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Fusarium wilt of brinjal: impact, pathogenicity, and sustainable management strategies

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Abstract
Brinjal (*Solanum melongena* L.) is an economically significant vegetable crop cultivated worldwide. However, its productivity is severely affected by various soil-borne pathogens, among which *Fusarium oxysporum* and *Fusarium solani* is a major causal agent of wilt disease. This review comprehensively discusses the etiology, epidemiology, symptomatology, and pathogenicity of *F. oxysporum* and *F. solani*, along with its impact on brinjal production. The pathogen invades the vascular system, leading to wilting, yellowing, and eventual plant death, resulting in significant yield losses. Various environmental factors, such as soil moisture, temperature, and host susceptibility, influence disease progression. Additionally, this paper explores current management strategies, including cultural practices, biological control, resistant cultivars, and chemical treatments, to mitigate the disease. Advances in molecular techniques for pathogen identification and resistance breeding are also highlighted. A better understanding of the host–pathogen interactions and integrated disease management approaches can aid in developing sustainable solutions to combat *F. solani* induced wilt in brinjal while also promoting environmentally friendly management practices to support global food security.

Keywords Brinjal wilt, Disease management, *F. solani*, Host–pathogen interaction, Resistance breeding

1 Introduction

Brinjal (*Solanum melongena* L.), also known as eggplant or aubergine, is one of the most important vegetable crops cultivated worldwide, particularly in Asia, Africa, and the Mediterranean regions. It is a rich source of fiber, vitamins, minerals, and bioactive compounds, contributing significantly to human health and nutrition [1]. Despite its importance, brinjal cultivation faces challenges from various biotic and abiotic stresses, with soil-borne diseases being particularly damaging. Among these, Fusarium wilt caused primarily by *F. oxysporum* f. sp. *melongenae* and *F. solani* is one of the most severe, leading to significant yield losses worldwide [2, 3]. *Fusarium* spp., survive in soil for extended periods as chlamydospores and initiate infection by entering plant roots, ultimately disrupting the vascular system and leading to wilt symptoms. Infected plants

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exhibit yellowing, wilting, and stunted growth due to vascular disruption caused by the pathogen [4, 5].

The disease is favoured by warm, humid conditions and spreads through infected soil, seeds, and plant debris, making management particularly difficult [6]. Yield losses in susceptible cultivars can reach up to 80%, posing a serious economic threat especially to small-scale farmers with limited access to advanced control measures [7]. Reliance on chemical fungicides for disease control has led to issues such as fungicide resistance, environmental contamination, and disruption of beneficial soil microbes [3]. Therefore, sustainable and integrated disease management strategies are essential. Cultural practices like crop rotation and organic amendments, use of biological control agents (BCAs) such as *Trichoderma harzianum*, *Pseudomonas fluorescens*, and *Bacillus subtilis* suppress *Fusarium* and promote plant growth [7, 8]. As a result, there is a pressing need for sustainable and integrated disease management strategies to mitigate the impact of Fusarium wilt and ensure long-term agricultural sustainability.

The pathogenicity of *Fusarium* spp., is facilitated by various virulence factors, including toxin production, enzyme secretion, and host-specific genetic determinants [9]. Several studies have identified resistance-related genes (R-genes) and defence signalling pathways in brinjal plants that contribute to plant immunity against *Fusarium* spp., However, due to the high genetic variability of the pathogen, breeding for resistance remains challenging [6]. Understanding the molecular mechanisms of host–pathogen interactions is essential for the development of effective disease-resistant brinjal cultivars. Organic soil amendments like compost and neem cake enhance soil health and plant immunity [8]. Molecular tools such as PCR and CRISPR aid in early pathogen detection and genetic resistance development [5].

The objective of this review is to provide a comprehensive understanding of Fusarium wilt in brinjal, covering its pathogenicity, economic impact, host–pathogen interactions, and sustainable management strategies. By analysing recent advancements in plant pathology, disease diagnostics, and integrated disease management, this study aims to highlight potential solutions for mitigating Fusarium wilt and ensuring sustainable brinjal production.

2 Diseases reported on brinjal

Diseases are a common occurrence in brinjal cultivation, and certain significant diseases are attributed to fungi. Soil-borne pathogens commonly lead to diseases that mainly affect the underground parts of plants, causing problems like seed decay, seedling damping off, root and crown rots, and wilting through root infections [9]. Plant pathogens include Phomopsis blight caused by (*Phomopsis vexons*) Sacc and Syd (1914), Anthracnose (*Colletotrichum coccodes*) Hughes (1958), Cercospora leaf spot (*Cercospera melongenae*) Welles (1922), Phytophthora root rot (*Phytophthora melongenae*) Formosa (1914), Fusarium wilt (*Fusarium oxysporum*) schitdl *et al*. (1824), Powdery Mildew (*Leveillula taurica*) Arnaud (1921), Damping-off (*Pythium aphanidermatum*) Fitzn (1923). These diseases are a major obstacle to crop growth, causing around 68% of yield losses in vegetables when continuous cropping is practiced [10]. More than 100 types of *Fusarium* vascular wilts exist globally [11]. These pathogens are a significant challenge because they typically endure in the soil for extended periods, and various species of

these pathogens can affect multiple types of vegetable crops [12]. Several major fungal diseases affecting brinjal plants are listed in Table 1.

3 Causal pathogen—*Fusarium* Spp.

The genus *Fusarium* is a widely distributed group of filamentous fungi that includes numerous agriculturally important plant pathogens. Historically, *Fusarium* was first described as Fusisporium by the German mycologist Link in 1809. The name was later changed to *Fusarium*, derived from the Latin word fusus, meaning "spindle-shaped," referencing the typical morphology of its macroconidia. This genus encompasses a wide array of fungal species found globally in both plants and soil. Members of *Fusarium* spp., can act as pathogens, endophytes, or saprophytes [13]. They are commonly considered soil-dwelling fungi due to their prevalence in the rhizosphere and their frequent interaction with plant roots, either as parasites or decomposers [14].

One of the key features of *Fusarium* is its complex taxonomy. The genus comprises more than 300 phylogenetically distinct species grouped into multiple species complexes, with over 20 commonly reported as plant pathogens. The *Fusarium* genus comprises more than 20 morphologically distinct and agriculturally important species [15]. Some *Fusarium* spp., are also known to exhibit a teleomorphic (sexual) state, typically classified under the genus Gibberella [16]. The most common pathogenic species associated with vascular wilt and root rot in solanaceous crops include *F. solani, F. oxysporum, F. equiseti, and F. chlamydosporum* [17]. Identification and differentiation of *Fusarium* spp., rely on a combination of macroscopic and microscopic characteristics. Macroscopic features include colony morphology (colour, texture, and growth rate), as well as microscopic features such as the length and shape of macroconidia, the number, shape, and arrangement of microconidia, and the presence or absence of chlamydospores [18].

3.1 Taxonomic position and nomenclature

Recent advances in molecular phylogeny have significantly refined the taxonomy of the genus *Fusarium*, which was historically based on morphological traits. *F. oxysporum*, a member of the phylum Ascomycota, class Sordariomycetes, order Hypocreales, and family Nectriaceae, is now placed within the *F. oxysporum* species complex (FOSC), as supported by multilocus sequence analyses [19]. Molecular markers such as TEF1-α (translation elongation factor 1-alpha), RPB1, and RPB2 have been widely used for precise delimitation within *Fusarium* lineages, helping to distinguish pathogenic and non-pathogenic forms at the forma specialis level. Current consensus classifies *F. oxysporum* as a species complex encompassing both saprophytic and pathogenic strains, with *formae speciales* used to denote host specificity rather than strict taxonomic separation. These updates reflect a shift from traditional morphology-based taxonomy to a more

Table 1 Diseases reported on brinjal

Sr. No.	Disease name	Causal organism	Reference
1	Phomopsis blight	*Phomopsis vexans*	Sacc and Syd (1914)
2	Anthracnose	*Colletotrichum coccodes*	Hughes (1958)
3	Cercospora leaf spot	*Cercospera melongenae*	Welles (1922)
4	Phytophthora root rot	*Phytophthora melongenae*	Formosa (1914)
5	*Fusarium* wilt	*Fusarium oxysporum*	Schitdl et al. (1824)
6	Powdery Mildew	*Leveillula taurica*	Arnaud (1921)
7	Damping-off	*Pythium aphanidermatum*	Fitzn (1923)

robust, DNA-based classification, which enhances the accuracy of identification, diagnosis, and disease management strategies [20].

The *Fusarium* genus is known for its diversity and encompasses a multitude of species, some of which hold significant agricultural and ecological significance [21]. *Fusarium* genus includes both pathogenic and non-pathogenic species. Some *Fusarium* spp., are beneficial and can act as antagonists to plant pathogens, while others are destructive plant pathogens responsible for diseases such as Fusarium wilt [22].

3.2 Fusarium wilt: *F. oxysporum* and *F. solani*

Fusarium wilts are primarily caused by *F. oxysporum* and *F. solani*, two of the most destructive fungi affecting brinjal plants and many other economically important crops such as tomato (*Solanum lycopersicum*), chili (*Capsicum annuum*), potato (*Solanum tuberosum*), cotton (*Gossypium spp.*), banana (*Musa spp.*), okra (*Abelmoschus esculentus*), and legumes like chickpea (*Cicer arietinum*) and pigeon pea (*Cajanus cajan*) [5, 21]. These pathogens invade the vascular tissues, obstructing water and nutrient transport, which leads to wilting, yellowing, and eventual plant death. *F. solani*, in particular, has been noted for its host specificity and pathogenicity in both greenhouse and field-grown brinjal. It produces mycotoxins that pose serious threats to both plants and animals [23, 24]. In Iraq, *F. oxysporum* is recognized as a prominent wilt pathogen of brinjal wilt and other Solanaceae crops under both open and protected cultivation systems [25]. It persists in soil via mycelia and chlamydospores and is capable of causing significant losses [26].

Not all *F. oxysporum* strains are pathogenic. Non-pathogenic isolates, frequently recovered from healthy roots and rhizosphere soils, have shown potential for biological control. These strains can outcompete pathogenic forms through mechanisms like nutrient competition, rhizosphere colonization, and induction of systemic resistance in host plants [27]. Moreover, some non-pathogenic strains are isolated from soils naturally suppressive to Fusarium wilt and are under investigation as sustainable biocontrol agents [28]. Although field performance is often reduced due to microbial competition, early application of adequate inoculum densities has demonstrated promising disease suppression.

Fusarium spp., exhibit considerable morphological variability [29], particularly in *F. oxysporum*, which typically produces white, cottony colonies with diverse structures when grown on PDA [30, 31]. It reproduces asexually through three main spore types. Macroconidia are slightly curved or sickle-shaped, 3–5 septate, multinucleate, and exhibit rapid germination [32]. Microconidia are oval to round, single-celled, typically uninucleate, and show poor germination efficiency (1–20%) under standard conditions, although their germination is significantly enhanced in the presence of root exudates [33]. Chlamydospores, which form through the thickening of hyphal or conidial cells, serve as long-term survival structures and can germinate up to 98% within 10 hours in root-exudate assays [34]. In contrast, *F. solani* macroconidia germinate poorly (~ 10%) in minimal media [35]. These morphological traits play a critical role in fungal reproduction, survival, and soil persistence.

The genus *Fusarium* was systematically classified by Wollenweber in his monographic work. [36]. Among its diverse species, *F. oxysporum* is of particular interest due to its complex intraspecific diversity, including both pathogenic and non-pathogenic forms.

Host specificity in *F. oxysporum* is determined by *formae speciales*, each adapted to a specific plant host [37]. Conidia play a central role in initiating infection and disseminating the disease [38]. Microconidia germinate variably, from 14 to 63% in root exudates and ~16% in buffer solutions [33]. Chlamydospore and macroconidial germination patterns, along with micro-conidiophore structures, further aid in species-level identification [39].

3.3 Climatic conditions required for growth

Environmental and soil factors, such as humidity, rainfall, temperature, evaporation rate, soil moisture, and sunshine hours, influence Fusarium wilt disease development [40]. *Fusarium* spp., thrives in warm and humid conditions, although specific environmental preferences vary. High humidity is essential for spore germination and infection, with disease development being more likely when relative humidity exceeds 70% [41]. Fusarium wilt is more severe in soils with a pH below 6.4 and above 7. Seven factors showed a significant positive correlation across all plant varieties, including KHBR-205-E, KHBR-202-B, EP-900, WER2, WER-3, and DESI/Local. Prolonged rainfall and excessive moisture in poorly drained soils facilitate spore spread and infection, while poor air circulation raises humidity, further promoting disease. Effective soil drainage, irrigation control, and improved airflow can help reduce its impact. [42]. Fusarium wilt development in brinjal plant is influenced by temperature, with optimal growth occurring between 25 and 30 °C (77°F–86°F), creating favourable conditions for disease progression [43].

3.4 Symptoms of Fusarium wilt

Fusarium wilt disease in brinjal begins with veinlet clearing, leaving the primary vein green, followed by one-sided yellowing of young leaflets and eventual wilting and death of mature leaves [44]. Hyphae were often aligned parallel to the long axis of xylem vessels and frequently coated with an amorphous electron-opaque material deposited on vessel walls and pit membranes, leading to occlusion of pit cavities [45]. Diseased brinjal plants often display chlorotic foliage, lamina sagging, sequential abscission of leaves, and noticeable suppression of canopy development [46], along with leaf epinasty [47], and vascular tissue discoloration, chlorosis, and stem bending, which may result in plantlet death [48]. The pathogen gains entry via the root system and progressively infiltrates the vascular tissues, impairing xylem functionality and resulting in systemic wilting and necrosis of aerial plant structures [49]. Under conducive environmental conditions, particularly in the winter season, the disease may reach epidemic proportions, potentially leading to complete crop loss [50].

Fusarium oxysporum f. sp. *melongenae*, the etiological agent of wilt in brinjal, exhibits high virulence and possesses the ability to persist in soil for extended durations in the absence of a host [51]. This persistence makes control difficult, and the frequent use of chemical fungicides has raised concerns due to potential risks to human health, the environment, and non-target organisms [52]. The fungus causes severe damage to brinjal by blocking water flow in the xylem, resulting in wilting and plant death [53]. Characteristic symptoms include reddish-brown vascular streaks and the collapse of aboveground parts of the plant [54]. Its spores remain viable in the soil for extended durations [5], and xylem blockage often leads to sudden plant mortality [55].

Pathogenicity involves the production of conidia that infect roots and colonize the vascular system [56]. The infection process includes hyphal attachment to the root surface and secretion of cell wall-degrading enzymes that allow entry into vascular tissues like the xylem and phloem [57]. The plant's defense mechanisms such as tyloses, gels, and proliferation of parenchyma cells further contribute to vessel blockage and disease severity [58].

3.5 Disease cycle of Fusarium wilt

Traditionally, the colonization of plant roots by fungi in soil has been investigated using indirect methods like microbial isolation. These methods do not provide the means for directly observing infection sites or the interactions between fungal pathogens and the organisms that counteract them [59]. The disease cycles of most Fusarium wilts exhibit similarities [60]. The life cycle of *Fusarium* spp., can be categorized into dormant, parasitic, and saprophytic stages [61]. The life cycle can be divided into three stages. First, the dormant stage involves inhibiting and germinating resting structures in the soil. It then breaches the endodermis and gains access to the xylem vessels through the pit membranes. Once inside the vascular system, the pathogen enters its parasitic phase, leading to systemic colonization and ultimately resulting in wilting and host death. Lastly, the saprophytic stage includes the formation of resting structures in the deceased host [62]. In the dormant phase, mycelia, chlamydospores, macroconidia and microconidia (propagules) present in infested soil are inhibited from germinating because of mycostasis or microbiostasis [63]. *Fusarium* spp., transition to the parasitic phase when their propagules or spore germ tubes penetrate the host through openings created by emerging lateral roots, wounds, or at various points on the root structure such as the root cap, root hairs, or branch roots [64–68]. *Fusarium* spp. initiates infection by intercellularly penetrating the root surface, first colonizing the cortical tissues. Inside the vessels, the mycelia move upward, and effective colonization is genetically regulated [69]. They produce microconidia, which travel upward in the plant's transpiration stream, germinating into new hyphae to infect neighbouring vessel elements and further colonize [70]. As the *Fusarium* hyphae spread within the plant's cell apoplast, it causes significant cytological changes leading to symptom expression. Vessel clogging by mycelia, fungal spores, and plant defences like gels, gums, tyloses, and proliferation of adjacent parenchyma cells contribute to water transport disruption in the plant, resulting in wilting and eventual plant death [37, 71, 72]. New spores can return to the soil as infected plants decompose or be carried to new areas by wind or water. Conidia are formed on dead leaves, and various propagules, including chlamydospores, are produced [73]. Chlamydospores can lie dormant in the soil for years, germinating when they encounter a new host. Some weeds can harbour *Fusarium* without showing symptoms [74]. A detailed representation of the life cycle of the plant have been provided in (Fig. 1).

3.6 Pathogenicity and associated genes of fusarium wilt

The pathogen exhibits high genetic variability, enabling it to evolve into different physiological races that infect various plant species across diverse geographical regions [75]. *Fusarium* colonies typically exhibit rapid growth, with pale or bright-coloured mycelium that is extensive and has a cottony appearance [76]. Conidiophores can be varied, ranging from slender and simple to short and irregularly branched, sometimes forming

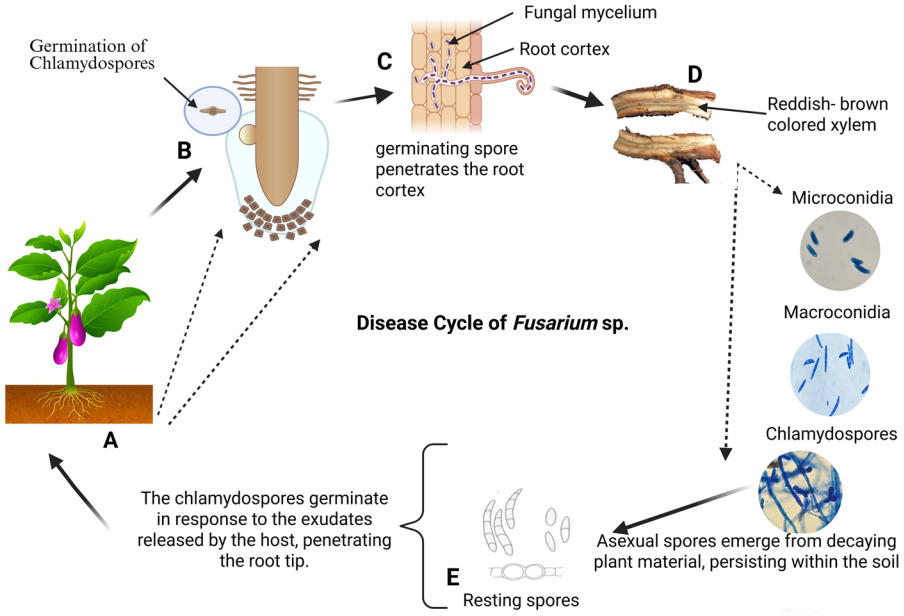

Fig. 1 Life cycle and infection mechanism of *Fusarium* spp., associated with wilt disease in brinjal (*Solanum melongena* L.). Schematic representation of the infection process and disease cycle of *Fusarium* spp., in brinjal. **A** Brinjal plant showing wilt symptoms. **B** Germination of chlamydospores in response to root exudates. **C** Penetration of the root cortex by germinating spores and development of fungal mycelium. **D** Vascular discoloration due to colonization of the xylem tissue. **E** Formation of resting structures including microconidia, macroconidia, and chlamydospores which persist in the soil and contribute to disease recurrence

clusters of phialides. Conidia, which are hyaline macroconidia, which are slightly curved and have multiple cells, and microconidia, which are single-celled, oval to oblong, and may be solitary or in chains [77]. *Fusarium solani* is a soil-dwelling fungus responsible for causing Fusarium wilt in brinjal. This disease is highly prevalent in regions where plants are grown and can lead to substantial yield losses. This pathogen was first documented in Japan and subsequently in China [78]. Pathogenesis is the process of how a disease develops in a host, from infection to symptom production [79].

Fusarium solani was identified by examining its colony appearance, as well as the shapes of its conidiophores and spores, which are all visual characteristics [80]. The microorganism can persist for extended periods as chlamydospores within plant and soil remnants, even following the cultivation of crops that are not susceptible to it [81]. *Fusarium solani* is a globally distributed pathogen that causes Fusarium wilt, a lethal vascular syndrome affecting a wide range of plant species. [82]. Fusarium wilt is a fungal disease that thrives in warm conditions, particularly around 28 °C. Disease progression is hindered at temperatures exceeding 34 °C or falling below 17 °C [83]. The pathogen can disseminate between different regions through various means, such as the transportation of infected planting materials and seeds, as well as the use of farm tools and machinery, especially when they have been in contact with contaminated soils and are subsequently used in uninfected fields. Additionally, both air and water contribute to the dispersal of this fungus [84]. *Fusarium* spp., is the pathogenic microorganism responsible for Fusarium wilt, a highly destructive disease that impacts a broad spectrum of plants, including both wild weeds and commercially cultivated crops [85]. *Fusarium oxysporum* develops hyphae for the attachment and penetration of both wounded and unwounded roots epidermal cells [57]. Certain species infect floral tissue with fusaric

acid during flowering, then spread to the flower's central axis, ultimately contaminating grains with toxins [86]. *Fusarium* spp., produce a cancer-causing mycotoxin, posing a direct risk to both food safety and human well-being [87]. The chlamydospores were spherical to nearly spherical, measuring an average of 10 µm in diameter, and the hyphae exhibited branching at sharp angles. These tough chlamydospores, which are alive and contain lipid-like material, can remain in the soil for a long time [60].

In addition to its morphological and ecological adaptations, *Fusarium* pathogenesis is intricately regulated at the molecular level. One of the major virulence factors is the fusaric acid biosynthetic cluster (FUB1–FUB12), which includes genes like *FUB1*, encoding a polyketide synthase essential for the synthesis of fusaric acid a phytotoxin that disrupts water balance and cellular structure in host plants, thereby enhancing fungal virulence [88]. Another hallmark of virulence in *F. oxysporum* is the presence of Secreted in Xylem (SIX) effector proteins, small cysteine rich proteins encoded on mobile pathogenicity chromosomes that suppress plant immune responses and determine host specificity. Notably, horizontal transfer of these chromosomes can convert nonpathogenic strains into pathogenic ones [89]. Besides the SIX effectors, more than 26 other candidate effector genes have been found, some of which are similar to RNA binding and ToxA-like proteins, expanding the range of known effectors [90]. Comparative genomic studies across *Fusarium* spp. have revealed a wide diversity of toxin biosynthesis and secondary metabolite biosynthetic gene clusters, such as those responsible for trichothecene and fusarin production, which contribute significantly to pathogenicity and fungal fitness [91] These molecular components collectively reinforce the pathogen's ability to colonize host tissues, evade plant defenses, and cause severe vascular damage.

3.7 Pathogenicity assessment and advanced testing methods

Pathogenicity testing plays a crucial role in confirming the virulence of *Fusarium* spp., infecting brinjal. Traditionally, Fusarium wilt pathogens are identified using Koch's postulates, which involve isolation of the suspected pathogen, reinoculation onto a healthy host, and reproduction of disease symptoms. Differentiation is typically based on morphological features, genetic sequencing of the internal transcribed spacer (ITS) and elongation factor 1-alpha (TEF1-α) regions, as well as pathogenicity tests [92].

The most widely adopted technique is the root-dip inoculation method, wherein seedlings are immersed in a conidial suspension and then transplanted into sterilized soil or potting medium [93]. Disease symptoms are evaluated after 15–30 days post-inoculation, including leaf yellowing, wilting, vascular discoloration, and root necrosis. These symptoms are scored using a standardized disease rating scale, typically 0–5 or 0–9, to assess severity [94]. Quantitative metrics such as Disease Incidence (%) and Disease Severity Index (DSI) are commonly employed:

$$\text{Disease Incidence}\,(\%\,) = \frac{\text{Number of infected plant (s)}}{\text{Number of total plant (s)}} \times 100$$

$$\text{Disease Severity Index} = \frac{\sum(\text{Disease score} \times \text{Number of plants at that score})}{\text{Total number of plants} \times \text{Maximum score}} \times 100$$

[95]. These indices allow for reproducible comparison of pathogenicity across isolates, treatments, or genotypes.

Beyond phenotypic assays, molecular tools have significantly enhanced pathogen characterization. PCR-based marker identification using ITS, TEF1-α, and teleomorphic gene loci enable precise differentiation between pathogenic and non-pathogenic isolates and between different *formae speciales* [96]. These tools are critical for early detection and are especially useful in resistance breeding and quarantine regulation.

Recent advances in functional genomics have deepened our understanding of virulence mechanisms. For example, targeted deletion of *FUB1*, part of the fusaric acid biosynthetic gene cluster, reduces phytotoxin production and virulence, indicating the role of fusaric acid in host colonization and symptom expression [88]. Likewise, SIX (Secreted in Xylem) effector proteins encoded on mobile chromosomes modulate host immune responses, determining host specificity and aggressiveness in *F. oxysporum* [96].

Live-cell imaging using GFP-tagged *Fusarium* strains provides a powerful method for visualizing the infection process in planta. This technology enables real time tracking of fungal colonization, from root surface adhesion to xylem invasion, greatly advancing our understanding of host pathogen interactions at cellular and tissue levels [97]. In summary, integration of classical inoculation methods with molecular diagnostics, gene function studies, and in vivo imaging creates a comprehensive framework for pathogenicity assessment. These tools are essential for devising effective and sustainable management strategies for Fusarium wilt in brinjal.

3.8 Enzymatic mechanisms of pathogenesis

Fusarium spp., secretes a range of extracellular enzymes that contribute to pathogenicity and host tissue colonization. Among these, pectinases, cellulases, hemicellulases, and cutinases are particularly important in degrading plant cell walls, facilitating penetration and systemic infection. *F. solani* has been identified as a significant producer of pectinases, enzymes extensively utilized in the food and wine industries, but which in planta degrade pectic substances of the middle lamella, allowing hyphal invasion [98].

Phytopathogenic fungi typically employ various pathogenic mechanisms, including the formation of specialized structures such as appressoria, and the secretion of enzymes that degrade plant cell walls, along with toxins, growth regulators, and effector proteins to facilitate infection and colonization [99]. *F. oxysporum* f. sp. *melongenae* expresses a set of cell wall degrading enzymes including endopolygalacturonases (PG1), exopolygalacturonases (PG2 and PG3), an endoxylanase (XYL1), and an endo-pectatelyase (PL1) which are actively expressed during infection [100].

Cutinases are particularly critical during the early stages of infection, as they hydrolyze the cuticle a protective waxy layer thereby allowing fungal entry through the epidermis [99]. In *F. solani*, knockout mutants lacking the cutinase gene showed up to 90% reduction in cutinase activity and significantly diminished virulence, underscoring its essential role in host penetration [101].

Additionally, proteases such as aspartic and serine proteases degrade host defense-related proteins, further aiding in fungal colonization. An aspartic protease secreted by *F. oxysporum* was shown to suppress host immunity and contribute directly to virulence by interfering with host protease inhibitors [102]. Proteomic analyses of *F. oxysporum* f. sp. *melongenae* have identified hundreds of candidate secreted effector proteins (CSEPs), including enzymes with hydrolase, cutinase, and protease domains, supporting their extensive involvement in pathogenicity [98]. These molecular components work

synergistically to degrade host barriers, suppress immune responses, and facilitate vascular colonization, leading to wilt symptoms and ultimately plant death.

3.9 Isolation techniques

Isolation of *Fusarium* spp. from infected brinjal tissues is a crucial step in understanding disease etiology and developing effective management strategies. A common approach involves culturing surface-sterilized segments of diseased roots, stems, or vascular tissues on Potato Dextrose Agar (PDA), a nutrient-rich medium that promotes rapid fungal growth and sporulation [103, 104]. However, because PDA is non-selective, it often permits overgrowth by saprophytic fungi and bacteria. To increase selectivity, antibiotics such as streptomycin and penicillin are often incorporated into PDA, enhancing recovery of *Fusarium* by suppressing bacterial contaminants [103]. For purification and pathogenicity assays, single-spore isolation is widely employed to obtain genetically uniform cultures [105]. Additionally, tissue segment culturing, combined with microscopic morphological assessment, is effective for isolating seed and *Fusarium* spp., from various solanaceous hosts, including brinjal [106].

To enhance both the specificity and sensitivity of *Fusarium* isolation, several selective and semi-selective media have been developed. Komada medium, originally formulated for *F. oxysporum*, contains pentachloronitrobenzene (PCNB) and other antimicrobial agents such as streptomycin and oxgall. It exhibits moderate to high selectivity and, with adjusted PCNB concentrations, can also support the growth of *F. solani* [107]. Nash and Snyder medium, a more widely adopted semi-selective medium, incorporates PCNB, chloramphenicol, streptomycin, and neomycin, and is known for its high sensitivity and specificity in isolating *Fusarium* spp., from both soil and plant materials [108]. Snyder medium, a simplified version that omits neomycin, offers moderate selectivity at lower cost and is suitable for samples with lower microbial competition [109].

Several studies have successfully isolated *Fusarium* spp., from solanaceous hosts. *F. oxysporum* has been isolated from diseased tissues using selective media such as Komada or Nash and Snyder medium, with isolates confirmed through subculturing and morphological or molecular identification [110]. Similarly, *F. oxysporum* was re-isolated from infected brinjal tissues, and Koch's postulates were confirmed using morphological and molecular methods. [111]. The pathogenicity of *Fusarium* spp., on brinjal was established through root-dip inoculation of healthy seedlings followed by re-isolation of the pathogen from infected vascular tissues [112].

Multiple isolation methods including PDA with antibiotics, Komada medium, and Nash and Snyder medium enhances the accuracy and efficiency of *Fusarium* spp., recovery. Each medium offers varying degrees of specificity and sensitivity, and their use should be tailored based on sample type, pathogen load, and downstream applications [13, 80].

3.10 Yield loss assessment

Fusarium wilt, caused by *F. solani* f. sp. *melongenae*, is a devastating vascular disease of brinjal, posing a significant threat to sustainable vegetable production. Globally, brinjal plants is cultivated across more than 1.8 million hectares, yielding around 56 million metric tonnes annually [113]. Yield losses due to wilt diseases especially *Fusarium* are estimated at 30–50%, depending on environmental conditions and varietal susceptibility

[114]. Globally, these reductions result in an economic loss of approximately USD 2 to 4 billion per year, accounting for both direct yield losses and indirect costs associated with disease management, replanting, and reduced market quality [113].

In India, the second-largest producer of brinjal after China, cultivation spans approximately 0.72 million hectares, with total production reaching 13.4 million tonnes annually [115]. Fusarium wilt and related vascular wilts are responsible for 20–50% yield losses, resulting in an estimated economic loss of INR 1500–3500 crore per year, disproportionately affecting small and marginal farmers [116]. Disease prevalence is notably high in endemic zones such as Uttar Pradesh, Bihar, and West Bengal, where incidence rates can fluctuate between 10 and 90%, depending on local soil conditions and the susceptibility of cultivated varieties [117].

At the state level, consistent yield reductions of 20–30% have been documented in parts of eastern Rajasthan, Uttar Pradesh, and Maharashtra, with field-level mortality occasionally exceeding 50%, especially during peak infection periods between November and December [118]. In neighbouring Bangladesh, which shares agro-climatic similarities with eastern India, household-level cultivation of brinjal has experienced up to 100% plant mortality due to unmanaged *Fusarium* outbreaks [119].

Furthermore, post-harvest losses contribute substantially to overall economic impact. *Fusarium*-infected fruits are prone to rapid quality deterioration, causing losses of around 7% at the farm level and up to 15% during transportation and marketing [120]. Overall post-harvest losses in vegetables range from 20 to 50%, depending on storage and transport conditions [121].

Fusarium wilt is particularly severe in acidic ($pH < 6.4$) and alkaline ($pH > 7.0$) soils, which provide a conducive environment for pathogen proliferation. Therefore, effective disease management remains a critical challenge to improving brinjal productivity, safeguarding economic returns, and ensuring food security for vulnerable farming communities in affected regions [119].

4 Integrated disease management (IDM)

Effective management of Fusarium wilt in brinjal (*F. solani* f. sp. *melongenae*) requires an integrated approach combining cultural, mechanical, chemical, biological, host resistance, and advanced biotechnological strategies.

The management of brinjal wilt requires an integrated approach involving cultural practices, resistant cultivars, biological agents, and chemical treatments to reduce disease incidence and sustain productivity. Fusarium wilt is generally monocyclic, with limited in-season spread due to delayed formation of infectious propagules; however, variation in symptom onset can mimic secondary spread [122]. Plant defence mechanisms like the hypersensitive response (HR) restrict pathogen infection through localized cell death [123]. In Japan, grafting brinjal onto resistant rootstocks is commonly practiced to manage wilt [124, 125]. The nature of the pathogen, its broad host range across 44 plant families, and multiple transmission routes (root contact, soil, tools, insects) complicate control efforts [126]. Biological control using non-pathogenic *F. oxysporum* and other soil antagonists has shown promise in suppressive soils [127], while the use of resistant varieties remains the most effective long-term solution [128].

4.1 Chemical management

The chemical management of brinjal plants involves the strategic application of pesticides and fertilizers to optimize crop health, minimize pests, and enhance yield, as detailed in agricultural practices and studies. Nitrogen is recognized as a crucial macronutrient that plants require for their growth, development, and yield [129]. Azole fungicides like Difenoconazole, Hexaconazole, Propiconazole, and Tebuconazole are particularly effective at inhibiting pathogens by targeting sterol biosynthesis in the cell membrane [130]. In vitro condition, @ 1.0% concentration of chitosan displayed the strongest inhibitory effect on the test pathogen [131]. Mancozeb at 0.20% concentration was successful in preventing pathogen development, increasing plant growth, and HR, and providing significant protection in vitro and greenhouse investigations. Mancozeb's effectiveness against *F. oxysporum* f.sp. *melongenae* was evaluated in a controlled laboratory setting using the poisoned food technique [132]. Carbendazim and Captan were applied at a concentration of 0.20%, and they resulted in a notable increase in brinjal seed germination, reaching a rate of 95% [133]. The fungicidal treatment yielded significant results compared to the control group, with seed germination reaching 68% and a vigor index of 306 being recorded [134]. Using the synthetic fungicide M-45 at a concentration of 50% was found to boost tomato seed germination to 90% and enhance seedling vigor to 1406.7 [135]. Carbendazim (0.1%) was found to effectively inhibit the growth of the tomato wilt-inducing pathogen *F. oxysporum* f.sp. *lycopersici* in both in-vitro and in-vivo conditions. this fungicide outperformed other fungicides [136]. Prochloraz and bromuconazole, along with Benomyl and Carbendazim, exhibited significant effectiveness against the pathogen and Carbendazim (Autostin 50 WP) demonstrated effectiveness in the control of *F. oxysporum* both in vitro and field conditions [137]. Plant-derived pesticides with biological activity are anticipated to have a growing importance in crop protection strategies [138]. The selective and excessive use of chemical pesticides can have detrimental impacts on the environment and ecology, often resulting in residual effects on brinjal plants [139]. As a consequence of this situation, the export of brinjal from Bangladesh faces restrictions, posing a significant concern for the host country [140]. The summary of chemical constituents of Brinjal have been mentioned in Table 2.

4.1.1 Effect of agrochemicals

Controlling wilt disease through chemical means can be quite challenging, and at times, finding suitable chemicals that are readily accessible can be impractical [141, 142]. Furadan 5G is being employed to manage the pathogen affecting brinjal plants [142, 143]. Acibenzolar-S-methyl utilised as an abiotic plant stimulant, and a non-host strain of *F. oxysporum* applied to brinjal seedlings, were employed to enhance resistance against Fusarium wilt, caused by *F. oxysporum* f. sp. *melonis*, in brinjal plants [144]. Efficacy of four systemic fungicides (Topsin-M, Difenoconazole, Alliete, and Nativo) and *Bacillus subtilis* in both laboratory and greenhouse conditions. The poison food technique was used in the lab, while soil drenching was applied in the greenhouse [145]. Potassium application and soil liming are employed as measures to create unfavourable conditions for the fungus [82]. The use of chemical pesticides carries the risk that pests can develop resistance to these pesticides. Autostin 50WP (Carbendazim), a fungicide, was tested for its efficacy against *F. oxysporum* using the Food Poisoning Technique, specifically employing the cup or groove method [23]. The use of chemical pesticides

Table 2 Chemical constituents of Brinjal (*Solanum melongena* L.)

Sr. No.	Chemical constituents	Type and amount (%)	References
1	Carbohydrate	Starch, simple sugars, and dietary fiber (5–6%)	Naeem and Ugur (2019)
2	Proteins	1–2% [Leucine (7–10%), Lysine (5–7%), Phenylalanine (4–6%)] [Alanine (5–8%), Glutamine (8–12%), Glycine (2–4%)]	Naeem and Ugur (2019)
3	Lipids	0.1–0.3%	Naeem and Ugur (2019)
4	Saturated fatty acid	Palmitic and stearic acid (20–30%)	Naeem and Ugur (2019)
5	Unsaturated fatty acid	Oleic acid and linoleic acid (60–70%)	Naeem and Ugur (2019)
6	Phospholipids	Phosphatidylcholine and Phosphatidylethanolamine (10–15%)	Naeem and Ugur (2019)
7	Polyphenols	Polyphenols Caffeic acid, Rutin	Oladosu et al., (2021)
8	Flavonoids	Quercetin, Kaempferol	USDA (2012)
9	Alkaloids	Solanine, Solasonine	Oladosu et al. (2021)
10	Terpenoids	α-Sitosterol, β-Sitosterol	Oladosu et al. (2021)
11	Vitamins	Vitamin K (Phylloquinone) Vitamin E (α-tocopherol)	USDA (2012)
12	Minerals	Zn, Na, K, P, Mg, Fe, Ca	USDA (2012)
13	Water	90%	USDA (2012)

carries the risk that pests can develop resistance to these pesticides. The effectiveness of eight systemic fungicides was assessed at concentrations of 500, 1000, and 1500 ppm against *F. oxysporum* f. sp. *melongenae* using the poisoned food technique *in vitro* [146]. The order of mycelial growth observed was as follows: Difenconazole, Propiconazole, Azoxystrobin, Tebuconazole, Hexaconazole, Thiophanate methyl, and Fosetyle AL [147].

4.1.2 Regulatory status of key fungicides

Several fungicides such as Carbendazim, Thiophanate-methyl, and Mancozeb have been widely reported in the literature for the control of Fusarium wilt in brinjal. However, their current regulatory status differs significantly across regions [148]. Carbendazim and Thiophanate-methyl, both belonging to the benzimidazole class of fungicides, have been banned in the European Union due to concerns over their toxicity, endocrine-disrupting potential, and environmental persistence [149].

Mancozeb, a dithiocarbamate fungicide, was also prohibited in the EU in 2021 under Regulation (EU) No. 2020/2087 because of its potential to impair reproduction and its classification as a suspected endocrine disruptor [150].

In contrast, these fungicides remain registered and widely used in many non-European countries, including India, Bangladesh, and several nations across Africa and Latin America [151]. In these regions, they continue to be part of integrated disease management programs and are often applied under regulated conditions, sometimes in combination with biological control agents to enhance their efficacy and mitigate resistance development [152]. Therefore, while historical data supports the efficacy of these fungicides against *Fusarium* spp., it is essential to consider regional regulatory frameworks when making recommendations. The inclusion or exclusion of such chemicals in integrated pest management strategies should reflect both their proven effectiveness and compliance with current national or international safety regulations.

4.2 Biological management

Using helpful microorganisms and eco-friendly practices, the biological approach to managing fungi in brinjal farming is both natural and chemical-free. Bioactive substances derived from an antagonistic fungus were identified for their role in suppressing Fusarium wilt in tomato and brinjal [153, 154]. Biological control [155]. Bacteria have been investigated as biocontrol agents for plant diseases. *Bacillus subtilis* strains display advantageous characteristics for the host plant and have demonstrated encouraging outcomes when used as bioinoculants. They have the potential to be utilized the development of novel, safer, and more efficient formulations as a substitute for chemical fungicides [156]. Biochar, created through pyrolysis, was found to reduce the growth rate of *Fusarium* spp., in soils [157]. Soils treated with biochar showed a 50% reduction compared to untreated soils [158]. Vermicomposting refers to the natural decomposition of organic matter through the actions of earthworms in controlled environments [159]. For every 1 kg of mushroom production, approximately 5 kg of SMS is generated [160]. Due to its high cation exchange capacity (CEC), slow mineralization rate, and nutrient-rich composition, spent mushroom compost (SMC) has been identified as a valuable nutrient source for agriculture [161]. It naturally manages soil infections, minimizes plant damage, and enhances crop yield. Nine organic amendments tested against *F. oxysporum* f. sp. *melongenae*, neem seed kernel cake was the most effective in reducing disease incidence, followed by mustard cake. Other amendments like safflower cake, groundnut cake, and vermicompost also had positive effects [126]. SMS is abundant in various microorganisms, including beneficial bacteria and fungi that combat diseases. It is biodegradable, safe to use, and cost-effective to produce [162]. The initial step in biological control is to identify and utilize highly effective strains [163]. Poultry manure has a high nutritional value since it contains nitrogen, phosphorus, and potassium [164]. The incidence of wilt can be reduced by 41.15% by poultry manure [165]. *Bacilli* and *Pseudomonas* in the rhizosphere can compete with root pathogens [166]. To reduce the environmental risks associated with fungicides, biotic agents such as rhizobacteria, non-pathogenic fungi, plant extracts, and inducers like acibenzolar S-methyl and salicylic acid have been employed as substitutes in controlling *F. oxysporum* f.sp. *melongenae*, replacing the use of chemical fungicides [167]. Where *Trichoderma harzianum* was applied individually or in combination with Furadan 3G, Krosin 10SP, Autostin 50WP, and poultry manure, no wilt incidence was observed [104].

4.2.1 Effect of trichoderma

In summary, when assessing cost-benefit ratios, Sawdust application had the highest ratio at 4.17, followed by Furadan 5G, *Trichoderma harzianum* Tn, and Grafting, all with favourable ratios, while the Control group had a much lower ratio at 0.70. *Trichoderma* spp., is seen as effective bio-control agents and promoters of crop growth [168]. In a pot culture experiment, The beneficial roles of biocontrol agents such as *Trichoderma harzianum, Trichoderma viride, Glomus mosseae, and Glomus fasciculatum* in combating *F. oxysporum* on brinjal [169]. Aqueous exudates from neem trees (*A. indica*) and willow trees (*Salix babylonica*) both had a 10% influence on Fusarium wilt [170]. A biological control agent fungus (*Trichoderma harzianum*) for the in vitro and in vivo biocontrol of *F. oxysporum*, which causes Fusarium wilt diseases in brinjal plants and They concluded that using *T. harzianum* to control *F. oxysporum* significantly reduced the damage to

brinjal plants [54]. *Trichoderma harzianum* is a helpful natural organism that can protect plants from diseases like Fusarium wilt [171]. A method known as the dual culture technique was employed to assess how *T. harzianum* interacts with *F. oxysporum* in a controlled laboratory environment [54]. Both individual and combined use of biological control agents reduced the severity of wilt disease caused by the pathogen [172]. Among them, the *T. atroviride* isolate, particularly when combined with bacteria, proved to be the most effective treatment [103]. Among all the isolates tested, *Trichoderma asperellum* (TB3) was highly effective in suppressing the radial mycelial growth of the pathogen, with an inhibition rate of 85.77%. Applying *Trichoderma* suspension before brinjal flowering effectively suppresses wilt disease, while combining Metalaxyl and Captan with *Trichoderma virens* and *T. harzianum* enhances efficacy beyond their individual effects [116, 173]. Five *Trichoderma* strains, including *T. harzianum* and *T. reesei*, were tested using the dual culture technique to combat brinjal wilting caused by *F. solani* [174].

4.2.2 Effect of plant extract

Ginger (*Zingiber officinale*) and the fungicide Shincar (carbendazim) showed strong effectiveness against wilt disease. *T. harzianum*, a non-pathogenic fungus, thrives by outcompeting harmful microbes for space and nutrients in the root zone [175, 176]. *T. harsianum* and *T. viride* demonstrated complete suppression of the mycelial growth of *F. oxysporum* [177]. Various plant parts from different species, including leaves and seeds of *Lantana camara, Luffa cylindrica* leaves*, Moringa oleifera* leaves, *Putranjiva roxburghii* leaves and seeds, *Salvadora persica* leaves, *Senna alata* leaves, *Trema orientales* leaves, and *Trichosanthes dioica* leaves, showed potential to inhibit the growth of specific seed-borne fungi and improved the germination of brinjal seeds [178].

4.3 Mechanical management

Mechanical management of fungi in brinjal cultivation involves physical methods, like pruning affected parts and using barriers, as practical strategies to control fungal diseases, detailed in agricultural practices. The most commonly employed method for disease control is the utilization of resistant varieties. However, the effectiveness of this approach has been hindered due to the substantial variability exhibited by the pathogen [179]. Wilt management involves crop rotation, development of resistant cultivars, soil solarization or sterilization, and selective application of fungicides [180]. *F. oxysporum* is employed as a biological control agent to manage *Cannabis sativa* cultivation, specifically targeting marijuana production [181]. *F. oxysporum* has been linked to biocontrol strategies targeting Striga weed [182]. Derosal and Benomyl achieved a 100% reduction in disease incidence, while Vitavax and Cabrio-top showed disease incidences of 96.33% and 83.37%, respectively [183].

4.4 Cultural management

Cultural management forms the foundational layer of integrated disease management (IDM) strategies against Fusarium wilt in brinjal, primarily targeting the reduction of initial inoculum and improving soil and plant health. Crop rotation with non-host plants such as cereals (e.g., maize, sorghum) or legumes (e.g., cowpea, chickpea) is highly effective in breaking the pathogen's lifecycle and minimizing soil inoculum loads [184]. Soil solarization, involving the use of transparent polyethylene sheets during peak summer

months, significantly reduces *Fusarium* spp., propagules by raising soil temperatures to lethal levels and enhancing competitive microbial populations [185]. Proper field drainage, construction of raised beds, and regulated irrigation schedules further reduce root zone moisture and suppress disease development [186]. Sanitary practices such as removal and destruction of infected plant debris, disinfection of tools, and avoidance of continuous monocropping are essential to prevent mechanical dissemination and inoculum buildup [80]. Additionally, maintaining soil pH within a neutral range (6.5–7.0) has been shown to suppress pathogen activity, as acidic or alkaline conditions tend to favor disease proliferation [187]. Timely planting and optimal plant spacing can also reduce disease incidence by minimizing canopy humidity and improving plant vigor. When integrated with other components like biological agents and host resistance, these cultural practices significantly contribute to reducing the severity and spread of Fusarium wilt under field conditions.

4.5 Host resistance

Host resistance is considered one of the most effective and sustainable strategies for managing Fusarium wilt in brinjal. The use of resistant or tolerant varieties reduces reliance on chemical inputs and can significantly lower disease incidence and severity under endemic conditions. Several brinjal genotypes have been screened for resistance to *F. solani* f. sp. *melongenae*, and varying levels of field tolerance have been reported. Varieties such as 'Arka Keshav', 'Arka Nidhi', and 'Arka Neelkanth' developed by the Indian Institute of Horticultural Research (IIHR), Bengaluru, have shown moderate resistance in multilocational trials [188] Similarly, 'Pusa Ankur' and 'Pusa Purple Long' from ICAR-IARI, and local varieties like 'Punjab Neelam' and 'CO-2' have demonstrated field-level tolerance against wilt pathogens under variable soil conditions [189].

In addition to cultivated varieties, wild relatives such as *Solanum torvum*, *S. integrifolium*, and *S. sisymbriifolium* have been identified as potential resistance sources due to their robust root systems and low susceptibility to *Fusarium* spp., These wild species are widely used as rootstocks in grafting techniques, particularly in East Asian countries like Japan and Korea, where they are integrated into IDM programs to confer root-level resistance and enhance plant vigor [190, 191]. Grafted plants have not only shown improved resistance but also better yield performance under wilt-infested soils. The continuous screening of germplasm and development of resistant hybrids through classical breeding and marker-assisted selection remains a priority in brinjal improvement programs aimed at long-term wilt management [190].

4.6 Biotechnological approaches

Recent advances in biotechnology have opened up promising avenues for managing Fusarium wilt in brinjal, especially through CRISPR/Cas9 genome editing, RNA interference (RNAi), and rhizosphere microbial consortia. CRISPR/Cas9 has been used to edit susceptibility-related genes in brinjal to improve defense traits. For example, SmPG1 (polygalacturonase) was successfully knocked out to reduce fungal invasion, demonstrating enhanced resistance to vascular pathogens [192]. CRISPR/Cas9 editing of three *PPO* genes in brinjal significantly reduced enzymatic browning in fruit flesh, establishing a proof-of-concept for targeted quality trait improvement [193]. Similarly, CRISPR/

Cas9-mediated editing of the *SmPPO* gene enhanced postharvest quality in brinjal, reinforcing the potential of genome editing for trait enhancement in brinjal [194].

RNAi-based strategies are also gaining ground. Biosynthesized zinc oxide (ZnO) nanoparticles not only reduced Fusarium wilt severity in brinjal but also enhanced plant immune responses, indicating that RNAi and nanotechnology could be integrated to silence fungal virulence genes and boost host immunity [195]. In parallel, microbial consortia involving beneficial organisms like *Trichoderma harzianum*, *Pseudomonas fluorescens*, and *Bacillus subtilis* have shown consistent success under field conditions. These bioagents, when combined with nanoformulations or low-dose fungicides, enhance soil suppressiveness and systemic resistance. A 58% reduction in wilt incidence was achieved using *T. harzianum*-fortified amendments [196], while combining *T. harzianum* with carbendazim significantly improved both plant growth and disease suppression [197].

Marker-assisted selection (MAS) has further supported resistance breeding, with QTLs like *FM1* on chromosome 2 accounting for up to 92% of resistance to *F. oxysporum* f. sp. *melongenae* [198]. Together, these strategies offer sustainable, targeted, and integrative options for combating Fusarium wilt and enhancing brinjal resilience.

5 Conclusion

Fusarium wilt of brinjal, caused by *F. solani and F. oxysporum* is a destructive soil-borne disease that significantly impacts plant health and yield. The pathogen invades the vascular system, disrupting water transport and causing wilting, yellowing, and plant death. Its ability to produce persistent chlamydospores enables long-term survival in soil, complicating eradication efforts. Environmental factors such as warm temperatures, acidic to neutral pH, and high humidity promotes disease development. Pathogenicity assessment and understanding of the pathogen's enzymatic and virulence factors are essential for early diagnosis and resistance screening. Yield losses can be severe, often exceeding 50% under favourable conditions.

Managing Fusarium wilt requires an integrated disease management (IDM) strategy that combines cultural, biological, chemical, and genetic approaches. Cultural methods like crop rotation, soil solarization, and sanitation reduce inoculum levels. Biocontrol agents such as *Trichoderma* spp., *Bacillus subtilis*, and natural plant extracts have shown efficacy in suppressing the pathogen. Although fungicides like carbendazim and mancozeb are effective, their regulatory status varies by region, and overuse may lead to resistance and environmental harm. Host resistance remains the most sustainable strategy, with ongoing efforts to develop resistant varieties supported by biochemical and molecular tools. Recent advances such as CRISPR/Cas9 gene editing, RNA interference, marker-assisted selection, and microbial consortia present innovative tools for improving resistance and managing the disease sustainably. Continued research on host–pathogen interactions, resistance breeding, and eco-friendly technologies will be vital to safeguard brinjal cultivation against Fusarium wilt what is the effective.

Abbreviations

DM	Disease management
SBP	Soil-borne pathogen
HPI	Host–pathogen interaction
RB	Resistance breeding
SMS	Spent mushroom substrate
SMC	Spent mushroom compost
BCAs	Biological control agents

FS *Fusarium solani*
FO *Fusarium oxysporum*

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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Author contributions
RSS: Collected the data & written the manuscript. JBS: contributed as a coauthor. MBW: reviewed and corrected it. All authors have read and approved the final manuscript.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
In this experiment author not collecting any plant samples from field, they record the data from farmers feld only. So, plant ethical not applicable for this survey study.

Consent for publication
All author have given their concern for publication of this manuscript.

Competing interests
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References

1. Choudhary B, Gaur K. Nutritional and economic significance of brinjal (*Solanum melongena* L.). Hortic Sci J. 2021;10(2):45–52. https://doi.org/10.4236/blr.2022.134051.
2. Kumar R, Khan A, Singh P, Singh A, Srivastava A. *Fusarium* infection of eggplant: disease cycle and management strategies. In: Shahid M, Gaur R, editors. Molecular dynamics of plant stress and its management. Singapore: Springer Nature Singapore; 2024. p. 281–306. https://doi.org/10.1007/978-981-97-1699-9_12.
3. Das S, Rabha J, Narzary D. Assessment of soil yeasts *Papiliotrema laurentii* S-08 and *Saitozyma podzolica* S-77 for plant growth promotion and biocontrol of *Fusarium* wilt of brinjal. J Appl Microbiol. 2023;134(11):1–12. https://doi.org/10.1093/jambio/lxad252.
4. Jatav NK, Shekhawat KS, Balai LP. Chemical control of wilt of brinjal (*Solanum melongena* L.) caused by *Fusarium oxysporum* f. sp. melongenae (Schlecht) Mutuo and Ishigami. Eco Env Cons. 2013;18(4):1049–52. https://doi.org/10.13140/RG.2.2.27054.64323.
5. Patil VM, Patole KR, Paprikar MS, Rajput JC. Biological control of brinjal wilt caused by *Fusarium oxysporum* f. sp. *melongenae* using soluble powder formulation of *Aspergillus niger*. Int J Adv Res Biol Sci. 2017;4(11):66–71. https://doi.org/10.22192/ijarbs.2017.04.11.009.
6. Anuradha B, Kumar AK, Narayana DL, Laxmi KV, Goud CR, Mishra RP. Tomato and brinjal germplasm screening and rootstock identification for Fusarium wilt resistance. Int J Plant Soil Sci. 2023;35(4):1–9. https://doi.org/10.9734/IJPSS/2023/v35i102938.
7. Rao VG, Viswanath HS, Ambadkar CV, Navgire KD, Apet KT. Management of Fusarium wilt (*Fusarium oxysporum* f. sp. melongenae) using organic soil amendments in eggplant. Int J Plant Soil Sci. 2022;34(24):47–56. https://doi.org/10.9734/ijpss/2022/v34i242612.
8. Sundaramoorthy S. Spatial distribution, isolation, and characterization of *Fusarium* spp., causing wilt in brinjal. Asian J Adv Res. 2024;7(3):1–10. https://doi.org/10.9734/acri/2025/v25i31093.
9. Suansia A, Samal KC. Vegetable grafting: a sustainable and eco-friendly strategy for soil-borne pest and disease management. J Pharmacogn Phytochem. 2021;10(1):1634–42.
10. Mahadevakumar S, Janardhana GR. *Phomopsis vexans* (Sacc. & Syd.) harter: current research and future perspectives (1914–2015). Res Rev J Bot Sci. 2016;5(S3):1–9.
11. Himabindu P, Kumar V. Methods for management of Fusarium wilt in tomato. Int J Curr Microbiol App Sci. 2021;10(1):363–71. https://doi.org/10.20546/ijcmas.2021.1001.045.
12. Yadav RA. A comprehensive review on management of soil-borne diseases through vegetable grafting. GRIN eBook; 2020. p. 1–13.

13. Leslie JF, Summerell BA. The *Fusarium* laboratory manual. Blackwell Publishing; 2006. https://doi.org/10.1002/9780470278376.
14. Nelson PE, Dignani MC, Anaissie EJ. Taxonomy, biology, and clinical aspects of *Fusarium* species. Clin Microbiol Rev. 1994;7(4):479–504. https://doi.org/10.1128/CMR.7.4.479.
15. Van Diepeningen AD, Al-Hatmi AMS, Brankovics B, de Hoog GS. Taxonomy and clinical spectra of *Fusarium* species: Where do we stand in 2014? Curr Clin Microbiol Rep. 2014;1:10–8. https://doi.org/10.1007/s40588-014-0003-x.
16. Booth C. Perfect states (teleomorphs) of *Fusarium* species. In: Nelson PE, Toussoun TA, Cook RJ, editors. *Fusarium:* diseases, biology, and taxonomy. Pennsylvania State University Press; 1981. p. 446–52.
17. Chimbekujwo IB. Frequency and pathogenicity of *Fusarium* wilts (*Fusarium solani* and *Fusarium equiseti*) of cotton (*Gossypium hirsutum*) in Adamawa, Nigeria. Rev Biol Trop. 2000;48(1):1–5. https://doi.org/10.15517/rbt.v48i1.18129.
18. Larone DH. Medically important fungi: a guide to identification. 3rd ed. Washington: ASM Press; 1995.
19. Geiser DM, Aoki T, Bacon CW, Baker SE, Bhattacharyya MK, Brandt ME, Zhang N, et al. One fungus, one name: defining the genus *Fusarium* in a scientifically robust way that preserves longstanding use. Phytopathology. 2021;111(4):599–601. https://doi.org/10.1094/PHYTO-08-20-0330-LE.
20. O'Donnell K, Rooney AP, Proctor RH, et al. Phylogenetic and genomic analyses support reclassification of *Fusarium solani* species complex. Stud Mycol. 2020;95:245–92. https://doi.org/10.1016/j.simyco.2020.02.001.
21. Summerell BA. Resolving *Fusarium*: current status of the genus. Annu Rev Phytopathol. 2019;57:323–39. https://doi.org/10.1146/annurev-phyto-082718-100117.
22. Mohd-Hafifi AB, Mohamed Nor NMI, Zakaria L, Mohd MH. Molecular phylogeny and genetic diversity of *Fusarium oxysporum* from various hosts in Malaysia. Sci Rep. 2024;14(1):29708. https://doi.org/10.1038/s41598-024-29708-9.
23. Mekhaeel S, Abdulhameed T, Abduljawad A. Horticulture and vegetable diseases. Mosul: Mosul University Press; 1981. p. 281.
24. Bogale M, Steenkamp ET, Wingfield MJ, Wingfield BD. Diverse *Fusarium solani* isolates colonise agricultural environments in Ethiopia. Eur J Plant Pathol. 2009;124(3):369–78.
25. Theradimani M, Amutha C, Parthasarathy S. Efficacy of antagonists, oil cakes, and fungicides against *Fusarium* wilt disease in brinjal. Madras Agric J. 2019;106(10–12):604–8.
26. Tiwari V, Singh R, Pandey AK. Efficacy of some antagonistic fungi and botanicals against *Fusarium solani* causing damping-off disease in eggplant (*Solanum melongena* L.). J Appl Biosci. 2017;43(1):47–56. https://doi.org/10.13140/RG.2.2.23978.72648.
27. Shishido M, Miwa C, Usami T, Amemiya Y, Johnson KB. Biological control efficiency of *Fusarium* wilt of tomato by non-pathogenic *Fusarium oxysporum* Fo-B2 in different environments. Phytopathology. 2005;95(9):1072–80. https://doi.org/10.1094/PHYTO-95-1072.
28. Sajeena A, Nair DS, Sreepavan K. Non-pathogenic *Fusarium oxysporum* as a biocontrol agent. Indian Phytopathol. 2020;73(2):177–83. https://doi.org/10.1007/s42360-020-00226-x.
29. Salim HA, Simon S. Effect of carbendazim and solarized soil with *Pseudomonas fluorescens*, spent mushroom compost against *Fusarium oxysporum* f. sp. *lycopersici* in tomato. Eur Acad Res. 2015;2(12):15997–6010.
30. Nelson AJ, Elias KS, Arévalo G, Darlington LC, Bailey BA. Genetic characterization by RAPD analysis of isolates of *Fusarium oxysporum* f. sp. *erythroxyli* associated with an emerging epidemic in Peru. Phytopathology. 1997;87(12):1220–5. https://doi.org/10.1094/PHYTO.1997.87.12.1220.
31. Joshi M, Srivastava R, Sharma AK, Prakash A. Screening of resistant varieties and antagonistic *Fusarium oxysporum* for biocontrol of *Fusarium* wilt of chilli. J Plant Pathol Microbiol. 2012;3(5):134. https://doi.org/10.4172/2157-7471.1000134.
32. Rana A, Sahgal M, Johri BN. *Fusarium oxysporum*: genomics, diversity, and plant–host interaction. In: Gupta VK, Tuohy MG, Ayyachamy M, Turner KM, O'Donovan A, editors. Developments in fungal biology and applied mycology. Singapore: Springer; 2017. p. 159–99. https://doi.org/10.1007/978-981-10-4768-8_10.
33. Steinkellner S, Langer I, García-Garrido JM. A comparison of *Fusarium oxysporum* f. sp. lycopersici germination in the presence of root exudates from tomato cultivars. Plant Soil. 2005;269(1):251–60. https://doi.org/10.1007/s11104-004-0599-1.
34. Akhter A, Hage-Ahmed K, Soja G, Steinkellner S. Potential of *Fusarium* wilt-inducing chlamydospores, in vitro behaviour in root exudates and physiology of tomato in biochar and compost amended soil. Plant Soil. 2016;406(1):425–40. https://doi.org/10.1007/s11104-016-2897-3.
35. Schippers B, van Eck WH. Formation and survival of chlamydospores in *Fusarium*. In: Nelson PE, Toussoun TA, Cook RJ, editors. Fusarium: diseases, biology, and taxonomy. University Park: Pennsylvania State University Press; 1981. p. 250–60. https://doi.org/10.1023/A:1015104903622.
36. Wollenweber HW. *Fusarium*-monographie. Fungi parasitici et saprophytici Z Parasitenkd. 1931;3(3):269–516. https://doi.org/10.1007/BF02120198.
37. Armstrong GM, Armstrong JK. Formae speciales and races of *Fusarium oxysporum* causing wilt diseases. In: Nelson PE, Toussoun TA, Cook RJ, editors. Fusarium: Diseases, Biology, and Taxonomy. University Park: Pennsylvania State University Press; 1981. p. 391–9.
38. Ohara T, Inoue I, Namiki F, Kunoh H, Tsuge T. REN1 is required for development of microconidia and macroconidia, but not of chlamydospores, in the plant pathogenic fungus *Fusarium oxysporum*. Genetics. 2004;166(1):113–24. https://doi.org/10.1534/genetics.166.1.113.
39. Beckman CH. The nature of wilt diseases of plants. Am Phytopathol Soc Press. 1987. https://doi.org/10.1086/415877.
40. Bhandari S, Niraula D, Adhikari K. *Fusarium* and Verticillium wilt in cotton: a review. Environ Contam Rev. 2020;3(1):48–52. https://doi.org/10.26480/ecr.01.2020.48.52.
41. Agrios GN. Plant pathology. 5th ed. Burlington: Elsevier Academic Press; 2005. p. 952. https://doi.org/10.1016/C2009-0-02037-6.
42. Ploetz RC. *Fusarium* wilt of banana. Phytopathology. 2005;95(12):1468–9. https://doi.org/10.1094/PHYTO-95-1468A.
43. Egbuta MA, Mwanza M, Babalola OO. A review of the ubiquity of ascomycetes filamentous fungi in relation to their economic and medical importance. Adv Microbiol. 2016;6(14):1140–58. https://doi.org/10.4236/aim.2016.614103.
44. Matsuo T, Ishigami K. On the wilt of *Solanum melongena* L. and its causal fungus *Fusarium oxysporum* f. sp. melongenae. Ann Phytopathol Soc Jpn. 1958;23(4):189–92. https://doi.org/10.3186/jjphytopath.23.189.
45. Bishop CD, Cooper RM. An ultrastructural study of vascular colonization in three vascular wilt diseases: I. Colonization of susceptible cultivars. Physiol Plant Pathol. 1983;23(3):323–43. https://doi.org/10.1016/0048-4059(83)90048-2.

46. Srinivas C, Nirmala Devi D, Narasimha Murthy K, Mohan CD, Lakshmeesha TR, Singh B, Kalagatur NK, Niranjana SR, Hashem A, Alqarawi AA, Tabassum B, Abd-Allah EF, Chandra Nayaka S, Srivastava RK. *Fusarium oxysporum* f. sp. *lycopersici*, causal agent of vascular wilt disease of tomato: biology to diversity—a review. Saudi J Biol Sci. 2019;26(7):1315–24. https://doi.org/10.1016/j.sjbs.2019.06.002.
47. Oelke IM, Bosland PW. *Fusarium* diseases of *Capsicum* and eggplant. Capsicum Eggplant Newsl. 2001;20:86–9.
48. Katan J. Symptomless carriers of the tomato *Fusarium* wilt pathogen. Phytopathology. 1971;61(10):1213–7. https://doi.org/10.1094/Phyto-61-1213.
49. Altınok HH. First report of *Fusarium* wilt of eggplant caused by *Fusarium oxysporum* f. sp. *melongenae* in Turkey. Plant Pathol. 2005;54(4):577. https://doi.org/10.1111/j.1365-3059.2005.01235.
50. Meah MB. Diseases in Rabi crops under crop diversification program. Dhaka: Department of Agricultural Extension; 1997. Report Crop Diversification Program, 10.
51. Banu N, Sharada MS. Inhibitory effects of mancozeb on growth and stimulation of resistance against *Fusarium* wilt of brinjal. Bioscan. 2018;13(1):285–90. https://doi.org/10.13140/RG.2.2.20824.16649.
52. Basco MJ, Bisen K, Keswani C, Singh HB. Biological management of *Fusarium* wilt of tomato using biofortified vermicompost. Mycosphere. 2017;8(3):467–83. https://doi.org/10.5943/mycosphere/8/3/8.
53. Biswas MK, Ghosh T. Evaluation of phyto-extracts, biological agents, and chemicals against the development of *Alternaria* brassicae in vitro and in vivo. Eur J Med Plants. 2018;22(3):1–9. https://doi.org/10.9734/EJMP/2018/40412.
54. Kareem HJ, Al-Araji AM. Evaluation of *Trichoderma harzianum* biological control against *Fusarium oxysporum* f. sp. melongenae. Iraqi J Sci. 2017;58(4B):2051–60. https://doi.org/10.24996/ijs.2017.58.4B.7.
55. Di Pietro A, Madrid MP, Caracuel Z, Delgado-Jarana J, Roncero MI. *Fusarium oxysporum*: exploring the molecular arsenal of a vascular wilt fungus. Mol Plant Pathol. 2003;4(4):315–25. https://doi.org/10.1046/j.1364-3703.2003.00180.
56. Chung GF. Effect of pests and diseases on oil palm yield. In: Lai OM, Tan CP, Akoh CC, editors. Palm oil: production, processing, characterization, and uses. Urbana: AOCS Press; 2012. p. 163–210. https://doi.org/10.1016/B978-0-9818936-9-3.50011-0.
57. Mendgen K, Hahn M, Deising H. Morphogenesis and mechanisms of penetration by plant pathogenic fungi. Annu Rev Phytopathol. 1996;34(1):367–86. https://doi.org/10.1146/annurev.phyto.34.1.367.
58. Ortiz E, Cruz M, Melgarejo LM, Marquínez X, Hoyos-Carvajal L. Histopathological features of infections caused by *Fusarium oxysporum* and *F. solani* in purple passionfruit plants (*Passiflora edulis* Sims). Summa Phytopathol. 2014;40(2):134–40. https://doi.org/10.1590/0100-5405/1910.
59. Olivain C, Humbert C, Nahalkova J, Fatehi J, L'Haridon F, Alabouvette C. Colonization of tomato root by pathogenic and nonpathogenic *Fusarium oxysporum* strains inoculated together and separately into the soil. Appl Environ Microbiol. 2006;72(2):1523–31. https://doi.org/10.1128/AEM.72.2.1523-1531.2006.
60. Agrios GN. Plant pathology. 5th ed. Burlington: Elsevier Academic Press; 2006. p. 922. https://doi.org/10.1016/C2009-0-02037-6.
61. Beckman CH. The nature of wilt diseases of plants. St. Paul: American Phytopathological Society Press; 1987. p. 175. https://doi.org/10.1086/415877.
62. Schnathorst WC. Life cycle and epidemiology of *Verticillium*. In: Mace ME, Bell AA, Beckman CH, editors. Fungal wilt diseases of plants. New York: Academic Press; 1981. p. 81–111.
63. Huisman OC. Interrelations of root growth dynamics to epidemiology of root-invading fungi. Annu Rev Phytopathol. 1982;20(1):303–27. https://doi.org/10.1146/annurev.py.20.090182.001511.
64. Inoue I, Namiki F, Tsuge T. Plant colonization by the vascular wilt fungus *Fusarium oxysporum* requires FOW1, a gene encoding a mitochondrial protein. Plant Cell. 2002;14(8):1869–83. https://doi.org/10.1105/tpc.002576.
65. Rodríguez-Molina MC, Medina I, Torres-Vila LM, Cuartero J. Vascular colonization patterns in susceptible and resistant tomato cultivars inoculated with *Fusarium oxysporum* f. sp. *lycopersici* races 0 and 1. Plant Pathol. 2003;52(2):199–203. https://doi.org/10.1046/j.1365-3059.2003.00810.
66. Hardham AR. The cell biology behind *Phytophthora* pathogenicity. Australas Plant Pathol. 2001;30(2):91–8. https://doi.org/10.1071/AP01006.
67. Wanjiru MW, Kang Z, Buchenauer H. Importance of cell wall degrading enzymes produced by *Fusarium graminearum* during infection of wheat heads. Eur J Plant Pathol. 2002;108:803–10. https://doi.org/10.1023/A:1020847216155.
68. Mandeel QA. Modeling competition for infection sites on roots by nonpathogenic strains of *Fusarium oxysporum*. Mycopathologia. 2007;163(1):9–20. https://doi.org/10.1007/s11046-006-0080-3.
69. Mace ME, Bell AA, Beckman CH, editors. Fungal wilt diseases of plants. Elsevier; 2012.
70. Magyar D, Vass M, Li D-W. Dispersal strategies of microfungi. In: Li D-W, editor. Biology of microfungi. Cham: Springer; 2016. p. 315–71. https://doi.org/10.1007/978-3-319-29137-6_14.
71. VanderMolen GE, Beckman CH, Rodehorst E. Vascular gelation: a general response phenomenon following infection. Physiol Plant Pathol. 1977;11(1):95–100. https://doi.org/10.1016/0048-4059(77)90012-9.
72. Moreau M, Catesson AM, Péresse M, Czaninski Y. Comparative dynamics of cytological reactions in carnation xylem in the presence of vascular parasites. Phytopathol Z. 1978;91:289–306.
73. Doohan FM, Zhou B. Fungal pathogens of plants. In: Kavanagh K, editor. Fungi: biology and applications. 2nd ed. Wiley-Blackwell; 2017. p. 355–87. https://doi.org/10.1002/9781119374340.ch14.
74. Fassihiani A. Symptomless carriers of the causal agent of tomato wilt pathogen. J Agric Sci Technol. 2000;2:27–32. https://doi.org/10.1094/PHYTO-61-1213.
75. Lal D, Dev D, Kumari S, Pandey S, Aparna S, Sharma N, Nandni S, Jha RK, Singh A. *Fusarium* wilt pandemic: current understanding and molecular perspectives. Funct Integr Genomics. 2024;24(2):41. https://doi.org/10.1007/s10142-024-01319-w.
76. Barnett HL, Hunter BB. Illustrated genera of imperfect fungi. 3rd ed. Burgess Publishing; 1972.
77. Ma LJ, Geiser DM, Proctor RH, Rooney AP, O'Donnell K, Trail F, Gardiner DM, Manners JM, Kazan K. *Fusarium* pathogenomics. Annu Rev Microbiol. 2013;67:399–416. https://doi.org/10.1146/annurev-micro-092412-155650.
78. Ganesh P, Dwivedi SK. Biological and chemical management of *Fusarium solani* causing wilt in eggplants. Int J Sci Res Rev. 2018;7(4):1914–25. https://doi.org/10.3390/medicines5010002.
79. Lucas JA. Plant Pathology and Plant Pathogens. 3rd ed. Blackwell Science; 1998. p. 274.
80. Nelson PE, Toussoun TA. Marasas WFO. Fusarium Species: An Illustrated Manual for Identification. The Pennsylvania State University Press; 1983. p. 193.

81. Gordon TR, Martyn RD. The evolutionary biology of *Fusarium oxysporum*. Annu Rev Phytopathol. 1997;35:111–28. https://doi.org/10.1146/annurev.phyto.35.1.111.
82. Flood J. A review of *Fusarium* wilt of oil palm caused by *Fusarium oxysporum* f. sp. *elaeidis*. Phytopathology. 2006;96(6):660–2. https://doi.org/10.1094/PHYTO-96-0660.
83. Zitter TA. *Fusarium* diseases of cucurbits. Fact Sheet No. 733. Department of Plant Pathology, New York: Cornell University; 1998.
84. Munkvold GP, Gullino ML. Seed and propagative material. In: Gullino ML, Albajes R, Nicot PC, editors. Integrated pest and disease management in greenhouse crops. 2nd ed. Cham: Springer; 2020. p. 331–54. https://doi.org/10.1007/978-3-030-22304-5_11.
85. Joshi R. A review of *Fusarium oxysporum* on its plant interaction and industrial use. J Med Plants Stud. 2018;6(3):112–5. https://doi.org/10.22271/plants.2018.v6.i3b.07.
86. Gardiner DM, Kazan K, Manners JM. Cross-kingdom gene transfer facilitates the evolution of virulence in fungal pathogens. Plant Sci. 2013;210:151–8. https://doi.org/10.1016/j.plantsci.2013.06.004.
87. Zhang DY, Luo HS, Cheng T, Li WF, Zhou XG, Wei G, Gu CY, Diao Z. Enhancing wheat *Fusarium* head blight detection using rotation Yolo wheat detection network and simple spatial attention network. Comput Electron Agric. 2023;211: 107968. https://doi.org/10.1016/j.compag.2023.107968.
88. Palmieri D, Segorbe D, López-Berges MS, De Curtis F, Lima G, Di Pietro A, Turrà D. Alkaline pH, low iron availability, poor nitrogen sources and CWI MAPK signaling are associated with increased fusaric acid production in *Fusarium oxysporum*. Toxins. 2023;15(1):50. https://doi.org/10.3390/toxins15010050.
89. Chen D, Ju M, Xie J, Chen X-L, Peng J, et al. Current progress on pathogenicity-related genes in *Fusarium oxysporum* f. sp. cubense tropical race 4. Phytopathol Res. 2024;6:53. https://doi.org/10.1186/s42483-024-00291-x.
90. Lin C, Feng XL, Liu Y, Li ZC, Li XZ, Qi J. Bioinformatic analysis of secondary metabolite biosynthetic potential in pathogenic *Fusarium*. J Fungi. 2023;9(8): 850. https://doi.org/10.3390/jof9080850.
91. Zhao C, Wiemann P, Xu W, Green H, Cane DE, Keller NP. Functional characterization of *Fusarium* secondary metabolite gene clusters in pathogenicity. Mol Plant Microbe Interact. 2014;27(6):578–89. https://doi.org/10.1094/MPMI-12-13-0375-R.
92. Mwaniki PK, Abang MM, Wagara IN, Wolukau JN, Schroers HJ. Morphology, pathogenicity and molecular identification of *Fusarium* spp., from wilting eggplants in Tanzania. Afr Crop Sci Conf Proc. 2011;10:217–21.
93. Ramaiah AK, Garampalli RKH. In vitro antifungal activity of some plant extracts against *Fusarium oxysporum* f. sp. lycopersici. Asian J Plant Sci Res. 2015;5:22–7. https://doi.org/10.4236/ajps.2014.518281.
94. Islam MM, Islam MR, Stevens C, Meah MB, Miah MAT. Molecular characterization of *Phomopsis vexans* isolates of eggplant of Bangladesh. Bangladesh J Agric Res. 2011;36(3):389–96. https://doi.org/10.3329/bjar.v36i3.10632.
95. Bock CH, Barbedo JGA, Del Ponte EM, Bohnenkamp D, Mahlein AK. From visual estimates to fully automated sensor-based measurements of plant disease severity: status and challenges for improving accuracy. Phytopathol Res. 2020;2:1–30. https://doi.org/10.1186/s42483-020-00049-8.
96. Stoeva DC, Gencheva D, Yordanova R, Beev G. Molecular marker-based identification and genetic diversity evaluation of *Fusarium* spp.: a review. Acta Microbiol Bulgar. 2023;39(4):376–85. https://doi.org/10.59393/amb23390402.
97. Zvirin A, Nitzan N, Mor H, Goldman GH, Sharon A, Yarden O, et al. Differential colonization and defence responses of resistant and susceptible melon lines infected by *Fusarium oxysporum* race 1.2 expressing GFP. Plant Pathol. 2010;59(1):135–44. https://doi.org/10.1111/j.1365-3059.2009.02225.x.
98. Roncero MI, Di Pietro A, Ruiz-Roldán MC, Huertas-González MD, Garcia-Maceira FI, Méglecz E, Páez M. Role of cell wall-degrading enzymes in pathogenicity of *Fusarium oxysporum*. Rev Iberoam Micol. 2000;17(1):S47–53.
99. Cheng J, Peng Y, Yan J, Zhou ML, Tang Y, Gao AJ, Zhou M. Research progress in phytopathogenic fungi and their role as biocontrol agents. Front Microbiol. 2021;12:1209. https://doi.org/10.3389/fmicb.2021.670135.
100. Xia Y, Xu X, Zhang J, Liu H, Chen L, Huang C, et al. The ubiquitous wilt-inducing pathogen *Fusarium oxysporum*: pathogenicity factors and CWDE expression during host infection. Pathogens. 2023;13(10):823. https://doi.org/10.3390/pathogens13100823.
101. Chen M, Wang Y, Zhao Z, Li X, Zhang H, Liu Y, et al. A novel secreted protein of *Fusarium oxysporum* promotes infection via cell wall-degrading enzymes. Plant Cell Environ. 2024;47(1):18–30. https://doi.org/10.1111/pce.15200.
102. Wu J, Wang P, Wang W, Hu H, Wei Q, Bao C, Yan Y. Comprehensive genomic and proteomic analysis identifies effectors of *Fusarium oxysporum* f. sp. *melongenae*. J Fungi. 2024;10(12): 828. https://doi.org/10.3390/jof10120828.
103. Chandrika R, Theradimani M, Thiruvudainambi S, Shanthi M, Renuka R, Brindhadevi S, Ayyandurai M. Evaluation of antifungal activity of *Trichoderma* spp., against the wilt disease of brinjal (*Solanum melongena* L.) caused by *Fusarium solani* f. sp. *melongenae*. Pharma Innov J. 2021;10(10):2112–5.
104. Akter N, Islam MR, Hossain MB, Islam MN, Chowdhury SR, Hoque S, Nitol RH, Tasnin R. Management of wilt complex of eggplant (*Solanum melongena* L.) caused by *Fusarium oxysporum*, *Ralstonia solanacearum* and *Meloidogyne* spp. Am J Plant Sci. 2021;12(7):1155–71. https://doi.org/10.4236/ajps.2021.127080.
105. Das SN. Field efficacy of brinjal wilt with potential fungicides, biocontrol agents, and plant extracts. Nat Volatiles Essent Oils. 2021;8(5):9328–41.
106. Ghosh T, Biswas MK, Guin C, Roy P, Aikat K. A review on seed-borne mycoflora associated with different cereal crop seeds and their management. Plant Cell Biotechnol Mol Biol. 2018;19(3–4):107–17.
107. Komada H. Development of a selective medium for quantitative isolation of *Fusarium oxysporum* from natural soil. Rev Plant Prot Res. 1975;8:114–25. https://doi.org/10.4236/fns.2012.37125.
108. Nash SM, Snyder WC. Quantitative estimations by plate counts of propagules of the bean root rot fungi in field soils. Phytopathology. 1962;52:567–72.
109. Singh RS. Techniques for selective isolation of some soil-borne pathogens. Consultants' Group Discussion on the Resistance to Soil-borne Diseases. 1979;75.
110. Barman KL, Kalita RB, Jha DK. Inductions of resistance in brinjal (*Solanum melongena* L.) by aqueous extract of vermicompost against *Fusarium* wilt. Int J Plant Anim Environ Sci. 2013;3(1):141–8.
111. Baysal Ö, Siragusa M, Gümrükcü E, Zengin S, Carimi F, Sajeva M, Teixeira da Silva JA. Molecular characterization of *Fusarium oxysporum* f. sp. melongenae by ISSR and RAPD markers on eggplant. Biochem Genet. 2010;48(5):524–37. https://doi.org/10.1007/s10528-010-9336-1.

112. Begum N, Robab MI, Wu MD, Ahmed SM, Danish M. Fusarium wilt disease of vegetable crops and its management through biocontrol agent: a review. IJRAS. 2025;12(1):1–12.
113. Food and Agriculture Organization of the United Nations. FAOSTAT Statistical Database: Crops and livestock products. Rome: FAO; 2023. Available from: https://www.fao.org/faostat/en
114. Thompson J. Nomination for pre-plant soil use for eggplants grown in open fields. USA CUN Soil. 2010;12:7–15.
115. National Horticulture Board (NHB). Horticultural Statistics at a Glance 2022. Ministry of Agriculture and Farmers Welfare, Government of India. Available at: http://nhb.gov.in (2022). Accessed 5 July 2025.
116. Adhikary MC, Begum HA, Meah MB. Possibility of recovering *Fusarium* wilt-affected eggplants by *Trichoderma*. Int J Agric Res Innov Technol. 2017;7(1):38–42. https://doi.org/10.3329/ijarit.v7i1.38384.
117. Sahoo R. Biorational approach: an alternative approach to control the wilt diseases of crops. Acta Sci Agric. 2022;6(9):71–7. https://doi.org/10.31080/ASAG.2022.06.1181.
118. Jatav NK, Lal B, Singh SP, Bairwa SK, Meena AK, Koli RS. Evaluation of plant extracts against wilt of brinjal caused by *Fusarium oxysporum* f. sp. *melongenae* (Schlecht) Mutuo and Ishigami. Int J Curr Microbiol App Sci. 2019;8(12):2754–61. https://doi.org/10.20546/ijcmas.2019.812.322.
119. Ali M, Alam MZ, Akanda MA. Grafting: a technique of control soil borne diseases of tomato and eggplant. IPSA-JICA publ. 1994;4:10.
120. Haque M, Miah M, Khatun F, Shajahan M. Postharvest loss assessment of brinjal in selected areas of Bangladesh. Bangladesh J Agric Res. 2004;29(2):313–7.
121. Kader AA, ed. Postharvest Technology of Horticultural Crops. 2nd ed. Oakland: University of California, Division of Agriculture and Natural Resources; 1992. p. 15–17. https://doi.org/10.4236/oalib.1105509
122. Egel DS, Martyn RD. *Fusarium* wilt of watermelon and other cucurbits. The Plant Health Instr. 2007;10:1094. https://doi.org/10.1094/PHI-I-2007-0122-01.
123. Mehdy MC. Active oxygen species in plant defense against pathogens. Plant Physiol. 1994;105(2):467–72. https://doi.org/10.1104/pp.105.2.467.
124. Matsuzoe N, Okubo H, Fujieda K. Resistance of tomato plants grafted on *Solanum* rootstocks to bacterial wilt and root-knot nematode. J Jpn Soc Hortic Sci. 1993;61(4):865–72. https://doi.org/10.2503/jjshs.61.865.
125. Ali M, Matsuzoe N, Okubo H, Fujieda K. Resistance of non-tuberous *Solanum* to root-knot nematode. J Jpn Soc Hortic Sci. 1991;60(4):921–6.
126. Rao VG, Dhutraj DN, Apet KT, Ambadkar CV, Kumar BP, Daunde AT, Bhalerao SR, Sontakke PL, Patil AG. Characterization and variability of *Fusarium oxysporum* f. sp. *melongenae* (Schlecht) Mutuo and Ishigami from wilting eggplants in Marathwada region of Maharashtra. J Pharmacogn Phytochem. 2019;8(5):1436–43.
127. Paulitz TC, Park CS, Baker R. Biological control of *Fusarium* wilt of cucumber with nonpathogenic isolates of *Fusarium oxysporum*. Can J Microbiol. 1987;33(5):349–53. https://doi.org/10.1139/m87-061.
128. Corley RHV, Tinker PB. The oil palm. 4th ed. Oxford: Blackwell Publishing; 2003. https://doi.org/10.1002/9780470750971.
129. Aminifard MH, Aroiee H, Fatemi H, Ameri A, Karimpour S. Responses of eggplant (*Solanum melongena* L.) to different rates of nitrogen under field conditions. J Cent Eur Agric. 2010;11(4):453–8. https://doi.org/10.5513/JCEA01/11.4.863.
130. Ravichandra NG. Agrochemicals in plant disease management. Jodhpur: Scientific Publishers; 2018. p. 230–7.
131. Chaterjee S, Jannat R, Hossain MM, Amin MR, Rubayet MT. Chitosan for suppression of *fusarium* wilt and plant growth promotion of brinjal. J Agric Appl Biol. 2021;2(2):124–37. https://doi.org/10.11594/jaab.02.02.07.
132. Najiya B, Sharada MS. Inhibitory effects of mancozeb on growth and stimulation of resistance against *Fusarium* wilt of brinjal. Bioscan. 2018;13(1):285–90.
133. Thippeswamy BMK, Chakravarthy CN, Sathisha AM, Jyothi SU, Vasantha KK. Pathogenicity and management of *Phomopsis* blight and leaf spot in brinjal caused by *Phomopsis vexans* and *Alternaria solani*. Indian Phytopathol. 2006;59(4):475–81.
134. Choudhary CS, Jain SC, Kumar R, Choudhary JS. Efficacy of different fungicides, biocides, and botanical extract seed treatment for controlling seed-borne *Colletotrichum* sp. in chilli (*Capsicum annuum* L.). Bioscan. 2013;8(1):123–6.
135. Vikhe AM, Reddy PG. Effect of bio-fungicide (leaf extract) and synthetic fungicide on seed germination, seedling development, and seedling vigor in tomato var. NS-2535. Adv Biores. 2015;6(5).
136. Musmade NA, Pillai T, Thakur KD. Biological and chemical management of tomato wilt caused by *Fusarium oxysporum* f.sp. *lycopersici*. J Soils Crops. 2009;19(1):118–21.
137. Amini J, Sidovich D. The effects of fungicides on *Fusarium oxysporum* f. sp. lycopersici associated with *Fusarium* wilt of tomato. J Plant Prot Res. 2010;50(2):172–8. https://doi.org/10.2478/v10045-010-0029.
138. Satish S, Mohana DC, Raghavendra MP, Raveesha KA. Antifungal activity of some plant extracts against important seed borne pathogens of *Aspergillus* sp. J Agric Technol. 2007;3(1):109–19.
139. Parvin RA, Al-Subeihi AA, Mahmud MC, Chowdhury MT, Prodhan MDH, Islam MA. Determination of pesticide residues and health risk assessment in cucumber and eggplant sold in northern part of Bangladesh. Pollut Res. 2021;40(4):1180–7.
140. Rahman Z. Lack of regulations affect vegetable export. The Financial Express. 2016.
141. Mondal B, Bhattacharya R, Ranjan RK, Khatua DC. Bacterial wilt disease of horticultural plants in West Bengal and its chemical control. Green Technol. 2004;6:70–4.
142. Hillocks RJ, Waller JM, editors. Soilborne diseases of tropical crops. Wallingford: CAB International; 1997.
143. Hossain MM, Ali MR, Goswami BK, Hossain MS, Ali MR. Management of root-knot nematode of brinjal with nematicide and organic amendment to soil. Bangladesh J Plant Pathol. 2003;19(1/2):29–32.
144. Altinok HH. Activation of systemic disease resistance by acibenzolar-S-methyl and a non-pathogen *Fusarium oxysporum* melonis (FOM) strain against *Fusarium* wilt disease in eggplant seedlings. J Turk Phytopathol. 2009;38:21–32.
145. Shahid M, Zaidi A, Khan MS, Rizvi A, Saif S, Ahmed B. Recent advances in management strategies of vegetable diseases. In: Zaidi A, Khan MS, editors. Microbial strategies for vegetable production. Cham: Springer; 2017. p. 197–226. https://doi.org/10.1007/978-3-319-54401-4_9.
146. Harshita AS, Khan JB, Tripathi UK, Ratan V, Trivedi S, Mishra A, Verma A. In-vitro evaluation of systemic fungicides against *Fusarium oxysporum* f. sp. lycopersici and their compatibility with bioagents. J Pharmacogn Phytochemistry. 2019;8(3):3117–23.
147. Poudel S, Osti S. In vitro evaluation of efficacy of fungicides against *Fusarium oxysporum* f. sp. lycopersici (Sacc.) Snyder and Hansen causing wilt disease of tomato. NPI J Sci Technol. 2024;4(2):31–48. https://doi.org/10.3126/npijst.v1i1.68938.

148. European Commission. Commission decision concerning the non-inclusion of carbendazim in Annex I to Council Directive 91/414/EEC. Off J Eur Union. 2010. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX:32010D0807
149. European Food Safety Authority (EFSA), Arena M, Auteri D, Barmaz S, Bellisai G, Brancato A, Villamar-Bouza L. Peer review of the pesticide risk assessment of the active substance thiophanate-methyl. EFSA J. 2018;16(1):e05133. https://doi.org/10.2903/j.efsa.2018.5133.
150. European Commission. Commission Implementing Regulation (EU) 2020/2087 of 14 December 2020 concerning the non-renewal of the approval of the active substance mancozeb. Off J Eur Union. 2020. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32020R2087.
151. Central Insecticides Board and Registration Committee (CIBRC). List of registered pesticides under the Insecticides Act, 1968. Ministry of Agriculture, Government of India. 2024. https://ppqs.gov.in.
152. Dhar SS, Chakraborty S, Tumpa FH, Hossain MA, Khokon MAR. Effectiveness of *Glomus mosseae* inoculation in enhancing Fusarium wilt tolerance in eggplant. J Bangladesh Agric Univ. 2024;18(3):593–8. https://doi.org/10.5455/JBAU.113635.
153. Khan Z, Kim YH, Kim SG, Kim HW. Observations on the suppression of root-knot nematode (*Meloidogyne arenaria*) on tomato by incorporation of cyanobacterial powder (*Oscillatoria chlorina*) into potting field soil. Bioresour Technol. 2007;98(1):69–73. https://doi.org/10.1016/j.biortech.2005.11.014.
154. Kanokmedhakul S, Kanokmedhakul K, Prajuabsuk T, Soytong K, Kongsaeree P, Suksamrarn A. A bioactive triterpenoid and vulpinic acid derivatives from the mushroom *Scleroderma citrinum*. Planta Med. 2003;69(6):568–71. https://doi.org/10.1055/s-2003-40639.
155. Singh SS, Gupta P, Gupta AK. Handbook of agricultural sciences. 4th ed. New Delhi: Kalyani Publishers; 2015. p. 184–5.
156. Gerhardson B. Biological substitutes for pesticides. Trends Biotechnol. 2002;20(8):338–43. https://doi.org/10.1016/S0167-7799(02)02021-8.
157. Saha D, Purkayastha GD, Ghosh A, Isha M, Saha A. Isolation and characterization of two new *Bacillus subtilis* strains from the rhizosphere of eggplant as potential biocontrol agents. J Plant Pathol. 2012;94(1):109–18.
158. Akanmu AO, Sobowale AA, Abiala MA, Olawuyi OJ, Odebode AC. Efficacy of biochar in the management of *Fusarium verticillioides* Sacc. causing ear rot in *Zea mays* L. Biotechnol Rep. 2020;26: e00474. https://doi.org/10.1016/j.btre.2020.e00474.
159. Gudeta K, Bhagat A, Julka JM, Sinha R, Verma R, Kumar A, Kumari S, Ameen F, Bhat SA, Amarowicz R, Sharma M. Vermicompost and its derivatives against phytopathogenic fungi in the soil: a review. Horticulturae. 2022;8(4): 311. https://doi.org/10.3390/horticulturae8040311.
160. Zied DC, de Abreu CG, Alves LDS, Prado EP, Pardo-Gimenez A, de Melo PC, Dias ES. Influence of the production environment on the cultivation of lettuce and arugula with spent mushroom substrate. J Environ Manag. 2021;281: 111799. https://doi.org/10.1016/j.jenvman.2020.111799.
161. Verma D, Didwana VS, Maurya B. Spent mushroom substrate: a potential sustainable substrate for agriculture. Int J Grid Distrib Comput. 2020;13(1):667–75. https://doi.org/10.33832/ijgdc.2020.13.1.59.
162. Adedeji K, Aduramigba MA. In vitro evaluation of spent mushroom compost on growth of *Fusarium oxysporum* f. sp. lycopersici. Adv Plants Agric Res. 2016;4(4):332–9. https://doi.org/10.15406/apar.2016.04.00149.
163. Dubey RK, Kumar P, Singh N, Kumar R. Effect of *Trichoderma viride* and *Pseudomonas fluorescens* on growth and flowering of gladiolus. Indian J Ecol. 2008;35(1):97–8.
164. Richa, Kumar V, Singh J, Sharma N. Poultry manure and poultry waste management: a review. Int J Curr Microbiol Appl Sci. 2020;9(6):3483–95. https://doi.org/10.20546/ijcmas.2020.906.409.
165. Jat MK, Ahir RR, Choudhary S, Kakaraliya GL. Management of coriander wilt (*Fusarium oxysporium*) through cultural practices as organic amendments and date of sowing. J Pharmacogn Phytochem. 2017;6(4):1499–501. https://doi.org/10.20546/IJCMAS.2017.609.108.
166. Baker KF, Cook RJ. Biological control of soil-borne pathogens. San Francisco: W.H. Freeman & Co.; 1974. p. 433.
167. Yildiz HN, Altinok HH, Dikilitas M. Screening of rhizobacteria against *Fusarium oxysporum* f. sp. melongenae, the causal agent of wilt disease of eggplant. Afr J Microbiol Res. 2012;6(15):3700–6. https://doi.org/10.5897/AJMR12.307.
168. Abdel-Monaim MF, Abdel-Gaid MA, Zayan SA, Nassef DM. Enhancement of growth parameters and yield components in eggplant using antagonism of *Trichoderma* spp., against *Fusarium* wilt disease. Int J Phytopathol. 2014;3(1):33–40. https://doi.org/10.33687/phytopath.003.01.0510.
169. Wani AH. Biological control of wilt of brinjal caused by *Fusarium oxysporum* with some fungal antagonists. Indian Phytopathol. 2012;41(3):177–85. https://doi.org/10.1094/PHYTO-58-0228.
170. Hanaa RF, Abdou ZA, Salama DA, Ibrahim MA, Sror HAM. Effect of neem and willow aqueous extracts on *Fusarium* wilt disease in tomato seedlings: Induction of antioxidant defensive enzymes. J Agric Sci. 2011;56(1):1–7. https://doi.org/10.1016/j.aoas.2011.05.007.
171. Vinale F, Marra R, Scala F, Ghisalberti EL, Lorito M, Sivasithamparam K. Major secondary metabolites produced by two commercial *Trichoderma* strains active against different phytopathogens. Lett Appl Microbiol. 2006;43:143–8. https://doi.org/10.1111/j.1472-765X.2006.01939.x.
172. Bilginturan M, Karaca GH. Effects of *Trichoderma* and PGPR applications on growth and *Verticillium* wilt of eggplant. Mediterr Agric Sci. 2021;34(3):267–72. https://doi.org/10.29136/mediterranean.897989.
173. Tariq RR, Razdan VK, Tewari AK, Shanaz E, Bhat ZA, Hassan MG, Wani TA. Integrated management of wilt complex disease in bell pepper (*Capsicum annuum* L). J Agric Sci. 2012;4(1):122–6. https://doi.org/10.5539/jas.v4n7p122.
174. Chakraborty MR, Chatterjee NC. Control of *Fusarium* wilt of *Solanum melongena* by *Trichoderma* spp. Biol Plant. 2008;52:582–6. https://doi.org/10.1007/s10535-008-0116-2.
175. Bari MA. Biological control of soil-borne diseases of vegetables. Contract Research Project, Plant Pathology Division, Bangladesh Agricultural Research Institute, Joydebpur, Gazipur. 2001. p. 21–49.
176. Khan MA, Ishaq H, Ali S, Gogi D. Management of *Fusarium* wilt of eggplant in relation to soil and environmental factors [dissertation]. 2019.
177. Ramaiah AK, Garampalli RKH. *In vitro* antifungal activity of some plant extracts against *Fusarium oxysporum* f. sp. *lycopersici*. Asian J Plant Sci Res. 2015;5(1):22–7.
178. Kuri SK, Islam RM, Mondal U. Antifungal potentiality of some botanical extracts against important seedborne fungal pathogen associated with brinjal seeds (*Solanum melongena* L.). J Agric Technol. 2011;7(4):1139–53.
179. Aspires RB, Cruz AR. Potential biological control of bacterial wilt in tobacco and potato with *Bacillus plymyxa* and *Pseudomonas fluorescens*. In: Persley GJ, editor. Bacterial wilt disease in Asia and South Pacific. ACIAR Proc. 1985;(13):89–92.

180. Yucel S, Elekcioğlu YH, Can C, Soout MA, Ozarslandan A. Alternative techniques to control wilts in vegetables. Turk J Agric. 2007;31:47–53.
181. McPartland JM, McKernan KJ. Contaminants of concern in cannabis: microbes, heavy metals, and pesticides. In: *Cannabis sativa* L. Botany and Biotechnology. 2017. p. 457–74. https://doi.org/10.1007/978-3-319-54564-6_22.
182. Ciotola M, DiTommaso A, Watson AK. Chlamydospore production, inoculation methods and pathogenicity of *Fusarium oxysporum* M12–4A, a biocontrol for *Striga hermonthica*. Biocontrol Sci Technol. 2000;10(2):129–45. https://doi.org/10.1080/09583150029279.
183. Magnucka EG, Suzuki Y, Pietr SJ, Kozubek A, Zarnowski R. Action of benzimidazole fungicides on resorcinolic lipid metabolism in rye seedlings depends on thermal and light growth conditions. Pestic Biochem Phys. 2007;88(2):219–25. https://doi.org/10.1016/j.pestbp.2006.11.008.
184. Gamliel A. Soil and substrate health. In: Gullino ML, Albajes R, Nicot PC, editors. Integrated pest and disease management in greenhouse crops. Cham: Springer; 2020. p. 355–83. https://doi.org/10.1007/978-3-030-22304-5_12.
185. Katan J. Soil solarization. In: Chet I, editor. innovative approaches to plant disease control. New York: Wiley; 1987. p. 77–105. https://doi.org/10.1007/978-81-322-0723-8_11.
186. Koike ST, Gladders P, Paulus AO. Vegetable diseases: a colour handbook. 1st ed. CRC Press; 2006. p. 320. https://doi.org/10.1201/b15147.
187. Baker KF, Cook RJ. Biological control of plant pathogens. San Francisco: W.H. Freeman & Co.; 1974. p. xv–433.
188. Kumar AP, Ranjan JK, Tomar BS, Lata S, Singh J, Mishra GP, Gayacharan, et al. Phenotypic reaction of cultivated and wild brinjal genotypes to Fusarium wilt under sick plot and artificially inoculated pot conditions. J Phytopathol. 2025;173(1):e70018. https://doi.org/10.1111/jph.70018.
189. Gotame TP, Poudel S, Shrestha SL, Shrestha J. Evaluation of yield and yield components of eggplant (*Solanum melongena* L.) genotypes in the Terai region of Nepal. Int J Environ. 2020;9(2):67–80. https://doi.org/10.3126/ije.v9i2.32517.
190. Rivard CL, Louws FJ. Grafting to manage soilborne diseases in heirloom tomato production. HortScience. 2008;43(7):2104–11. https://doi.org/10.21273/HORTSCI.43.7.2104.
191. Lee JM, Kubota C, Tsao SJ, Bie Z, Echevarria PH, Morra L, Oda M. Current status of vegetable grafting: diffusion, grafting techniques, automation. Sci Hortic. 2010;127(2):93–105. https://doi.org/10.1016/j.scienta.2010.08.003.
192. Bánfalvi Z, Kondrák M, Bánfalvi G. CRISPR/Cas9-mediated mutation of *SmPG1*, a polygalacturonase gene in eggplant, improves resistance to fungal infection. Int J Mol Sci. 2022;23(14):7815. https://doi.org/10.3390/ijms23147815.
193. Maioli A, Gianoglio S, Moglia A, Acquadro A, Valentino D, Milani AM, Comino C, et al. Simultaneous CRISPR/Cas9 editing of three PPO genes reduces fruit flesh browning in *Solanum melongena* L. Front Plant Sci. 2020;11: 607161. https://doi.org/10.3389/fpls.2020.607161.
194. Kaur S, Dhaka N, Sharma M, Ahuja PS. CRISPR/Cas9 editing of *SmPPO* gene reduces enzymatic browning in eggplant (*Solanum melongena*). Front Plant Sci. 2023;14:1175203. https://doi.org/10.3389/fpls.2023.1175203.
195. Abdelaziz AM, Salem SS, Khalil AMA, El-Wakil DA, Fouda HM, Hashem AH. Potential of biosynthesized zinc oxide nanoparticles to control *Fusarium* wilt disease in eggplant (*Solanum melongena*) and promote plant growth. Biometals. 2022;35(3):601–16. https://doi.org/10.1007/s10534-022-00391-8.
196. Rao VG, Dhutraj DN, Navgire KD. Efficacy of Trichoderma-fortified organic amendments on Fusarium wilt suppression, growth and yield of eggplant. Asian J. Adv. Agric. Res. 2021:1–16.
197. Jamil A, Kumar M. Suppression of Fusarium wilt of eggplant using *Trichoderma harzianum* and Carbendazim. Int J Veg Sci. 2022;28(2):144–55. https://doi.org/10.1080/19315260.2021.1998478.
198. Miyatake K, Saito T, Negoro S, Yamaguchi H, Nunome T, Ohyama A, Fukuoka H. Detailed mapping of a resistance locus against Fusarium wilt in cultivated eggplant (*Solanum melongena*). Theor Appl Genet. 2016;129(2):357–67. https://doi.org/10.1007/s00122-015-2632-8.

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