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Interaction of Gene Effect and Number of Loci on Heritability and Heterosis for Downy Mildew Resistance in Nigerian Elite Pearl Millet Lines

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Abstract: The inter-relationship among genetic parameter is of great importance as this tells on the phenotypic selection. Five pearl millet parental lines; BUDUMA, SOSAT-C88, LCICDMR36-4, 20B-2 and 25B-4 were obtained from Lake Chad Research Institute Maiduguri. From the five parents $6F_1$s, $6F_2$s $6BC_1P_1$s and $6BC_1P_2$s were generated using factorial mating design of North Carolina Design II, during November, 2004 and May, 2005 Off-season at Lake Chad Research Institute Maiduguri. The five parents, twenty-four crosses and one check (7042S) were evaluated using Randomized Complete Block Design (RCBD) with three replications at the downy mildew field nursery of Lake Chad Research Institute and the Experimental Farm of Federal University of Technology, Yola during 2005 and 2006 seasons. Results from this studies suggest that while dominance gene effects is important in expressing especially heterosis with 34.05% contribution, for resistance to downy mildew incidence and severity, it is higly limited by number of loci involved. Number of loci contributes 17.4% and 47.7% to total inheritance of resistance to downy mildew incidence and severity and about 8.39% and 7.33% to heterosis for resistance to downy mildew incidence and severity, respectively. Crosses involving SOSAT-C88, which had high estimates for number of loci (6, 7) controlling resistance for incidence expressed high narrow-sense (93.00%, 78.46%) heritability estimates and high significant negative higher-parent [(-79.78% and -94.79%), (-90.04% and -89.08%)] heterosis (considered advantageous) for downy mildew resistance.

Key Words: *Downy mildew · Dominance · Heritability · Heterosis*

INTRODUCTION

Breeding for diseases resistance of economic importance, more especially downy mildew contributed to increase productivity and stability of pearl millet grain, stover and forage yields. To increase the durability of resistance several gene pyramiding and deployment strategies have been proposed by Hash *et al*. [1] and Witcombe and Hash, [2]. The choice of selection and breeding procedure to be used for genetic improvement of any crop plant largely depends on the magnitude of genetic variability and the nature of gene action governing the nature of inheritance of the desirable traits. For practical and feasible breeding programmes, it is important for the breeder to be familiar with the genetic inheritance and potentials of local materials before embarking on population improvement. Therefore the inheritance of downy mildew resistance as well as yield and yield components characters are better understood when genetic parameters are evaluated. The inter-relationship among genetic parameter is of great importance as this tells on the phenotypic selection. This study was therefore designed to determine the heritability for downy mildew resistance, number of loci governing the nature of inheritance and the inter-relationship among the genetic parameters studied.

MATERIALS AND METHODS

Five Pearl Millet Parental Lines: BUDUMA, SOSAT-C88, LCICDMR36-4, 20B-2 and 25B-4 were obtained from Lake Chad Research Institute Maiduguri. Using factorial mating design of North Carolina Design II, $6F_1$s, $6F_2$s $6BC_1P_1$s and $6BC_1P_2$s were generated during November, 2004 to May, 2005 Off-season at Lake Chad Research Institute Maiduguri under irrigation. The five genotypes, twenty-four crosses and one check (7042S) were evaluated using randomized complete block design

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(RCBD) with three replications at the downy mildew field nursery of Lake Chad Research Institute and the Experimental Farm of Federal University of Technology, Yola during 2005 and 2006 seasons. The evaluations were done to estimate the inheritance of downy mildew resistance and the number of loci involved.

Downy Mildew incidence (number of diseased plants showing downy mildew symptoms expressed as a percentage of total number of plants in a plot were assessed at 30 and 60 days after sowing (DAS) by scoring for chlorosis of infected plants and at dough stage by scoring for green ears. Downy Mildew incidence was computed using the formula developed by James, [3]. Disease severity was scored on a 1-5 scale as described by Williams *et al*. [4].

Where: 1 = no disease; 2 = symptoms on aerial tillers only; 3 = symptoms on less than 50% basal tillers; 4 = symptoms on more than 50% basal tillers; and 5 = total destruction of stand or no production of normal head. Percent disease severity index was calculated using a formula described by Williams *et al.* [4].

Estimation of gene effects was based on a Six-Parameter Model using components of generation mean, a non-weighted analysis approach developed by Hayman [5] as used by St. Amandi and Wehner [6]. The genetic component calculation were based on an estimate of the net effects of all loci at which the parents differ in the measured traits assuming epistatic effect is not significant. Genetic parameters of appropriate model were tested within the limit of their standard errors where significance of the genetic effects is tested in a similar manner as done in the scaling tests.

Degree of dominance was calculated as the ratio of dominance variance to additive variance = $\sqrt{D/A}$.

Minimum number of loci involved in genetic control of the inheritance of the character were calculated using the least-square means and variances, a formula developed by Mather and Jinks [7] and as used by Yeye and Nwasike [8] and Wilson *et al.* [9].

$$n = \frac{0.25\,(0.75\text{-}h + h^2)D^2}{d^2\,F_2\text{-}d^2\,F_1}$$

Where n = minimum number of loci involved in the inheritance of the character being considered $h = F_1\text{-}P_1/P_2\text{-}P_1$ and F_1, P_1 and P_2 are means of F_1, P_1 and P_2, respectively

D^2 = square deviation of either parents from the mid-parent (P_2-P_1) value based on the following assumptions.

- no linkage between relevant loci,
- each loci contributes only positively or alleles effects of all loci are equal,
- the degree of dominance is the same and
- no espistatic interactions exist between loci.

Estimates of broad sense and narrow sense heritability were calculated for downy mildew incidence (DM) and downy mildew severity (DMS) in pearl millet by using the variance of the parent, F_1, F_2 and backcross generations to estimate phenotypic (V_P), environmental (V_E), total genetic (V_G), additive genetic (V_A) and dominance genetic variances (V_D). Where:

Broad sense heritability = $h^2_b = (V_A + V_D)/V_{F2}$, = $\delta^2F_2\text{-}\delta^2e/\delta^2F_2 = \delta^2g/\delta^2F_2$

where; $V_A + V_D$ represent the genetic variance of F_2 according to Allard, [10] and used by (Erin, [11].

Narrow sense heritability = $h^2_n = V_A/V_{F2} = 2\delta^2F_2 - (\delta^2B_1 + \delta^2B_2) / \delta^2F_2$ described Warner, [12] as used by Karen *et al.* [13].

Heterosis: was calculated from the mean of F1 over mean of mid-parent (Mp) and high- parent (Hp) expressed in percentage suggested by Liang, *et al.,* [14].

$$H(\%)Mp = F_1 - Mp/Mp \times 100$$

$$H(\%)Hp = F_1 - Hp/Hp \times 100$$

Where; F_1 = mean of F_1 generation, Mp = mean of mid-parent 1 and 2, Hp = mean of higher parent

RESULT AND DISCUSSION

Result from Table 1 indicated that, dominance×dominance genes effects were influential in controlling the resistance of downy mildew incidence and severity in the materials studied. This is as observed from the estimates dominance values which ranged from 0.91-2.99 with a mean value of 1.95 for incidence and from 1.78-8.88 with overall mean value of 5.33 for severity index. The results are in agreement with the earlier observations reported by Singh, *et al.* [15] and Singh, [16]. Results also indicated that gene effects were controlled at several loci as demonstrated by the average estimates of 10.15 loci for incidence and 5.14 for severity. This study further confirms the findings of Appadurai, *et al.* [17], Singh, [16] and Gill *et al*. [18, 19], who demonstrated that resistance for downy mildew resistance in pearl millet is controlled

Table 1: Estimates for Degree of Dominances and Number of Loci controlling resistance to downy mildew incidence and severity on pearl millet for combined locations (Maiduguri andYola) and years (2005 and 2006 seasons)

	DMI		DMS	
Crosses	Degree of dominance	Number of loci	Degree of dominance	Number of loci
20B-2×BUDUMA	1.90	4.95	1.78	8.96
25B-4×BUDUMA	2.33	3.11	6.16	1.01
20B-2×SOSAT-C88	2.81	6.13	2.12	1.18
25B-4×SOSAT-C88	2.99	7.94	2.77	9.99
20B-2×DMR36-4	0.91	0.32	2.90	0.28
25B-4×DMR36-4	2.72	19.97	8.83	3.88
Range	0.91-2.99	0.32-19.97	1.78-8.88	0.28-9.99
Mean	1.95	10.15	5.33	5.14

DMI = downy mildew incidence (%), DMS = downy mildew severity index (%)

Table 2: Estimates of heritabilities (%) and heterosis for resistance to downy mildew incidence and severity on pearl millet for combined locations (Maiduguri andYola) and years (2005 and 2006 seasons)

	DMI				DMS			
	Heritability (%)		Heterosis (%)		Heritability (%)		Heterosis (%)	
Crosses	BS	NS	Mp	Hp	BS	NS	Mp	Hp
20B-2×BUDUMA	39.25	84.65	-37.50	-49.97	-13.99	8.20	-40.88	-20.71
25B-4×BUDUMA	76.34	43.48	-63.74	-62.91	88.50	92.26	-76.81	-74.73
20B-2×SOSAT-C88	82.34	87.69	-94.86	-90.34	81.96	93.00	-99.78	-89.08
25B-4×SOSAT-C88	88.64	80.01	-92.98	-86.55	47.24	78.46	-94.79	-90.04
20B-2×DMR36-4	85.09	86.98	-52.19	-21.47	93.06	96.62	-72.35	-54.46
25B-4×DMR36-4	37.78	52.22	-51.10	-15.12	74.20	72.10	-85.63	-74.49
Range	37.78-88.64	43.48-87.69	-37.50-94.86	-15.12-90.34	-13.99-93.06	8.2-96.62	-40.88-99.78	-20.71-90.04
Mean	63.21	66.06	-66.18	-52.73	81.07	52.41	-70.33	-55.34

DMI = downy mildew incidence (%), DMS = downy mildew severity index (%), BS = Broadsense, NS = Narrowsense, MP = Midparent, HP = high-parent

by one or two dominant genes. Estimates of gene effects for downy mildew severity index (DMS) showed that additive×dominance gene effect was highly significant for crosses involving SOSAT-C88 as male or donor parent, which demonstrated that there were genes in the nucleus controlling downy mildew resistance. Dominance gene effects (d) for downy mildew severity index was highly significant for all crosses involving 25B-4 as female or recipient parent as evidenced in its crosses with either BUDUMA, SOSAT-C88 or DMR36-4. In the case where one gene with complete dominance was responsible for downy mildew resistance in pearl millet, mode of inheritance is simple and its utilization is straightforward. Results from this study confirms the findings of several authors [19, 20, 21], that dominance of downy mildew in pearl millet is controlled by two to several genes (oligogenically or polygenically controlled) considering the fact that an average of 10 and 5 loci were involved in the control of downy mildew incidence and severity, respectively. Thus quantitative inheritance studies of downy mildew resistance in pearl millet have been more successful in identifying parental materials with the ability to transmit high levels of resistance and be exploited for further breeding programme as suggested by Singh and Talukdar, [22]. Thus the need for marker assisted selection (MAS) is critical in breeding pearl millet hybrids with broad genetic base resistance to downy mildew infestation.

Results from Table 2 indicated that average broad-sense heritability for resistance to downy mildew incidence was 63.21% while narrow-sense heritability was 66.06%. Crosses involving SOSAT-C88 as a donor parent had both the highest broad and narrow-sense heritability for incidence. Broad-sense heritability estimates for resistance to downy mildew severity was 81.07% and narrow-sense heritability estimates were 52.41%. This

shows that genes conferring resistance to downy mildew is highly heritable, predictive, repeatable, stable and amenable to selection techniques which could be facilitated by the modern biotechnological tools, such as marker assisted selection techniques. According to Erin, [11], high heritability estimates can increase the prevalence of a particular trait under selection. In this study the high heritability estimates for downy mildew incidence and severity indicates resistance to recipients parents by donor parents was highly possible.

From Fig. 1, dominance gene effect contributes 8.1% to inheritance of resistance to downy mildew incidence while only 5.6% to inheritance of resistance to severity index. On the other hand, number of loci contributes 17.4% and 47.7% to total inheritance of resistance to downy mildew incidence and severity, respectively on the materials studied as indicated in Fig. 3 and 4. This however, is associated with steady decline as the number of loci increases.

The results in Table 2 and in Fig. 5 and 6, combined effects showed that, as the number of loci increases heritability (narrow-sense) of resistance to downy mildew incidence and severity decreases. This for example is clearly demonstrated on Fig. 5 and 6, where heritability (NS), for resistance to downy mildew and severity, decrease from 87.69 to 52.22 %, number of loci increased from 6.13 to 19.97 and from 93.0 to 8.2% when it increased from 1.18 to 8.96, respectively. This is further explained by the fact that, all the crosses involving SOSAT-C88, which had estimates of up-to 6 - 7 number of loci controlling resistance for incidence (Table 1) were those expressing high narrow-sense heritability (87.69%, 80.01%) as indicated on Table 2. Similar trend was also observed for inheritance of resistance to downy mildew severity in the cross involving 20B-2×SOSAT-C88 which narrow-sense heritability estimates was 93.0% when only one loci was involved (Table 1 and 2). It therefore suggests that, although inheritance of resistance to downy mildew appears quantitative involving several loci under the influence of dominance gene effects, there is a limit. This implies that, optimum number of quantitative trait loci (QTLs) (which needs to be investigated), should be determine for effective deployment strategies in gene pyramiding to increase the durability of resistance.

Estimates for mid-parent heterosis for resistance to downy mildew incidence as presented on Table 2 varied from -37.50 to -94.86% with a mean value of -66.18%. Higher-parent heterosis ranged from -15.12 to -90.34% with mean value of -52.73%. Mid-parent heterosis estimates for resistance to downy mildew severity ranged from -40.88 to -99.78% with a mean of -70.33 and higher-parent heterosis estimates were between -20.71 to -90.04% with a mean of -55.34%. Negatives values for downy mildew incidence and severity are considered advantageous as it suggest reduction in infestation. The higher the negative values the better the resistance. All crosses involving SOSAT-C88 as a donor parent had the highest negative mid-parent and high-parent heterosis for resistance to both downy mildew incidence and severity index, suggesting that there is possibility for exploiting hybrid vigor from these crosses.

Figure 7 showed that the expression of high negative high-parent heterosis for resistance to downy mildew was observed to be controlled by dominance gene effects contributing 34.05%. However, it was only influence 4.29% to expression of high-parent heterosis for resistance to downy mildew severity on the materials studied (Fig. 8). But as the number of loci increases, there was a decline in the expression of high negative heterosis for resistance to both downy mildew incidence and severity index contributing only about 8.39 and 7.33%, respectively as presented on Fig. 9 and 10. As represented on Fig. 11 and 12, high-parent heterosis for resistance to downy mildew and severity, decreased from 90.34 to 15.22% when number of loci increased from 6.13 to 19.97 and from 99.78 to 40.88% when it increased from 1.18 to 8.96, respectively. Further explanation can be illustrated by the fact that, all the crosses involving SOSAT-C88, which had estimates of up-to 6 or 7 number of loci controlling resistance for incidence (Table 2) were those expressing high significant negative higher-parent heterosis of -90.34% and -86.55% for resistance to downy incidence as indicated on Table 3. Similar trend was also observed for inheritance of resistance to downy mildew severity which high negative high-parent estimates were -89.08% and -90.04% (Tables 2 and 3). This still further suggests that number of loci is critical in expressing heterotic vigor for resistance to downy incidence and severity on pearl lines studied.

Results from this studies suggest that while dominance gene effects is important in expressing especially heterosis with 34.05% contribution, for resistance to downy mildew incidence and severity, it is higly limited by number of loci involved. This is evident in the fact, number of loci contributes 17.4% and 47.7% to total inheritance of resistance to downy mildew incidence and severity and about 8.39% and 7.33% of heterosis for resistance to downy mildew incidence and severity, respectively. Therefore optimum number of loci is an important area of investigation to understand the

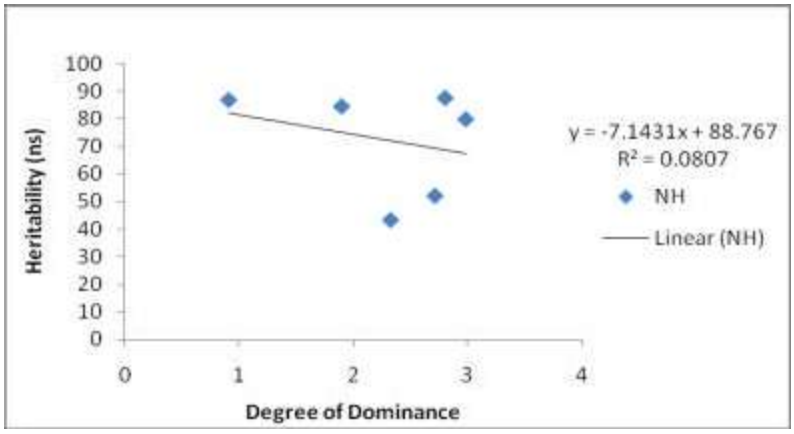

Fig.1: Influence of dominance on the heritability (ns) of resistance to downy mildew incidence on pearl millet

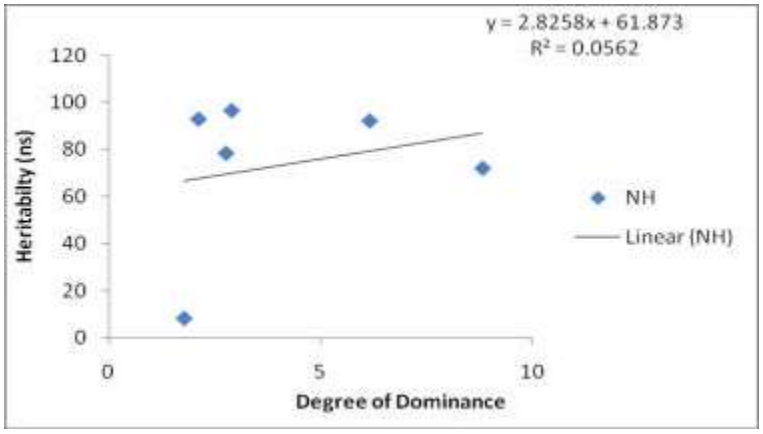

Fig. 2: Influence of dominance on heritability (ns) for resistance to downy mildew severity on pearl millet

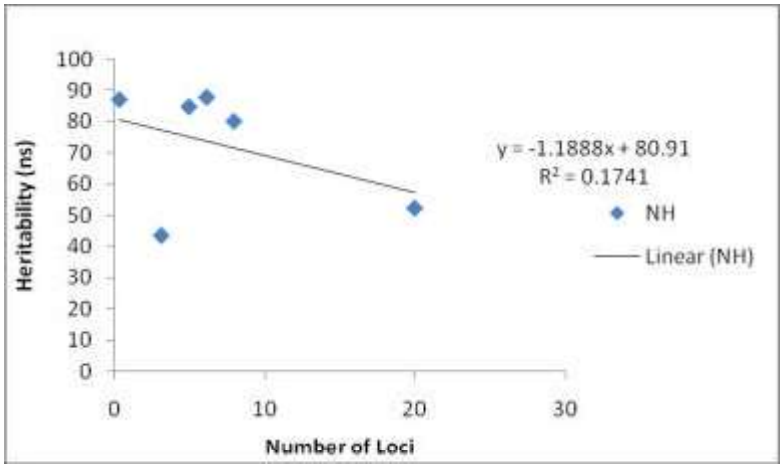

Fig. 3: Influence of number of loci on the heritability (ns) of resistance to downy mildew incidence on pearl millet

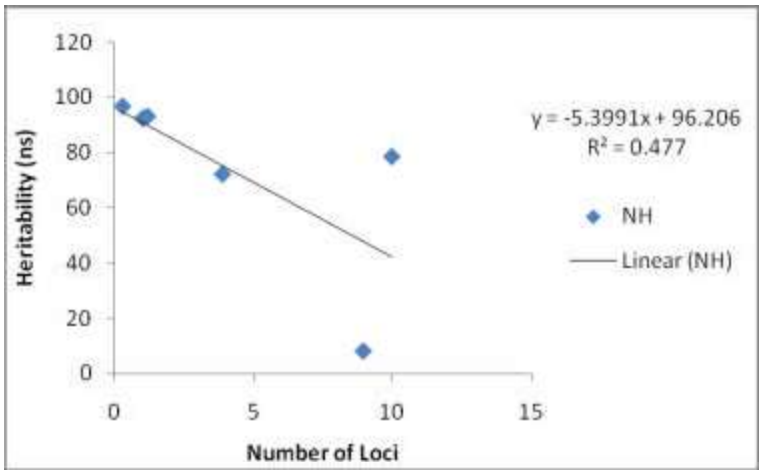

Fig. 4: Influence of number of loci on heritability (ns) for resistance to downy mildew severity on pearl millet

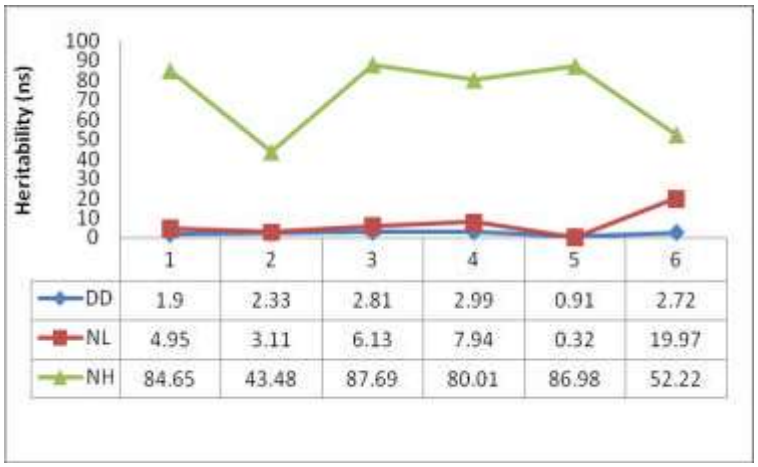

Fig. 5: Interaction of degree of dominance and number of loci on the heritability (ns) of resistance to downy mildew incidence on pearl millet

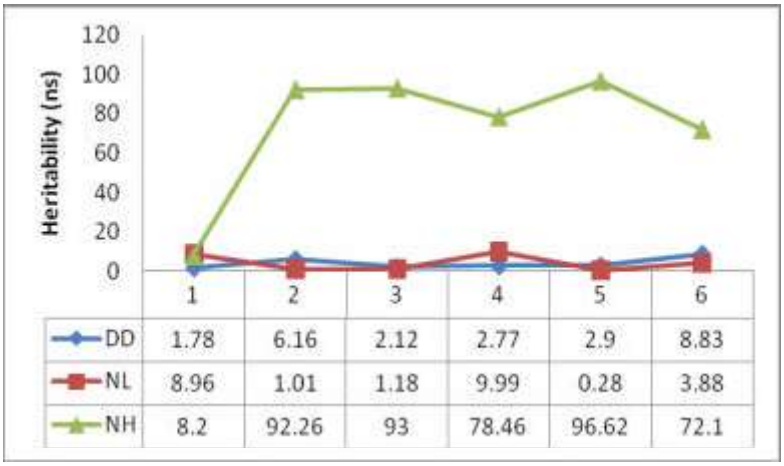

Fig. 6: Interaction of degree of dominance and number of loci on the heritability (ns) of resistance to downy mildew severity on pearl millet

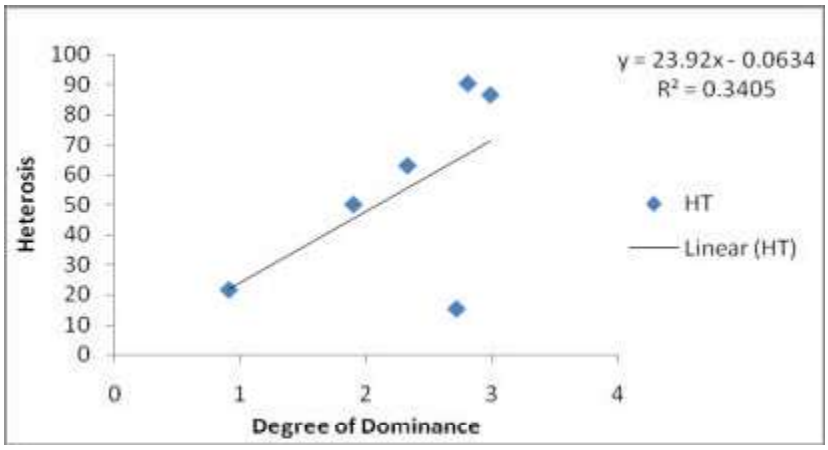

Fig. 7: Influence of dominance on the heterosis for resistance to downy mildew incidence on pearl millet

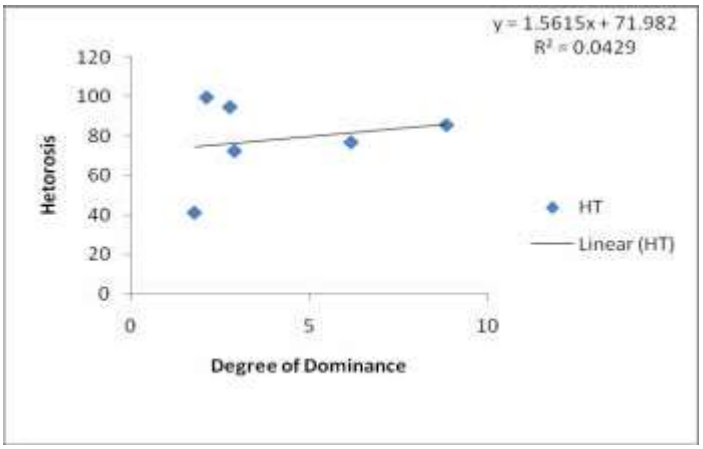

Fig. 8: Influence of dominance on heterosis for resistance to downy mildew severity on pearl millet

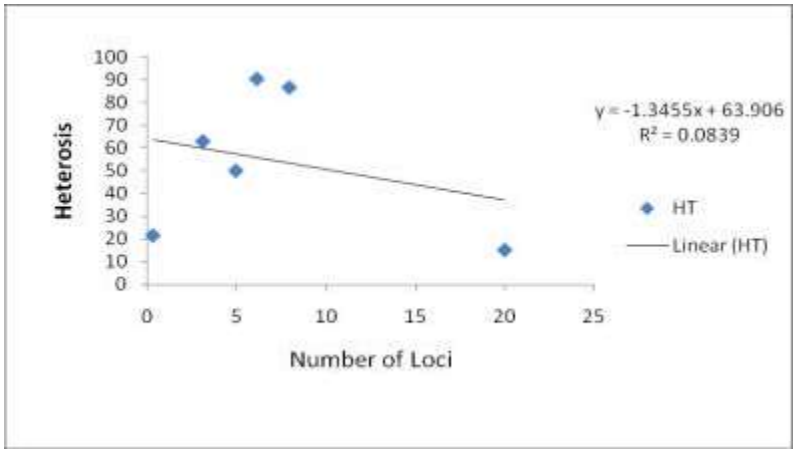

Fig. 9: Influence of number of loci on the heterosis for resistance to downy mildew incidence on pearl millet

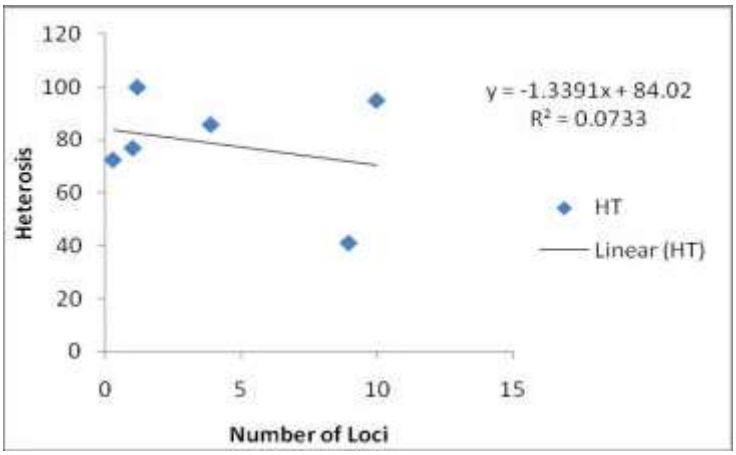

Fig. 10: Influence of number of loci on heterosis for resistance to downy mildew severity on pearl millet

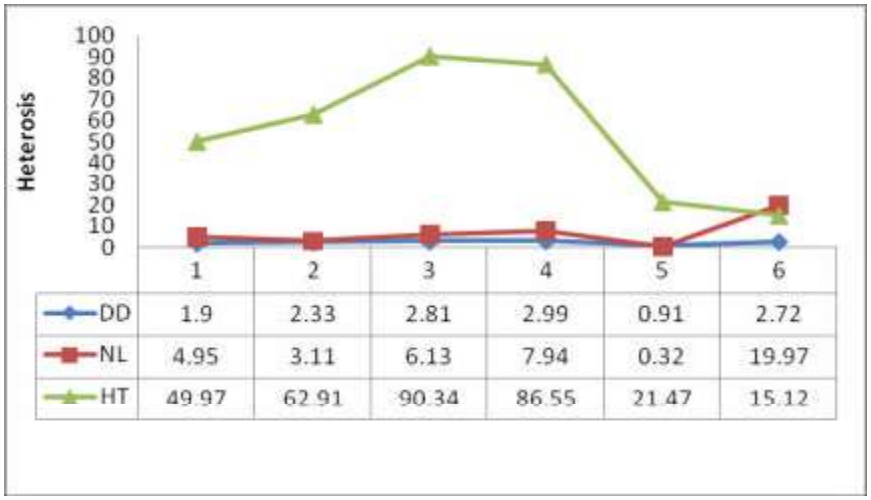

Fig. 11: Interaction of degree of dominance and number of loci on the heterosis for resistance to downy mildew incidence on pearl millet

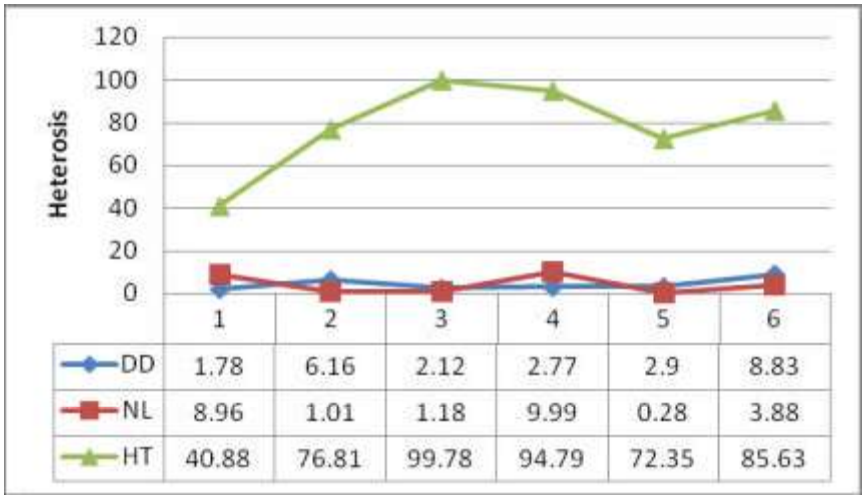

Fig.12: Interaction of Degree of dominance and Number of loci on heterosis for resistance to downy mildew severity on pearl millet

principles of gene pyramiding while deploying QTLs in elite pearl millet lines.

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