Journal of Environmental Biology
p-ISSN: 0254-8704 • e-ISSN: 2394-0379 • CODEN: JEBIDP

JEB®

Review Article
Volume 47 Issue 6 November 2026 pp. 1189-1198
DOI: *http://doi.org/10.22438/jeb/47/6/MRN-5990*

***J. Environ. Biol.*, 2026**
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Journal website : www.jeb.co.in ★ **E-mail :** editor@jeb.co.in

Fusarium sugarcane pathosystem: Etiology, diversity, host-pathogen interactions, epidemiology, diagnosis and management of Pokkah Boeng disease of sugarcane

L. Sharma[1]*, D. Singh[1], S.K. Shukla[2] and L. Rawat[3]

[1]*Division of Crop Protection, ICAR - Indian Sugarcane Research Institute, Lucknow-226 002, India*
[2]*Division of Crop Production, ICAR-Indian Sugarcane Research Institute, Lucknow - 226 002, India*
[3]*Department of Plant Pathology, College of Hill Agriculture, Ranichauri, V.C.S.G. Uttarakhand University of Horticulture and Forestry, Pauri Garhwal-246 123, India*

Received: 19 February 2026 **Revised:** 16 June 2026 **Accepted:** 02 September 2026

***Corresponding Author Email:** sharmanbaim@gmail.com ***ORCiD:** https://orcid.org/0000-0002-1873-2803

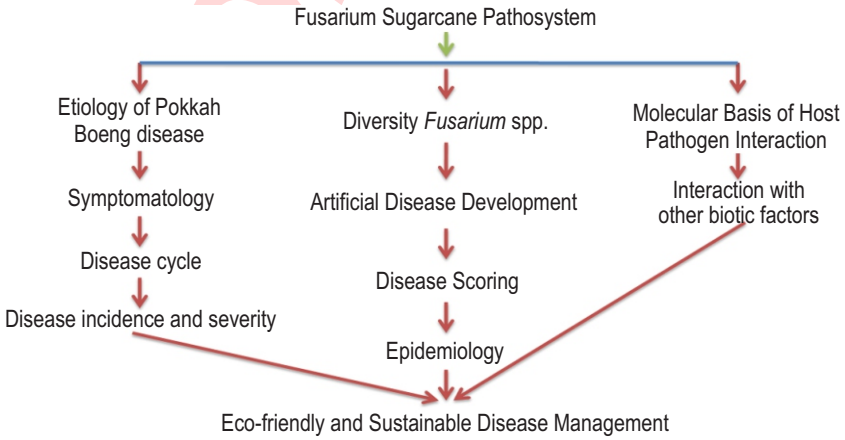
Abstract

Pokkah Boeng is an emerging and economically significant disease of sugarcane (*Saccharum* spp.), causing yield losses of 10-15% in India and posing an increasing threat to sustainable cane production worldwide. This review provides a comprehensive synthesis of current knowledge on the *Fusarium Sugarcane Pathosystem,* encompassing disease etiology, pathogen diversity, host-pathogen interactions, epidemiology, diagnostics and integrated management strategies. Particular emphasis is laid on the taxonomic complexity, phylogenetic relationships and pathogenic variability of the *Fusarium* species complex associated with Pokkah Boeng disease of sugarcane.

The review further explores the influence of climatic drivers, crop production practices, inoculum dynamics and pathogen dissemination mechanisms in shaping disease outbreaks, with particular relevance to Indian agro-ecosystems. Recent advances in understanding molecular interactions between *Fusarium* and sugarcane are discussed including fungal effectors, virulence-associated factors, host defence responses and emerging biotechnological approaches. Current disease management practices, including resistant cultivar deployment, cultural measures, biological control, fungicide application and RNA based technologies are critically evaluated.

Finally, key knowledge gaps and future research priorities are highlighted, emphasising the integration of precision diagnostics, genomics-assisted resistance breeding, climate based disease forecasting and sustainable agro-ecological approaches for durable management of Pokkah Boeng disease of sugarcane.

Key words: Epidemiology, *Fusarium* spp., Host Pathogen interaction, Pokkah Boeng, Sugarcane



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How to cite: Sharma, L., D. Singh, S.K. Shukla and L. Rawat: *Fusarium* sugarcane pathosystem: Etiology, diversity, host-pathogen interactions, epidemiology, diagnosis and management of Pokkah Boeng disease of sugarcane. *J. Environ. Biol.*, **47,** 1189-1198 (2026).

Introduction

Sugarcane (*Saccharum* spp.) is one of the most important commercial crops in India, serving as a major source of sugar and an increasingly important feedstock for bio-ethanol production. India is the world's second largest sugar producer country after Brazil, with annual sugar consumption ranging from 28 to 31 million metric tonnes (Sharma *et al.,* 2023). During 2024-25, sugarcane occupied approximately 5.6 million ha area with an average productivity of 83.6 t ha^{-1}, while Uttar Pradesh contributed the largest share (50%) of total sugarcane cultivated area of India. The expanding emphasis on renewable energy has further enhanced the strategic importance of sugarcane as a leading bio-fuel crop owing to its exceptional biomass productivity and efficient C_4 photosynthetic pathway (Sharma *et al.,* 2023). Despite its economic importance and wide ecological adaptability, sugarcane is vulnerable to numerous biotic stresses including insect pest, nematodes and pathogenic microorganisms. Among these, fungal diseases account for some of the most severe sugarcane yield and quality losses often ranging from 10 to 15% (Blackburn, 1984; Sharma *et al.,* 2024).

Major fungal diseases such as red rot (*Colletotrichum falcatum*), wilt (*Fusarium sacchari*) and smut (*Sporisorium scitamineum*) continue to threaten sugarcane production across the country. However, Pokkah Boeng has emerged as a major concern in recent years with increasing disease severity reported from several sugarcane growing states of India (Sharma *et al.,* 2019; Sharma *et al.,* 2022; Tiwari *et al.,* 2021). Once regarded as a sporadic disease, Pokkah Boeng now exhibits epidemic behaviour under contemporary climatic conditions, particularly during pre-monsoon periods characterised by alternating dry spells and high humidity (Sharma *et al.,* 2024; Poorniammal *et al.,* 2024). Pokkah Boeng is a foliar disease caused by several phylogenetically related *Fusarium* species predominantly *Fusarium verticillioides, F. proliferatum* and *F. sacchari* (Sharma *et al.,* 2024, Poorniammal *et al.,* 2024). Initial symptoms include chlorotic streaks on young leaves followed by twisting, crinkling and the characteristic knife-cut lesions. Severe infections may progress to top rot, resulting in death of the growing point and significant reduction in cane yield and juice quality (Wang *et al.,* 2016; Sharma *et al.*, 2024).

The prolonged crop duration of sugarcane (10-12 months) exposes plants to fluctuating environmental conditions that intensify interactions among pathogen populations, host plants and climatic factors thereby complicating disease prediction and management (Shukla *et al.,* 2017). Pokkah Boeng disease outbreaks are strongly influenced by weather conditions, particularly hot and dry periods followed by humid spells that favour conidial dispersal, stomatal opening and leaf infection (Dixon *et al.,* 1994; Minnatullah *et al.,* 2023). At the physiological level, *Fusarium* infection disrupts photosynthesis, water relations, nutrient transport and hormonal balance, ultimately reducing vegetative growth and sucrose accumulation (Wang *et al.,* 2016; Jiang *et al.,* 2024). Screening programmes conducted across Indian sugarcane research institutions have revealed substantial variation in varietal responses to Pokkah Boeng disease although only a limited number of genotypes exhibit stable resistance under natural field conditions (Shukla *et al.,* 2017). Effective disease management also depends on accurate pathogen identification and a clear understanding of pathogen diversity. However, morphological identification of *Fusarium* species associated with Pokkah Boeng pathogen is often constrained by phenotypic plasticity and symptom overlap with other foliar diseases. Consequently, molecular approaches such as internal transcribed spacer (ITS) sequencing, multilocus phylogenetics and quantitative PCR assays are increasingly employed for reliable pathogen detection and discrimination (Poorniammal *et al.,* 2024).

Management of Pokkah Boeng in India currently relies on integrated disease management (IDM) strategies involving host resistance, field sanitation, balanced nutrition, biological control agents and the judicious use of fungicides (Blackburn, 1984). Fungicides such as carbendazim (0.1%), copper oxychloride (0.2%) and mancozeb (0.3%) are commonly recommended during epidemic conditions although their long term sustainability remains questionable because of environmental concerns and the risk of fungicide resistance (Singh *et al.,* 2025). Consequently, increasing attention is being directed toward environmentally sustainable approaches, including microbial consortia, soil amendments such as biochar, antifungal metabolites and host-directed biotechnological interventions such as Host-Induced Gene Silencing (HIGS) targeting essential fungal genes (Liu *et al.,* 2023; Jiang *et al.,* 2024; Poorniammal *et al.,* 2024). This review consolidates available information on the *Fusarium* Sugarcane Pathosystem with particular emphasis on pathogen diversity, host pathogen interactions, epidemiology, diagnostic advances and integrated disease management strategies. It further identifies key research gaps and future priorities for the sustainable management of Pokkah Boeng disease in sugarcane.

Pokkah Boeng disease

Pokkah Boeng is an emerging pre-monsoon foliar disease of sugarcane caused by several species within the genus *Fusarium*. The history of Pokkah Boeng disease of sugarcane dates back to 1896, when Walker and Went introduced the Javanese term "Pokkah Boeng" for the fungal disease caused in sugarcane. Sheldon (1904) first described the disease caused by *Fusarium moniliforme,* while Butler and Khan (1913) reported *Cephalosporium sacchari* as the causal organism in India. Sawada (1917) described the perfect stage as *Gibberella fujikuroi.* During 1927–37, Bolle conducted isolation, pathogenicity and survey studies in Java and identified sugarcane variety POJ 2878 as highly susceptible. McRae (1932) isolated *Fusarium moniliforme* from diseased sugarcane, and Padwick (1940) reported the first disease incidence of 2.4–2.8% on Co 317 in India. Severe epidemics on POJ 2878 in Java were subsequently reported by Dillewijn (1950). Martin *et al.* (1961) confirmed airborne transmission of *Fusarium* pathogen and

reported serious losses in Java. Ahmed and Padmanabhan (1964) observed conspicuous disease in Tamil Nadu following the introduction of Co 658. Subsequently, Zummo (1970) and Booth (1971) proposed that Pokkah Boeng is a disease complex involving multiple *Fusarium* species, while Higgy *et al.* (1977) confirmed the involvement of *Fusarium* spp. in Asia.

Under favourable climatic conditions, the disease occurs in most sugarcane-growing regions although its severity and economic impact differ substantially among locations and cultivars (Barnes, 1974). Burgess (1981) highlighted airborne spore dispersal, and Nelson *et al.* (1983) demonstrated pathogenic diversity within the genus *Fusarium*. Blackburn (1984) listed Pokkah Boeng among the major *Fusarium* related diseases of sugarcane. Economic losses were reported in Maharashtra on Co 7219 and CoC 671 by Patil and Hapase (1987), while Raid and Lentini (1991) demonstrated greater susceptibility of young sugarcane plants. Patil (1995) classified four disease phases in India. The occurrence of Pokkah Boeng in Haryana and Uttar Pradesh, together with regional variation in symptom expression, was reported by Kamal and Singh (1979) and Singh and Virk (1997). Nirenberg and O'Donnell (1998) reconfirmed the sexual stage as *Gibberella fujikuroi.* By Whittle and Irawan (2000), Pokkah Boeng had been recorded in most sugarcane-growing countries. Owing to its distinctive symptomatology, it is also known as Distorted Top Disease. Summerell *et al.* (2001) linked *Fusarium* section Liseola species with sugarcane diseases and mycotoxin production, whereas Dohare *et al.* (2003) documented 40.8–64.5% sugar losses under severe disease infection. Leslie *et al.* (2006) reported rain-splash dispersal of curved macroconidia, and Siddique (2007) confirmed the vulnerability of young sugarcane stages. Disease development is strongly influenced by environmental factors particularly temperatures ranging from 20 to 32°C and weather patterns characterised by hot, dry periods followed by humid conditions that favour pathogen growth, sporulation and dispersal (Leslie *et al.,* 2001).

Large-scale occurrence in Maharashtra was again reported by Viswanathan and Padmanaban (2008). Govender *et al.* (2010) confirmed *F. proliferatum* and *F. andiyazi* as causal species in South Africa and described disease progression, while Mohammadi *et al.* (2012) further supported the role of *Fusarium* spp. in Pokkah Boeng in Asia. In India, Vishwakarma *et al.* (2013) recorded 1-90% disease incidence across major sugarcane cultivars in Uttar Pradesh. Lin *et al.* (2014) highlighted continuing uncertainty regarding the dominant *Fusarium* species involved in sugarcane epidemics, while Karuppian *et al.* (2015) demonstrated the influence of planting season on disease severity. Wang *et al.* (2016) recognized Pokkah Boeng as an emerging major foliar disease in China, and Wang *et al.* (2017) identified resistant and moderately resistant varieties. Vishwanathan *et al.* (2017) reported *F. sacchari* as a causal species associated with wilt and Pokkah Boeng in India. Severe infections reduce stem elongation, tiller production and sucrose accumulation, resulting in significant losses in cane yield and quality (Muqing and Muralidharan, 2018). Abhay *et al.* (2018) recorded 1.1–21.2% disease incidence across sugarcane varieties in Bihar. Wang *et al.* (2021) developed a trait-based modelling framework for understanding airborne fungal dispersal threats. Yield and quality losses caused by Pokkah Boeng vary considerably depending on cultivar susceptibility, environmental conditions, and the crop growth stage at the time of infection (Poorniammal *et al.,* 2024; Liu *et al.,* 2023). More recently, Goswami *et al.* (2024) reported *F. verticillioides* as a cause of Pokkah Boeng in India, while Siva *et al.* (2025) reported that several *Fusarium* species can separately cause wilt and Pokkah Boeng. Vishwanathan (2026) reported *F. sacchari, F. andiyazi* and *F. verticillioides* as causal agents of wilt and Pokkah Boeng in different sugarcane varieties, emphasizing that Pokkah Boeng does not always progress to wilt and that such progression depends on the variety, season and region.

Despite the agronomic significance of improved sugarcane cultivars, several commercially grown varieties remain highly susceptible to Pokkah Boeng disease. Disease incidence ranging from 2 to 40% has been reported in the cultivars. The severe disease manifestations including the characteristic knife-cut symptoms and top rot stage have been particularly documented in cultivars Co 0238 and CoS 08279 (Vishwakarma *et al.,* 2013; Abhay *et al.,* 2018). The extensive cultivation of these susceptible varieties across tropical and subtropical sugarcane-growing regions of India considerably elevates the risk of substantial yield and economic losses during disease-conducive periods.

Diversity of *Fusarium* spp. associated with Pokkah Boeng disease: Pokkah Boeng disease of sugarcane is caused by a complex of *Fusarium* species. The genus *Fusarium,* first described by Link in 1809, is among the most diverse and economically important groups of phytopathogenic fungi, infecting cereals, legumes, grasses and numerous other crops. Early *Fusarium* taxonomy relied primarily on morphological characteristics. Wollenweber and Reinking (1935) proposed one of the earliest monographic classification systems and described nearly 1,000 taxa distributed among three major species groups. Snyder and Hansen (1945) subsequently simplified the taxonomy by recognising nine species complexes. Booth (1971) further refined species identification using characteristics such as conidiogenous cells, colony morphology, cultural behaviour on different media and incubation requirements. He highlighted the diagnostic significance of phialide morphology, particularly monophialides versus polyphialides and also variations in microconidiophore and microconidial morphology for distinguishing species such as *F. oxysporum, F. solani* and *F. moniliforme.* Booth (1971) also incorporated sexual structures called perithecia into the taxonomy and classified the genus into twelve sections comprising forty four species and seven varieties. Gerlach and Nirenberg (1982) expanded the number of recognised species through extensive examination of infected plant materials while Nelson *et al.* (1983) revised sectional classification using microconidial morphology, macroconidial

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dimensions and chlamydospore position. Alexopoulos *et al.* (1996) clarified the dual taxonomic status of Fusarium recognising the genus as anamorphic within the Deuteromycetes and teleomorphic within the Ascomycota (Class Sordariomycetes, Order Hypocreales, Family Nectriaceae). Subsequent studies recognised sixteen sections within the genus namely Eupionnotes, Macroconia, Spirarioides, Submicrocera, Pseudomicrocera, Arachnites, Sporotrichiella, Roseum, Arthrosporiella, Gibbosum, Discolour, Lateritium, Liseola, Elegans, Martiella and Ventricosum (Leslie *et al.,* 2001).

Summerell *et al.* (2003) further formalised the morphological species concept based on conidial morphology, sporulation patterns and colony characteristics. Species belonging to section *Liseola* are particularly important in Pokkah Boeng epidemiology and have traditionally been distinguished by morphological traits. Globally, several *Fusarium* species have been associated with the disease including *F. verticillioides* in Iran; *F. moniliforme* and *F. moniliforme* var. *subglutinans* in the United States; *F. andiyazi* and *F. sacchari* in South Africa; *F. verticillioides* and *F. proliferatum* in China; *F. sacchari* in Australia; and *F. moniliforme* in Thailand. In Indonesia, reported taxa include *F. anguioides, F. bulbigenum* var. tracheiphilum, *F. moniliforme* and its varieties (subglutinans and anthophilum), *F. neoceras, F. orthoceras* var. longius and *F. semitectum.* In India, *Cephalosporium sacchari* was first reported in association with Pokkah Boeng by Butler and Khan (1913). Subsequently, Patil and Hapse (1987) differentiated *C. sacchari* and *F. moniliforme* based on enzymatic activity and pathogenic behaviour during infection.

Comparative studies have demonstrated substantial variation among Fusarium species in colony morphology, growth characteristics, phialide structure and conidial morphology. For example, *F. proliferatum* isolates from rice, maize and sugarcane typically produce abundant aerial mycelia, pyriform to clavate conidia and numerous polyphialides, whereas *F. fujikuroi* generally forms cottony colonies with abundant sporodochia and fewer polyphialides. Similarly, *F. sacchari* characteristically produces pyriform macroconidia whereas *F. subglutinans* produces predominantly oval macroconidia. Microconidial production also provides important diagnostic characters. *F. proliferatum* forms long chains containing more than twenty microconidia, whereas *F. fujikuroi* generally produces shorter chains containing fewer than twenty conidia. In contrast, F. sacchari and *F. subglutinans* lack microconidial chains and instead produce microconidia in false heads allowing their differentiation from *F. verticillioides, F. proliferatum* and *F. fujikuroi.* Additional distinctions between *F. sacchari* and *F. subglutinans* are provided by macroconidial morphology. Vegetative compatibility group (VCG) analysis has been widely used to assess genetic relatedness among *Fusarium* isolates. Isolates capable of forming stable heterokaryons are considered members of the same VCG (Leslie, 1993; Leslie and Summerell, 2006; Patil *et al.,* 2007). The technique involves generation of nitrate non-utilising (*nit*) mutants on potassium chlorate-amended media, followed by phenotypic characterisation on minimal media containing nitrate, nitrite, hypoxanthine or ammonium (Mohammadi *et al.,* 2012). Mutants are classified as *nit1, nit3* or *NitM,* and complementary pairings are used to determine heterokaryon formation and VCG membership (Correll *et al.,* 1987). Morphological traits alone cannot reliably distinguish more than 400 phylogenetically diverse *Fusarium* species, molecular identification now relies primarily on sequencing of housekeeping genes such as translation elongation factor 1-α (TEF1-α) and the RNA polymerase II subunit genes (RPB1 and RPB2). These markers provide substantially greater phylogenetic resolution than the internal transcribed spacer (ITS) region which is relatively conserved within the genus (O'Donnell *et al.,* 2022). Among them, approximately 680 bp 5' region of *TEF1-α*, containing three introns is regarded as one of the most informative single locus markers for species discrimination. Current identification protocols typically combine *TEF1-α* sequencing with *RPB1* or *RPB2* analyses followed by BLASTn searches against curated databases such as FUSARIUM-ID v.3.0 and GenBank and validation through maximum-likelihood phylogenetic analyses (O'Donnell *et al.,* 2022). Beyond the sequence based diagnostics, biosensor technologies are emerging as promising alternatives for rapid detection of *Fusarium* pathogens and associated mycotoxins. Compared with conventional culture-based and molecular methods, biosensors offer faster analysis, minimal sample preparation, portability and suitability for field deployment, thereby supporting early disease diagnosis and improved management strategies.

Symptomatology: The initial symptoms of Pokkah Boeng appear as chlorosis and yellowing (irregular whitish to pale yellow areas) at the basal portion of young emerging leaves, especially near the midrib representing the early chlorotic phase of the disease. As infection progresses in the mature leaves, affected leaves become wrinkled, twisted and are noticeably narrower than healthy leaves (Fig. 1). Subsequently, irregular reddish streaks and specks develop within the chlorotic areas / tissues which later give rise to lens shaped perforations or ladder like lesions bordered by dark margins (Leslie and Summerell, 2006).

Under severe infection, the pathogen advances from the leaf base into the stalk, producing dark reddish streaks that extend across internodes. Infected stem tissues exhibit dark-brown discolouration and cellular rupture as diseased tissues fail to expand synchronously with adjacent healthy tissues resulting in characteristic ladder like depressions. In advanced stages, these lesions resemble sharply incised cuts giving rise to the characteristic knife-cut symptom of Pokkah Boeng disease (Govender *et al.,* 2010). The most severe manifestation of the disease is the top rot stage, where the growing point collapses and dies. Top death has been frequently reported in highly susceptible cultivars such as Co 0238. Symptom expression varies according to the timing of infection, duration of favourable environmental conditions, inoculum availability and varietal susceptibility, but similar symptom patterns have been consistently reported across sugarcane growing regions worldwide (Raid and Lentini, 1991; Whittle and Irawan, 2000).

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Fig. 1: (a) initial Pokkah Boeng symptoms showing chlorosis and yellowing of young leaves (b) twisting and curling of top young leaves (c) sever necrosis and death of top portion of sugarcane, called Top rot (d) downward pathogen infection marked like knife made, called Knife-cut symptom.

Epidemiology and disease cycle of *Fusarium* pathogen: *Fusarium* pathogen is soil borne in nature. The primary inoculum of Pokkah Boeng pathogen originates from infected contaminated soil, seed setts, irrigation water and crop residues where *Fusarium* spp. can survive for 9-12 months under field conditions, particularly in cool and dry environments (Vishwakarma *et al.,* 2013). Maximum survival has been reported in infected debris buried at depths of approximately 30 cm. During the pre-monsoon period, the pathogen produces abundant conidia that are dispersed by wind and rain splash, including the Puff and Tap mechanism characteristic of rain dispersed fungi (Raid and Lentini, 1991; Deacon, 2006). Airborne conidia settle on partially unfolded leaves and are subsequently washed into the spindle and basal leaf-sheath regions by rainfall (Blackburn, 1984). Germination occurs on young, succulent tissues and the emerging hyphae penetrate through soft cuticle and poorly lignified epidermis of developing leaves (Barnes, 1974; Vishwanathan *et al.,* 2017). The pathogen then colonises leaf tissues and advances into immature stalks, invading vascular bundles and disrupting water and nutrient translocation, resulting in tissue distortion, rupture and development of characteristic ladder like lesions (Holliday, 1980) and ultimately, top rot involving

death of the growing point (Raid and Lentini, 1991; Whittle and Irawan, 2000). Secondary inoculum is continuously produced on infected tissues and disseminated throughout the cropping season by wind-driven rain, irrigation splash, contaminated soil and insect vectors such as stem borers and crown mealy bugs (Whittle and Irawan, 2000). The pathogen survives for extended periods in infected plant debris and produces multiple spore types that facilitate both local and long distance dispersal (Raid and Lentini, 1991; Deacon, 2006).

Standardized artificial inoculation techniques: Successful and reproducible development of Pokkah Boeng disease under experimental conditions depends the crop growth stage, inoculum quality and inoculation method employed. *Fusarium* spp. associated with Pokkah Boeng exhibit considerable ecological adaptability and produce multiple spore types that serve as both primary and secondary inoculum sources. Once these propagules reach susceptible tissues, particularly the spindle and leaf-sheath regions, infection is initiated under favourable environmental conditions, especially warm temperatures and high relative humidity (Wang *et al.,* 2017). Consequently, several artificial inoculation methods have been standardised for uniform disease development and screening sugarcane genotypes for resistance under field conditions.

Foliar spray inoculation: Fresh pathogen cultures are grown under laboratory conditions and harvested at peak sporulation (5-7 days). Conidial suspensions are adjusted to approximately 2×10^6 conidia ml^{-1} and sprayed uniformly onto the leaf sheath and young leaves of sugarcane plants at three to five leaf stage using a hand sprayer or atomiser. Inoculated plants are generally maintained under high humidity for 24-48 hr to promote infection, and disease symptoms are typically assessed 15 days after inoculation (Sharma *et al.,* 2024; Siddique, 2007; Poorniammal *et al.,* 2024; Hilton *et al.,* 2017; Wang *et al.,* 2016).

Spindle inoculation: In this method, freshly prepared conidial suspensions (2×10^6 conidia ml^{-1}) are injected directly into the spindle region of sugarcane plants at four to five leaf stage using a sterile syringe. Direct inoculum delivery to actively growing tissues ensures reliable symptom development. Disease expression is generally recorded 15 days after inoculation. (Tiwari *et al.,* 2021; Siddique, 2007; Poorniammal *et al.,* 2024; Hilton *et al.,* 2017; Wang *et al.,* 2016).

Soil inoculation: This technique is commonly used to evaluate early infection processes and varietal responses to the pathogen under pot or field conditions. Soil is treated with a conidial suspension (2×10^6 conidia ml^{-1}) at the time of cane planting. Disease incidence is generally recorded around 60 days after planting, when foliar symptoms and growth abnormalities become evident. (Tiwari *et al.,* 2021; Siddique, 2007; Poorniammal *et al.,* 2024; Hilton *et al.,* 2017; Wang *et al.,* 2016).

Molecular basis of host-pathogen interaction: Pokkah Boeng represents a complex *Fusarium* sugarcane pathosystem involving multiple species, particularly *F. verticillioides* and *F. proliferatum* which employ diverse molecular strategies to colonise host tissues, suppress plant immunity and alter host metabolism. Recent advances in fungal genomics, transcriptomics and functional genetics have significantly improved understanding of virulence mechanisms and host defence responses involved in disease development. A major determinant of *Fusarium* pathogenicity is the secretion of effector proteins that manipulate host cellular processes. In *F. sacchari*, the metalloproteinase effector FsMEP1 interacts with sugarcane thiamine thiazole synthase (ScTHI2), causing its mislocalisation and disrupting thiamine biosynthesis, a pathway associated with plant defence signalling. This interaction reduces reactive oxygen species (ROS) accumulation, suppresses callose deposition and inhibits programmed cell death, thereby facilitating fungal colonisation (Wang *et al.,* 2024). Similarly, the secreted protein Fs00367 enhances fungal virulence by targeting sugarcane susceptibility-associated protein ScPi21 and suppressing ScPi21-mediated defence signalling during early infection (Huang *et al.,* 2024). Besides effectors, *Fusarium* species produce a range of cell wall-degrading enzymes, including pectinases, cellulases and xylanases which facilitate host tissue penetration. The pectate lyase gene *FsPL* from *F. sacchari* is essential for full virulence; however, it also acts as a pathogen-associated molecular pattern (PAMP) that activates host defence responses. Disruption of *FsPL* reduced pathogenicity, whereas its transient expression induced ROS accumulation and callose deposition through receptor-like kinase-associated pathways involving BAK1 and SOBIR1 (Huang *et al.,* 2024).

Successful infection further depends on fungal regulatory networks controlling growth, development and pathogenicity. The C_2H_2 zinc-finger transcription factor *FsCGBP* (cutinase G-box-binding protein) regulates mycelial growth, conidiation and virulence in *F. sacchari.* Deletion of *FsCGBP* impaired infection ability and altered expression of genes associated with cuticle degradation and MAP kinase signalling, indicating its role as a key regulator of host invasion and stress adaptation (Liang *et al.,* 2024). Similarly, the conserved 14-3-3 proteins FsBmh1 and FsBmh2 regulate hyphal differentiation, metabolism and pathogenicity, with loss-of-function mutants showing reduced growth and attenuated virulence (Chen *et al.,* 2024). Sugarcane responds to *Fusarium* infection through multilayered defence mechanisms, including oxidative bursts, cell wall reinforcement and activation of defence-related genes. However, pathogen effectors and regulatory networks can suppress or redirect these responses, particularly in susceptible cultivars.

Understanding these molecular interactions has facilitated the development of innovative resistance strategies, including Host Induced Gene Silencing (HIGS). RNA interference-mediated silencing of essential ergosterol biosynthesis gene *FsCYP51* significantly reduced fungal growth and disease severity and enhanced resistance in transgenic sugarcane lines under greenhouse and field conditions (Yin *et al.,* 2025). Such approaches represent promising, environmentally sustainable

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tools to complement conventional breeding and integrated disease management strategies, particularly in regions affected by diverse *Fusarium* populations.

Interaction of Pokkah Boeng disease pathogen with other biotic factors: *Fusarium* species associated with Pokkah Boeng particularly *F. verticillioides* and *F. proliferatum* persist in soil and crop residues and can colonise sugarcane tissues as endophytes (Wang *et al.,* 2016; Poorniammal *et al.,* 2024). Insect pests and other arthropods including crown mealy bugs, ants, borers, leaf feeders and mites by causing tissue injuries that serve as infection courts for *Fusarium* propagules. These wounds may facilitate interactions between Pokkah Boeng, stalk borers and secondary rot causing fungi resulting in synergistic disease outcomes. Such tritrophic interactions among host plant, pathogen and insect pest are increasingly recognised as important drivers of disease complexity in sugarcane monoculture systems. The frequent association of Pokkah Boeng with major diseases such as red rot, smut and stalk rot highlights the need to consider the disease as a component of a broader sugarcane disease complex rather than an isolated disorder. Therefore, sustainable management requires integrated approaches targeting multiple biotic stresses through deployment of multi disease resistant cultivars, crop residue management, crop rotation, insect-pest suppression, balanced nutrient management and climate-based optimisation of fungicide applications (Singh *et al.,* 2025).

Pokkah Boeng disease incidence and disease severity: During host-pathogen interactions, sugarcane genotypes exhibit varying levels of response to Pokkah Boeng infection. Based on percentage disease incidence, five reaction categories have been reported for screening sugarcane clones/genotypes against Pokkah Boeng disease. The Pokkah Boeng disease scoring system devised by Elmer (2002) employs a 0 - 5 disease rating scale based on the severity of visible symptoms. A rating of 0 represents no visible symptoms, while 1 indicates slight chlorosis on young leaves. A rating of 2 is assigned when less than 10% of the leaf area exhibits chlorosis and/or approximately 10% of the plant shows twisted-leaf symptoms. A rating of 3 represents 11 - 25% of the plant exhibiting twisted-leaf symptoms. A rating of 4 is assigned when 26 - 50% of the plant shows twisted leaves and/or reddish specks develop within chlorotic areas. The most severe rating, 5, represents 51 - 100% of the plant exhibiting twisted, wrinkled and shortened leaves or plant death. The disease severity index (DSI) is calculated as $DSI = \Sigma(A \times n) \times 100 / [\Sigma B \times \text{Maximum rating scale}]$, where A is the disease rating scale (0-5), n is the number of stalks evaluated at each disease rating, B is the total number of stalks evaluated, and the maximum rating scale is 5.

Disease management: Sugarcane is a long duration crop requiring approximately 10-12 months to attain maturity stage, and Pokkah Boeng symptoms generally appear soon after sett germination or around 60-70 days after planting. Disease severity progressively increases during the grand growth phase (3-7 months after planting), after which effective disease management becomes increasingly difficult. Therefore, early detection and timely integrated disease management are essential for minimizing disease development and associated losses. Major management strategies for Pokkah Boeng include deployment of resistant or moderately tolerant varieties, which remains the most effective, economical and sustainable approach for reducing disease incidence and losses (Han, 1956; Vishwakarma *et al.,* 2013; Karuppaiyan *et al.,* 2015). The use of healthy, disease-free planting material is also important to minimize pathogen carry-over and reduce the transmission of primary inoculum between cropping cycles (Vishwakarma *et al.,* 2013; Ranjan *et al.,* 2022). Crop sanitation, including the removal and destruction of infected crop residues and severely diseased stalks exhibiting top rot or knife-cut symptoms, can further reduce inoculum survival and secondary disease spread (Vishwakarma *et al.,* 2013). Fungicide application at the initial stages of disease development is another important component of management, copper oxychloride (Blitox-50, 0.2%), carbendazim (0.1%) and mancozeb (0.3%) have been recommended, with two to three foliar applications at 15-day intervals reported to suppress pathogen development and minimize yield and juice-quality losses (Vishwakarma *et al.,* 2013; Karuppaiyan *et al.,* 2015). In addition, biological and eco-friendly approaches involving biological control agents and beneficial microbial consortia capable of suppressing *Fusarium* spp. or inducing systemic resistance in sugarcane represent promising components of sustainable Pokkah Boeng management. Although these approaches require further validation, they offer considerable potential for developing environmentally compatible disease management strategies (Karuppaiyan *et al.,* 2015; Sharma *et al.,* 2024).

Conclusion and future perspectives: *Fusarium* Sugarcane Pathosystem associated with Pokkah Boeng represents a multifaceted and evolving disease complex shaped by the interplay among pathogen diversity, host susceptibility and environmental variability. Pokkah Boeng disease of sugarcane has emerged as a significant constraint to sustainable sugarcane production, particularly under changing climatic scenarios that favour *Fusarium* proliferation, dispersal and infection. Recent advances in comparative genomics, transcriptomics and functional biology have transformed our understanding of Fusarium pathogenicity by revealing sophisticated mechanisms involving secreted effectors, cell wall degrading enzymes, regulatory networks and host immune manipulation. However, the molecular basis of host specificity, pathogen adaptation and variation in virulence among *Fusarium* populations associated with sugarcane remains poorly understood. Integrating pathogen genomics with host genetic resources, phenotyping platforms and environmental data will be essential for identifying durable resistance mechanisms and predicting disease emergence under future climate conditions.

Acknowledgments

The authors thank the Director, ICAR - Indian Sugarcane Research Institute, Lucknow for providing research facilities and support.

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Authors' contribution: L. Sharma: Data collection, literatures, analysis and first draft was prepared; **D. Singh:** Assisted in data collection, literatures and analysis; **S.K. Shukla:** Assisted in data collection, literatures and analysis and manuscript writing; **L. Rawat:** Assisted in preparing manuscript .

Funding: No external funding.

Research content: The research content of manuscript is original and has not been published elsewhere.

Ethical approval: This research article does not contain any studies with human participants or animals performed by any authors.

Conflict of interest: The authors declare that is there is no conflict of interest.

Data availability: The datasets generated during and analyzed during the current study are available from the Corresponding author upon reasonable request.

Consent to publish: All authors agree to publish the paper in *Journal of Environmental Biology.*

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