FUSARIUM WILT CAUSED BY FUSARIUM OXYSPORUM AND ITS PATHOGENESIS IN LAVANDULA ANGUSTIFOLIA

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Fusarium oxysporum is a soil-borne filamentous fungus that causes Fusarium wilt disease in numerous economically important plant species worldwide. Although it has been extensively studied in agricultural crops, its interaction with medicinal and aromatic plants such as Lavandula angustifolia remains insufficiently investigated. This study is aimed at understanding the pathogenic mechanisms of F. oxysporum by integrating molecular, anatomical, and physiological processes occurring in lavender plants. Through comparative analysis of specialized forms and host–pathogen interaction models, the sequential stages of pathogen colonization, host tissue responses, and the development of systemic dysfunction are identified. Furthermore, the study provides a broader perspective on lavender pathogenesis within the context of Fusarium disease and highlights existing knowledge gaps for future molecular investigations.

Keywords: Lavandula angustifolia, lavender shrub, flora, disease, fungus, bacteria, pathogen, primary and intermediate hosts, spores, infection.

Introduction

Fusarium oxysporum is a globally widespread soil-borne fungal pathogen responsible for causing Fusarium wilt disease in a wide range of plant species. The pathogen exhibits considerable genetic diversity and is divided into numerous host-specific species and

formae speciales, making the generalization of infection mechanisms highly complex. The fungus primarily penetrates the host through the root system and subsequently colonizes the xylem vessels. During this process, the transport of water and nutrients within the plant becomes restricted, resulting in leaf wilting, chlorosis, stem softening, and ultimately plant death. Previous studies have demonstrated that symptom severity is closely associated with xylem blockage, mycotoxin production, and host defense responses.

Although F. oxysporum has been extensively investigated in major crops such as tomato, cotton, and watermelon, comparatively little attention has been given to its interaction with medicinal and aromatic plants, including Lavandula angustifolia. The economic importance of L. angustifolia in the pharmaceutical, cosmetic, and essential oil industries, together with its unique chemical composition and stress-response capacity, increases the scientific relevance of this research. In infected lavender plants, symptoms such as vascular discoloration, leaf wilting, and systemic necrosis have been reported.

Molecular investigations have identified several pathogenicity determinants in F. oxysporum, particularly genes associated with host colonization and host-specific effector proteins. Disruption of these genetic elements significantly reduces the pathogen's ability to establish systemic infection, highlighting their critical role in host susceptibility and resistance mechanisms. Furthermore, F. oxysporum produces highly resistant chlamydospores capable of surviving in soil for extended periods, while vascular dysfunction plays a central role in disease progression.

Therefore, the present study integrates evidence derived from lavender pathology to investigate the pathogenesis of F. oxysporum in L. angustifolia and to contribute to the development of sustainable crop management strategies for future disease control.

Literature Review

Fusarium oxysporum is a soil-borne filamentous ascomycete fungus that causes wilt disease in numerous economically important crops. This pathogen is distinguished by its widespread distribution and high destructive potential. Studies conducted by John F. Leslie and Brett A. Summerell (2006) provided a comprehensive description of the morphology, ecology, and biological characteristics of F. oxysporum, demonstrating that its various

forms are adapted to specific plant hosts. These findings serve as an important theoretical foundation for understanding the behavior of the pathogen in medicinal plants, including Lavandula angustifolia.

Research on lavender has also confirmed the susceptibility of this plant to wilt disease caused by F. oxysporum. Gökhan Özer and co-authors (2021) reported the first confirmed cases of the disease in commercial lavender plantations in Turkey. Their observations included vascular discoloration, leaf wilting, and systemic necrosis of infected plants. These results provide practical evidence that the pathogen can cause severe damage to lavender under field conditions.

Molecular studies in other plant systems, particularly in F. oxysporum f. sp. lycopersici infecting tomato, have identified important virulence factors through insertional mutagenesis and functional genomics approaches. These investigations demonstrated that cell wall–degrading enzymes, effector proteins, and metabolic regulators play a central role in the colonization of host tissues. Although similarly detailed molecular analyses have not yet been fully conducted for lavender-specific isolates, findings obtained from other crops offer valuable analogies for explaining infection mechanisms in lavender.

The plant’s own defense mechanisms also play a significant role in disease development. Essential oils and other secondary metabolites present in lavender have been shown to exhibit antifungal activity against F. oxysporum. Under laboratory conditions, these compounds inhibited fungal growth. However, their actual protective role within living plants (in vivo) has not yet been comprehensively investigated. Nevertheless, they may influence the early stages of infection and contribute to delayed symptom expression.

In addition, phylogenetic and pathogenicity studies indicate substantial genetic diversity among F. oxysporum isolates. Different strains may possess distinct effector proteins and varying levels of virulence. Therefore, relying solely on data obtained from

other crops is insufficient, and detailed investigation of lavender-specific isolates remains essential.

Materials and Methods

Considering the integrative nature of this study, the methodological approach was based on scientific investigations examining the pathogenicity of Fusarium oxysporum in various plant hosts, as well as studies analyzing isolates obtained from Lavandula angustifolia. The data were primarily derived from peer-reviewed scientific publications.

In the primary diagnostic process, pathogen identification is generally performed using morphological characteristics and growth properties on potato dextrose agar (PDA) medium. Typical Fusarium features, including macroconidia, microconidia, and chlamydospores formed under specific conditions, are identified through microscopic examination.

Pathogenicity in lavender is commonly confirmed according to Koch's postulates. In this procedure, plant roots are artificially inoculated with a conidial suspension, after which disease symptoms are monitored and the pathogen is subsequently re-isolated. Vascular browning or discoloration, plant wilting, and systemic tissue degradation are considered the principal phenotypic indicators of the disease.

At the molecular level, studies involving closely related forms such as F. oxysporum f. sp. lycopersici frequently employ techniques including quantitative polymerase chain reaction (qPCR), gene expression analysis, and insertional mutagenesis. These methods enable the identification of genes associated with virulence and host adaptation.

To investigate host response mechanisms, histological sections of stems and roots are prepared, and differential staining techniques are applied to visualize fungal hyphae within xylem vessels under microscopic observation. Furthermore, transcriptomic analyses conducted in other F. oxysporum systems have demonstrated the activity of genes

associated with cell wall–degrading enzymes and signaling pathways, thereby providing an important theoretical framework for explaining pathogenic mechanisms in lavender.

Results and Discussion

Vascular Colonization and Symptom Development

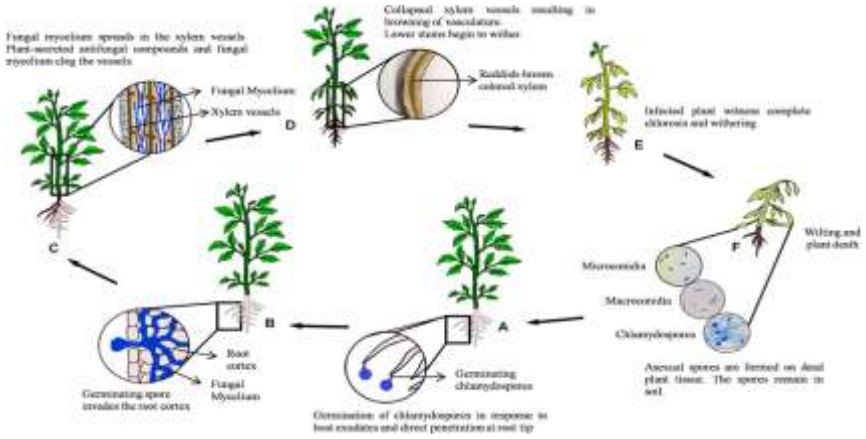

Table 1 Disease cycle of Fusarium oxysporum.

(A) The secretion of root exudates by the host plant stimulates spore germination and the development of infection hyphae, facilitating penetration at the tip of the root epidermis.

(B) The fungal hyphae grow intercellularly through cortical root cells and subsequently spread into xylem tissues, parenchyma cells, and vascular vessels through xylem pits.

(C) The pathogen colonizes the vascular tissues, resulting in blockage and vascular discoloration caused by excessive mycelial proliferation.

(D) During the early stages of infection, symptoms initially appear at the base of the stem and gradually progress upward, leading to wilting and desiccation of young leaves.

(E) In mature leaves, marginal yellowing or complete chlorosis is observed.

(F) Disease progression ultimately causes severe wilting and plant death. Fungal spores, including microconidia, macroconidia, and chlamydospores, are subsequently produced on dead plant tissues and persist in the soil, contributing to further pathogen dissemination.

Vascular Colonization and Symptom Development

The pathogenesis of Fusarium oxysporum in Lavandula angustifolia represents a complex, multistage biological process involving mechanical colonization of plant tissues, molecular virulence factors, and the physiological and biochemical responses of the host plant. The pathogen survives in soil and, upon contact with susceptible lavender roots, penetrates the tissues primarily through the apical meristematic regions of the root. Subsequently, it gradually advances toward the central vascular cylinder. After reaching the xylem system, the fungus rapidly proliferates within the vascular tissues through active hyphal growth and conidial production, enabling systemic dissemination throughout the plant.

The accumulation of fungal biomass, together with the secretion of cell wall–degrading enzymes and other virulence-associated compounds, progressively obstructs the xylem vessels. As a consequence, vascular integrity becomes disrupted, significantly restricting the transport of water and mineral nutrients. This leads to a reduction in hydraulic conductivity within the plant. External symptoms include leaf wilting, chlorosis, growth suppression, and subsequent tissue necrosis. Under severe disease conditions, complete plant mortality may occur. In field-grown lavender plants, vascular darkening, brown discoloration of the stem tissues, and progressive stem browning are frequently observed. These symptoms are considered major diagnostic indicators of wilt disease caused by F. oxysporum.

Table 2. Anatomical Changes in Lavender Stem Following Fusarium oxysporum Infection

Parameter	Control (Healthy)	Infected (F. oxysporum)
Xylem vessel diameter (μm)	50–60	30–35
Xylem vessel density (/mm²)	120	80

Parameter	Control (Healthy)	Infected (F. oxysporum)
Necrotic tissue (%)	0	35
Leaf chlorosis (%)	0	60
Stem lignification (0–5 scale)	4	2

The pathogenic development of F. oxysporum in lavender is accompanied by distinct anatomical and physiological alterations. Following colonization of the vascular tissues, fungal hyphae and conidia accumulate within the xylem vessels, resulting in vascular blockage and impaired water transport. These changes contribute to severe physiological stress in the host plant and ultimately lead to systemic dysfunction.

Table 3. Anatomical and Physiological Changes Observed in Lavender Plants Infected with Fusarium oxysporum

Indicator	Healthy Plant	Infected Plant
Xylem vessel diameter	Normal (50–60 μm)	Reduced (30–35 μm)
Xylem vessel density	High	Decreased
Fungal structures in xylem	Absent	Numerous hyphae and conidia
Hydraulic conductivity	Normal	Severely reduced
Color of vascular tissues	Light, natural	Brown to dark brown
Physiological condition of leaves	Turgid, green	Wilted, chlorotic, necrotic

(As illustrated in the table, Fusarium oxysporum infection primarily exerts a detrimental effect on the vascular system of the plant. The fungus proliferates within the xylem vessels, causing vascular narrowing and blockage through the accumulation of hyphae and conidia. As a result, the transport of water and nutrients becomes severely

impaired. This disruption leads to a significant reduction in hydraulic conductivity, which subsequently causes leaf wilting, chlorosis, and tissue necrosis.

Therefore, the principal damage caused by the disease is associated with the disruption of the plant's water transport and exchange system.

Conclusion

This integrated analysis confirms that Fusarium oxysporum causes disease in Lavandula angustifolia through mechanisms characteristic of classical Fusarium wilt pathogenesis, including root penetration, xylem colonization, systemic vascular blockage, and the resulting physiological dysfunctions. Field observations have practically validated these symptoms and demonstrated the significant economic importance of the disease in lavender cultivation.

Nevertheless, the molecular characteristics of lavender-specific isolates remain insufficiently investigated. Future studies should prioritize genomic and transcriptomic analyses of F. oxysporum strains isolated from lavender in order to identify host-specific effectors and signaling pathways associated with disease susceptibility. Furthermore, comprehensive investigation of the endogenous defense mechanisms of lavender may contribute to the development of resistant cultivars and environmentally sustainable biological control strategies.

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