameters recorded for both net blotch and scald diseases were in the negative direction. Similarly, the [i] epistatic interaction gene effects for AUDPC as per cent of days for net blotch, final percent severity for scald, and AUDPC for scald were also negative indicating gene effects were involved in increasing resistance. For a character that showed non-significant Chi-square (χ), the additive dominance model is adequate. The results of the individual test scaling and joint scale tests indicated that the additive dominance model did not fit the data for all characters (Table 2) and the digenic epistatic model failed to explain variation in generation means for all characters would be due to the presence of higher-order interaction or linkage genes according to Kearsey and Pooni [14]. Highly significant additive [d] and significant additive x additive [i] gene effects was obtained for AUDPC as per cent of days for net blotch and initial percent severity for scald. Besides this, additive x dominance (j) epistatic gene action (partly fixable) was significant for initial percent severity for net blotch and final percent severity for scald.

Generation means analysis report on barley indicated the existence of non-allelic interaction for all studied traits in all five crosses studied and dominance × dominance epistatic component was greater in the magnitudes than additive × additive and additive × dominance in the most studied traits [36]. The negative sign of additive x dominance [j] interaction in most cases also suggests the dispersion of genes in the parents [37]. The significance of additive x dominance [j] epistasis effects of either positive or negative estimates indicates that dominance was towards the direction of increasing and decreasing the studied traits, respectively [38]. The negative or positive sign for [d] depends on which parent is chosen as P_1 [39]. Moreover, the opposite signs of dominance [h] and dominance x dominance [l] were assumed as an indicator of duplicate epistasis, while when both effects had the same sign, the effects were complementary [14,16]. Therefore, in this study, all the characters showed a duplicate type of epistasis (Table 2). and based on the direction of [h] and [l], for all the disease parameters indicates duplicate epistasis between dominant decreasers. This suggest that duplicate dominant epistatic types are the predominant non-allelic interaction gene effects in the barley population studied.

Reports showed that in the presence of a duplicate type of epistasis, genetic improvement through conventional selection at early generation will be unsuccessful so that selection should be delayed after several generations until a high level of gene fixation is attained [40]. Therefore, in such cases, to achieve more improvement, researcher suggest various methods, such as through recurrent selection in biparental progenies that would help in exploiting the duplicate type of non-allelic interaction and allow recombination and concentration of genes having cumulative effects in population as this method helps break up of undesirable linkage [41]. And Alake et al. [42] indicated in the presence of a significant amount of all types of gene actions, recurrent selection and reciprocal recurrent selection methods are recommended as efficient techniques for selecting a desirable cultivar. Furthermore, Gopikannan and Ganesh [43] emphasized the importance of inter mating of selected segregants or selective diallel mating system as useful to utilize both additive and non-additive gene effects and break any linkage in crop improvement.

Table 2. Estimates of scaling tests, joint scale test and gene effects for scald and net blotch diseases studied in six basic generations of 'HB42' x 'Sabini' cross

Parameter	NBI	AUDPCNB	SCDI	SCDF	AUDPCSCD
			Scale test		
A	1.0+2.3	-71.4±85.6	-1.1+1.1	-16.6± 8.2*	-203.1±109.6
B	-10.5+4.2**	-173.9±75.0*	-9.5+7.8	-0.5±6.9	-113.8 ±120.2
C	-13.9+3.9**	-200.0±165.1	-13.1±6.6*	25.7±18.5	77.9 ±218.3
D	-12.9+3.8**	-102.7±108.3	-17.3±6.6*	2.2±16.9	-370.6 ±133.1*
			Three parameter model		
m	2.7+4.1	158.2**	9.3±7.5	40.40	673.4**
[d]	-7.2+1.7**	84.3**	-12±2.5**	30.52	596.0**
[h]	-6.5+11.7	1.0**	-16.2±20.9	-13.57	-402.91**
			Estimates of gene effects (six parameter model)		
m	0.7+0.4	114.7±20.9**	3.2±1.0**	38.8±3.7**	499.8 ±26.6**
[d]	-1.4+1.7	-37.3±17.9*	-7.6±3.0*	-33.8±3.7**	-641.2±40.5**
[h]	-1.5+4.1	-48.1± 115.4	-8.2±7.6	-53.5±17.5**	-804.3±164.2**
[i]	4.5+3.7	-45.4± 90.8	2.5±7.1	-42.8±17**	-394.8±134**
[j]	5.8+2.4*	51.3 ± 32.9	4.2±3.9	-8.0±3.9*	-44.7±50.2
[l]	5.0+7.8	290.8± 180.0	8.0±13.6	59.9±23.6*	711.7±271.7*
χ^2	373.7**	8688701.2**	197.6**	23806.4**	560206198.2**
Type of epistasis	D	D	D	D	D

*, ** = 0.05 and 0.01, respectively; D = Duplicate; C = Complementary, mean (m), additive [d], dominance [h], additive x additive [i], additive x dominance [j] and dominance x dominance [l] interaction genic effects,χ^2 –Chi-square, NBI= Initial per cent net blotch severity, AUDPCNB:=Area under disease pressure curve as per cent of days for net blotch, SCDI= Initial per cent scald severity, SCDF= Final per cent scald severity ,AUDPCSCD= Area under disease pressure curve as per cent of days for scald.

3.3. Components of Genetic Variance

The adequacy of the additive dominance model may also be further tested by the type of allelic and non-allelic relationships from components of the variances by using the three-parameter model (E, D, and H). Hence, dominance variance (H) was greater than the additive variance (D) for all traits measured except the final per cent severity of net blotch where D was greater than H (Table 3). And the components of genetic variance estimates all the characters measured except final net blotch severity showed the preponderance of dominance gene effects. This result also confirmed the estimate of main gene effects and interaction gene effects for characters studied (Table 3) making selection difficult.

The average degree of dominance $[(H/D)^{1/2}]$ is used to determine the importance of dominance effects concerning the additive deviations of genes and is interpreted as partial, complete, or over-dominance [14]. If the ratio of $F/\sqrt{DxH}$ is equal to or near one confirms that the magnitude and sign of dominance for all the genes monitoring the character are equal, therefore the ratio $(H/D)^{½}$ is a good estimator of dominance. While if the ratio $F/\sqrt{DxH}$ is equal to zero or close to zero, the magnitude and the sign of gene effects controlling the character is not equal, and hence $(H/D)^{½}$ explains average dominance [44]. Accordingly, in this study the ratio of $(H/D)^{½}$ explained average dominance for the final percent severity for net blotch while all the rest traits showed overdominance (Table 3).

The direction of dominance (F) which indicates the relative frequency of dominant and recessive alleles in the parent was positive for all characters studied except for initial percent of net blotch, initial percent of scald, and AUDPC for scald. This indicated that more dominant alleles were found in parents than recessives for those traits that showed positive F values while excessive recessive alleles exhibited in the parents for initial percent of net blotch and initial percent of scald, AUDPC for scald (Table 3). The positive value of F- for some characters studied suggested that dominant alleles were more abundant than the recessive alleles in the parents and indicated the importance of dominance gene action in the inheritance of the traits under study [38]. Generally, the F value and average degree of dominance estimates indicated that dominance was unidirectional negative decreasing alleles at all loci for all characters studied. Negative F- estimate shows also dominant genes are in the low-performance parent. This all confirms the predominance of non-additive gene effects to additive gene effects indicated in generation mean analysis (Table 3).

3.4. Heritability for Net Blotch and Scald Diseases

The comparison between broad and narrow sense heritability is an indication of additive and non-additive gene effects. Heritability estimates in a narrow sense were very low to moderate for all studied traits (Table 3). Genetic advance as a percentage of F_2 mean (ΔG %) estimates of all characters was relatively higher. High narrow sense heritability associated with the highest genetic advance was obtained for thousand kernel weight and grain yield per plant.

The lowest narrow-sense heritability was due to higher influence of non-additive and environmental effects which makes selection difficult. However, high heritability coupled with high genetic advance as percent of mean was recorded for the characters like final per cent net blotch severity and final per cent scald severity (Table 3) indicating efficient selection. A similar result has been reported by Zerihun et al [45]. And moderate heritability values were also recorded for scald severity (0.491) and net blotch severity (0.581) [46]. Thus in this study high genetic advance as percent of F_2 mean was found coupled with high narrow-sense heritability and genetic variability for thousand kernel weight followed by a number of kernels per spike and days to heading indicating high genetic gain from the selection in these characters.

Table 3. Estimated components of variance and genetic parameters for net blotch and net blotch Disease studied in HB42xSabini barley cross

Parameter	NBI	NBF	AUDPCNB	SCDI	SCDF	AUDPCSCD
D	-3.5	31.2	2390.65	-19.9	43.2	-662.7
H	-26.5	-5.8	-34581.2	-30.0	-109.2	-49951.6
E	8.9	34.8	8754.0	20.2	47.4	14943.8
F	-0.8	4.2	217.4	-26.7	11.7	-1790.4
$(H/D)^{1/2}$	2.8	-0.43	-3.8	1.2	-2.5	8.7
$F/\sqrt{DxH}$	-0.08	0.31	-0.02	-1.1	-0.17	-0.31
h^2b	0.771	0.515	0.809	0.712	0.763	0.772
h^2n	0.090	0.435	0.052	0.284	0.216	0.010
ΔG	0.09	15.86	139.7	1.6	18.56	43.77
ΔG%	7.7	4749.2	149535.0	284.7	11559	97844.5

D=Estimates of additive variance, H= dominance variance, E=environmental variance, (H/D) = Average degree of dominance,- F direction of dominance, h^2b =broad sense heritability, h^2n =narrow sense heritability, ΔG= genetic advance, ΔG%=genetic advance as percentage of F_2 mean, NBI= Initial % net blotch severity, NBF= Final % net blotch severity, AUDPCNB:=Area under disease pressure curve as % of days for net blotch, SCDI= Initial % scald severity, SCDF= Final % scald severity ,AUDPCSCD= Area under disease pressure curve as % of days for scald.

4. Conclusion

The result showed that AUDPC rate of disease development for net blotch was lower than that of AUDPC of scald. And it appears that HB42 and BC_1 showed better resistance to both diseases which was followed by F_1 for both diseases indicating transfer of resistance gene from the resistant 'HB42' parent. The scale test and generation mean analysis showed greater influence of non-additive gene effects including epistasis or non-allelic genic interaction type of gene effects in the control of all parameters studied which was also confirmed by components of variances. Moreover, the joint scale test revealed the predominance of non-additive gene effects than additive (fixable) gene effects in all disease parameters studied. The joint scale tests and individual scaling tests confirmed the additive- dominance model did not fit to the data for all characters and also the digenic epistatic model failed to explain variation in generation means for all characters due to the presence of higher-order interaction or linkage. Generally, the comparison of various models and analysis revealed the predominance of non-additive genes gene action to additive gene effects in governing the inheritance of all parameters studied making selection difficult. And 'HB42' parent contained double resistance to scald and net blotch that requires further study for effective utilization in barley breeding for resistance.

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Statement of Competing Interests

The authors declare that they have no competing interest.

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