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Characterization and Epidemiology of *Fusarium* Wilt in Tomato under Subtropical Conditions

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Abstract

Tomato (Solanum lycopersicum L.) is one of the most important vegetable crops cultivated globally and plays a significant role in food security, nutrition, and income generation, particularly in subtropical regions. However, tomato production is severely constrained by Fusarium wilt, a destructive vascular disease caused by Fusarium oxysporum f. sp. lycopersici (FOL). The disease is particularly aggressive under subtropical conditions characterized by warm temperatures, moderate to high humidity, and intensive cultivation practices. Recent advances in molecular biology have improved understanding of pathogen diversity, race structure, virulence mechanisms, and host–pathogen interactions. This review provides a comprehensive overview of the molecular characterization and epidemiology of Fusarium wilt of tomato under subtropical conditions, with emphasis on genetic variability, pathogenicity determinants, environmental influences, and integrated management strategies.

Keywords: *Tomato, microconidia, macroconidia, Fusarium wilt, Chlamydospores.*

Introduction

Tomato (*Solanum lycopersicum*) is widely cultivated in tropical and subtropical agro-ecological zones due to its adaptability and high market demand. Despite its economic importance, tomato production is threatened by several diseases, among which Fusarium wilt is one of the most devastating. The disease is caused by the soil-borne fungal pathogen *Fusarium oxysporum* f. sp. *lycopersici*, which infects plants through the roots and colonizes the vascular tissues, leading to wilting, yellowing of leaves, vascular browning, and eventual plant death. Yield losses may range from moderate to severe depending on environmental conditions, cultivar susceptibility, and pathogen race [1]. Subtropical climates provide ideal conditions for pathogen growth and disease development, making the study of molecular characterization and epidemiology particularly relevant for sustainable management.

Taxonomy and Biological Characteristics of the Pathogen

Fusarium oxysporum f. sp. *Lycopersici* belongs to the phylum Ascomycota and is part of the *Fusarium oxysporum* species complex, which includes numerous formae speciales distinguished by host specificity. Morphologically, the fungus produces, and thick-walled chlamydospores. Microconidia are single-celled and oval-shaped, while macroconidia are multicellular and sickle-shaped. Chlamydospores serve as survival structures that enable the pathogen to persist in soil for many years, even in the absence of host plants [2]. This long-term survival capacity makes *Fusarium wilt* particularly difficult to eradicate in subtropical cropping systems where continuous tomato cultivation is common.

Molecular Characterization of the Pathogen

Molecular characterization has significantly advanced the understanding of genetic diversity and race differentiation within *Fusarium oxysporum* f. sp. *lycopersici*. The pathogen is classified into three physiological races—Race 1, Race 2, and Race 3—based on their ability to overcome specific resistance genes in tomato. Race identification is commonly performed using pathogenicity assays on differential cultivars and molecular markers targeting avirulence genes. Techniques such as random amplified polymorphic DNA (RAPD), amplified fragment length polymorphism (AFLP), simple sequence repeats (SSR), internal transcribed spacer (ITS) sequencing, and elongation factor-1 alpha (EF-1α) gene sequencing have revealed considerable genetic variability among isolates. Subtropical regions often exhibit greater pathogen diversity due to climatic variability, diverse

cropping patterns, and long-term pathogen persistence in soil. Whole-genome sequencing studies have further clarified the genetic architecture of FOL. The genome consists of core chromosomes containing essential housekeeping genes and accessory or lineage-specific chromosomes that harbor pathogenicity-related genes [3]. These accessory chromosomes can be horizontally transferred between strains, contributing to the rapid evolution of new virulent races. The presence of transposable elements and mobile genetic elements enhances genomic plasticity, allowing the pathogen to adapt efficiently to environmental stresses common in subtropical climates.

Virulence Factors and Pathogenicity Mechanisms

The pathogenicity of FOL is largely determined by secreted effector proteins known as SIX (Secreted In Xylem) proteins. These small proteins are expressed during infection and play critical roles in host colonization and suppression of plant immune responses. Certain SIX genes correspond to specific avirulence functions and interact with tomato resistance genes. Mutations or deletions in these genes can enable the pathogen to overcome host resistance. In addition to effectors, the fungus produces cell wall-degrading enzymes such as pectinases, cellulases, and xylanases, which facilitate root penetration and vascular invasion. The production of mycotoxins, particularly fusaric acid, contributes to symptom development by interfering with plant physiological processes, including water transport and ion balance [4]. Together, these virulence factors enable the pathogen to colonize xylem vessels, block water movement, and induce characteristic wilting symptoms. The expression of virulence genes is often influenced by environmental conditions, and high temperatures typical of subtropical regions can enhance pathogenic activity.

Host–Pathogen Interaction

Resistance to Fusarium wilt in tomato is governed by specific resistance (R) genes, notably I, I-2, and I-3, which correspond to resistance against Races 1, 2, and 3, respectively. These genes encode proteins that recognize pathogen avirulence factors and activate defense responses, including the hypersensitive response. Upon recognition, the plant initiates rapid localized cell death to restrict pathogen spread. Additional defense mechanisms include the production of reactive oxygen species, synthesis of pathogenesis-related proteins, activation of salicylic acid signaling pathways, and reinforcement of cell walls through lignification, environmental stresses such as high temperature and irregular moisture, common in subtropical climates, can compromise host resistance [5]. Heat stress may weaken plant immune responses, allowing even moderately virulent isolates to cause severe disease. This interaction between environmental factors and host resistance plays a critical role in disease epidemiology.

Table 1: Molecular and Epidemiological Features of Fusarium oxysporum f. sp. lycopersici in Subtropical Regions

Parameter	Description	Relevance under Subtropical Conditions
Pathogen	*Fusarium oxysporum* f. sp. *lycopersici*	Soil-borne fungus causing vascular wilt in tomato
Survival Structure	Chlamydospores	Long-term survival in warm soils for many years
Optimal Temperature	25–30°C	Favors rapid growth and disease development
Physiological Races	Race 1, Race 2, Race 3	Overcome specific tomato resistance genes (I, I-2, I-3)
Major Virulence Factors	SIX proteins, CWDEs, fusaric acid	Promote xylem colonization and wilting symptoms
Mode of Spread	Contaminated soil, water, seedlings, tools	Enhanced by intensive cultivation practices
Management Strategies	Resistant cultivars, crop rotation, biocontrol, soil solarization	Essential for sustainable control in subtropical climates

Epidemiology under Subtropical Conditions

The epidemiology of Fusarium wilt is strongly influenced by climatic and soil factors prevalent in subtropical regions. The optimal temperature for pathogen growth ranges between 25°C and 30°C, which coincides with typical subtropical growing conditions. Warm temperatures accelerate spore germination, root infection, and vascular colonization. Soil moisture also influences disease development; moderate moisture levels favor fungal growth, while waterlogging can predispose plants to infection by causing root injury. Soil pH between 5.5 and 7.0 is generally conducive to disease development. The pathogen survives in soil primarily as chlamydospores, which can remain viable for extended periods. Root exudates from tomato plants stimulate chlamydospore germination, initiating infection. Disease spread occurs through contaminated soil, irrigation water, infected seedlings, and farm equipment. Continuous monocropping, a common practice in subtropical vegetable production systems, leads to inoculum buildup and increased disease incidence over time.

The disease cycle begins with survival of the pathogen in soil, followed by germination in response to host signals. The fungus penetrates roots, colonizes xylem vessels, produces toxins and enzymes, and causes systemic wilting. Infected plant debris returns the pathogen to the soil, completing the cycle [6]. Under favorable subtropical conditions, this cycle can progress rapidly, resulting in significant yield losses.

Molecular Epidemiology and Population Structure

Molecular epidemiology utilizes phylogenetic analyses, multilocus sequence typing, microsatellite markers, and single nucleotide polymorphism (SNP) genotyping to study population structure and genetic relationships among FOL isolates [3]. These approaches help identify sources of outbreaks, track the emergence of new races, and understand evolutionary dynamics. In subtropical regions, where environmental conditions favor rapid pathogen multiplication, monitoring genetic diversity is essential for early detection of virulent strains capable of overcoming existing resistance genes.

Integrated Disease Management

Effective management of Fusarium wilt under subtropical conditions requires an integrated approach. The use of resistant cultivars remains the most economical and environmentally sustainable strategy. However, the emergence of new pathogen races necessitates continuous breeding programs incorporating multiple resistance genes. Cultural practices such as crop rotation with non-host crops, soil solarization during hot seasons, use of certified disease-free seedlings, and improved drainage through raised beds can significantly reduce disease pressure. Biological control agents, including species of *Trichoderma* and *Bacillus*, as well as non-pathogenic strains of *Fusarium*, have shown promise in suppressing pathogen populations through mechanisms such as competition, antibiosis, and induction of systemic resistance. Chemical control is generally less effective due to the soil-borne nature of the pathogen and environmental concerns associated with fungicide use [2]. Therefore, emphasis is placed on preventive and sustainable management practices.

Future Perspectives and Conclusion

Advances in genomics, transcriptomics, and molecular diagnostics have greatly enhanced understanding of *Fusarium oxysporum* f. sp. *lycopersici*. Emerging technologies such as CRISPR-based genome editing, RNA interference strategies, and metagenomic analysis of soil microbiomes offer new opportunities for durable disease management. Predictive epidemiological models integrating climate data may further assist in forecasting disease outbreaks under changing climatic conditions. Fusarium wilt of tomato remains a major threat to tomato production in subtropical regions. Molecular characterization has revealed significant genetic diversity and complex pathogenic mechanisms that enable adaptation to warm climates. Epidemiological studies underscore the influence of environmental conditions, cropping practices, and host resistance on disease dynamics. Sustainable management requires integration of resistant cultivars, cultural practices, biological control, and advanced molecular monitoring to mitigate the impact of this persistent soil-borne pathogen.

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