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EPIDEMIOLOGICAL ASPECTS OF LEAF BLIGHT (EXSEROHILUM TURCICUM) OF SORGHUM, *SORGHUM BICOLOR* (L.) MOENCH

K, Kanaka Durga[1,2*], Belum V. S. Reddy[1], M. S. S. Reddy[2], and M Ganesh[2]

[1]ICRISAT Asia Center, Patancheru, 502 324, Andhra Pradesh, India

[2]Acharya N G Ranga Agricultural University, Hyderabad 500-030, Andhra Pradesh, India

ABSTRACT

Late appearance of symptoms (72 h after inoculation), minimum rate of lesion growth (0.056 cm^2) and less number of spores (2.33 ml^{-3}) are considered useful because genotypes with such traits could offer stability in resistance and be deployed in different environments. The parents and the hybrids, which confirm to the above conditions, are of paramount importance in this context. These were SPLB 94025A, SPLB 94025B, 296A among parents and SPLB 94025B x ICSR 90030 and 296A x A 2267-2 among hybrids. The latent period for sporulation

* **Corresponding Author**

inheritance (dominance / recessivity) was not found to be consistent and appeared to be dependent on the genotype.

Key Words: Epidemiology, leaf blight, sorghum.

INTRODUCTION

Leaf blight of sorghum *(Exserohilum turcicum),* reported first by Butler (1918) and later by Mitra (1923) in Punjab has been observed in all the major sorghum growing areas of the world (Tarumoto *et al.,* 1977) and severe damage was observed in USA, Argentina, Mexico, and Israel (Frederiksen, 1980). Effect of leaf blight on grain loss is usually low as compared to the damage caused by grain diseases. Nonetheless, it can take a severe toll in susceptible cultivars if the disease occurs prior to or at the booting stage.

The disease is prevalent and actively widespread particularly in the states of Andbra Pradesh, Haryana, Maharashtra, Madhya Pradesh, Karnataka, Tamilnadu and Rajasthan (Sundarmn *et al.,* 1972). A disease intensity of 32 to 69% (ICRISAT, 1979) has been reported with a loss in grain Yield of 45% (Sharma, 1978).

Early research work was restricted to taxonomical aspects but no serious systematic efforts on the pathological aspects of leaf blight were made. The information on latent period, rate of growth of lesion and sporulation capacity is not available. Hence, the present studies were undertaken to study some of the pathological aspects of the disease and the results are presented in this paper.

MATERIAL AND METHODS

Material and field evaluation:

The material used in the present investigation comprised of resistant (SPLB 94007A and SPLB 94007B), moderately resistant (SPLB 94011A and SPLB 94011B), less susceptible (SPLB 94025A and SPLB 94025B), and susceptible (296A) female lines. The male lines included were A 2267-2 (resistant) and ICSR 90030 (susceptible). The male-sterile lines were crossed with restorer lines and the male-fertile lines were emasculated with restorer lines during *kharif* 1996 and 1997, respectively. The corresponding 14 hybrids along with their parents were sown during *rabi* 1997 to evaluate for leaf blight resistance under artificial disease epiphytotic conditions. The experiment was carried out at ICRISAT Asia Center,

Patancheru, Andhra Pradesh.

The recommended package of practices was followed to raise a healthy crop. Plot size consists of 2 rows of 4 m length each (75 cm between rows and 12 cm within the row). Chemical spraying with fungicides to control powdery mildew and shoot fly was avoided immediately after the inoculation to prevent its adverse effect on the spread of the inoculum. However, need based plant protection measures were taken up at the initial stages of the crop to safeguard the crop from the incidence of shoot fly. Highly leaf blight susceptible entries such as Kundi Jowar and H 112 were planted in two rows each as infector or spreader rows all round the field and after every 12 rows of the test material.

Inoculum preparation:

The inoculum was prepared as follows for artificial whorl-drop method of inoculation (Frederiksen and Franklin, 1978) in the field. The leaves affected with leaf blight *(Exserohilum turcicum* (Pass.) were collected from the field and cut into small pieces and surface sterilized with 0.1 % mercuric chloride for one minute followed by washing with sterile distilled water. Leaf pieces were aseptically transferred to sterilized petri plates containing 20 ml of sterilized Potato Dextrose Agar media (PDA) and incubated at 20°C for encouraging the fungal growth. The fungal growth was aseptically transfered to flasks containing sterilized sorghum grains and incubated at 20°C for 15 days so that the sorghum grains were covered with mycelia and the conidia of fungus colonized grains was removed from the flasks, allowed to airdry and separated as far as possible.

Inoculation:

Sorghum plants were artificially inoculated following whorl-drop method of inoculation (Frederiksen and Franklin, 1978) with the inoculum prepared as follows. The inoculation was carried out 21 and 30 days after emergence of coleoptile during *rabi* 1996 and 1997, respectively. The second inoculation was given one week after the first inoculation. All the plants in each entry were inoculated by placing two or three grains of seed inoculum in the whorl. The high humid conditions were created by providing overhead sprinklers on the same day after inoculation until the disease has spread. It took 40 days for the disease to Spread.

Observations:

The following detailed plot-wise observations viz., latent period, average

rate of lesion growth and sporulation capacity of the pathogen were recorded and average values were computed.

RESULTS AND DISCUSSION

Disease development is detennined essentially by four factors: infection, latent period, pathogen sporulation and loss of infectious tissue (Vander Plank, 1963). Resistance affects one or more of these processes and thus influences the outcome of a potential epidemic. The types of resistance currently employed against *Helminthosporium turcicum* act in a diverse way (Hooker, 1961 and Hooker, 1975). Polygenic resistance reduces the number of lesions produced, while chlorotic lesion resistance primarily suppresses fungal growth and sporulation (Raymundo and Hooker, 1981).

The data collected on the latent period i.e., time of appearance of symptoms after inoculation (HAI), area of the lesion (cm2) at different time intervals after inoculation viz., (10 DAI, 12 DAI, 14 DAI, 17 DAI, 19 DAI, 21 DAI, 26 DAI and 28 DAI) and sporulation capacity (ml-3) for various genotypes are presented in Table 1. However, the grouping of the genotypes was done based on the average disease damage score for two consecutive years.

Latent period:

The data presented in Table 1, shows that among the male-sterile lines, the susceptible genotype (296A) exhibited early symptoms of the disease (36 h) and the resistant line, SPLB 94011, showed symptoms at 98 h after inoculation. Moderately resistant and less susceptible genotypes expressed symptoms at the same time ie. at 72 h after inoculation. Among the restorer lines, susceptible (ICSR 90030) and resistant (A 2267-2) lines exhibited symptoms at 48 and 72 h after inoculation, respectively The hybrid, 296A x ICSR 90030 (S x S) showed symptoms at early stages i.e. at 36 h after inoculation compared to the rest of the hybrids where R x S (SPLB 94011 x ICSR 90030) group, LS x S (SPLB 94025 x ICSR 90030) group and S x R (296A x A 2267-2) group expressed symptoms at 48 h after inoculation. On the other hand, in the crosses SPLB 94011 x A 2267-2 (R x R), SPLB 94007 x A 2267-2 (MR x R), SPLB 94007 x ICSR 90030 (MR x S) and SPLB 94025 x A 2267-2 (LS x R) symptoms were noticed at 72 h after inoculation.

Table 1. Latent period. area of lesion at different time intervals after inoculation and sporulation capacity for selected genotypes of sorghum, rabi season 1997.

Genotypes	Disease reaction group!	Time of app earance symptoms (HAI)	Area of the lesion (cm^2) at different time intervals after inoculation 10 DAI	12 DAI	14 DAI	17 DAI	19 DAI	21 DAI	24 DAI	26 DAI	28 DAI	Mean	Sporulation capacity (ml^{-3})
SPLB 940118	R	98	0.020	0.040	0.060	0.063	0.063	0.067	0.120	0.123	0.123	0.073	4.37
SPLB 94011A	R	98	0.010	0.013	0.023	0.043	0.060	0.060	0.097	0.097	0.097	0.070	4.80
SPLB 94007B	MR	72	0.047	0.097	0:190	0.360	0.420	0.480	0.480	0.480	0.480	0.102	3.83
SPLB 94007 A	MR	72	0.013	0.050	0.207	0.210	0.253	0.270	0.257	0.257	0.257	0.469	8.77
SPLB 94025B	LS	72	0.043	0.160	3.470	6.860	7.060	7.060	7.060	7.060	7.060	0.076	7.04
SPLB 94025A	LS	72	0.090	0.340	4.830	8.207	8.207	8.207	8.207	8.207	8.207	0.056	11.92
296A	S	36	0.010	0.017	0.160	0.520	0.600	1.500	2.240	4.500	5.830	0.197	13.13
A 2267-2	R	72	0.010	0.010	0.033	0.037	0.083	0.090	0.090	0.090	0.090	0.102	18.56
ICSR 90030	S	48	0.010	0.013	0.440	0.770	1.120	1.400	1.670	2.020	2.020	1.690	7.61
SPLB 94011 B X A 2267-2	RxR	72	0.020	0.040	0.060	0.060	0.067	0.067	0.113	0.117	0.117	0.337	5.84
SPLB 94011A X A 2267-2	RxR	72	0.020	0.030	0.033	0.060	0.080	0.100	0.103	0.103	0.103	0.041	7.83
SPLB 94011 B X ICSR 90030	RxS	48	0.010	0.010	0.013	0.040	0.150	0.173	0.173	0.173	0.173	1.697	5.03
SPLB 94011 A X ICSR 90030	RxS	48	0.017	0.033	0.093	0.127	0.647	0.720	0.860	0.860	0.860	6.056	10.71
SPLB 94007B X A 2267-2	MRxR	72	0.010	0.020	0.023	0.033	0.033	0.060	0.063	0.063	0.063	0.188	6.80
SPLB 94007 A X A 2267-2	MRxR	72	0.013	0.020	0.023	0.057	0.103	0.150	0.180	0.183	0.183	0.717	2.93
SPLB 94007B X ICSR 90030	MRxS	72	0.060	0.090	0.840	1.590	1.970	2.230	2.590	2.710	3.190	5.093	2.33
SPLB 94007 A X ICSR 90030	MRxS	72	0.077	0.160	1.043	1.620	2.060	2.180	2.690	2.690	2.690	0.088	6.36
SPLB 94025B X A 2267-2	LS x R	72	0.013	0.013	0.060	0.080	0.083	0.087	0.150	0.153	0.153	0.205	4.77
SPLB 94025A X A 2267-2	LS x R	72	0.013	0.013	0.013	0.053	0.147	0.360	0.363	0.363	0.363	1.709	5.75
SPLB 94025B X ICSR 90030	LS x S	48	0.013	0.023	0.030	0.293	0.297	0.297	0.297	0.297	0.297	0.804	9.99
SPLB 94025A X ICSR 90030	LS x S	48	0.010	0.010	0.140	0.810	0.917	1.080	1.160	1.163	1.163	1 630	4.16
296A X A 2267-2	SxR	48	0.010	0.010	.0.013	0.110	0.430	0.667	2.000	2.000	2.000	0.059	4.91
296A X ICSR 90030	SxS	36	0.023	0.030	0.290	1.020	1.550	1.920	3.200	3.320	3.320	1.052	2.91
Mean		64.890	0.025	0.054	0.526	1.001	1.148	1.271	1.485	1.610	1.689	0.979	6.972
S.Ed.		T =0.026		G=0.136			TxG=0.123						
LSD (5%)		0.051		0.315			0.246						

1. The groups are based on the average disease score for two years.
OAI = Oays after inoculation. T= Time T x G = Time x Genotype interaction
HAI = Hours after inoculation. G= Genotypes

Rate of growth of area of the lesion:

The data presented in Table 1 shows significant differences among the genotypes, time intervals and genotypes x time intervals. The maximum leaf area affected among the parents was observed for SPLB 94025A (6.056 cm2) and SPLB 94025B (5.093 cm^2) followed by 296A (1.709 cm2). However, SPLB 94025A and SPLB 94025B exhibited significant differences (CD=0.136) at 5% level of significance. On the other hand, minimum area of the lesion was recorded by the resistant male-fertile line (SPLB 94011B, 0.076 cm^2) which was not statistically different from its corresponding malesterile line ie., SPLB 94011A (0.056 cm^2). Among testers, A 2267-2 (0.059 cm^2) exhibited minimum disease affected area, and ICSR 90030 recorded maximum affected area.

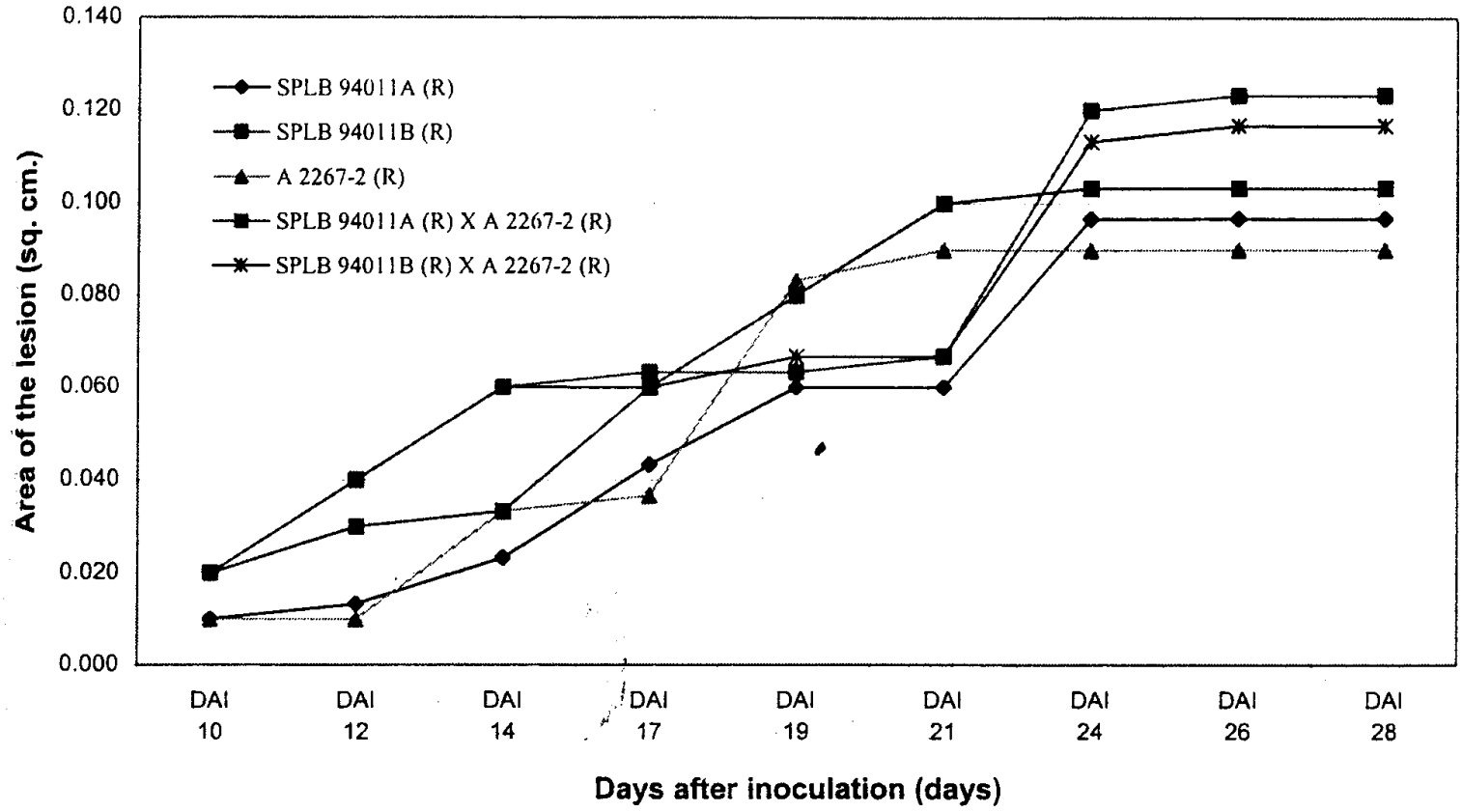
The area of the lesion showed a continuous increase from 10 DAI to 28 DAI. The maximum area of the lesion observed at 28 DAI was statistically significant from the lesion area recorded at 26 DAI. Considering the rate of increase in lesion area at different time intervals, the pattern of lesion spreading was different for different genotypes. The difference was also noticed among the genotypes with different cytoplasm backgrounds i.e., sterile and fertile cytoplasms.

The resistant parents, SPLB 94011B and SPLB 94011A, and A 2267-2 showed an increase from 10 DAI to 19 and 21 DAI, respectively (Fig 1). Thereafter the lesion area remained more or less constant till 28 DAI.

The hybrids involving ICSR 90030 as the male parent recorded greater increase in lesion affected area and average lesion affected area over different time intervals. Considering resistant x resistant hybrids, significant difference was not observed with respect to cytoplasms and any two-time intervals. The significant difference between cytoplasms was observed from 19 DAI for SPLB 94011 x ICSR 90030 (Rx S) (Fig. 1),17 DAI for SPLB 94025A x ICSR 90030 (LS x S) (Fig. 3) and at 28 DAI for SPLB 94007A x ICSR 90030 (MR x S) (Fig 2). The hybrid between susceptible and resistant parents *(296A* x A 2267-2) and S x S parents (296A x ICSR 90030) exhibited a continuous increase in lesion area upto 24 and 26 DAI, respectively (Fig 4). Thereafter, the lesion area remained constant till 28 DAI.

The rate of growth of lesion differs and the increase is not always linear. This implies that multiple measurements might accurately assess the reaction to *Exserohilum turcicum.* Sigulas *et al.,* 1988 also suggested that a genetic interaction exists with time and the entries with similar means early in the season could be markedly different at a later date. The limited spread of the disease as observed in the resistant lines is in confirmity with the findings of Raymundo (1978) and

Figure 1. Growth of blight lesion area at different intervals after inoculation for resistant (R) x resistant (R) and resistant (R) x susceptible (S) hybrids



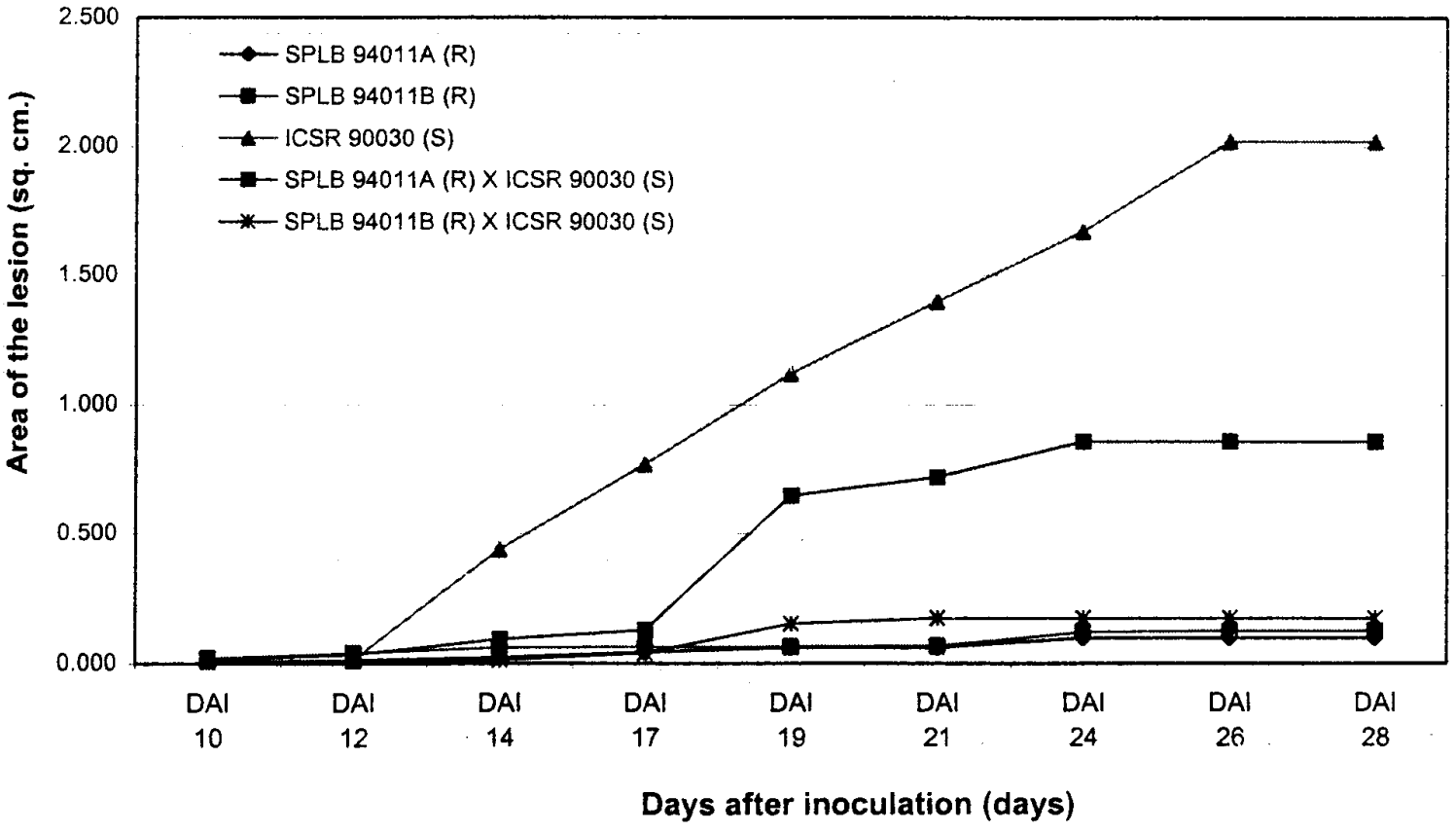
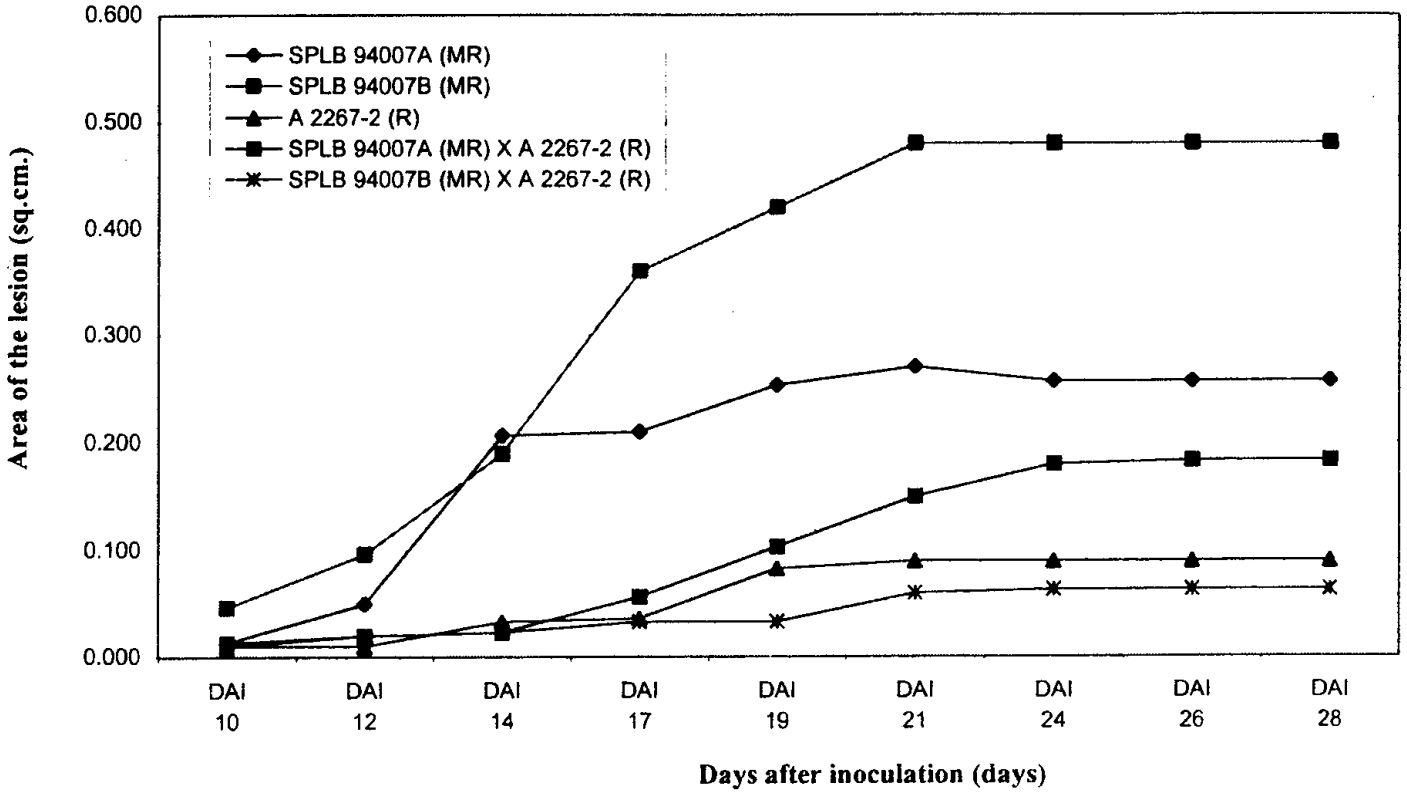


Figure 2. Growth of blight lesion area at different intervals after inoculation for moderately resistant (MR) x resistant (R) and moderately resistant (MR) x susceptible (S) hybrids



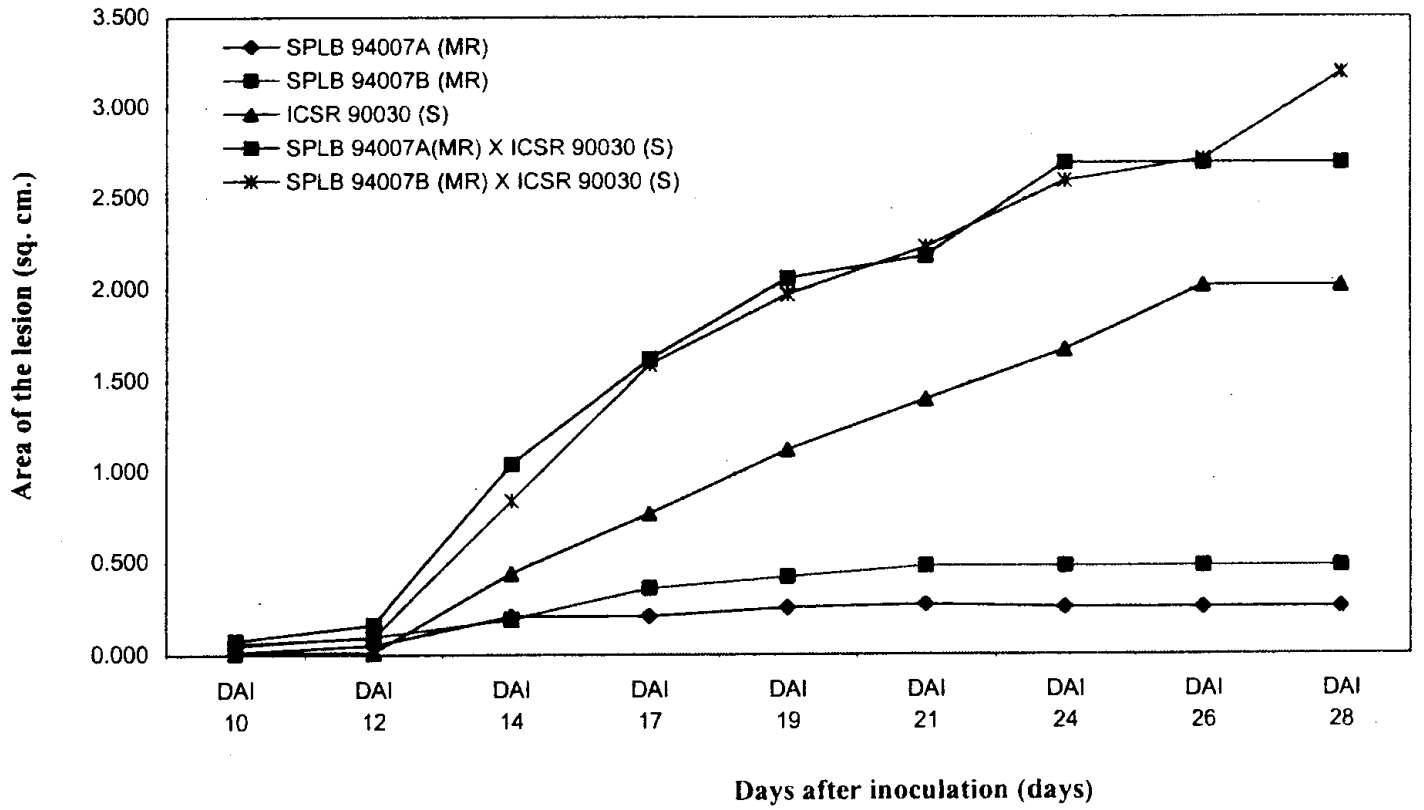
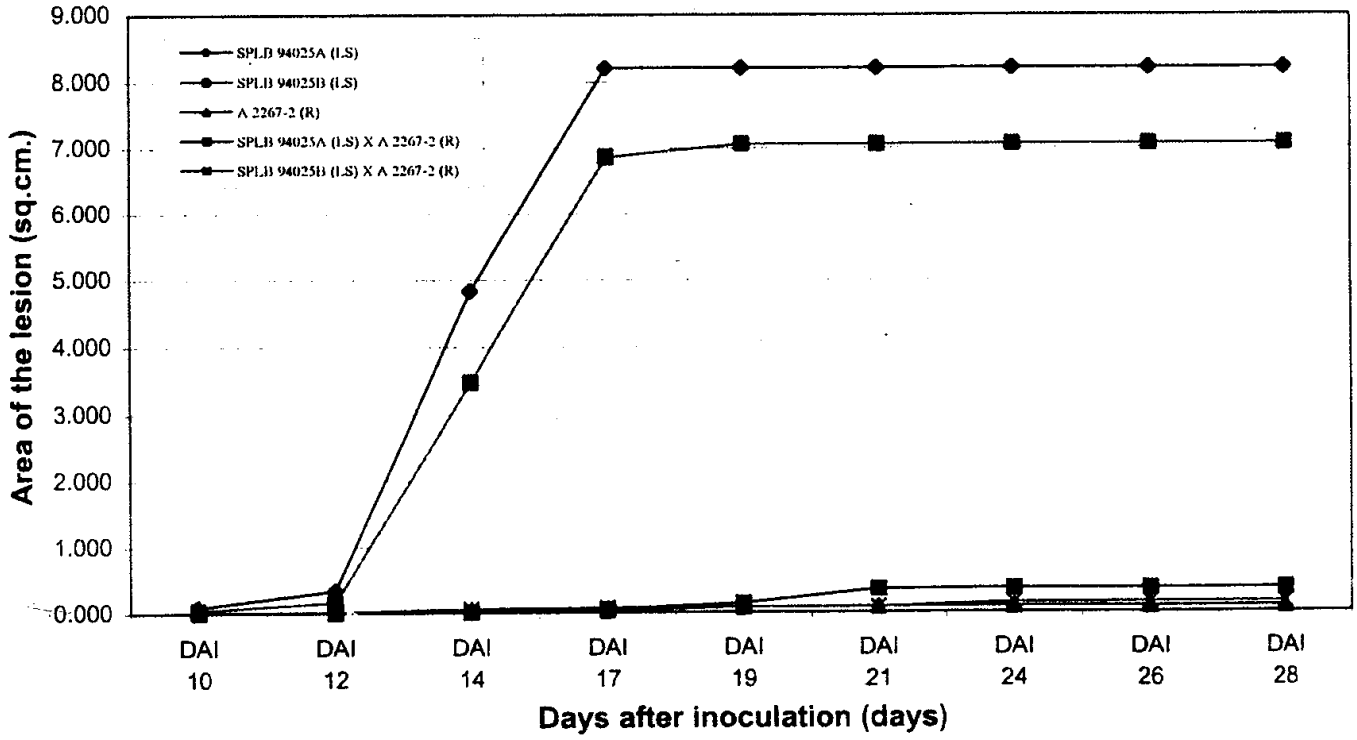


Figure 3. Growth of blight lesion area at different intervals after inoculation for less susceptible (LS) x resistant (R) and less susceptible (LS) x susceptible (S) hybrids



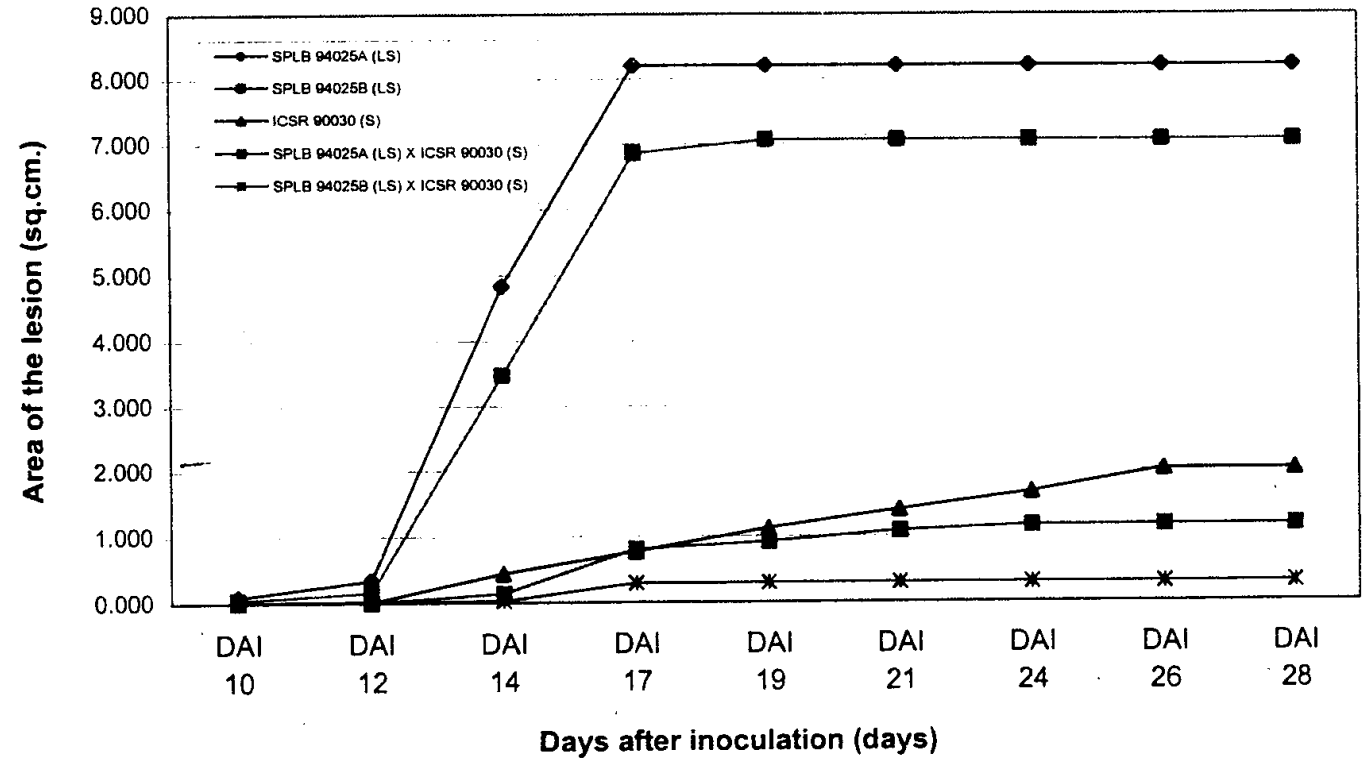
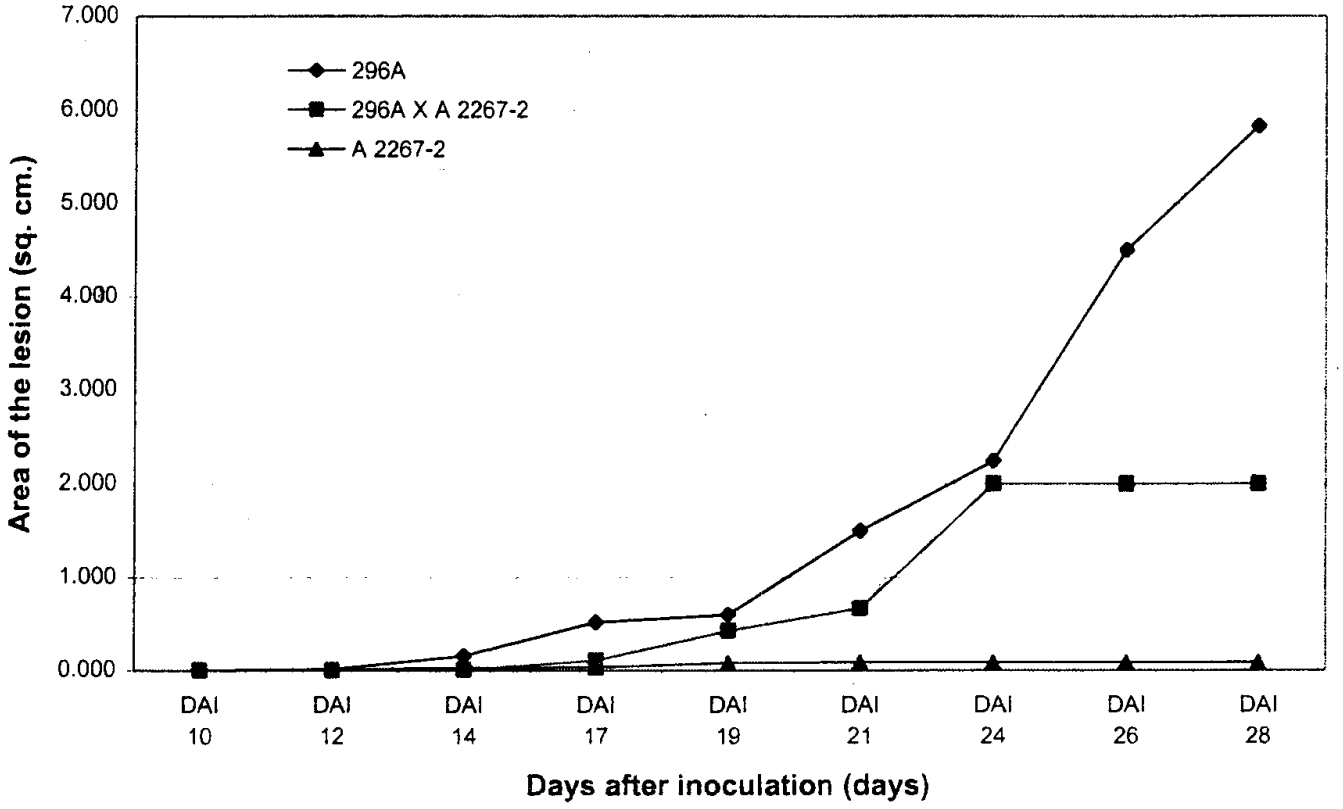
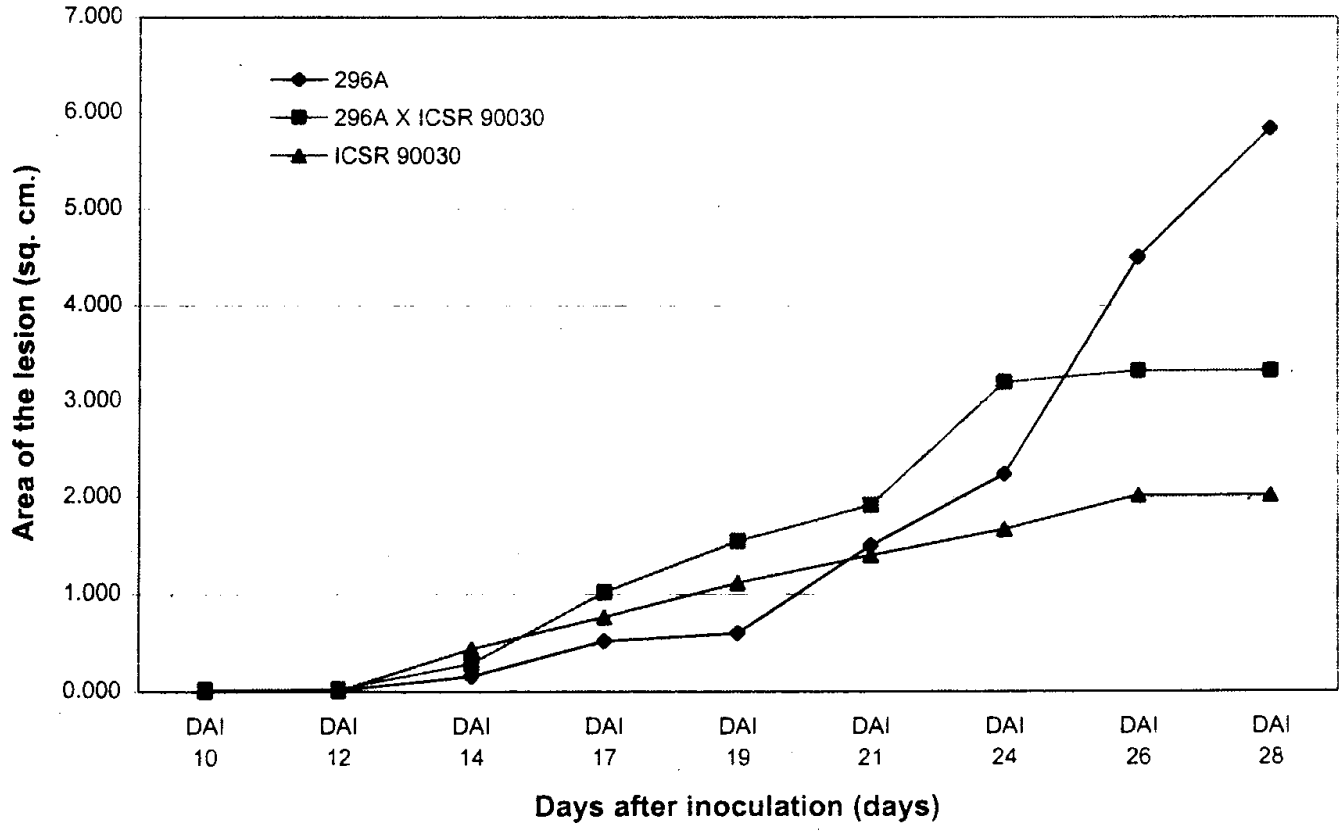


Figure 4. Growth of blight lesion area at different intervals after inoculation for susceptible (S) x resistant (R) and susceptible (S) x susceptible (S) hybrids





Pataky *et al.* (1986) who suggested that the spread of leaf blight in the hybrids of maize with qualitative and quantitative resistance is limited primarily to neighbouring plants and would not reach levels that would cause significant yield reduction.

Sporulation capacity:

The data on sporulation capacity revealed that among the parental lines SPLB 94011B (R, 4.37 spores ml^{-3}) and SPLB 94011A (R, 4.80 spores ml^{-3}) and SPLB 94007B (MR, 3.83 spores ml^{-3}) recorded minimum number of spores. Of all the hybrids studied, SPLB 94007B x ICSR 90030 (MR x S) was reported to have minimum number of spores of 2.33 spores ml-3, while its corresponding A line hybrid recorded 6.36 spores ml-3. Similar behaviour was noticed for the hybrids SPLB 94007 A x A 2267-2 (MR x R, 2.93 spores ml^{-3}), SPLB 94007B x A 2267-2 (MR x R, 6.80 spores ml^{-3}) and 296A x ICSR 90030 (S x S, 2.91 spores ml^{-3}).

Late appearance of symptoms, minimum rate of lesion growth and less number of spores are considered useful because genotypes with such traits could offer stability in resistance and be deployed in different environments. Sigulas *et al.*, 1988 opined that favourable genotypes would be those in which disease development was delayed and growth rate was slow. The parents and the hybrids, which confirm to the above conditions are of paramount importance in this context. These were SPLB 94025 A, SPLB 94025B, 296A, among parents and SPLB 94025B x ICSR 90030 and 296A x A 2267-2 among hybrids. The latent period for sporulation inheritance (dominance / recessivity) was not found to be consistent and appeared to be dependent on the genotype. Turner and Hart (1975) also have suggested that the spread of *Exserohi/um lurcicum* is localized and that spore production is affected significantly by the host genotype. Consequently, large amounts of initial inoculum or conditions that result in excessive secondary inoculum production and dissemination are probably necessary for severe epidemics of leafblight in maize (Pataky *et al.*, 1986).

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