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Comprehensive Review of Coriander Wilt: From Pathogen Characterization to Management Strategies

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Abstract

Coriander (*Coriandrum sativum* L.) is one of the important seed spice andfresh green leaves have a pleasant aromatic odour. It is widely used throughout the world. Fusarium wilt of coriander caused by *Fusarium oxysporum*f. sp.*coriandri*now became a major problem worldwide. When the funguscolonizes the xylem region, leading progressive chlorosisinitiatsfrom the basal leaves and reaches up topermanent wilting and also distinct by vascular browning. Enzymes *viz*., Polygalacturonase, β-1,4-glucanase, Xylanase, Protease, Phospholipase and Laccase are involved in disease development. Crop rotation with non-host crops, deep plowing and soil solarization with transparent polyethylene sheets (25-50 μm) during summer may disrupts the chlamydospores and the fungal mycelia. Biocontrol agents are effectively reduce the disease incidence.

Keywords: Coriander, wilt, *Fusarium,* symptoms, management

Introduction

Coriander (*Coriandrum sativum* L.), often referred to as Cilantro or Dhania, is a pivotal annual herb belonging to the Apiaceae family (Mahleyuddin*et al.*,2022)Addmissing in ref. It holds a unique position in global agriculture as a multi-purpose crop used extensively for its fresh foliage, dried seeds and essential oil. The plant's dual utility drives distinct breeding objectives: fresh-market growers prioritize "bolt tolerance" to extend the leaf-harvesting window in warm climates, whereas seed producers seek early-flowering genotypes to shorten the crop cycle and reduce resource inputs. Despite its adaptability as a summer and winter crop, the industry faces significant hurdles due to the short shelf-life of the greens and the increasing pressure of soil-borne pathogens in intensive coriander cultivation systems.

India remains the global leader in coriander production, with recent data from 2023–2024 indicating a cultivated area of approximately 7.10 lakh hectares and a total output exceeding 9.74 lakh tons

(Anonymous, 2024). The bulk of this production is concentrated in the "Coriander Belt," spanning Rajasthan, Madhya Pradesh, Gujarat and Andhra Pradesh. In these regions, coriander is often grown in the same fields year after year either in rainfed vertisols or irrigated inceptisolsa practice that has carelessly led to the crop that faces significant threats from soil-borne pathogens (Kaushik andNema, 2024).

Pests and diseases are the major constraints in the production of coriander. Coriander cultivation is affected by several diseases like wilt caused by *Fusarium oxysporum* (Narula and Joshi PS (1963), stem gall causedby *Protomyces macrospores*(Das, 1971), grain mould diseases caused by *Helminthosporium* spp.,*Fusarium* spp., *Curvularia* spp. and *Alternaria* spp. (Rajan *et al*., (1990), powdery mildew (*Erisyphepolygoni*),rust and leaf spots. Among these the wilt of Corianderhas recently emerged as the single most destructive threat to global coriander production (Netzer *et al.*, 2024).

Fusarium wilt is a primary limiting factor in coriander production, capable of causing up to 60–80 per cent yield loss globally under favorable environmental conditions (Sowmya *et al*., 2025; Chaimovitsh*et al*., 2024). In India, the economic impact varies by region: historical and recent surveys indicate that seed yield losses in Rajasthan range from 5 per cent to 60 per cent, while Gujarat experiences losses between 15 per cent and 25 per cent (Jat *et al*., 2017; Sowmya *et al*., 2025).In regions like Israel, the phenomenon has reached a crisis point in netted tunnels, leading to total field collapse (Netzer *et al*., 2024). The complexity of the disease is further intensified by the presence of secondary pathogens like *Rhizoctonia* spp. and *Pythium* spp. which contribute to damping-off and root rot, forming a lethal "complex" that attacks the plant from the seedling stage through to maturity(Koike & Gordon, 2015).

Fusarium wilt of coriander: Pathogen overview

Fusarium oxysporum is an exceptionally destructive plant pathogen, recognized for its ability to cause a diverse array of diseases including vascular wilts, head and seed blights, stem rots, root and crown rots, and cankers. In many instances, this fungus can simultaneously cause multiple or overlapping syndromes depending on the host and the surrounding environment. It possesses one of the most extensive host ranges among fungal phytopathogens, affecting a wide variety of economically vital agricultural and horticultural crops. Unlike other pathogens, *F. oxysporum* is divided into over 150 *formaespeciales* (f. sp.) based on host specificity, which are often further subdivided into races depending on their virulence toward specific host cultivars (Armstrong and Armstrong (1981)). This pathogen is a primary threat to global agriculture and horticulture, causing devastating vascular wilt in a wide range of crops (MacHardy *et al*., (1981)). Key examples of these specialized forms (*formaespeciales*) include:

Crop Type	Host Plant (Genus)	Specialized Pathogen (*Fusarium oxysporum*f. sp.)
Fruits & Vegetables	Banana (*Musa*)	f. sp.*cubense*
	Tomato (*Lycopersicon*)	f. sp.*lycopersici*
	Onion (*Allium*)	f. sp.*cepae*
	Watermelon (*Citrullus*)	f. sp. *niveum*
	Pea (*Pisum*)	f. sp. *pisi*
	Cabbage (*Brassica*)	f. sp. *conglutinans*
	Muskmelon (*Cucumis*)	f. sp. *melonis*
Industrial Crops	Cotton (*Gossypium*)	f. sp. *vasinfectum*
	Flax (*Linum*)	f. sp. *lini*
Ornamental Flowers	Tulip (*Tulipa*)	f. sp. *tulipae*
	Chrysanthemum	f. sp. *chrysanthemi*
	Carnation (*Dianthus*)	f. sp. *dianthi*
	Gladioli (*Gladiolus*)	f.sp. *gladioli*

Several reports on Fusarium wilt caused by *Fusarium oxysporum* in coriander have recently become more frequent: the disease was initially reported in India (Srivastava,1969). The pathogen identified in India was named *F. oxysporum* f. sp. *coriandrii* (Narula and Joshi, 1963, Ashwathi 2017) and other countries, like Argentina (Gaetán *et al.,* 1999), California (Koike and Gordon, 2005), and Egypt (Nada 2014). Sequencing of *F. oxysporum* f. sp. *coriandrii* was first deposited for an isolate from Italy (Gilardi *et al*. 2019). *Fusarium oxysporum* in coriander was also reported in two areas of China (Li *et al*., 2021; Yang *et al*. 2022), in Israel (Chaimovitsh, 2024) and recently *Fusarium pallidoroseum* (Cooke) Sacc also reported in Gujarat (Pithiya and Kanzaria, 2024).

F.oxysporum f.sp. *coriandrii* is a highly specialized and destructive soil-borne fungus responsible for coriander wilt disease and capable of transmission through contaminated seeds and persisting in the soil for over a decade (Sowmya *et al*., 2025; Chaimovitsh, *et al*., 2024). It affects the plant throughout its phenological development, with symptoms typically intensifying from the seedling stage through to flowering and seed setting (Jat *et al.,* 2021).

The infection process manifests initially as a subtle drooping of the apical leaves, which rapidly progresses into systemic chlorosis and severe vascular wilting. A diagnostic hallmark of *F.oxysporum* f.sp.*coriandri* is the distinct reddish-brown discoloration of the internal xylem vessels, which becomes clearly visible upon cross-sectioning the root or lower stems tissues (Sowmya *et al*.,2025). The incidence and severity of the disease are heavily influenced by environmental variables; warm soil temperatures between 25°C and 30°C provide optimal conditions for fungal proliferation. Furthermore, the pathology is often exacerbated by a sequence of high soil moisture followed by abrupt dry spells, a transition that intensifies the transpiration pull on the already compromised and obstructed vascular system (Dahikar and Nagarkar, 2024; Ali *et al*.,2024).

Biologically, the pathogen maintains a dual lifestyle, alternating between a saprophytic existence in the soil and an active pathogenic phase within the host tissue. This persistence is mediated by three specialized fungal propagules that ensure long-term survival and rapid colonization. Primarily, thick-walled chlamydospores act as hardy resting structures, capable of surviving at soil depths of up to 25 cm for 10–15 years, even in the absence of a viable host. For dispersal, the fungus produces multi-celled, sickle-shaped macroconidia that facilitate short-distance spread. Finally, within the host's vascular system, the pathogen abundantly produces microconidia, which are transported via the transpiration stream to ensure rapid systemic colonization of the entire plant (Sassenrath *et al.,* 2024).

The pathogenicity mechanism of *Fusarium oxysporum* f.sp*. coriandri* is a highly specialized, multi-stage process that transitions from a soil-borne saprophyte to a systemic vascular parasite. The cycle initiates in the rhizosphere, where dormant chlamydospores possess G-protein coupled receptors (GPCRs) detect specific coriander root exudates such as sugars and organic acids which triggering germ tube emergence and directed growth toward the host via chemotropism (Sowmya *et al*., 2025; Chaimovitsh *et al*., 2024). Unlike pathogens that rely solely on mechanical pressure, this fungus utilizes an arsenal of Cell Wall Degrading Enzymes (CWDEs)—including cellulases, xylanases, and pectinases to enzymatically macerate the host tissues, allowing the hyphae to penetrate the root epidermis primarily through the root cap, lateral root junctions, or existing wounds (Wahab *et al*., 2024).

Once the fungus reaches the root cortex, it enters a brief biotrophic phase, colonizing the intercellular spaces before invading the xylem vessels. Within the vascular system, the pathogen proliferates and produces abundant microconidia, which are transported upward by the plant's transpiration stream, facilitating rapid systemic colonization from the roots to the apical meristems (Narula and Joshi, 1963). To suppress the coriander plant's innate immune response, the fungus secretes specialized SIX (Secreted in Xylem) effector proteins that disarm host defense signaling (Michielse and Rep, 2009).

The characteristic wilting symptoms are the result of a dual physiological attack: physical obstruction and chemical toxicity. The accumulation of fungal mycelia and microconidia, combined with the host's defensive production of tyloses and pectic gels creates a physical blockage that severely restricts water transport (Sowmya *et al*., 2025). Concurrently, the pathogen secretes phytotoxins, most notably fusaric acid, which disrupts cellular membrane permeability and inhibits mitochondrial respiration (Wahab *et al*., 2024). This synergy between vascular clogging and toxin-mediated cell death results in the rapid chlorosis, stunting, and permanent wilting observed in susceptible coriander genotypes, eventually leading to plant death and the return of the fungus to the soil in the form of resting spores (Chaimovitsh *et al*., 2024; Jat *et al*., 2021).

Symptomatology

The clinical progression of coriander wilt begins with subtle physiological shifts that rapidly escalate into systemic failure. During early growth, the presence of *F. oxysporum*f. sp.*coriandri* often induces pre-emergence decay, leading to uneven or patchy germination. Seedlings that emerge successfully frequently exhibit epinasty (fig.1a), characterized by the downward curving of leaves, alongside significant stunting. This early phase is marked by a specific "chlorotic progression," where a unique veinal chlorosis initiates at the margins of the oldest leaves as a metabolic consequence of fungal colonization. Simultaneously, the subterranean tissues suffer from root hypoplasia, involving a marked reduction in secondary and tertiary root hairs. This structural degradation severely limits nutrient and water uptake even before macroscopic wilting becomes evident (Kumar *et al*., 2024).However, as *F.oxysporum* f. sp. *coriandrii* colonizes the xylem, this recovery ceases, leading to permanent wilting and progressive chlorosis starting from the basal leaves (Netzer *et al*.,2024; Guo and Chen, 2021). Internally, the disease is marked by distinct vascular browning; upon transecting the root or stem, the xylem exhibits a dark brown discoloration (fig. 1b). This is caused by the deposition of gums and tyloses which, while intended to wall off the fungus, paradoxically results in total vascular occlusion and the "sudden collapse" of the plant (Henry *et al*., 2020; Saccardi *et al*., 2022). Brown discoloration of vascular tissue was exhibited at crown region and white mycelium growth in vascular region (Fig.1c).

In later infections or partially tolerant varieties, the disease manifests as reproductive deterioration rather than immediate mortality. Infected plants leaves exhibited yellowing (fig.2a), and necrotic lesion at crown region and it extended to streak on stem (fig.2b) caused partial wilting symptoms (Fig.2c). later infected plants were dried (fig.2d). Infected plants often suffer from "umbel blasting," producing shriveled, non-viable seeds and exhibiting a significant reduction in Linalool synthesis,which compromises essential oil quality (Netzer *et al*., 2024).Symptoms in roots showed dark brownish discoloration (fig.2e). Browni discoloration of cortex and vascular tissue of roots (fig.2f). White to pinkish mycelium growth in vascular region of roots are obserbed (fig.2g) . White- pinkish mycelium growth and discoloration showed in vascular region of stem (fig.2h). This late-stage infection accounts for the widespread 15 to 25 per cent yield reductions reported in major hubs like Gujarat (Patel and Kumar, 2023).

Morphological characterization

Morphological characterization of *F. oxysporum*f. sp.*coriandri* reveals a rapid growth rate on Potato Dextrose Agar (PDA), typically reaching full plate colonization within seven days at 28° C. The cultural morphology is characterized by aerial, cottony mycelia that transition from an initial pure white to varying shades of pink, peach, or violet as the colony ages, a result of the biosynthesis of secondary metabolites like bikaverin (Ali *et al*., 2024). The reverse side of the culture often exhibits intense pigmentation, ranging from pale orange to deep magenta. Microscopic evaluation is critical for identification, focusing on the ontogeny of three spore types (Leslie and Summerell, B. 2006).

Microconidia: Microconidia are typically produced on short monophialides and are predominantly non-septate, elliptical, or reniform, measuring 5-15 × 2-4 µm (fig.3).

Macroconidia: Multi-septate (usually 3–5 septa), sickle-shaped with a distinct pedicellate foot cell, measuring 25.50-42.80 × 2-5µm (fig.3).

Chlamydospores: Thick-walled, globose resting structures formed terminally or intercalary in the mycelium, capable of surviving in the soil for 5–10 years even in the absence of a host (Leslie and Summerell, B. 2006).

Fig.1: Coriander plants showed symptoms due to wilt disease as
A- Early stage coriander plants leave showed yellow and later dried.
B- In early seedlings root showed brown to reddish vascular discoloration.
C- Brown discoloration of vascular tissue at crown region and white mycelium growth in vascular region

Fig.2: Coriander plants showed symptoms due to wilt disease as
A- Young plant leaves showed yellowing.
B- Necrotic lesion on stem.
C- Young plant showed partial wilting .
D-Plants dried
E- Young plant roots showed dark brownish discoloration. f- Brown discoloration of cortex and vascular tissue of roots.
G- White to pinkish mycelium growth in vascular region of roots .
H- White- pinkish mycelium growth and discoloration showed in vascular region.

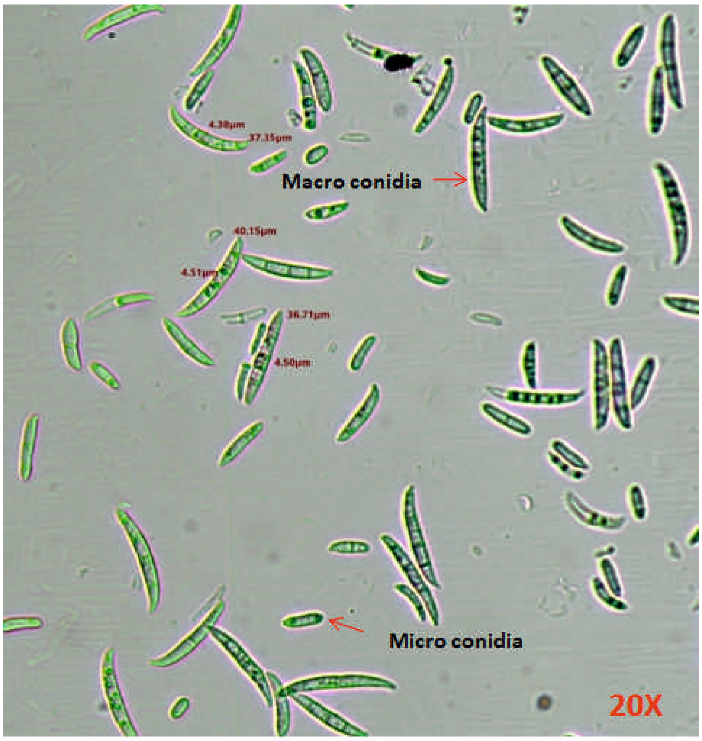

Fig.3: Conidia of *F.oxysporum* f.sp.*coriandrii* causing wilt disease in coriander

Integrated disease management

Cultural Management:

Cultural practices modify the cropping environment to be less conducive to the pathogen while improving host vigor.

Host Starvation through Crop Rotation (Basic):

The most fundamental cultural method is a 3–4 year rotation with non-host crops. *F. oxysporum* f. sp. *coriandrii* is highly host-specific, rotating coriander with cereals such as wheat (*Triticum aestivum*) or barley (*Hordeum vulgare*) or oilseeds like mustard (*Brassica juncea*) deprives the fungus of coriander-specific root exudates needed for germination. This leads to a natural decline in soil inoculum levels over time (Jat *et al.*, 2023; Meena *et al.*, 2023).

Disease Escape through Sowing Date Adjustment:

Epidemiological studies indicate that pathogenic fungus is most virulent at soil temperatures between 25 to 30° C. Advancing or delaying the sowing date to ensure that the sensitive seedling stage coincides with cooler soil temperatures (<20 ℃) can significantly reduce infection rates (Pathak *et al.,* 2024).

Rhizosphere pH and Nutrient Manipulation:

Recent research suggests that the form of nitrogen fertilizer significantly impacts wilt severity. Using Nitrate-nitrogen (NO_3^-) instead of Ammonium-nitrogen (NH_4^+) helps maintain a higher rhizosphere pH, which suppresses the germination of *Fusarium* spores. Additionally, the application of Potash has been shown to thicken cell walls, providing a mechanical barrier against fungal penetration (Kumar *et al.,* 2024; Patel *et al.*, 2025).

Physical Management:

Physical methods utilize heat and mechanical exposure to directly neutralize fungal propagules.

Deep Summer Plowing:

Exposing the lower soil layers to the intense heat and UV radiation of the summer sun (May–June) is a highly effective mechanical method. Deep plowing to a depth of 25-30 cm desiccates the chlamydospores and disrupts the fungal mycelial network within the soil profile (Meena *et al.*, 2023).

Soil Solarization:

This technique involves covering moist, tilled soil with transparent polyethylene sheets (25-50 μm) for 4-6 weeks during peak summer. This raises the soil temperature to 45-55° C which is lethal to *Fusarium* spores.

Aerated Steam and Thermotherapy:

For seed-borne inoculums, Hot Water Treatment 52°C for 15 minutes) can eradicate internal fungal infections without compromising seed viability (Singh *et al.*, 2025).

Biological Management:

Seed Bio-Priming:

Treating coriander seeds with *Trichoderma harzianum* or *Trichoderma viride* at a rate of 4 g/kg of seed is a proven basic method. Recent studies show that *T. harzianum* can inhibit pathogenic fungus growth by up to 83.69 per cent under *in vitro* condition (Patel *et al.*,2025) and reduce field wilt incidence by 61.75 per cent (Singh *et al.*,2025).

Rhizosphere Colonization with *Bacillus*:

Advanced biocontrol involves the use of *Bacillus subtilis* and *Bacillus toyonensis* which produce cellulolytic and proteolytic enzymes that degrade the fungal cell wall. Furthermore, they induce Systemic Acquired Resistance (SAR), priming the coriander plant's own immune system against future infection (Patel *et al.*, 2025).

Endophytic Protection :

The use of non-pathogenic *Fusarium oxysporum*entophytes is an emerging advanced technique. These "friendly" fungi occupy the same ecological niche as the pathogen within the plant's vascular system, effectively "blocking" the Foc from colonizing the xylem vessels (Sowmya *et al.*, 2025).

Future Prospects:

Fusarium wilt of coriander due to*Fusarium oxysporum*f. sp.*coriandrii*, focusing on its geographical distribution, host-pathogen interactions and vascular infection mechanisms. Furthermore, understanding of the pathogen's interaction with coriander genotypes is crucial for developing integrated disease management (IDM) strategies. These insights are essential for managing the spread of the fungus and minimizing economic losses, ultimately fostering the productivity and long-term sustainability of coriander cultivation globally.

Conclusion

Based on literature showed that Fusarium wilt of coriander due to *Fusarium oxysporum* f. sp. *coriandrii* now became a major problem in worldwide. When funguscolonizes the xylem region, leading progressive chlorosis starting from the basal leaves to permanent wilting and also distinct by vascular browning.Enzymes *viz.*, Polygalacturonase, β-1,4-glucanase, Xylanase, Protease, Phospholipase and Laccase are involved in disease development. Crop rotation with non-host crops, deep plowing and soil solarization with transparent polyethylene sheets (25-50 μm) during summer may disrupts the chlamydospores and the fungal mycelia. Biocontrol agents effectively reduce the disease incidence.

Conflict of Interest

The authors declare that they have no conflict of interest.

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