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Resistance screening of *Luffa aegyptiaca* genotypes to *Fusarium* cf. *proliferatum*

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Abstract

Luffa (*Luffa aegyptiaca*) is an economically important crop cultivated for its vegetable, fibrous, and bioactive properties, yet its productivity is severely constrained by Fusarium diseases. This study aimed to evaluate the resistance responses of eight luffa genotypes—comprising five indigenous Iranian accessions (Northern Black Seed, Angled, Northern White Seed, Touri, and Northern Large) and three non-indigenous accessions (Afghani, Long luffa, and Brazilian)—against a *Fusarium* isolate obtained from melon fields in Iran. Pathogenicity assays were conducted using the root-dip inoculation method with a conidial suspension (1×10^6 spores mL^{-1}), and disease progression was monitored for two months. Disease severity was quantified using disease index (DI) and area under the disease progress curve (AUDPC). Significant genotypic differences in disease response were observed. Three genotypes—Brazilian, Northern Black Seed, and Northern White Seed—exhibited complete resistance (DI = 0%; AUDPC = 0), showing no visible symptoms throughout the evaluation period. Three additional genotypes (Angled, Long luffa, and Afghani) demonstrated partial resistance with low DI values (0–15%), while Northern Large and Touri were classified as sensitive, exhibiting DI values of 64% and 44%, respectively. No genotype displayed highly sensitive responses. These findings identify valuable sources of genetic resistance, particularly the completely resistant indigenous genotypes, which represent promising candidates for integration into breeding programs aimed at developing Fusarium-resistant luffa cultivars. The resistant accessions may harbour effective defense mechanisms, including physical and chemical barriers, that restrict pathogen establishment and progression. This study provides foundational knowledge for sustainable luffa production through genetic improvement and emphasizes the necessity of conserving and utilizing indigenous germplasm as reservoirs of disease resistance traits.

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Introduction

Luffa (*L. aegyptiaca* Mill syn. *Luffa cylindrica* (L.) Roem; Cucurbitaceae) is an annual climbing crop of considerable economic and industrial importance. Beyond its traditional use as a vegetable, luffa fibers and extracts are widely utilized in pharmaceutical, cosmetic, orthopedic, paper, and decorative industries owing to their biodegradable, fibrous, and bioactive properties (Reyes-Prezas et al., 2025; Alhijazi et al., 2020). The crop is primarily cultivated in tropical and subtropical regions, where favorable climatic conditions support large-scale production (Oboh & Aluyor, 2009). Despite its versatility and growing demand, luffa productivity and product quality are severely constrained by plant diseases, leading to substantial economic losses.

Luffa is susceptible to a wide range of fungal pathogens, several of which cause destructive diseases throughout the crop cycle. Previous studies have documented infections caused by *Alternaria alternata*, *Rhizoctonia solani*, *Pythium theobromae*, *Pythium aphanidermatum*, *Verticillium dahlia*, *Fusarium* spp., and waxy rot caused by *Geotrichum candidum* (Manthachitra, 1971; Richardson, 1979; Shakir & Mirza, 1992; Abdel-Kader et al., 2024; Delaramifar et al., 2023; Shirzaei et al.,

2025; Miri et al., 2026). Among these, *Fusarium*-induced diseases are particularly damaging due to persistence in agricultural fields, and difficulty of management.

Fusarium wilt is among the most economically important diseases affecting cucurbit crops worldwide, causing vascular discoloration, plant wilting, yield losses, and, in severe cases, total crop failure (Egel & Martyn, 2007; Sharma & Shukla, 2021). In luffa and other cucurbits, several fungal pathogens have been reported, including *Alternaria alternata*, *Rhizoctonia solani*, *Pythium theobromae*, *Fusarium* spp., and waxy rot caused by *Geotrichum candidum* (Manthachitra, 1971; Richardson, 1979; Shakir & Mirza, 1992; Naureen et al., 2009). Fusarium wilt, a soilborne disease with major economic consequences for cucurbit production, is caused by multiple *Fusarium* species that differ in host range and specialization (Sharma & Arora, 2016; Asma et al., 2018). *Fusarium oxysporum* formae speciales typically exhibit high host specificity within *Cucurbitaceae* and other plant families; however, the *F. oxysporum* species complex has been segregated into several distinct species in recent years (Lombard et al. 2019; Irandegani et al., 2023). *Fusarium solani* is recognized as an important pathogen with a broad host range, infecting numerous plant species, including cucurbits, albeit with lower host specialization (Nelson et al., 1981; Coleman, 2016). The ecological adaptability and long-term persistence of *F. solani* in soil further complicate effective disease management strategies in cucurbit cropping systems.

The management of Fusarium wilt relies on an integrated approach, including crop rotation, soil disinfestation, and chemical or biological control; however, these methods often provide inconsistent results and may pose environmental concerns (Fravel et al., 2003; Gordon, 2017). Consequently, recent methods including green synthesized zinc oxide nanoparticles (Jomeyazdian et al., 2024), application of phytohormone to enhance resistance against Fusarium wilt (Mirrahimi et al., 2023), and the deployment of genetically resistant cultivars remains the most effective, economical, and environmentally sustainable strategy for long-term disease management (Velpula & Gaibriyal, 2022; Michielse & Rep, 2009). The identification and utilization of resistant germplasm constitute a critical step in breeding programs aimed at improving crop resilience against Fusarium pathogens.

Genetic diversity within cultivated and wild germplasm collections serves as a valuable reservoir of resistance genes against major plant pathogens. Indigenous landraces, in particular, often harbor adaptive traits shaped by long-term exposure to local biotic stresses and may represent underexplored sources of disease resistance (McCouch et al., 2013; Zhang et al., 2020). However, systematic evaluations of resistance to Fusarium wilt in luffa genotypes remain limited.

Therefore, the present study aimed to assess the resistance responses of indigenous and non-indigenous luffa genotypes to *Fusarium* infection, with the objective of identifying resistant genetic resources that can be exploited in future breeding programs for sustainable luffa production.

Materials and Methods

Pathogen source and inoculum preparation

A *Fusarium* sp. isolate was obtained from the Iranian Research Institute of Plant Protection (Tehran, Iran, Strain No. IRAN1042C), and used for pathogenicity and resistance screening assays. The *Fusarium* sp. had been previously collected from *Cucumis melo* L. fields (Iran, Razavi Khorasan Province, Torbat-e Jam) which is closely related to Luffa in terms of its cropping environment and shares several common pathogens. The fungal isolate was cultured in Czapek–Dox liquid medium to produce conidia. After incubation, the spore suspension was filtered and adjusted to a final concentration of 1×10^6 conidia mL^{-1} using a hemocytometer.

DNA extraction and PCR amplification

The *Fusarium* isolate used in the present study had been not molecularly identified. To detect the pathogen, genomic DNA was extracted from from the fungal isolate following the protocol of Dellaporta et al. (1983) with minor modifications. DNA quality and integrity were assessed using 1% agarose gel electrophoresis, and concentration was measured using a UV–Vis spectrophotometer (Unico UV-2100, USA).

PCR amplification of the internal transcribed spacer (ITS) region of ribosomal DNA was carried out using primers ITS1 (5′-TCCGTAGGTGAACCTGCGG-3′) and ITS4 (5′-TCCTCCGCTTATTGATATGC-3′) (White et al., 1990). The PCR program consisted of an initial denaturation at 95 °C for 2 min, followed by 30 cycles of denaturation at 94 °C for 1 min, annealing at 54 °C for 30 s, extension at 72 °C for 1 min, and a final extension at 72 °C for 10 min using a thermal cycler (Eppendorf, Hamburg, Germany).

DNA sequencing

PCR products were purified and sequenced commercially by Takapouzist Company (Iran). The

resulting sequences were analyzed using MEGA version 7 software, and sequence identity was confirmed through BLAST analysis against the NCBI GenBank database.

Pathogenicity assay

Plant Material and Genotypes: Eight genotypes of luffa (*L. aegyptiaca*) were evaluated in this study, comprising five indigenous and three non-indigenous accessions. The indigenous genotypes included Northern black seed (NB), Angled (An), Northern white seed (NW), Touri (Tou), and Northern large (NL), collected from different agroecological regions of Iran, including Mazandaran, Kerman, and Sistan and Baluchistan provinces. The non-indigenous genotypes—Afghani (Af), Long luffa (LL), and Brazilian (Br)—originated from Afghanistan, China, and Brazil, respectively, and were obtained from Nasima Seed Company (Qom, Iran).

Inoculation Procedure: Seeds of each luffa genotype were germinated in disposable containers lined with moist sterile paper towels and maintained under controlled conditions. After approximately two weeks, seedlings at the 2–4 true leaf stage were selected for inoculation. Inoculation was performed using the root-dip method. Seedling roots were gently washed to remove adhering substrate and immersed in the conidial suspension (1×10^6 spores mL^{-1}) for 5 min. Control plants were treated similarly using sterile distilled water. Following inoculation, seedlings were transplanted into plastic pots (containing a sterile soil–peat moss mixture), in a completely randomized block design with three replicates of ten plants per treatment, and incubated in a growth chamber at 25 ± 1 °C with a 12 h light/12 h dark photoperiod. A susceptible cucumber (*Cucumis sativus* L.) cultivar was included as a positive control to validate pathogen virulence.

Disease assessment and resistance classification

Disease development was monitored for up to two months post-inoculation (mpi). Disease severity was assessed using a three-point scale: 0 = healthy plant with no visible symptoms, 1 = wilted plant, 2 = complete seedling death. The disease index (DI) was calculated as a percentage using the following formula (Cachinero et al., 2002):

$$DI = \frac{\sum(n_i \times s_i)}{N \times S} \times 100$$

where n_i is the number of plants in each severity category, s_i is the corresponding severity score, N is the total number of evaluated plants, and S is the maximum severity score. Based on DI values, genotypes were classified as: Resistant (DI = 0–20), Sensitive (DI = 21–70%), Highly sensitive (DI = 71–100%). In addition, the area under the disease progress curve (AUDPC) was calculated to quantify disease progression over time following the method described by Campbell and Madden (1990).

$$\text{AUDPC} = \sum_{i=1}^{n-1} \left(\frac{y_i + y_{i+1}}{2}\right)(t_{i+1} - t_i)$$

AUDPC values were calculated separately for each replicate (n = 3). Data were analyzed using a two-way analysis of variance (ANOVA) with genotype as a fixed factor and replicate as a blocking factor. Normality of residuals and homogeneity of variances were verified using the Shapiro–Wilk and Levene's tests, respectively. Mean comparisons among genotypes were performed using Tukey's honestly significant difference (HSD) test at $P \leq 0.05$. All statistical analyses were conducted using standard parametric procedures. All analyses were performed in IBM SPSS Statistics 27.

Pathogen re-isolation

To confirm the pathogenicity of the isolate, symptomatic plants were collected and thoroughly washed under running tap water. Stem and leaf segments (approximately 0.5 cm) were excised under aseptic conditions, surface-sterilized in 1% sodium hypochlorite for 1 min, rinsed with sterile distilled water, and air-dried on sterile filter paper. The tissue segments were placed onto potato dextrose agar (PDA) plates and incubated at 25 °C. Emerging fungal colonies were examined and identified based on colony morphology and microscopic characteristics.

Pathogen identity

To molecularly confirm the identity of the fungus re-isolated from symptomatic seedlings, the internal transcribed spacer (ITS) region was amplified using the universal fungal primers ITS1 and ITS4. The resulting amplicons were purified and sequenced. Sequencing chromatograms were assessed for quality using MEGA7 software; high-quality reads with clear base calls were obtained for subsequent analysis. The resulting sequence was edited and assembled using BioEdit software. For identification, the consensus sequence was compared to the NCBI nucleotide database using the BLASTn algorithm (http://blast.ncbi.nlm.nih.gov).

Results

Pathogenicity test

Disease index (DI) values revealed clear differences in the response of *Luffa* genotypes to *Fusarium* infection. Based on the predefined classification criteria (DI 0–20% = resistant; DI 21–70% = sensitive; DI 71–100% = super sensitive), six genotypes—Br, NB, NW, An, LL, and Af—were categorized as resistant, exhibiting low DI values ranging from 0 to 15%. Among these, Br, NB, and NW showed complete resistance, with no visible disease symptoms throughout the evaluation period (DI = 0%). In contrast, two genotypes, NL and Tou, displayed significantly higher DI values of 64% and 44%, respectively, and were therefore classified as sensitive. These genotypes showed pronounced wilt symptoms and progressive disease development following inoculation (Fig 1A). No genotype exhibited DI values exceeding 70%, indicating the absence of super sensitive responses under the experimental conditions (Cachinero et al., 2002).

Significant differences in disease development were observed among genotypes based on AUDPC values. Two-way ANOVA revealed a highly significant effect of genotype on AUDPC ($F_{7,14} = 15.33$, $P < 0.001$), while the replicate (block) effect was not significant ($P = 0.47$), indicating consistent disease assessments across experimental replicates. Mean AUDPC values ranged from 0 to 22.0. The genotype NL exhibited the highest AUDPC, indicating pronounced susceptibility, followed by Tou, which showed moderate disease development. LL, Af, and An displayed intermediate responses, suggesting partial resistance. In contrast, NB, Br, and NW showed complete resistance, with no detectable disease development across all replicates. Tukey's HSD test confirmed clear separation of genotypes into statistically distinct susceptibility groups (Fig 1B).

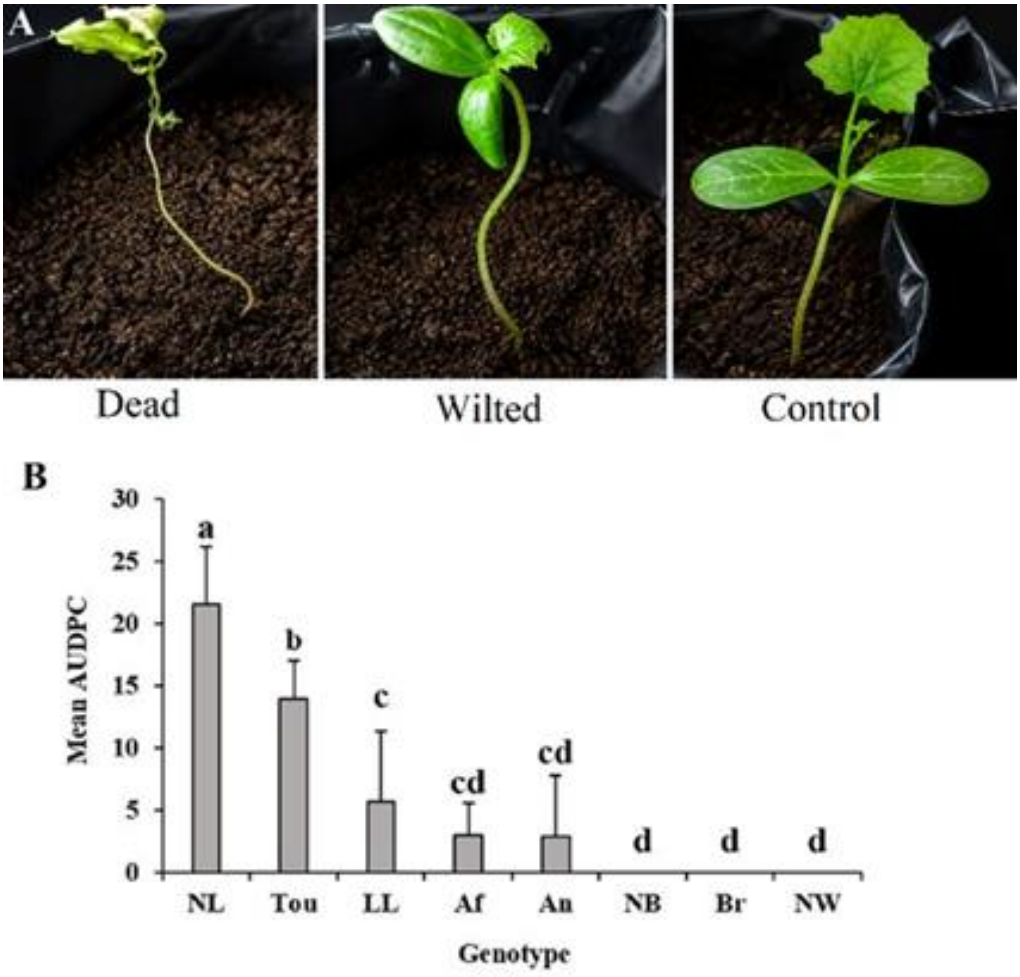

Fig. 1. The progression of disease severity in the inoculated plants. (A) The reaction of *Luffa* seedlings to fungus at 2 mpi; (B) Genotypic differences in disease development quantified by the area under the disease progress curve (AUDPC); Bars represent mean AUDPC values (n=3). Different letters above bars indicate significant differences among genotypes according to Tukey's honestly significant difference (HSD) test at $P \leq 0.05$.

Pathogen re-isolation

Fungal colonies that emerged on PDA from symptomatic plant specimens were examined morphologically to confirm pathogen infection. The fungal isolate exhibited rapid radial growth on potato dextrose agar, producing dense, cottony to floccose aerial mycelium within 5–7 days of incubation. Colonies were initially white and gradually developed pale pink to salmon pigmentation in the central region, with light brown coloration toward the margins. Microscopic examination revealed hyaline, septate, and branched hyphae. Collectively, these morphological features were similar to those described for *Fusarium* cf. *proliferatum* by Leslie and Summerell (2006).

Molecular identification

Molecular analysis of the ITS region confirmed the identity of the fungal isolate recovered from symptomatic seedlings. PCR amplification using primers ITS1 and ITS4 yielded a single amplicon of approximately 500–600 bp, which was successfully sequenced. Following quality trimming and assembly, a consensus sequence of 400 bp was obtained. BLASTn analysis against the NCBI nucleotide database revealed that the isolate shared 99–100% sequence identity with reference sequences of Fusarium species. The sequence generated in this study was deposited in GenBank under accession number PP916731.1. The molecular analysis supported the morphological identification of the re-isolated fungus as *Fusarium* cf. *proliferatum*.

Discussion

Luffa is a plant species that grows in most regions of Iran and exhibits a high level of diversity between indigenous and non-indigenous genotypes (Arbabi *et al*. 2023, 2022, 2020). The identification of resistant germplasm represents a fundamental step toward the development of sustainable strategies for managing Fusarium diseases in cucurbit crops. In the present study, significant variation in disease severity was observed among indigenous and non-indigenous *Luffa aegyptiaca* genotypes following inoculation with a *F. proliferatum*. The resistant responses exhibited by genotypes such as NB, Br, and NW demonstrate that valuable genetic variation for resistance exists within the evaluated germplasm and may serve as an important resource for future breeding programs. Similar studies have shown that the exploitation of naturally occurring resistance remains one of the most effective approaches for reducing crop losses caused by soil-borne fungal pathogens (Zhang et al., 2017; McCouch et al., 2013).

The broad range of disease reactions observed among the evaluated genotypes indicates that host genetic background plays a major role in determining susceptibility to *Fusarium* infection. Disease development results from the complex interaction between host defense mechanisms and pathogen virulence, leading to continuous variation in symptom expression rather than distinct resistant and susceptible classes. Similar patterns have been reported in several host–*Fusarium* pathosystems and reflect the quantitative nature of resistance to vascular wilt pathogens (Gordon, 2017; Michielse & Rep, 2009). The identification of moderately and highly resistant genotypes suggests that resistance in *L. aegyptiaca* is likely controlled by multiple genes acting together to restrict pathogen colonization and disease progression.

The differences in disease severity observed among the luffa genotypes are consistent with current understanding of plant defense against soil-borne fungal pathogens. Resistant plants are generally able to suppress fungal invasion through rapid activation of structural and biochemical defense responses, including reinforcement of cell walls, accumulation of antimicrobial compounds, and induction of defense-related pathways (Cachinero et al., 2002). Although the present study did not investigate the molecular mechanisms underlying resistance, the phenotypic variation observed among the evaluated genotypes strongly suggests differences in the efficiency of these defense responses.

The identification of resistant luffa genotypes has important implications for disease management. Soil-borne *Fusarium* pathogens can survive for prolonged periods in plant debris and soil, making their control by chemical means both difficult and economically unsustainable (Fravel et al., 2003; Gordon, 2017). Consequently, the deployment of resistant cultivars remains one of the most practical and environmentally sound strategies for reducing disease incidence and minimizing yield losses. The resistant genotypes identified in this study therefore represent valuable genetic resources for breeding programs aimed at improving disease resistance in *L. aegyptiaca*.

In addition to their value for cultivar development, these resistant genotypes provide useful parental material for

broadening the genetic base of luffa breeding programs. The incorporation of resistant germplasm into breeding populations can facilitate the accumulation of favorable alleles while maintaining desirable agronomic characteristics. Modern breeding approaches, including marker-assisted selection, further increase the efficiency of transferring quantitative resistance into elite cultivars and contribute to the development of durable resistance (Pilet-Nayel et al., 2017).

Although this study successfully identified resistant germplasm, additional research is needed to elucidate the genetic basis of resistance and the mechanisms governing the interaction between *L. aegyptiaca* and *Fusarium*. Future investigations integrating genetic mapping, molecular characterization of resistance-associated loci, and functional analyses of defense-related genes will improve our understanding of host resistance and accelerate the development of resistant cultivars.

Overall, the present study demonstrates substantial genetic variation in the response of *L. aegyptiaca* genotypes to *Fusarium* infection and identifies promising sources of resistance for future breeding programs. These findings provide valuable baseline information for integrated disease management and support the use of host resistance as a sustainable strategy for reducing the impact of *Fusarium* diseases in luffa cultivation.

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Conflict of interest

The authors have no competing interests to declare that are relevant to the content of this article.

CRediT author statement

N. Mollashahi: Material preparation, data collection. **M. Keykhasaber, M. Pirnia, and Sh. Sarani:** Material preparation, data collection and analysis. All authors contributed to the study conception and design, read and approved the final manuscript.

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Data Availability

All data generated or analysed during this study are included in this published article.

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