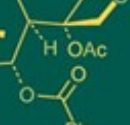
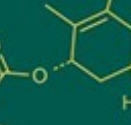
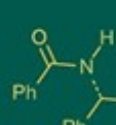


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Integrated field and greenhouse assessment of phenotypic variation and anthracnose resistance in diverse soybean germplasm

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ABSTRACT

A comprehensive investigation was conducted to evaluate anthracnose disease response among 40 soybean genotypes under field and greenhouse conditions. The genotypes were evaluated for plant survival rate (PSR), percentage disease incidence (PDI), and percentage disease severity (PDS), followed by disease reaction classification and comparative analysis between environments. Considerable variation was found among genotypes for all three disease-related traits, indicating substantial differences in their response to anthracnose infection. Field to greenhouse association was strong for PSR, PDI and PDS, with Pearson correlation coefficients of 0.93, 0.94 and 0.93, respectively, indicating consistent genotype responses across environments. Based on disease severity, several genotypes exhibited resistance in both environments, whereas others showed moderate resistance, moderate susceptibility or susceptibility with environment-dependent responses. The results identified promising soybean genotypes with stable resistance to anthracnose, providing valuable genetic resources for resistance breeding and further investigation of the mechanisms underlying soybean *Colletotrichum* interactions.

Keywords: severity, environments, interaction, resistant, susceptible

Introduction

Soybean is one of the world's most valuable and versatile legume crops. It has recognized for its high nutritional importance. It is widely cultivated across diverse agro-environments under various management practices and serves a broad range of food, feed, and industrial purposes. In India soybean cultivation has expanded from 6.42 million ha in 2000 to 12.80 million ha in 2026 with production increasing from 5.28 million tons to 13.50 million tons, while productivity fluctuated across years and increased from 822.21 kg ha⁻¹ in 2000 to 1,050 kg ha⁻¹ in 2026 with the highest area of 13.26 million ha recorded in 2023 with the highest production of 15.27 million tons in 2024 and the highest productivity of 1,352.95 kg ha⁻¹ achieved in 2012 [28]. Soybean production in India remains geographically concentrated in Madhya Pradesh, Maharashtra and Rajasthan. Madhya Pradesh consistently remained a major production center with soybean area ranging from 5.01 to 6.50 million ha and production from 4.61 to 7.80 million tons. Maharashtra exhibited a marked expansion in cultivated area increasing from 3.01 million ha in 2011-12 to 5.00 million ha in 2025-26 with production varied between 2.06 and 7.37 million tons. Rajasthan maintained contribution in soybean area ranging from 0.89 to 1.20 million ha and production from 0.52 to 1.47 million tonnes. Telangana, Karnataka, Gujarat and Chhattisgarh were additional important soybean producing states [28]. *Glycine gracilis* represents an evolutionary product of soybean domestication. There are no gene flow from *Glycine max* to either *Glycine gracilis* or *Glycine soja* but detected significant gene flow from *Glycine soja* to *Glycine gracilis* and then transfer from *Glycine gracilis* to *Glycine max*. This evidence supports *Glycine soja* as the progenitor of both *Glycine max* and *Glycine gracilis* [13].

Soybean belong to family Fabaceae, genus *Glycine* and subgenus *Soja*. The cultivated soybean, *Glycine max* is believed have been domesticated from the wild annual soybean *Glycine soja* (2n = 40). In addition, the subgenus *Glycine* comprises 26 wild perennial species that are indigenous to Australia and are rich in genes conferring resistance to insect pests and enabling the development of climate-smart genotypes. The growth habit of the soybean plant can be classified as determinate, semi-determinate, or indeterminate in relation to how the plant develops. For the determinate type, the plant's vegetative development comes to an end once flowering has finished, whereas in the indeterminate type, vegetative development and flowering take place at the same time. In the semi-determinate type, the plant does continue to grow for a restricted period following the onset of flowering [5].

Colletotrichum constitutes a genus of ascomycete fungi encompassing hemi biotrophic plant pathogens that induce anthracnose in broad range of spectrum in host species. The infection process involves spore adhesion, appressorium development, and penetration of the host, and this genus occupies eighth place in a sur

vey-based ranking of the most significant fungal plant pathogens. The genus ranks among the ten most economically important fungal pathogens worldwide [11]. Species belonging to the genus *Colletotrichum* are capable of infecting cereals, legumes, vegetables, perennial crops and tree fruits [29]. Anthracnose leaf blight and stalk rot are caused by *C. graminicola* [4]. Anthracnose represents a substantial economic limitation across tropical and subtropical areas, being attributable to multiple species, among them *C. acutatum* and *C. capsici* [29]. In common bean, anthracnose is caused by *C. lindemuthianum*, with isolates of differing geographic provenance displaying variation in pathogenicity and genetic makeup [1], the greatest damage from this disease occurs when temperatures are cool and humidity is high [14]. It is the most important disease across nearly all producing regions, and *C. alienum*, *C. asianum*, *C. fruticola*, *C. siamense* and *C. tropicale*, each belonging to the *C. gloeosporioides* species complex, were linked to disease symptoms in Mexico [30]. The principal causal species is regarded as *C. truncatum*, even though additional species have been reported [7], while isolates from India have likewise been characterized as *C. clivicola* and *C. chlorophyt* [6]. In India, anthracnose attributable to *C. truncatum* ranks among the foremost diseases of soybean [17], making dependable sources of resistance indispensable for the genetic improvement of this crop [33]. The infection process of *Colletotrichum truncatum* is initiated when conidia germinate upon the soybean surface, after which a melanized appressorium differentiates and produces the pressure necessary to penetrate the host. As demonstrated by recent functional studies, the UDP-glycosyltransferase CtYjC plays an indispensable role in melanisation, conidial germination, appressorium formation, and the full virulence of *C. truncatum* on soybean tissues [10]. Once penetration has occurred, the fungus develops close associations with host cells and secretes candidate effector proteins that are thought to promote host colonization and influence cellular defence mechanisms. The pathogen's subsequent proliferation coincides with the induction of antioxidant and phenolic defence responses in soybean, whereas disease progression is additionally governed by isolate-specific virulence [17]. Thus, comprehending these infection-associated processes is essential for identifying durable resistance sources and elucidating the molecular mechanisms governing soybean resistance to anthracnose.

Material and Method

Experimental site and plant material

The experiment was conducted at experimental farm of ICAR-National institute of seed science and technology, Kushmaur, Mau, Uttar Pradesh, India during *kharif* season 2024-25 and disease reaction was validated at green house condition. This institute is located in eastern Uttar Pradesh, situated at about 25.94°N latitude and 83.56°E longitude with an elevation of around 63 meters above mean sea level. The soil at the experimental site is sandy loam to loam. A composite soil sample collected from the experimental field prior to sowing was analyzed at the Crop Physiology Laboratory of the ICAR- NISST, Kushmaur, Mau. The results revealed a soil pH of 6.8-7.2 (slightly acidic to neutral). The greenhouse used for the study was a naturally ventilated polyhouse of 24 m × 10 m dimensions. It was covered with 200-micron UV stabilized transparent polyethylene film on the roof and enclosed with 50-mesh insect-proof nylon net on the sidewalls. The mean day time temperature ranged between 28 °C and 40 °C, and night time temperatures between 24 °C and 30 °C. Relative humidity was maintained at 65-80%. The plants were grown in earthen pots of 30 cm diameter. This study utilized 40 genotypes consisted with some germplasm and some cultivated variety (Table 1). The genotypes were evaluated in unreplicated trial with a row-to-row distance 50 cm and plant to plant distance of 10 cm. Statistical analysis estimated through R studio with suitable R package.

Fungal inoculum preparation

The authenticated characterized fungal virulent fungal pathogen *Colletotrichum truncatum* procured from the Indian Type Culture Collection (ITCC) at the Division of Plant Pathology in Indian Agriculture Research Institute (IARI), New Delhi. The culture media was prepared by the Standardized procedure Ricker and Ricker (1936) [26] at seed pathology laboratory in ICAR-NISST, Mau. Select actively growing 7-12 days old *C. truncatum* cultures on PDA plates that showed dense mycelial growth and sporulation. Maintain incubation at 25 °C ± 2 °C with 12 hours light and 12 hours dark.

Riker, A. J., & Riker, E. S. (1936). Introduction to research on plant diseases. A guide to the principles and practice for studying various plant-disease problems.

Artificial inoculation procedure

Artificial inoculation was applied by using the freshly prepared conidial suspension of *C. truncatum*. The suspension culture was sprayed 13 days before flowering and 15 days before flowering to the development and expression of disease symptom moderately susceptible at reproductive phase.

Disease scoring procedure

Screening of soybean genotypes was conducted during the two *Kharif* season 2024-25 and 2025-26. The disease response was recorded on the basis of plant survival rate (PSR), plant disease incidence (PDI) and plant disease severity (PDS) by selecting random 10 plants in each row. The Plant survival rate was recorded by calculating the proportion of surviving plant in each plot. Percent disease incidence was recorded by the number of diseased plants and it express as percentage. *Colletotrichum truncatum* severity in row was recorded on reproductive stage in term of 0-9 scaling that was given by Mayes and Dattar (1986) [18]. The Plant disease severity was calculated by formula that was given by (Wheeler (1969) [32].

$$\text{PDS (\%)} = \frac{\sum \text{Disease ratings}}{\text{Number of plants scored} \times \text{Maximum disease score}} \times 100$$

Result

The phenotypic observation of 40 soybean genotypes evaluated under field and greenhouse conditions. Under field evaluation, the mean values recorded for plant survival rate (PSR), percent disease incidence (PDI) and percent disease severity (PDS) were 39.6, 63.6 and 37.1%, respectively. PSR spanned from 10.0 to 72.2%, with a standard deviation (SD) of 17.5 and a coefficient of variation (CV) of 44.22%. PDI ranged between 10.0 and 100.0%, exhibiting a mean of 63.6%, an SD of 32.6% and a CV of 51.26%. PDS showed a broad range of 2.4%-100.0%, with a mean of 37.1%, an SD of 32.2% and a CV of 86.71%. Under the green house, the average PSR rose to 47.9%, while the average PDI and PDS were 66.0% and 40.8%, respectively. PSR varied between 8.9 and 98.3%, with an SD of 22.7 and a CV of 47.40%. PDI spanned 8.5 to 100.0%, with an SD of 33.7 and a CV of 51.05%, whereas PDS ranged from 0.2 to 97.6%, with an SD of 34.5 and a CV of 84.63%. The median values of PSR, PDI and PDS rose from 35.1, 75.0 and 33.41% in the field to 45.47, 77.97 and 42.13% under greenhouse conditions, respectively. PDS exhibited the highest CV in both environments, which revealing variation in disease severity (Table II).

Agreement between field and green house evaluation

A strong positive relationship was found between field and greenhouse assessments for all three disease-related traits. Pearson's correlation coefficients between field and greenhouse were 0.93 for PSR, 0.94 for PDI, and 0.93 for PDS, with each significant correlation at $P < 0.001$. The matching 95% confidence intervals were 0.872–0.963 for PSR, 0.888–0.968 for PDI, and 0.869–0.962 for PDS. Spearman's rank correlation coefficients were 0.95 for PSR, 0.85 for PDI, and 0.90 for PDS, all of which were significant at $P < 0.001$. Consequently, the field and greenhouse assessments showed close

agreement for the three disease-related traits, with PDI exhibiting the highest Pearson correlation and PSR the highest rank correlation.

Correlation among disease related traits within environments

A significant correlation was recorded among PSR, PDI and PDS in both environments. Significant associations were observed among PSR, PDI and PDS in both environments. In the field evaluation, PSR showed a significant negative correlation with PDI ($r = -0.526$, $P < 0.001$) and PDS ($r = -0.608$, $P < 0.001$), while PDI showed a strong positive correlation with PDS ($r = 0.805$, $P < 0.001$). The Spearman's rank correlations were -0.535, -0.629 and 0.805, respectively. Under greenhouse conditions, PSR was negatively correlated with PDI ($r = -0.408$, $P = 0.009$) and PDS ($r = -0.540$, $P < 0.001$), whereas PDI showed a strong positive association with PDS ($r = 0.836$, $P < 0.001$). The corresponding Spearman's correlations were -0.374, -0.592 and 0.786, respectively. PDI and PDS consistently showed the strongest positive association in both environments, whereas PSR was negatively associated with both disease incidence and severity.

Genotype-wise validation of anthracnose reaction under field and greenhouse conditions

The comparison of PDS values conducted on a genotype-by-genotype basis revealed pronounced discrepancies in disease response under the field and greenhouse conditions. In both environments, thirteen genotypes consistently displayed a resistant (R) reaction (figure II) 15_KDS-1045_W, GW 104_W, GW 114_W, GW 250_W, GW 256_W, GW 260_BR, GW 264_B, GW 27_BR, GW 291_B, GW 303_W, GW 32_W, GW 39_G and NRC130. In the field, their PDS values spanned from 2.36 to 9.83%, whereas in the greenhouse these values extended from 0.20 to 9.07%. Within this set of genotypes, GW 27_BR exhibited the lowest PDS under both field (2.36%) and greenhouse (0.20%) evaluations. Four genotypes GW 213_W, GW 257_W, GW 284_W, and GW 96_W showed moderately susceptible response. A susceptible (S) reaction was consistently observed in both environments for thirteen genotypes e.g. GW 140_W, GW 143_W, GW 160_W, GW 175_W, GW 179_W, GW 204_W, GW 211_G, GW 273_W, GW 313_W, GW 86_W, GW 89_G, JS 22-12. The result indicated that that ten distinct genotypes demonstrated shifts in their disease reaction that were contingent upon the surrounding environment. The genotypes 1_NRC-86_W shifted from a reaction of resistant when grown under field conditions to moderately resistant within the greenhouse condition, and the genotype JS 20-98 showed a shift that was exactly the same, moving from resistant to moderately resistant. In contrast, GW 102_W, GW 134_W, GW 227_W, and GW 289_B all transitioned from MODERATLY SUSCEPTIBLE to S, whereas GW 252_W moved from moderately resistant to moderately susceptible. The genotype GW 206_W changed from moderately resistant to resistant, GW 274_W changed from Susceptible to moderately susceptible, and GW 320_W changed from moderately susceptible to resistant. The genotype GW 320_W showed a significant reduction in plant disease severity, which is declined from 43.41% under field conditions to 3.95% within the greenhouse. The integrated field and greenhouse assessment distinguished genotypes based on the stability of their anthracnose response across the two experimental environments and pinpointed those genotypes that exhibited repeatedly resistant reactions.

Discussion

This study revealed considerable phenotypic variation in the response to anthracnose among the soybean genotypes with a high degree of concordance between field and greenhouse assessments. *Colletotrichum truncatum* is a major causal agent of soybean anthracnose, even though additional *Colletotrichum* species have likewise been documented as capable of infecting soybean [7, 12, 23]. Anthracnose is capable of damaging various above-ground tissues of the plant and can result in pod infection, premature defoliation, seed infection and yield reduction, which makes dependable sources of resistance essential for soybean improvement programmes [7, 12]. The

broad variation detected for plant survival rate, plant disease incidence and plant disease severity suggests to significant differences among the genotypes in how they respond to anthracnose. PDS exhibited the greatest CV under both field (86.71%) and greenhouse (84.63%) conditions, which reflects considerable phenotypic differentiation in disease severity. Comparable variation in the soybean response to *C. truncatum* has been mentioned in resistance screening investigations, which reinforces the value of phenotypic diversity for selecting contrasting sources of resistance [22, 34]. This variation provides appropriate material for subsequent genetic and molecular characterization. The strong correlations between field and greenhouse assessments provide additional confirmation of the validity of the screening approach. Pearson's correlation coefficients reached 0.93 for PSR, 0.94 for PDI and 0.93 for PDS, whereas Spearman's correlations were 0.95, 0.85 and 0.90, respectively. These findings suggest that the majority of genotypes maintained their relative disease response across both environments. Significant associations between controlled and field-based disease evaluations in previous investigation in soybean, which illustrates the value of controlled screening for predicting field disease response [3, 8, 31]. Controlled inoculation has similarly been employed with success to distinguish soybean genotypes for anthracnose resistance. Distinct reactions among soybean cultivars and germplasm were recorded following greenhouse inoculation and field evaluation with *Colletotrichum truncatum*, indicating the value of controlled assays for resistance screening [9, 21, 33]. More recently, appreciable variation in soybean responses to virulent *C. truncatum* isolates has been reported with multiple resistant germplasm sources identified [17]. These results lend support to the combined application of field and greenhouse evaluations in the present study.

The pronounced correlation between field and greenhouse condition for PDS ($r = 0.93$) is particularly relevant, since PDS provides a quantitative measure of disease progression. Quantitative disease phenotyping is able to differentiate partial, moderate and strong resistance more effectively than categorical scoring alone, thus provides valuable information for resistance breeding [16]. These findings support that PDS as a valuable trait for identifying contrasting anthracnose responses in soybean. Environmental conditions can influence diseases expression. Fluctuations in temperature, rainfall, humidity, canopy microclimate and inoculum distribution may alter disease development in the field, whereas greenhouse conditions provide relatively greater environmental control [24, 25]. As a result, the strong correlations found in this study suggest general consistency between the two environments, whereas the genotype-specific shifts imply that environmental factors affect disease expression. The genotype-wise comparison recognized thirteen genotypes that maintained a resistant response in both environments. The consistently low PDS values observed for these genotypes reinforce that their resistant phenotype is reproducible. NRC130 reported as resistant to a highly virulent *C. truncatum* isolate, which provides independent corroboration of the resistance reported in the present study [17]. Soybean germplasm had genetically valuable sources of anthracnose resistance [22]. GW 27_BR had the lowest PDS with values of 2.36% in the field and 0.20% under greenhouse conditions. When low disease severity stay across multiple environments provides stronger evidence of reproducible resistance than a low disease value recorded under a single screening condition. Similar multi-environment strategies have been used to identify soybean genotypes with stable resistance to other diseases [20, 24]. GW 27_BR represents promising candidate for additional validation of anthracnose resistance. Four genotypes, namely GW 213_W, GW 257_W, GW 284_W and GW 96_W stayed moderately susceptible in both environments. Their stable intermediate response could imply partial or quantitative resistance instead of than complete resistance. Quantitative resistance can provide useful and potentially more durable protection, since it may entail multiple genetic factors instead of a single major resistance mechanism [19]. Genetic variation for anthracnose resistance in soybean further accentuates the potential value of such intermediate phenotypes for breed

ing and genetic investigations [22]. The consistently susceptible genotypes provided contrasting phenotypes for later molecular studies. Resistant and susceptible soybean genotypes have been successfully used to study differential defence responses during interaction with *C. truncatum* [34]. Thus, the differing phenotypes found in this study may provide as valuable resources for comparative histological, biochemical, and gene-expression studies. A number of genotypes showed environment dependent change like 1_NRC-86_W and JS 20-98 moved from resistant in field to moderately resistant in greenhouse, GW 206_W moderately resistant from to resistant, GW 102_W, GW 134_W, GW 227_W and GW 289_B from moderately susceptible to susceptible, GW 252_W from moderately resistant to moderately susceptible, GW 274_W from susceptible to moderately susceptible. GW 320_W from moderately susceptible to resistant which concurred with reports of the effect of the environment on resistance expression and genotype ranking in soybean disease screening [20, 24, 25]. Such shifts suggested the occurrence of a quantitative disease response influenced by the environments in a number of genotypes.

GW 320_W showed the most pronounced shift with a PDS falling from 43.41% (field) to 3.95% (greenhouse), which implied a strong effect of the environment. However, this greenhouse response should not be interpreted as evidence of complete genetic resistance since field and greenhouse environments differ in term of disease pressure emphasizing the need to complement environments in phenotyping to obtain reliable resistance estimates [2, 16]. GW 134_W recorded the opposite trend with a plant disease severity rising from 34.31% (field) to 61.46% (greenhouse) possibly as a result of variations in temperature, moisture, inoculum pressure and other factors that affect pathogen development, which conformed to earlier reports on genotype-specific environmental [20, 24]. Such shifts thus provide an environment dependent disease expression rather than a definitive gain or loss of genetic resistance. The diversity of pathogens may also contribute to the variation in the expression of anthracnose. The different responses of soybeans to *C. truncatum* genotypes and isolates suggested the occurrence of host pathogen interaction during the development of the disease [12] and sufficient differences in virulence among isolates, plus isolate $\times$ genotype interactions, have been reported in recent studies [17] alongside genetic diversity among soybean associated *Colletotrichum* isolates in India [6]. The field and greenhouse results supported a hierarchical resistance screening approach with field evaluation capturing the natural disease response. Greenhouse assessment providing a controlled validation, consistent with studies that successfully integrated field, greenhouse and detached-leaf assays as complementary methods [15, 27, 33]. From a breeding perspective, the thirteen resistant genotypes represented the most reliable resistance sources. Nevertheless, resistance observed in just two environments is not enough to establish durable, broad-spectrum resistance, which requires validation across pathogen populations, environments and seasons [19], the intricate nature of soybean-

Colletotrichum interactions also emphasized the need for multi-isolate, multi environment testing before advancing resistance sources in breeding programmes [7]. The resistant and susceptible phenotypes identified in this study also provided suitable appropriate material for molecular characterization since differences in defense responses between resistant and susceptible genotypes during *C. truncatum* infection had been previously reported [15, 34], supporting their use in future histological, biochemical and gene-expression studies. Taken as a whole, the study demonstrated a strong correspondence between field and greenhouse results together with sufficient genotype specific variation towards anthracnose response. The strong correlations among PSR, PDI and PDS suggested to a largely consistent pattern of disease response in multiple environments, even though the genotype specific changes indicated that disease expression depends on the environment. The thirteen resistant genotypes showed reproducible resistance phenotypes that justified further validation. The integrated field greenhouse approach provided a useful framework for selecting stable anthracnose resistance and for identifying contrasting material for molecular characterization.

Conclusion

This study provides an integrated field and greenhouse evaluation of anthracnose resistance across 40 soybean genotypes that provide a validated and reproducible screening for use in resistance breeding. The strong correlation was observed between field and greenhouse evaluations (Pearson $r = 0.93-0.94$ for PSR, PDI and PDS) suggests that greenhouse inoculation constitutes a dependable proxy for field disease response, thus decreasing dependency on lengthy, resource demanding multi-season field testing. Thirteen genotypes, especially NRC130 and GW 27_BR maintained a resistant phenotype in both screening environments, establishing them as reliable and immediately applicable sources of resistance for cultivar improvement. Simultaneously, the environment-dependent changes in reaction identified in genotypes such as GW 320_W and GW 134_W indicate a genotype $\times$ environment component underlying anthracnose expression, an aspect that has attracted only limited attention in previous investigations. Collectively, these findings not only deliver validated resistance sources but also establish a dual-environment screening strategy that can be readily implemented in future soybean improvement programmes, while the contrasting resistant and susceptible genotypes identified provides a natural beginning point for subsequent molecular dissection of anthracnose resistance mechanisms.

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Table 1. List of genotypes used in the present study.

S. No.	Genotypes	S. No.	Genotypes
1	15_KDS- 1045_W	21	GW 260_BR
2	1_NRC-86_W	22	GW 264_B
3	GW 102_W	23	GW 273_W
4	GW 104_W	24	GW 274_W
5	GW 114_W	25	GW 27_BR
6	GW 134_W	26	GW 284_W
7	GW 140_W	27	GW 289_B
8	GW 143_W	28	GW 291_B
9	GW 160_W	29	GW 303_W
10	GW 175_W	30	GW 313_W
11	GW 179_W	31	GW 320_W

(continued on next page)

S. No.	Genotypes	S. No.	Genotypes
12	GW 204_W	32	GW 32_W
13	GW 206_W	33	GW 39_G
14	GW 211_G	34	GW 86_W
15	GW 213_W	35	GW 89_G
16	GW 227_W	36	GW 96_W
17	GW 250_W	37	JS 20-98
18	GW 252_W	38	JS 22-12
19	GW 256_W	39	JS 335
20	GW 257_W	40	NRC130

Table 2. Disease severity scoring scale and disease reaction classification of soybean genotypes against anthracnose.

Disease Rating Scale (Mayes and Dattar, 1989)			
S. No.	Score	Description	Disease Reaction
1	0-1	No visible symptoms	Immune
2	1-2	1 - 10 % plants exhibiting few lesion	Resistant (<i>R</i>)
3	3-4	11- 20 % plants exhibiting brown lesion on leaves and stems	Moderately Resistant (MR)
4	5-6	21 - 50% plants exhibiting lesion leaves, stems and pods	Moderately Susceptible (MS)
5	7-8	51 - 75% plants exhibiting lesion with tissue damage	Susceptible (<i>S</i>)

Table 3. Descriptive statistics

Environment	Trait	Mean	Median	SD	Min	Max	Range	CV
Field_2024-25	PSR	39.61	35.1	17.52	10	72.15	62.15	44.22
Field_2024-25	PDI	63.55	75	32.58	10	100	90	51.26
Field_2024-25	PDS	37.09	33.41	32.16	2.36	100	97.64	86.71
Greenhouse_2025-26	PSR	47.86	45.47	22.68	8.87	98.3	89.43	47.4
Greenhouse_2025-26	PDI	66.04	77.97	33.71	8.54	100	91.46	51.05
Greenhouse_2025-26	PDS	40.76	42.13	34.5	0.2	97.58	97.38	84.63

PDS- Plant Disease severity, *R*- Resistant, MR- Moderately Resistant,
S- Susceptible, MS- Moderately Susceptible

Table 4. Disease severity (PDS %) and reaction of genotypes under both environmental conditions, classified by consistency of resistance response

S. No.	Genotype	Field PDS (%)	Greenhouse PDS (%)	Field Disease Reaction	Greenhouse Disease Reaction	Consistency Category
1	15_KDS-1045_W	5.58	8.016	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
2	1_NRC-86_W	9.74	14.655	Resistant (<i>R</i>)	Moderately Resistant (MR)	Environment-dependent / variable
3	GW 102_W	39.29	51.652	Moderately Susceptible (MS)	Susceptible (<i>S</i>)	Environment-dependent / variable
4	GW 104_W	6.61	4.103	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
5	GW 114_W	4.54	4.993	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
6	GW 134_W	34.31	61.46	Moderately Susceptible (MS)	Susceptible (<i>S</i>)	Environment-dependent / variable
7	GW 140_W	55.97	85.561	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
8	GW 143_W	50.91	60.911	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
9	GW 160_W	86.87	97.578	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible

(continued on next page)

S. No.	Genotype	Field PDS (%)	Greenhouse PDS (%)	Field Disease Reaction	Greenhouse Disease Reaction	Consistency Category
10	GW 175_W	59.79	71.474	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
11	GW 179_W	100	97.216	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
12	GW 204_W	94.94	93.781	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
13	GW 206_W	12.46	8.175	Moderately Resistant (MR)	Resistant (<i>R</i>)	Environment-dependent / variable
14	GW 211_G	61.19	85.524	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
15	GW 213_W	40.71	44.126	Moderately Susceptible (MS)	Moderately Susceptible (MS)	Environment-dependent / variable
16	GW 227_W	38.74	50.213	Moderately Susceptible (MS)	Susceptible (<i>S</i>)	Environment-dependent / variable
17	GW 250_W	6.783	6.783	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
18	GW 252_W	13.99	21.885	Moderately Resistant (MR)	Moderately Susceptible (MS)	Environment-dependent / variable
19	GW 256_W	3.28	2.695	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
20	GW 257_W	34.34	40.132	Moderately Susceptible (MS)	Moderately Susceptible (MS)	Environment-dependent / variable
21	GW 260_BR	3.2	6.198	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
22	GW 264_B	3.37	4.806	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
23	GW 273_W	88.03	72.032	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
24	GW 274_W	52.1	46.272	Susceptible (<i>S</i>)	Moderately Susceptible (MS)	Environment-dependent / variable
25	GW 27_BR	2.36	0.203	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
26	GW 284_W	30.3	34.027	Moderately Susceptible (MS)	Moderately Susceptible (MS)	Environment-dependent / variable
27	GW 289_B	32.5	51.852	Moderately Susceptible (MS)	Susceptible (<i>S</i>)	Environment-dependent / variable
28	GW 291_B	8.02	9.071	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
29	GW 303_W	4.24	4.609	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
30	GW 313_W	61.88	92.309	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
31	GW 320_W	43.41	3.953	Moderately Susceptible (MS)	Resistant (<i>R</i>)	Environment-dependent / variable
32	GW 32_W	9.41	7.462	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
33	GW 39_G	2.56	6.343	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant
34	GW 86_W	66.45	56.281	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
35	GW 89_G	95.65	96.31	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
36	GW 96_W	30.87	46.853	Moderately Susceptible (MS)	Moderately Susceptible (MS)	Environment-dependent / variable
37	JS 20-98	6.25	13.137	Resistant (<i>R</i>)	Moderately Resistant (MR)	Environment-dependent / variable
38	JS 22-12	78.97	63.236	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
39	JS 335	94	96.837	Susceptible (<i>S</i>)	Susceptible (<i>S</i>)	Consistently susceptible
40	NRC130	9.83	7.83	Resistant (<i>R</i>)	Resistant (<i>R</i>)	Consistently resistant

(continued on next page)

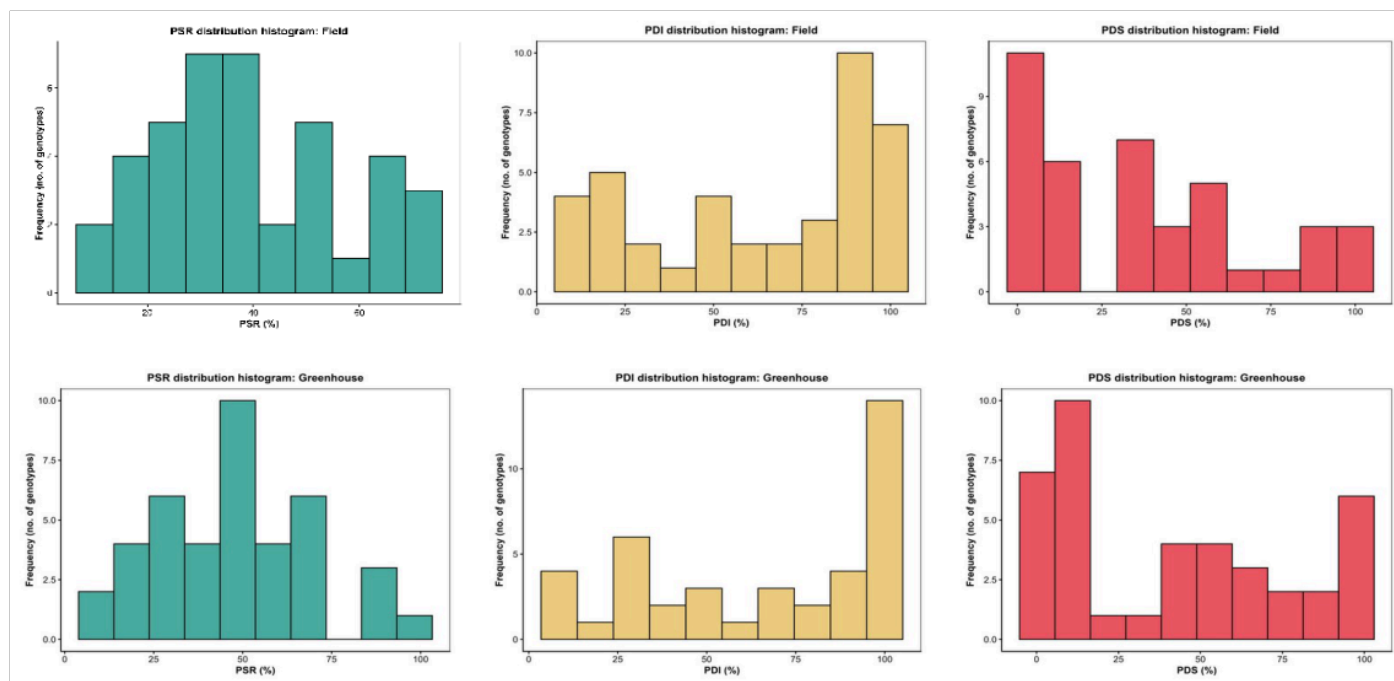


Figure 1. Frequency distribution histograms of Percent Severity Rating (PSR), Percent Disease Index (PDI) and Percent Disease Severity (PDS) of genotypes evaluated under field and greenhouse conditions.

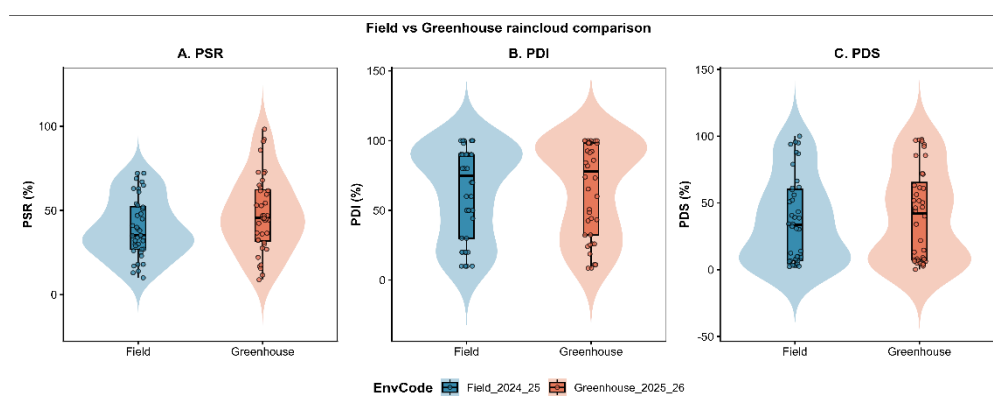


Figure 2. Raincloud plots showing distribution of (A) Percent Severity Rating (PSR), (B) Percent Disease Index (PDI) and (C) Percent Disease Severity (PDS) of genotypes under field (2024-25) and greenhouse (2025-26) conditions.

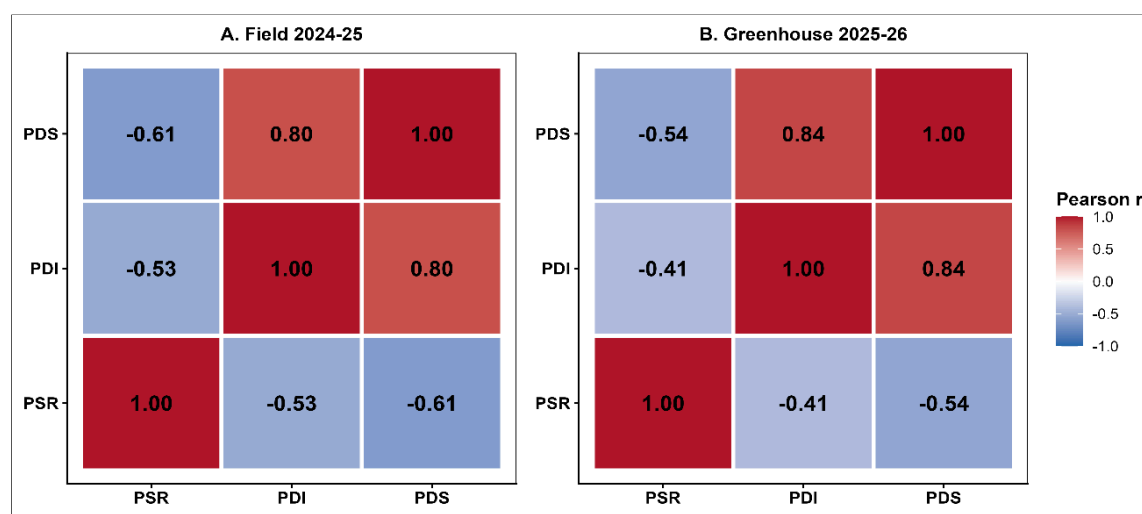


Figure 3. Pearson correlation heatmaps among Percent Severity Rating (PSR), Percent Disease Index (PDI) and Percent Disease Severity (PDS) under (A) Field 2024-25 and (B) Greenhouse 2025-26 conditions.

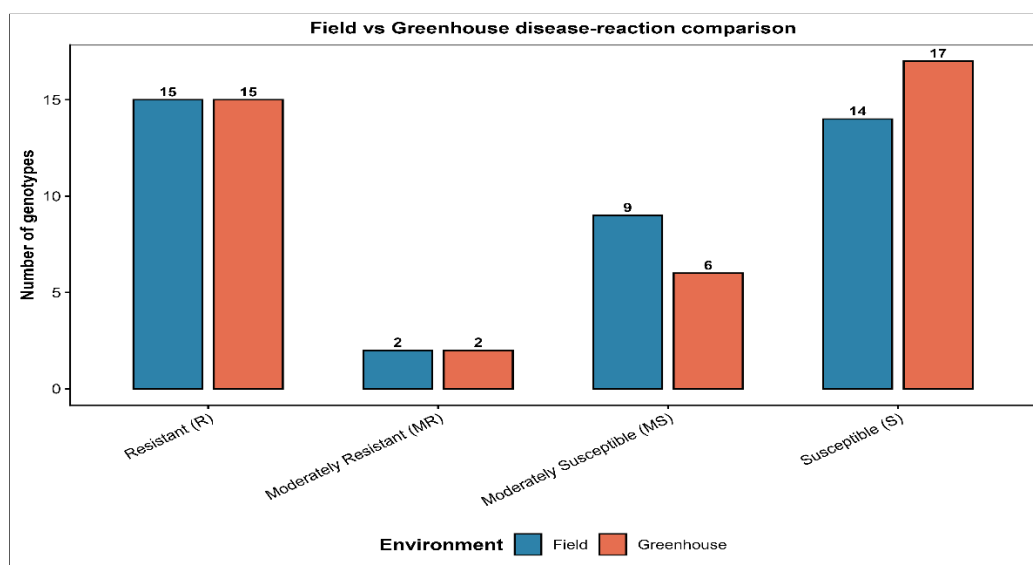


Figure 4. Number of genotypes classified into disease reaction categories under natural field and greenhouse conditions.

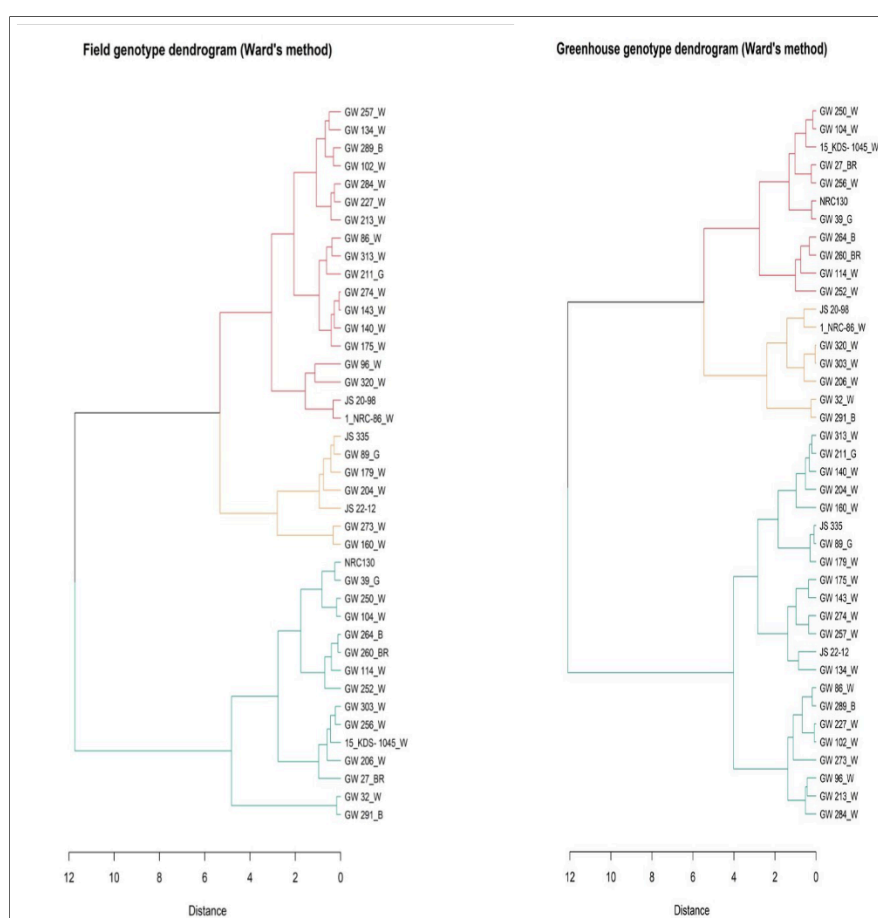


Figure 5. Ward's hierarchical clustering dendrogram showing genetic divergence among soybean genotypes based on anthracnose disease response under field and greenhouse conditions.

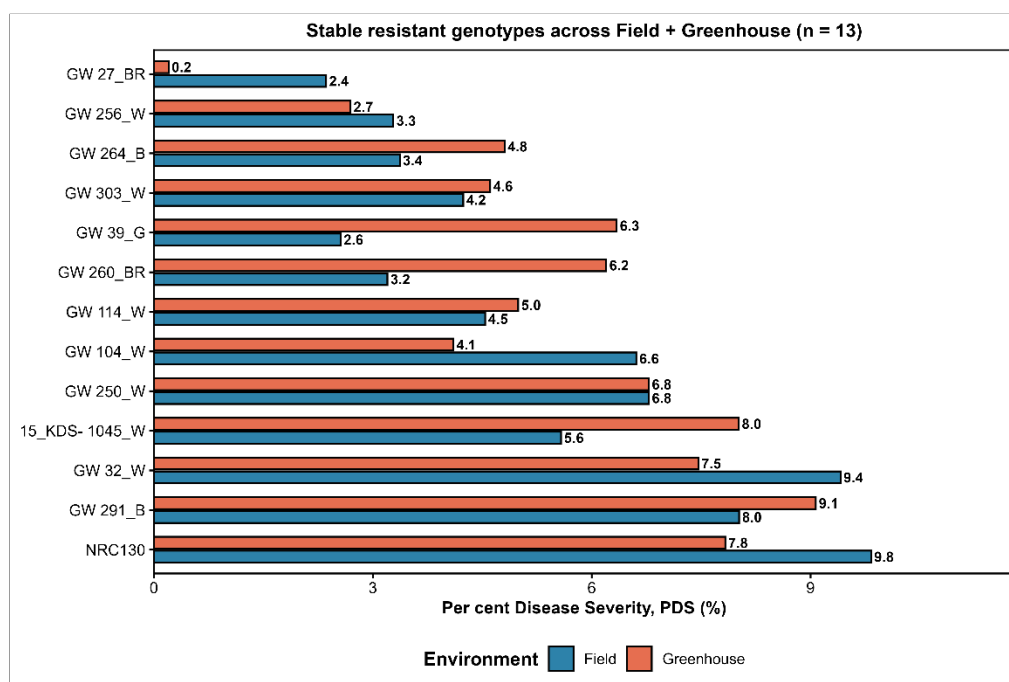


Figure 6. Comparative disease severity (PDS, %) of 13 soybean genotypes showing stable resistance to anthracnose under field and greenhouse screening conditions.

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