



Screening and genetic studies of certain maize genotype for resistance to southern corn leaf blight in India

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Received: 26 August 2016

Revised Accepted: 15 October 2016

ABSTRACT

Southern Corn Leaf Blight (SCLB) incited by *Helminthosporium maydis*, is a serious disease of Maize. The 53 maize inbreds were screened for SCLB during rainy season consequently for two year 2013 and 2014. A standard visual scale of 1 to 5 is being used by CIMMYT pathologist was considered appropriate for disease scale. Based on disease scale Percent Disease Index (PDI) and Area under Disease Progressive curve (AUDPC) considered as disease trait for screening of maize germplasm as well as estimation of Genetics of SCLB. On an average over four environments 25 resistance, 17 partial resistances, 3 susceptible lines were identified. In general there were less diseases in E₁ (Varanasi in Northern India) than E₂ (Nagenhalli in Southern India) though equal load of inoculums were applied in both the cases. Genetics of *Helminthosporium maydis* were studied in three crosses viz, CM- 212 (P₁) x V- 336 (P₂), CM- 212 (P₁) x V- 338 (P₂) and CM212 (P₁) x CM145 (P₂) using Scaling test, Joint Scaling Test and Jinks and Jones (1959) six parameters model. Diseases scores of P₁, P₂, F₁, F₂, B₁ and B₂ generations of the 3 crosses were used for genetics studies. All the effects (additive, dominance, additive x additive, additive x dominance and dominance x dominance) were important for expression of SCLB in all three crosses. Study of generation mean analysis revealed that all three crosses exhibited duplicate type of epistasis. Thus analysis revealed that SCLB resistance could be population non specific, although various types of gene effects were observed.

Key Words: Resistance, Screening, Southern corn leaf blight, *Zea mays L.*

INTRODUCTION

Maize (*Zea mays L.*) is the world leading and staple cereal crop belongs to tribe *maydaea* and grass family, Poaceae and also provides raw materials for the livestock and many agro-allied industries in the world (ali *et al* 2011, Randjelovic *et al* 2011). Almost 65 pathogens infect maize crop (Rahul and Singh 2002), out of which Southern Corn Leaf Blight (SCLB) is one of the most important diseases causing drastic reduction in maize yield which is caused by *Helminthosporium maydis* (Syn. *Bipolaris maydis*

(Nisik.) Shoemaker), (telomorph: *Cochliobolus heterostrophus*) and prevailing throughout the world where maize is grown under warm, humid conditions (White 1999). The blight spreads from the basal leaves to the developing ear and then flag leaf of maize plant (CIMMYT 1985). Under severe conditions depending upon the susceptibility of the variety, SCLB may cause significant grain yield losses from 9.7 to 11.7% (Singh and Srivastava, 2012). Losses have tended to be effectively controlled in high-intensity agricultural systems where it has been economical to invest in resistant germplasm. Resistance against SCLB has been described as quantitative, with a predominance of additive gene action as well as significant dominance effects present in some populations (Thompson and Bergquist 1984, Burnette and White 1985, Holly and Goodman 1989). Utilization of host resistance is the

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most cost effective and environmentally sound method of controlling of SCLB. Also studying the genetic basis of resistance is imperative in breeding for SCLB resistance. The aim of this study was to identify sources of resistance against SCLB for use in a hybrid breeding programme.

MATERIALS AND METHODS

Screening Studies: An experiment was conducted involving 53 inbreds lines during the year 2012-13 and 2013-14 to screen the maize inbreds lines to understand their reaction to Southern Corn Leaf Blight (SCLB). The 53 maize inbreds were obtained from DMR, New Delhi, VPKAS, Almora and Maize programme BHU, Varanasi. The inbreds were early to medium in maturity which fit well for development of early medium hybrid exhibited for rainfed condition. An experiment was conducted in Randomized Block Design (RBD) with two replications during 2013 and 2014 in two

environments at BHU, Varanasi and Mandya, Karnataka.

Inoculation: Plants were artificially inoculated 25-30 days after seedling emergence. Local sources of inoculum were used at both locations for artificial inoculation. At Naganahalli, the infected leaf tissues were collected, sterilized with $HgCl_2$, washed thrice with sterile water, cultured on potato dextrose agar medium, and multiplied on sorghum seeds. For this, the sorghum seeds were soaked overnight, transferred to sterilized conical flasks next day, and the pathogen inoculum was added. The flasks were shaken once in two days, and equal amounts of fresh sorghum seeds were mixed after one week. The infected sorghum with pathogen inoculums were ground to fine powder, and 1–1.5g of the ground inoculum was added to each leaf whorl, followed by a light spray of water to moisten the tissue and initiate infection. The inoculation procedure was repeated twice at 10-day interval to ensure no disease escapes.



A. Deployment of artificial inoculation in field.



B. Symptoms of *maydis* leaf blight

Figure 1 Inoculation procedure of Southern Corn Leaf Blight.

Disease severity: Each inbred line was scored for SCLB disease severity during the flowering stage at both locations. A standard visual scale of 1 to 5, in which score of ‘1’ indicating least severity and ‘5’ indicating highest severity was utilized. Since

intermediate ratings between two numbers (1.5, 2.5, 3.5 etc.) were also considered appropriate by the CIMMYT Pathologists, a modified rating scale was adopted in this study (Table 1).

Table 1 Modified 1-5 point scale used for screening maize genotypes for SCLB.

| Disease Score | Description | Disease reaction | Disease severity (%) |
|---------------|--------------------------------------------------------------------------------------------------------------|----------------------|----------------------|
| 1.0 | Very slight to slight infection, one or two to few scattered lesions on lower leaves | Highly resistant | <10 |
| 2.0 | Light infection, moderate number of lesions on lower leaves only | Resistant | 11-25 |
| 3.0 | Moderate infection, abundant lesions on lower leaves, few on middle leaves | Moderately resistant | 26-50 |
| 4.0 | Heavy infection, lesions abundant on lower and middle leaves, extending to upper leaves | Susceptible | 51-75 |
| 5.0 | Very heavy infection, lesions abundant on almost all leaves, plants prematurely dry or killed by the disease | Highly susceptible | 75-100 |

Percent Disease Index (PDI): Percent Disease Index (PDI) was worked out by disease score using formula given by (Wheeler 1969).

$$PDI = \frac{\text{Sum of all ratings}}{\text{Total no. of observations} \times \text{Maximum rating scale}} \times 100$$

Area under Disease Progressive Curve (AUDPC): Disease progress curve, which consist of proportions of diseased plants (i.e. disease severity %) recorded at 15 days interval starting from the onset of disease 3 times throughout the growing period. To ensure consistent disease evaluation in the field, a disease progress curve was made. This curve was developed from percent disease severity reading. The Area Under Disease Progress Curve (AUDPC) is used to quantify repressing of the beginning of the epidemic and the time until the blight reached peak. Leaf blight for whole plant was converted to AUDPC to compare relative level of resistance and highly susceptible varieties. The derived disease parameter, AUDPC was calculated according to the equation of

(Campbell and Madden, 1991) using the following formula:

$$AUDPC = \sum_{i=1}^{n-1} \{ [(X_{i+1} + X_i) / 2] * (t_{i+1} - t_i) \}$$

Where:

- ✓ X_i is the disease index expressed as a proportion at the i^{th} observation.
- ✓ t_i is the time (days after planting) at the i^{th} observations.
- ✓ And n is the total number of observations

Inbreds were classified in different categories (Resistant 600-950, Partial resistant 951-1350, partial susceptible 1351-1650, Susceptible 1651-2000, highly susceptible 2001-2350) based on AUDPC value for both the environments.

Genetics of Southern Corn Leaf Blight: For study of gene action six generations (P_1 , P_2 , F_1 , F_2 , B_1 and B_2) for each of the following three crosses were evaluated in this study: CM- 212 (P_1) x V- 336 (P_2), CM- 212 (P_1) X V- 338 (P_2) and CM212 (P_1) x CM145 (P_2). Initial crosses were made at Agricultural

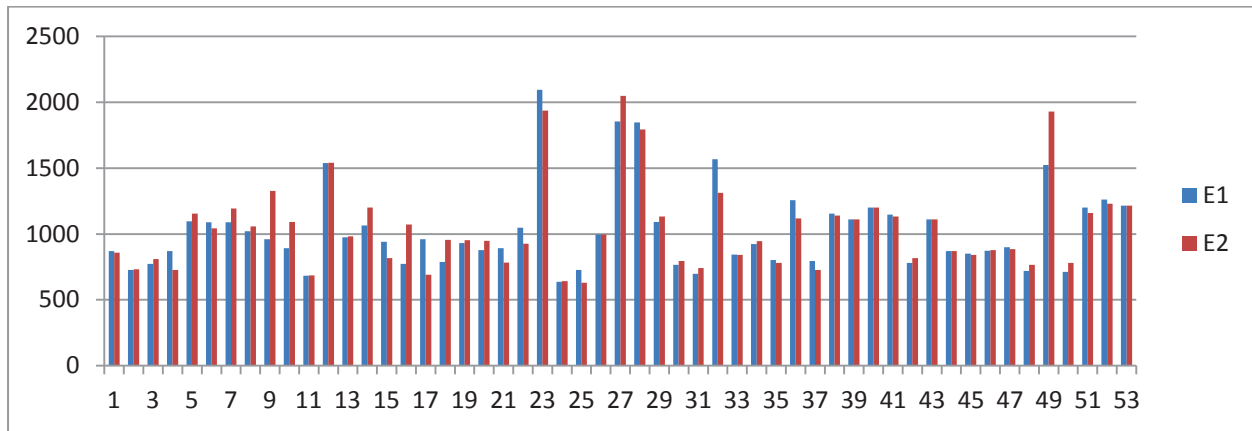


Figure 2 Comparison of 53 maize inbreds in two environments on the basis of AUDPC value.

Table 2 Combined analysis of variance for PDI, AUDPC and Yield evaluated in two different environments.

| Source of variation | Degree of freedom(DF) | Mean sum of square PDI | AUDPC | YIELD |
|---------------------------------|-----------------------|------------------------|------------|----------|
| Replication | 1 | 27.97** | 8983.019* | 3.78* |
| Environment | 1 | 14.27* | 3694.458** | 6.70** |
| Treatment | 52 | 551.916** | 384084.5* | 764.31** |
| Environment x Treatment (E x T) | 52 | 29.45156** | 14876.31** | 15.39** |
| Pooled error | 105 | 3.766981 | 2058.376 | 0.933 |
| CV% | | 4.795067 | 4.38 | 2.30 |

research station, Mandya, Karnataka during *Rabi* 2012. All that generations required for the study were developed during *Kharif* 2013 at same location. Six generations (P_1 , P_2 , F_1 , F_2 , B_1 and B_2) from the Cross of CM212 x V-336, CM212 x V-338 and CM212 x CM145 were sown and evaluated in artificial epiphytotic condition during *Kharif* 2014 at Agricultural research farm of Banaras Hindu University. A scaling test was performed to test the adequacy of additive-dominance model. The four scaling tests, as given by Hayman and Mather (1955), were adapted as follows: $A = 2B_1 - P_1 - F_1$, $B = 2B_2 - P_2 - F_1$, $C = 4F_2 - 2F_1 - P_1 - P_2$ and $D = 2F_2 - B_1 - B_2$. Wherever the additive dominance model was inadequate, the Six Parameter Model of Hayman (1958) was used for estimation of various genetic components, where m = Mean, d = Additive effect, h = Dominance effect, i = Additive x Additive type of gene interaction, j =

Additive x Dominance type of gene interaction, and l = Dominance x Dominance type of gene interaction.

The genetic parameters were also estimated using joint scaling test (Cavalli 1952). Instead of testing the relationship individually, the joint scaling test provides the full set of scaling tests into one. Since the six generation mean to which the model was fitted was not known in equal precision, the generation means and their expectations were weighted using the reciprocals of variance of means as the weight. Various generation means were predicted based on the parameters estimated and the test for goodness-of-fit was conducted using chi-square statistic. If the P value for the calculated chi-square (c^2) was >0.05 , the models were considered adequate. Different combinations of reduced number of parameters were tested in order to identify the best fitting model.

Table 3 Comparison of response of different maize genotypes towards disease reaction in two different environments.

| GENOTYPE | AUDPC E1 | DR | AUDPC E2 | DR | YIELD |
|--------------------|----------|----|----------|----|-------|
| 219-J | 870 | R | 858.75 | R | 29.65 |
| V-341 | 727.5 | R | 731.25 | R | 35.75 |
| HKI-586 | 772.5 | R | 810 | R | 39.2 |
| HKI-323 | 870 | R | 727.5 | R | 66.4 |
| CM-141 | 1095 | PR | 1155 | PR | 42.3 |
| CML140 | 1087.5 | PR | 1042.5 | PR | 40.85 |
| V335 | 1087.5 | PR | 1192.5 | PR | 34.3 |
| HUZM-97-1-2 | 1020 | PR | 1057.5 | PR | 45.7 |
| HUZM-80-1 | 960 | PR | 1327.5 | PR | 45.15 |
| HUZM-185 | 892.5 | R | 1091.25 | PR | 41.75 |
| HKI536 | 682.5 | R | 686.25 | R | 53.85 |
| V-351 | 1537.5 | PR | 1541.25 | PS | 34.65 |
| HKI-287 | 975 | PR | 982.5 | PR | 37.3 |
| HUZM-478 | 1065 | PR | 1200 | PR | 41.2 |
| HKI-193 | 941.25 | R | 817.5 | R | 28.05 |
| HKI-164-4-(1-3)-2 | 772.5 | R | 1072.5 | PR | 65.65 |
| HUZM-53 | 960 | R | 690 | R | 37.2 |
| V-386 | 787.5 | R | 956.25 | PR | 41.5 |
| HKI-1352-5-8-9 | 930 | R | 952.5 | PR | 36.2 |
| HUZM-121 | 877.5 | R | 948.75 | R | 31.75 |
| HKI-1105 | 892.5 | R | 783.75 | R | 29.9 |
| HUZM-36 | 1046.25 | PR | 926.25 | R | 24.5 |
| CM-212 (Sus Check) | 2092.5 | HS | 1935 | S | 18 |
| CM-145 (Res Check) | 637.5 | R | 641.25 | R | 70.5 |
| V-336 | 727.5 | R | 630 | R | 53.65 |
| V-338 | 997.5 | PR | 997.5 | PR | 48.7 |
| V-25 | 1852.5 | S | 2047.5 | HS | 20.55 |
| HKI-162 | 1845 | S | 1792.5 | S | 22.15 |
| HUZM-88 | 1091.25 | PS | 1132.5 | PR | 43.75 |
| HUZM-211-1 | 765 | R | 795 | R | 66.2 |
| HKI-PC-8 | 697.5 | R | 742.5 | R | 77.1 |
| V-388 | 1567.5 | PS | 1312.5 | PR | 34.2 |
| HUZM-47 | 843.75 | R | 840 | R | 46.55 |
| HUZM-509 | 922.5 | R | 945 | R | 40.9 |
| HUZM-60 | 802.5 | R | 780 | R | 46.35 |
| HUZM-69 | 1256.25 | PR | 1117.5 | PR | 26.55 |
| HUZM-81-1 | 795 | R | 727.5 | R | 80.95 |
| HUZM-356 | 1155 | PR | 1140 | PR | 65.45 |
| HUZM-457 | 1110 | PR | 1110 | PR | 44.3 |
| HKI-335 | 1200 | PR | 1200 | PR | 46.25 |
| CML-451 | 1147.5 | PR | 1132.5 | PR | 41.75 |
| CML-161 | 780 | R | 817.5 | R | 44.5 |
| HKI-209 | 1110 | PR | 1110 | PR | 25.35 |
| CML-172 | 870 | R | 870 | R | 42.35 |
| CML-150 | 851.25 | R | 840 | R | 42.7 |
| CML-395 | 873.75 | R | 877.5 | R | 37.4 |
| CML-152 | 900 | R | 885 | R | 35.9 |
| CML-192 | 720 | R | 765 | R | 25.4 |
| CM-126 | 1522.5 | S | 1927.5 | S | 26.4 |
| V-348 | 712.5 | R | 780 | R | 43 |
| V-342 | 1200 | PR | 1158.75 | PR | 44.2 |
| V-346 | 1260 | PR | 1230 | PR | 46.8 |
| V-273 | 1215 | PR | 1215 | PR | 47.7 |

Table 4 Mean performance of six generations of three crosses for disease score of SCLB.

| Crosses | Character | Generations | | | | | |
|-----------------|---------------|-------------|---------|---------|---------|---------|---------|
| | | P1 | P2 | F1 | F2 | B1 | B2 |
| CM 212 x V-336 | Disease score | 3.89 | 1.7 | 1.6 | 2.0 | 2.4 | 3.2 |
| | PDI | 75.84 | 20.33 | 39.33 | 34.83 | 54.16 | 63.83 |
| | AUDPC | 2063.75 | 610.00 | 1003.75 | 950.00 | 1365.00 | 1697.50 |
| CM 212 x V-338 | Disease score | 3.89 | 3.08 | 2.95 | 3.78 | 2.84 | 3.62 |
| | PDI | 77.83 | 61.67 | 58.33 | 76.16 | 56.83 | 72.00 |
| | AUDPC | 2078.75 | 1621.25 | 1445.00 | 1975.00 | 1465.25 | 1935.00 |
| CM-212 x CM-145 | Disease score | 3.900 | 1.000 | 1.967 | 1.877 | 2.760 | 3.083 |
| | PDI | 75.83 | 34.00 | 32.33 | 40.67 | 58.34 | 68.34 |
| | AUDPC | 2063.75 | 898.75 | 878.75 | 1097.50 | 1493.75 | 1741.25 |

Table 5 Showing estimates of scaling test (A, B, C, and D) based on six generations of disease score of SCLB in maize.

| Crosses | Character | Scaling test | | | | Joint scaling test | χ^2 value |
|------------------|---------------|--------------|------------|-----------|----------|--------------------|----------------|
| | | A | B | C | D | | |
| CM- 212 X V- 336 | Disease score | 0.625** | -3.267** | 0.0558** | -1.600** | ** | 142.36 |
| | PDI | 6.83 | -68.00** | 0.35.50** | -48.33** | ** | 375.40 |
| | AUDPC | 337.50 | -1781.25** | 881.25** | -1162** | ** | 1095.89 |
| CM- 212 X V- 338 | Disease score | 1.167** | -1.158** | -2.242** | 1.125** | ** | 145.51 |
| | PDI | 22.50** | -24.40** | -45.50** | 23.50** | ** | 156.64 |
| | AUDPC | 611.25** | -803.75** | -1310** | 558375** | ** | 138.53 |
| CM 212 xCM 145 | Disease score | 0.347 | -3.200** | 1.327** | -2.090** | ** | 402.85 |
| | PDI | -8.50 | -70.33** | 11.83** | -45.33** | ** | 406.42 |
| | AUDPC | -45.00 | -1750** | 330.00** | -1040** | ** | 265.47 |

Table 6 estimates of gene effect obtained from six parameter models of Jinks and Jones (1985) in three crosses based on disease score for SCLB in maize.

| Crosses | [m] | [d] | [h] | [i] | [j] | [l] | |
|------------------|---------------|-----------|-----------|------------|------------|------------|------------|
| CM 212 x V- 336 | Disease score | 2.067** | -0.850** | 2.021** | 3.200** | -3.892** | -5.842** |
| | PDI | 34.83** | -9.68** | 87.91** | 96.67** | -74.83** | -157.83** |
| | AUDPC | 950.00** | -332.50** | 1998.87** | 2325.00** | -2118.75** | -3768.75** |
| CM- 212 x V- 338 | Disease score | 3.78** | -0.758** | -2.779** | -2.250** | -2.325** | 2.258** |
| | PDI | 76.17** | -15.17** | -58.41** | -47.00** | -46.50** | 45.50** |
| | AUDPC | 1975.00** | -478.75** | -1552.50** | -1117.50** | -1415.00** | 925.00** |
| CM 212 x V 145 | Disease score | 1.877** | -0.323** | 3.697** | 4.180** | -3.547** | -7.033** |
| | PDI | 40.66** | -10.00** | 68.08** | 90.66** | -61.83** | -169.50** |
| | AUDPC | 1097.50** | -247.50** | 1477.50** | 2080.00** | -1660.00** | -3830.00** |

RESULTS

The two separate experiments were conducted First (A) Screening 53 maize inbred lines in two environments during two years and second (B) Genetics of SCLB in three crosses.

Screening of maize inbred for SCLB resistance:

High disease pressure was achieved through artificial inoculations, as was evident from the disease severity of susceptible checks used at the test locations.

During the course of the study two fold screening has been performed to get a genotype which is resistant over the environment in a year. In first experiment genotypes were screened in Varanasi in epiphytic condition during 2013. Secondly same set of genotypes were screened for resistance in the hot spot and National Screen House Test Center at Nagenahalli, University of Agricultural Science

Bangalore during 2013. In both center fifty-three genotypes were planted in two replications with plot size randomized block design.

The combined/pooled analysis of variance showed that mean squares for yield were significantly different for environment ($P < 0.01$), treatment ($P < 0.01$) and $E \times T$ ($P < 0.01$), whereas treatment (T) and $E \times T$ mean squares was significant for PDI ($P < 0.01$), AUDPC ($P < 0.01$) and Yield ($P < 0.01$). However, significant differences were observed for environments in PDI ($P < 0.05$), AUDPC ($P < 0.01$) and Yield ($P < 0.01$) (Table 2). The lowest PDI were observed for V-336 (23.75) while highest PDI was observed for CM-212 (71.5) and with the combined mean of 40.47. The mean AUDPC value across the 53 maize inbreds was 1034.43 with the lowest value for CM-145 (646.87) and highest value for CM-126 (1728.75) while mean value for yield was 42.10 with the highest value for HUZM-81-1 (80.82) and lowest value for V-25 (20.32).

There was significant variation among the varieties for AUDPC values. The severity of the disease (AUDPC), however, varied from location to location dependent on the different in the environmental conditions, appearance of disease and other factors. Inbreds were allotted in different categories (Resistant 600-950, Partial resistant 951-1350, partial susceptible 1351-1650, Susceptible 1651-2000, highly susceptible 2001-2350) based on AUDPC value for both the environments (Table 3 and Figure 2).

AUDPC value for E_1 was found to vary from 637.5 in CM-145 to highest score 2092.5 in CM-212. Out of 53 maize inbred lines, 28 inbreds namely 219-6, V-341, HKI-586, HKI-323, HUZM-185, HKI-536, HKI-193, HKI-1352-5-8-9, HUZM-53, V-386, HKI-164-4-(1-3)-2, HUZM-121, HKI-1105, CM-145, V-336, HUZM-211-1, HKI-PC-8, HUZM-47, HUZM-509, HUZM-60, HUZM-81-1, CML-161, CML-172, CML-150, CML-395, CML-152, CML-192 and V-348 exhibited AUDPC value between 600 to 950 in

E_1 and therefore grouped as resistant inbreds. While in E_2 25 inbred lines namely 219-6, V-341, HKI-586, HKI-323, HKI-536, HKI-193, HUZM-53, HUZM-121, HKI-1105, CM-145, V-336, HUZM-211-1, HKI-PC-8, HUZM-47, HUZM-509, HUZM-60, HUZM-81-1, HUZM-36, CML-161, CML-172, CML-150, CML-395, CML-152, CML-192 and V-348 while disease reaction of HUZM-185, HKI-164-4-(1-3)-2, V-386 and HKI-1352-5-8-9 changed from resistant to partial resistant as compared to E_1 and HUZM-185 behaved as resistant in E_2 as compared to E_1 where it was partial resistant.

Another 18 inbreds namely CM-141, CML-140, V-335, HUZM-97-1-2, HUZM-80-1, HKI-287, HUZM-478, HUZM-36, V-338, V-351, HUZM-69, HUZM-356, HUZM-457, HKI-335, CML-451, HKI-209, V-342, V-346 and V-273 exhibited AUDPC value between 951 to 1350 in E_1 and therefore grouped into partial resistant and E_2 17 maize inbreds namely CM-141, CML-140, V-335, HUZM-97-1-2, HUZM-80-1, HKI-287, HUZM-478, V-338, HUZM-69, HUZM-356, HUZM-457, HKI-335, CML-451, HKI-209, V-342, V-346 and V-273 grouped into partial resistant while V-351 partial susceptible in E_2 as compared to E_1 where it was partial resistant HUZM-356 was resistant in E_2 as compared to E_1 where it was partial resistant. HUZM-88 and V-338 were partial susceptible in E_1 while in E_2 V-351 was partial susceptible with the AUDPC value between 1351 to 1650. 3 inbreds V-25, HKI-162 and CM-126 were susceptible in E_1 and HKI-162, CM-212 and CM-126 were susceptible in E_2 with the AUDPC value ranged from 1651 to 2000. CM-212 was highly susceptible in E_1 and in E_2 V-25 was highly susceptible as they exhibited AUDPC value between 2001 to 2350 (Table 3).

Genetics of Southern Corn Leaf Blight: The examinations of mean values for disease score, PDI and AUDPC of six generations of three crosses are presented in Table 4. The F_1 of all three crosses for disease score showed reduced than the parents. The F_2 in cross I and cross II showed exceeded expression

than F_1 for disease score indicating heterobeltosis, but cross III showed reduced expression for disease score. The mean values of the B_1 exceeded the mean value of their respective P_2 , F_1 and F_2 in cross I and III, whereas value of B_1 is reduced mean value of their P_2 , F_1 and F_2 . The mean value of B_2 is higher than the mean values of their respective P_2 , F_1 , F_2 , B_1 in all three crosses for disease score. For PDI and AUDPC F_1 of cross II and Cross III showed reduced than the parents but in cross I F_1 was higher than P_1 but Lower than P_2 . The F_2 in cross II and cross III showed exceeded expression than F_1 for disease score indicating heterobeltosis, but cross I showed reduced expression for PDI as well as AUDPC. B_1 exceeded the mean value of their respective P_2 , F_1 and F_2 in cross I and III for PDI and AUDPC whereas value of B_1 is reduced than the mean value of their P_2 , F_1 and F_2 in cross II for PDI. B_2 also exceeded the mean value of their respective P_2 , F_1 , F_2 and F_2 in cross I and III for PDI and AUDPC whereas value of B_2 is lower than F_2 for PDI and AUDPC (Table 4).

The parameter estimates for all the four scaling tests were highly significant indicating the inadequacy of additive/dominance model and the importance of gene interaction. Scaling test and joint scaling test indicated the presence of epistasis in all the three crosses studied (Table 5). All the crosses also showed significant estimates of additive, dominance, additive x additive, additive x dominance and dominance x dominance component. Highest magnitude of additive component was obtained for cross 1(CM 212 x V 336) followed by 2(CM 212 x V 338) and cross 3(CM212 x CM145). All crosses were exhibited duplicate epistasis (Table 6).

DISCUSSION

Screening of maize inbred for SCLB resistance:

Disease resistance is the major aim of the breeding community and to provide such genotype having a resistant genetic background is the primary goal of plant breeding. The resistant material will drastically reduce the inputs of the farming community and will ultimately increase the grain yield. The variability

observed in this experiment for all the traits showed the maize possesses remarkable genetic diversity for almost all traits of economic importance including disease resistant. It is therefore, important to collect information about the extent of disease severity, genetics of disease, variability, for different characters.

Environment (E), treatment (T) and Environment x Treatment (E x T) were significant for GY. This suggests differences in yield performance of different inbreds in both environments. The T and E x T were significant for PDI, AUDPC and yield. Ibikunle *et al* (2009) and Ahmad *et al* (2011) noted similar observations earlier. Maize inbred lines such as 219-6, V-341, HKI-586, HKI-323, HKI-536, HKI-193, HUZM-53, HUZM-121, HKI-1105, CM-145, V-336, HUZM-211-1, HKI-PC-8, HUZM-47, HUZM-509, HUZM-60, HUZM-81-1, HUZM-36, CML-161, CML-172, CML-150, CML-395, CML-152, CML-192 exhibited low AUDPC value and showed resistant type of disease reaction in both the environment and these results were similar with (Rai *et al* 2009 and Chandrashekara *et al* 2014), who observed that low AUDPC values showed disease resistance. This is also in accordance with the work of earlier reports (Mallikarjuna 1998) who has reported that AUDPC values took care of initial and terminal severity and also rate of infection.

The resistant maize inbred lines reported here appear adopted and have potential to be used as source material in the breeding of disease resistant, high yielding and stable inbred in the area of Nagenahalli, Mandya, Karanataka, BHU, Varanasi and similar environment in India. The severity of the disease was found more at Nagenahalli, Karnataka, due to the moderate temperate were prevailing which favours Southern Corn Leaf Blight development during the crop season, whereas, the disease severity was found less in BHU, Varanasi as compared to Nagenahalli, Karnataka which known to be the hot spot for disease. From the current evaluation it appeared that there are potential losses incurred by Southern Corn

Leaf Blight on maize yield. Therefore, it is justifiable for estimation of a resistance breeding programme to develop varieties with increase resistant plant, which will be most effective and affordable way to overcome the problem of leaf blight disease of maize in diverse Agro-ecological environment of Nagenahalli, Mandya, Karnataka, BHU, Varanasi and similar environment in India. SCLB development was influenced by humidity and susceptibility of maize variety. The appearances of disease were varied from location to location that might be due to the influence of environment, but the increment of the AUDPC values was consistent in resistant and susceptible inbreds. In the present investigation, the GY of resistant and moderately resistant inbreds were in general high in comparison to susceptible inbred.

Genetics of Southern Corn Leaf Blight: Generation mean analysis clearly revealed that the nature of inheritance could be population non specific. Quantitative resistance is expressed independently of the physical environment and has never succumbed to MLB pathotypes in the field. Lim and Hooker (1976) reported that the predominant mode of inheritance for SLB resistance in the set of crosses was additive based on diallel analysis. Burnette and White (1985) studied the inheritance of resistance to race o in crosses of nine diverse, resistant inbred lines with three susceptible inbreds.

Generation means analysis for the 12 families from crosses of resistant x susceptible inbreds indicated that additive effects accounted for 49 to 97% of the variance and dominance effects for 2 to 47%. There is evidence of isolate x line specificity in the polygenic resistance to both northern and southern leaf blight. In controlled tests, it has been possible to partially overcome polygenic resistance to both diseases by recurrent selection in the pathogen (Leonard 1993). Based on Diallele analysis Rai *et al* (2003) studied gene action in four inbred lines of contrasting resistance level namely CM 104 (Resistant), CM 105 (Resistant), CM 110 (Moderate resistant) and CM 210 (Susceptible). Resistance to maydis leaf blight

was found to be predominantly under the influence of additive gene action along with significant contribution from additive x additive epistasis. However, significant role of dominant gene action along with epistasis could not be ruled out entirely. Holley and Goodman (1989) identified both additive and recessive forms of gene action in an evaluation of tropical inbreds and their hybrid progeny, however recovery of moderately resistant progeny from crosses involving inbreds with recessive sources of resistance indicated that epistatic interactions could contribute to resistance phenotypes.

The results obtained in the present study were in general, congruent with the above findings. Although various types of gene effects, namely additive, dominance and epistasis (i.e., additive x additive, additive x dominance and dominance x dominance) were observed in this study, the general tendency was for additive genetic component to be of predominant importance. The additive nature of resistance also emphasises the utility of procedures such as gene/QTL pyramiding to attain higher levels of resistance.

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