Plant Archives Vol. 25, No. 2, 2025 pp. 1257-1268 e-ISSN:2581-6063 (online), ISSN:0972-5210

Plant Archives

Journal homepage: http://www.plantarchives.org

DOI Url : https://doi.org/10.51470/PLANTARCHIVES.2025.v25.no.2.181

LASIODIPLODIA THEOBROMAE: A FACULTATIVE PATHOGEN OF COCONUT- A COMPREHENSIVE REVIEW

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(Date of Receiving-04-07-2025; Date of Acceptance-19-09-2025)

ABSTRACT

Lasiodiplodia theobromae is a globally significant facultative parasitic fungus implicated in severe diseases of coconut and other economically important crops. As an endophyte capable of shifting to a pathogenic lifestyle under stress, its infection process is influenced by various abiotic factors such as drought, heat, and nutrient imbalance, which weaken host defenses and facilitate tissue colonization. In coconut, *L. theobromae* is associated with stem bleeding, fruit rot and leaf blight, often exacerbated by poor agronomic conditions, hybrid susceptibility, and compromised host physiology. The pathogen's arsenal of cell wall-degrading enzymes, phytotoxins, and stress-responsive genes contributes to its virulence and adaptability. Host–pathogen interactions are shaped by hormonal crosstalk, oxidative responses and structural vulnerabilities, especially under nutrient and water stress. Recent research highlights the role of integrated management, including biocontrol agents (e.g., *Trichoderma* spp., *Bacillus* spp.), novel fungicides, and cultural practices in mitigating disease impact. Understanding the complex interplay of environmental stressors, host susceptibility, and pathogen biology is essential for devising effective and sustainable control strategies in coconut cultivation and beyond.

Key words : *Lasiodiplodia theobromae*, Coconut, Facultative pathogen, Abiotic factors, Management strategies, Infection process, Predisposing factors, Host-pathogen interaction.

Introduction

Coconut (*Cocos nucifera* L.), a vital tropical crop, serves as an essential source of food, oil, fiber, and income for millions across Asia, Africa, and Latin America. However, its productivity is significantly threatened by a range of biotic stresses, among which fungal pathogens play a dominant role. One of the most destructive and globally distributed fungal pathogens affecting coconut is *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl., a member of the family Botryosphaeriaceae.

L. theobromae is a facultative pathogen exhibiting significant genetic and morphological variability, which complicates accurate diagnosis. Recent advances in molecular phylogenetics, particularly the use of multilocus sequencing (ITS, TEF1-α, tub2), have enhanced the resolution of species identification, helping distinguish *L. theobromae* from closely related congeners. Despite its increasing incidence, particularly under stress conditions such as drought, mechanical injury, and nutrient deficiencies, the epidemiology and management of *L. theobromae*-induced diseases in coconut remain poorly understood. Given the economic significance of coconut and the growing number of reported infections worldwide, there is an urgent need to consolidate current knowledge on the pathogen's pathogenicity, distribution, symptomatology and effective control measures.

L. theobromae is a widespread fungal pathogen affecting over 500 plant species, including coconut, especially under stress conditions. It is responsible for severe diseases such as stem-end rot and leaf blight, with

notable reports from Brazil (Machado *et al.*, 2016), India (Arunkumar *et al*., 2022), Southeast Asia and Africa (FAO, 2015). In 2025, *L. theobromae* was first identified in the USA, where it caused rachis blight in Florida (Ploetz *et al*., 2025). The global spread of this pathogen highlights the urgent need for enhanced surveillance, effective disease management strategies, and stress management practices in coconut plantations.

This review aims to synthesize existing literature on *L. theobromae* as a pathogen of coconut, highlighting disease manifestations, regional case studies, identification challenges, and future research directions.

Infection context

L. theobromae is widely recognized as a facultative pathogen that can exist asymptomatically within coconut tissues as a latent endophyte (Machado *et al*., 2016). This latent phase enables the fungus to persist without causing immediate harm to the host. However, disease symptoms typically manifest when the coconut palm is subjected to abiotic stress factors such as drought, high temperature, nutrient deficiencies, or mechanical injuries that compromise the plant's natural defenses (Arunkumar *et al*., 2022). These stress conditions trigger the pathogen to switch from a quiescent to an aggressive phase, leading to the onset of disease.

The pathogen infects multiple plant parts, each exhibiting characteristic symptoms. The leaves often develop blight, with necrotic lesions expanding along the leaf blades, which significantly reduces photosynthetic capacity and overall plant vigor (Arunkumar *et al*., 2022). Immature coconut fruits are susceptible to postharvest stem-end rot, a major cause of economic losses, particularly under conditions of high humidity and poor handling practices (Machado *et al*., 2016; Ploetz *et al*., 2025). The fungus also infects the inflorescence and rachis, causing wilting and progressive tissue breakdown, which adversely affects flowering and nut development (Ploetz *et al*., 2025). Additionally, wounds on the trunk or pruning sites serve as critical entry points for *L. theobromae*, especially in environments with high humidity, facilitating fungal colonization and systemic infection (Batista *et al*., 2019).

Such multifaceted infection strategies underscore the opportunistic nature of *L. theobromae* in coconut palms, where environmental and physiological stressors create favorable conditions for disease emergence. Effective management, therefore, requires integrated approaches targeting both host resilience and environmental conditions to suppress latent infections before they develop into severe outbreaks.

Post-harvest diseases further exacerbate economic losses associated with *L.theobromae*. For instance, in Brazil, stem-end rot of papaya caused by *L. theobromae* can reach incidences of 70–80%, significantly diminishing fruit quality and reducing its marketability (European Food Safety Authority [EFSA], 2020). Similarly, in Nigeria, *L. theobromae* has been identified as a major storage pathogen of kola nut (*Cola* spp.), where its rapid post-harvest spread not only reduces yield but also threatens the sustainability of smallholder kola nut production systems (Oladigbolu *et al*., 2023). In Saudi Arabia, a field survey found approximately 40% disease severity and 15% incidence of mango dieback due to *L. theobromae*, underscoring the substantial yield losses and regional vulnerability associated with this pathogen (Al-Sadi *et al*., 2022).

Global incidence and severity of *L. theobromae* in Coconut

L. theobromae is a notable fungal pathogen that affects coconut palms across diverse tropical and subtropical regions worldwide. The incidence and severity of diseases caused by this pathogen vary significantly depending on environmental conditions, host susceptibility, and agricultural practices.

In Brazil, *L. theobromae* has been identified as the primary causal agent of postharvest stem-end rot in immature coconut fruits. This disease is particularly problematic in the northeastern states, which are key coconut-producing areas. The pathogen's aggressive behavior under humid conditions and poor postharvest management leads to considerable economic losses, underscoring the urgent need for effective control measures (Machado *et al*., 2016).

A recent study from Florida, USA documented a 30% disease incidence of *L. theobromae* causing rachis blight in coconut palms. The severity of infection varied widely, affecting between 20% to 100% of the canopy, thus signaling a potential threat to coconut cultivation in non-traditional growing regions previously considered free of this pathogen (Ploetz *et al*., 2025).

Moreover, China has reported the presence of *L. theobromae* causing postharvest stem-end rot in coconuts grown in Guangdong Province. This represents the first confirmed report of the pathogen in the country, indicating an expanding geographical distribution and raising concerns for regional coconut production (Zhang and Niu, 2019).

In India, *L. theobromae* is implicated in leaf blight and fruit rot diseases of coconut. Field studies have

reported disease incidences ranging from 10% to 25%, with the highest prevalence observed during the dry, summer months when palms are stressed. These infections contribute to significant reductions in yield and highlight the importance of adopting timely and integrated management strategies (Arunkumar *et al*., 2022).

In Andhra Pradesh, as in other regions of South India, *L. theobromae* has emerged as a significant pathogen of coconut particularly under conditions of abiotic stress. Although specific incidence data for Andhra Pradesh are limited, evidence from neighboring Tamil Nadu provides valuable insights due to similar agroclimatic conditions leaf blight causes substantial nut yield losses ranging from 10% to 25%, with peak incidence typically occurring during the hot summer months (TNAU, 2015). The disease is characterized by symptoms such as necrotic lesions on the lower leaflets, wilting, and premature nut drop, resulting in reduced productivity (TNAU, 2015; Parachute Kalpavriksha, 2022).

Field studies on disease management in Tamil Nadu have demonstrated that the severity of *L. theobromae* infections can be reduced significantly through chemical and biological interventions. For instance, the use of systemic fungicides such as propiconazole and tebuconazole via root feeding led to a 5.66% reduction in disease incidence and increased average nut yield from 99 to 135 nuts per palm per year (Latha *et al*., 2024). In addition, integrated biological control strategies involving root feeding with *Pseudomonas fluorescens*, in combination with soil application of microbial consortia and organic inputs, have been shown to reduce disease incidence to as low as 6.5% to 12.9%, depending on seasonal and regional factors (Johnson *et al*., 2014).

Although, region-specific Percent Disease Index (PDI) values for Andhra Pradesh are not extensively documented, comparable disease pressure is assumed, given the similarity in environmental stressors. These include frequent droughts, high summer temperatures, nutrient-deficient lateritic soils, and mechanical injuries, all of which compromise host vigor and facilitate opportunistic fungal invasion (TNAU, 2015; Parachute Kalpavriksha, 2022). Crop losses in affected plantations are typically estimated at 10%–25%, aligning with field observations and extension data from South India (TNAU, 2015; Johnson *et al*., 2014).

The recent upsurge in disease prevalence in Andhra Pradesh is likely due to a combination of factors: increasing abiotic **stress**, particularly heat and water scarcity during pre-monsoon months; inadequate fertilization and soil health degradation; mechanical damage from pruning or harvesting practices that act as infection courts; and humid microclimates during monsoon transitions that promote sporulation and pathogen dissemination (TNAU, 2015; Parachute Kalpavriksha, 2022). These conditions collectively enable *L. theobromae* to shift from a latent endophyte to a destructive pathogen, significantly affecting both the yield and quality of coconut production in Andhra Pradesh.

Disease Symptoms of *L. theobromae* in coconut

L. theobromae manifests through a variety of symptoms across different parts of the coconut palm, reflecting its opportunistic and aggressive nature, particularly under stress conditions.

Leaf Blight : Characteristically, the leaflets of the outer whorls begin desiccating from the tips downward, adopting a charred or "burnt" appearance. On immature nuts, dark grey to brown lesions with wavy margins develop near the apex. The fungus penetrates the kernel through the mesocarp, leading to endosperm decay, and resulting in shriveled, malformed nuts that drop prematurely a disease dynamic that can cause yield losses of 10–25% (Parachute Kalpavriksha, 2022).

Immature Nut Rot : Dark grey to brown lesions that initiate at the nut apex expand across the surface, with the mesocarp and endosperm deteriorating. Severely infected nuts become desiccated, deformed, and frequently drop early, especially when associated with mite infestation (Parachute Kalpavriksha, 2022).

Postharvest Stem-End Rot : Observed in China and Brazil, this symptom manifests as black rot beginning at the peduncle below the bracts, progressing inward to the endosperm. Inoculation trials confirm its pathogenic nature (Zhang and Niu, 2019).

Rachis Blight : In Florida, disease onset appears on older fronds and spreads toward the younger ones. Affected tissues exhibit internal discoloration, followed by profuse sporulation and fruiting body formation. In severe cases, the entire frond dies; incidence reached approximately 30%, with canopy severity between 20%–100% (Ayika *et al*., 2025).

Etiology of *L. theobromae* in coconut

L. theobromae, a member of the Botryosphaeriaceae family, is a facultative fungal pathogen widely distributed in tropical and subtropical zones (Alves *et al*., 2008) APS Journals. Its ability to adopt both endophytic and pathogenic lifestyles is central to its etiology in coconut. Under favorable conditions, the pathogen resides latently within plant tissues; however, environmental stressors such as drought, nutrient deficiency, and mechanical injury

can trigger its transition to active disease-causing stages (Marsberg *et al*., 2017).

Several studies have highlighted the role of *L. theobromae* in key disease complexes affecting coconut. In Brazil, a study employing both morphological characteristics and multilocus phylogenetic analysis (using ITS, TEF1-α, and β-tubulin gene regions) investigated isolates associated with postharvest stem-end rot in immature coconuts. The findings confirmed that *L. theobromae*, along with closely related species such as *L. brasiliense, L. egyptiacae* and *L. pseudotheobromae* were pathogenic. Among them, *L. theobromae* was identified as the most aggressive species, particularly under humid environmental conditions and suboptimal postharvest handling practices (Rosado *et al.*, 2016).

Diagnostic protocols in leaf blight of coconut in India have similarly relied on morphological characterization and ITS sequencing to confirm *L. theobromae* as the causal agent. Ramjegathesh *et al*. (2019) used pin-prick inoculation and mycelial disc application on multiple coconut varieties, successfully reproducing disease symptoms and fulfilling Koch's postulates.

In China, Zhang and Niu (2019) documented the first occurrence of *L. theobromae* causing postharvest stem-end rot in the coconut-growing regions of Guangdong Province. Molecular identification and disease characterization confirmed the pathogen's etiology. Rosado *et al*. (2016) also noted similar findings regarding species identification in Brazil.

Phylogeographic studies have revealed noteworthy intraspecific diversity, particularly in major disease hotspots such as Brazil and Central Africa, likely shaped by both host diversity and variable management practices. These findings suggest multiple independent introductions or selection events that have influenced the pathogen's evolutionary trajectory and geographic spread (Santos *et al*., 2017).

Morphologically, *L. theobromae* is discerned by characteristic features, including pycnidial paraphyses and longitudinal striations on mature conidia. However, morphological identification alone often proves insufficient due to species overlap. Hence, researchers now prioritize multilocus DNA sequencing with emphasis on ITS, TEF1 α and β tubulin gene sequences for accurate species-level resolution (Slippers *et al*., 2014; Rosado *et al*., 2016).

Nature of survival and spread of *L. theobromae* in coconut

L. theobromae exhibits remarkable resilience by surviving in various forms such as mycelia, pycnidia, and potentially chlamydospores, residing on and within plant debris, soil, and infected tissues a strategy that enables persistence through unfavorable periods (Michereff *et al*., 2005; Kuswinanti *et al*., 2019) This survival mechanism allows the pathogen to remain latent until environmental conditions become conducive for disease development.

The pathogen's spread is primarily driven by conidial (pycnidiospore) dispersal, facilitated by rain splash, irrigation droplets and wind-driven rainfall, which efficiently carry spores to susceptible host tissues (Vásquez López *et al*., 2019) While not extensively documented for *L. theobromae*, it is plausible that insect vectors may also contribute to its dispersal, as has been observed in closely related *Lasiodiplodia* species (Ploetz, 2003)

Entry into the host often occurs through wounds from pruning, harvesting, mechanical damage, insect feeding, or climatic injury though direct penetration into fruit or other tissues has been recorded even without specialized structures like appressoria (Navarro *et al*., 2022; Ploetz, 2003). Following entry, *L. theobromae* is capable of colonizing the vascular system and spreading ahead of visible symptoms, allowing it to establish systemic infections (Burgess *et al*., 2006; Shahbaz *et al*., 2009) Additionally, the pathogen can persist as a latent or quiescent infection within plant tissues, transitioning to an active pathogenic phase when host stress or environmental triggers weaken host defenses (Ventura *et al*., 2004)

Although, specific survival and epidemiological dynamics in coconut remain underexplored, evidence from other hosts suggests similar behavior. For example, in Cucurbitaceae, the pathogen survives on infected seeds and plant debris, with its pycnidia and mycelia enabling survival up to twelve months, depending on the host tissue's infection level (e.g., calabash vs. squash) ephytia.inra.fr. Moreover, *L. theobromae* conidia are known to germinate and sporulate under humid, warm conditions conditions common to many coconut-growing regions (Ellis and Pitt, 2012).

Environmental factors such as temperature and light significantly influence pathogen development. For instance, fungal growth and sporulation peak around 25–30°C, with conidial germination favored under alternating photoperiods rather than extended darkness, which suggests adaptive flexibility to day night cycles (Xiao *et al*., 2021).

Characterization of *L. theobromae*

L. theobromae (Pat.) Griffon & Maubl., is a key fungal pathogen affecting coconut palms globally. Accurate identification of this pathogen is fundamental for effective disease management. Morphological and cultural characteristics on Potato Dextrose Agar (PDA) reveal initially white colonies that turn grayish to dark brown with abundant aerial mycelium; mature cultures produce pycnidia measuring 0.7–1.0 mm in diameter with aseptate paraphyses. Immature conidia are hyaline, single-celled, and ovoid (approximately 24.92–26.05 × 13.54–15.12 µm), maturing into dark brown, septate, ellipsoid spores exhibiting longitudinal striations caused by melanin granule deposition on the inner wall surface (Ramjegathesh *et al*., 2019; Ploetz *et al*., 2024).

Molecular characterization through amplification and sequencing of the Internal Transcribed Spacer (ITS) region using primers ITS1 and ITS4 has been widely employed. For example, isolates from Bangladesh showed ITS sequences confirming their identity as *L. theobromae* via phylogenetic analyses submitted to GenBank (Al Noman *et al*., 2025). Similarly, in Florida, USA, ITS sequencing of isolates showed 100% identity to *L. theobromae* sequences in GenBank, validating pathogen identification (Ploetz *et al*., 2024). Pathogenicity tests conducted in Tamil Nadu, India, involving pinprick inoculation with conidial suspensions and mycelial plugs on various coconut cultivars resulted in characteristic necrotic leaf lesions, fulfilling Koch's postulates and confirming isolate virulence (Ramjegathesh *et al*., 2019).

Disease Indexing of *L. theobromae*

Disease indexing is a crucial tool used to quantify the extent of infection caused by *L. theobromae* in coconut and other hosts, enabling effective disease monitoring and management. The Percent Disease Index (PDI) is commonly employed, which integrates both disease incidence and severity to provide a comprehensive measure of disease impact in the field. The PDI is calculated using the formula:

$$\text{PDI} = \frac{\text{Sum of numerical ratings}}{\text{Number of leaves examined}} \times \frac{100}{\text{Max. Grade in score chart}}$$

Different studies have used standardized disease rating scales, typically ranging from 0 (no symptoms) to 5 or 9 (severe symptoms) depending on the host and symptom expression (Arunkumar *et al*., 2022). For instance, in coconut, leaf blight severity caused by *L. theobromae* has been assessed on a 0–5 scale based on the percentage of affected leaf area, with PDI values ranging from 10% to 25% reported in South Indian plantations under stress conditions (Agritech TNAU, 2023).

Disease indexing not only assists in evaluating the severity of *L. theobromae* infections but also plays a vital role in assessing the efficacy of fungicides, biocontrol agents, and cultural practices for disease management (Ramjegathesh *et al*., 2019). Regular monitoring through disease indexing allows for timely intervention and mitigation of crop losses.

Pathogen Diversity of *Lasiodiplodia* Spp.

The genus *Lasiodiplodia* (family Botryosphaeriaceae) comprises a complex group of fungal pathogens with considerable genetic and morphological diversity, which contributes to their broad host range and variable pathogenicity. *L. theobromae* is recognized as the most economically important species within this genus, but recent molecular and phylogenetic studies have revealed a rich diversity of cryptic species closely related to *L. theobromae* that are also pathogenic to various crops, including coconut.

Early morphological classification based on colony characteristics, conidial size and shape was often insufficient to accurately differentiate species within *Lasiodiplodia*, leading to misidentifications (Phillips *et al*., 2013). The advent of molecular tools, especially multilocus sequence analyses targeting regions such as ITS, translation elongation factor 1-á (TEF1-á), â-tubulin, and RNA polymerase II subunit (RPB2), has greatly refined species delimitation within the genus (Alves *et al*., 2008; Phillips *et al*., 2013).

For instance, Machado *et al*. (2016) conducted a comprehensive study on *Lasiodiplodia* species infecting coconut in Brazil and identified four distinct species: *L. theobromae*, *L. brasiliense*, *L. pseudotheobromae* and *L. egyptiacae*. Among these, *L. theobromae* was the most prevalent and aggressive, but the presence of other species suggests a complex pathogenic community contributing to disease severity and variability. This diversity has significant implications for diagnosis and management, as species can differ in virulence, host specificity, and fungicide sensitivity (Marin-Felix *et al*., 2019).

Globally, similar patterns of *Lasiodiplodia* diversity have been documented in coconut and other hosts. Studies in Asia, Africa and the Americas report the coexistence of multiple *Lasiodiplodia* species, highlighting the need for molecular identification methods to distinguish closely related pathogens accurately (Arunkumar *et al*., 2022; Burgess *et al*., 2006). Understanding pathogen diversity

is critical for effective quarantine measures and tailored disease control strategies, especially under changing climatic conditions that may favor the emergence or spread of more virulent or fungicide-resistant species.

Role of abiotic stress in Modulating the Virulence of *L. theobromae*

Abiotic stressors such as drought, heat, salinity, and osmotic stress play a pivotal role in modulating the virulence of *L. theobromae* and influencing the outcome of disease development in a wide range of host plants. This fungus is frequently found as an endophyte in asymptomatic plant tissues, but it transitions to a pathogenic phase when the host is physiologically weakened by environmental stress. For example, studies on nutmeg (*Myristica fragrans*) and other woody hosts demonstrate that abiotic factors like waterlogging or prolonged drought predispose plants to disease expression, manifesting as twig dieback, canker formation, and internal stem necrosis symptoms otherwise absent under optimal growing conditions (Pavani *et al*., 2021).

In grapevine (*Vitis vinifera*), the interaction between drought stress and *L. theobromae* infection is highly dependent on the timing of the two events. When fungal inoculation preceded the onset of drought, survival rates decreased by nearly 50%, correlating with altered hormonal signaling, notably elevated jasmonic acid and suppressed malondialdehyde (MDA) levels. In contrast, when drought stress occurred before infection, vines exhibited higher levels of proline and MDA, yet maintained better overall survival, suggesting activation of protective stress-response pathways (Sosnowski *et al*., 2022; Pitt *et al*., 2022).

Heat stress also enhances the virulence potential of *L. theobromae*. Multi-omics analyses have shown that elevated temperatures (35–37°C) induce expression of genes coding for plant cell wall–degrading enzymes, salicylic acid hydroxylases, and components of L-tyrosine catabolism, collectively contributing to increased tissue colonization and disease severity (Yan *et al*., 2019). Transcriptomic profiling under combined heat stress and grapevine wood exposure further reveals metabolic shifts in the pathogen, including upregulation of purine biosynthesis, sterol pathways, and pathogenicity-related enzymes like pectate lyases and hydroxylases (Blanco-Ulate *et al*., 2016; Pitt *et al*., 2016).

Additionally, oxidative and osmotic stress tolerance are critical to *L. theobromae*'s pathogenic fitness. A recent study identified glutathione peroxidase LtGPX3 as a key enzyme required for oxidative stress resistance and virulence in peach. Knockout mutants lacking LtGPX3 were hypersensitive to reactive oxygen species (ROS), showed impaired growth under osmotic stress, and triggered stronger defense responses in host tissues highlighting the role of redox signaling in fungal aggressiveness (Hu *et al*., 2024).

Environmental parameters such as temperature, pH, and osmolarity directly influence the pathogen's growth dynamics. Optimal mycelial growth and sporulation are observed at ~25°C, while extreme cold (e.g., 12°C) significantly restricts development. Moreover, high-sucrose or saline environments mimic osmotic stress and can favor the proliferation of more virulent fungal isolates, potentially explaining the prevalence of *L. theobromae*-associated rot in sugar-rich fruit during ripening or postharvest handling (Yan *et al*., 2022).

Environmental pollution and the incidence of *L. theobromae*

Environmental pollution including heavy metals, soil and water contaminants, and other anthropogenic stressors has emerged as a critical factor influencing the biology and epidemiology of plant-associated fungi. Although *L. theobromae* is widely recognized for its ability to transition from a latent endophyte to an aggressive pathogen under abiotic stress, the role of environmental pollution in modulating this transition is not yet fully understood. However, emerging studies in fungal ecology and environmental microbiology suggest that pollution may indirectly influence pathogen dynamics by altering host physiology, microbial interactions, and fungal adaptability.

Heavy Metals and Soil Pollution

Heavy metal contamination in soils is a well-documented stressor that affects plant health, microbial diversity, and pathogen dynamics. Certain *Lasiodiplodia* species, including endophytic isolates, have demonstrated remarkable tolerance to heavy metals such as cadmium (Cd), lead (Pb), and zinc (Zn). For example, an endophytic *Lasiodiplodia* strain isolated from *Portulaca oleracea* in metal-contaminated soils exhibited significant resistance to heavy metal toxicity and even promoted plant growth under stress conditions (Li *et al*., 2013). While such findings are promising for phytoremediation, they also raise concerns regarding the adaptability of this genus to polluted soils and its potential pathogenic activation in stressed hosts.

Furthermore, *L. theobromae* has shown the capacity to degrade polycyclic aromatic hydrocarbons (PAHs), such as benzo[a]pyrene (BaP), in polluted environments. In vitro experiments have reported degradation rates of up to 53% of BaP within 10 days, a process facilitated by

lignin-degrading enzymes such as laccases and lignin peroxidases (Sarkar *et al*., 2020). This metabolic versatility suggests a high degree of environmental plasticity, which may enhance survival under chemically stressed conditions. Though direct links between heavy metal exposure and disease expression in plants are yet to be established, these biochemical capabilities may facilitate persistence in polluted ecosystems, increasing the likelihood of opportunistic infections.

Water Pollution and Chemical contaminants

While there is limited direct evidence connecting water pollution with increased *L. theobromae* incidence, the broader impacts of pesticide runoff, industrial effluent, and nutrient loading on soil health and plant-pathogen interactions are well established. Agrochemical contamination has been shown to disrupt soil microbial communities, alter nutrient cycling and weaken plant immune responses (Kaur and Mukherjee, 2021). Such disruptions may inadvertently benefit opportunistic pathogens like *L. theobromae* by reducing microbial antagonism or impairing the host's systemic defenses.

Moreover, pollutants that cause oxidative or osmotic stress in plants may increase susceptibility to endophyte-derived infections. While specific experimental data for *L. theobromae* are lacking, parallels can be drawn from other Botryosphaeriaceae pathogens, which are known to exploit stress-compromised tissues for colonization and pathogenesis.

Additional Environmental stressors and pathogenic shifts

Beyond chemical pollution, environmental factors such as drought, waterlogging, and temperature extremes are well-documented inducers of pathogenicity in *L. theobromae*. Observations from nutmeg (*Myristica* fragrans) plantations in Kerala, India, revealed heightened incidence of twig dieback and canker formation during periods of abiotic stress, particularly prolonged drought or flooding (Pavani *et al*., 2021). These findings underscore the fungus's opportunistic lifestyle, with stress conditions acting as triggers for its transition from endophyte to pathogen.

In this context, it is plausible that environmental contaminants by imposing physiological stress on plants and altering soil microbial equilibrium may create conditions conducive to latent infection and disease outbreak. This hypothesis aligns with the broader understanding of how environmental stress modifies plant-pathogen dynamics, particularly in woody perennials and tropical fruit crops.

Predisposing factors for *L. theobromae* infection: The role of Hybrid vigor, Plant physiology and Nutrient deficiencies

L. theobromae, is a latent endophyte and opportunistic pathogen affecting a broad range of woody and horticultural plants. While environmental factors like drought, heat and mechanical injury are known triggers of pathogenicity, plant intrinsic factors such as hybrid vigor, physiological health, and nutrient status also play critical roles in disease susceptibility. This review explores these predisposing host factors and their mechanistic influence on *L. theobromae* infection dynamics.

Low Hybrid vigor and Genetic vulnerability

Hybrid vigor, or heterosis, is the phenomenon where hybrid offspring display superior traits compared to their parental lines, including enhanced growth, yield, stress tolerance, and disease resistance (Chen, 2013). Conversely, plants with low hybrid vigor often due to inbreeding or lack of genetic diversity tend to exhibit compromised physiological robustness and immune competence. Although, no direct studies currently establish a causal link between hybrid vigor and susceptibility to *L. theobromae*, it is well understood in plant pathology that genetic uniformity can reduce a plant's resilience to opportunistic pathogens. In monocultures or genetically narrow breeding lines, this vulnerability may enhance disease outbreaks under stress.

Weakened physiological state and host susceptibility

The physiological status of the host plant is a critical determinant of its susceptibility to infection. Stresses such as senescence, drought, excessive pruning, and prior pathogen damage can weaken plant tissues and reduce their capacity to resist or contain fungal invasion. For instance, in grapevine, *L. theobromae* infection has been associated with significant physiological alterations including a decline in chlorophyll fluorescence and an increase in reactive oxygen species (ROS) even in asymptomatic tissues (Valencia-López *et al*., 2019). Such physiological impairment compromises photosynthetic efficiency and antioxidant capacity, thereby facilitating the colonization of host tissues by the fungus.

Furthermore, *L. theobromae* has been shown to actively exploit stressed tissues, often remaining latent until host defenses are weakened. In mango, cocoa, and nutmeg, environmental stressors like drought or flooding have been documented to trigger symptom expression in previously asymptomatic infections (Slippers and Wingfield, 2007; Pavani *et al*., 2021).

Nutrient and Mineral deficiencies as facilitators of infection

Micronutrients and macronutrients are vital for plant metabolism, cell wall integrity, enzyme function, and immune signaling pathways. Deficiencies in key nutrients such as nitrogen (N), phosphorus (P), potassium (K), calcium (Ca), *magnesium* (Mg), iron (Fe) and zinc (Zn) are known to compromise plant structural defenses and immune responses (Marschner, 2012). Although specific studies on *L. theobromae* in nutrient-deficient hosts are limited, extrapolation from broader plant–pathogen systems suggest several plausible pathways by which nutritional stress predisposes plants to fungal infection:

Nitrogen deficiency reduces chlorophyll content and photosynthetic efficiency, leading to general plant debilitation and lower synthesis of defense proteins. Calcium deficiency disrupts cell wall stability and membrane integrity, weakening the plant's first line of defense against pathogen ingress. Iron and magnesium deficiencies impair energy metabolism and reduce the plant's capacity to produce reactive oxygen species and phytoalexins key elements of pathogen response. Micronutrient imbalances, particularly in zinc, boron, and copper, affect enzyme activity and hormone signaling, potentially altering defense gene expression.

Integrated Mechanisms of Susceptibility

The interaction between low hybrid vigor, weakened physiological state, and nutrient imbalance creates a synergistic vulnerability that can be exploited by *L. theobromae*. Several mechanistic processes underpin this susceptibility

Impaired Immune Signaling: Nutritional deficits, particularly in micronutrients, can reduce biosynthesis of critical defense hormones such as salicylic acid, jasmonic acid, and ethylene.

Compromised Structural Barriers: Calcium and boron deficiencies reduce the mechanical strength of cell walls, making tissues more susceptible to enzymatic degradation by fungal cell wall-degrading enzymes.

Energy Constraints and Metabolic Imbalance: Plants under nutrient or oxidative stress have reduced ATP availability, impairing defense-related processes such as ROS production, phytoalexin synthesis, and callose deposition. Similar energy-related constraints were demonstrated in fruit crops, where energy-depleted conditions accelerated pathogen invasion (Lin *et al*., 2017).

Delayed or Inadequate Stress Responses: Physiologically stressed plants may exhibit delayed activation of antioxidant enzymes and pathogenesis-related proteins, allowing the pathogen to establish and spread before effective resistance can be mounted.

Mode of entry and infection process for *L. theobromae* pathogenesis

L. theobromae is a widespread, polyphagous fungus known for its dual lifestyle as both a latent endophyte and an aggressive necrotrophic pathogen. Its infection process is highly opportunistic, often triggered by environmental or physiological stress in the host. The fungus infects a wide range of woody and horticultural crops, including grapevine, mango, coconut, papaya, and nutmeg, causing symptoms such as stem cankers, dieback, fruit rot, and post-harvest decay (Slippers and Wingfield, 2007; Pavani *et al*., 2021).

Mode of Entry into the Host

The initial entry of *L. theobromae* into plant tissues generally occurs through wounds which may be caused by pruning, mechanical injury, insect damage, or environmental factors such as wind or hail. The fungus can also penetrate through natural openings, such as lenticels, stomata, or abscission scars, particularly in fruits and young shoots (Úrbez-Torres *et al*., 2010). In many host species, *L. theobromae* behaves as an endophyte, colonizing tissues without inducing symptoms until stress or senescence triggers a shift to its pathogenic phase.

Infection process and Tissue colonization

Following entry, the pathogen rapidly colonizes intercellular and vascular tissues, secreting a suite of cell wall degrading enzymes such as cellulases, pectinases, xylanases, and laccases that facilitate tissue maceration and penetration. The fungus also produces phytotoxic metabolites, including jasmonic acid analogs and aromatic compounds, which disrupt host metabolism and accelerate cell death (Yan *et al*., 2018). Colonization of the vascular system impairs water and nutrient transport, resulting in wilting, dieback and canker formation.

Molecular and transcriptomic studies have shown that *L. theobromae* modulates its gene expression based on host cues and environmental conditions. Pathogenicity-related genes are upregulated in response to heat, drought, and host-derived signals (Rampadarath *et al*., 2021). In particular, genes encoding salicylic acid–degrading proteins and detoxification enzymes enable the fungus to suppress host immune responses and tolerate oxidative stress during infection (Salvatore *et al*., 2020).

Favorable conditions for Disease development

The pathogenic shift of *L. theobromae* from a quiescent endophyte to an aggressive invader is highly

dependent on environmental and physiological stress in the host. Key favorable conditions include:

High temperature and humidity: The fungus thrives in warm, humid environments (25–35/ °C), with optimal mycelial growth and sporulation occurring under such conditions (Slippers *et al*., 2009).

Drought or water stress: Host water deficit has been shown to increase susceptibility. In grapevines, pre-inoculation drought stress reduced plant survival by 50%, accompanied by physiological shifts favoring fungal colonization (Yan *et al*., 2021).

Wounding and pruning events: Mechanical damage creates direct entry points and reduces the host's ability to compartmentalize infection.

Nutritional deficiencies: Mineral imbalances, particularly involving calcium, magnesium, and micronutrients, impair structural and immune defenses, facilitating tissue invasion (Marschner, 2012).

Post-harvest conditions: The fungus frequently causes rot during fruit storage and transportation, particularly when fruits are bruised or stored under poor sanitary conditions. In papaya, for example, stem-end rot caused by *L. theobromae* can reach incidences as high as 80% under high humidity storage (EFSA, 2020).

Latent Infection and Disease Reactivation

An important epidemiological feature of *L. theobromae* is its ability to persist in plant tissues as a latent infection, only becoming pathogenic when triggered by environmental cues or host weakening. Slippers and Wingfield (2007) described this phenomenon as "opportunistic pathogenesis," in which the fungus switches from a silent endophytic phase to active colonization upon sensing stress signals. This allows the pathogen to remain undetected for long periods, contributing to its wide distribution and significant economic impact in both field and post-harvest contexts.

Pathogenicity of *L. theobromae*: A Facultative Parasitic Strategy in Plant–pathogen interactions

L. theobromae (Pat.) Griffon & Maubl., is a globally distributed, opportunistic fungal pathogen with a broad host range, including economically important crops such as grapevine (*Vitis vinifera*), mango (*Mangifera indica*), coconut (*Cocos nucifera*), papaya (*Carica papaya*), nutmeg (*Myristica fragrans*), and several forest species (Slippers & Wingfield, 2007; EFSA Panel on Plant Health [PLH], 2020). The fungus is characterized by its facultative parasitism existing as a latent endophyte under non-stressful conditions and transitioning to a necrotrophic pathogen under conducive environmental or host-related stress.

Facultative Parasitism and Latency

Unlike obligate pathogens, *L. theobromae* does not rely exclusively on living host tissues for survival. It colonizes asymptomatic plant tissues as an endophyte, remaining quiescent until stress conditions in the host, such as wounding, drought, heat, or nutrient deficiency, trigger a pathogenic switch (Slippers and Wingfield, 2007; Pavani *et al*., 2021). This facultative lifestyle provides a strategic advantage, allowing the fungus to persist undetected within the host and exploit weakened tissues when defenses are compromised.

Infection Strategy and Virulence Factors

Pathogenicity in *L. theobromae* is primarily driven by its arsenal of virulence factors, including cell wall–degrading enzymes (CWDEs), secondary metabolites, and detoxifying enzymes. The fungus secretes cellulases, pectinases, xylanases and laccases to breach host cell walls and facilitate tissue invasion (Salvatore *et al*., 2020). Concurrently, the production of phytotoxins such as jasmonic acid analogs and phenolic compounds interferes with host cellular signaling and induces programmed cell death, supporting its necrotrophic colonization strategy (Yan *et al*., 2018).

Recent transcriptomic and multi-omics studies have revealed that *L. theobromae* dynamically modulates gene expression in response to abiotic stress and host-derived cues. Genes encoding detoxifying enzymes, heat shock proteins, and salicylic acid–degrading enzymes are upregulated under stress, enabling the fungus to suppress host immunity and adapt to hostile environments (Rampadarath *et al*., 2021). These adaptive mechanisms underline its plasticity as a facultative parasite capable of fine-tuning virulence according to environmental context.

Host response and Tissue susceptibility

The host response to *L. theobromae* is often inadequate when physiological or environmental stress weakens plant immunity. For instance, drought-stressed grapevines exhibit reduced photosynthetic activity, disrupted redox homeostasis, and delayed activation of defense-related enzymes, making them more susceptible to infection (Valencia-López *et al*., 2019). In nutmeg trees, stem necrosis caused by *L. theobromae* is significantly exacerbated under abiotic stress, including waterlogging and drought, indicating the pathogen's reliance on host weakening for successful colonization (Pavani *et al*., 2021).

Moreover, calcium and boron deficiencies have been

implicated in the breakdown of structural defenses, particularly cell wall integrity, further facilitating pathogen ingress (Marschner, 2012). Such interactions exemplify the critical role of plant physiology in determining the outcome of infection.

Ecological and Economic implications

The facultative nature of *L. theobromae* has significant ecological and epidemiological implications. Its ability to remain dormant in host tissues or survive as a saprophyte on dead plant material contributes to long-term persistence in agricultural and natural ecosystems. Under favorable environmental conditions such as high humidity, elevated temperatures, and increased incidence of pruning or mechanical injury latent infections can lead to aggressive outbreaks, often with devastating effects on crop yield and quality (EFSA Panel on Plant Health [PLH], 2020).

Recent advances in Managing *L. theobromae*

L. theobromae continues to pose a significant threat to horticultural and woody crops worldwide. Its ability to act as both an endophyte and an aggressive necrotroph under stress conditions complicates management strategies. Recent international innovations span molecular diagnostics, biocontrol agents, novel fungicides, and integrated production approaches, offering promising avenues for mitigation and future research.

Molecular Insights into Fungicide resistance

The increasing resistance of *L. theobromae* to conventional fungicides highlights the urgent need for molecular-level insights. In a recent study, He *et al.* (2024) investigated the mechanisms underlying resistance to prochloraz in *L. theobromae* isolates from mango. Their analysis identified specific genetic mutations and altered gene expression patterns linked to reduced fungicide sensitivity. These findings not only deepen our understanding of resistance development in this pathogen but also support the advancement of next-generation fungicides and tools for monitoring resistance.

Biocontrol agents from Diverse origins

Biological control has emerged as a sustainable alternative to chemical fungicides. *Bacillus velezensis* strains, isolated from avocado rhizospheres, have demonstrated strong antagonistic activity against *L. theobromae* in vitro and in field conditions. In trials, application of these strains reduced the incidence of branch blight by up to 74%, attributed to lipopeptide-mediated suppression of spore germination (Li *et al.*, 2024). Concurrently, the use of *Trichoderma hamatum* an endophytic and antagonistic fungus has shown effective suppression of *L. theobromae* infections in macadamia, validating its potential as a biocontrol agent in tropical tree crops (Li *et al.*, 2022).

Chemical innovations: New Fungicidal compounds

Chemical control remains a cornerstone in disease management, though innovation is needed to overcome resistance and minimize environmental impact. A novel sulfone derivative (designated JHXJZ) has demonstrated potent antifungal activity against *L. theobromae*. Its mechanism involves disruption of ergosterol biosynthesis and alteration of hyphal morphology, offering a distinct mode of action from conventional fungicides and presenting a candidate for development in integrated disease management programs (Dong *et al.*, 2021).

Integrated Bioproduction platforms

In a novel development, *L. theobromae* is being investigated for its potential in industrial applications. Marchioro *et al.* (2025) reported that an endophytic strain (MMPI) was successfully utilized for the sustainable production of both mycoprotein and extracellular β-glucan. This dual-purpose strategy capitalizes on the fungus's metabolic versatility, offering valuable applications in the food and pharmaceutical industries, while also encouraging further research into its ecological dynamics under controlled conditions.

Traditional Management reinforced by Empirical research

Traditional disease control methods continue to be refined through scientific validation. A Ghanaian study investigated the efficacy of both chemical and plant-based biopesticides against mango decline caused by *L. theobromae*. While systemic fungicides such as carbendazim and copper-based formulations (e.g., funguran) showed efficacy, notable inhibitory effects were also observed for botanical extracts from *Chromolaena odorata*, *Azadirachta indica*, and *Carica papaya*, offering eco-friendly alternatives particularly relevant for smallholder and organic farming systems (Ablormeti *et al.*, 2021).

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