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Pokkah boeng or twisted top disease – A new threat to sugarcane cultivation

Anuradha*, Manisha Meena, Rajinder Kumar and Gulzar Singh Sanghera

Punjab Agricultural University, Regional Research Station, Kapurthala-144601, Punjab, India.

ABSTRACT

Pokkah boeng or twisted top disease caused by the Fusarium species complex is a fungal disease reported to cause economic losses in sugarcane crops throughout the world. Currently, the disease has become of major constraint in sugarcane production in many sugarcane-growing countries. The incidence of the disease is highly affected by epidemiological factors and the pathogen spreads through the air currents, with secondary infections occurring in irrigation water, rain splashes, and the soil. Several Fusarium species are reported to be involved in causing pokkah boeng disease, including F. verticillioides, F. sacchari, F. proliferatum, F. subglutinans and F. andiyazi. The disease causes yield losses up to 90 per cent. The traditional methods for identification of Fusarium species are based on the morphology of macroconidia, microconidia, and conidiophores but this process is tedious and needs expertise. Hence, both morphological and molecular phylogenetic analysis is important for the identification of this fungus up to species level. Management practice for this disease includes the use of healthy seed material, the use of resistant varieties, cultural practices, and fungicidal and biological control. However, the development and use of resistant varieties is the most viable and economical approach for this disease. This review summarized the various aspects of pokkah boeng disease like distribution, epidemiology, and disease management strategies.

Keywords: *Pokkah boeng, Symptoms, Epidemiology, Transmission, Management, Fusarium*

Introduction

Pokkah boeng or twisted top disease (TTD) in sugarcane is a Javanese term that means malformed and distorted top. It was first reported in Java by Walker and Went in 1896 and later on, by Edgerton [1]. It is one of the serious fungal diseases of sugarcane and its occurrence has been described in almost all sugarcane-producing countries of the world [2]. The 3 to 7-month-old rapidly growing plants of sugarcane are more vulnerable to disease rather than grown-up plants [3]. Pokkah boeng was first reported in India 1930s and 1940s [4], but the severity of the disease was documented in two commercial types, Co 7219 and CoC 671, in Maharashtra between 1983 to 1984 [5]. In Haryana, pokkah boeng was reported on varieties Co 1148, CoS 767, and CoJ 64 [6].

Climatic conditions where a hot and dry season was followed by a wet season were shown to be conducive for pokkah boeng spread [7]. Pokkah boeng disease is caused by *Fusarium* but it is not well identified, which *Fusarium* species is responsible to cause the disease. The disease is well-studied in Asia by many researchers [8] [9] [10]. The incidence of pokkah boeng disease is increasing in states like Uttar Pradesh, Haryana, Punjab, Maharashtra, Tamil Nadu, Assam, Bihar, and other sugarcane-growing parts of India [11],[12] [13] [14]. The disease was observed to have a low to moderate incidence on most commercial types, but the severity was significant in Uttar Pradesh and Maharashtra. Pokkah boeng disease symptoms have been documented in Andhra Pradesh (CoA 99082, Co 7805, CoV 94102, 98V95, and 2000V59) and Haryana (CoH 133, CoJ 85, CoH 151, and CoS 8436), whereas top rotting symptoms have been reported in Haryana (CoS 8436, CoH 152, Co 89003, and CoH 136). Pokkah boeng disease is developing as a major danger to sugarcane output in Maharashtra, with cases reported in a wide range of types (Co 86032, Co 94012, CoV 19805, Co 05002**,** VSI 434, CoC 671, CoM 08090, Co7527 and Co 8014) [4]. In Bihar, trace to moderate disease incidence was observed in several varieties, whereas in western Uttar Pradesh (Saharanpur, Muzzafarnagar, Meeru, and Bijnour districts), the disease was observed in varieties CoS 94257, CoS 8436, CoS 767, CoSe 98231, CoSe 95422, CoS 88230, CoS 94270, and CoJ 64, and eastern Uttar Pradesh (Baharaich Kushinagar and Gonda districts) [4]. CoPant 97222, Co Pant 99214, CoPant 99259, CoS 8432, CoS 8436, CoS 767, and Co 1148 were also found to have severe conditions in Uttarakhand [13].

Economic importance

In India, pokkah boeng disease was first observed in Maharashtra in 1983-1984 on two commercial varieties of sugarcane, CoC 671, and Co7219, and was identified as the state's biggest constraint for sugarcane output [15]. Pokkah boeng disease is reported to reduce the quality up to 40.8 to 64.5 per cent every year in all cultivated varieties [16]. Uttar Pradesh reported up to 90 per cent incidence on genotype S224/10 [13]. Similarly, Goswami et al. [17] also observed yield losses from 40 to 60 per cent in sugarcane by pokkah boeng disease. Lin et al. [18] reported the occurrence of pokkah boeng throughout the growing season. Pokkah boeng sickness has historically been more common after heavy rains or during the post-monsoon season. The disease, however, is now present throughout the growing season, in both wet and dry conditions.

Disease symptoms

Pokkah boeng disease symptoms appear in four stages: chlorotic phase I, chlorotic phase II, knife cut phase, and acute/top-rot (Fig. 1).

*Corresponding Author: ***Anuradha***

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The symptoms of the disease are easy to distinguish as it generally infects the upper portion of plants and causes chlorosis of younger leaves from the basal areas [1]. Later on, the infected leaves showed wrinkling, curling, and twisting and get reduced [1], within the chlorotic region irregular reddish stripes and specks are observed, which occasionally turn into lens-shaped holes [19]. Ladder-shaped lesions in longitudinal rows may also appear on leaves which later on turned into dark reddish to brown colour. The next advanced and serious stage is top rot where during favorable conditions, soft rot develops in stalks of susceptible varieties [1].

Pokkah boeng disease is spread by spores or ascospores, and the pathogen enters the host tissues through any type of injury, such as being injected by insects or borers, or through naturally developed fissures. In Australia, Bangladesh, China, Pakistan, Indonesia, Taiwan, and Vietnam, it has been described as a devastating disease of sugarcane cultivars [2]. The whole base of the spindle gets rotten and top rot of the cane tissue occurs. The pathogenic infection penetrated the stalk and continued downward. Top rot resulted in the death of the top of the cane which is the most serious [3] [19]. Sometimes, the knife-cut symptoms are observed in association with the top rot phase in the form of one two, or more transverse cuts in the rind of the stalk. In India, Kamal and Singh [20] first time observed the knife cut symptoms in mature cane stalks affected by pokkah boeng disease.

Due to the splitting of diseased cells that were unable to grow alongside healthy tissues, a lengthy lesion formed in the internodal areas, giving an external and internal ladder-like appearance [3]. If the infection is limited to the leaves, the plant usually recovers, but if an internal ladder-like disease develops in the stem, the plant would die. Soft rot develops in infected areas during damp weather [1]. When the fungus infects the growth points and kills the entire top of the cane, the most extreme harm occurs. This is referred to as top rot or acute phase [3] [19]. In extremely sensitive kinds, heavily infected plants have a deformed or twisted top, and death of the upper sections of the plant might ensue [19].

Causal Organism

Pokkah boeng disease was first reported to be caused by *Gibberella fujikuroi* (Sawada) by Sheldon [21]. *Fusarium moniliforme* as a causal pathogen of pokkah boeng disease is confirmed and established by many workers [8] [9] [10] [14]. Govender et al. [9] reported the association of *F. sacchari* with Pokkah boeng disease. In China, pokkah boeng has been described as one of the most important diseases of sugarcane [18]. *Fusarium* is also reported to be a causal pathogen of many other sugarcane diseases like wilt [22], stalk and root rot [23], and knife cut [20]. Leslie and Frederiksen [24] studied the incidence of pokkah boeng disease in sorghum and reported it to be caused by *F. moniliforme*. Martin et al. [19] reported *F. moniliforme* var. *subglutinans* as the causative pathogen of maize (*Zea mays* L.) diseases like seedling blight, root stalk and kernel rot, and sugarcane pokkah boeng disease. *Fusarium moniliforme* Sheldon and *Gibberella fujikuroi* (Saw.) were also reported to be a cause of pokkah boeng disease of sugarcane in Thailand [25].

In recent studies, several *Fusarium* species were found to be involved in causing pokkah boeng disease, including *F. verticillioides* [10] [26], *F. sacchari* [4] [17] [27], *F. proliferatum* and *F. subglutinans* [28], *F. verticillioides* or *F. subglutinans* [26], *F. verticillioides* and *G. fujikuroi* [25], *F. sacchari*, *F. proliferatum* and *F. andiyazi* [9] and *F. verticillioides* and *F. proliferatum* [18].

Costa et al. [29] studied the detection and pathogenicity of pokkah boeng associated with various *Fusarium* species in Brazil which is the biggest sugarcane producing country in the world. They identified 39 isolates under study as *F. sachhari*, *F. proliferatum* and other unknown species having phylogenetic lineage to *F. andiyazi*. *F. verticillioides* and *F. proliferatum* were detected as the causal agent of pokkah boeng disease in China [18]. Recently, the involvement of *F. sacchari* with pokkah boeng was also detected by Meng et al. [30]. Three *Fusarium* species *viz*., *F. verticillioides*, *F. proliferatum*, *F. subglutinans,* and *F. semitectum* were reported as pokkah boeng causal pathogen from Iran by Khani et al. [28]. In Mexico, two *Fusarium* species (*F. verticilliodes* and *F. proliferatum*) were identified from pokkah boeng isolates [31].

In India, Patil and Hapase [14] reported the association of *Fusarium moniliforme* with sugarcane pokkah boeng disease. *F. sacchhari* and *F. proliferatum* were reported in association with the occurrence of pokkah boeng disease which is known to occur in all sugarcane growing states and recorded different severity levels in almost all commercial cultivars [4] [12] [13].

Taxonomy and Nomenclature

Alexopoulos et al. [32] identified *Fusarium* sp. as an anamorphic fungus belonging to the class *Deutromycetes*, order *Moniliale*s, and family *Tuberculariaceae*. *F. moniliforme* has been identified as the major representative of the Liseola section. Later scientists proposed that the pathogen *F. moniliforme* has a variety status. On morphological basis, *F. subglutinans var. subglutinans* can easily distinguished from *F. moniliforme* [33]. Booth [34] transferred *F. moniliforme* again into the section Liseola. This decision was based on the fact that morphological characteristics such as conidiophores and conidiogenous cells as well as spore and colony colour, chlamydospores, and mono or polyphialids were used to distinguish *F. moniliforme* var. *subglutinans* and *F. subglutinans* var. *subglutinans* in the section Liseola. The presence and absence of microconidia, macroconidia, and chlamydospores, as well as their shape and size, were used to separate each section by Nelson et al. [35]. Snyder and Hansen [36] restricted the definitions of species, eventually proposing a nine-species classification scheme. They were dubbed "dramatic lumpers" for their contribution. Finally, morphological traits, as well as phylogenetic and molecular approaches, were used to classify *Fusarium* [37].

Epidemiology

The incidence of pokkah boeng is highly affected by epidemiological factors and pathogens spread through air currents and infected setts [2] [13] [38]. The rainy season is beneficial for conidia to move down to the sensitive regions of the spindles at the margin of partially unfolded leaves and to germinate there. Because the epidermal tissues are still fragile and are not protected by the plant system, the germinated conidia produce mycelium, which passes through the flimsy cuticle of immature spindle leaves to the interior tissues. According to Holliday [39], the ladder-like lesions form when mycelium spreads to vascular bundles and blocks the vessels which results in distortion of growth. The symptoms of distortion and rupture, as well as their progression, resemble those of a ladder. Hot and dry weather causes leaves to partially unfurl, allowing flying conidia to land on the leaves.

Sugarcane stem borers larvae and adults also transmit the fungus [40]. *Chilo* spp., a type of top borer, causes deformation and shortening of the top leaves, which are remarkably similar to the symptoms of pokkah boeng disease [41].

Pokkah boeng disease of sugarcane can also be disseminated through fungus-infected setts [42]. Temperature influences the distribution of pathogen. The minimum, optimal, and maximum temperatures required for pathogen to cause disease are 10 to 15 °C, 30 °C, and 35 to 40 °C, respectively. The disease thrives in temp ranging from 20 to 30 °C, with high humidity of 70 to 80 per cent and gloomy weather during the monsoon season, which runs from July to September. Following big rain events or the end of the monsoon season, the incidence of pokkah boeng sickness has historically increased. Lin et al. [18] found that the disease is now present throughout the growing season, during both rainy and dry times. Karuppaiyan et al. [14] also discovered that without the fungicidal spray, over 90 per cent of pokkah boeng infected plants recovered their growth when the plant grew older and the weather changed. The disease incidence was lower in autumn-planted canes than in spring-planted canes.

Transmission and viability

The pathogen that causes pokkah boeng disease is spread by air currents, which transport spores from one location to another [3] [19]. Pokkah boeng is an airborne disease that spreads through the air, with secondary infections occurring in irrigation water, rain splashes, and the soil. In natural environments, the pathogen (*F. moniliforme*) has been shown to persist for one year in plant debris and soil, while in laboratory conditions, it has been found to survive for more than eight months. At 50 °C, the fungus could not develop, although it was still viable for at least six months. *F. moniliforme* can live for up to a year, while the occurrence drops after nine months. At 30 cm deep in the soil, maximum survival was reported for more than 11 months in natural conditions. The survival of a fungus in plant debris was aided by cool and dry conditions [20]. The spore of the pokkah boeng disease is spread by air currents from one location to another, and the spore settles on plants and begins colonizing their leaves, flowers, and stems [43]. The dispersal of spores is affected by the weather conditions (windy, rainy, or dry), which necessitates diverse dispersal tactics. There are mainly two ways of mechanism for the dispersal of pathogen described by Deacon [44].

The "puff" and "tap" mechanisms cause the dry spore to become airborne and, in most cases, the spores of the fungus are dispersed by rain splashes. Fungi grew on leaf surfaces and produced spore chains that were blown away by the wind, misted air, or hygroscopic (drying) movements, causing the spore to buckle. It also mentioned that hot, dry weather caused the opening of leaves between partially unfurled leaves, allowing airborne conidia to land on the leaves. Conidia are swept down to the vulnerable regions of the spindles near the border of partly unfurled leaves when rains begin, where they germinate. Because the epidermal tissues are still frail and not protected by the plant system, the conidia sprout and the mycelium penetrates through the flimsy cuticle of young leaves to the interior tissues. Mycelium spreads to the embryonic stem's vascular bundles and plug vessels, causing growth deformities and rupture, and the formation of ladder-like lesions. Dissemination of fungal spores may also be reported by pupae and adults of sugarcane stem borers [40].

Morphological characterization

Fusarium species can be identified and characterized by using different identification approaches. Different workers used morphological observation, pathogenicity test, and phylogenetic analysis approaches as diagnostic tools to differentiate *Fusarium* species from one to another [4] [12] [29].

Various cultural differences have been reported in isolates of *Fusarium* spp. (Fig 2). Summerell et al. [45] considered the shape and size of conidia to study and identify species of *Fusarium*. In Florida, white and purple color strains of *F. moniliforme* were identified by Bourne [40].

Burgess [43] studied the morphology of *Fusarium* by using 21 days 21-day-old culture. They reported one, two, and three septations in conidia ranging from 6 -14 × 5 µm, 14-21 × 2-4 µm and 19-28 × 2-5 µm, respectively**.** Nirenberg and O'Donnell [46] observed delicately floccose felted with powdery form due to the formation of macroconidia in the mycelium of *F. moniliforme*. Khanna and Rafay [47] discovered that a low or high amount of glucose affects the length of *Fusarium moniliforme* conidia. Chattopadhyay and Gupta [48] reported the size of conidia of *Fusarium* spp. in the range of 9.3 to 29.7 µm in length and 2.7 to 6.0 µm in width in potato dextrose agar medium (PDA). Sharma and Kumar [49] investigated *F. moniliforme* (Sheldon) cultures raised from 12 infected samples of sugarcane in diverse areas of Uttar Pradesh between 2012 and 2014. In a pathogenicity test, four isolates (Fm122, Fm126, Fm129, and Fm1212) were found to be more virulent than the rest. In different strains, the size of the macro and microconidia ranged in size from 12.4–46.8 × 2.4–6.8 µm and 4.6–9.9 × 1.8–4.8 µm, respectively were observed. Thaware et al. [50] investigated the cultural, morphological, and molecular properties of *F. oxysporum* f. sp. *ciceri*. Of the eight isolates studied, FOC-2 (Jalna) had the highest mycelial growth of 90.00 mm. On the other hand, Jalna (FOC-2) and Beed (FOC-3) formed partially submerged (FOC-2) to submerged (FOC-3) white sparse dense growth and bright white substrate pigmentation. Maximum micro conidial, macro conidial, and chlamydospore size (17.20 µm, 30.50 × 7.00 µm, and 21.80 × 19.60 µm) was formed in isolate Jalna (FOC-2). The microconidia were oval to cylindrical and lacked septation. According to Somu et al. [51], lactose and sucrose promoted good growth of *Fusarium* spp., but maltose promoted poor growth. *Fusarium* spp. grew well in magnesium nitrite, followed by sodium nitrate [52].

Molecular characterization

The traditional diagnostic methods based on the morphology of macroconidia, microconidia, and conidiophores for identification of *Fusarium* species are though considered as primary characteristics [35] [38], but this process is tedious and needs expertise. Molecular biology has provided fungal taxonomists with several useful tools, such as methods for identifying isolates and methods for illuminating the connections between fungal species. DNA markers are easily inherited, phenotypically neutral, polymorphic, plentiful, and appropriate for analyzing complex characteristics. To describe genetic links in fungi, many types of molecular markers have been utilized. The introduction of molecular tools has expanded the variability, taxonomy, and evolutionary investigations of *Fusarium*. Various molecular techniques are restriction fragment length polymorphism (RFLP) [53], amplified fragment length polymorphism (AFLP) [54], simple sequence repeats (SSR) analysis [55], random amplified of polymorphic DNA (RAPD) analysis [56] and DNA amplification fingerprinting (DAF) analysis [57].

Polymerase chain reaction (PCR) is a sensitive and fast method for identifying and detecting *Fusarium* species in pure culture and diseased cane samples. Various Polymerase chain reaction (PCR)-based techniques have been widely used in the identification and detection of *Fusarium* spp. [58].

Unique bands of DNA from random amplified polymorphic DNA (RAPD) analysis have been used to distinguish this fungus [59]. Several *Fusarium* species have been detected or identified by PCR using species-specific primers [60] [61]. These techniques have the advantages of speed and sensitivity [58] [59]. Ingle and Rai [62] identified 10 isolates of *F. semitectum* based on morphological and cultural features, which were verified by RAPD-PCR using 40 random primers. Based on an unweighted paired group method of arithmetic average (UPGMA) cluster analysis, the genetic similarity coefficients between pairwise isolates ranged from 0.00 to 1.95. Vogelsang et al. [63] characterized two species of *Fusarium viz*., *F. graminearum* and *F. culmorum* using SSR primers.

ISSR, unlike SSR, does not need prior sequence information and generates specific and reproducible banding patterns [64]. Singh and Kapoor [65] identified and quantified *Fusarium oxysporum* f. sp. *carthami* using ISSR markers and sequence-characterized amplified region (SCAR) markers. Mahmoud and Fatah [66] revealed the feasibility of using ISSR, sequence-related amplified polymorphism (SRAP), SSR, and biochemical markers in genetic diversity analysis in Fusarium wilt disease of faba bean (*Vicia faba*).

Techniques like Internal Transcribed Spacer (ITS) and cDNA markers have also been used widely. The ITS techniques have been effectively used by various workers to do phylogenetic analysis in *Fusarium* spp. [67]. Hilton et al. [68] described the identification and characterization of pathogenic and endophytic species associated with sugarcane pokkah boeng disease. They extracted DNA from five fungus strains and amplified and sequenced the ITS regions. Sequencing results indicated that the ITS region (475 bp) amplified from strain 20 showed 100 percent similarity with *F. verticillioides, F. begonia,* and *F. bactridioides*. Kumar et al. [69] examined the alignments for *Fusarium* species and reference sequences using the BLASTn method. The maximum likelihood approach was used to generate phylogenetic trees with 1000 bootstrap replications, and the trees with the highest log likelihood were observed. Lin et al. [70] described the *F. verticillioides* strains ITS, *TEF-*$\boldsymbol{\alpha}$, and ATP-6 (ATPase) gene sequences, and deposited them in GenBank (accession numbers ranging from KJ765857 to KJ765871). Phylogenetic trees were constructed using PCR amplification sequences from *F. verticillioides* isolates and other matching sequences from GenBank.

Species-specific PCR technique (TaqMan as well as conventional) was used for the detection and identification of *Fusarium* species complex causing pokkah boeng disease in China [18].. This technique has also been used for the identification of *Fusarium graminearum* causing Fusarium head blight (FHB) disease on cereal crops [71]. *TEF1-α* gene has been used as a useful genetic marker to study the different species of *Fusarium* [4] [72]. Costa et al. [29], identified the 39 isolates as *F. sacchari, F. proliferatum*, and an unidentified phylogenetic branch sister to *F. andiyazi*. All three species produced pokkah boeng symptoms in infected sugarcane plants and stem rot in maize, sorghum, and millet.

Tiwari et al. [73], identified two *Fusarium* isolates (F2 and F7) as *Fusarium fujikuroi* and *Fusarium proliferatum* causing pokkah boeng of sugarcane by phylogenetic analysis based on the internal transcribed spacer 1, partial sequence; 5.8S ribosomal RNA gene and internal transcribed spacer 2, complete sequence and large subunit ribosomal RNA gene, partial sequence.

Management

Disease control strategies in sugarcane are mainly based on integrated approaches like legislative measures, planting of healthy seed material, use of resistant varieties, and other management approaches [11]. Fungicide applications aiming at managing the pokkah boeng disease are useful as sett treatment and/or as spraying using fungicides such as Bavistin (1gm/lit with water) or Copper oxychloride (2 gm/lit with water)) or Dithane M-45 (3gm/lit of water) [13]. Treatment of setts by overnight dipping in fungicidal solution or by using a mechanized sett treatment device followed by two to three sprays within 15 days limits the spread of the pathogen inoculum [13]. The use of mechanized sett treatment device to treat the planting material (setts/buds) helps in providing effective delivery of fungicides in small quantity within a short period [74]. Sett treatment helps in improving germination by protecting the planting material from sources of inoculum present in the soil [75]. Chemical control measures are often expensive and due to the harmful impacts (short-term and long-term) of fungicides on the environment, eco-friendly approaches like biological control agents are also explored for the management of this disease. Biocontrol agents rely on various mechanisms to inhibit their target pathogen either act directly through competition, mycoparasitism, and antibiosis or indirectly by induction of disease resistance in plants [76]. *Trichoderma* spp. is one of the most widely used biocontrol agents worldwide for the management of several plant diseases in various crops including sugarcane [73]. Species of *Pseudomonas* and *Bacillus* have also been recognized as potential endophytic bacteria to suppress plant diseases induced by fungal pathogens [77].

The best way to prevent the incidence of pokkah boeng disease is to use healthy seed cane as planting material. The infected plants should be roughed out of the field as and when appear. Regular examination of clones at various levels of selection throughout the breeding program is essential for the development of resistant varieties.

In recent years, pokkah boeng disease has become an important biotic constraint that seriously affects sugarcane production in India. The disease is spreading rapidly in sugarcane growing areas where monoculture of single variety exists and due to difficulties associated with the widespread use of chemical control measures and drawbacks of biological control measures and also lack of effective chemical management strategy use of resistant varieties is the most effective measure for its management [13] [14]. Evaluation of germplasm for resistance to multiple diseases is a crucial step in managing plant diseases through host plant resistance. Resistance to pokkah boeng varies among different sugarcane clones/varieties. Several sugarcane clones have been screened for disease resistance in India. Wang et al. [78] assessed four different artificial inoculation methods and concluded that the syringe method and spindle inoculation method were the most reliable for varietal screening against the sugarcane disease pokkah boeng. They also screened the sugarcane germplasm for pokkah beong disease in Subtropical China and reported eleven clones as highly susceptible and moderately susceptible. Karuppaiyan et al. [14] studied the incidence of pokkah boeng in 360 indigenous and exotic sugarcane clones and reported that spring-planting canes had higher disease incidence than autumn-planted canes. They also reported that the majority of the clones were resistant or moderately resistant to pokkah boeng, whereas subtropical varieties such as BO 137, Co 0238, CoH 110, CoJ 83, CoJ 85, CoS 8432, CoS 8436, CoS 88230, CoS 03251, CoS 03252, CoSe 95436,

CoSe 01434 and UP 0097 were moderately susceptible and fourteen clones, namely Co 425, Co 12027, Co 10036, Co 0331, M 165/38, H 45-2120, B 46-199, Q 64, PR 1058, CP 81-1384, F 65-554, POJ 6688, EPC 37-069, and H 44-3098 were susceptible. Lin et al. [70] studied the natural disease incidence of pokkah boeng in 13 cultivars and 52 cross combinations of sugarcane and reported that the incidence in most of the cultivars was between 0 to 14 per cent. Ranjan et al. [79] tested 22 sugarcane varieties under natural conditions for pokkah boeng disease and found that twelve clones (BO 154, CoP 9301, BO 91, BO 153, CoP 112, CoP 9301, CoP 16437, CoP 2061, CoP 3437, CoP 3438, CoP 16440 and CoSe 13452) were resistant and four clones (BO 156, BO 155, Co11438 and CoP 13436) were moderately susceptible to pokkah boeng. Anuradha et al. [80] screened the seventy-one clones/varieties of early and mid-late maturity groups and state-release varieties of sugarcane in Punjab under natural conditions. During the study, forty-four clones/varieties were found resistant, nineteen clones/varieties exhibited moderately susceptible reaction and eight were susceptible to disease. Variety Co 0238 was found highly susceptible to pokkah boeng disease.

The main factors which led to the outbreak of pokkah boeng disease are the planting of susceptible varieties along with conducive weather conditions. The screening of sugarcane germplasm for resistance to pokkah boeng disease should be strengthened as being the most viable and economic approach in a variety of breeding programs. The planting of susceptible varieties should be avoided in susceptible zones and the use of resistant varieties should be promoted to control the outbreak of pokkah boeng disease.

In India, during recent years, pokkah boeng disease emerged as the most important fungal disease of sugarcane that causes both qualitative and quantitative losses. Timely surveys and surveillance for pokkah boeng disease incidence have to be undertaken to assess the crop losses caused by the disease and for the adoption of proper management practices.

Future Scope

The future research on pokkah boeng of sugarcane encompasses investigating environmental and climatic influences on disease spread, and exploring host-pathogen interactions to identify resistance mechanisms. Use of integrated disease management approaches, combining cultural practices, biological controls, and judicious chemical use, alongside economic impact assessments and farmer education programs, will be helpful to manage the disease and enhance sugarcane productivity sustainably.

Conclusion

Sugarcane is one of the major cash crops in tropical and sub-tropical regions of the world. India ranks second globally in sugarcane production and is the world's biggest consumer of sugar. The crop is attacked by numerous pathogenic micro-organisms including fungi, bacteria, viruses, and mycoplasma as well as nematodes. Pokkah boeng which caused both qualitative and quantitative losses in sugarcane has become a major fungal disease in recent years. There are four stages to the disease viz., chlorotic phase I, chlorotic phase II, knife cut phase, and acute/top-rot. Epidemiological factors have a significant impact on disease. It has been reported to be caused by several *Fusarium* species complex. Therefore, it is important to identify the species involved by using both morphological and molecular phylogenetic studies to develop proper management practices for the disease. Various approaches are used for the management of the disease like the use of healthy seed material, resistant varieties, various cultural practices, use of fungicides, and biological control measures. However, the most practical and cost-effective way to combat the disease is to develop disease-resistant varieties.

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Conflict of Interest We state that there is no conflict of interest in publishing this article.

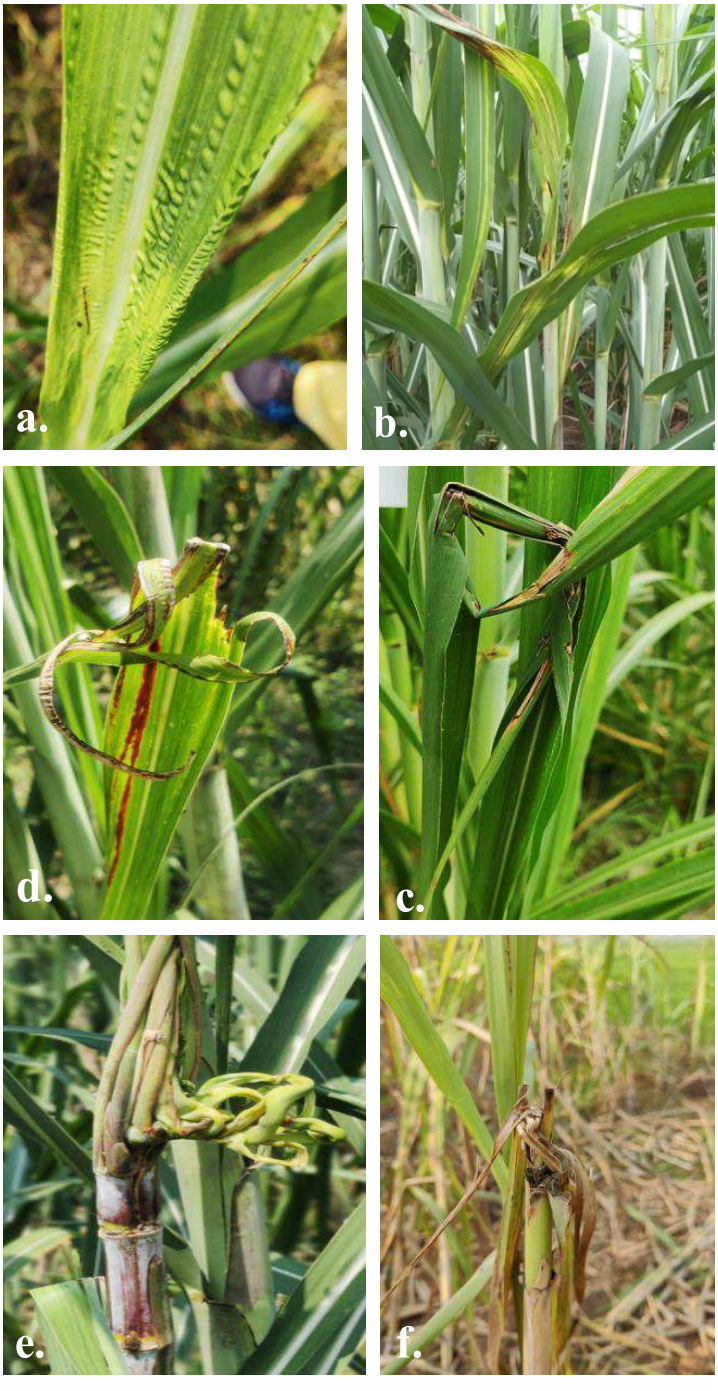

Fig. 1 Pokkah boeng disease symptoms include chlorosis (a), leaf tearing and chlorosis (b), wrinkling and curling (c), lead tearing (d), knife cut (e) and top rot (f)

Fig. 2 Morphological variability among isolates of Fusarium spp. isolated from pokkah boeng disease samples.

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