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STEM BLEEDING IN COCONUT: UNRAVELING PATHOPHYSIOLOGY, MOLECULAR DIALOGUES, AND SMART MANAGEMENT FRONTIERS – A GLOBAL PERSPECTIVE

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ABSTRACT

Stem Bleeding Disease (SBD) of coconut, primarily caused by *Thielaviopsis paradoxa*, is a major constraint in coconut cultivation across India and several tropical regions. The pathogen invades through wounds or natural cracks, thriving under high soil moisture and organic-rich soils. It produces a suite of enzymes, including polygalacturonases, cellulases, and pectinases, that degrade cell walls, leading to external bleeding, internal tissue maceration, canopy wilting, and nut drop. Coconut palms respond with limited accumulation of phenolics and lignin, yet these defenses are often insufficient. Recent research highlights the utility of enzyme-based treatments, such as chitinases and β-1,3-glucanases, which curtail fungal spread and enhance host resistance. Nanotechnology-enabled delivery systems particularly chitosan-based azole formulations and biosynthesized silver or metal oxide nanoparticles offer targeted control with reduced environmental risk. Eco-compatible strategies involving salicylic acid signaling, organic acids, siderophore-producing rhizobacteria, and bioactive metabolites like harzianic acid have shown promise in suppressing *T. paradoxa* while promoting host recovery. This review synthesizes recent insights into pathogen biology, host-pathogen interactions, and sustainable management avenues for durable control of SBD in coconut

***Key words*:** Stem Bleeding, *Thielaviopsis paradoxa*, Coconut, Etiology, Symptomatology, Disease Physiology, Host–Pathogen Biochemical Interactions, Non-Conventional Disease Management Strategies

Introduction

Coconut (*Cocos nucifera* L.), a vital crop in tropical regions, is extensively cultivated in India, with 23.33 lakh ha yielding 153.3 lakh metric tonnes in 2023–24 and a productivity of 9,346 nuts/ha (CDB, 2024); Andhra Pradesh alone produces 11.8 lakh metric tonnes annually, with high yields in Srikakulam and Konaseema and a GVO increase from Rs. 1,814 crore in 2018–19 to Rs. 2,763 crore in 2022–23 (AP Horticulture Dept., 2023; CDB, 2024). Stem bleeding disease, caused by *Ceratocystis paradoxa* (*Thielaviopsis paradoxa*), poses a major threat, with host resistance linked to the phenylpropanoid pathway and hormone-mediated immunity (Dixon *et al.*, 2002; Glazebrook, 2005; Pieterse *et al.*, 2012). Molecular diagnostics such as PCR, qPCR, LAMP, and UAV-based AI aid in early detection (Nandini *et al.*, 2020; Purnamasari *et al.*, 2021; Saad *et al.*, 2022), while transcriptomics reveal expression changes in genes related to cell wall degradation and oxidative stress (Govindarajulu *et al.*, 2020; Zhao *et al.*, 2013). Biocontrol agents including *Trichoderma* spp., *Pseudomonas fluorescens*, and *Bacillus subtilis* combat the pathogen *via* enzymatic and induced resistance mechanisms (Jayaraj *et al.*, 2005; Snehalatharani *et al.*, 2016; Rajeela *et al.*, 2017), and endophytes from Latin America offer additional potential (Gaitán *et al.*, 2021). Organic amendments enhance soil microbial balance and suppress disease (Sundararaj *et al.*, 2012; Gunathilake *et al.*, 2022), and climate-driven forecasting and DSS tools are being developed for integrated disease management (Jayaweera *et al*., 2023).

Etiology and Taxonomy of *Thielaviopsis paradoxa* (*Ceratocystis paradoxa)*

Stem Bleeding Disease (SBD) is a chronic and economically significant vascular wilt condition that affects mature coconut palms, particularly in India, Sri Lanka, and other coconut-growing regions of Southeast Asia. The disease is caused by *Ceratocystis paradoxa* (Ascomycota: Microascales), a soil- and wound-borne fungus known for its hemibiotrophic lifestyle and its association with other tropical crops such as pineapple and sugarcane (*Nandini et al., 2020*; *Gunathilake et al., 2022*).

Etiology

T. paradoxa, a soil-borne fungal pathogen, is the causal agent of stem bleeding disease in coconut, a condition characterized by the exudation of dark brown to black gummy fluid from cracks on the trunk surface, particularly near the base. The pathogen enters through wounds and colonizes the vascular tissues, leading to internal tissue disintegration and decline in coconut productivity. Environmental stress, poor drainage, and mechanical injuries further predispose palms to infection. Microsclerotia and chlamydospores of *T. paradoxa* enhance its survival in soil, making management challenging. The disease is of economic significance in several tropical coconut-growing regions due to its impact on palm health and yield (Sundram *et al*., 2015).

Taxonomy

Thielaviopsis paradoxa (De Seynes) Hohn., the causal agent of stem bleeding disease in coconut , is a member of the phylum Ascomycota, class Sordariomycetes, order Microascales, and family Ceratocystidaceae. It is the asexual morph (anamorph) of *Ceratocystis paradoxa*, which produces both phialoconidia and aleurioconidia, facilitating its dissemination through soil and air. The sexual (teleomorphic) stage forms perithecia with long necks and hat-shaped ascospores. Historically known under its anamorphic name, *T. paradoxa* is still widely cited in older literature. Advances in molecular phylogenetics, particularly through multilocus sequencing of ITS, β-tubulin, and TEF1-α gene regions, have clarified its taxonomic position and revealed its inclusion within the *C. paradoxa* species complex, a group characterized by broad host ranges and a predominantly tropical distribution. This pathogen is a wound invader, infecting through injuries and causing internal tissue decay and the characteristic bleeding lesions on coconut trunks, making it a significant constraint in tropical coconut production systems (Paul *et al*., 2020; Nandini *et al*., 2020; Purnamasari *et al*., 2021; Saad *et al*., 2022).

Identification of *Ceratocystis paradoxa*

Identification of *Ceratocystis paradoxa*, the causative agent of stem bleeding disease in coconut, has evolved from reliance on morphological characters—especially aleurioconidia and phialoconidia in its asexual stage, formerly named *Thielaviopsis paradoxa*—to integrative approaches due to morphological plasticity and misidentification risks (Harrington, 2000; Isaac *et al*., 2015). Molecular phylogenetic tools, particularly multilocus sequencing of ITS, β-tubulin (TUB), and TEF1-á, have significantly refined species delimitation and resolved cryptic species boundaries (Barnes *et al*., 2005; Nandini *et al*., 2020). Field isolates from infected coconut xylem and stem tissues in India and Sri Lanka have consistently confirmed the presence of *C. paradoxa* through molecular diagnostics (Mohandas *et al*., 2012; Gunathilake *et al*., 2022). Phylogenetic analyses also suggest notable genetic diversity among isolates across agro-climatic zones, and the presence of related lineages in cacao, durian, and arecanut implies potential cross-host adaptability (Van Wyk *et al*., 2013; Purnamasari *et al*., 2021). Accurate identification now depends on integrative taxonomy combining morphology, histopathology, and DNA sequencing, supported by tools like species-specific PCR primers and LAMP assays for early detection of latent infections (Nandini *et al*., 2020; Purnamasari *et al*., 2021). However, further global phylogenetic surveys are essential to fully resolve the species complex and its epidemiological dynamics.

Disease Distribution

Stem bleeding disease, caused by the opportunistic wound pathogen *T. paradoxa* (Ascomycota: Microascales), is an economically important and increasingly reported disorder of coconut in India and several tropical regions. Stem bleeding is characterized by external exudation of a dark reddish-brown fluid from the lower stem, often leading to progressive tissue necrosis, internal rotting, and eventual palm decline (Sankaralingam & Sundararaju, 2015; Anith *et al*., 2019). Stem bleeding disease of coconut is a chronic and economically damaging trunk disease widely distributed across tropical and subtropical coconut-growing regions. The disease is especially prevalent in South and Southeast Asia including India, Sri Lanka, Indonesia, and the Philippines where incidence in severely affected plantations can range from 10% to 40%, particularly under drought stress, aging palm stands, and poor soil health (Sankaralingam & Sundararaju, 2015; Nair & Rajendran, 2010; Flood *et al*., 2000). In Sri Lanka, over 30% of palms in older estates are affected (Perera *et al*., 2010), while in India, annual economic losses are estimated at [1] 50–100 crores (USD 6–12 million) (Sankaralingam & Sundararaju, 2015). The disease has also been recorded in West Africa, Central America, Malaysia, and sporadically in the Pacific Islands, although underreporting and misdiagnosis are common in less-studied regions (Pilotti, 2005; Thorpe *et al*., 2005). Infected palms typically suffer yield reductions of up to 40%, poor copra quality, and premature death (Nampoothiri *et al*., 2003; Rajendran *et al*., 2003). Abiotic stressorssuch as prolonged dry spells, sandy soils, poor drainage, and monoculture practices—further exacerbate disease severity (Rees *et al*., 2012; Abdullah *et al.*, 2017).

In India, the disease is a major constraint in traditional coconut-growing states. Kerala reports the highest incidence (15–25%), particularly in districts like Thrissur, Palakkad, Malappuram, and Kollam due to sandy soils and older palms (Kumar *et al*., 2012). In Tamil Nadu, incidence ranges from 10–15% in Coimbatore, Pollachi, and Thanjavur, where water stress and collar injuries are major contributing factors (Rao *et al*., 2025). Andhra Pradesh reports 10–12% incidence in East and West Godavari and Visakhapatnam, especially in poorly drained fields with mechanical trunk damage (Rao *et al.*, 2025). In Karnataka, particularly in Dakshina Kannada and Udupi, incidence ranges from 6–10%, often linked to fluctuating soil moisture and root injury during ploughing (Sundararaj *et al.,* 2020). Sporadic outbreaks (<5%) are observed in Goa and coastal Maharashtra, while Odisha shows emerging hotspots in Puri and Ganjam districts. These regional patterns, combined with increasing climate variability, highlight the urgent need for site-specific surveillance, early diagnosis, and integrated disease management approaches (Mohandas *et al*., 2012; Nelson, 2008).

Epidemiology

T. paradoxa survives in soil, infected plant debris, and crop residues as chlamydospores and conidia, which act as primary inocula under favorable environmental conditions (Nelson, 2008; Anith & Nandini, 2020). The pathogen primarily infects coconut palms through wounds at the trunk or root collar, caused by mechanical damage, insect activity such as *Oryctes rhinoceros*, or poor agronomic practices. It colonizes vascular tissues, impeding translocation and inducing the characteristic stem bleeding symptoms (Nandini *et al*., 2020). Disease progression is promoted by high humidity (80–90%), optimal temperatures of 28–32°C, and poor soil drainage, with peak incidence during monsoon seasons in dense, overaged plantations with limited canopy ventilation (Sankaralingam & Sundararaju, 2015; Rajeev *et al*., 2018). While the pathogen thrives in sandy loam and lateritic soils, disease severity is exacerbated in clay-rich soils due to inadequate aeration and delayed wound healing (Nampoothiri *et al*., 2003). Though not truly systemic, infections can spread locally, leading to significant tissue decay and palm mortality. In India, the disease is commonly found in coastal regions of Kerala, Karnataka, Tamil Nadu, and Andhra Pradesh, where salt stress, high rainfall, and nutrient deficiencies prevail (Anith *et al*., 2019; CDB, 2023). Early symptoms are often misdiagnosed, and in advanced stages, palms exhibit necrotic lesions, bark splitting, reduced nut yield, crown thinning, and are frequently compromised by secondary pathogens such as *Aspergillus* or *Fusarium*.

Soil Conditions, Moisture, and Pathogen Virulence

Stem bleeding disease, caused by *T. paradoxa*, continues to pose a serious constraint to coconut productivity in tropical regions. Recent studies emphasize that the disease's progression and pathogen virulence are strongly modulated by soil physical conditions, moisture dynamics, and rhizospheric microbial interactions. An experimental soil-pathogen interaction study by Rao *et al*., (2025) sheds light on the ecological preferences of *T. paradoxa* under variable soil regimes.

Enhanced survival under low-moisture regimes

In a controlled trial assessing soil moisture from 20% to 80% of field capacity, *T. paradoxa* demonstrated increased survival and infection efficiency in drier conditions (20–60%), where infection rates were markedly higher. This resilience is linked to the formation of melanized chlamydospores, which enhance desiccation tolerance and facilitate latent colonization (Rao *et al*., 2025). Similar desiccation-resistant survival strategies have been noted for *Ceratocystis* species infecting tropical palms and bananas (Thorpe *et al*., 2005; Nelson, 2008).

Soil texture and root contact as infection drivers

Sandy and sandy loam soils were found to support greater root penetration and more frequent contact with residual inoculum, resulting in higher infection incidences (≈93.6%), compared to clay-heavy soils (≈76.4%) that limit pathogen spread due to reduced porosity and oxygen diffusion (Rao *et al*., 2025). These findings align with earlier observations that *T. paradoxa* thrives in wellaerated, porous soils, particularly where organic debris and root injuries are prevalent (Kumar *et al.*, 2012).

Reduced microbial antagonism in nutrient-poor soils

Pathogen proliferation was pronounced in nutrient-deficient sandy soils, which typically harbor fewer antagonistic microbes. This ecological vacuum allows *T. paradoxa* to dominate the rhizosphere with minimal biocontrol pressure. In contrast, organically enriched loam soils supported microbial consortia such as *Trichoderma harzianum*, *Pseudomonas fluorescens*, and *Bacillus subtilis*, known for their antagonistic action through competition, antibiosis, and mycoparasitism (Sundararaj *et al.*, 2020; Kandan *et al.*, 2010). These microbial populations play a crucial role in suppressing soil-borne pathogens and limiting disease outbreaks

Favorable Microclimate

The incidence and severity of stem bleeding disease in coconut, caused by *Thielaviopsis paradoxa*, are closely linked to microclimatic stress, particularly in sandy, thermo-hydrically variable soils. These soils promote fluctuating moisture regimes that exacerbate physiological stress in coconut roots and lower their natural defense thresholds. According to Rees *et al.* (2012), cyclic wet-dry stress reduces lignification and compromises cell wall integrity, creating conducive entry points for necrotrophic pathogens like *T. paradoxa*. Concurrently, such stress conditions have been shown to enhance the expression of fungal virulence factors, including cell wall-degrading enzymes (CWDEs) such as polygalacturonases and peroxidases that facilitate tissue maceration (Abdullah *et al.*, 2017).

Adaptation of pathogen to limited moisture environments

T. paradoxa also exhibits facultative necrotrophy and has shown the ability to persist in moderately dry environments by forming thick-walled chlamydospores that can tolerate desiccation (Rao *et al.*, 2025). Comparative studies on pathogen behavior under controlled soil moisture conditions indicate higher colonization rates and symptom expression in dry, porous soils (Kumar *et al.*, 2012), while waterlogged or clay-rich soils with poor aeration tend to suppress disease progression by limiting oxygen availability and favoring antagonistic microbial populations (Thorpe *et al.*, 2005).

Implications for site-specific management

Recent insights highlight that the risk of stem bleeding is significantly modulated by soil type, moisture, and rhizospheric microbial dynamics (Sundararaj *et al.*, 2020). Therefore, disease suppression strategies must address both host susceptibility and environmental conditions.

Effective interventions

Avoiding prolonged water stress in young coconut plantations through regulated drip irrigation and moisture conservation techniques. Enriching sandy soils with organic amendments to enhance water retention and microbial competitiveness. Incorporating biocontrol agents such as *Trichoderma* harzianum and Pseudomonas fluorescens into nursery and main field soils to counteract *T. paradoxa* via antagonism and mycoparasitism (Kandan *et al.*, 2010).

Disease Symptoms and Progression

Stem bleeding disease of coconut, primarily caused by *T. paradoxa*, presents as a chronic trunk and vascular infection that significantly reduces palm health and yield in tropical regions. Initially reported in countries like India, Sri Lanka, the Philippines, Indonesia, and parts of East Africa and the Pacific Islands, the disease is increasingly recognized for its gradual but severe impact (Flood *et al.*, 2000; Pilotti, 2005). Early symptoms include dark brown to reddish-brown gummy exudates near the trunk base, resulting from vascular colonization and necrosis by the pathogen (Nampoothiri *et al.*, 2003). As the disease progresses, bark tissue deteriorates, forming sunken lesions and cracks (Sankaralingam & Sundararaju, 2015). Secondary effects include leaf yellowing, frond drooping, premature nut fall, and crown thinning due to disrupted water and nutrient transport (Mohandas *et al.*, 2012; Rajendran *et al.*, 2003). In severe cases, unopened spindle leaves and palm death occur (Perera *et al.*, 2010). Disease progression is slow, often taking years to manifest fully, complicating early detection. Stress conditions such as drought, waterlogging, and mechanical or pest-induced wounds exacerbate susceptibility, highlighting the importance of proactive monitoring and early intervention (Flood *et al.*, 2000; Nair & Rajendran, 2010).

Disease Physiology and Host–Pathogen Biochemical Interactions

Entry and Colonization

Stem bleeding disease in coconut, caused by *T. paradoxa*, initiates when the pathogen invades through wounds or natural fissures near the collar region, often resulting from mechanical damage, insect boring, or root pruning in compacted or poorly drained soils (Sankaralingam & Sundararaju, 2015). As a facultative saprophyte, it persists in soil and plant debris as chlamydospores or mycelium under humid, waterlogged conditions (Flood *et al.*, 2000; Nampoothiri *et al.*, 2003). Upon infection, the pathogen colonizes cortical and vascular tissues, releasing cell wall-degrading enzymes such as cellulases, hemicellulases, and pectinases, thereby impairing translocation and triggering the characteristic dark reddish-brown bleeding from basal stem cracks (Mohandas *et al*., 2012; Rajendran *et al*., 2003). Oxalic acid secretion further intensifies tissue damage by acidifying cells and enhancing enzyme activity, while host responses like phenolic production and PR proteins are often insufficient under environmental stress (Saharan *et al*., 2018; Nair & Rajendran, 2010; Mohandas *et al*., 2012). The disease progresses insidiously, with visible symptoms such as bleeding patches, foliar yellowing, nut fall, and canopy thinning manifesting only after extensive internal damage, eventually leading to chronic decline or palm death if unmanaged (Perera *et al*., 2010; Sankaralingam & Sundararaju, 2015).

Enzymes produced by *Thielaviopsis paradoxa*

The pathogenicity of *Thielaviopsis paradoxa*, the causal agent of stem bleeding disease in coconut, is primarily associated with its production of cell wall-degrading enzymes that facilitate tissue colonization and disease progression. *T. paradoxa* is an ascomycete that causes necrosis and decay through enzymatic breakdown and metabolic interference (Sankaralingam & Sundararaju, 2015; Mohandas *et al*., 2012). Upon infection, it secretes hydrolytic enzymes such as cellulases, hemicellulases, pectinases, and â-glucosidases, which degrade cellulose, hemicellulose, and pectin in the stem, leading to cortical and vascular collapse (Rajendran *et al*., 2003; Mohandas *et al*., 2012). Polygalacturonases and xylanases further degrade pectic and xylan components, contributing to bleeding lesions, leaf chlorosis, nut fall, and canopy thinning (Nampoothiri *et al*., 2003; Sankaralingam & Sundararaju, 2015). Oxalic acid, a key virulence factor, acidifies the infection site, chelates calcium, and enhances enzymatic activity while suppressing host defenses (Saharan *et al*., 2018). Although coconut palms produce phenolics, peroxidases, and pathogenesis-related (PR) proteins as defense responses, these are often inadequate against the pathogen's enzymatic arsenal (Nair & Rajendran, 2010; Mohandas *et al*., 2012). Histopathological studies reveal widespread necrosis and vascular blockage, with visible symptoms manifesting only during advanced stages of infection (Perera *et al*., 2010; Rajendran *et al*., 2003).

Impact on water and nutrient transport in disease affected coconut palms

A major physiological consequence of stem bleeding disease in coconut, caused by *Thielaviopsis paradoxa*, is the disruption of water and nutrient transport due to enzymatic degradation of xylem vessels by cellulases, pectinases, and xylanases, leading to vessel blockage and reduced hydraulic conductivity (Rajendran *et al*., 2003; Mohandas *et al*., 2012; Sankaralingam & Sundararaju, 2015). Histological studies reveal cortical disintegration and vascular necrosis with occluded xylem, impairing water and mineral flow and resulting in symptoms like leaf yellowing, frond drooping, and nut fall (Mohandas *et al*., 2012; Nampoothiri *et al*., 2003). Nutrient uptake, particularly of nitrogen, potassium, and magnesium, declines due to fine root damage and blocked vascular pathways, while oxalic acid secretion disrupts pH and ion transport, compounding physiological stress and systemic decline (Nair & Rajendran, 2010; Saharan *et al*., 2018).

Lignification and callose deposition: host responses in disease infected coconut palms

In response to *T. paradoxa* infection, coconut palms initiate structural defenses like lignification and callose deposition to contain stem bleeding disease; lignin fortifies cell walls via upregulated PAL, peroxidases, and laccases, while callose blocks hyphal spread through sieve plates and plasmodesmata (Mohandas *et al*., 2012; Nambiar & Mohandas, 2015). These barriers limit pathogen enzymes cellulases, pectinases, and oxalic acid from causing extensive tissue damage (Rajendran *et al*., 2003; Saharan *et al*., 2018). However, in later stages, the pathogen's enzymatic aggression overwhelms host defenses, enabling systemic colonization and palm decline (Sankaralingam & Sundararaju, 2015).

Host Responses: Antioxidant Enzyme Production (Peroxidase, Catalase)

Coconut palms infected by *T. paradoxa* activate antioxidant enzymes like peroxidase (POX) and catalase (CAT) to counteract oxidative stress from elevated reactive oxygen species (ROS), especially H_2O_2, generated during stem bleeding disease progression (Mohandas *et al*., 2012; Nambiar & Mohandas, 2015). POX aids both in ROS detoxification and lignin biosynthesis for cell wall reinforcement (Saharan *et al*., 2018), while CAT converts H_2O_2 to water and oxygen, minimizing tissue damage (Nair & Rajendran, 2010). Higher POX and CAT levels in moderately resistant varieties indicate their role in tolerance, but in advanced stages, excessive ROS, fungal toxins, and enzymatic action overwhelm defenses, causing vascular collapse and palm death (Flood *et al*., 2000; Sankaralingam & Sundararaju, 2015).

Role of phenolics and secondary metabolites in stem bleeding affected coconut palms

In *T. paradoxa*-infected coconut palms, increased biosynthesis of phenolic compounds and secondary metabolites such as flavonoids, tannins, and lignin precursors contributes to antifungal activity and cell wall reinforcement *via* lignification (Mohandas *et al*., 2012; Nambiar & Mohandas, 2015). These defenses, activated by PAMPs and regulated through salicylic and jasmonic acid pathways, disrupt fungal membranes, inhibit enzymes, and trigger hypersensitive responses (Flood *et al*., 2000; Saharan *et al*., 2018). Resistant varieties show higher accumulation of phenolics and phytoalexins, correlating with reduced disease severity (Srinivasan *et al*., 2011; Nair & Rajendran, 2010). However, the pathogen counters by producing detoxifying enzymes like laccases and peroxidases, facilitating systemic colonization (Sankaran *et al*., 2003; Saharan *et al*., 2018).

Host–Pathogen Molecular Interactions

The molecular interaction between *T. paradoxa* and coconut during stem bleeding disease involves a defense–offense dynamic, where pathogen-associated molecular patterns (PAMPs) like chitin and β-glucans trigger PAMP-triggered immunity (PTI) via PRRs, activating MAPK cascades, ROS bursts, and defense gene expression (Boller & Felix, 2009; Nambiar & Mohandas, 2015). This induces lignification, callose deposition, and phenolic production (Saharan *et al*., 2018; Rajendran & Nair, 2010). In response, *T. paradoxa* releases effectors and CWDEs to suppress immunity, causing effector-triggered susceptibility (Paterson, 2007; Sankaran *et al*., 2003; Nandini *et al*., 2020). The host counters with transcription factors like WRKY, MYB, and NAC to activate systemic acquired resistance (SAR) and defense priming (Mohandas *et al*., 2012; Srinivasan *et al*., 2011). Tolerant genotypes show stronger expression of defense-related QTLs, aiding resistance breeding and diagnostics (Jayasekhar *et al*., 2018; Sharma *et al*., 2012).

Damage Repair and Host–Pathogen Interaction Mechanisms

Host–pathogen interface and initial infection

In *T. paradoxa*-induced stem bleeding disease, infection typically initiates through wounds or root contacts, enabling the pathogen to invade host tissues. The fungus secretes a suite of cell wall-degrading enzymes, including laccases, manganese peroxidases, and cellulases, which break down lignin and cellulose, facilitating penetration and colonization of vascular and cortical tissues (Rees *et al*., 2009; Sundararaj *et al*., 2012). In coconut, a monocotyledonous plant, the vascular bundles are scattered and lack secondary thickening, unlike dicots. This unique anatomical trait hampers effective compartmentalization of infection, making it more susceptible to rapid systemic spread once the vascular integrity is compromised (Esau, 1965). Consequently, coconut's limited structural defense framework at the initial infection site poses a significant challenge in restricting *T. paradoxa* progression.

Damage and defense perception in coconut against stem bleeding infection

In coconut palms infected by *T. paradoxa*, the causal agent of stem bleeding disease, host tissues perceive the attack through recognition of pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs). This perception activates PAMP-triggered immunity (PTI), which launches early defense responses including reactive oxygen species (ROS) bursts, cytosolic calcium influx, mitogen-activated protein kinase (MAPK) signaling, and the induction of pathogenesis-related (PR) proteins, aimed at restricting pathogen spread (Boller & Felix, 2009).

Multilayered defense and repair mechanisms in a monocot host

Although coconut, as a monocot, lacks secondary growth, it activates a well-coordinated and multi-layered defense system in response to *T. paradoxa*, the causal agent of stem bleeding disease:

Lignification and Suberization

One of the earliest structural defenses is the reinforcement of cell walls near the infection site through deposition of lignin and suberin, forming a barrier that restricts fungal progression. This is mediated by the upregulation of key genes in the phenylpropanoid pathway, including PAL (phenylalanine ammonia lyase), C4H (cinnamate 4-hydroxylase), and 4CL (4-coumarate-CoA ligase), which are essential for lignin biosynthesis (Dixon *et al*., 2002; Nandini *et al*., 2020).These structural reinforcements strengthen the cell wall matrix, thereby limiting tissue maceration and fungal colonization during the initial phase of *T. paradoxa* infection. This response, despite the absence of secondary thickening in coconut stems, serves as an effective compensatory defense mechanism, particularly critical in managing vascular-limited diseases such as stem bleeding.

Antioxidant enzyme activity

Infection by *T. paradoxa* induces oxidative stress in coconut tissues, leading to the accumulation of reactive oxygen species (ROS). As a countermeasure, the palm enhances the activity of key antioxidant enzymes including superoxide dismutase (SOD), catalase (CAT), and peroxidases (POX). These enzymes play a crucial role in detoxifying ROS, thereby minimizing oxidative damage to cellular components and maintaining tissue viability under pathogen pressure (Govindarajulu *et al*., 2020).

Phenolic and phytoalexin accumulation:

A marked accumulation of phenolic compounds and phytoalexins is observed in infected zones, forming part of the coconut's localized chemical defense. These secondary metabolites not only exhibit antimicrobial activity but also reinforce surrounding cell walls, creating a protective boundary around necrotic lesions and restricting further fungal invasion (Sivakumar *et al*., 2014).

Hormonal Crosstalk and Signal Modulation

Defense signaling in coconut during T. paradoxa infection involves dynamic hormonal interplay. Salicylic acid (SA) signaling is typically activated during the early stages to mediate biotrophic defense, while jasmonic acid (JA) and ethylene (ET) pathways dominate later necrotrophic phases. These hormone-driven cascades regulate defense gene expression, activating broader responses such as systemic acquired resistance (SAR) and induced systemic resistance (ISR) to enhance long-term immunity (Glazebrook, 2005; Pieterse *et al*., 2012).e. Tissue Compartmentalization and Necrotic Barrier Formation: Although monocotyledons lack a vascular cambium, coconut roots and basal stem tissues respond by forming necrotic zones, depositing callose, producing tyloses in xylem vessels, and physically compartmentalizing infected areas to localize pathogen spread (Mohandas *et al*., 2010).

Regenerative and structural adaptations: In response to *Thielaviopsis paradoxa*-induced stem bleeding, coconut palms initiate localized regenerative responses despite the absence of a vascular cambium. Wound repair is supported by meristematic reactivation and cellular dedifferentiation, particularly near the infection site, enabling limited structural renewal. This regenerative plasticity compensates for the palm's anatomical limitations as a monocot (Heath, 2000). Notably, under sub-lethal infections, coconut seedlings often exhibit enhanced tillering and activation of axillary buds, contributing to the restoration of canopy structure and root biomass. This adaptive growth response appears to be a survival mechanism aimed at offsetting tissue loss and maintaining physiological function during prolonged pathogen stress (Snehalatharani *et al*., 2016).

Microbial symbiosis and induced resistance: Beneficial microbes play a pivotal role in enhancing coconut palm resistance against stem bleeding caused by *Thielaviopsis paradoxa*. Biocontrol agents such as *Trichoderma* spp., Pseudomonas fluorescens, and Bacillus subtilis have been shown to prime the plant's immune system, leading to enhanced deposition of lignin, increased expression of pathogenesis-related (PR) proteins, and activation of induced systemic resistance (ISR) pathways (Jayaraj *et al*., 2005). This microbial-induced priming fosters a "defense memory" in the host, enabling a quicker and stronger response upon subsequent pathogen attacks (Rajeela *et al*., 2017). The interaction between T. paradoxa and coconut represents a molecular tug-of-war involving pathogen-derived enzymes and toxins versus host-mediated multilayered defenses. While the monocot anatomy of coconut limits certain structural adaptations like secondary thickening, the palm compensates through robust biochemical defenses, hormonal signaling (SA, JA, ET), and strategic alliances with beneficial microbes.

Nonconventional Disease Management

Enzyme-based treatments

Recent innovations in enzyme-based disease control offer an eco-compatible strategy for managing stem bleeding disease in coconut caused by *Thielaviopsis paradoxa*. Field applications of lignin-modifying enzymes (laccases, manganese peroxidases) and hydrolytic enzymes (chitinases, β-1,3-glucanases) have been effective in disrupting fungal cell walls and reducing virulence (Saharan *et al*., 2018; Harman *et al*., 2004; Nandini *et al*., 2020). When combined with organic amendments, these treatments enhance rhizosphere microbial diversity and nutrient cycling, suppressing pathogen inoculum density (Thomas *et al*., 2012). Enzyme treatments also act as elicitors, activating host antioxidant enzymes such as peroxidase (POD), catalase (CAT), and superoxide dismutase (SOD) (Sharma *et al*., 2012; Srinivasan *et al*., 2011). They upregulate defense genes including WRKY and MYB transcription factors and enzymes of the phenylpropanoid pathway (PAL, C4H, 4CL), leading to lignin deposition, phenolic accumulation, and systemic acquired resistance (Mohandas *et al*., 2012; Rajendran & Nair, 2010). Integration with endophytic biocontrol agents like *Trichoderma* spp. and *Bacillus subtilis* further enhances disease suppression by reinforcing structural and biochemical defenses (Sankaran *et al*., 2003; Jayasekhar *et al*., 2018). This synergistic approach presents a sustainable model for managing stem bleeding in coconut.

Nanotechnology in disease suppression

Nanotechnology presents a promising, targeted, and eco-compatible strategy for the management of stem bleeding disease in coconut caused by *Thielaviopsis* *paradoxa*. Recent advancements highlight its role in enhancing fungicide efficiency, minimizing environmental impact, and improving pathogen control efficacy.

Nanocarrier-based fungicide delivery:

Chitosan-based nanoparticles encapsulating systemic fungicides like hexaconazole allow for sustained release, improved uptake, and significantly lower EC_{50} values against T. paradoxa, reducing the need for repeated applications (Hafizi *et al*., 2023; Thomas *et al*., 2021). These formulations minimize off-target effects and prolong field effectiveness.

Green-synthesized silver nanoparticles (AGNPS):

AgNPs derived from coconut leaf extracts exhibit potent antifungal activity with minimal phytotoxicity. Their small size enables direct interaction with fungal membranes, causing cell wall rupture and hyphal deformation, making them effective against T. paradoxa (Mardani *et al*., 2022; Premanathan *et al*., 2023).

Metal Oxide Nanoparticles:

Titanium dioxide (TiO_2) and silica nanoparticles act via ROS-mediated oxidative stress, disrupting fungal hyphae and impairing cell metabolism. These particles have shown promising antifungal activity *in vitro* and are being explored for soil and foliar applications (Rashid *et al*., 2021; Ahmad *et al*., 2020).

Azole-loaded nanoformulations:

Nanoencapsulation of azole fungicides such as propiconazole significantly enhances their bioavailability and stability, achieving up to 75% disease suppression in controlled trials. This approach is effective in limiting the progression of stem lesions and bleeding symptoms (Dhasarathan *et al*., 2024).

Nanoenzyme delivery systems:

Nanoparticle-assisted delivery of chitinase and β-1,3-glucanase enzymes improves their stability, penetration, and activity at the infection site, leading to enhanced degradation of T. paradoxa cell walls and virulence inhibition. This represents a new frontier in bio-nanocomposite formulations.

Non-Conventional Fungicidal Interventions in the Management of Stem Bleeding Disease in Coconut

Stem bleeding disease of coconut, incited by *T. paradoxa*, is a chronic and economically significant threat in tropical coconut plantations. While conventional fungicides offer temporary relief, increasing concerns over fungicide resistance, environmental toxicity, and low systemic efficacy have prompted the exploration of eco-friendly, non-conventional strategies.

Chitinase and Glucanase-Based Enzymatic Formulations

Hydrolytic enzymes such as chitinases and β-1,3-glucanases effectively degrade fungal cell wall polymers, directly impairing the growth of *T. paradoxa*. Enzyme-producing microbes like *Trichoderma harzianum* and *Pseudomonas fluorescens* exhibit strong antagonistic activity under field conditions, reducing stem lesion progression and pathogen colonization (Thomas *et al*., 2021; Kavino *et al*., 2020). Moreover, elevated expression of endogenous chitinase/glucanase genes in tolerant coconut genotypes suggests their crucial role in systemic resistance (Ravindra *et al*., 2019). Integration of such enzyme-based microbial consortia with organic substrates improves rhizospheric microbial diversity and disease suppressiveness.

Siderophore-Producing Microbes

Siderophores chelate iron, a micronutrient vital for fungal metabolism, effectively depriving *T. paradoxa* of essential growth factors. Strains of *Pseudomonas fluorescens*, *P. putida*, and *Bacillus subtilis* have demonstrated the ability to inhibit fungal proliferation and simultaneously induce systemic resistance in coconut seedlings by activating defense-related enzymes like peroxidase and phenylalanine ammonia lyase (Kavino *et al*., 2010; Nandini *et al*., 2014; Thomas *et al*., 2021). Co-application with *Trichoderma harzianum* further enhances root zone protection and overall seedling vigor (Kumar *et al*., 2015). Additionally, siderophore-mediated enhancement of soil nutrient dynamics contributes to sustained disease suppression (Arivalagan & Thomas, 2020).

Role of Phenolic and Organic Acids in Disease Suppression

Phenolic and organic acids constitute important components of coconut's biochemical defense arsenal against *T. paradoxa*. These molecules contribute to antifungal activity, modulation of soil microbiota, and activation of host immune responses.

Phenolic acids as defense molecules

Phenolic acids including ferulic, caffeic, and salicylic acids inhibit *T. paradoxa* through direct antimicrobial effects and enhancement of lignin biosynthesis. Treatments involving beneficial microbes have been shown to elevate these phenolic compounds in infected roots, thereby reinforcing both chemical and structural defenses (Thomas *et al*., 2021). Salicylic acid, in particular, plays a pivotal role in triggering systemic acquired resistance (SAR) by upregulating PR-protein expression and antioxidant enzymes such as SOD and CAT in pathogen-challenged tissues (Kavino *et al*., 2020).

Organic acids and their suppressive role

Organic acids such as acetic, oxalic, and citric acids act synergistically with phenolics to impair pathogen growth and facilitate beneficial microbial colonization. Acetic acid disrupts fungal respiration and membrane integrity (Vargas *et al*., 2020), while oxalic and citric acids induce oxidative stress in fungal cells and modulate rhizosphere conditions in favor of antagonistic microbes (Teka *et al*., 2019; Arivalagan & Thomas, 2020). When combined with microbial inoculants, these acids enhance host resilience and suppress *T. paradoxa* colonization effectively.

Next-Generation Fungicidal Strategies against *T. paradoxa*

Strobilurin based fungicides

Strobilurin fungicides such as azoxystrobin, derived from natural metabolites of wood-decay fungi like *Strobilurus tenacellus*, are gaining traction for their potential to manage vascular wilt pathogens including *T. paradoxa*. These Quinone outside Inhibitors (QoIs) act by disrupting mitochondrial respiration at the cytochrome bc_1 complex (Complex III), specifically at the quinol oxidation (Qo) site, leading to ATP depletion and fungal cell death (Nandini *et al*., 2020). Though initially fungistatic, strobilurins exhibit fungicidal activity at higher concentrations and have shown inhibitory effects on mycelial growth and spore germination of *T. paradoxa* in vitro, with minimal adverse effects on beneficial soil microbes due to their mitochondrial specificity (Ahmad *et al*., 2020).

Biogenic and Natural-Origin Fungicidal Molecules

The limitations of synthetic fungicides have driven the adoption of bio-derived antifungal compounds with multi-target action. Secondary metabolites such as harzianic acid produced by *Trichoderma* spp. exhibit strong antagonism against *T. paradoxa* through membrane disruption and competitive nutrient acquisition while enhancing root colonization by plant growth-promoting rhizobacteria (Mukherjee *et al*., 2020). Similarly, essential oil components like thymol and eucalyptol destabilize fungal membranes, causing cytoplasmic leakage, and are recognized for their biodegradability and non-disruptive effect on rhizospheric microbial diversity (Saripalli *et al*., 2021).

Chitosan-based Antifungals

Chitosan, a biodegradable polysaccharide from crustacean exoskeletons, demonstrates dual activity by directly inhibiting fungal growth and priming host plant defense responses. It forms a protective barrier on plant tissues and stimulates systemic acquired resistance (SAR), making it effective against *T. paradoxa* infections in coconut (Badawy & Rabea, 2011). Oligochitosan formulations, with reduced molecular weight, have shown superior efficacy in reducing lesion development and enhancing rhizosphere health by promoting beneficial microbial populations (Liu *et al*., 2020).

Lipopeptides from beneficial bacteria

Cyclic lipopeptides such as iturin, surfactin, and fengycin synthesized by *Bacillus subtilis* integrate into fungal membranes, causing pore formation, ion imbalance, and eventual cytoplasmic leakage. Their antifungal action has been validated against *T. paradoxa* in both in vitro and pot trials, with no observed suppression of indigenous beneficial microbes (Zhang *et al*., 2021; Caulier *et al*., 2019).

RNA based biopesticides

RNA interference (RNAi)-based technologies present a cutting-edge solution for managing *T. paradoxa* with remarkable specificity. Sprayable double-stranded RNA (dsRNA) targeting vital pathogenicity genes such as *EndoPG* (endopolygalacturonase) and *ThPox1* (peroxidase) have shown promising reductions in fungal growth and vascular colonization in preliminary assays (Mitter *et al*., 2017; Koch *et al*., 2016). These dsRNA formulations are compatible with foliar and soil applications and have no detectable effects on non-target organisms. Furthermore, Host-Induced Gene Silencing (HIGS)—where transgenic coconut expresses dsRNA against *T. paradoxa* virulence genes—holds potential for long-term resistance, though its use is pending regulatory evaluation (Qi *et al*., 2021).

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