



RESEARCH ARTICLE

Integrating host resistance and botanical agents: Screening pigeonpea [*Cajanus cajan* (L.) Huth] genotypes for *Fusarium* wilt (*Fusarium udum* Butler) management

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Abstract

Fusarium wilt, caused by *Fusarium udum* Butler, is a major biotic constraint limiting pigeonpea (*Cajanus cajan* L. Huth) productivity in tropical and subtropical regions. Wilt incidence varied widely among 143 pigeonpea genotypes, ranging from 0.40 to 15.60 % at 30 days after sowing and escalating to 2.00 to 78.00 % at 75 DAS. Area under disease progress curve (AUDPC) values ranged from 57.9 to 2258.1, indicating diverse disease progression. Based on the percent disease index (PDI), relative AUDPC (rAUDPC) and *r*-values, genotypes were categorised as resistant (38.5 %), moderately resistant (37.8 %), susceptible (11.9 %) and highly susceptible (11.9 %). Notable resistant genotypes included Pusa Arhar 21-60, ICP 8863 and NAM 88. *In vitro* evaluation of seven plant extracts at 10, 15 and 20 ppm using the poisoned food technique revealed significant variation in antifungal activity. *Thuja occidentalis* L., *Cannabis sativa* L. and *Mentha spicata* L. consistently exhibited the highest inhibition in a dose-dependent manner, whereas *Chenopodium album* L. was least effective. Integration of resistant genotypes with potent botanical extracts offers an eco-friendly strategy for sustainable management of *Fusarium* wilt in pigeonpea.

Keywords: area under disease progress curve; botanicals; disease management; *Fusarium* wilt; pigeonpea

Introduction

Pigeonpea (*Cajanus cajan* L. Huth) is a vital leguminous crop predominantly cultivated in tropical and subtropical regions, with India being the leading producer, contributing approximately 80–90 % of global production (1, 2). Despite its significance, pigeonpea productivity has remained stagnant over the past three decades due to various biotic and abiotic stresses. Among the biotic constraints, *Fusarium* wilt caused by *Fusarium udum* is a major threat, leading to substantial yield losses. The pathogen's ability to persist in soil and infect plants at various growth stages exacerbates the challenge (3). Traditional management strategies, including the use of synthetic fungicides, have limitations such as environmental concerns and the development of pathogen resistance. Consequently, there is a growing interest in exploring alternative, eco-friendly approaches for disease management. Botanicals, known for their antifungal properties, present a promising avenue. Recent studies have highlighted that plant-derived extracts possess

significant antifungal activity against *Fusarium* species, effectively inhibiting their growth. For instance, *Mentha spicata* L. essential oil has demonstrated strong inhibitory effects against *Fusarium oxysporum* under *in vitro* conditions, while other plant-derived essential oils and extracts have also shown broad-spectrum antifungal potential against phytopathogenic fungi (4, 5). In addition to botanical interventions, breeding for genetic resistance remains a cornerstone of disease management. Screening pigeonpea genotypes for resistance to *Fusarium* wilt is crucial for developing cultivars with durable resistance. Previous studies have identified genotypes with varying degrees of resistance, underscoring the importance of genetic diversity in breeding programs (6). The study aims to screen diverse pigeonpea genotypes for *Fusarium* wilt resistance and evaluate selected botanicals under laboratory and field conditions. By integrating genetic resistance with botanical treatments, the research seeks to develop sustainable, eco-friendly disease management strategies for pigeonpea cultivation.

Materials and Methods

Screening of pigeonpea genotypes

Location

Field experiments were conducted during the kharif season of 2021–22 at the Research Farm of Tirhut College of Agriculture, Dholi, Muzaffarpur (Bihar), situated at an elevation of 160 m above mean sea level (latitude 25.98° N, longitude 85.6° E). The experimental site featured uniform topography with silty loam soil and a slightly alkaline pH of 8.5. Controlled laboratory studies were performed at the Department of Plant Pathology, Tirhut College of Agriculture, Dholi.

Wilt sick plot preparation

Fusarium udum Butler was isolated from wilt-infected pigeonpea plants collected from naturally infested fields. Infected vascular tissues were surface sterilised (1% NaOCl, 1–2 min), rinsed and plated on potato dextrose agar (PDA), followed by incubation at $25 \pm 2^\circ\text{C}$ for 5–7 days. Pure cultures were obtained using the hyphal tip method and identified based on morphological characteristics (7).

The pathogen was mass-multiplied on sterilised sand-pigeonpea flour medium (8:2, w/w; ~40–50 % moisture), autoclaved at 121°C for 30 min on two consecutive days and incubated at $25 \pm 2^\circ\text{C}$ for 10–15 days until full colonisation. The inoculum was mixed with sterilised soil at 20 % (w/w) and incorporated into an already wilt-infested experimental plot during final land preparation to ensure uniform disease pressure. Uninoculated sterilised soil served as a control in pot experiments. The study was conducted in a pre-infested research field using a native pathogen isolate, following standard phytopathological practices for sick plot development and institutional biosafety guidelines to avoid unintended spread (8).

Genotypes and planting

A total of 143 pigeonpea genotypes obtained from the Indian Institute of Pulses Research (IIPR), Kanpur, were evaluated under field conditions. Each genotype was sown in two rows with plant spacing of 20 cm between plants and 50 cm between rows. To maintain uniform disease pressure, infector rows of the highly susceptible check ICP 2376 were planted after every two test entries, following the standard sick plot technique. Recommended agronomic practices were followed to ensure optimal crop growth and health (9, 10).

Observations on wilt incidence

Wilt incidence was recorded at 30, 45, 60 and 75 days after sowing (DAS), continuing until February 2022. At each interval, the number of wilted plants per genotype was recorded. Percent disease incidence (PDI) was calculated using the following formula:

$$\text{PDI} = \frac{\text{Number of wilted plants}}{\text{Total number of plants}} \times 100 \quad (\text{Eqn. 1})$$

This provided a quantitative estimate of wilt severity, helping assess resistance levels across genotypes. The mean PDI (%) was calculated as the average of PDI values recorded at 30, 45, 60 and 75 DAS, providing a measure of sustained resistance or susceptibility overtime.

Quantification of disease progression

Area under the disease progress curve: To quantify disease progression over time, AUDPC was computed using the standard method (11).

$$\text{AUDPC} = \sum_{i=1}^{n-1} \left[\left(\frac{Y_i + Y_{i+1}}{2} \right) \times (t_{i+1} - t_i) \right] \quad (\text{Eqn. 2})$$

where Y_i is the PDI at the i^{th} observation and t_i is the time (days) at the i^{th} observation.

The AUDPC integrates both severity and duration of disease, offering a comprehensive assessment of disease impact.

Relative AUDPC: To facilitate comparison among genotypes, relative AUDPC (rAUDPC) was calculated by normalising each genotype's AUDPC to the maximum AUDPC recorded in the experiment. This allows for standardised interpretation across varying disease levels.

Relative rate of disease progression (r-value): The r -value was calculated using regression analysis of PDI over time, reflecting the rate of disease development. A lower r -value indicates slower disease progression, signifying better resistance.

Classification of genotypes

Based on PDI at 75 DAS and AUDPC, genotypes were categorised as:

Disease incidence (%)	Reaction
1–10	Resistant
> 10–30	Moderately resistant
> 30–50	Susceptible
> above 50	Highly susceptible

This classification allows clear identification of genotypes with varying degrees of disease resistance, facilitating breeding and selection programs.

Efficacy of botanicals against the pathogen *in vitro*

The antifungal activity of aqueous extracts from seven botanicals (*Aegle marmelos* (L.) Corrêa, *Cannabis sativa* L., *Chenopodium album* L., *Mentha spicata* L., *Psidium guajava* L., *Solanum nigrum* L. and *Thuja occidentalis* L.) was evaluated against *F. udum*, the causal agent of pigeonpea wilt. The pathogen was isolated from wilted pigeonpea plants collected from naturally infested fields. Pure cultures were obtained using the hyphal tip method and identified based on morphological characteristics following standard taxonomic keys (7).

Aqueous extracts were prepared by soaking 10 g of shade-dried leaf powder in 100 mL of sterile distilled water for 24 hr, then filtering to obtain stock solutions. Working concentrations of 10, 15 and 20 % (v/v) were prepared and incorporated into sterilised PDA using the poisoned food technique (12). A 5 mm mycelial disc from a 7-day-old culture of *F. udum* was placed centrally on each plate, with untreated PDA serving as a control. Plates were incubated at $25 \pm 2^\circ\text{C}$ for 7 days and radial growth was recorded. Percent inhibition of mycelial growth was calculated relative to the control. The experiment was conducted in a completely randomised design (CRD) with 3 replicates.

Statistical analysis

PDI values were used to calculate mean PDI, AUDPC, (rAUDPC) and the r -value. These parameters were computed using R Statistics Software. For the *in vitro* evaluation of botanical extracts against the mycelial growth of the pathogen, statistical analysis was performed. Treatment means were made at the 1 % level of significance (13).

Results

Screening of genotypes

A wide variation in wilt incidence was observed among the genotypes, ranging from 0.40 to 15.60 % at 30 days after sowing (DAS) and increasing to 2.00 to 78.00 % at 75 DAS. The AUDPC values varied considerably, ranging from 57.9 to 2258.1, indicating diverse disease progression rates among the genotypes. Comprehensive data of genotypes, including PDI (%), AUDPC, rAUDPC, *r*-value and disease reaction, are presented in Supplementary Table 1.

A total of 143 red gram genotypes were classified into four disease reaction categories based on their PDI, rAUDPC and *r*-value (Table 1; Fig. 1). Among these, 55 genotypes were categorised as resistant, exhibiting a mean PDI of 5.79 %, a mean rAUDPC of 3.72 % and a mean *r*-value of 0.04. A group of 54 genotypes were classified as moderately resistant with a mean PDI of 16.96 %, a mean rAUDPC of 10.91 % and a similar *r*-value of 0.04. In contrast, 17 genotypes were found to be susceptible, having a mean PDI of 42.88 %, a mean rAUDPC of 27.59 % and an *r*-value of 0.05. Additionally, 17 genotypes were identified as highly susceptible, with the highest disease expression characterised by a mean PDI of 63.02 %, a mean rAUDPC of 40.55 % and a mean *r*-value of 0.06.

Based on the PDI recorded at 75 DAS, the genotypes were categorised into four distinct resistance groups: resistant, moderately resistant, susceptible and highly susceptible (Table 2 and Fig. 2).

A total of 55 genotypes (38.5 %) were classified as resistant (PDI ≤ 10 %), indicating a high level of tolerance to the disease. Notable genotypes in this category included Pusa Arhar 21-60, ICP 8863, NAM 88, TJT 501, BDN 2019-5, BDN 2019-9, WRGE 134, ICPDL

17103, AKTE 16-12, PT 11-4-4-1, BDN 716, JKN 189, NAM 151, BDN 2019-34, AKTM 1914, PT 12-5-5-1, SKNP 1715, BAUPP 16-38, BAUPP 18-7, WRG 391, LRG 471, IPAL 20-24, PT 2017-1, WRGE 124, PT 10-1-1-2, BDN 2013-2, PT 11-16, GRG 622, GJP 1915, AKTM 1637, IPA 19-26, IPA 19-43, IPA 19-6, IPA 18-8, IPA 14-2, IPA 15-19, IPA 13-1, BRG 4, BRG 5164, BSMR 203, BSMR 736, BWR 243, BWR 853, BWR 254, BWR 553, BWR 571, BWR 316, BSMR 2, BSMR 65, BSMR 1, BSMR 339, BWR 23, BWR 164, BWR 153 and BWR 133.

Fifty-four genotypes (37.8 %) exhibited moderate resistance (PDI > 10–30 %), demonstrating intermediate tolerance to the disease. These included AL 2324, Pusa Arhar 21-1, Pusa Arhar 21-27, Pusa Arhar 21-29, CRG 16-01, PT 0012, BDN 711, PT 12-19-2, AKTE 1905, LRG 467, RVKT 383, NAM 92, BDN 2013-5, RVSA 2020-6, RVSA 2020-7, RVSA 2020-8, LRG 224, LRG 275, AKTM 1917, GJP 2008, WRG 390, RKPV 821-01, NPD 18-03, KA 19-05, IPAL 20-11, RVSA 14-2, WRGE 138, PT 2017-2, PUSA 197, MAL 53, IPA 15-21, IPA 14-6, IPA 11-45, IPA 15-1, IPA 16-18, GTH 1, SKNPCH 1906, WRG 65, PAH 16, PAH 17, PAH 5, PAH 10, WRG 1, MAL 13, MAL 57, BRG 1, BRG 2, BRG 3, BSMR 175, BSMR 164, BSMR 79, BSMR 26, BWR 254 and BWR 134.

Seventeen genotypes (11.9 %) were classified as susceptible (PDI > 30–50 %), indicating higher disease incidence. These genotypes included PAU 881, Pusa Arhar 21-61, Pusa Arhar 21-57, PUSA 992, PA 291, PA 662, AL 2276-1, Pusa Arhar 21-14, Pusa Arhar 21-24, PA 656, RKPV 810-01, CRG 18-007, PUSA-198, AH 1713, IPA 18-4, IPA 7-2 and MA 6. Another 17 genotypes (11.9 %) were categorised as highly susceptible (PDI > 50 %), reflecting severe disease reaction. This group comprised ICP 2376, UPAS 120, PA 663, PA 669, AL 2250, Pusa Arhar 21-45, PA 676, SKNP 1710, PUSA-199, Pusa 2020-2, PA 650, IPAE 15-08, RVKT 333, IPAE 15-05, CRG 16-12, IPA 19-42 and IPA 18-33.

Table 1. Classification of 143 red gram genotypes based on disease reaction, along with their mean PDI, rAUDPC and *r*-value

Reaction class	PDI range (%)	No. of genotypes	Mean PDI (%)	Mean rAUDPC (%)	Mean <i>r</i> -value
Resistant (R)	≤ 10	55	5.79	3.72	0.04
Moderately resistant (MR)	> 10–30	54	16.96	10.91	0.04
Susceptible (S)	> 30–50	17	42.88	27.59	0.05
Highly susceptible (HS)	> 50	17	63.02	40.55	0.06

PDI: percent disease index; rAUDPC: relative area under disease progress curve; *r*-value: relative rate of disease increase.

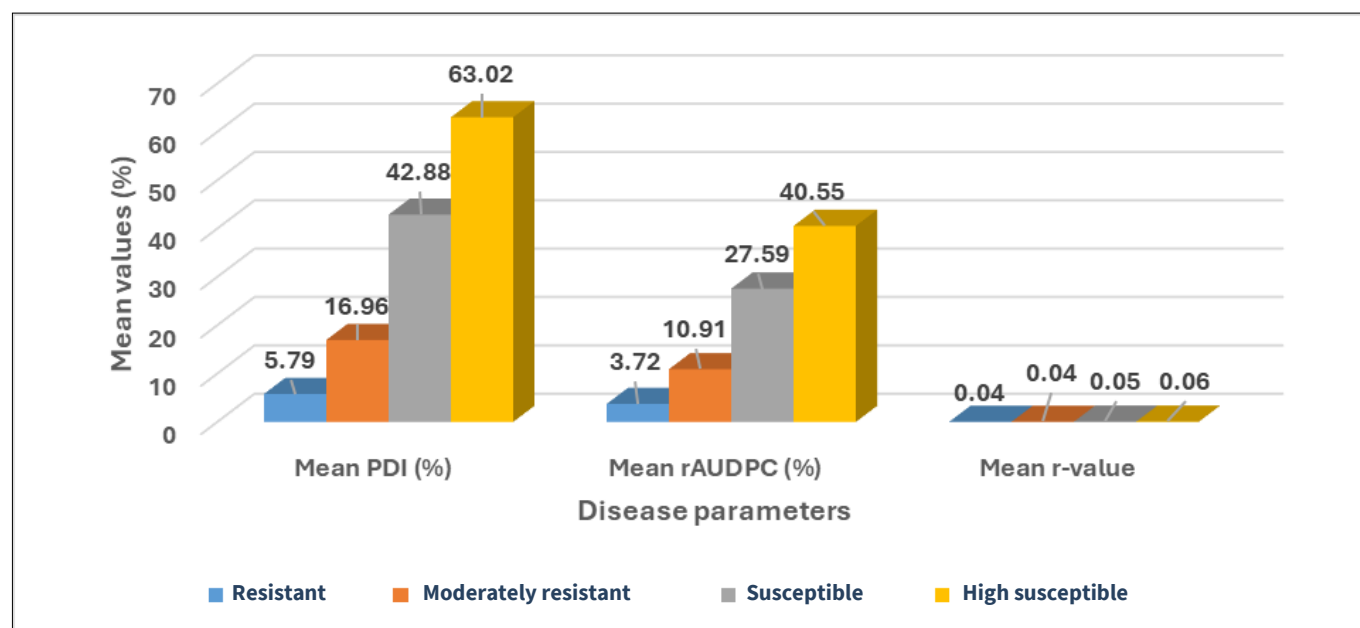
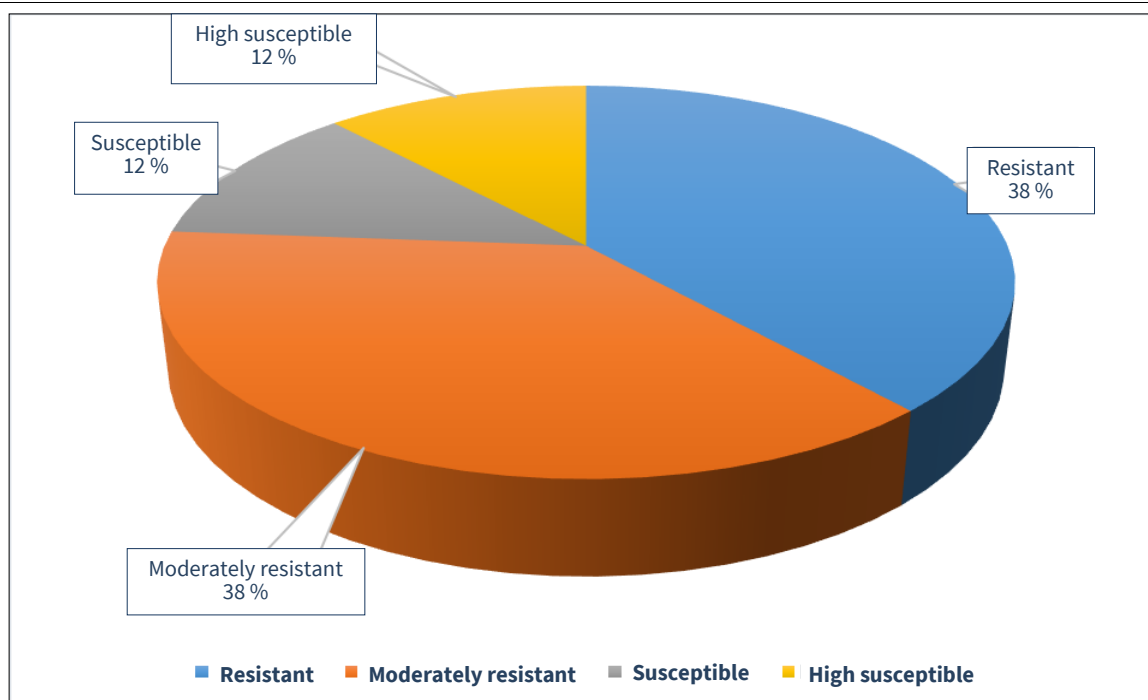


Fig. 1. Comparison of disease parameters across different resistance categories.

Table 2. List of red gram genotypes classified into different resistance categories based on percent disease index (PDI) at 75 DAS

Category	No. of genotypes	Genotypes
Resistant ($\leq 10\%$)	55	Pusa Arhar 21-60, ICP 8863, NAM 88, TJT 501, BDN 2019-5, BDN 2019-9, WRGE 134, ICPDL 17103, AKTE 16-12, PT 11-4-4-1, BDN 716, JKN 189, NAM 151, BDN 2019-34, AKTM 1914, PT 12-5-5-1, SKNP 1715, BAUPP 16-38, BAUPP 18-7, WRG 391, LRG 471, IPAL 20-24, PT 2017-1, WRGE 124, PT 10-1-1-2, BDN 2013-2, PT 11-16, GRG 622, GJP 1915, AKTM 1637, IPA 19-26, IPA 19-43, IPA 19-6, IPA 18-8, IPA 14-2, IPA 15-19, IPA 13-1, BRG 4, BRG 5164, BSMR 203, BSMR 736, BWR 243, BWR 853, BWR 254, BWR 553, BWR 571, BWR 316, BSMR 2, BSMR 65, BSMR 1, BSMR 339, BWR 23, BWR 164, BWR 153, BWR 133.
Moderately resistant ($> 10-30\%$)	54	AL 2324, Pusa Arhar 21-1, Pusa Arhar 21-27, Pusa Arhar 21-29, CRG 16-01, PT 0012, BDN 711, PT 12-19-2, AKTE 1905, LRG 467, RVKT 383, NAM 92, BDN 2013-5, RVSA 2020-6, RVSA 2020-7, RVSA 2020-8, LRG 224, LRG 275, AKTM 1917, GJP 2008, WRG 390, RKPV 821-01, NPJ 18-03, KA 19-05, IPAL 20-11, RVSA 14-2, WRGE 138, PT 2017-2, PUSA 197, MAL 53, IPA 15-21, IPA 14-6, IPA 11-45, IPA 15-1, IPA 16-18, GTH 1, SKNPCH 1906, WRG 65, PAH 16, PAH 17, PAH 5, PAH 10, WRG 1, MAL 13, MAL 57, BRG 1, BRG 2, BRG 3, BSMR 175, BSMR 164, BSMR 79, BSMR 26, BWR 254, BWR 134.
Susceptible ($> 30-50\%$)	17	PAU 881, Pusa Arhar 21-61, Pusa Arhar 21-57, PUSA 992, PA 291, PA 662, AL 2276-1, Pusa Arhar 21-14, Pusa Arhar 21-24, PA 656, RKPV 810-01, CRG 18-007, PUSA-198, AH 1713, IPA 18-4, IPA 7-2, MA 6.
Highly susceptible ($> 50\%$)	17	ICP 2376, UPAS 120, PA 663, PA 669, AL 2250, Pusa Arhar 21-45, PA 676, SKNP 1710, PUSA-199, Pusa 2020-2, PA 650, IPAE 15-08, RVKT 333, IPAE 15-05, CRG 16-12, IPA 19-42, IPA 18-33.

**Fig. 2.** Genotypic distribution by resistance category (%).

In vitro evaluation of different plant extracts against *Fusarium udum*

The antifungal potential of seven plant extracts was evaluated against *Fusarium udum* at concentrations of 10, 15 and 20 ppm using the poisoned food technique (Table 3 and Fig. 3). Significant differences were observed among treatments and concentrations, indicating variable fungitoxic potential of the botanicals. At 10 ppm, *T. occidentalis* exhibited the lowest mycelial growth (16.62 mm) and highest inhibition (78.62 %), being statistically superior to all other treatments. *Cannabis sativa* (27.47 mm; 64.67 %) formed the next

most effective group, significantly higher than *M. spicata* (32.15 mm; 58.65 %) but statistically at par with *P. guajava* (44.68 mm; 42.53 %). *Solanum nigrum* (56.17 mm; 27.76 %) and *C. album* (63.81 mm; 17.93 %) were the least effective, showing lower inhibition than the control (77.75 mm). At 15 ppm, *T. occidentalis* again recorded the minimum mycelial growth (16.48 mm) and maximum inhibition (79.90 %), remaining statistically at par with *C. sativa* (20.24 mm; 75.32 %) and *M. spicata* (22.48 mm; 72.59 %). *Psidium guajava* (29.44 mm; 64.10 %) exhibited moderate inhibition, while *S. nigrum* (40.44 mm; 50.68 %) and *C. album* (56.92 mm; 30.59 %) were comparatively less effective. At 20 ppm, *T. occidentalis* (15.72 mm;

Table 3. Comparative antifungal efficacy of selected plant extracts against *Fusarium udum* expressed as mycelial growth and percent inhibition at different concentrations

Treatments		Mycelial growth (mm)			Growth inhibition (%)		
Common name	Scientific name	10 ppm	15 ppm	20 ppm	10 ppm	15 ppm	20 ppm
Bael	<i>Aegle marmelos</i>	52.54	34.78	30.84	32.42	57.59	62.39
Bhang	<i>Cannabis sativa</i>	27.47	20.24	16.44	64.67	75.32	79.95
Bathua	<i>Chenopodium album</i>	63.81	56.92	49.18	17.93	30.59	40.02
Mentha	<i>Mentha spicata</i>	32.15	22.48	20.42	58.65	72.59	75.10
Guava	<i>Psidium guajava</i>	44.68	29.44	24.96	42.53	64.10	69.56
Makoy	<i>Solanum nigrum</i>	56.17	40.44	34.92	27.76	50.68	57.41
Thuja	<i>Thuja occidentalis</i>	16.62	16.48	15.72	78.62	79.90	80.83
Control		77.75	82.00	82.00	0.00	0.00	0.00
C.D. at 1 %		2.52	1.52	1.33	-	-	-
CV		3.12	2.29	2.23	-	-	-

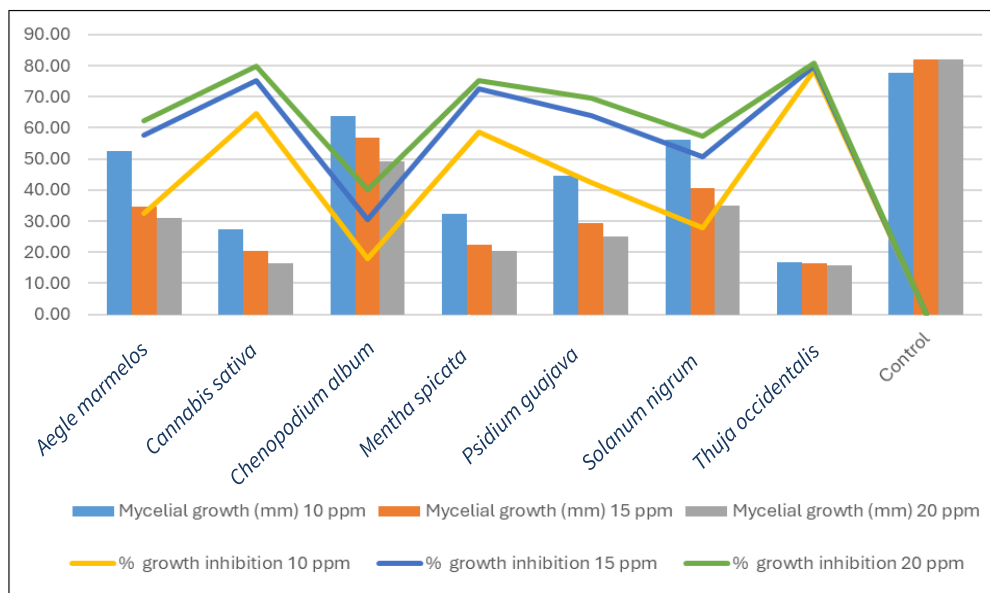


Fig. 3. Comparative antifungal efficacy of plant extracts against *Fusarium udum* at different concentrations.

80.83 %) again showed the highest inhibition, statistically at par with *C. sativa* (16.44 mm; 79.95 %) and *M. spicata* (20.42 mm; 75.10 %), which together formed the most effective group. *Psidium guajava* (24.96 mm; 69.56 %) constituted the next significant group, followed by *S. nigrum* (34.92 mm; 57.41 %) and *C. album* (49.18 mm; 40.02 %), which were least effective. *Thuja occidentalis*, *C. sativa* and *M. spicata* consistently exhibited superior antifungal activity across all concentrations, showing a clear dose-dependent increase in inhibition. *Chenopodium album* remained the least effective, likely due to lower concentrations of bioactive antifungal compounds.

Discussion

The present screening of 143 pigeonpea genotypes revealed substantial variation in *Fusarium* wilt incidence and disease progression, with wilt incidence ranging from 0.40 to 15.60 % at 30 DAS and increasing to 2.00 to 78.00 % by 75 DAS. The wide range of AUDPC values (57.9 to 2258.1) underscores differential disease dynamics across genotypes. Such variability in host response aligns with earlier reports documenting significant genetic diversity for *Fusarium* wilt resistance in pigeonpea and other pulse crops (14).

The distribution of genotypes across resistance categories from resistant to highly susceptible confirms broad-spectrum disease responses, consistent with prior studies in pigeonpea and legumes. Research indicates the considerable differences in *Fusarium* wilt resistance among pigeonpea genotypes, largely due to inherent genetic variability and environmental interactions (14). Similarly, breeding, genetics and genomics studies emphasise that allelic diversity and genotype $\times$ environment ($G \times E$) interactions strongly influence resistance expression (15).

Identification of 55 genotypes (~38.5 %) as resistant (mean PDI 5.79 %) and 54 (~37.8 %) as moderately resistant provides a valuable genetic reservoir for breeding programs. Notable resistant genotypes, such as Pusa Arhar 21-60, ICP 8863, NAM 88 and TJT 501, may serve as donor parents in crosses with high-yielding but susceptible lines. Their relatively low relative AUDPC (rAUDPC) and r -values suggest durable resistance across temporal disease progression, enhancing their suitability for both conventional and marker-assisted breeding. SSR markers such as ASSR-1, ASSR-23,

ASSR-148 and ASSR-363 have been employed to tag *Fusarium* wilt resistance loci in pigeonpea (8). Integrating phenotypic screening with molecular marker-based approaches can significantly accelerate the introgression of resistance alleles into elite cultivars by enhancing selection efficiency and precision (16, 17).

Dynamic disease assessment parameters such as AUDPC and rAUDPC, alongside endpoint PDI, provide a comprehensive understanding of disease progression, latent period and resistance durability. Genotypes with low AUDPC and r -values exhibit slower disease development and delayed epidemic buildup, desirable traits for durable resistance. This approach effectively distinguishes between escape, tolerance and true resistance mechanisms and has been recommended in multiple patho-systems (18–20).

Despite promising results, resistance expression may be influenced by $G \times E$ interactions. Previous studies in pigeonpea documented significant environmental effects on wilt incidence, indicating that genotypes may perform differently across agro-ecologies (21). Multi-location evaluation is therefore essential to confirm the stability of resistance. Additionally, the genetic control of wilt resistance in pigeonpea is complex, involving single dominant genes, duplicate dominant genes or complementary gene systems depending on the genetic background (14). Fine mapping and molecular characterisation of resistance loci in the screened material would further elucidate gene action and support precision breeding.

Transcriptomic analyses have revealed key pathways involved in pigeonpea wilt resistance, including jasmonic acid and salicylic acid signalling, cell wall remodelling, reactive oxygen species (ROS) regulation and vascular development (22). Integrating transcriptomic or gene expression studies with phenotypic data from resistant and susceptible genotypes could provide mechanistic insights and identify candidate genes for functional validation. Collectively, these findings highlight a rich resource of resistant germplasm for developing durable, high-performing pigeonpea cultivars.

In vitro evaluation revealed significant variation in antifungal activity among the seven plant extracts against *F. udum*. *Thuja occidentalis*, *C. sativa* and *M. spicata* consistently demonstrated the strongest inhibitory effects in a dose-dependent manner. The superior performance of *T. occidentalis* aligns with recent studies reporting its broad-spectrum antifungal activity against

phytopathogenic and opportunistic fungi, including *Fusarium*, *Aspergillus* and *Saccharomyces* species (23). The antifungal effect is primarily attributed to monoterpenoids such as thujone, camphor and borneol, which disrupt fungal cell membranes and interfere with metabolic pathways (24). GC-MS analysis showed that *T. occidentalis* essential oil contains ~21.6 % thujone, 18.9 % camphor and 9.7 % isobornyl acetate, significantly inhibiting *Aspergillus flavus* and downregulating the *afIR-2* gene involved in mycotoxin production (25).

Although direct reports of *C. sativa* extracts against *F. udum* are limited, *C. sativa* is susceptible to other *Fusarium* species and its extracts are known to possess antimicrobial properties. The presence of cannabinoids, terpenes and phenolics likely contributes to membrane disruption or enzyme inhibition in fungal cells (26). The antifungal potential of *M. spicata* is well documented with menthol, menthone and related compounds impairing fungal cell wall integrity, inhibiting spore germination and disrupting ergosterol biosynthesis (27). The observed dose-dependent inhibition corroborates previous studies on botanical extracts against *Fusarium* species. In the context of pigeonpea wilt, *F. udum* remains a major pathogen and recent studies emphasise biocontrol and botanical alternatives due to limitations of chemical fungicides (28, 29). Integrative strategies, such as deploying microbial consortia in the rhizosphere, have been shown to effectively suppress *Fusarium udum*. Combined application of biocontrol agents such as *Trichoderma viride* along with other rhizosphere microbes significantly reduced wilt incidence to 2.97–3.15 % compared to much higher levels in the untreated control, demonstrating strong disease suppression under field conditions (30).

Conclusion

The study revealed significant genetic variability among pigeonpea genotypes against *Fusarium udum*, with 55 resistant lines identified as promising breeding resources. Among the botanicals evaluated, *Thuja occidentalis*, *Cannabis sativa* and *Mentha spicata* exhibited strong, dose-dependent antifungal activity, while *Chenopodium album* was least effective. The integration of resistant genotypes with effective botanicals offers a sustainable and eco-friendly approach for managing pigeonpea wilt and reducing dependence on chemical fungicides.

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Authors' contributions

JK, SC and AK¹ conducted the field experiments, laboratory work and data collection and contributed to manuscript preparation. BK conceptualised and supervised the study. RB assisted with statistical analysis, preparation of figures and tables and critically reviewed the manuscript. AK² and MBT contributed to the final editing of the manuscript. All authors read and approved the final manuscript. [AK¹ stands for Arti Kumari and AK² stands for Anupam Kumari].

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) to enhance language clarity and improve the overall readability of the text. After using this tool, the authors properly reviewed and revised the content as necessary and take full responsibility for the content of the publication.

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