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Potential Interactions Between Huanglongbing and *Lasiodiplodia*-Associated Branch Dieback in Citrus Orchards: A Review of Co-occurrence and Host Responses

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Abstract

Huanglongbing (HLB), also known as citrus greening, is currently one of the most destructive diseases threatening citrus production worldwide, having been reported globally. The disease is associated with the phloem-limited, vector-transmitted bacterium *Candidatus* Liberibacter asiaticus, and its management is strongly dependent on early and reliable diagnosis. However, symptom development is often slow and nonspecific, frequently overlapping with nutritional disorders and abiotic stress. Recent evidence indicates that pathogen detection is more consistent and can be achieved earlier in root tissues than in leaves, emphasizing the diagnostic value of root sampling for early-stage HLB surveillance. Given the economic relevance of citrus as a global commodity, the capacity to identify infections at incipient stages is essential to reduce disease spread and mitigate severe yield and quality losses. In parallel, citrus branch dieback associated with *Lasiodiplodia* spp. represents another major constraint in citrus orchards. This disease involves complex pathogen-host interactions that disrupt vascular function, impair phloem transport, and induce phloem necrosis, ultimately leading to branch decline and tree weakening. Several *Lasiodiplodia* species have been linked to citrus dieback, and field assessments suggest that certain citrus relatives may exhibit partial resistance. Understanding the pathogen biology, host responses, and disease mechanisms at the plant–microbe interface is critical for the development of effective control strategies. Furthermore, recent advances in citrus and fungal genomics have improved our understanding of citrus evolution, pathogen diversity, and potential targets for disease management. This review highlights current knowledge on HLB and *Lasiodiplodia*-associated dieback, emphasizing diagnostic challenges, vascular dysfunction, and emerging molecular tools that may support integrated management approaches for citrus decline syndromes.

Keywords: HLB; Botryosphaeriaceae; Citrus Decline Syndrome; Co-Infection; Vascular Dysfunction

Introduction

Huanglongbing (HLB), also known as citrus greening, is one of the most destructive diseases affecting citrus production worldwide. The disease has been reported in more than 50 countries across Africa, Asia, Oceania, and the Americas, including the Caribbean, and continues to expand its geographic range [4, 12]. Since its first detection in Florida in 2005, HLB has rapidly spread to most citrus-producing regions of the United States, severely compromising orchard productivity and profitability [12, 30]. HLB is primarily associated with the phloem-limited, vector-transmitted bacterium *Candidatus* Liberibacter asiaticus (CLas), which is disseminated mainly by the Asian citrus psyllid (*Diaphorina citri*) [4, 12]. Effective disease management depends strongly on early and accurate diagnosis; however, symptom expression is often slow, variable, and frequently confounded with nutritional deficiencies and abiotic stress, complicating visual assessments in the field (Figure 1A) [12, 30] . Notably, recent evidence indicates that CLas detection may be more consistent and achieved earlier in root tissues than in leaves, highlighting the diagnostic relevance of root sampling for early-stage HLB surveillance and improved disease monitoring [16, 27]. Given the global economic importance of citrus as a high-value commodity crop, early pathogen detection is essential to limit disease spread and mitigate severe yield and fruit quality losses [12, 15].

In parallel with HLB, citrus orchards are increasingly challenged by branch dieback caused by

Lasiodiplodia species, which contribute to wood necrosis, canker development, gummosis, and progressive decline of the canopy [26, 29]. The genus *Lasiodiplodia* comprises nearly 50 recognized species and is considered one of the most significant pathogen groups within the Botryosphaeriaceae, with a broad host range and cosmopolitan distribution [7, 33]. Several *Lasiodiplodia* species have been documented in association with citrus dieback in diverse geographic regions, suggesting that this disease complex may involve multiple species with differing ecological and pathogenic profiles [7, 26]. Symptom expression often involves systemic necrosis of lignified tissues, branch mortality, and mummified fruit, which collectively reduce tree vigor and yield potential (Figure 1B) [26] .

Despite being taxonomically and mechanistically distinct, HLB and *Lasiodiplodia*-associated dieback share key features related to vascular dysfunction and the progressive loss of physiological integrity in affected trees. Both diseases disrupt phloem transport and compromise the capacity of citrus trees to maintain carbon allocation, water use efficiency, and defense responses [12, 29]. This convergence in symptomatology, particularly under field conditions where mixed infections may occur, highlights the need to investigate potential interactions between these pathogens and their cumulative effects on tree health. A deeper understanding of the pathogen-host interface for both HLB and *Lasiodiplodia* species is essential for developing integrated management strategies that account for complex disease scenarios in modern orchards. Furthermore, advances in citrus and fungal genomics, along with multi-omics approaches, are providing new insights into citrus evolution, pathogen diversity, and candidate molecular targets for improved diagnostics and control [11, 21, 31].

Although HLB and *Lasiodiplodia*-associated branch dieback have been widely studied as independent pathosystems, their potential interaction under field conditions remains poorly understood. In citrus orchards, trees frequently experience simultaneous biotic and abiotic stresses, and chronic HLB infection may predispose hosts to opportunistic vascular pathogens by altering carbon partitioning, weakening defense responses, and impairing vascular integrity. Conversely, wood-colonizing fungi such as *Lasiodiplodia* spp. may exacerbate canopy decline by accelerating xylem dysfunction and intensifying physiological stress in already HLB-compromised trees. Despite increasing reports of citrus decline syndromes involving multiple pathogens, there is still limited integrative information regarding the combined effects of CLas infection and Botryosphaeriaceae-associated dieback on citrus physiology, disease progression, and orchard productivity. Therefore, this review synthesizes current knowledge on HLB and *Lasiodiplodia* branch dieback, highlighting potential converging mechanisms related to vascular dysfunction, diagnostic challenges, and host susceptibility, while discussing emerging molecular tools and research directions to support integrated management strategies for complex citrus decline scenarios [12, 29, 30].

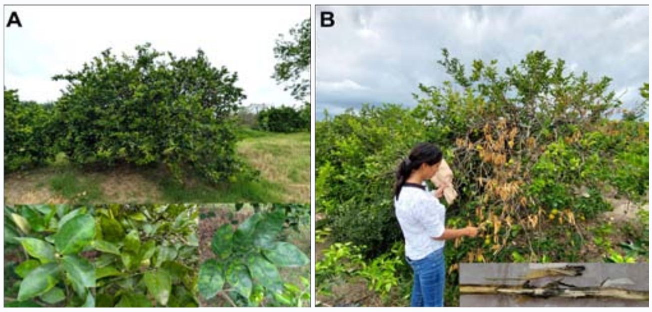

Figure 1: (A) Symptoms of Huanglongbing (HLB) in Persian lime (*Citrus latifolia*), lower panel shows a close-up of symptomatic leaves on branches. (B) Dieback disease symptoms in Persian lime (*C. latifolia*), lower right panel displays internal tissue necrosis.

Taxonomy and Biology

Lasiodiplodia-associated citrus dieback, historically referred to in some regions as citrus foot rot or black foot, is a fungal disease affecting citrus trees and is frequently associated with the ascomycete *Lasiodiplodia theobromae*. The disease typically causes twig and branch dieback and may be accompanied by bark cracking, creasing, and longitudinal lesions near the basal trunk. These symptoms have sometimes contributed to confusion with other basal trunk disorders, including Phytophthora foot rot, which is caused mainly by *Phytophthora nicotianae* and *P. palmivora* and primarily affects the root collar region [13, 23]. *Lasiodiplodia theobromae* is a cosmopolitan species widely recognized as an important agent associated with grafting wounds, stem cankers, wood discoloration, and fruit rot in citrus. It is often considered an opportunistic pathogen, frequently colonizing stressed or weakened host tissues and behaving as an endophyte or latent pathogen under favorable conditions [29, 33].

Description and Geographic Distribution of *Lasiodiplodia* spp.

Phylogenetic studies place *Lasiodiplodia* within the family Botryosphaeriaceae (order Botryosphaeriales), and multilocus analyses support the recognition of numerous cryptic species previously grouped under the *L. theobromae* species complex [24, 33]. The global relevance of *Lasiodiplodia* spp. in citrus decline has been increasingly documented through regional surveys. In North Africa, *L. mediterranea* and *L. mitidjana* have been associated with shoot mortality and wood necrosis in citrus orchards in Algeria [3]. In the Middle East, citrus cankers and gummosis have been linked to species such as *L. iraniensis*, *L. hormozganensis*, and *L. theobromae* in Iran and Oman [1, 2, 7]. In Egypt, *L. laeliocattleyae* and other *Lasiodiplodia* species have been reported as pathogenic to *Citrus reticulata* and other citrus hosts [6]. In South America, *L. theobromae* has been frequently reported in association with citrus branch dieback and fruit rot in Brazil [5, 10]. In the United States, recent studies have identified *L. iraniensis* and *L. parva* from symptomatic citrus tissues, further supporting the expanding distribution of this genus in commercial citrus systems [22]. In Mexico, several species have been reported infecting *C. latifolia* and C. sinensis, including *L. iraniensis*, *L. lignicola, L. mexicanensis, L. pseudotheobromae, L. subglobosa* and *L. theobromae* [25, 26]. Notably, extensive surveys in China identified nine distinct *Lasiodiplodia* species associated with trunk and branch disorders across seven citrus hosts, all of which exhibited pathogenicity in inoculation assays, underscoring the diversity and potential epidemiological importance of this genus in citrus orchards [32].

Economic and Agricultural Impact of HLB

Citrus Huanglongbing is among the most devastating diseases affecting citrus production, causing substantial economic losses through reduced yield, impaired fruit quality, increased production costs, and shortened tree lifespan [12, 14]. In heavily affected regions, even orchards with moderate disease incidence may experience severe economic constraints due to declining productivity and the increasing costs associated with vector management, tree replacement, and intensified cultural practices [14, 28]. Since its first detection in Florida in 2005, HLB has spread throughout most citrus-producing areas of the United States and has contributed to a dramatic reduction in citrus acreage and processing capacity, with broad socioeconomic consequences for growers and associated industries [14, 28]. Recent estimates suggest that the cumulative economic impact of HLB in the U.S. citrus industry exceeds several billion dollars, including major losses in labor income and employment opportunities [14, 28].

The rapid expansion of HLB, including its dissemination through infected propagation material and nursery stock, highlights the urgent need for reliable detection methods in both commercial orchards and certified plant production systems [12, 30]. Surveillance strategies therefore emphasize early identification of infected plants, since symptom development is often delayed and asymptomatic infections may persist for extended periods, allowing unnoticed pathogen spread [12, 15]. Accurate pathogen detection is also essential prior to planting to prevent the establishment of infected orchards, as secondary spread by psyllid vectors can rapidly amplify disease incidence once inoculum sources are present [12, 30]. Molecular diagnostics based on quantitative PCR (qPCR) remain the most widely used tools for detecting *Candidatus* Liberibacter asiaticus in citrus tissues [18]. Importantly, recent studies indicate that root tissues may provide earlier and more consistent detection compared to leaf tissues, reinforcing the value of root sampling for early-stage surveillance and improved diagnostic reliability [16, 34]. Root-based detection is particularly relevant because CLas colonization and decline in the root system may precede the development of visible canopy symptoms, allowing earlier identification of infected trees before major aboveground damage occurs [16, 34].

Beyond its direct economic impact, HLB also increases the vulnerability of citrus trees to secondary biotic constraints that contribute to decline syndromes in commercial orchards. Chronic CLas infection disrupts carbohydrate allocation, weakens root systems, and alters vascular function, potentially predisposing trees to opportunistic wood-colonizing pathogens such as *Lasiodiplodia* spp., which frequently infect stressed tissues through wounds or environmental damage. Under field conditions, this overlap may intensify canopy dieback, accelerate tree decline, and further reduce orchard longevity, thereby amplifying economic losses beyond those attributed solely to HLB. Consequently, understanding how HLB may interact with branch dieback pathogens is essential to develop integrated management strategies capable of addressing complex citrus decline scenarios in modern production systems [12, 29, 30].

Co-Occurrence in Citrus Orchards

The co-occurrence of multiple plant diseases within the same host populations and geographic regions is common and may involve complex ecological, epidemiological, and physiological interactions that influence disease development and severity [15, 17]. In citrus orchards, trees affected by HLB are frequently observed to exhibit branch dieback symptoms resembling those associated with *Lasiodiplodia* spp., particularly in Mexico [26] and in several citrus-producing regions of the United States [15]. Recent advances in the taxonomy and phylogenetics of *Lasiodiplodia* have improved the accuracy of pathogen identification, enabling more systematic documentation of species diversity, host range, and geographic distribution. Nevertheless, comprehensive studies evaluating spatial overlap, temporal patterns, and potential disease interactions between HLB and *Lasiodiplodia*-associated dieback within the same orchards remain scarce. This lack of integrative epidemiological evidence represents a key knowledge gap, particularly given the increasing frequency of citrus decline syndromes involving multiple stressors.

HLB is associated with phloem-limited bacteria of the genus CLas and is primarily transmitted by *D. citri*, with long-distance spread also occurring through infected nursery stock and vegetative propagation material [4, 12]. In contrast, dieback caused by *Lasiodiplodia* spp. is not known to involve insect-mediated transmission and is typically associated with infection through wounds, pruning injuries, or natural openings, often under conditions of host stress [23, 29]. Although both diseases are frequently observed within the same orchards and in some cases within the same trees current evidence does not demonstrate direct epidemiological dependence between CLas and *Lasiodiplodia* spp. Therefore, it remains unclear whether their co-occurrence reflects independent disease processes driven by shared environmental conditions, or whether HLB-induced physiological alterations predispose trees to colonization by opportunistic wood pathogens. Distinguishing between these scenarios is essential because each has different implications for disease diagnosis, epidemiological interpretation, and the design of integrated management strategies for citrus decline.

Geographic Distribution and Overlap

Co-occurrence patterns between *Lasiodiplodia* spp. and HLB in citrus orchards remain poorly characterized. Nevertheless, available reports suggest a substantial geographic overlap between both pathosystems, particularly in regions where HLB-positive orchards coincide with high population densities of the Asian citrus psyllid. In Mexico, field observations and disease timelines indicate that HLB was detected before widespread recognition and formal reporting of *Lasiodiplodia*-associated branch dieback, suggesting that HLB may have become established earlier in several production areas [26, 31]. Similarly, in the United States, reports of branch dieback and citrus decline have been increasingly documented in areas with high HLB incidence, although systematic pathogen identification and species-level characterization of Botryosphaeriaceae in citrus have only expanded more recently [12, 15]. Collectively, these observations provide preliminary support for the hypothesis that CLas became established before *Lasiodiplodia* spp. were formally recognized as major contributors to decline in certain citrus systems; however, both pathogens may also have been introduced independently and subsequently converged in the same orchards.

Given this apparent overlap, synthesizing the geographic distributions of HLB and *Lasiodiplodia* spp. is essential to clarify the epidemiological context and potential constraints associated with co-infection. HLB, associated mainly with the phloem-limited bacterium CLas, is widely regarded as one of the most destructive diseases affecting citrus worldwide and continues to expand into new citrus-growing regions, generating severe economic losses [4, 14]. The insect vector D. citri has been recorded across a broad range of citrus-producing regions, including areas where HLB has not yet been officially detected. This widespread distribution of the vector increases the risk of further HLB expansion and may contribute indirectly to the frequent spatial association between HLB and decline syndromes involving opportunistic pathogens such as *Lasiodiplodia* spp. [12, 29].

Mapping the reported distribution of *Lasiodiplodia* spp. together with HLB provides a useful framework for evaluating their co-occurrence in citrus production systems. Early descriptions of *Lasiodiplodia* (historically referred to as *Botryodiplodia* spp.) documented its broad host range, including citrus species, long before the global expansion of HLB was recognized [29]. In the United States, citrus decline and dieback symptoms consistent with Botryosphaeriaceae-associated disorders were reported decades before the official detection of HLB in 2005, suggesting that wood-inhabiting pathogens may have been present as latent or secondary agents prior to the emergence of CLas as a dominant limiting factor [12, 23]. However, the recent expansion of multilocus phylogenetics and systematic disease surveys has revealed that citrus dieback may involve multiple *Lasiodiplodia* species with distinct ecological and pathogenic profiles, complicating comparisons across historical and contemporary reports [24, 33].

Field reports and survey data indicate that dieback symptoms are widespread across multiple citrus varieties, although disease incidence and severity vary among genotypes, orchard management systems, and environmental conditions. The frequent association of *Lasiodiplodia* spp. with stressed trees, combined with its reported co-occurrence with other pathogens, highlights the need for integrative epidemiological assessments. Such approaches are essential to clarify the contribution of *Lasiodiplodia* spp. to citrus decline syndromes and to determine whether its association with HLB reflects direct biological interactions, shared predisposing factors, or independent infections occurring within the same orchards [17, 29].

Seasonal Dynamics and Environmental Drivers

Approaches used to characterize the seasonal dynamics of *Lasiodiplodia*-associated dieback and HLB in citrus orchards have been recently applied in different production systems, including studies conducted across multiple agroecological zones in Pakistan [15]. In these assessments, seasonal variation was identified as a major factor influencing disease incidence, whereas the relative contribution of individual environmental variables appeared less consistent across locations. Based on meteorological analyses, temperature and precipitation are frequently considered key climatic parameters for evaluating potential shared environmental drivers that may influence the development of both pathosystems.

Available evidence suggests that both *Lasiodiplodia*-associated dieback and HLB can be favored within comparable temperature ranges, with increased disease expression commonly reported under mild-to-warm conditions (approximately 25-35°C), which are also conducive to citrus growth and vector activity [12, [29]. However, the two diseases may exhibit contrasting associations with precipitation and water availability. HLB symptom development and disease progression are often linked to seasonal flush cycles and periods of higher psyllid activity, which frequently coincide with moderate rainfall and favorable humidity conditions [12, 30]. In contrast, *Lasiodiplodia* dieback is frequently reported to intensify under drought stress, high evaporative demand, or prolonged dry periods, consistent with the opportunistic behavior of Botryosphaeriaceae as latent pathogens that become aggressive when host physiological integrity is compromised [29,32]. These contrasting seasonal patterns suggest that *Lasiodiplodia*-associated decline may be strongly influenced by climatic stress conditions, and that its apparent association with HLB in the field could reflect shared environmental drivers or stress-mediated host predisposition rather than a direct pathogen-pathogen interaction.

Commonalities and Distinctions in Disease Symptoms

Citrus branch dieback associated with *Lasiodiplodia* spp. has been reported in many citrus-growing regions worldwide and is frequently observed in orchards where Huanglongbing is also present. Because HLB is a highly regulated disease in several citrus-producing countries, its characteristic symptomatology is often used as a primary reference for orchard inspection, surveillance programs, and phytosanitary decision-making [4, 12]. However, HLB-like symptoms may overlap with those induced by other biotic and abiotic disorders, including nutritional deficiencies, drought stress, and fungal trunk and branch diseases, which complicates symptom-based diagnosis under field conditions [12, 30].

At the foliar level, symptom expression associated with *Lasiodiplodia*-associated dieback and HLB does not fully coincide, and their diagnostic differences do not exclude the possibility of simultaneous infections within the same tree. HLB typically produces blotchy mottle, asymmetric chlorosis, vein corking, and small, lopsided fruits with color inversion, whereas *Lasiodiplodia* infections are more commonly associated with twig blight, localized necrosis, cankers, gummosis, and progressive dieback originating from branches or pruning wounds [4, 26, 29]. Nonetheless, canopy thinning, premature leaf drop, and generalized yellowing may occur in both conditions, particularly in advanced stages of decline. Under such circumstances, dieback symptoms may be incorrectly attributed solely to HLB, especially in orchards where CLas is already widespread.

Field observations from different citrus-producing regions suggest that canopy decline and defoliation linked to *Lasiodiplodia* infections may be more pronounced in physiologically compromised trees, supporting the hypothesis that chronic HLB infection could predispose citrus hosts to opportunistic fungal colonization [29, 30]. Therefore, the presence of HLB should not exclude *Lasiodiplodia* spp. as contributing causal agents of branch decline, particularly when necrotic lesions, wood discoloration, and gummosis are evident.

Although knowledge regarding the population diversity, epidemiology, and pathogenic mechanisms of *Lasiodiplodia* species affecting citrus remains incomplete, symptom monitoring and disease progression assessments can provide valuable information for orchard-level decision-making. Importantly, overlapping symptoms such as chlorosis, canopy thinning, and leaf abscission (commonly associated with HLB) may be intensified when *Lasiodiplodia*-associated dieback is also present, thereby complicating field diagnosis and reinforcing the need for pathogen-specific molecular detection methods and systematic isolation-based confirmation [12, 23, 26].

Potential Interactions between *Lasiodiplodia* and HLB

Lasiodiplodia-associated citrus dieback and Huanglongbing may interact within citrus trees, potentially influencing symptom expression and accelerating decline in co-infected hosts. Although direct experimental evidence remains limited, the frequent field co-occurrence of both diseases supports the hypothesis that their combined presence may amplify physiological disruption beyond the effects of either pathogen alone [17, 26]. To explain possible interaction mechanisms, several non-mutually exclusive hypotheses can be proposed. First, *Lasiodiplodia*-derived pathogenicity factors and the host responses induced by wood colonization may alter carbon allocation, nutrient uptake, and resource redistribution, thereby modulating HLB symptom development. Second, competition for host assimilates and mineral nutrients may intensify the metabolic imbalance caused by CLas infection, contributing to earlier canopy decline. Third, disruption of vascular integrity caused by fungal necrosis of woody tissues may exacerbate the systemic effects of HLB by further impairing translocation processes and accelerating water stress and canopy collapse. Understanding the timing of CLas establishment relative to subsequent fungal infection, as well as the extent to which both pathogens converge on shared physiological pathways, is essential to evaluate these potential interactions [20, 30].

Potential interactions may also be mediated through overlapping defense and stress-response signaling pathways. Co-infection could promote immune suppression, antagonistic hormonal crosstalk, or defense trade-offs, potentially facilitating higher pathogen colonization or increased symptom severity. Transcriptomic and metabolomic studies have shown that HLB induces extensive reprogramming of citrus gene expression and is associated with strong alterations in phytohormone homeostasis, carbohydrate metabolism, and defense-related pathways [8, 20, 19]. In particular, Salicylic Acid (SA)-associated responses are frequently upregulated in CLas-infected tissues, while Jasmonic Acid (JA) and ethylene signaling may be altered depending on infection stage and tissue type, potentially influencing the ability of citrus to respond to necrotrophic or opportunistic fungi [20, 30]. Because *Lasiodiplodia* spp. behave as stress-associated pathogens capable of rapidly colonizing weakened woody tissues, hormonal imbalances and carbon depletion caused by HLB could predispose trees to fungal invasion and accelerate branch mortality [26, 29]. Collectively, these observations suggest that co-infection may intensify physiological dysfunction in citrus, although the net outcome is likely to vary depending on host genotype, orchard conditions, and the sequence of pathogen establishment.

Mechanistic Hypotheses for Interaction

Under co-infection scenarios, the physiological status of citrus trees may be substantially altered, potentially influencing symptom severity and disease progression associated with either pathogen. Current mechanistic hypotheses regarding potential interactions between *Lasiodiplodia*-associated dieback and HLB mainly involve: (i) competition for water, mineral nutrients, and photoassimilates; (ii) disruption of metabolic homeostasis and resource allocation, which may indirectly constrain the synthesis of defense-related signaling molecules such as Salicylic Acid (SA) and Jasmonic Acid (JA); and (iii) the combined capacity of both pathogens to impair vascular function, thereby compromising the structural and physiological integrity of phloem and xylem associated tissues [19, 29]. These mechanisms are not mutually exclusive and may operate simultaneously depending on the infection stage and environmental context.

Several host responses reported during the progression of both diseases support the possibility of convergent or partially overlapping stress-response pathways, including altered phytohormone homeostasis, transcriptional reprogramming of defense-related genes, and changes in carbohydrate metabolism and transport. In CLas-infected citrus, transcriptomic and metabolomic studies have consistently reported major disturbances in immune signaling, Reactive Oxygen Species (ROS)-related processes, and phloem function, which collectively contribute to progressive decline [19, 20]. In contrast, Botryosphaeriaceae fungi such as *Lasiodiplodia* spp. are frequently associated with necrosis of woody tissues and stress-induced disease expression, often involving the activation of ethylene-associated responses and host senescence pathways [23, 29]. These overlapping host responses may contribute to additive or synergistic symptom development under co-infection, although the magnitude and direction of such effects likely depend on host genotype, environmental conditions, and the temporal sequence of pathogen establishment.

An integrated approach combining pathogen-specific detection methods with systematic monitoring of host physiological parameters such as, carbohydrate reserves, water relations, hydraulic conductivity, and vascular integrity, together with phytohormone profiling, would provide critical insights into the biological relevance of these interactions. Such information is essential to refine diagnosis and develop more effective management strategies for citrus decline syndromes involving multiple pathogens.

Conceptual Model of Co-Infection and Decline Progression

A conceptual framework may help explain the frequent field association between HLB and *Lasiodiplodia*-associated branch dieback in citrus orchards. In this model, CLas infection is proposed to act as an early, systemic driver of chronic physiological decline by disrupting phloem transport, altering source–sink relationships, and progressively impairing root health. Root loss and reduced nutrient uptake capacity can lead to long-term carbohydrate depletion and increased sensitivity to abiotic stress, particularly drought and high temperature, which are common in many citrus-growing regions [12, 16, 30]. These changes may reduce the capacity of infected trees to sustain normal canopy renewal and defense responses, thereby creating a favorable physiological context for secondary colonization by opportunistic pathogens.

Within this decline trajectory, *Lasiodiplodia* spp. may act as stress-associated fungi that exploit weakened woody tissues through pruning wounds, sunburn damage, or natural openings. Once established, the fungus may accelerate decline by inducing localized necrosis, canker formation, gummosis, and vascular discoloration, ultimately contributing to branch mortality and canopy collapse [23, 26, 29]. In this scenario, fungal infection may not be directly dependent on CLas presence, but HLB-mediated physiological disruption may increase host susceptibility and enhance the probability of successful fungal colonization. Conversely, fungal-driven wood necrosis may intensify the systemic effects of HLB by reducing water transport efficiency and further limiting carbon allocation to roots and developing tissues.

This conceptual model supports the hypothesis that citrus decline in HLB-endemic orchards may represent a multifactorial syndrome driven by the combined effects of phloem dysfunction, root deterioration, and opportunistic wood colonization. Importantly, the outcome of co-infection is likely to depend on environmental stress intensity, orchard management practices (e.g., pruning frequency, irrigation regimes, and wound protection), and the sequence of pathogen establishment. Therefore, integrated studies combining spatial epidemiology, temporal infection tracking, and physiological monitoring are required to validate this framework and determine whether HLB functions primarily as a predisposing factor, a synergistic partner, or an independent contributor to decline in orchards affected by *Lasiodiplodia* spp. [17, 19].

Shared Plant Responses and Immune Modulation

Transcriptomic studies of Huanglongbing (HLB) associated with CLas have revealed extensive transcriptional reprogramming in infected citrus tissues, including the induction of multiple stress-responsive and defense-related gene networks, together with the repression of a smaller subset of genes involved in growth, metabolism, and transport [8, 19]. These findings support the concept that HLB is characterized by strong immune dysregulation and chronic activation of host defense pathways. In this context, many HLB symptoms may reflect a long-term damage-response condition in which citrus trees progressively decline while maintaining elevated defense signaling and oxidative stress responses [19, 30].

Comparable immune modulation has also been reported during *Lasiodiplodia*-associated citrus dieback. Members of the Botryosphaeriaceae frequently behave as latent pathogens that transition to aggressive colonization under host stress, and infection is often accompanied by marked activation of stress-associated hormonal pathways. Increased accumulation of Salicylic Acid (SA) and Jasmonic Acid (JA), as well as the upregulation of SA- and JA-responsive genes, has been reported in citrus and other woody hosts infected by Botryosphaeriaceae fungi, suggesting that hormonal imbalance and immune activation are key components of disease development [29, 31]. Notably, elevated SA levels have been detected in citrus leaves prior to the appearance of severe necrotic symptoms, indicating that defense-related signaling may precede visible lesion formation and contribute to early physiological decline [31].

Citrus trees co-infected by *Lasiodiplodia* spp. and CLas may therefore exhibit altered symptom expression and disease progression compared with trees affected by either pathogen alone. Studies on plant disease complexes indicate that co-colonization can modify host physiological status, nutrient allocation, and immune regulation, leading to disease outcomes that may differ from those expected under single-pathogen infections [17]. In the specific context of HLB and *Lasiodiplodia* dieback, co-occurrence may influence symptom severity through overlapping hormonal signaling networks, resource competition, or cumulative disruption of vascular function. However, mechanistic evidence directly demonstrating synergistic interactions remains limited, and further experimental work is required to determine whether these interactions are additive, synergistic, or largely independent across citrus genotypes and environmental conditions.

Influence of Co-Infection on Disease Severity and Tree Physiology

Citrus trees simultaneously affected by HLB and *Lasiodiplodia*-associated branch dieback may experience compounded physiological stress due to the combined disruption of vascular transport, nutrient allocation, and carbon partitioning. Under co-infection scenarios, trees may shift resource allocation toward defense-related processes at the expense of growth, potentially increasing susceptibility to abiotic stresses such as drought, heat, and nutrient limitation. Because HLB is strongly associated with phloem dysfunction and progressive root decline, infected trees often exhibit impaired nutrient uptake and reduced carbohydrate availability, which may predispose them to opportunistic wood-colonizing fungi that preferentially infect weakened tissues [12, 16, 29].

In this context, *Lasiodiplodia* spp. may further intensify decline by colonizing woody tissues and inducing necrosis, cankers, and vascular discoloration, processes that can compromise xylem integrity and water transport. Reduced vascular reinforcement and weakened cell wall responses in stressed trees may facilitate fungal spread through lignified tissues, accelerating branch dieback and canopy collapse [23, 26]. Moreover, the combined nutritional and hydraulic limitations imposed by both diseases could reduce the capacity of citrus trees to sustain effective defense responses and maintain physiological homeostasis, thereby amplifying symptom severity.

Although direct experimental evidence demonstrating synergistic interactions between HLB and *Lasiodiplodia* infection is still lacking, studies of HLB physiology support the plausibility of stress-mediated predisposition. HLB-infected trees commonly exhibit altered mineral nutrient profiles, carbohydrate depletion, and reduced photosynthetic performance, which may indirectly favor colonization by secondary pathogens and accelerate decline syndromes [30]. Therefore, the frequent field co-occurrence of both diseases may reflect convergent effects on tree physiology rather than direct pathogen–pathogen interactions, reinforcing the need for controlled co-infection experiments and longitudinal field monitoring.

Diagnostic Considerations in Co-Infected Trees

The potential co-occurrence of Huanglongbing and *Lasiodiplodia*-associated citrus dieback requires an integrated diagnostic framework capable of detecting both pathogens and distinguishing overlapping decline symptoms. Reliable diagnosis is particularly important because canopy thinning, chlorosis, and dieback may be incorrectly attributed solely to HLB in endemic areas, while wood-inhabiting fungi may remain undetected unless systematic isolation and molecular confirmation are performed [12, 26].

Diagnosis of *Lasiodiplodia*-associated dieback is typically based on a combination of symptom assessment (e.g., twig and branch dieback, cankers, gummosis, and wood discoloration), fungal isolation from symptomatic tissues, and morphological identification of characteristic pycnidia and conidia produced on infected plant material or artificial substrates [23, 26]. In culture, *Lasiodiplodia* isolates often produce rapidly growing, initially white mycelium that later darkens, although morphological traits alone are insufficient for species-level identification due to overlap among Botryosphaeriaceae taxa. Therefore, molecular tools based on amplification and sequencing of the rDNA ITS region, frequently complemented with additional loci such as *tef1-α*, *tub2*, and *rpb2*, are currently recommended to ensure accurate identification within the *Lasiodiplodia* species complex [24, 33]. Although primer pairs targeting ITS fragments have been widely used, multilocus approaches provide improved taxonomic resolution and are essential for distinguishing cryptic species with potentially different aggressiveness and epidemiological relevance [33].

Table 1: Comparative diagnostic features of Huanglongbing (HLB) and Lasiodiplodia-associated citrus dieback [4, 12, 16, 18, 23, 26, 27, 33].

Feature	Huanglongbing (HLB)	*Lasiodiplodia*-associated dieback
Causal agent	*Candidatus* Liberibacter asiaticus (CLas) (phloem-limited bacterium)	*Lasiodiplodia* spp. (Botryosphaeriaceae; wood-inhabiting fungi)
Primary plant tissue affected	Phloem tissues (systemic infection)	Woody tissues (xylem-associated colonization, bark, cambium)
Key foliar symptoms	Blotchy mottle, asymmetric chlorosis, vein corking, leaf thickening, upright leaves	General chlorosis, wilting, leaf drop, canopy thinning
Key branch/trunk symptoms	Twig dieback may occur in advanced stages; usually secondary to canopy decline	Cankers, gummosis, bark cracking, internal wood discoloration, progressive twig and branch dieback
Fruit symptoms	Small, lopsided fruit; aborted seeds; color inversion; poor juice quality	Fruit rot, premature fruit drop, mummified fruit
Pattern of symptom distribution	Often sectorial/asymmetric; blotchy mottle irregular within the canopy	Often localized, starting from infected branches; dieback progresses from tips inward
Vector involvement	Yes, primarily *Diaphorina citri*	No confirmed insect vector; infection commonly associated with wounds and stress
Predisposing factors	Psyllid pressure, infected nursery stock, flush cycles	Drought stress, heat stress, pruning wounds, sunburn, mechanical injury
Recommended field sampling tissue	Roots (preferred), leaf midribs/petioles, psyllids	Symptomatic branches, canker margins, necrotic wood tissues, fruit lesions
Best tissue for early detection	Roots (more consistent detection before foliar symptoms)	Woody tissues at lesion margins (active infection zone)
Main laboratory diagnostic method	qPCR targeting CLas (16S rDNA, *rplJ/rplL*, etc.)	Isolation + morphology + multilocus sequencing (ITS + *tef1-α*, *tub2*, *rpb2*)
Culture-based detection	Not culturable under routine conditions	Routinely culturable on PDA/MEA; pycnidia and conidia often produced
Morphological confirmation	Not applicable	Pycnidia formation; pigmented conidia; fast-growing mycelium
Molecular confirmation	Conventional PCR / qPCR	PCR + sequencing
Recommended complementary approach	Vector scouting and spatial mapping	Evaluation of wound history, pruning intensity, and abiotic stress indicators
Diagnostic limitations	Uneven bacterial distribution; false negatives in leaves; latent infection	Species complexes; morphology overlaps; latent/endophytic presence complicates interpretation
Interpretation in co-infection	May explain systemic decline but not localized cankers	May explain localized necrosis and dieback but not classic blotchy mottle fruit symptoms

In contrast, HLB diagnosis requires pathogen-specific detection of CLas, primarily through PCR-based methods, including conventional PCR and quantitative real-time PCR (qPCR), which remain the most widely adopted diagnostic tools for regulatory and surveillance programs [30]. Monitoring psyllid populations is also an important complementary component of disease management, as vector presence and population peaks are strongly associated with increased risk of pathogen introduction and spread [12, 14]. CLas can be detected during asymptomatic stages; however, early detection is challenged by uneven bacterial distribution and low titers in aboveground tissues. Several studies indicate that CLas detection is more consistent in root tissues than in leaves and that root colonization may precede visible canopy symptoms by several months, reinforcing the diagnostic value of root sampling for early-stage surveillance [16, 27]. Root sampling near the trunk or from structural lateral roots is often recommended because CLas colonization may occur earlier in proximal root tissues before becoming uniformly distributed throughout the root system [16].

Because co-infection may exacerbate physiological disruption and accelerate decline, quantifying pathogen abundance and documenting symptom progression may help determine whether one disease is intensifying the effects of the other. In this context, integrating pathogen detection (qPCR for CLas and multilocus sequencing for *Lasiodiplodia* spp.) with field symptom monitoring, vector scouting, and spatial–temporal disease mapping may improve diagnostic accuracy and guide management decisions. Additionally, dieback syndromes are often influenced by predisposing biotic factors such as pruning wounds and wood-boring insects, which may facilitate fungal infection by increasing entry points and weakening vascular tissues [23, 29]. Therefore, diagnostic frameworks should incorporate orchard history, pest pressure, and environmental stress indicators to support more accurate interpretation of decline symptoms and to inform preventive interventions.

Recommended Diagnostic Workflow for Co-Infected Orchards

Accurate diagnosis of citrus decline in orchards where HLB and *Lasiodiplodia*-associated dieback may co-occur requires a structured workflow combining field inspection, pathogen-specific detection, and contextual epidemiological information. Because both diseases can produce overlapping canopy symptoms, a stepwise diagnostic strategy is recommended to reduce misdiagnosis and to support timely management decisions.

The first step should involve systematic field scouting focused on distinguishing foliar and fruit symptoms typical of HLB (e.g., blotchy mottle, asymmetric chlorosis, lopsided fruit, and color inversion) from dieback-associated symptoms such as localized branch necrosis, cankers, gummosis, and wood discoloration. Visual inspection should be complemented by recording orchard management practices, recent pruning events, drought stress indicators, and the presence of wood injuries that may predispose trees to fungal colonization. Concurrent monitoring of *Diaphorina citri* populations is recommended, as vector presence strongly increases the likelihood of active CLas spread and supports epidemiological interpretation [12].

For HLB confirmation, sampling should prioritize root tissues and symptomatic leaves, followed by CLas detection using qPCR-based assays. Root sampling near the trunk and from major lateral roots is advisable because CLas can be detected more consistently in roots than in canopy tissues during early infection stages [16, 27]. For *Lasiodiplodia* diagnosis, sampling should target symptomatic branches showing cankers, gummosis, or internal vascular discoloration. Isolation on culture media followed by morphological observation can provide preliminary identification, but species-level confirmation should rely on molecular sequencing of ITS combined with the loci, *tef1-α, tub2,* and *rpb2*, given the high prevalence of cryptic species within the genus [23, 33].

Finally, integrating diagnostic results with spatial mapping of infected trees and temporal monitoring of symptom development can improve interpretation of disease progression and potential interactions. Orchards showing consistent co-detection of CLas and *Lasiodiplodia* spp. should be prioritized for longitudinal sampling to assess whether fungal colonization occurs preferentially after HLB establishment, and whether disease severity correlates with pathogen load, water stress intensity, or orchard practices. This workflow provides a practical framework to support surveillance programs and improve management decisions in citrus production systems affected by complex decline syndromes (Table 1).

Management and Mitigation Strategies

In the absence of durable long-term solutions for Huanglongbing, integrated management remains essential to sustain productivity in orchards where HLB and *Lasiodiplodia*-associated dieback may co-occur. Under these conditions, mitigation strategies should aim to reduce pathogen pressure, minimize physiological stress, and preserve canopy function through a combination of cultural, chemical, and biological approaches. Because both diseases are strongly influenced by host stress status, management should be implemented adaptively and supported by continuous monitoring of symptom progression, vector abundance, and environmental conditions [12, 29].

Cultural practices that improve tree vigor and reduce abiotic stress may indirectly limit disease progression by strengthening host defenses and reducing susceptibility to opportunistic pathogens. Optimized irrigation management, balanced fertilization, and avoidance of nutrient deficiencies are particularly important in HLB-endemic orchards, as CLas infection is associated with progressive root decline and impaired nutrient uptake [16, 30]. Micronutrient supplementation and soil amendments have been explored as supportive measures to maintain canopy health and stimulate root activity, although such interventions do not eliminate the pathogen and should be considered part of an integrated mitigation program rather than a curative solution [15].

Management of *Lasiodiplodia* dieback primarily relies on reducing infection opportunities and limiting inoculum reservoirs. Sanitation practices such as pruning and removal of symptomatic branches, destruction of infected debris, and disinfection of pruning tools are essential to reduce local fungal spread. Because *Lasiodiplodia* spp. frequently infect through wounds, pruning should be performed during periods of low rainfall, and large cuts should be minimized or protected to reduce fungal entry [23, 29]. Orchard practices that reduce mechanical injury, sunburn damage, and branch breakage are also important, particularly in high-density systems or under strong winds. In addition, irrigation practices should avoid prolonged water stress, as drought predisposition is strongly associated with Botryosphaeriaceae-related dieback outbreaks [29].

Chemical and biological control options have been evaluated for both HLB and *Lasiodiplodia*, although field-level efficacy remains inconsistent. In HLB management, insecticide-based psyllid suppression and the use of certified pathogen-free nursery stock remain fundamental measures to reduce pathogen introduction and secondary spread [12]. For fungal dieback, fungicides may provide limited protection when applied preventively on pruning wounds, but they are generally less effective once internal wood colonization has occurred. Therefore, chemical control should be regarded mainly as a preventive tool rather than a curative strategy for established infections [23]. Biological approaches, including the use of endophytic bacteria and fungi with antagonistic activity, have shown promise under experimental conditions; however, their persistence, colonization stability, and reproducibility under orchard environments remain key limitations [26].

Long-term mitigation efforts increasingly emphasize the development of tolerant scion–rootstock combinations and the deployment of HLB-tolerant or HLB-resistant rootstocks. Rootstock breeding programs are now recognized as critical components of sustainable citrus production because rootstocks influence nutrient uptake, stress tolerance, and defense responsiveness. Importantly, breeding strategies may also consider tolerance to decline-associated pathogens such as *Lasiodiplodia* spp., thereby supporting broader resilience against multifactorial orchard stress syndromes [11, 31]. Ultimately, integrated approaches combining genetic resistance, vector management, orchard sanitation, and stress reduction practices will be essential to mitigate citrus decline under scenarios involving HLB and opportunistic fungal pathogens.

Applicable Management Strategies Under HLB-*Lasiodiplodia* Co-Occurrence

Management of orchards affected by HLB and *Lasiodiplodia*-associated dieback should be addressed as a multifactorial decline syndrome. Because symptoms overlap, the first priority is accurate diagnosis, combining CLas detection by qPCR with fungal isolation and multilocus identification of *Lasiodiplodia* spp. from symptomatic branches. This integrated approach improves decisions on pruning, sanitation, and tree removal.

Since HLB spread is driven mainly by *D. citri*, vector monitoring and suppression remain essential, particularly during flushing periods, together with the use of certified pathogen-free nursery stock. For *Lasiodiplodia* dieback, management should emphasize sanitation and wound prevention, including pruning during dry periods, removing infected branches below necrotic tissue, destroying debris, and disinfecting pruning tools. Preventive protection of major pruning cuts may reduce infection risk, although fungicides are generally ineffective once internal colonization is established.

Because drought and heat stress strongly favor Botryosphaeriaceae outbreaks and aggravate HLB decline, stress reduction practices are critical. Optimized irrigation and balanced fertilization can sustain canopy renewal and root function, slowing decline progression. In severely affected orchards, removing chronically declining trees and replanting with tolerant rootstock-scion combinations may be more cost-effective than continued maintenance. Spatial monitoring of hotspots can further support targeted interventions and reduce orchard level deterioration.

Conclusion

Huanglongbing and *Lasiodiplodia*-associated branch dieback represent two major constraints to citrus production that increasingly co-occur in modern orchards, particularly in tropical and subtropical regions. Although these diseases differ markedly in etiology and epidemiology, HLB being a systemic, vector-transmitted bacterial disease and *Lasiodiplodia* dieback being a stress-associated woody tissue infection, both converge in their capacity to impair vascular function, disrupt carbohydrate allocation, and accelerate canopy decline. This convergence contributes to overlapping symptom expression under field conditions, complicating diagnosis and potentially masking the contribution of opportunistic fungal pathogens in HLB-endemic areas.

Evidence reviewed here supports the hypothesis that HLB may predispose citrus trees to *Lasiodiplodia* infection by promoting chronic physiological stress, root deterioration, and nutritional imbalance. Conversely, fungal colonization of woody tissues may intensify decline by accelerating xylem dysfunction, increasing water stress, and amplifying the physiological costs of CLas infection. While mechanistic evidence demonstrating direct synergism remains limited, the frequent spatial overlap of both pathosystems and the shared host responses involving immune activation, oxidative stress, hormonal imbalance, and resource depletion suggest that co-infection may represent an important component of citrus decline syndromes. Consequently, distinguishing whether these interactions are additive, stress-mediated, or truly synergistic remains a priority for future research.

Improved diagnostic frameworks are essential to address complex decline scenarios. Root-based molecular detection for CLas, combined with culture-dependent and multilocus molecular identification of *Lasiodiplodia* spp., provides a robust approach to differentiate causal agents and reduce misdiagnosis. In parallel, integrated orchard management should incorporate strict sanitation, pruning wound protection, vector suppression, and cultural practices aimed at reducing abiotic stress, particularly drought and heat, which strongly influence disease expression. Long-term sustainability will likely depend on the deployment of tolerant scion–rootstock combinations and the integration of genomic and multi-omics tools to identify key host traits and pathogen targets for improved resistance and disease surveillance.

Overall, this review highlights that citrus decline in HLB-endemic production systems should be increasingly viewed as a multifactorial syndrome rather than a single-pathogen disease outcome. Addressing the combined impact of CLas infection and opportunistic fungal dieback pathogens will require coordinated efforts integrating epidemiology, molecular diagnostics, plant physiology, and orchard-level management strategies to protect citrus productivity and mitigate the long-term economic consequences of decline.

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