International Journal of Plant & Soil Science

Volume 38, Issue 1, Page 99-113, 2026; Article no.IJPSS.145282
ISSN: 2320-7035

Screening of Amaranthus Genotypes for Resistance to Leaf Blight Caused by *Rhizoctonia solani*

S. Akhila [a*], Flemine Xavier [b], C. R. Rashmi [b], P. Anitha [c] and P. K. Reshmika [a]

[a] Department of Vegetable Science, College of Agriculture, Vellanikkara, Kerala Agricultural University, India.
[b] AICRP on Vegetable Crops, College of Agriculture, Vellanikkara, Kerala Agricultural University, India.
[c] Department of Plantation, Spices, Medicinal and Aromatic Crops, College of Agriculture, Vellanikkara, Kerala Agricultural University, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: https://doi.org/10.9734/ijpss/2026/v38i15929

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://pr.sdiarticle5.com/review-history/145282

Original Research Article

Received: 24/09/2025
Published: 09/01/2026

Abstract

Amaranthus is a nutrient-rich leafy vegetable grown in tropical and subtropical regions. Leaf blight caused by *Rhizoctonia solani* is a major constraint in amaranthus production. In the present study 45 amaranthus genotypes were screened twice under field conditions for resistance against *R. solani* and the resistant lines identified were screened *in-vitro* with artificial inoculation. Disease severity varied widely, with PDI values ranging from 0.0 to 88.48 in 2024 and 0.0 to 96.66 in 2025. The accessions *VLKAM-25*, *VLKAM-41*, and *VLKAM-42* were completely immune, while *VLKAM-*

**Corresponding author: E-mail: akhila-2023-12-024@student.kau.in;*

Cite as: *Akhila, S., Flemine Xavier, C. R. Rashmi, P. Anitha, and P. K. Reshmika. 2026. "Screening of Amaranthus Genotypes for Resistance to Leaf Blight Caused by Rhizoctonia Solani". International Journal of Plant & Soil Science 38 (1):99-113. https://doi.org/10.9734/ijpss/2026/v38i15929.*

36 was highly resistant in both field screenings. The lines showing immune to moderate resistance in the field when screened *in vitro,* the accession *VLKAM-41* remained free of symptoms, and *VLKAM-25*, *VLKAM-36*, and *VLKAM-42* showed only limited lesion development. In contrast, the checks Arun and KAU Vaika developed large, coalescing lesions. The immune and highly resistant genotypes were identified as *Amaranthus spinosus (VLKAM-41)*, *A. hypochondriacus (VLKAM-25 & VLKAM-36*), and *A. dubius (VLKAM-42)*.

Keywords: Amaranthus; leaf blight; genotypes; disease screening; Rhizoctonia solani.

1. Introduction

Amaranthus (*Amaranthus* spp.), belonging to the family Amaranthaceae, is a widely cultivated leafy vegetable in tropical and subtropical regions, especially in Southern India. The genus *Amaranthus* includes three subgenera and has 75-80 species, both cultivated and wild, used for grains and leaves (Schmid *et al*., 2007). Due to its rapid growth, high nutritional value and low cultivation cost, it is often referred to as the “poor man’s spinach.” Amaranthus serves as an affordable nutritional source in tropical and subtropical regions and offers considerable economic returns for small scale farmers (Schafleitner *et al*., 2022). The crop is well-equipped to withstand adverse climatic conditions as a C4 photosynthetic plant, which makes it an ideal candidate for sustainable agricultural systems (Maughan *et al*., 2011). Amaranth leaves are valuable source of protein, essential amino acids (lysine, methionine), minerals (iron, calcium, potassium, zinc), and dietary fiber. Additionally, they possess health promoting bioactive molecules like betalains, Vitamin C, phenolic acids and flavonoids, which contribute to their antioxidant and nutraceutical benefits (Sarker *et al*., 2015; 2018).

The leaf blight disease caused by the soil-borne fungal pathogen *Rhizoctonia solani* Kühn (teleomorph: *Thanatephorus cucumeris*) is a serious threat to amaranthus production. It severely diminishes both the yield potential and marketability of amaranthus crop (Nayar *et al*., 1996; Gokulapalan *et al*., 2000). The disease initially appears as small, irregular creamy white spots on the leaves, which expand under humid conditions to form large translucent and light green lesions, ultimately leading to the development of shot hole symptoms (Gokulapalan *et al*., 2000). Severely affected leaves become unfit for human consumption causing a marked reduction in the economic and marketable value of the produce.

The causal agent, *R. solani* infects an extensive range of hosts, approximately 250 plant species belonging to families such as Poaceae, Fabaceae, Solanaceae, Amaranthaceae, Brassicaceae, Rubiaceae, Malvaceae, Asteraceae, Araceae and Moraceae (Chahal *et al*., 2003). In India, the pathogen has been reported on 62 major crop species and 20 families of weeds (Roy, 1993). *R. solani* is a seed and soil-borne pathogen that persists in the form of sclerotia and mycelium within infected seeds or contaminated soil. Infection of the pathogen produces diverse symptoms such as sheath blight, leaf blight, web blight, head rot, root rot, seed rot, bottom rot, stem canker, black scurf, seedling blight, brown patch, and damping-off in various crops (Ajayi-Oyetunde & Bradley, 2018; Senapati *et al*., 2022). Anastomosis, the fusion of vegetative hyphae, is an essential process in many filamentous fungi, enabling the exchange of cellular components such as cytoplasm and nuclei between genetically compatible isolates. Based on this ability to undergo hyphal fusion, isolates of *R. solani* are classified into anastomosis groups (AGs) (Parmeter *et al*., 1969). *R. solani* has been divided into 14 AGs, designated AG-1 to AG-13, along with a bridging group (AG-BI). These AGs differ considerably in colony morphology, nutritional requirements, pathogenic aggressiveness, and host specificity (Carling *et al*., 2002a,b; Ajayi-Oyetunde & Bradley, 2018).

Effective control of the pathogen is challenging due to its ecological adaptability, wide host range, and the ability of its sclerotia to survive under diverse environmental conditions (Anderson, 1982; Ogoshi, 1987). Fortnightly application of Mancozeb (0.4%) as a foliar spray is a suggested chemical control measure for controlling the disease (Gokulapalan *et al*., 1999). However, reliance on chemical fungicides is not a sustainable approach, particularly in the case of leafy vegetables that are typically consumed with minimal processing, as it raises public health concerns due to the potential accumulation of pesticide residues. Developing resistant varieties stands out as the most efficient strategy for the management of leaf blight disease. Since amaranthus is a highly nutritious crop, recent breeding initiatives are focused on boosting foliage yield and disease resistance. Identifying sources of resistance will support breeding efforts aimed at developing leaf blight-resistant amaranthus varieties, thereby enhancing crop productivity. Thus, the present investigation was undertaken to identify amaranthus genotypes with resistance to *Rhizoctonia* leaf blight.

2. Materials and Methods

2.1 Field Screening of Amaranthus Genotypes for *Rhizoctonia* Leaf Blight

The experiment was conducted at the Department of Vegetable Science, College of Agriculture, Vellanikkara, Thrissur, Kerala, during the *Rabi* season (October to January, 2024-25) and *Kharif* season (July to September, 2025). The experimental material comprised 45 genotypes of amaranthus, including three released varieties, 16 accessions from the All India Coordinated Research Project on Vegetable Crops (AICRP-VC), and 26 local collections (Table 1).

The genotypes were evaluated in a Randomized Block Design (RBD) with two replications. Twenty five days old amaranthus seedlings were transplanted in the field with a spacing of 30 x 20 cm. A total of ten plants were maintained in each replication. All cultural and agronomic practices were carried out in accordance with the Package of Practices Recommendations: Crops (KAU, 2024). Under natural conditions, these 45 amaranthus genotypes were screened for *Rhizoctonia* leaf blight resistance.

Table 1. List of amaranthus genotypes used in the study

Sl no	Genotypes	Source
1	VLKAM-1	AICRP (VC)
2	VLKAM-2	AICRP (VC)
3	VLKAM-3	AICRP (VC)
4	VLKAM-4	AICRP (VC)
5	VLKAM-5	AICRP (VC)
6	VLKAM-6	AICRP (VC)
7	VLKAM-7	AICRP (VC)
8	VLKAM-8	AICRP (VC)
9	VLKAM-9	AICRP (VC)
10	VLKAM-10	AICRP (VC)
11	VLKAM-11	AICRP (VC)
12	VLKAM-12	AICRP (VC)
13	VLKAM-13	AICRP (VC)
14	VLKAM-14	AICRP (VC)
15	VLKAM-15	Kanjikuzhy,Alappuzha
16	VLKAM-16	Thaikkal,Alappuzha
17	VLKAM-17	Palappur,Trivandrum
18	VLKAM-18	Palappur,Trivandrum
19	VLKAM-19	Kalliyoor,Trivandrum
20	VLKAM-20	Kalliyoor,Trivandrum
21	VLKAM-21	Panangad, Ernakulam
22	VLKAM-22	Pallichal,Trivandrum
23	VLKAM-23	Neyyattinkara,Trivandrum
24	VLKAM-24	Amaravila,Trivandrum
25	VLKAM-25	Sulthan Bathery,Wayanad
26	VLKAM-26	Poovachal,Trivandrum
27	VLKAM-27	Perumbuzha,Kollam
28	VLKAM-28	Sreekanteswaram,Trivandrum
29	VLKAM-29	Sreekanteswaram,Trivandrum
30	VLKAM-30	AICRP (VC)
31	VLKAM-31	Kannara,Thrissur
32	VLKAM-32	Sreekanteswaram,Trivandrum

Sl no	Genotypes	Source
33	VLKAM-33	Perumbuzha,Kollam
34	VLKAM-34	Vlathankara,Trivandrum
35	VLKAM-35	Thiruvizhamkunnu,Palakkad
36	VLKAM-36	Thiruvizhamkunnu,Palakkad
37	VLKAM-37	Thiruvizhamkunnu,Palakkad
38	VLKAM-38	Arka Arunima, IIHR
39	VLKAM-39	AICRP (VC)
40	VLKAM-40	Harippad,Alappuzha
41	VLKAM-41	Harippad,Alappuzha
42	VLKAM-42	Harippad,Alappuzha
43	VLKAM-43	Harippad,Alappuzha
44	Arun	Kerala Agricultural University
45	KAU Vaika	Kerala Agricultural University

The response of the genotypes to leaf blight was assessed in terms of disease incidence and disease severity. The percent disease incidence was calculated by using the following formula,

$$\text{Disease incidence (DI) \%} = \frac{\text{No. of plants infected}}{\text{Total no. of plants observed}} \times 100$$

The leaf blight severity on the accessions was scored thirty days after transplanting on fifty randomly selected leaves from each replication, based on a 0 to 9 scale (Nair & Anith, 2009) as given below (Fig. 1),

0: no disease
1: 1 to 10% infected leaf area
3: >11 to 25% infected leaf area
5: >26 to 50% infected leaf area
7: >51-75% infected leaf area
9: >76-100% leaf area infected

Percent disease severity or Percentage Disease Index (PDI) was calculated using the following formula (Wheeler, 1969).

$$\text{Percentage Disease Index (PDI)} = \frac{\text{Sum of grades of each leaf}}{\text{Number of leaves assessed}} \times \frac{100}{\text{Maximum grade used (9)}}$$

Based on percentage disease index, the accessions were grouped into five categories as defined by Rajkumar *et al*. (1995), as follows

Chart 1. Disease resistance scale

0	Immune
1-10%	Highly resistant
10.1-25%	Moderately resistant
25.1-50%	Moderately susceptible
>50%	Highly susceptible

Fig. 1. ***Rhizoctonia*** **leaf blight disease severity scale**

2.2 Artificial Screening of Selected Amaranthus Genotypes for *Rhizoctonia* Leaf Blight Resistance

To validate the resistance observed under natural field conditions, the selected genotypes were subjected to artificial inoculation with *R. solani* in a separate pot culture experiment conducted under controlled conditions. Isolation of the pathogen was carried out from naturally infected amaranthus plants displaying typical leaf blight symptoms. Infected leaf portions were excised into small fragments and surface sterilized using 0.1% mercuric chloride solution for 45 seconds. The fragments were then rinsed thoroughly in sterile distilled water to eliminate residues of the disinfectant. The sterilized segments were then aseptically placed on potato dextrose agar (PDA) medium in sterile Petri dishes and incubated at 28 ± 1 °C. Fungal growth emerging from the infected tissues was sub-cultured onto fresh PDA after 72 hours to establish pure culture. The culture appeared creamy white initially, later turning to light brown (Fig. 2). The mycelia of the isolated fungus were observed for the morphological features after staining with lactophenol cotton blue. The hyphae showed typical 90 degree branching and characteristic constriction and septum at the branching point. The characters were in compliance with those described by Parmeter and whitney (1970) and thus the fungus was identified accordingly as *R. solani.* Pathogenicity was proved on detached leaves of the susceptible cultivar *Arun* and the culture obtained upon re-isolation was identical to the one isolated initially. The isolate was maintained through periodic subculturing on PDA, and the pure culture was utilized for the subsequent experimental procedures.

Amaranthus seedlings grown in the protrays were transplanted to pots containing sterilized potting mixture. Genotypes were grown in separate pots with three replications. The commercial varieties *Arun* and *KAU Vaika* were used as susceptible checks. A non-inoculated control was maintained for each genotype to facilitate comparative assessment. Two weeks after transplanting, lower surface of four selected leaves of each amaranthus genotypes were mildly injured by giving pin pricks over an area of 1 cm^2. Artificial inoculation was performed by placing mycelial bits cut from actively growing fresh culture of the fungal pathogen on the injured area (Fig. 3). Moistened cotton was placed over the inoculated region to ensure adequate moisture. Pots were covered with perforated polythene sheets after inoculation to maintain high humidity for 24 hours. After artificial inoculation, plants were regularly observed for the development and progression of disease symptoms.

2.2.1 Disease Assessment and Categorization

For each genotype, three replications were maintained, and the plants were regularly monitored for symptom development. Lesion size was measured in terms of length and breadth in cm. The lesion area (cm^2) was calculated for each lesion, and the mean lesion area per genotype was computed by averaging the measurements across all replicates. In addition to lesion size measurements, observations were recorded on the number of days to symptom appearance, number of lesions per leaf, and the nature of symptoms, including lesion expansion and coalescence patterns. Since no predefined lesion area based scale exists for *Rhizoctonia* leaf blight in amaranthus, genotypes were classified into five resistance categories under artificial screening, based on mean lesion area and statistical grouping using Duncan's Multiple Range Test (DMRT). The genotype with zero lesion area was classified as immune; those in the lowest DMRT groups were classified as highly resistant; those in the highest groups as highly susceptible; and intermediate groups were categorized as moderately resistant or moderately susceptible. The methodology aligns with previous screening studies utilizing lesion severity and statistical mean separation for resistance grouping reported in cocoa black pod disease and sunflower powdery mildew (Surujdeo-Maharaj *et al*., 2001; Kulkarni *et al*., 2015; Suresha *et al*., 2017).

The resistance categories were defined as follows:

Chart 2. Lesion area categories (cm^2)

Lesion Area (cm^2)	Category
0.00	Immune
0.01–0.12	Highly Resistant
0.13–0.77	Moderately Resistant

Lesion Area (cm²)	Category
0.78–1.26	Moderately Susceptible
>1.26	Highly Susceptible

Fig. 2. a. *Rhizoctonia solani* culture, b. Microscopic view of the fungus

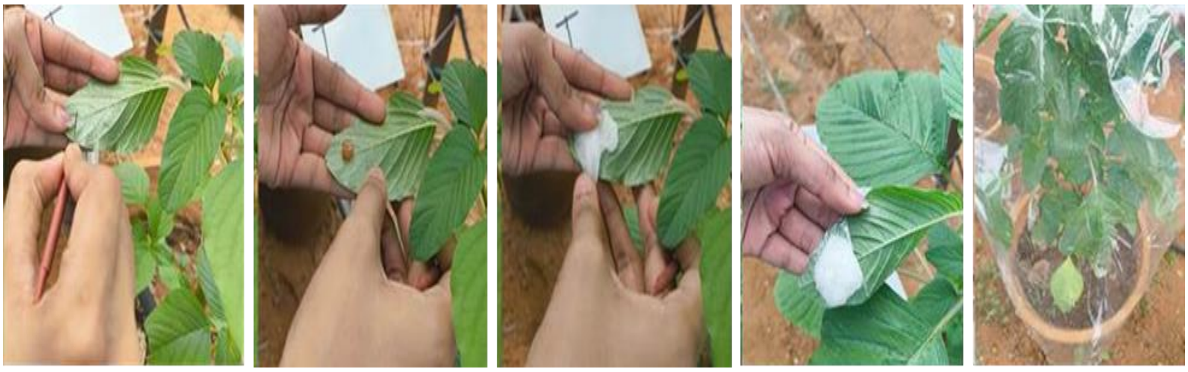

Fig. 3. Artificial inoculation with *R. solani*

3. Results and Discussion

3.1 Field screening of Amaranthus Genotypes for *Rhizoctonia* Leaf Blight Resistance

In the present study, leaf blight disease caused by *R. solani* was assessed across 45 amaranthus genotypes under field conditions during 2024 (Fig. 4) and 2025, revealing a wide range of disease severity. As per the initial field screening during 2024, the Percent Disease incidence (DI%) varied significantly from 0.0% to 100.0% among the 45 genotypes (Table 2). Genotypes such as *VLKAM-25, VLKAM-41, and VLKAM-42* recorded a DI% of 0.0, showing complete absence of symptom expression. *VLKAM-10, 14, 36*, and *40* exhibited a low disease incidence within a range of 20 % to 45%. Eighteen genotypes exhibited high disease incidence, with DI% values ranging from 75 to 100 %. The susceptible checks, *Arun* and *KAU Vaika*, exhibited the highest DI% (100 %), with symptoms appearing on all the field plants, while *VLKAM-1,4,19,38*, and *39* were among the most severely affected, with a DI% of 90.0%.

The Percent Disease Index (PDI) values demonstrated considerable variation, ranging from complete immunity (0.0) to severe infection (88.48), thereby indicating significant variability in disease response among the genotypes (Table 2). Three genotypes, *VLKAM-25*, *VLKAM-41*, and *VLKAM-42,* exhibited complete immunity, with no visible symptoms of infection under field conditions. The genotype *VLKAM-36* displayed a very low PDI (6.44) and was classified as highly resistant. Six genotypes were identified as moderately resistant, with PDIs ranging from 12.13 to 24.21, including *VLKAM-10, VLKAM-14, VLKAM-24, VLKAM-34, VLKAM-35*, and *VLKAM-40.* A substantial proportion of the screened genotypes fell into the susceptible categories. Among them, 15 genotypes demonstrated moderate susceptibility with their PDI values ranging from 26.23(*VLKAM-13*) to 48.34 (*VLKAM-17*). Twenty genotypes exhibited high levels of disease infection with PDIs exceeding 50 and were classified under the highly susceptible category. This group included *VLKAM* accessions with PDI values ranging from 54.12 (*VLKAM-2*) to 85.9 (*VLKAM-19*). Additionally, both the commercial check varieties, *Arun* and *KAU Vaika* with PDIs of 88.48 and 86.7, respectively were extremely susceptible and showed very high disease severity under the field conditions.

Table 2. Field screening of amaranthus genotypes for *Rhizoctonia* leaf blight resistance (*Rabi* 2024)

Sl. N0.	Genotype	Leaf Colour	Disease Incidence (%)	PDI	Disease Reaction
1	VLKAM-1	Green	90.0 (71.57)	85.15 (67.34)	Highly susceptible
2	VLKAM-2	Red	65.0 (53.73)	54.12 (47.37)	Highly susceptible
3	VLKAM-3	Red	70.0 (56.79)	76.55 (61.04)	Highly susceptible
4	VLKAM-4	Red	90.0 (71.57)	84.79 (67.05)	Highly susceptible
5	VLKAM-5	Red	60.0 (50.77)	65.36 (53.95)	Highly susceptible
6	VLKAM-6	Red	70.0 (56.79)	44.58 (41.89)	Moderately susceptible
7	VLKAM-7	Green	80.0 (63.44)	63.73 (52.97)	Highly susceptible
8	VLKAM-8	Green	50.0 (45)	29.52 (32.91)	Moderately susceptible
9	VLKAM-9	Green with dark brown blotch on basal area	80.0 (63.44)	83.65 (66.15)	Highly susceptible
10	VLKAM-10	Green	35.0 (36.28)	12.13 (20.39)	Moderately resistant
11	VLKAM-11	Green	50.0 (45)	31.19 (33.96)	Moderately susceptible
12	VLKAM-12	Green	50.0 (45)	27.15 (31.41)	Moderately susceptible
13	VLKAM-13	Green	50.0 (45)	26.23 (30.81)	Moderately susceptible
14	VLKAM-14	Green	40.0 (39.24)	12.53 (20.74)	Moderately resistant
15	VLKAM-15	Red	70.0 (56.79)	60.45 (51.03)	Highly susceptible
16	VLKAM-16	Red	70.0 (56.79)	74.84 (59.9)	Highly susceptible
17	VLKAM-17	Red	80.0 (63.44)	48.34 (44.06)	Moderately susceptible
18	VLKAM-18	Green	80.0 (63.44)	62.12 (52.02)	Highly susceptible
19	VLKAM-19	Red	90.0 (71.57)	85.9 (67.95)	Highly susceptible
20	VLKAM-20	Purple red	60.0 (50.77)	29.43 (32.86)	Moderately susceptible
21	VLKAM-21	Green	75.0 (60)	70.53 (57.13)	Highly susceptible
22	VLKAM-22	Purple red	75.0 (60)	29.52 (32.91)	Moderately susceptible
23	VLKAM-23	Red	80.0 (63.44)	56.48 (48.73)	Highly susceptible
24	VLKAM-24	Purple red	55.0 (47.87)	24.21 (29.48)	Moderately resistant
25	VLKAM-25	Reddish green	0.0 (9.09)	0.0 (0.0)	Immune
26	VLKAM-26	Red	70.0 (56.79)	32.35 (34.67)	Moderately susceptible
27	VLKAM-27	Green with dark	75.0 (60)	32.31 (34.65)	Moderately

Sl. N0.	Genotype	Leaf Colour	Disease Incidence (%)	PDI	Disease Reaction
		brown blotch on basal area			susceptible
28	VLKAM-28	Green with dark brown blotch on basal area	70.0 (56.79)	28.69 (32.39)	Moderately susceptible
29	VLKAM-29	Light green	65.0 (53.73)	33.21 (35.19)	Moderately susceptible
30	VLKAM-30	Green	85.0 (67.22)	71.75 (57.9)	Highly susceptible
31	VLKAM-31	Red	80.0 (63.44)	68.66 (55.96)	Highly susceptible
32	VLKAM-32	Green	75.0 (60)	59.73 (50.62)	Highly susceptible
33	VLKAM-33	Light green	65.0 (53.73)	32.25 (34.61)	Moderately susceptible
34	VLKAM-34	Purple red	50.0 (45)	17.41 (24.67)	Moderately resistant
35	VLKAM-35	Red	65.0 (53.73)	24.12 (29.42)	Moderately resistant
36	VLKAM-36	Reddish green	20.0 (26.57)	6.44 (14.71)	Highly resistant
37	VLKAM-37	Red	80.0 (63.44)	39.24 (38.79)	Moderately susceptible
38	VLKAM-38	Red	90.0 (71.57)	80.78 (64.0)	Highly susceptible
39	VLKAM-39	Red	90.0 (71.57)	80.13 (63.53)	Highly susceptible
40	VLKAM-40	Purple red	45.0 (42.14)	20.58 (26.98)	Moderately resistant
41	VLKAM-41	Green	0.0 (9.09)	0.0 (4.05)	Immune
42	VLKAM-42	Green	0.0 (9.09)	0.0 (4.05)	Immune
43	VLKAM-43	Purple red	60.0 (50.77)	35.23 (36.41)	Moderately susceptible
44	Arun	Red	100.0 (80.9)	88.48 (70.16)	Highly susceptible
45	KAU Vaika	Red	100.0 (80.9)	86.7 (68.62)	Highly susceptible
	CD(0.05)		9.8	8.79	
	C.V.		9.02	10.32	

**Data in parenthesis are arcsine transformed values*

Fig. 4. Field screening of amaranthus genotypes

The evaluated genotypes displayed noticeable variation in leaf colour, which ranged from green, light green, reddish green, and red to purple red. Green foliage types displayed a range of disease reactions, from complete immunity (*VLKAM-41, VLKAM-42*) to high susceptibility (*VLKAM-1, VLKAM-7, VLKAM-18* & *VLKAM-30*). A set of green genotypes including *VLKAM-8, VLKAM-11, VLKAM-12, VLKAM-13,* and *VLKAM-27* showed intermediate responses with DI of 50–80% and moderate PDI (26.23 - 32.31). Majority of the red genotypes were classified under the susceptible category. Genotypes like *VLKAM-2, VLKAM-3, VLKAM-4, VLKAM-5, VLKAM-15, VLKAM-16, VLKAM-19, VLKAM-23, VLKAM-31, VLKAM-38, VLKAM-39, Arun,* and *KAU Vaika* recorded high DI (65-100%) and PDI (54.12-88.48). A smaller group (*VLKAM-6, VLKAM-17, VLKAM-26,* and *VLKAM-37*) showed moderate susceptibility with DI of 60–80% and PDI between 32.35 and 48.34. Only the accession *VLKAM-35* recorded a relatively low PDI (24.12) and was categorized as moderately resistant.

Reddish green leaf types generally showed a resistant reaction. Since no disease symptoms appeared in case of the accession *VLKAM-25*, it was categorized as completely immune, while *VLKAM-36* showing only 20% disease incidence and a very low PDI (6.44), was grouped as highly resistant. Among the purple red genotypes, *VLKAM-20, VLKAM-22,* and *VLKAM-43* were moderately susceptible, with DI of 60-75% and PDI values ranging from 29.43 to 35.23. In contrast, *VLKAM-24, VLKAM-34,* and *VLKAM-40* exhibited moderate resistance with DI values between 45 and 55% and comparatively low PDI (17.41- 24.21). Light green genotypes (*VLKAM-29* and *VLKAM-33*) were moderately susceptible with PDI values of 33.21 and 32.25 respectively.

Among the 45 genotypes evaluated during the year 2024, *VLKAM-25*, *VLKAM-41*, and *VLKAM-42* displayed complete immunity to leaf blight under field conditions, while *VLKAM-36* exhibited a highly resistant reaction with a Percent Disease Index (PDI) of 6.44. Six genotypes, including *VLKAM-10, VLKAM-14, VLKAM-24, VLKAM-34, VLKAM-35,* and *VLKAM-40*, were categorized as moderately resistant, with PDIs ranging from 12.13 to 24.21, whereas the susceptible checks Arun and KAU Vaika recorded severe infection with PDIs exceeding 85.

The field screening of forty five amaranthus genotypes in 2025 revealed wide variation in per cent disease incidence (DI%) and per cent disease index (PDI), (Table 3). Disease incidence ranged from 0 to 100%, while the percent disease index (PDI) varied between 0.0 and 96.66. Three accessions (*VLKAM-25, VLKAM-41,* and *VLKAM-42*) remained completely free from infection. These accessions recorded zero disease incidence and zero PDI, and were therefore classified as immune. The genotype *VLKAM-36* displayed a highly resistant response with a low DI of 20% and a PDI of 8.52. These results were identical to that observed during the preliminary field screening in 2024, confirming the stability of their resistance. The genotype *VLKAM-14* expressed a moderately resistant reaction with a PDI of 23.81 and 50% DI. The moderately susceptible category comprised eleven accessions, which recorded PDI values ranging from 31.58 to 48.22. The remaining twenty nine accessions, including the checks *Arun* and *KAU Vaika* exhibited high susceptibility with disease incidence ranging from 80 to 100% and PDI values extended from 55.70 to 96.66. The highest disease severity was observed in *VLKAM-19* (96.66), followed by *Arun* (94.63) and *VLKAM-17* (91.35).

Overall, the results indicated that while most of the genotypes are highly vulnerable to *R. solani*, a few promising sources of resistance exist, particularly the immune lines (*VLKAM-25, VLKAM-41, VLKAM-42*) and the highly resistant genotype *VLKAM-36*, which may be valuable in future breeding works.

3.2 Artificial Screening of Selected Amaranthus Genotypes for *Rhizoctonia* Leaf Blight Resistance

Out of the 45 amaranthus genotypes screened under natural field conditions, ten genotypes were found to be resistant with comaparitively lower PDI values. In order to validate the resistance to *R. solani*, the amaranthus accessions that were found to be resistant in field screening were screened *in-vitro* with artificial inoculation. The commercial varieties *Arun* and *KAU Vaika* were included in the artificial screening trial as susceptible checks. Early symptom development was observed in *VLKAM-24* as well as in the susceptible checks, *Arun* and *KAU Vaika*, 2 dpi (days post inoculation) (Table 3) suggesting the rapid establishment of disease. Remaining genotypes developed symptoms within 3- 4 dpi except *VLKAM-41* which remained symptomless throughout the experiment period.

Table 3. Field screening of amaranthus genotypes for *Rhizoctonia* leaf blight resistance (*Kharif* 2025)

Sl. No.	Genotype	Disease Incidence (%)	PDI	Disease Reaction
1	VLKAM-1	95.0 (77.08)	87.76 (69.53)	Highly susceptible
2	VLKAM-2	80.0 (63.44)	65.83 (54.23)	Highly susceptible
3	VLKAM-3	80.0 (63.44)	71.14 (57.51)	Highly susceptible
4	VLKAM-4	80.0 (63.44)	68.86 (56.09)	Highly susceptible
5	VLKAM-5	80.0 (63.44)	72.33 (58.27)	Highly susceptible
6	VLKAM-6	90.0 (71.57)	83.88 (66.33)	Highly susceptible
7	VLKAM-7	80.0 (63.44)	74.24 (59.5)	Highly susceptible
8	VLKAM-8	60.0 (50.77)	31.58 (34.2)	Moderately susceptible
9	VLKAM-9	100.0 (80.9)	88.38 (70.07)	Highly susceptible
10	VLKAM-10	70.0 (56.79)	40.76 (39.68)	Moderately susceptible
11	VLKAM-11	70.0 (56.79)	42.21 (40.52)	Moderately susceptible
12	VLKAM-12	70.0 (56.79)	37.85 (37.97)	Moderately susceptible
13	VLKAM-13	75.0 (60.0)	47.38 (43.5)	Moderately susceptible
14	VLKAM-14	50.0 (45.0)	23.81 (29.21)	Moderately resistant
15	VLKAM-15	90.0 (71.57)	84.88 (67.12)	Highly susceptible
16	VLKAM-16	90.0 (71.57)	82.16 (65.02)	Highly susceptible
17	VLKAM-17	100.0 (80.9)	91.35 (72.9)	Highly susceptible
18	VLKAM-18	80.0 (63.44)	79.73 (63.25)	Highly susceptible
19	VLKAM-19	100.0 (80.9)	96.66 (79.47)	Highly susceptible
20	VLKAM-20	80.0 (63.44)	57.31 (49.21)	Highly susceptible
21	VLKAM-21	90.0 (71.57)	85.11 (67.31)	Highly susceptible
22	VLKAM-22	70.0 (56.79)	62.22 (52.08)	Highly susceptible
23	VLKAM-23	90 .0 (71.57)	81.65 (64.64)	Highly susceptible
24	VLKAM-24	70.0 (56.79)	48.22 (43.98)	Moderately susceptible
25	VLKAM-25	0.0 (9.09)	0.0 (4.05)	Immune
26	VLKAM-26	90.0 (71.57)	84.87 (67.11)	Highly susceptible
27	VLKAM-27	85.0 (67.22)	82.77 (65.48)	Highly susceptible
28	VLKAM-28	80.0 (63.44)	80.27 (63.63)	Highly susceptible
29	VLKAM-29	80.0 (63.44)	80.98 (64.15)	Highly susceptible
30	VLKAM-30	80.0 (63.44)	78.08 (57.51)	Highly susceptible
31	VLKAM-31	80.0 (63.44)	55.7 (48.28)	Highly susceptible
32	VLKAM-32	70.0 (56.79)	73.41 (58.96)	Highly susceptible
33	VLKAM-33	75.0 (60.0)	73.82 (59.23)	Moderately susceptible
34	VLKAM-34	80.0 (63.44)	38.28 (38.23)	Moderately susceptible
35	VLKAM-35	70.0 (56.79)	40.22 (39.37)	Moderately susceptible
36	VLKAM-36	20.0 (26.57)	8.52 (16.98)	Highly resistant
37	VLKAM-37	70.0 (56.79)	56.81 (48.92)	Highly susceptible
38	VLKAM-38	95.0 (77.08)	86.25 (68.24)	Highly susceptible
39	VLKAM-39	95.0 (77.08)	85.55 (67.66)	Highly susceptible
40	VLKAM-40	60.0 (50.77)	35.04 (36.3)	Moderately susceptible
41	VLKAM-41	0.0 (9.09)	0.0 (4.05)	Immune
42	VLKAM-42	0.0 (9.09)	0.0 (4.05)	Immune
43	VLKAM-43	70.0 (56.79)	43.82 (41.46)	Moderately susceptible
44	Arun	100.0 (80.9)	94.63 (76.61)	Highly susceptible
45	KAU Vaika	90.0 (71.57)	83.93 (66.37)	Highly susceptible
	CD(0.05)	9.77	9.58	
	C.V.	8.02	9.10	

**Data in parenthesis are arcsine transformed values*

Lesion counts ranged from 0 (*VLKAM-41*) to 5 (*KAU Vaika* and *VLKAM-10*) (Table 3). The accessions with fewer lesions, such as *VLKAM-34, VLKAM-25* and *VLKAM-42* displayed restricted disease proliferation. Remarkably, genotype *VLKAM-41* exhibited complete absence of lesions across replications and observation periods. The twelve amaranthus genotypes showed significant variation in lesion size (length × breadth), which suggests varying degrees of resistance or susceptibility. Arun and KAU Vaika, the susceptible checks, had the most extensive lesion development, with lesions of 1.7-2.1 × 1.1-1.7 cm and 1.8-2.3 × 1.4-1.8 cm, respectively. In contrast, the genotypes *VLKAM-25*, *VLKAM-36*, and *VLKAM-42* exhibited very small sized lesions.

Mean lesion area among the genotypes spanned from 0 cm² (*VLKAM-41*) to 2.64 cm² (KAU V*aika*) (Table 3). The lesion area values recorded under artificial screening experiment were statistically analyzed, and the genotypes were grouped using DMRT at 5% significance level. The DMRT analysis categorized the twelve genotypes into eight statistically distinct groups, reflecting significant variation in lesion development among the tested genotypes (Table 3). This indicates differential levels of resistance to *R. solani* under controlled conditions. Genotypes were classified into five resistance categories under artificial screening (Immune, HR, MR, MS, HS), based on lesion area and DMRT grouping. The genotype *VLKAM-41* which displayed no symptoms can be categorised as immune to leaf blight under artificial conditions. The genotypes, *VLKAM-25* and *VLKAM-42* produced small localised lesions strictly restricted to the inoculation site whereas *VKLAM-36* developed localized lesions with mild expansion. These lesions did not coalesce, and the surrounding tissues remained unaffected, indicating restricted fungal growth.

Genotypes *VLKAM-25*, *VLKAM-42*, and *VLKAM-36*, which recorded mean lesion areas ranging from 0.05 to 0.12 cm², were classified under the highly resistant category. These genotypes were statistically on par with each other and exhibited significantly lower lesion areas than all other genotypes. They developed only pin-point lesions localized to the inoculation site indicating an effective hypersensitive response to pathogen spread. The accessions *VLKAM-34, VLKAM- 14, VLKAM-10* and *VLKAM-24* exhibited small lesions with limited expansion and were classified under moderately resistant group based on the mean lesion area spanning from 0.22 to 0.77 cm². Although some necrotic spots developed, they did not coalesce, suggesting partial resistance in these genotypes. The accessions *VLKAM-40* and *VLKAM-35* developed coalescing lesions with mean lesion areas of 1.06 cm² and 1.26 cm², respectively and were therefore categorized as moderately susceptible. These genotypes exhibited noticeable lesion expansion and moderate leaf tissue damage. Arun and KAU Vaika, recorded the highest mean lesion areas of 2.24 cm², 2.64 cm², respectively and were classified under highly susceptible category. They exhibited severe coalescing lesions and rapid spread of the leaf blight.

Artificial screening validated findings of field level screening as*VLKAM-41* remained symptomless, confirming its immune reaction to the leaf blight. Genotypes such as *VLKAM-25*, *VLKAM-36*, and *VLKAM-42* exhibited localized lesions that did not expand beyond the inoculation point, indicating effective restriction of fungal colonization. In contrast, the susceptible checks displayed large coalescing lesions with mean lesion areas exceeding 2.2 cm². Based on the IBPGR amaranthus descriptors (Grubben & van Sloten, 1981), the immune genotype *VLKAM-41* was identified as *Amaranthus spinosus* while the highly resistant genotype *VLKAM-42* belonged to *A. dubius*, and *VLKAM-25* along with *VLKAM-36* were classified under *A. hypochondriacus*. These findings also align with earlier reports by Celine *et al.* (2013), who screened 89 amaranth accessions and classified *Amaranthus dubius* and *A*. *hypochondriacus* accessions as immune or highly resistant, whereas *A. tricolor* genotypes displayed variable susceptibility.

Krishnakumary *et al.* (2001), also identified immune and highly resistant genotypes to leaf blight among diverse amaranthus germplasm. Similarly, Sindhu (2002) conducted an experiment using 32 accessions of *A. dubius* and the variety '*Arun*' *(A. tricolor)* as the susceptible check and categorised genotypes as immune, highly resistant and moderately resistant. The consistent resistance expression under both screening conditions suggests that resistance is genetically stable and not strongly influenced by the environmental factors. Similar trends have been reported in Brachiaria, where accessions resistant to *R. solani* under field conditions maintained their performance under controlled inoculation (Alvarez *et al*., 2013). Slight differences observed in lesion development under artificial conditions in certain genotypes compared to field may be attributed to higher inoculum pressure, high relative humidity, and optimal temperature favouring pathogen

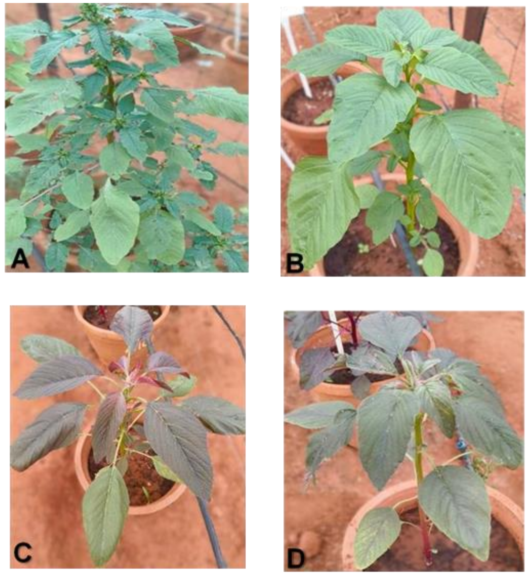

Fig. 5. Best performing genotypes after artificial inoculation- A. VLKAM-41 (Immune) B. VLKAM-42 (Highly resistant) C. VLKAM-25 (Highly resistant) D. VLKAM-36 (Highly resistant)

Table 4. Artificial screening of selected amaranthus genotypes

Sl. No.	Genotype	Days to Symptom Development	No. of Lesions	Lesion Size (L x B in cm)	Mean Lesion Area (cm^2)	Nature of Symptom	DMRT Group	Disease Reaction
1	VLKAM-10	3	5	0.4-1 x 0.3-0.5	0.61	Moderately expanding lesions	ef	MR
2	VLKAM-14	3	4	0.4-0.9 x 0.2-0.3	0.42	Limited lesion expansion	f	MR
3	VLKAM-24	2	4	0.8-1.4 x 0.3-0.6	0.77	Expanding lesions	e	MR
4	VLKAM-25	3	3	0.1-0.4 x 0.1-0.3	0.05	Localized lesions restricted to inoculation site	gh	HR
5	VLKAM-34	3	3	0.6-0.8 x 0.3-0.4	0.22	Small, discrete lesions	g	MR
6	VLKAM-35	3	4	1.2-1.8 x 0.8-1.1	1.26	Coalescing lesions	c	MS
7	VLKAM-36	4	4	0.5-0.6 x 0.2-0.3	0.12	Localized lesions with mild expansion	gh	HR
8	VLKAM-40	3	4	1.2-1.7 x 0.7-1	1.06	Coalescing lesions	d	MS
9	VLKAM-41	0	0	0	0	No visible symptoms	h	I
10	VLKAM-42	4	3	0.3-0.4 x 0.2-0.3	0.09	Localized lesions restricted to inoculation site	gh	HR
11	Arun	2	4	1.7-2.1 x 1.1-1.7	2.24	Severe and coalescing lesions	b	HS
12	KAU Vaika	2	5	1.8-2.3 x 1.4-1.8	2.64	Severe and coalescing lesions	a	HS

proliferation in artificial setups (Ajayi-Oyetunde & Bradley, 2018).

Resistance mechanisms to *R. solani* are complex and involves activation of defense pathways within the host plant to curb the disease development. In the present study, resistant genotypes showed restricted lesion expansion, suggesting hypersensitive responses. According to the studies of Copley *et al*. (2017), resistance to *Rhizoctonia* foliar blight in soybean is governed by multiple defense mechanisms involving hormonal signaling, oxidative responses, and pathogenesis-related proteins. Jasmonic acid (JA)-mediated pathways plays a major role in defense against necrotrophic pathogens such as *R. solani*, by activating lipoxygenase (*LOX*) genes and phenylalanine ammonia-lyase (*PAL1*) pathways which lead to lignification and phenolic accumulation for cell wall strengthening.

Plant defense responses mediated by phytohormones exhibit variability in disease response against *R. solani*, influenced by the infection site, isolate differences and host species. Research studies indicate that different isolates of *R. solani* employ unique infection strategies that plants counter through either SA or JA-dependent pathways, irrespective of their AG classification (Kouzai *et al*., 2018; Koley *et al*., 2022; Taheri & Tarighi, 2010; He *et al*., 2023). In addition, pathogenesis-related (PR) proteins, including chitinases and glucanases, play a critical role in degrading fungal cell walls. Additionally, an early oxidative burst, mediated by reactive oxygen species (ROS), is a critical component of host defense as it restricts fungal spread and triggers hypersensitive-like responses (Taheri & Tarighi, 2011; Samsatly *et al*., 2018). From a breeding perspective, the identification of immune and highly resistant genotypes provides promising donor sources for introgression of resistance into susceptible varieties. However, conventional breeding faces challenges such as linkage drag, poly genic nature of resistance and limited genetic resources. Hence, molecular breeding approaches such as marker-assisted selection (MAS), QTL pyramiding, RNA interference and genome editing offer promising solutions (Senapati *et al*., 2022). These modern breeding strategies have been successfully employed in cereals to improve sheath blight resistance (Chen *et al*., 2023; Senapati *et al*., 2022) and can be adapted for amaranthus breeding initiatives.

4. Conclusion

The present study revealed pronounced variability in disease response among amaranthus genotypes to *R. solani* leaf blight under natural and artificial inoculation conditions. The genotype *VLKAM-41* was completely immune and three entries (*VLKAM-25, VLKAM-42,VLKAM-36*) exhibited a high resistance reaction to the disease. The immune and highly resistant genotypes identified in this study serve as valuable donor sources in resistance breeding programmes for developing leaf blight resistant cultivars. Considering the extensive genetic and pathogenic variability in *R. solani* populations, future research should focus on validating resistance against multiple isolates across diverse environments. Integrating the resistant sources with advanced breeding approaches such as marker-assisted selection, QTL mapping, and genome editing can accelerate the development of resistant varieties. A holistic approach that combines resistant genotypes with advanced breeding methods and eco-friendly management practices, including biological control will enable sustainable management of leaf blight in amaranthus and enhance its role as a climate resilient, nutrient-rich crop for future food systems.

Disclaimer (Artificial Intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

Acknowledgements

Authors are grateful to Kerala Agricultural University, Thrissur, Kerala, India for the financial support and Department of Vegetable Science, College of Agriculture, Vellanikkara for providing necessary facilities to carry out this study.

Competing Interests

Authors have declared that no competing interests exist.

References

Ajayi-Oyetunde, O. O., & Bradley, C. A. (2018). *Rhizoctonia solani*: Taxonomy, population biology and management of rhizoctonia seedling disease of soybean. *Plant Pathology, 67*(1), 3–17.

Alvarez, E., Latorre, M., Bonilla, X., Sotelo, G., & Miles, J. W. (2013). Diversity of *Rhizoctonia* spp. causing foliar blight on *Brachiaria* in Colombia and evaluation of *Brachiaria* genotypes for foliar blight resistance. *Plant Disease, 97*(6), 772–779.

Anderson, N. A. (1982). The genetics and pathology of *Rhizoctonia solani*. *Annual Review of Phytopathology, 20*, 329–347.

Carling, D. E., Baird, R. E., Gitaitis, R. D., Brainard, K. A., & Kuninaga, S. (2002a). Characterization of AG-13, a newly reported anastomosis group of *Rhizoctonia solani*. *Phytopathology, 92*, 893–899.

Carling, D. E., Kuninaga, S., & Brainard, K. A. (2002b). Hyphal anastomosis reactions, rDNA-internal transcribed spacer sequences, and virulence levels among subsets of *Rhizoctonia solani* anastomosis group-2 (AG-2) and AG-BI. *Phytopathology, 92*, 43–50.

Celine, V. A., Girija, V. K., Sreelathakumary, I., & Vahab, M. A. (2013). Selection of amaranth genotypes for resistance to *Rhizoctonia solani*. *International Journal of Vegetable Science, 19*, 157–163.

Chahal, K. S., Sokhi, S. S., & Rattan, G. S. (2003). Investigations on sheath blight of rice in Punjab. *Indian Phytopathology, 56*(1), 22–26.

Chen, J., Xuan, Y., Yi, J., Xiao, G., Yuan, D. P., & Li, D. (2023). Progress in rice sheath blight resistance research. *Frontiers in Plant Science, 14*, 1141697. https://doi.org/10.3389/fpls.2023.1141697

Copley, T., Bayen, S., & Jabaji, S. (2017). Biochar amendment modifies expression of soybean and *Rhizoctonia solani* genes leading to increased severity of *Rhizoctonia* foliar blight. *Frontiers in Plant Science, 8*, 221. https://doi.org/10.3389/fpls.2017.00221

Gokulapalan, C., Nayar, K., & Umamaheshwaran, K. (2000). Foliar blight of amaranthus caused by *Rhizoctonia solani* Kühn. *Journal of Mycology and Plant Pathology, 30*, 131–132.

Grubben, G., & van Sloten, D. (1981). *Genetic resources of amaranth—a global plan of action*. IBPGR, Wageningen, 62p.

He, Y., Zhang, K., Li, S., Lu, X., Zhao, H., Guan, C., et al. (2023). Multiomics analysis reveals the molecular mechanisms underlying virulence in *Rhizoctonia* and jasmonic acid–mediated resistance in Tartary buckwheat (*Fagopyrum tataricum*). *The Plant Cell, 35*(8), 2773–2798. https://doi.org/10.1093/plcell/koac180

KAU [Kerala Agricultural University]. (2024). *Package of practices recommendations: Crops* (16th ed.). Kerala Agricultural University, Thrissur, 435p.

Koley, P., Brahmachari, S., Saha, A., Deb, C., Mondal, M., Das, N., et al. (2022). Phytohormone priming of tomato plants evoke differential behavior in *Rhizoctonia solani* during infection, with salicylate priming imparting greater tolerance than jasmonate. *Frontiers in Plant Science, 12*, 766095. https://doi.org/10.3389/fpls.2021.766095

Kouzai, Y., Kimura, M., Watanabe, M., Kusunoki, K., Osaka, D., Suzuki, T., et al. (2018). Salicylic acid-dependent immunity contributes to resistance against *Rhizoctonia solani*, a necrotrophic fungal agent of sheath blight, in rice and *Brachypodium distachyon*. *New Phytologist, 217*(2), 771–783. https://doi.org/10.1111/nph.14854

Krishnakumary, K. (2000). *Genotypic and seasonal influence on leaf spot disease in amaranth* [PhD thesis, Kerala Agricultural University].

Kulkarni, V. V., Shankergoud, I., & Govindappa, M. R. (2015). Identification of sunflower powdery mildew resistant sources under artificial screening. *SABRAO Journal of Breeding and Genetics, 47*(4), 502–509.

Maughan, P., Smith, S., Fairbanks, D., & Jellen, E. (2011). Development, characterization, and linkage mapping of single nucleotide polymorphisms in the grain *Amaranthus* (*Amaranthus* spp.). *The Plant Genome, 4*, 92–101.

Nair, C. B., & Anith, K. N. (2009). Efficacy of acibenzolar-S-methyl and rhizobacteria for the management of leaf blight disease of amaranth. *Journal of Tropical Agriculture, 47*, 43–47.

Nayar, K., Gokulapalan, C., & Nair, C. (1996). A new foliar blight of amaranthus caused by *Rhizoctonia solani*. *Indian Phytopathology, 49*(4), 407.

Ogoshi, A. (1987). Ecology and pathogenicity of anastomosis and intraspecific groups of *Rhizoctonia solani* Kühn. *Annual Review of Phytopathology*, *25*, 125–143.

Parmeter, J. R. J., Sherwood, R. T., & Platt, W. D. (1969). Anastomosis grouping among isolates of *Thanatephorus cucumeris*. *Phytopathology*, *59*, 1270–1278.

Parmeter, J. R., & Whitney, H. S. (1970). Taxonomy and nomenclature of the imperfect state. In J. R. Parmeter (Ed.), *Rhizoctonia solani: Biology and Pathology* (pp. 7–19). University of California Press.

Rajkumar, G., Kaloo, & Pandey, P. K. (1995). Resistance in cowpea to *Pseudocercospora*. In *Proceedings of the National Symposium on Recent Developments in Vegetable Improvement*, Raipur, 2–5 February 1995 (pp. 23–24).

Roy, A. K. (1993). Sheath blight of rice in India. *Indian Phytopathology*, *46*, 197–205.

Samsatly, J., Copley, T. R., & Jabaji, S. H. (2018). Antioxidant genes of plants and fungal pathogens are distinctly regulated during disease development in different *Rhizoctonia solani* pathosystems. *PLoS One*, *13*(2), e0192682. https://doi.org/10.1371/journal.pone.0192682

Sarker, U., Islam, M. T., Rabbani, M. G., & Oba, S. (2015). Genotype variability in composition of antioxidant vitamins and minerals in vegetable amaranth. *Genetika*, *47*, 85–96.

Sarker, U., Islam, T., Rabbani, G., & Oba, S. (2018). Antioxidant leaf pigments and variability in vegetable amaranth. *Genetika*, *50*(1), 209–220.

Schafleitner, R., Lin, Y. P., Dinssa, F. F., N'Danikou, S., Finkers, R., Minja, R., et al. (2022). The World Vegetable Center *Amaranthus* germplasm collection: Core collection development and evaluation of agronomic and nutritional traits. *Crop Science*, *62*(3), 1173–1187. https://doi.org/10.1002/csc2.20720

Schmid, R., Judd, W. S., & Campbell, C. S. (2007). *Plant systematics: A phylogenetic approach*. *Taxon*, *56*(4), 1316.

Senapati, M., Tiwari, A., Sharma, N., Chandra, P., Bashyal, B. M., Ellur, R. K., et al. (2022). *Rhizoctonia solani* Kühn pathophysiology: Status and prospects of sheath blight disease management in rice. *Frontiers in Plant Science*, *13*, 881116. https://doi.org/10.3389/fpls.2022.881116

Sindhu, L. (2002). *Variability in vegetable amaranth (Amaranthus dubius Mart. ex. Thell.) for yield, quality and resistance to leaf blight* (M.Sc. thesis). Department of Olericulture, Kerala Agricultural University, Thrissur, India.

Suresha, P. G., Kulkarni, V. V., Supriya, S. M., & Govindappa, M. R. (2017). Evaluation of experimental hybrids for powdery mildew tolerance in sunflower (*Helianthus annuus* L.). *International Journal of Current Microbiology and Applied Sciences*, *6*(7), 2716–2728.

Surujdeo-Maharaj, S., Umaharan, P., & Iwaro, A. D. (2001). A study of genotype-isolate interaction in cacao (*Theobroma cacao* L.): Resistance of cacao genotypes to isolates of *Phytophthora palmivora*. *Euphytica*, *118*, 295–303.

Taheri, P., & Tarighi, S. (2010). Riboflavin induces resistance in rice against *Rhizoctonia solani* via jasmonate-mediated priming of phenylpropanoid pathway. *Journal of Plant Physiology*, *167*(3), 201–208.

Taheri, P., & Tarighi, S. (2011). A survey on basal resistance and riboflavin-induced defense responses of sugar beet against *Rhizoctonia solani*. *Journal of Plant Physiology*, *168*(10), 1114–1122.

Wheeler, B. E. J. (1969). *An introduction to plant diseases*. John Wiley and Sons Ltd.

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of the publisher and/or the editor(s). This publisher and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.

© Copyright (2026): Author(s). The licensee is the journal publisher. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
https://pr.sdiarticle5.com/review-history/145282