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GENETIC VARIABILITY AND CLUSTER ANALYSIS FOR FUSARIUM WILT AND STERILITY MOSAIC DISEASE RESISTANCE IN PIGEONPEA (*CAJANUS CAJAN* L. MILLSP.)

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ABSTRACT

Pigeonpea is an important protein source, catering to the nutritional needs of the global population. The potential grain yield of pigeonpea is affected by many biotic stresses. Mainly fusarium wilt (FW) and sterility mosaic disease (SMD) are economically devastating diseases. To combat the losses, disease resistant cultivars should be developed. In this context, the current investigation was designed to study the genetic variability present in a diverse panel of 100 pigeonpea genotypes. The experimental material was screened under artificial epiphytotic conditions for FW and SMD diseases in alpha lattice design with two replications in two environments i.e., GKVK and ICRISAT. ANOVA results depicted significant differences between genotypes, environments and genotype $\times$ environment interaction effects. The genotypes were grouped into different disease response groups, based on the calculated PDI. The genotypes ICP 11259 and ICP 16264 showed combined and stable resistance over environments. The multivariate statistical technique using Ward's method was used to substantiate the genetic diversity and four clusters were formed from the experimental material; cluster I with 14 genotypes, cluster II with 40 genotypes, cluster III with 40 and cluster IV with 13 genotypes. The cluster II recorded lowest mean PDI scores. The genotypes from this cluster are cardinal to develop FW and SMD disease resistant cultivars in pigeonpea.

Keywords : Fusarium wilt, Sterility mosaic disease, Resistance, Variability, Clusters

Introduction

Pigeonpea [*Cajanus cajan* (L.) Millsp.], a versatile grain legume cultivated in the tropical and subtropical regions of the world is a rich source of vegetable protein with ~20% crude protein (Saxena, 2008). It is consumed in both vegetable and dal form, wherein it is said that the nutrient quotient of green forms is more than that of the dal (Saxena *et al.*, 2010). Pigeonpea is cultivated in an area of 6.09 million hectares yielding 5.01 million tonnes with an average yield of 822 kg ha⁻¹ (FAOSTAT, 2022). India is the predominant producer of pigeonpea worldwide, accounting for over 75% of global production (FAOSTAT, 2022). Though India is the leading producer of pigeonpea, the productivity is low compared to the global average. This yield stagnation

due to low productivity is due to various biotic and abiotic stresses that affect the yield of pigeonpea.

Among the biotic stresses, fusarium wilt and sterility mosaic disease pose significant challenges to pigeonpea production. Fusarium wilt (FW) caused by the fungal pathogen, *Fusarium udum* Butler is a vascular wilt disease predominant in Indian sub continent (Jain and Reddy, 1995). It is proven to be one of the most devastating diseases of pigeonpea. Remarkably, the annual crop loss due to FW alone reports a monetary loss of approximately US\$ 71 million in India (Kannaiyan and Nene, 1981; Dhar *et al.*, 2012). This soil-borne pathogen enters through the openings such as wounds and clogs the xylem vessels with mycelia, spores and polysaccharides produced by the fungus (Purohit *et al.*, 2017). The black streaks are

formed on the xylem, leading to visible brown to purple bands on the stem of partially wilted plants (Reddy *et al.*, 1993).

Another devastating disease sterility mosaic disease (SMD) is a viral disease caused by *Pigeonpea sterility mosaic virus (PPSMV)*, leading to 100% yield loss. Eriophyid mite (*Aceria cajani* Channabasavanna) transmits virus in a semi-persistent manner. The estimated monetary loss due to SMD disease in the Indian subcontinent approximates US\$ 300 million (Reddy *et al.*, 1998). Patches of light green plants with excessive vegetative growth appear in the field which spreads profusely under favourable conditions thereby known as “green plague” (Jones *et al.*, 2004). The severity of symptoms depends on the stage of infection: complete sterility and cessation of flowering occur when <45 days old plants are infected. When the SMD infection is noticed for >45 days old plants, it results in mild mosaic and reduced flowering symptoms (Kannaiyan *et al.*, 1984).

Various fungicides are employed to control FW disease, while different acaricides are used to prevent SMD infection by eliminating the mites that spread the virus. However these chemical approaches are costly and pose significant negative effect on environment. Deploying disease resistant cultivars is the most economical, ecologically safe and effective method. The presence of good variability for the disease response of FW and SMD will provide better opportunities for selection of better-performing genotypes (Bhatt *et al.*, 2024). Higher estimates of descriptive statistics parameters such as heritability, genetic advance as percentage mean, GCV and PCV (genotypic and phenotypic coefficients of variation) is desirable (Patil *et al.*, 2015, Mukherjee *et al.*, 2025). Considering the FW and SMD disease reaction, the genotypes will be grouped into clusters (Yang *et al.*, 2014). The present investigation was conducted to

study the genetic variability and identify genotypes for FW and SMD resistance in the panel of diverse pigeonpea genotypes.

Material and Methods

Experimental material

A diverse panel consisting of 100 pigeonpea genotypes was procured from the Genebank at ICRISAT, Hyderabad. The accessions were selfed for two generations. The resulting selfed genotypes were evaluated for FW and SMD disease resistance in the current investigation. The list of pigeonpea genotypes used to study the genetic variability is presented in Supplementary Table 1.

Experimental field and design

The experimental material was screened for FW and SMD disease reaction in two diverse environments viz., GKV, Bangalore and ICRISAT, Hyderabad. The weather parameters prevailing during the crop growth period are briefly presented in Table-1. The experiment was laid out in alpha lattice design with two replications in both environments. Each test genotype was planted in a 4 m long row with seed-to-seed spacing of 10 cm within rows and row-to-row spacing of 75 cm. All the recommended practices were followed to raise the crop apart from crop protection measures, such that disease pressure is not disturbed.

Disease screening for FW disease response

The diverse panel was assessed in a wilt sick plot under controlled epidemic conditions at GKV and ICRISAT. The wilt pathogen was maintained at a consistent level of 5×10^5 conidia m^{-2} by adding chopped wilted pigeonpea plants to the plot annually (Nene *et al.*, 1981). A susceptible cultivar ICP 2376 was used as a check and was planted after every 10 rows of test genotypes to act as both indicator and infector rows.

Table 1 : Weather parameters prevailing during the crop period

Details	GKV, Bangalore	ICRISAT, Hyderabad
Agro-climatic zone	Eastern dry zone of Karnataka (Zone 5)	Southern Telangana Zone of Telangana
Latitude	13° 05'N	17° 32'N
Longitude	77° 34'E	78° 16'E
Altitude	924 meters above mean sea level	545 meters above mean sea level
Annual average rainfall	915.8 mm	615 mm
Minimum temperature	17.0°C	8.6°C
Maximum temperature	32.0°C	40.2°C
Minimum relative humidity	37%	27%
Maximum relative humidity	96%	98%

Disease screening for SMD disease reaction

Artificial epiphytotic condition for SMD was created in the fields to screen the diverse panel of pigeonpea genotypes. SMD infected pigeonpea leaves harbouring viruliferous mites (*Aceria cajani*) were folded over the primary leaf of the test accession such that the lower side bearing mites come in contact with the primary leaf and it was then stapled using a small paper stapler to ensure successful infection with SMD virus. The susceptible cultivar ICP 8863 was planted for every tenth row to serve as a susceptible check and to maintain sufficient disease pressure.

Data collection

The number of FW and SMD infected plants were recorded at three growth stages i.e., seedling, flowering, and maturity stages at each environment. Total disease incidence for each disease across these stages was calculated using the following formula.

Per cent Disease Incidence (PDI)

$$= \left(\frac{\text{Number of infected plants}}{\text{Total number of plants}} \right) \times 100$$

Based on the calculated PDI for FW and SMD incidence, the test genotypes were classified into four disease response groups. The disease response groups formed based on disease PDI were resistant (<10.0% incidence), moderately resistant (10.1–20.0% incidence), moderately susceptible (20.1–50.0% incidence) and susceptible (>50% incidence). The PDI was transformed to normalize residuals using arcsine transformation (Gomez and Gomez, 1984). The transformed data was further used for analysis.

Statistical Analysis

The pooled analysis of variance (ANOVA) for GKV and ICRISAT environments was performed using random-effects model in R software ver. 4.4.1 (Chambers, 2008). Further one-way ANOVA was computed to know the significance/non-significance of differences between the disease response groups which justifies the grouping of disease response groups based on PDI. One-way ANOVA was computed in Microsoft Excel. Genetic variability parameters such as mean, variance, GCV, PCV, ECV, heritability and GAM were computed using the “variability” package in R software. One of the important variability parameter, heritability over environments was estimated using the following formula,

$$h^2_{(bs)} = \frac{\sigma_g^2}{\left(\sigma_g^2 + \frac{\sigma_{ge}^2}{s} + \frac{\sigma_r^2}{rs} \right)}$$

Where, σ_g^2 , σ_{ge}^2 and σ_r^2 are the genotypic variance, genotype $\times$ environment variance and residual variance, respectively; ‘r’ is the number of replications and ‘e’ is the number of environments.

Data standardization was done using the Euclidean approach to calculate the genetic distance matrix. Subsequently, hierarchical clustering was carried out using Ward’s distance method (Randriamihison *et al.*, 2021). Dendrograms were developed using Euclidean distances to explore the relationships within and between populations/clusters. Cluster analysis of 100 pigeonpea genotypes was conducted using the R statistical packages “dendextend” and “circlize” in R ver. 4.4.1. The significance of differences between the clusters was tested using a post-hoc test called Student-Newman-Keuls (SNK) method, which presents an ANOVA to detect the significant differences between the sample means (Carlson, 1975).

Results and Discussion

Combined analysis of variance

The results of pooled analysis of variance over two varied environments are presented in Table-2. Total variation was partitioned into different sources like genotypes, environments, genotype $\times$ environment interaction. Highly significant mean sum of squares for genotypes and environments indicates varied disease reaction of genotypes and cumulative effect of environmental factors on the development and spread of FW and SMD diseases (Sharma *et al.*, 2012a). Subsequently, genotype $\times$ environment interaction effects were found highly significant ($p=0.001\%$) envisaging that FW and SMD disease reaction of genotypes varied with environments. Genotypes with resistant response at one environment showed susceptibility reaction in other environments. The results obtained aligned with previous reports by Sharma and Pande, 2011.

The varied contribution of different sources towards the total variation tells the role of genotypes, environment and genotype $\times$ environment interaction to the disease response. For FW and SMD diseases, the responses were majorly contributed by the following components; genotypes (71.91% for FW; 66.72% for SMD) and G $\times$ E interaction (18.23% for FW; 25.41% for SMD). This signifies the confounding effect of genotypes on the FW and SMD disease response over environments. The resistance is the function of genotypes and the profound effect of genotypes on disease resistance was previously reported by Sharma *et al.*, 2012b; Sharma *et al.*, 2016; Kimaro *et al.*, 2020.

The genotype effect is followed by genotype × environment interaction effect, which has significant effect on the disease responses. The interaction of genotype with its environment will manifest resistance in certain genotypes. Consequently, genotypes showing

resistance in one environment may be susceptible in another. The genotype × environment interaction will significantly affect the disease response of genotypes (Sharma *et al.*, 2016).

Table 2: Pooled analysis of variance for alpha lattice design over two diverse environments

Source of variation	Df	FW			SMD		
		SS	MSS	Variation	SS	MSS	Variation
Environments	1	14294	14293***	6.7%	11799	11798***	6.2%
Replication (Environments)	2	822	410.8	0.38%	462	230.9	0.06%
Blocks (Environments × Replications)	16	1440	90.0	2.03%	1312	82	1.40%
Genotypes	99	155813	1573***	71.91%	128872	1301***	66.72%
Genotype × Environment	99	38437	388.3***	18.23%	47855	483.4***	25.41%
Residuals	182	30393	167		17383	95.5	

*, ** and *** Significant at 5%, 1% and 0.01% probability, respectively; df = Degrees of freedom; FW = Fusarium wilt; SMD = Sterility mosaic disease; SS = Sum of squares; MSS = Mean sum of squares.

Analysis of variance for FW and SMD disease response groups of pigeonpea genotypes

The classified FW and SMD disease response groups were subjected to analysis of variance. Significance of mean sum of squares between disease response groups justified the classification of genotypes into four different disease response groups.

It highlighted that there existed notable difference in disease response groups among the genotypes that were screened for FW and SMD disease resistance. For the hybridization programme, the parents can be selected between the disease response groups to have contrasting disease reaction as reported by Sanjeev and Onkarappa, 2018.

Table 3 : Analysis of variance for FW and SMD disease response groups of pigeonpea genotypes

Source of variation	Degrees of freedom	FW		SMD	
		SS	MSS	SS	MSS
Between response groups	3	137949.1	45983.03***	149123.9	49707.97**
Within response group	196	33769.00	172.2908	18576.32	94.77
Total	199	171718.00		167700.22	

*, ** and *** Significant at 5%, 1% and 0.01% probability, respectively; df = Degrees of freedom; FW = Fusarium wilt; SMD = Sterility mosaic disease; SS = Sum of squares; MSS = Mean sum of squares.

Disease reaction of pigeonpea genotypes for FW and SMD diseases under artificial epiphytotic conditions

The diverse panel of 100 pigeonpea genotypes showed varied reaction to FW and SMD diseases. Based on the PDI, genotypes were categorized into four disease response groups in two environments. The FW and SMD disease reaction of pigeonpea genotypes used in the study are presented in Supplementary Table-2. The number of genotypes in disease response groups varied with the particular disease and the environment as depicted in Figure-1. It emphasizes the role of the environment and genotype × environment interaction on the development of the diseases. In GKVK environment, 3 and 21 genotypes were

resistant; 5 and 13 genotypes were moderately resistant; 25 and 28 genotypes were moderately susceptible; 58 and 38 susceptible genotypes for FW and SMD respectively were obtained. The number of genotypes under each category varied with the environment. At ICRISAT environment, three resistant, three moderately resistant, 10 moderately susceptible and 84 susceptible genotypes were noted for FW disease. Similarly, the number of genotypes were 29 resistant, 18 moderately resistant, 34 moderately susceptible and 19 susceptible for SMD disease reactions. The resistant genotypes for FW, SMD and both the diseases were noted to make use of it in further breeding programs.

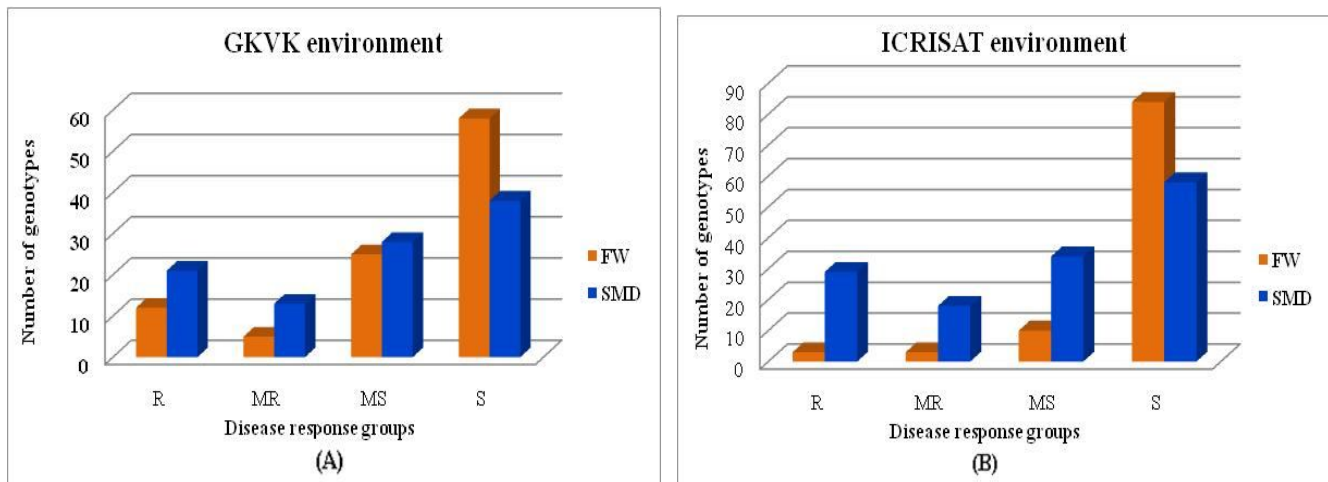


Fig. 1 : Fusarium wilt and sterility mosaic disease response groups over environments: A) GKVK environment B) ICRISAT environment.

Genotypes with resistance to both FW and SMD diseases are vital, as combinedly these two diseases can cause up to 100% crop loss. Combined resistance for both diseases was shown in genotypes such as ICP 10276, ICP 16264, ICP 13304, ICP 11505 and ICP 11259. Genotypes showing low PDI scores for both diseases show combined resistance, which is a prized possession in resistance breeding. It is important to identify stable sources of resistance over environments. The genotypes ICP 11259 and ICP 16264 showed resistance for both FW and SMD diseases over two environments. These genotypes were resistant to both the strains of SMD and *Fusarium* variants. These genotypes can be further studied for their performance and released as cultivars for the two environments and/or can be used as donors for transferring disease resistance to better performing variety (Sharma *et al.*, 2016). Either ways, these genotypes prove to be valuable assets in disease resistance breeding.

Genetic variability studies on FW and SMD disease responses

Genetic variability parameters define a trait and are essential to initiate successful breeding program. Good variability was noticed for FW and SMD disease response in pigeonpea genotypes. A range of resistant and susceptible genotypes were identified from the study. The PDI scores ranging from 0 – 100% was noticed for both FW and SMD disease response with a mean value of 58.78% for FW and 34.49% for SMD disease response (Table-4), indicating presence of good variability in the panel for the traits studied. Genotypic variance was lower than the phenotypic variance for both FW and SMD, indicating the role of environment on the disease response. However, the environmental variance was much lesser than genotypic variance conveying that the role of genetic components was higher on FW and SMD disease response and environmental influence was less (Shinde *et al.*, 2010; Divyadarshini *et al.*, 2016 and Suma *et al.*, 2025).

Table 4 : Genetic variability parameters for FW and SMD disease response in pigeonpea genotypes

Genetic parameters	SMD	FW
Maximum	100	100
Minimum	0	0
Mean	34.49	58.78
Standard error of the mean	11.45	11.91
Environmental variance	262.38	284.16
Genotypic variance	519.67	644.85
Phenotypic variance	782.06	929.017
ECV	46.96	28.67
GCV	66.09	43.19
PCV	81.08	51.84
Heritability (%)	66	69
Genetic advance as a percentage mean	68.28	74.13
CV	2.03	1.52

FW = Fusarium wilt; DMS = Sterility mosaic disease; ECV, PCV and GCV are environmental, phenotypic and genotypic coefficient of variation; CV = coefficient of variation.

GCV and PCV depict the variation present for the studied traits in the genetic material. The PCV and GCV estimates were 51.84 and 43.19 for FW; 81.08 and 66.09% for SMD disease response, respectively. GCV is lesser than PCV and greater than ECV. It tells that the traits were majorly contributed by genetic factors, while environment did have an effect on the trait but the effect was smaller in magnitude (Vange and Egbe, 2009; Oyiga and Uguru, 2011; Mahiboobsa *et al.*, 2012).

Heritability was studied as one of the important parameters for the improvement of a trait. Robinson *et al.* (1949) categorized the heritability values into low (30%), moderate (30 – 60%) and high (>60%). The estimates of heritability were 69% for FW and 66% for SMD disease response, emphasizing on the high heritable nature of the two diseases. These high estimates of heritability endorse the idea of selection based on phenotypic performance. Similarly, higher heritability estimates were noted by Patel and Patel, 1998; Linge *et al.*, 2010 for yield traits in pigeonpea. According to the GAM, genotypes are categorized into three groups: low (<10%), moderate (10 – 20%) and high (>20%) (Johnson *et al.*, 1955). GAM calculated for PDI was high for both FW (74.13%) and SMD (68.28%). A combination of substantial genetic advance and high heritability provides optimal conditions for selection; resulting in a more dependable indicator of selection values (Johnson *et al.*, 1955).

Thereby deciphers the gene action underlying the trait. FW and SMD disease response had high GAM coupled with high heritability values, which provided a more reliable index for selection value and additive gene action underlies the inheritance of the traits. High heritability and GAM values for yield and its attributing traits were previously reported in pigeonpea by researchers such as Saroj *et al.*, 2013; Gaur *et al.*, 2020; Vanniarajan *et al.*, 2023.

Cluster analysis

The hierarchical clustering is a learning algorithm to group similar genotypes/objects into same cluster and dissimilar genotypes/objects into different clusters. This visualization tool helps to understand data structure and identify natural groupings among observations (Sarkar *et al.*, 2024). A dendrogram was created from a cluster analysis of 100 pigeonpea genotypes based on the disease response of FW and SMD diseases. Previous researchers like Katiyar *et al.* (2004) and Pratap *et al.* (2011) noted significant genetic variation in pigeonpea. The genetic divergence was quantitatively assessed using Ward's minimum variance method, focusing on FW and SMD disease response. In the current study, the optimum number of clusters was determined to be four using the scree plot by silhouette method (Figure-2). The pigeonpea genotypes were categorized into four clusters and the same was depicted using a circular dendrogram (Figure-3).

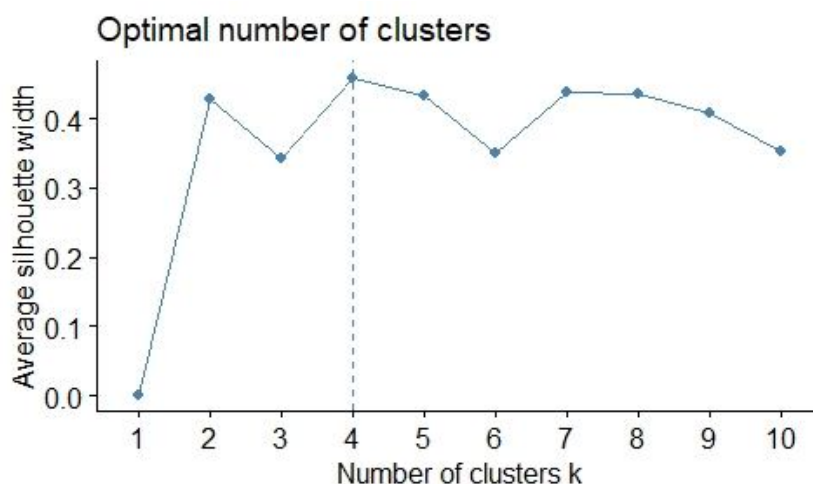


Fig. 2 : Scree plot showing the optimum number of clusters

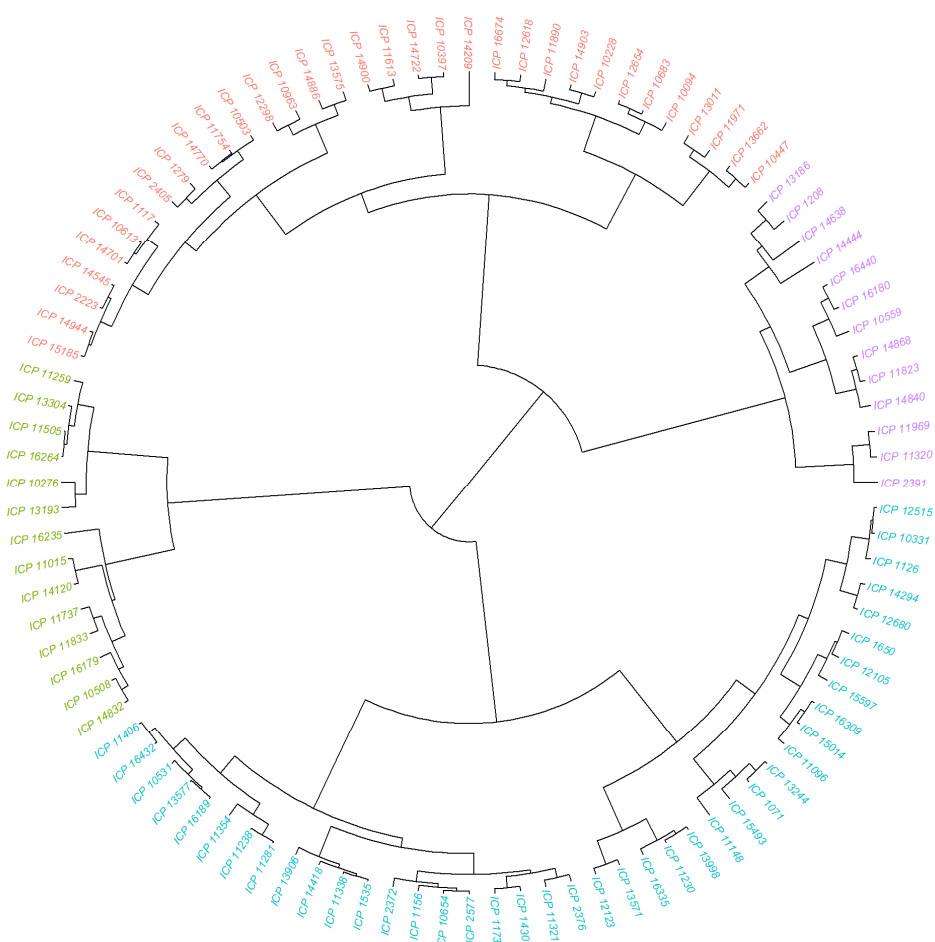


Fig. 3 : Circlize dendrogram depicting the genetic relationship of 100 pigeonpea genotypes pooled over two environments

The cluster size was 33, 14, 40 and 13 for clusters I, II, III and IV respectively. Cluster I had highest number of genotypes among the clusters. The cluster profile of each of the clusters is represented in Table-5, enlisting all the genotypes in a particular cluster. After clustering, measuring the cluster-specific parameters like mean tells about the characteristics of the cluster. The intra-cluster group means for two traits (Table-6)

showed significant variations among the clusters regarding their average values for the different traits. Cluster II had the lowest mean PDI scores for FW (26.23) and SMD (8.86) with superiority for disease resistance (Table-7). The genotype members of this cluster can be used to develop FW and SMD disease resistant cultivars in pigeonpea.

Table 5 : Cluster profile of pigeonpea genotypes

Clusters	Number of genotypes	Genotypes
Cluster I	33	ICP 10963, ICP 1117, ICP 11613, ICP 11754, ICP 11890, ICP 11971, ICP 12298, ICP 12618, ICP 12654, ICP 1279, ICP 13011, ICP 13575, ICP 13662, ICP 14209, ICP 14545, ICP 14701, ICP 14722, ICP 14770, ICP 14886, ICP 14900, ICP 14903
Cluster II	14	ICP 10276, ICP 10508, ICP 11015, ICP 11259, ICP 11505, ICP 11737, ICP 11833, ICP 13193, ICP 13304, ICP 14120, ICP 14832, ICP 16179, ICP 16235, ICP 16264
Cluster III	40	ICP 10331, ICP 10531, ICP 10654, ICP 1071, ICP 11096, ICP 11148, ICP 11230, ICP 11238, ICP 1126, ICP 11281, ICP 11321, ICP 11338, ICP 11354, ICP 11406, ICP 1156, ICP 11738, ICP 12105, ICP 12123, ICP 12515, ICP 12680, ICP 13244, ICP 13571, ICP 13577, ICP 13906, ICP 13998, ICP 14294, ICP 14303, ICP 14418, ICP 15014, ICP 1535, ICP 15493, ICP 15597, ICP 16189, ICP 16309, ICP 16335, ICP 16432, ICP 1650, ICP 2372, ICP 2376, ICP 2577
Cluster IV	13	ICP 10559, ICP 11320, ICP 11823, ICP 11969, ICP 1208, ICP 13186, ICP 14444, ICP 14638, ICP 14840, ICP 14868, ICP 16180, ICP 16440, ICP 2391

Table 6 : Estimates of inter and intra cluster distance among different cluster centroids in pigeonpea over two environments.

Clusters	I	II	III	IV
I	10.69	73.92	39.20	51.21
II		10.44	55.98	61.38
III			10.85	64.91
IV				11.84

Table 7 : Cluster means for FW and SMD disease response over two environments for pigeonpea genotypes

Clusters	FW	SMD
Cluster I	82.67	54.16
Cluster II	26.23	8.86
Cluster III	79.05	19.98
Cluster IV	36.74	66.68

Two genotypes ICP 11259 and ICP 16264 showing combined resistance for FW and SMD diseases in two environments were grouped in cluster II. This suggests that the disease resistance observed in these genotypes may have been introgressed from the same or a closely related ancestor (Saxena *et al.*, 2010). Similarly, the intra-cluster means for different quantitative traits were previously reported by Sawant *et al.*, 2009; Pratap *et al.*, 2011. Inter and intra cluster distance was calculated for four clusters. The maximum amount of inter cluster distance (73.92) was found between cluster I and II, which signifies greater genetic diversity between clusters. Likewise, substantial genetic divergence in the population was delineated by Satapathy and Panigrahi, 2014; Zavinon *et al.*, 2019; Ranjani *et al.*, 2021 in pigeonpea. This can be exploited by hybridization of genotypes between the clusters to implement heterosis breeding or made use of recombinant breeding.

Conclusion

In the current investigation, 100 pigeonpea genotypes were screened for FW and SMD disease response in two locations viz., GKVK and ICRISAT. The results revealed highly significant differences for genotypes, environments and genotype × environment interaction effects. Based on the FW and SMD PDI, genetic variability parameters were calculated. Higher values of heritability and GAM indicated the effectiveness of phenotypic selection and their inheritance was majorly controlled by additive gene action. Combined resistance for both diseases was shown in genotypes such as ICP 10276, ICP 16264, ICP 13304, ICP 11505 and ICP 11259. Notably, ICP 11259 and ICP 16264 showed consistent resistance to both FW and SMD across both GKVK and ICRISAT environments, making them valuable stable sources of resistance. The

cluster analysis was performed using Ward's method, which categorized the diverse panel into four clusters. Cluster II with lowest mean PDI provides prospects for improving the FW and SMD disease resistance in pigeonpea. Conversely, the genotypes from divergent clusters can be selected for hybridization programme to exploit greater amount of hybrid vigour. Conclusively the study provides an overview of the genetic variability and the stable resistant sources can be further used to develop FW and SMD disease resistant cultivars.

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Supplementary Table 1 : List of pigeonpea genotypes used in the study

Sl. No.	Genotypes	Sl. No.	Genotypes	Sl. No.	Genotypes	Sl. No.	Genotypes
1	ICP 10094	26	ICP 11321	51	ICP 13186	76	ICP 14900
2	ICP 10228	27	ICP 11338	52	ICP 13193	77	ICP 14903
3	ICP 10276	28	ICP 11354	53	ICP 13244	78	ICP 14944
4	ICP 10331	29	ICP 11406	54	ICP 13304	79	ICP 15014
5	ICP 10397	30	ICP 11505	55	ICP 13571	80	ICP 15185
6	ICP 10447	31	ICP 1156	56	ICP 13575	81	ICP 1535
7	ICP 10503	32	ICP 11613	57	ICP 13577	82	ICP 15493
8	ICP 10508	33	ICP 11737	58	ICP 13662	83	ICP 15597
9	ICP 10531	34	ICP 11738	59	ICP 13906	84	ICP 16179
10	ICP 10559	35	ICP 11754	60	ICP 13998	85	ICP 16180
11	ICP 10613	36	ICP 11823	61	ICP 14120	86	ICP 16189
12	ICP 10654	37	ICP 11833	62	ICP 14209	87	ICP 16235
13	ICP 10683	38	ICP 11890	63	ICP 14294	88	ICP 16264
14	ICP 1071	39	ICP 11969	64	ICP 14303	89	ICP 16309
15	ICP 10963	40	ICP 11971	65	ICP 14418	90	ICP 16335
16	ICP 11015	41	ICP 1208	66	ICP 14444	91	ICP 16432
17	ICP 11096	42	ICP 12105	67	ICP 14545	92	ICP 16440
18	ICP 11148	43	ICP 12123	68	ICP 14638	93	ICP 1650
19	ICP 1117	44	ICP 12298	69	ICP 14701	94	ICP 16674
20	ICP 11230	45	ICP 12515	70	ICP 14722	95	ICP 2223
21	ICP 11238	46	ICP 12618	71	ICP 14770	96	ICP 2372
22	ICP 11259	47	ICP 12654	72	ICP 14832	97	ICP 2376
23	ICP 1126	48	ICP 12680	73	ICP 14840	98	ICP 2391
24	ICP 11281	49	ICP 1279	74	ICP 14868	99	ICP 2405
25	ICP 11320	50	ICP 13011	75	ICP 14886	100	ICP 2577

Supplementary Table 2 : Disease reaction of 100 pigeonpea genotypes

GENOTYPES	GKVK environment				ICRISAT environment			
	FW		SMD		FW		SMD	
ICP 10094	90.42	S	7.74	R	78.79	S	60.61	S
ICP 10228	84.38	S	34.31	MS	100.00	S	41.25	MS
ICP 10276	13.35	MR	5.26	R	19.47	MR	0.00	R
ICP 10331	40.98	MS	34.71	MS	97.02	S	29.91	MS
ICP 10397	87.87	S	96.45	S	100.00	S	41.41	MS
ICP 10447	94.44	S	93.27	S	100.00	S	9.72	R
ICP 10503	43.75	MS	52.78	S	100.00	S	36.04	MS
ICP 10508	42.71	MS	5.56	R	44.17	MS	26.48	MS
ICP 10531	70.83	S	14.36	MR	100.00	S	5.56	R
ICP 10559	8.71	R	68.89	S	90.57	S	56.67	S
ICP 10613	74.52	S	58.37	S	86.06	S	54.17	S
ICP 10654	95.83	S	3.13	R	100.00	S	25.76	MS
ICP 10683	91.67	S	33.64	MS	88.83	S	33.64	MS
ICP 1071	47.86	MS	48.53	MS	70.06	S	22.50	MS
ICP 10963	60.00	S	96.15	S	87.50	S	29.17	MS
ICP 11015	45.56	MS	13.22	MR	13.25	MR	0.00	R
ICP 11096	86.88	S	58.89	S	35.00	MS	0.00	R
ICP 11148	44.17	MS	79.22	S	69.61	S	18.33	MR
ICP 1117	57.03	S	98.79	S	100.00	S	21.11	MS
ICP 11230	59.82	S	17.71	MR	90.45	S	6.25	R
ICP 11238	93.03	S	8.50	R	87.19	S	0.00	R
ICP 11259	8.71	R	9.44	R	0.00	R	0.00	R
ICP 1126	46.67	MS	43.53	MS	94.94	S	26.92	MS
ICP 11281	92.31	S	10.51	MR	87.46	S	0.00	R
ICP 11320	54.62	S	97.78	S	32.14	MS	74.11	S
ICP 11321	95.61	S	32.39	MS	100.00	S	6.85	R
ICP 11338	96.15	S	21.50	MS	74.11	S	18.73	MR
ICP 11354	93.75	S	0.00	R	100.00	S	0.00	R
ICP 11406	89.58	S	14.36	MR	83.33	S	11.11	MR
ICP 11505	1.12	R	3.33	R	13.64	MR	0.00	R
ICP 1156	100.00	S	10.71	MR	100.00	S	20.93	MR
ICP 11613	97.50	S	89.76	S	75.98	S	67.55	S
ICP 11737	42.86	MS	8.33	R	50.00	S	10.90	R
ICP 11738	86.67	S	19.38	MR	100.00	S	15.18	MR
ICP 11754	62.14	S	34.38	MS	82.91	S	60.68	S
ICP 11823	26.04	MS	82.99	S	45.99	MS	21.79	MS
ICP 11833	15.00	MR	3.85	R	67.65	S	8.83	R
ICP 11890	76.67	S	44.85	MS	86.43	S	35.00	MS
ICP 11969	33.03	MS	88.54	S	53.55	S	92.11	S
ICP 11971	100.00	S	58.33	S	100.00	S	53.57	S
ICP 1208	6.90	R	63.05	S	45.30	MS	80.13	S
ICP 12105	46.61	MS	29.41	MS	87.79	S	14.65	MR
ICP 12123	42.08	MS	4.17	R	82.26	S	12.13	MR
ICP 12298	51.36	S	89.49	S	92.53	S	48.75	MS
ICP 12515	38.10	MS	34.62	MS	98.58	S	33.94	MS
ICP 12618	88.54	S	37.78	MS	83.33	S	47.22	MS
ICP 12654	88.07	S	30.80	MS	90.91	S	31.82	MS
ICP 12680	70.83	S	27.08	MS	83.22	S	32.87	MS
ICP 1279	46.88	MS	46.88	MS	87.76	S	58.74	S
ICP 13011	91.18	S	97.68	S	100.00	S	22.22	MS
ICP 13186	0.00	R	72.92	S	68.06	S	79.17	S
ICP 13193	7.74	R	19.23	MR	27.78	MS	5.69	R
ICP 13244	42.56	MS	63.07	S	71.43	S	7.14	R

GENOTYPES	GKVK environment				ICRISAT environment			
	FW		SMD		FW	MR	SMD	
ICP 13304	0.00	R	3.33	R	20.00	MR	0.00	R
ICP 13571	61.65	S	15.34	MR	68.75	S	6.25	R
ICP 13575	46.88	MS	72.85	S	83.33	S	47.22	MS
ICP 13577	87.08	S	0.00	R	87.50	S	18.19	MR
ICP 13662	96.88	S	63.97	S	100.00	S	31.75	MS
ICP 13906	75.96	S	48.13	MS	100.00	S	0.00	R
ICP 13998	79.81	S	4.17	R	74.55	S	20.09	MR
ICP 14120	7.50	R	8.01	R	54.44	S	0.00	R
ICP 14209	100.00	S	90.84	S	65.00	S	91.43	S
ICP 14294	87.50	S	46.88	MS	72.27	S	10.29	R
ICP 14303	87.29	S	4.17	R	93.24	S	30.56	MS
ICP 14418	94.50	S	7.14	R	74.17	S	38.64	MS
ICP 14444	0.00	R	88.33	S	38.57	MS	14.57	MR
ICP 14545	71.88	S	79.17	S	93.54	S	27.62	MS
ICP 14638	9.63	R	56.70	S	10.00	S	92.62	S
ICP 14701	96.43	S	42.73	MS	61.25	S	72.50	S
ICP 14722	85.67	S	97.95	S	100.00	S	45.00	MS
ICP 14770	47.22	MS	49.41	MS	92.31	S	45.10	MS
ICP 14832	15.92	MR	15.88	MR	66.67	S	11.78	MR
ICP 14840	10.45	R	55.21	S	71.15	S	38.85	MS
ICP 14868	39.08	MS	59.65	S	39.58	MS	45.83	MS
ICP 14886	35.38	MS	54.17	S	87.38	S	75.96	S
ICP 14900	89.18	S	93.03	S	79.29	S	55.71	S
ICP 14903	86.36	S	63.33	S	100.00	S	19.24	MR
ICP 14944	82.50	S	88.10	S	76.67	S	13.48	MR
ICP 15014	45.00	MS	24.05	MS	80.36	S	26.79	MS
ICP 15185	96.34	S	90.63	S	66.56	S	7.41	R
ICP 1535	74.51	S	15.26	MR	93.90	S	26.54	MS
ICP 15493	56.67	S	44.79	MS	70.83	S	36.11	MS
ICP 15597	46.88	MS	35.38	MS	74.17	S	0.00	R
ICP 16179	14.96	MR	14.87	MR	55.68	S	13.92	MR
ICP 16180	34.52	MS	62.50	S	44.62	MS	63.19	S
ICP 16189	74.17	S	6.25	R	100.00	S	9.26	R
ICP 16235	35.90	MS	48.72	MS	34.27	MS	8.39	R
ICP 16264	6.67	R	3.13	R	9.55	R	0.00	R
ICP 16309	57.72	S	35.50	MS	71.79	S	16.03	MR
ICP 16335	67.92	S	30.59	MS	89.01	S	0.00	R
ICP 16432	96.32	S	24.26	MS	75.00	S	5.00	R
ICP 16440	19.17	MR	71.79	S	66.67	S	50.76	MS
ICP 1650	42.08	MS	10.00	R	89.64	S	23.78	MS
ICP 16674	100.00	S	90.42	S	80.00	S	0.00	R
ICP 2223	77.50	S	43.08	MS	85.71	S	60.39	S
ICP 2372	90.99	S	8.12	R	100.00	S	12.05	MR
ICP 2376	96.88	S	30.79	MS	100.00	S	17.78	MR
ICP 2391	34.86	MS	94.44	S	72.22	S	61.11	S
ICP 2405	51.04	S	76.19	S	94.44	S	31.94	MS
ICP 2577	100.00	S	11.44	MR	93.22	S	14.65	MR