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Identification of Elite Pigeonpea Genotypes against *Fusarium* Wilt and Sterility Mosaic Disease through AMMI and GGE Biplot Analysis

B. Deepak Reddy[1], B. Kumar[1], M.S. Sai Reddy[2], K. Sai Krishna[3], Somala Karthik[2], Rajeev Kumar[4] 10.18805/LR-4838

ABSTRACT

Background: Pigeonpea is an important legume crop in the world. The diseases *Fusarium* Wilt (FW) and Sterility Mosaic Disease (SMD) causes a complete yield loss *i.e.* up to 100% in susceptible pigeonpea genotypes. Amidst of these conditions the selection of stable resistant genotypes against these diseases under varying environmental conditions is the primary management choice for minimizing yield losses (Sharma *et al.*, 2012; Bhaskar *et al.*, 2016).

Methods: Fifty pigeonpea genotypes were evaluated against *Fusarium* wilt and SMD in artificial epiphytotic conditions during four *Kharif* seasons *i.e.,* 2017, 2018, 2019 and 2020. Additive main effects and multiplicative interaction (AMMI) analysis was used to decipher the interaction between genotype (G) and environment (E) for FW and SMD in pigeonpea.

Result: Analysis revealed that in both the diseases IPCA1 and IPCA2 collectively contributed more than 80% interaction. AMMI biplots revealed that *Kharif*- 2019 shows positive effect to FW disease and negative effect to SMD. The AMMI model integration with GGE biplot, identified stable and resistant genotypes (1, 6, 7, 9, 11, 14, 20, 25, 21, 30, 33, 35, 42, 44, 46, 47, 48 and 49) to both FW and SMD based on their performance across diverse environments. These genotypes will be referred for pigeonpea FW and SMD disease resistance breeding programmes.

Key words: AMMI, FW, GGE, Pigeonpea, SMD.

INTRODUCTION

Pigeonpea [*Cajanus cajan* (L.) Millsp.] is a significant legume crop in tropical and subtropical regions of the world (Behera *et al.*, 2020). Globally, pigeonpea is cultivated in 6.99 M ha with production of 5.93 Mt and with productivity of 852 kg/ha. In India it is cultivated in 45 Lha, with annual production of 42 Lt and contributing nearly 90% of world's acreage and production (FAOSTAT, 2020) and it is widely cultivated as a low-input, rain fed crop and has a straight impact on the pecuniary and economically well-being and on the nutritive status of the farmers in the country and gives high profitable returns from each part of the plant (Sharma *et al.*, 2019). Among the diseases *Fusarium* wilt (FW) caused by the fungus *Fusarium udum* and sterility mosaic disease (SMD) by the virus pigeonpea sterility mosaic virus (and vectored by the eriophyid mite) wreak havoc on this legume crop. The pathogen *Fusarium udum* survives saprophytically up to eight years in the soil, causing disease and produces various symptoms *i.e.*, loss of turgidity, inter veinal chlorosis, brown discoloration of xylem tissue and a purple band on stem extending upwards from the base. Sterility mosaic disease (SMD), often referred as "Green Plague", as affected plants are green with excessive vegetative growth but with no flower or pod formation, during favorable conditions spreads rapidly and lead to severe epidemics. Together, these two diseases appear in all growing seasons and during favourable conditions, they are reported to cause even a complete yield loss *i.e.* up to 100% in susceptible pigeonpea genotypes (Saxena *et al.*, 2021). During the conducive environmental conditions it is uneconomical to treat diseases by means of fungicides and cultural methods. Amidst of these conditions the selection of stable resistant genotypes against these diseases under varying environmental conditions is the primary management choice for minimizing yield losses (Sharma *et al.*, 2012). Despite considerable efforts to develop Host Plant Resistance (HPR) through various screening techniques for these diseases, there is

[1]Department of Plant Pathology, Dr. Rajendra Prasad Central Agricultural University, Pusa-848 125, Samastipur, Bihar, India.
[2]Department of Entomology, Dr. Rajendra Prasad Central Agricultural University, Pusa-848 125, Samastipur, Bihar, India.
[3]Department of Basic Sciences and Languages, Dr. Rajendra Prasad Central Agricultural University, Pusa-848 125, Samastipur, Bihar, India.
[4]Department of Agricultural Biotechnology and Molecular Biology, Dr. Rajendra Prasad Central Agricultural University, Pusa-848 125, Samastipur, Bihar, India.

Corresponding Author: B. Deepak Reddy, Department of Plant Pathology, Dr. Rajendra Prasad Central Agricultural University, Pusa-848 125, Samastipur, Bihar, India.
Email: deepakreddyagrico.108@gmail.com

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still a gap in the identification of promising genotypes against FW and SMD under varied environmental conditions. A deeper understanding of genotype (G)×environment (E) interactions with versatile models, like additive main effects and multiplicative interaction (AMMI) model analyses the additive and multiplicative effects of genotype, environment and G×E critically. This model uses ANOVA and principal component analysis (PCA). AMMI biplots and multiplicative model are highly useful for understanding the additive ANOVA model's interaction effect. The researchers have demonstrated the potential utility of AMMI models to understand G×E interactions and to identify resistant genotypes across varying environments (Abamu *et al*., 1998; Verma *et al*., 2016; Parihar *et al*., 2017). With this context, the current study used AMMI model analysis; (i) to test genotypes ability to resist FW and SMD (ii) to understand genotype output and their interactions with varied environments.

MATERIALS AND METHODS

The experiment was conducted at Dr. Rajendra Prasad Central Agricultural University, Tirhut College of Agriculture, Muzaffarpur (Bihar). The sick plots required for conducting experiments were maintained with optimum pathogen population at T.C.A, Dholi and monitored under AICRP pigeonpea wilt and SMD disease screening programmes.

The study was carried out under artificial epiphytotic conditions with fifty pigeonpea genotypes against FW and SMD resistance in their sick plots respectively (Table 1 and Table 1A). The test genotypes were subjected to four different environments *i.e., Kharif*-2017, 2018, 2019 and 2020 using a Randomized Block Design (RBD) with two replications and in each replication twenty five plants were maintained. Observations on wilt incidence were recorded at 30 days interval from sowing (August) to harvesting (March) by counting healthy plants and wilt diseased plants. Maximum wilt incidence on before harvesting was considered to categorize the genotypes to different disease classes as per the disease scoring scale adopted by AICRP on pulses.

Evaluation for FW and SMD Resistance

Fusarium wilt resistance

The wilt sick plot was maintained by incorporating chopped wilt-infected pigeonpea plants in the soil every year. Resistance studies were carried out by planting each genotype in two rows of 5m row length with spacing of 15×40 cm in the sick plot. A susceptible cultivar ICP 2376 was included for every 5 test genotypes as an indicator/ infector rows.

Sterility mosaic disease resistance

Resistance studies of SMD carried out through leaf staple technique in isolated disease nursery using SMD-infested susceptible cultivar ICP 8863. At two-leaf stage, SMD infected leaf was pinned to the primary leaf of the test seedling using a small paper stapler in such a way that the lower surface of infested leaf was in contact with the primary leaf thus facilitating in successful disease infection.

Each genotype was planted in two rows of 2 m length with 15×40 cm spacing. A SMD susceptible cultivar ICP 8863 was included for every 5 test genotypes as an indicator row. The ICP 8863 was planted in the sick plot one month in advance of the regular planting time to serve as an infector rows in order to have a good source of virus inoculum (Sharma *et al*., 2019).

Per cent Disease Incidence (PDI)

PDI of both FW and SMD recorded in four environments was pooled and categorized into resistant (0-10%), moderately resistant (10-30%) and susceptible (>30%).

$$PDI = \frac{\text{No of wilted plants}}{\text{Total no of plants}} \times 100$$

Weather data was taken for crop standing period *i.e.,* sowing (August) to harvesting (March) in weekly intervals from Meteorological station, RPCAU.

AMMI and GGE biplot analysis

The performance of fifty pigeonpea genotypes and four testing environments for FW and SMD resistance and the

Table 1: Details of pigeonpea genotypes and its reaction to fusarium wilt and SMD in experimental trials.

Genotype	Code	Genotype	Code
AKTE 1701	1	BDN711	26
AKTE 12-04	2	IPA16-18	27
WRGE93	3	PA291	28
GRG152	4	PA536	29
IPAM16-3	5	KPL43	30
IBTDRG-7	6	MAL13	31
BDN2013-1	7	ICP8863	32
IPA15-03	8	BSMR243	33
TRG111	9	IPA203	34
ICP8862	10	TDRG59	35
ICP8859	11	MAL-45	36
ICP9174	12	BSMR853	37
ICP8858	13	BAHAR	38
ICPL99048	14	BDN1	39
IPA8F	15	BDN2	40
KPL44	16	BRG4	41
MAL51	17	MAL-6	42
IPA15-06	18	WRGE121	43
IPA14-4A	19	ICP7035	44
TJT501	20	KRG33	45
GJP1606	21	BDN2013-2	46
JKM189	22	BSMR-736	47
IPA15-02	23	KA16-1	48
WRGE127	24	KA16-5	49
WRGE122	25	ICP2376	50

Table 1A: Details of Pigeonpea genotypes and its reaction to *Fusarium* Wilt and SMD in experimental trials.

		PDI-Wilt							PDI-SMD						
Genotype	Code	*kharif* -2017	*kharif* -2018	*kharif* -2019	*kharif* -2020	Mean	PC1	PC2	*kharif* -2017	*kharif* -2018	*kharif* -2019	*kharif* -2020	Mean	PC1	PC2
AKTE 1701	1	19.20	17	23.36	22.41	20.50	-0.00	-0.24	27.04	28	12.52	26.58	23.54	-0.35	0.13
AKTE 12-04	2	25.04	32	14	8.69	19.93	-0.00	-0.16	28.66	65	11.01	44.83	37.38	1.919	-1.25
WRGE93	3	22.87	18.52	18.69	26.5	21.65	0.80	0.42	38.18	78	35.41	47.27	49.72	1.90	0.42
GRG152	4	10.34	8	7.84	13.34	9.88	0.65	0.26	34.83	59.4	29.16	37.39	40.20	1.02	0.66
IPAM16-3	5	18.87	17.89	20.87	18.41	19.01	0.10	0.00	42.91	80.6	17.39	45	46.48	2.53	-0.66
IBTDRG-7	6	14.58	15.22	14	24.40	17.05	0.91	0.67	12	2.17	10.25	20.33	11.19	-1.66	0.28
BDN2013-1	7	10.59	13.07	2.08	6.16	7.98	0.55	0.21	16.66	16.82	8.99	16.41	14.72	-0.60	0.61
IPA15-03	8	67.5	65.17	94	70.10	74.19	-1.86	1.17	20	47.25	4.35	46	29.40	1.00	-1.96
TRG111	9	22.41	16.82	21.90	23.90	21.26	0.35	0.45	12.5	8.99	8.99	18	12.12	-1.10	0.42
ICP8862	10	20.41	21.36	20.41	14.85	19.26	0.02	-0.64	21.28	26	16.93	4.166	17.10	-0.01	2.25
ICP8859	11	13.36	12.14	15.15	4.16	11.21	-0.35	-0.73	80.65	8.89	16.66	100	51.55	-4.58	-5.48
ICP9174	12	6.431	4.17	10	0	5.15	-0.37	-0.35	28.26	24.42	12.68	8.16	18.38	-0.25	1.63
ICP8858	13	10	11.82	16.02	2	9.96	-0.61	-0.73	41.73	8.51	10.41	23.46	21.03	-1.95	0.18
ICPL99048	14	12.5	8.51	16.52	18.33	13.97	0.22	0.85	18.87	2.17	12.16	14.25	11.87	-1.74	0.94
IPA8F	15	34.75	6.81	13.63	16.18	17.85	0.68	-0.15	34.78	6	10	18.41	17.30	-1.86	0.52
KPL44	16	18.18	16.42	12.25	55	25.46	2.80	3.01	12.77	6.62	6.52	16.33	10.56	-1.17	0.36
MAL51	17	18.75	20.08	23.05	18.41	20.07	-0.05	-0.09	24.24	26	8.34	42.83	25.36	-0.65	-1.51
IPA15-06	18	77.66	76.61	77.38	63.5	73.79	-0.44	-1.23	16.41	54	6.34	18.33	23.77	2.094	0.47
IPA14-4A	19	10	6.167	6.54	4	6.68	0.22	-0.41	24.41	22.23	4.44	8.16	14.82	-0.09	1.014
TJT501	20	44.91	34	55.16	46.08	45.04	-0.67	1.20	10.25	8.17	0	17.61	9.01	-0.85	-0.21
GJP1606	21	6.25	0	2	2.17	2.61	0.36	-0.08	43.56	81.82	8.51	59.25	48.29	2.55	-2.46
JKM189	22	20.41	18	23.52	22.91	21.21	0.14	0.41	26.25	82.97	6.25	31.54	36.75	3.67	-0.41
IPA15-02	23	77.08	65.17	80	59.25	70.38	-1.09	-0.58	40.75	41.43	15.61	41.47	34.82	-0.09	-0.73
WRGE127	24	56.62	53.5	48	45	50.78	0.37	-1.12	30.66	60.64	8.71	30.58	32.65	1.92	-0.27
WRGE122	25	29.80	51.82	26.70	24.58	33.23	0.64	-2.39	18.41	29.57	4.54	31.47	21.00	0.07	-0.86
BDN711	26	55	56	54	46	52.75	0.69	0.23	16	2.27	6.34	14.25	9.72	-1.51	0.512
IPA16-18	27	8.16	14	2.27	22.41	11.71	1.69	0.47	54	93.45	8.17	42.83	49.62	3.50	-1.04
PA291	28	18.75	40	73.58	46.08	44.61	-2.59	2.34	30.58	65.22	21.90	32.18	37.47	1.85	0.58
PA536	29	75	44.89	90	63.25	68.29	-2.09	1.77	21.28	66	6.08	18.41	27.95	2.84	0.52
KPL43	30	29.54	10.76	38.34	24.41	25.77	-1.01	1.23	16.52	22	8.51	24	17.76	-0.37	-0.01
MAL13	31	17.61	23.5	11.31	60.11	28.14	2.49	1.66	27.71	8.08	8	36.83	20.16	-1.88	-1.13
ICP8863	32	31.04	34.6	6.43	6.16	19.56	1.13	-3.31	56.60	76.96	62	91.91	71.87	-0.26	-1.23
BSMR243	33	10.52	8.17	11.06	12.86	10.66	0.35	0.30	17.39	18.33	16.25	14.34	16.58	-0.67	1.31
IPA203	34	24	39.5	86	4	38.39	-5.87	-1.03	21.28	4.16	2.08	12.25	9.95	-1.33	0.39
TDRG59	35	13.06	9.09	15	12.25	12.35	0.04	0.21	13.63	6.43	8.16	18.33	11.64	-1.29	0.32
MAL-45	36	6.340	6.43	12	10.25	8.76	0.041	0.41	33.21	4	14.16	14.58	16.49	-2.00	1.12
BSMR853	37	12.34	13.87	32.95	23.21	20.60	-0.84	1.38	30.66	49.32	4.34	32	29.08	1.20	-0.76
BAHAR	38	68	59.08	59.66	67.25	63.50	0.85	0.27	21.04	2	4.16	8.16	8.84	-1.45	0.86
BDN1	39	8.54	10.52	32.03	2.17	13.32	-2.02	-0.03	8.166	8.42	8.16	6.16	7.73	-0.77	1.31
BDN2	40	30	16.36	20.81	15.03	20.55	0.07	-0.59	26.41	32.61	22.87	16.58	24.62	-0.08	1.72
BRG4	41	25.63	56	26.19	44.83	38.16	1.79	-0.83	36	68	4	8.16	29.04	2.93	1.28
MAL-6	42	16	10	4.08	18.87	12.24	1.40	0.24	17.78	6.25	2.38	12.77	9.80	-1.12	0.37
WRGE121	43	23.37	15.32	20.5	10.25	17.36	-0.27	-0.67	34.83	52	0	9.10	23.99	1.90	0.81
ICP7035	44	16.81	23.91	8.54	16.41	16.42	1.11	-0.96	22.41	4.27	4.34	12.25	10.82	-1.4	0.56
KRG33	45	21.73	18.29	2.17	9.41	12.90	1.27	-1.51	46	70.38	8.69	61.25	46.58	1.63	-2.67
BDN2013-2	46	23.04	17.09	14.81	18.41	18.34	0.67	-0.28	16.52	26.09	6.71	31.25	20.14	-0.18	-0.71
BSMR-736	47	20.41	20	17.88	18.69	19.25	0.43	-0.29	30.68	16.52	2	12.25	15.36	-0.66	0.49
KA16-1	48	33.39	32.85	39.39	26.66	33.08	-0.57	-0.43	20	12	8	16	14.00	-0.98	0.558
KA16-5	49	41.73	40.18	50	29.71	40.41	-1.07	-0.73	16.41	21	0	18.33	13.94	-0.08	-0.17
ICP2376	50	64	72	74	56	66.50	-0.85	-1.09	19.50	2.08	4.08	8.16	8.46	-1.41	0.85

PDI: Percent disease incidence, PC1: Principal component 1, PC2: Principal component 2, SMD: Sterility mosaic disease.

G×E interactions were analyzed in this study. The disease incidence data from four test environments were structured to fit the AMMI models. The AMMI statistical model and computational methods described in Gauch (2013) were used. Analysis of variance was utilized to partition the variation into genotype (G) and environment (E) main effects and the G×E interaction effect. The genotype and genotype by environment (GGE) biplot analysis was utilized to graphically ascertain the stability of genotypes over the four test environments (Yan, 2001; Parihar *et al.*, 2017). Both the AMMI and GGE biplots analyses were carried out using the softwares GEA-R developed by 'CIMMYT' and 'R' package Agricolae.

RESULTS AND DISCUSSION

AMMI analysis of FW and SMD

AMMI ANOVA of FW revealed that among total sum of squares (SS), 82.21% of the SS was observed for genotype effect, 0.47% of SS provides environment effect and 17.31% of SS was observed for interaction effect (G×E). G×E interaction effect was 4.74 times smaller than that for the genotype SS and it was also 36.85 times larger than that for environment SS thus indicating that the variation in the genotypes across the environments were significant. The G×E was further divided into Interaction Principal Component Axis (IPCA) and residuals, in which IPCA1 has contributed 55.85% of SS followed by IPCA2 which contributed 27.96% of SS and IPCA1 and IPCA2 cumulatively contributed to 83.81% of the total SS (Table 2). Similar trend of results were observed for SMD AMMI ANNOVA, *i.e.,* among the total sum of squares genotype effect was 47.99%, environment effect was 15.50% and G×E interaction effect was 36.49%. G×E interaction effect was 1.31 times smaller than genotype and 2.35 times larger than environment. IPCA1 and IPCA2 collectively contributed 89.79% of the total SS, in which IPCA1 contributed 67.70% SS and IPAC2 contributed 22.08% SS (Table 2).

The high SS for genotypes obtained in the AMMI ANOVA analysis for both FW and SMD indicated the diverse nature of the pigeonpea genotypes with significant differences in the mean PDI causing most of the variations within the reactions of the genotypes (Persaud and Saravanakumar, 2018; Persaud *et al.*, 2019). It also indicated that the resistance has also been influenced by the G×E effect. In the current research for both FW and SMD, IPCA1 and IPAC2 collectively contributed more than 80% of interaction effect. It explains that IPCA1 and IPAC2 will be sufficient for studying interaction between 50 pigeonpea genotypes over four environments. (Yan *et al.*, 2000; Nayak *et al.*, 2008).

AMMI biplot analysis

AMMI biplot explains the relationship between the per cent disease incidence of genotypes, test environments and IPCA1 scores. Genotypes or environments present left side to perpendicular line are resistant genotypes (lower disease incidence) and environments will be less favourable for disease screening. If they are present on the right side of perpendicular line genotypes will be highly susceptible (high disease incidence) and environments will be favourable for disease screening (Persaud *et al.*, 2019; Srivastava *et al.*, 2021).

AMMI1 biplot (Trait vs IPCA1)

AMMI1 biplot analysis of FW (Fig 1) revealed that genotypes 21, 12, 19, 7, 36, 4, 13, 33, 11, 27, 35, 42, 45, 39, 14, 44, 6,

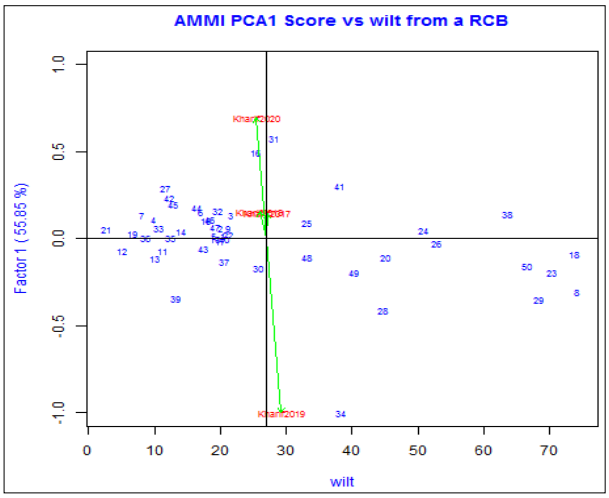

Fig 1: AMMI1 biplot displaying (*Fusarium* wilt) incidence and IPCA1 scores of genotypes at four test environments.

Table 2: AMMI ANOVA for Genotype X Environment interactions.

Source	Wilt						SMD					
	SS	%SS	Df	MSS	F	P	SS	%SS	Df	MSS	F	P
Env	859.43	0.47	3	286.47	2.91	S	26722.35	15.5	3	8907.45	108.74	S
Gen	150357	82.21	49	3068.51	31.26	S	82706.91	47.99	49	1687.89	20.6	S
Env*Gen	313673.72	17.31	147	215.46	2.19	S	62889.26	36.49	147	427.81	5.22	S
IPCA1	17690.2	55.85	51	346.86	3.51	S	42576.4	67.70	51	834.83	10.17	S
IPCA2	8857.65	27.96	49	180.76	1.83	S	13892.18	22.08	49	283.51	3.45	S
IPCA3	5125.86	16.18	47	109.06	1.10	NS	6420.67	10.20	47	136.61	1.66	S
Residuals	19631.98		200	98.15			16382.64		200	81.91		

Df: Degree of freedom, SS: Sum of squares, MSS: Mean sum of squares, IPCA: Interaction Principal Components Axis, F: F calculated value, P: Probability, S: Significant, NS: Non-significant.

43, 10, 46, 32, 47, 15, 22, 5, 17, 1, 40, 2, 9, 37, 30, 3 and 16 were resistant for FW and environments *Kharif*-2018 and *Kharif*-2020 are less favourable to FW due to the presence on the left side of perpendicular line. While, genotypes 18, 8, 23, 29, 50, 38, 26, 24, 20, 28, 49, 41, 34, 25, 48 and 31, environment *Kharif*-2019 on the right side of perpendicular line which are susceptible and favourable to FW respectively and *Kharif*-2017 was present on perpendicular line, it shows that it is a mean environment. Whereas, AMMI1 biplot analysis of SMD shows that genotypes 1, 25, 46, 10, 19, 49, 12, 30, 33, 47, 7, 48, 9, 39, 20, 50, 15, 31, 13, 36, 14, 16, 42, 35, 34, 38, 6, 44 and 26 are resistant for SMD disease and environment *Kharif*-2019 was less favourable for disease screening, however genotypes 32, 11, 23, 4, 45, 3, 27, 21, 5, 22, 24, 37, 2, 8, 28, 29, 41 and 17 were susceptible to disease and environments *Kharif*-2017, *Kharif*-2018 and *Kharif*-2020 were favourable for SMD disease screening. While genotypes 18, 43, 40 present exactly on the perpendicular line *i.e.,* representing mean genotypes (Fig 2) (Sharma *et al*., 2019).

Similarly, genotypes or environment with low IPCA1 scores have slight interactions, good stability and better adaptation over test environments, while large IPCA1 scores have big interaction effect and reflect more specific stability and adaptation to specific environments (Persaud and Saravanakumar, 2018). From our experiment it is observed that *Kharif*-2018, *Kharif*-2017 and larger proportion of genotypes recorded low IPCA1 scores and showed small interactions, which led to clustering of the genotypes on the FW biplot (Fig 1), While environments *Kharif*-2019, *Kharif*-2020 and genotypes 34, 16, 28 recorded the highest IPCA1 scores. In SMD, *Kharif*-2020, *Kharif*-2017 observed with low IPCA1 scores had small interactions, while a greater effect was observed for *Kharif*-2018, *Kharif*-2019. Similar to FW IPCA1 scores, in SMD also most of the genotypes exhibited low IPCA1 scores which leads to clustering. However, genotypes 11 and 22 recorded the highest IPCA1 scores (Table 3).

AMMI2 biplot (IPAC1 vs IPAC2)

AMMI2 biplot analysis differentiates environments and responsive genotypes. Genotypes which are placed near to test environments have better specific adaptions to test environments, while close to origin shows stable performance in all test environments, while away from origin show differential response to test environments. This analysis also illustrates an interaction between the genotypes and environments with reference to sector, if both are present in same sector then they interact positively, while in opposite sector interacts negatively and in adjacent sectors show complex interactions (Persaud and Saravanakumar, 2018; Persaud *et al*., 2019).

AMMI2 biplot analysis of FW illustrates genotypes 2 and 25 falling close to *Kharif*-2017; Genotype 32 falling close to *Kharif*-2018 and genotype 16 and 31 falling close to *Kharif*-2020, were particularly suitable and showed stable FW response in that environment. The genotypes 28 and 29 that were between *Kharif*-2019 and *Kharif*-2020 indicated highly stable disease response in those environments. While, *Kharif*-2019 and *Kharif*-2020 are the most differentiating environments; 16, 31, 28 and 34 are most responsive genotypes (Fig 3). Similarly, AMMI2 biplot analysis of SMD envisages that *Kharif*-2018, *Kharif*-2019 and *Kharif*-2020 were the most differentiating environments; while genotypes 11, 45 and 21 were the most responsive genotypes. From the biplot it is understood that genotypes

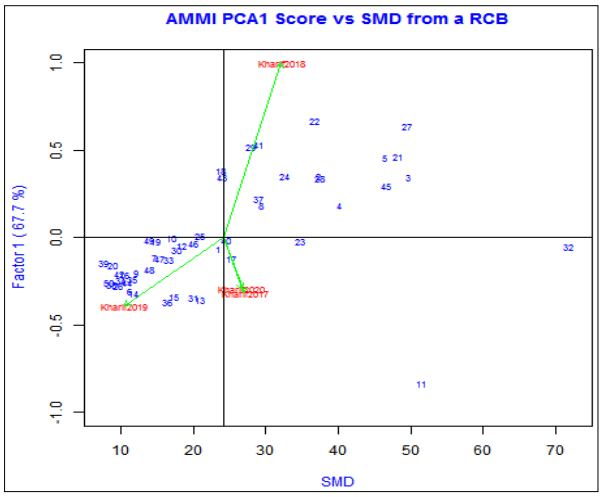

Fig 2: AMMI1 biplot displaying SMD disease incidence and IPCA1 scores of genotypes at four test environments.

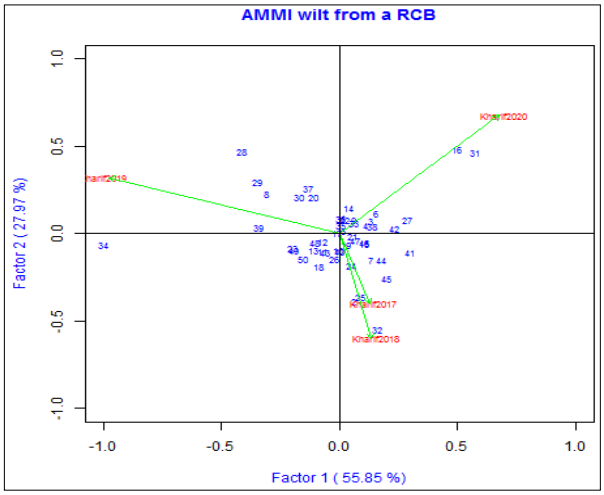

Fig 3: AMMI 2, Biplot (*Fusarium* Wilt) displaying genotypes and environment in first and second Principal Component (PC) axis.

Table 3: Environments IPCA scores of *Fusarium* Wilt and SMD.

Season	IPCA Scores	
	Wilt	SMD
Kharif-2017	1.473609	-3.30926
Kharif-2018	1.444307	10.44035
Kharif-2019	-7.90855	-4.08007
Kharif-2020	4.990629	-3.05102

42, 6, 13, 15, 26, 50, 48, 47, 7 and 35 falling close to *Kharif*-2017; Genotypes 22 and 27 falling close to *Kharif*-2018 were particularly suitable and showed stable SMD response in that environment. The genotype 31 was between *Kharif*-2017 and *Kharif*-2020; the genotype 36 was between *Kharif*-2017 and *Kharif*-2019 indicated highly stable disease response in those environments (Fig 4).

GGE biplot (discriminative vs representative)

Discriminating power of the environment is proportional to its length from the origin. Closer environments have less discriminative power and by increasing length from origin discriminative power of environment increases and it will explain more about the genotypes. Average environment axis will explain the ideal test environment for testing the genotypes (Persaud and Saravakumar, 2018; Srivastava *et al*., 2021). FW biplot shows that *Kharif*-2019 was the most discriminating environment and *Kharif*-2020 was the least discriminating environment (Fig 5). *Kharif*-2017 followed by *Kharif*-2018 were ideal test environments for FW testing because in biplot they were close to the "average environment" and "ideal test environment" and *Kharif*-2019 and *Kharif*-2020 were least representative because they were away from AEA (Fig 7).

The SMD biplot showed that *Kharif*-2018 to be the most discriminating (informative) environment, whereas *Kharif*-2019 was the least discriminating environment (Fig 6). Average environment axis explains *Kharif*-2020 was the most representative environment and *Kharif*-2019 least representative environments (Fig 8) (Tekalign *et al*., 2017; Tekdal and Kendal 2018; Sharma *et al*., 2019).

Which-won-where biplot

FW biplot revealed that two mega environments existed in the study (Fig 9), first includes 3 test environments (*Kharif*-2017, *Kharif*-2018 and *Kharif*-2020) and remaining test environment (*Kharif*-2019) befitted second mega environment. Genotypes with constant susceptibility in all test environments will be treated as winners. Each mega environment sector showed different winning genotypes (Yan and Tinker, 2006). From our investigation genotype 18 was identified as the winner in first mega environment and genotype 8 was the winner in the second mega environment. Genotypes moving away from origin and distanced from each other reveals their diverse in FW resistant status, having less stability and contributing great in Genotype and G×E interactions, while some of the genotypes (1, 2, 3, 4, 5, 6 , 7, 9, 10, 11, 14, 15, 17, 19, 20, 21, 22, 25, 30, 32, 33, 35, 36, 37, 40, 42, 43, 44, 45, 46, 47, 48 and 49) clustered close to the origin explains the similarity in FW resistant status (Fig 5).

Similarly, the SMD biplot demonstrated that two mega environments existed in the study. First mega environment includes (*Kharif*-2017, *Kharif*-2019 and *Kharif*-2020) while second mega environment includes (*Kharif*-2018). Genotypes 32 and 27 were winning genotypes in first and second mega environments respectively (Fig 10). Furthermore,

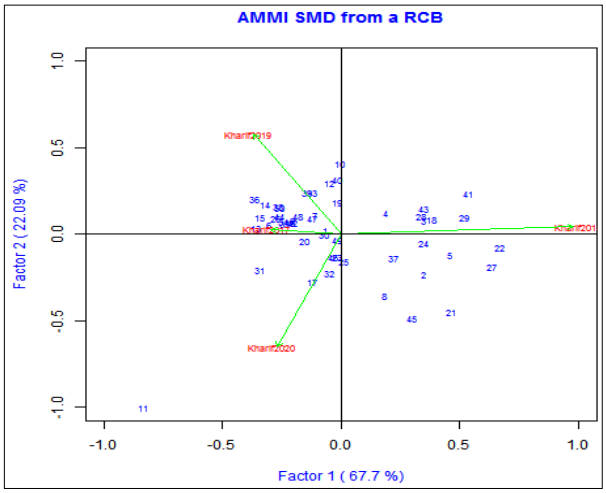

Fig 4: AMMI 2, Biplot (SMD) displaying genotypes and environment in first and second principal component (PC) axis.

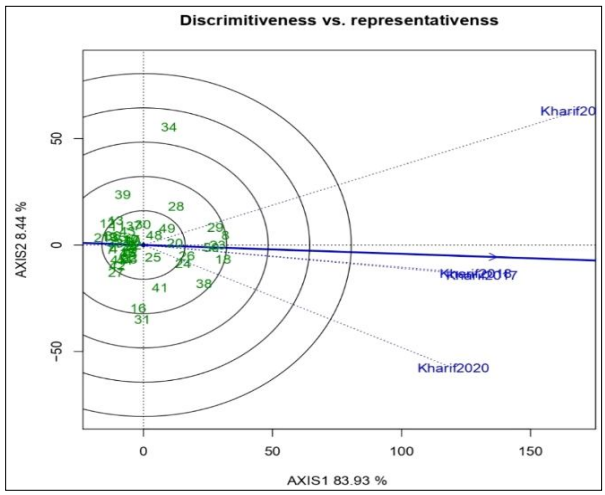

Fig 5: The GGE biplot (*Fusarium* Wilt) showing of genotypic performance in test environments.

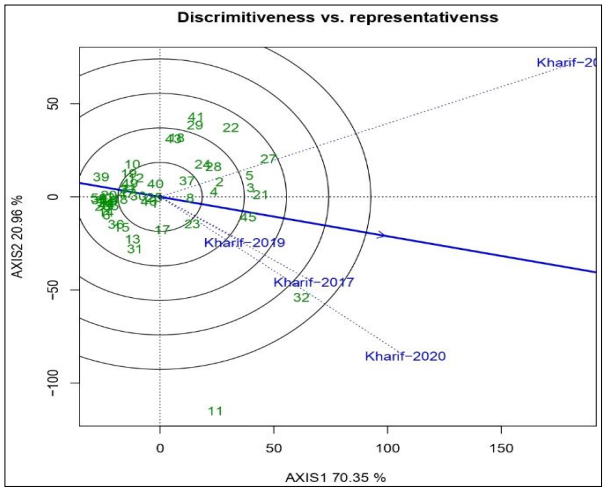

Fig 6: The GGE biplot (SMD) showing of genotypic performance in test environments.

the genotypes clustered towards the origin of the biplot (1, 6, 7, 9, 11, 12, 14, 16, 20, 25, 26, 30, 33, 34, 35, 42, 42, 44, 46, 47, 48, 49 and 50) have been referred to as consistent and stable resistant lines to SMD (Fig 6) (Sharma *et al.*, 2015; Tekalign *et al.*, 2017; Tekdal and Kendal, 2018).

Weather data

SMD

In AMMI1 biplot of SMD (Fig 2) it was observed that *Kharif*-2017, *Kharif*- 2018 and *Kharif*-2020 were present on the right side of the biplot and they were favourable for disease screening. Weather data also supports the above findings; temperature and wind speed were more in the *Kharif*-2017, *Kharif*-2018 and *Kharif*-2020 when compared to *Kharif*-2019, the temperature and wind speed may help in the mite population development and spread (Table 4).

FW

In AMMI1 FW biplot (Fig 1), *Kharif*-2019 on the right side of the biplot and it is more favourable for disease screening. Weather data also revealed that average rainfall also more and temperature was low in the year *Kharif*-2019 which may help in the pathogen development and spread.

Table 4: Weather data during crop season (September to March).

Season	Temperature (°C)	Rainfall (mm)	Wind speed (Km/hr)
Kharif-2017	27.50	23.75	2.87
Kharif-2018	28.10	23.142	2.89
Kharif-2019	26.10	70.20	1.10
Kharif-2020	27.10	33.48	2.85

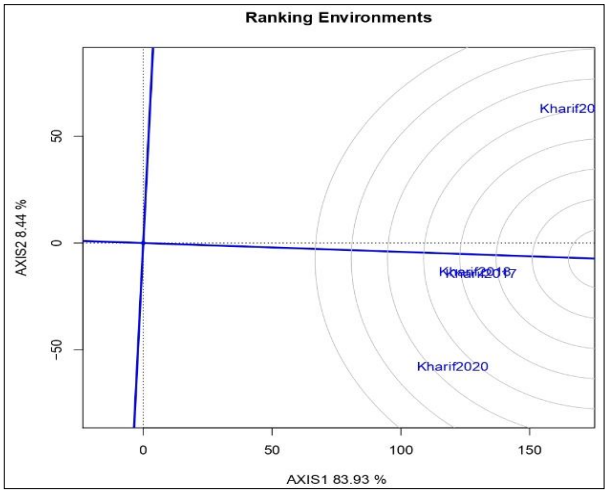

Fig 7: AEC (*Fusarium* wilt) view illustrating the rank of ideal environments.

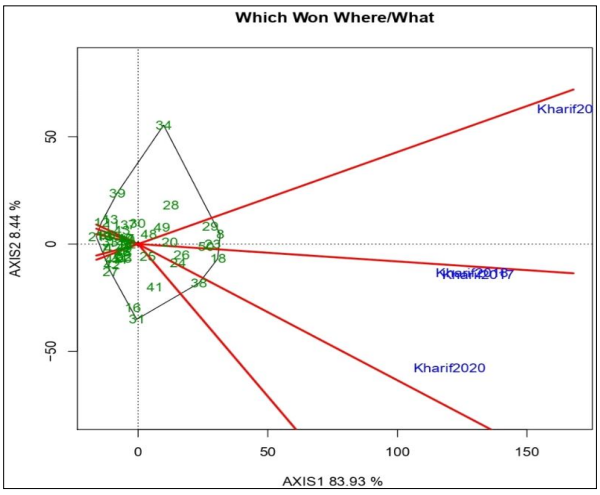

Fig 9: The which-won-where view of the GGE biplot (*Fusarium* Wilt) showing which genotypes, performed better in which environments.

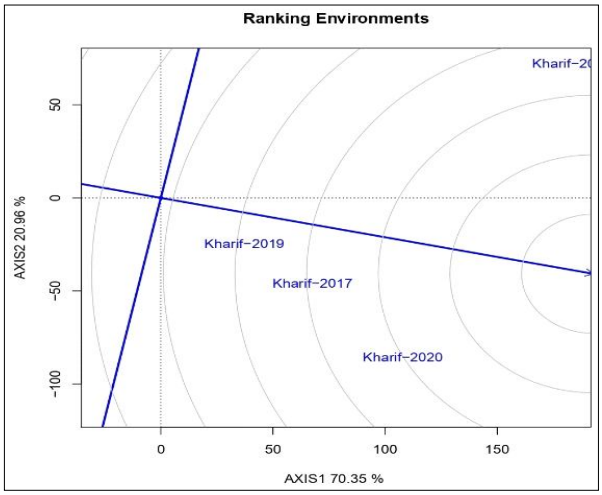

Fig 8: AEC (SMD) view illustrating the ideal rank of ideal environments.

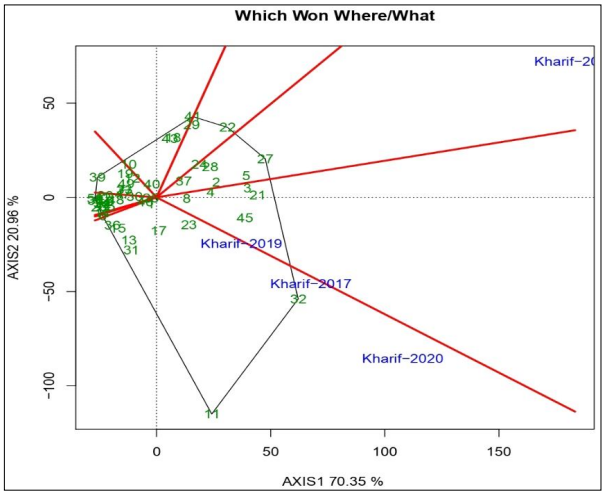

Fig 10: The which-won-where view of the GGE biplot (SMD) which genotypes, performed better in which environments.

CONCLUSION

The current research concluded that G×E interaction influencing FW and SMD incidence in pigeonpea and demands for the next studies to know how G×E interaction influences their incidence. The high SS for genotypes obtained in the AMMI ANOVA analysis for both FW and SMD indicated the diverse nature of the pigeonpea genotypes with significant differences in the mean PDI causing most of the variations within the reactions. It also indicated that the resistance has also been influenced by the G×E effect. In the current research for both FW and SMD, IPCA1 and IPAC2 collectively contributed more than 80% of interaction effect. It explains that IPCA1 and IPAC2 will be sufficient for studying interaction between 50 pigeonpea genotypes over four environments. The environment *Kharif-2019* suitable for FW disease screening and *Kharif*-2017, *Kharif*- 2018 and *Kharif*-2020 was suitable for SMD screening, the weather data also supports to above findings. AMMI model in integration with GGE biplot, enabled us to identify stable and resistant genotypes to FW and SMD (1, 6, 7, 9, 11, 14, 20, 25, 21, 30, 33, 35, 42, 44, 46, 47, 48 and 49) based on their performance across diverse environments.

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