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Fusarium diseases in sugarcane

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Introduction

Wilt is one of the earliest known diseases of sugarcane in India and was first reported by Butler (1906) from Bihar state. Wilt epidemics in India during the last century resulted in elimination of many commercial cultivars from cultivation (Kirtikar *et al*., 1972; Singh and Singh, 1974; Subba Raja and Natarajan, 1972). Later also very severe wilt incidences were noticed in South Gujarat and in different parts of Gangetic plains. Country-wide disease assessment revealed that wilt of 60% on Co 7717, 5-10% in CoJ 64, CoJ 79 and CoS 767 in Uttar Pradesh, severe wilt incidence in combination with red rot noticed on major varieties in Bihar, severe wilt incidence on Co 89003 and moderate wilt on Co 7717, CoS 8436 and CoS 88230 in Punjab, varying levels of wilt in most of the varieties in cultivation in South Gujarat, mild wilt on popular varieties in Maharashtra and in Madhya Pradesh (Agnihotri and Rao, 2002). Previous studies of Viswanathan *et al.* (2006) revealed that the disease intensity vary from trace to 75% in different states of India. Wilt in the cv. Co 7805, an elite variety in coastal Andhra Pradesh caused enormous loss to sugarcane production in the past two decades (Viswanathan, 2013a).

Butler and Khan (1913) for the first time described the disease in India in sugarcane under the term 'wilt' and noted *Cephalosporium sacchari* as the causal agent. However, Bourne (1922) recorded a stem rot disease of the basal portions of unwounded sugarcane stems having a species of

Fusarium associated with the disease in Barbados. Abbott (1932) noted a purple species of *Fusarium* constantly associated with diseased cuttings in Louisiana and also reported the presence of a purple and white strain of *Fusarium* in seed cuttings. Bourne (1953) recorded the *Fusarium* stem rot as prevalent during the period 1949-52 in the Everglades region of Florida, and considered it was just as important to breed canes resistance to it as to red rot itself. Apart from India, the disease has been reported from 34 countries in different continents (Rao and Agnihotri, 2000).

Impact of wilt in sugarcane

Wilt is a serious constraint to sugarcane production in India and is next to red rot caused by *Colletotrichum falcatum* in causing economic losses. In Louisiana, both white and purple strains of *Fusarium* caused serious reductions in germination of cuttings (Abbott, 1935). During 1960s, the disease caused severe damage to sugarcane crop in the Deccan plateau in India. Losses due to wilt are usually computed on the basis of quantum of canes, dried or dead, found in the field, after harvest and they varied from 2 to 10 tonnes/ha (Parthasarathy, 1972). Sarma (1976) reported that loss in the yield might go as high as 65% and the incidence of the disease is more in ratoon as compared to the plant crop.

Wilt affected canes show poor juice quality due to the pathogen infection in the stalk tissues. The deterioration in juice quality is due to the decrease in sucrose content and an increase in reducing sugars, gums, titrable acidity, flavonoids and soluble salts (Singh and Waraitch, 1981), such changes adversely affect processing of white sugar in the mills. The disease causes 14.6 to 25.8% reduction in juice extraction and 3 to 20% in sugar recovery (Gupta and Gupta, 1976). When the disease incidence was only 6% in cvs Co 658 and Co 449, ~9.97% reduction in sugar recovery was recorded (Subba Raja and Natarajan, 1972). During 2016, it was observed that severe wilt affected canes recorded only 1.5 to 2.0 brix as compared to 13-19.5 in apparently healthy canes in the cv. Co 0323 during harvest Kollegal areas in Karnataka state (Viswanathan, unpublished).

Wilt fungus in association with some insect pests of sugarcane particularly stalk borer and scale insects causes significant damages to the crop. In association with stalk borer, the disease has been reported to bring a loss of about 8.75 tonnes/ ha (Kulshreshtha and Avasthy, 1959). When the mean incidence of stalk borer-wilt complex was 51.4%, Singh (1973) found decline of 24.9% in cane weight. Waraitch (1981) reported high incidences of ~90% wilt in association with stalk borer in the popular variety Co 1148 and the crop was almost unfit for milling. Conservative

estimates of loss of 3-6 tonnes/ha, the disease may cause a loss of 12.7-25.4 million tonnes annually in different years accounting for several million dollars loss in India (Viswanathan *et al*., 2006). In addition to the losses in production to the farmers, the sugar industry also suffers due to loss of recoverable sugar in the mills and such unaccounted losses occur every year. Synergistic activity of red rot and wilt pathogens on many varieties is well established and also wilt aggravates red rot susceptibility in some of the varieties (Viswanathan, 2010, 2013a,b). Combination of wilt with red rot, stalk borer or root borer still makes the crop unfit for downstream processing in mills. Furthermore, losses caused by wilt go unnoticed in the field, since the pathogen infection takes place during maturity stage of the crop. The loss could be noticed only at the time of harvest, hence under normal situations the crop is harvested as healthy one, but still it suffers from the disease.

Symptomatology

Although the disease has been recorded a century ago, still confusion prevails on disease symptoms and it is mostly due to overlapping symptoms of the disease with other stalk diseases and lack of detailed publications on the disease. Recently, Viswanathan (2012a, 2013a) brought out a detailed account on the disease symptoms in sugarcane and for the first time, he elaborated many unknown symptoms of the disease. The disease affects the crop during the different phases *viz*., germination, young crop and maturity. The disease expression/symptoms in matured crop are well known as yellowing followed by drying of foliage and subsequent withering of infected plants. The germinating setts or germinated settlings also show the disease symptoms and usually such cases are ignored. Either disease infections during this stage are counted with sett rot (pineapple disease) or red rot. If the infections are severe, germinated settlings in patches may show drying. Careful examination of the dead settlings would reveal characteristic wilt in mother setts. The first author has witnessed such wilt infections in young crops in Gujarat and other places where the disease is epidemic. Here it was found that infected setts serve as the primary source for wilt development (Viswanathan, 2012a, 2013a).

Occurrence of wilt during tillering phase is inconspicuous until the canes are half grown. Initially the infected canes in a clump or all the canes in a clump show bright yellow /orange discolouration of leaves in the crown. The infected canes will be distinctly seen due to the foliage discolouration and stunted growth. Gradually the infected canes alone wither and dry in a background of healthy canes (Fig. 1) (Viswanathan,

2013a). In severe cases, yellowing/drying of canes occurs over a large area occurs, which leads to extensive damage to the crop (Fig. 2). Red rot also causes such extensive damage to the crop, however, here the clumps show varying levels of disease progress and such uniform crop damage as in wilt is not noticed.

Fig. 1. Wilt affected sugarcane plants exhibiting severe foliage discolouration

Fig. 2. Wilt affected sugarcane crop (cv. TNAU Si8) shows extensive crop damage in the field

Though wilt infected canes do not exhibit any rind disolouration as in the case of red rot, careful examination of the affected canes show loss of luster, paleness and the affected canes become shaky (Fig. 3). Internally the young canes show discolouration along with typical cavity formation in internodes. The discolouration starts as pinkish to pinkish-

brown and finally turns to deep brownish. Usually outer edges of the cavities show darker shades of discolouration and the colour intensity diminishes towards pith region (Fig. 4). The pith region exposes fibrous tissues from the core tissue. Matured canes display the disease symptoms similar to the young canes; however, the pith region showed more exposed fibers with deep pith cavities (Viswanathan, 2013a). The internal disolouration caused by the wilt pathogen vary from variety to variety. Occasionally different shades of intermodal discolouration are common and it may camouflage with other stalk rot related diseases. Such closely related and contrasting symptoms of diseases infecting sugarcane stalks such as red rot, wilt, pineapple disease and stalk rot were clearly described earlier (Viswanathan, 2012a). Unlike canes affected with red rot or pineapple diseases, the wilt-affected canes do not emit any odour. There are no white spots in internal tissues that are typical of red rot affected canes. Occasionally, scattered red streaks (vascular strands) in the internodes above the badly affected internodes are seen and such streaks pass from one internode to another. Initially, the root system of the affected plant does not show any discolouration, lesions or damage attributable to the disease, but subsequently the affected roots die. Severely affected plants show severe death of roots and reduction in root mass.

Fig. 3. *Fusarium sacchari* infection causes paleness of rind tissue in sugarcane stalks

Fig. 4. *Fusarium sacchari* infection causes characteristic pith cavities along with tissue discoloration to stalk tissue

Pathogen

Fusarium sacchari (E.J. Butler) W. Gams was first described as *Cephalosporium sacchari* Butler from sugarcane in India (Butler and Khan, 1913). Subsequently, *F. moniliforme* var. *subglutinans* was reported as the causative agent. Gams (1971) named *F. sacchari* (Butler) W. Gams as a new species and brought together both *C. sacchari* and *F. moniliforme* var. *subglutinans*. Later, two varieties of *F. sacchari* such as, *F. sacchari* var. *sacchari* and *F. sacchari* var. *subglutinans* were delineated (Nirenberg, 1976). In addition, Singh and Singh (1974) reported isolation of *Acremonium implicatum* and *A. furcatum* from wilt infected samples in subtropical India. Leslie *et al.* (2005) designated the neotype of the chosen isolates as *F. sacchari* (E.J. Butler) W. Gams after detailed sequencing and AFLP studies and teleomorph of was described as *Gibberella sacchari,* sp. nov. Studies of Viswanathan *et al*. (2006) on the pathogen recovery revealed that out of 95 cane samples collected nationwide, only 53 samples yielded wilt pathogens and all of them were found to be species of *Fusarium* and no *Acremonium* was isolated. Further, Poongothai *et al*. (2014a) compared recovery of *Fusarium* spp. from nodal and internodal tissues and it revealed that wilt pathogen was recovered from 84 and 70 nodal and internodal tissues of the 125 cane samples, respectively. Sugarcane is the most preferred host for *F. sacchari*, however reports are available on its infectivity on other crops such as maize, sorghum, wild rice, wheat etc. (Oren *et al.,* 2003; Petrovic *et al.,* 2013; Wang *et al.,* 2015).

Variation in *F. sacchari*

Recently detailed studies were carried out on phenotypic and genotypic variability in *F. sacchari* at ICAR-SBI, Coimbatore. From 346 wilt affected cane / soil samples collected from 15 states in India, 263 *Fusarium* isolates were recovered and no other fungal genera were recovered from the cane samples. The fungal isolates were characterized based on cultural, morphological, molecular and pathogenic variation.

Cultural variation: Cultural characterization of 117 isolates divided the *Fusarium* isolates into three groups based on radial growth as slow, moderate and fast. More than 75% of the isolates showed typical pinkish pigmentation and other cultures exhibited varying shades of pinkish pigmentation. The isolates were grouped into seven groups based on mycelial colour and they were assembled into 21 groups again based on mycelia colour/pigmentation. The isolates exhibited variation for topology of the mycelium and conidial frequency (Poongothai *et al.,* 2014a). The cultural characters were inconsistent and they tend to change with culture

conditions (Agnihotri and Rao, 2002). Viswanathan *et al.* (2012) reported production of macroconidia on carnation leaf agar.

Morphological variation: In addition to cultural characters of the wilt-associated fungal isolates, types of conidia produced, their shape, types of phialides production, conidial formation and germination and chlamydospore production in the same 117 isolates were characterized (Poongothai *et al.,* 2014b). All of the isolates produced microconidia at different frequencies. Macroconidia were straight to falcate and the apical and basal cells were either conical or blunt. Sporogenesis on CLA revealed macroconidia production by 9 to 12 h after inoculation and microconidia production either in false heads or chains after 12 to 15 h. This detailed report on morphological characteristics involving more than 100 *F. sacchari* isolates collected from different geographic regions in India clearly established a huge variation for the characters for the first time. Earlier, information on morphological features of sugarcane wilt pathogen was totally lacking. The extensive variation in cultural and morphological characters of the isolates may probably due to their origin from varied climatic conditions and host varieties in the country.

Molecular variation: Molecular tools such as RAPD, ISSR and IGS-RFLP were applied to characterize a set of 50 of 117 *F. sacchari* isolates. The morphologically distinct isolates formed separate clusters and isolates of *F. sacchari* grouped together in a cluster. Within this cluster, due to intraspecific variation *F. sacchari* isolates were further grouped into many subclusters (Poongothai *et al.,* 2015). However, use of other molecular markers such as AFLP and TEF may further refine grouping of *F. sacchari* isolates and other *Fusarium* spp associated with wilt of sugarcane. Studies of McFarlane *et al. (*2009) revealed variation in the *F. sacchari* group based on phylogenetic analysis of elongation factor-1α sequence and ISSR analysis and the results suggested existence of different genotypes of this species in sugarcane. Their studies further revealed the power and simplicity of ISSR analysis for the fingerprinting of fungal isolates and its use to confirm the identities of inoculated isolates.

Confirmation of *Fusarium sacchari* as wilt causative pathogen

Many reports were made on the association of different species of *Fusarium* and *Acremonium* with sugarcane wilt during different occasions in India (Agnihotri, 1983; Agnihotri and Rao, 2002). Since detailed studies were lacking on reproduction of the disease symptoms and Koch postulate on the disease, we carried out detailed pathogenicity studies on wilt associated *Fusarium* and established *F. sacchari* as the causative agent in

sugarcane (Viswanathan *et al.,* 2011). The study involved inoculation of a set of isolates collected from disease endemic region and a virulent isolate and an avirulent from non-endemic region in sugarcane, re-isolation of the isolates from the infected cane tissues and comparing molecular profile of them along with the recovered cultures. The molecular profile of pathogenic and nonpathogenic *Fusarium* isolates for the primers RAPD OPA 13, ISSR1 ISSR5 and ISSR9 clearly demarcated the virulent pathogenic isolates from the avirulent pathotypes (Poongothai *et al.,* 2015).

Simulation of wilt in sugarcane

Butler and Khan (1913) could not reproduce typical disease symptoms using *C. sacchari*, while later workers were successful. Four artificial inoculation methods viz., inoculation of setts, cut canes, standing canes and soil were reported during different periods with reasonable success in disease reproduction. Using these methods, claims and counter claims have been made regarding the reproduction of the disease on artificial inoculation. Singh *et al*. (1980) reported pathogenicity of *F. moniliforme* var. *subglutinans*, *A. furcatum* and *A. terricola* by inoculation into 6-7 months old standing stalks and by growing plants in the infected soil. Mohanraj and Alexander (1984) developed a quantitative numerical index for rating wilt severity based on all the effects of disease incidence on stalks. Based on the pathogenicity, a 0 to 4 scale was developed to rate wilt resistance in sugarcane genotypes. Viswanathan *et al.* (2015) made extensive studies on various methods to induce wilt in sugarcane such as plug method of inoculation, soil inoculation, planting of setts in infected soils etc. Although wilt was not earlier reported in young crops, Viswanathan (2012a) reported infection of the germinating shoots under disease endemic conditions. Similarly, findings of the study reiterated that soil inoculation of the pathogen caused both pre- and post- germination death of buds/shoots and pathogen recovery from the dead plants (Viswanathan *et al.,* 2015). Overall, different studies on pathogenicity clearly reveal that once the pathogen gets access into the host, it causes severe damage to the crop. But the pathogen entry into the host has not been studied in detail. We found roots are infected at early stages of crop growth. Further progress of the pathogen from roots to the stalk remains a mystery. Perhaps soil and environmental factors may play a vital role in disease development in the stalks and its aggravation.

Epidemiology

Infected planting materials setts and crop residues with the pathogen inoculum in the soil serve as primary sources of *F. sacchari* spread

in the field. *F. sacchari* is also reported to persist in the soil for 2.5 to 3 years. Wilt is very common in certain locations where conducive environment and susceptible hosts are available. However, the disease expression and its severity are being influenced by various biotic and abiotic factors in the field. Secondary spread from field to field occurs through rain and irrigation water. However, primary sources of the pathogen inoculum are important in disease perpetuation and spread in the field. If the inoculum comes from both the setts and soils, the disease severity is more as compared to the single source. Since sugarcane is a long duration crop with the practice of ratoons, soil borne inoculum plays a vital role in initiating the pathogen infection in plant or in ratoon crops. When pathogen infected setts are planted, the pathogen readily initiates infection on root and further infection progresses to the growing sprout. Perhaps depending on the pathogen load and its spread inside the host plant, disease manifestation is observed. Delayed expression of the disease indicates the need of a threshold in pathogen colonization in sugarcane root and stalk tissues.

Biotic factors

Apart from separate infections of *F. sacchari*, combined infections of *F. sacchari* and *Colletotrichum falcatum* causing red rot is found under field conditions in different states in India (Viswanathan *et al.,* 2006). The extent of damage caused to the crop is more in wilt-red rot complex compared to the crop with wilt alone. Similar observation was recorded by many workers in the past (Agnihotri and Rao, 2002). Combined infection of red rot and wilt in sugarcane yielded cultures of both *C. falcatum* and *F. sacchari* on culture medium (Viswanathan, 2012a). The observation points that presence of *C. falcatum* weakens the host thereby facilitate the entry of *F. sacchari*. Simasalit (1996) also found the stalk disease "red rot wilt" as a major sugarcane disease in Thailand.

Under Indian situations, wherever root borer, *Emmalocera depressella* infestation is severe, more wilt infection was reported. Earlier, Sardana *et al*. (2000) found a positive association between root borer and wilt. Viswanathan (2013a) found a clear association of wilt and root borer in the popular sugarcane cv. Co 89003 in subtropical India and in another cv TNAU Si8 (Si 2000-02) in Cauvery delta of Tamil Nadu. During the recent wilt epidemics in the plot with 611 parental clones at Coimbatore, it was found that clones expressed wilt either alone or in association with root borer, termites, internode and top borer. Out of the 611 clones, 12% of the clones exhibited wilt alone and 22% showed root borer infestation

alone. However, only 2% of the clones were affected by wilt and root borer together and 13% with wilt and other pests. (Viswanathan, unpublished). Overall, the association of root borer and wilt has been found to be variety specific. Viswanathan (2013a) observed that the popular sugarcane varieties from the tropical region succumb to wilt and root borer in the subtropical region in India as compared to the subtropical varieties. In addition to root borer (Agnihotri and Singh, 1989) association of scale insect and stalk borer have been reported to aggravate wilt infection. Other biotic agents, pathogens causing ratoon stunting, red stripe and leaf scald were also found to favour wilt (Viswanathan, 2013a).

Abiotic factors

The wilt pathogen survives in the soil and injury to the underground portion of stalk facilitates its entry. Experimental evidences are available on the influence of soil conditions such as moisture, pH and organic matter content on pathogen perpetuation and pathogenesis. Moisture stress during summer months coupled with high day temperature and low humidity may favour increase in wilt incidence. Drought at pre- and post-inoculation periods influenced the wilt severity significantly and varietal behaviour differs depending on the stress conditions even in uninoculated conditions. Variation in symptom development was found to be high under normal conditions followed by drought (Viswanathan *et al*., 2012, 2015). Abiotic factors like severe water logging also caused serious outbreak of the disease in the cv. Co 86032 in the tropical region.

Management

Wilt remains a serious production constraint in different parts of sugarcane growing regions in the country. Essentially it is due to the following reasons (i). Giving priority to red rot resistance while releasing sugarcane varieties and ignoring its wilt susceptibility, (ii). Influence of borer pests in inducing wilt under field conditions which was not anticipated while releasing new varieties, (iii). Climatic or soil factors which predispose sugarcane to wilt build up in both endemic and non-endemic regions. If wilt resistance is not assured in the varieties under cultivation an integrated disease management is to be practiced to sustain sugarcane cultivation.

Cultural practices

As vegetative propagation in sugarcane favours harbouring of the pathogen in the planting setts, adequate care should be taken while selecting seed canes. Monoculture of sugarcane over large areas provides conditions favourable for the rapid buildup of inoculum in the soil. Crop rotation

with paddy is recommended to reduce soil-borne inoculum of *F. sacchari* under puddled conditions. In ratoons, buildup of disease increases in successive years hence, when the disease is noticed in the plant crop it should be harvested on priority basis and ratooning should be avoided. Since wilt severity is predisposed by water logging and drought and borer pests, adequate care need be taken to minimize them. Providing optimum irrigation and drainage wherever required would be of paramount importance to manage the disease in endemic locations.

Chemical control

Ganguly (1964) reported that wilt incidence under field conditions can be appreciably reduced by applying 40ppm boron or manganese to wilt sick soils or by sett treatment. Later, Singh *et al.* (1985) reported that soil amendment with boric acid (15kg/ha) and sett treatment with aretan (0.1%) followed by 0.1% carbendazim considerably reduced wilt incidence and pathogen population. It has been found that dipping the setts in Carbendazim (0.1%) helps to prevent entry of the pathogen through cut ends. By applying Chloropyriphos 20EC or Confidor @ 0.5L a.i./ha or Furadon 3G or Fipronil @1.5 kg a.i./ha during last week of August root borer can be managed. Managing root borer by these insecticides helps to control wilt in the cv Co 89003 in subtropical India (Viswanathan and Padmanaban, 2008). Since the planting materials harbour the pathogen, detailed studies were conducted during 2015-16 season to manage the disease through fungicides and the results revealed that the untreated setts failed to germinate whereas the fungicide treatment Carbendazim (0.1% w/v) for overnight before planting recorded better germination of more than 20%.

Biological control

Bhatti and Chohan (1970) indicated possibility of wilt suppression using *Bacillus* and *Streptomyces* antagonists. 'Pressmud' is a by-product of sugar mills is a good organic substrate and its composition suits well for the establishment of *Trichoderma* on soil. The antagonistic *Trichoderma* isolates multiplied on pressmud is effective in managing wilt in endemic locations (Viswanathan *et al*., 2012). This approach is a viable strategy to combat wilt in disease endemic regions. Recently, we have identified two more effective *T. harzianum* isolates Th1 and Th2 which exhibited hyperparasitism against *F. sacchari* isolated from sugarcane at Coimbatore. Application of *Trichoderma* formulation in the rows at the time of planting @ 1 kg/3 m row at the time of planting significantly reduced wilt infections in the field (Viswanathan *et al.,* 2014).

Pokkah boeng

Pokkah boeng (PB) in sugarcane was first characterized and observed by Walker and Went (1896) in Java. The term '*pokkah boeng*', originally derived from 'Javanese term' stand for malformed or distorted top. Wind borne fungus settle in cane spindle causes the disease. Generally, a hot humid and rainfall condition favours the disease development and early stages of sugarcane were more prone to PB infection than the matured canes (Martin *et al.,* 1961; Raid and Lentini, 1991). In India, the disease was first recorded during 1930s and in 1940s. Earlier severe incidences of the disease were reported from Maharashtra and subsequently PB was reported from other states (Patil *et al.,* 2007; Viswanathan, 2012a,b). Vishwakarma *et al.* (2013) recently reported that the PB severity increased in the country based on detailed surveys during 2007-2013. Barnes (1974) reported that PB was one of the serious diseases of sugarcane and farmers often worry by its sudden spectacular appearance in their fields (King *et al.,* 1953). The disease occurs throughout the sugarcane growing countries in the world. Recently in China, PB has been reported as a major threat in sugarcane cultivation (Lin *et al.,* 2014).

Disease status

The disease occurs throughout the sugarcane growing regions in India. During the last 6-7 years PB occurred very severely across the country and few varieties succumbed to the disease in the field after their release for commercial cultivation. For e.g. the recently released high quality variety Co 99004 did not attain commercial status due to its susceptibility to PB. Many popular sugarcane varieties under cultivation in India exhibited PB from traces to 25%. In severe cases, top rot phase also recorded (Viswanathan and Rao, 2011). Recent field surveys conducted by Sharma *et al.* (2014) revealed 1.4-30% PB recorded in six popular sugarcane varieties in Uttar Pradesh.

The disease is one of the most serious fungal diseases in most, if not all, sugarcane-producing areas of the world (Whittle and Irawan, 2000). In most of the cane growing countries where the disease has been reported, the disease was of little effect in economic importance. However, the disease has the potential to arrest the crop growth temporarily and if the disease results in top rot phase, crop growth ceases and leads to loss of millable canes. The disease severity varies depending on the phase or symptom expression of the crop. In the past, considerable loss in the cv. POJ 2878 to a tune of 38% reduction was recorded to cane yield in Java due to high susceptibility to the disease and dry weather condition followed by a wet

season favoured the disease (Martin *et al.,* 1961). Patil *et al.* (2007) reported such favourable climate prevails in Maharashtra, India where infection is initiated after pre-monsoon showers and the disease prevails up to September. In the susceptible cultivars, PB causes yield losses ranging from 40% to 60% (Goswami *et al.,* 2013). Nearly 40.8-64.5% of sugars reduced in the canes affected with PB (Dohare *et al.,* 2003). The PB affected canes showed reduction in the weight, length of internodes, cane girth, juice, pol per cent and total sugars in juice than healthy canes (Singh *et al.,* 2006). The severe PB infection in stalk region, drastically reduced the internodal elongation of the canes (Viswanathan *et al.,* 2014). Due to the severe occurrence of the disease in different regions, the farmers were forced to take up fungicidal sprays to save the crop in parts of Tamil Nadu. It was also suspected that the PB infection may lead to wilt development in certain varieties (Viswanathan, 2012a,b).

Pathogen

PB is caused by the *Fusarium* species complex and association of different species of *Fusarium* was reported in the past. Initially the pathogen responsible for PB disease was reported as *Gibberella fujikuroi* (Sawada) in 1904. *F. sacchari,* which is closely related to PB caused by *F. verticillioides* occasionally, occurred on stems close to the top of plants (Sheldon, 1904). Later, in Java, Bolle (1927) found that PB disease is caused by *F. verticillioides* (*F. moniliforme*). Association of several species of *Fusarium*, including *F. verticillioides* (*F. moniliforme*) (Martin *et al.,* 1989; Mohammadi *et al.,* 2012), *F. sacchari* (Nordahliawate *et al.,* 2008), *F. proliferatum* and *F. subglutinans* (Khani *et al.,* 2013), *F. verticillioides* or *F. subglutinans* (McFarlane and Rutherford, 2005), *F. sacchari, F. proliferatum,* and *F. andiyazi* (Govender *et al.,* 2010) were reported. Gitatgong (1980) identified *F. verticillioides* and *G. fujikuroi* as the casual organism of PB on sugarcane in Thailand. Lin *et al.* (2014) identified *F. verticillioides* and *F. proliferatum* as the causal agents of PB in China. In Australia, *F. verticillioides* and *F. subglutinans* were the closely related to the PB. Under Indian conditions, we found association of *F. sacchari* and *F. proliferatum* with the disease (Viswanathan *et al.,* 2017). In South Africa, Govender *et al.* (2010) reported cause of typical PB symptoms by *F. andiyazi* and *F. sacchari.* The predominant species, *F. andiyazi*, was reported for the first time in sugarcane. Recently, Khani *et al.* (2013) categorized *Fusarium* spp. associated with PB in Iran into *F. verticillioides* (*F. moniliforme*), *F. proliferatum. F. subglutinans* and *F. semitectum* with 55%, 21.5%, 17.6% and 5.9% frequencies, respectively. Rosas-Guevara *et al.* (2014) reported association of *F. verticillioides* and *F. proliferatum* with

sugarcane PB samples collected from Veracruz, Morelos, Oaxaca and Puebla states of Mexico.

Recently about 79 isolates associated with PB in sugarcane were characterized at ICAR-SBI based on ITS sequencing. Among them, majority matched to *G. moniliformis* followed by *G. sacchari*, *G. thapsina*, *G. intermedia*, *F. oxysporum*, *F. pseudoanthophilum*, *F. subglutinans*, *Fusarium* sp. and *Gibberella* sp. (Scindiya, 2013). This study also revealed that ITS genome sequencing and their comparison with database sequences does not give reliable grouping of the isolates. Based on the morphological criteria (Leslie and Summerell, 2006) and molecular phylogenetic analysis, the pathogens of sugarcane PB belonged to two species, *F. verticillioides,* closely related to *F. sacchari* and *F. proliferatum,* closely related to *F. fujikuroi*.

Epidemiology

PB in sugarcane is common during monsoon/post monsoon months in the field in different parts of the country. It is highly influenced by the favourable climatic conditions *viz*., high humidity and temperature (Viswanathan, 2012a). The symptoms of this disease start to develop during 3-5 months old canes in which rainfall favours the infection of the pathogen. The symptomatic stages originated with young leaves or top portion of a plant or start to become chlorosis. After the infection, on the basal areas of young leaves as they emerge from the spindle became twisted, wrinkled and shortened. Earlier three phases of PB viz., chlorotic phase, acute phase and knife cut phase were described (Patil *et al.,* 2007) and recently Viswanathan (2012a) updated the different phases of disease symptomatology. In the recent years alarmingly high PB incidences were recorded from different parts of India (Viswanathan, 2012b) and severe occurrence of PB in the cvs. Co 0323, Co 95020 and Si 2002-02 in Tamil Nadu was recorded (Viswanathan, 2012b).

Pokkah boeng phase

The affected plants will be distinct and can be very easily noticed from a distance. The affected leaves in the crown show chlorotic patches at the base of the young leaves and occasionally on the other parts of the leaves and leaf sheath. Later, the disease expression is characterized by a pronounced yellowing, wrinkling, twisting and shortening of the leaves accompanied with malformation or distortion of young leaves (Fig. 5). Break-off of leaf lamina at malformed laminar region may also be seen during this stage. Occasionally the new emerging leaves will be destroyed

completely and only a projecting spindle core will be seen in the crown (Fig. 6). Knife-cut phase is seen during severe disease expression, where the fungus causes infection on stalk tissues. In this phase, shortened internodes exhibit varying levels of knife-cut symptoms due to partial or irregular infections of the pathogen.

Fig. 5. Characteristic twisted top symptoms with foliage deformation in sugarcane

Fig. 6. Severe pokkah boeng symptoms in sugarcane – severe reduction in leaf lamina

Top rot phase

The acute or top-rot phase is the advanced or very severe stage of the disease. Infection in the spindle may reach the growing point and growing point is killed, leading to development of top rot. The affected plant exhibits a dead-heart symptom in the crown with a whip like dried tissue in the spindle (Fig. 7). After killing the growing point, the pathogen may spread beneath the cane, becomes systemic and the top leaves may show yellowing. Although the prominent disease symptoms are noticed during 4-7 months stage, the symptoms can be seen throughout the crop stages. During the maturity phase, the newly initiated tillers also exhibit characteristic PB symptoms of leaf twisting, shortening and binding of the leaves.

Fig. 7. Top rot phase of pokkah boeng disease in sugarcane

Symptom recovery

Most of the PB affected canes generally recover from the disease symptoms; however, recovery of disease symptoms does not occur in case of top rot phase. Upon recovery we notice the normal whorl with remnants of twisted leaf portions of affected leaves still swing around the spindle with severe to moderate reduction in internodal elongation. The disease severity is directly related to retardation of internodal elongation in the crop and in case of severe infections, many internodes are shortened which significantly affect the cane yield (Viswanathan, 2012a, 2013a). Different weather parameters influence disease development and build up in sugarcane. Studies conducted at Shahjahanpur in Uttar Pradesh revealed that the maximum PB symptom intensity occur at maximum temperature of 32.8°C, minimum temperature of 26.3°C, relative humidity of 83.0 % and 489.0 mm rainfall in July month during 2013-14 season. However, in the tropical conditions of Andhra Pradesh, the disease incidence was initiated during the first fortnight of June and its severity gradually increased till November. The disease incidence was positively correlated with the number of rainy days, low temperature and high humidity.

Screening for PB resistance in sugarcane

PB is managed effectively by cultivating disease resistant varieties. During the course of breeding cycle susceptible clones are rejected and resistant ones are advanced. However, the newly released varieties with resistance to PB succumb to the disease probably due to a different environment or existence of a different pathotype. As the disease severity increased over the years in the country, a screening methodology has been developed to assess disease severity under natural conditions and the clones are rated as resistant (0-5%), moderately susceptible (5-10%), susceptible (10-20%) and highly susceptible (> 20%) in 11 sugarcane research centres (Viswanathan, unpublished). Nordahliawate et al. (2008) adopted two inoculation methods, spindle inoculation and soaking of single buds in the conidial suspension to simulate the disease in sugarcane. Recently, Das *et al*. (2015) attempted four methods of inoculation to assess sorghum genotypes for PB resistance under field conditions and found stem injection produced highest disease incidence. Viswanathan *et al.* (2014) conducted a detailed investigation on epidemiology of PB and wilt diseases in 600 sugarcane parental clones and found 32% of them were resistant, 17% moderately resistant, 20% moderately susceptible, 18% susceptible and 13% highly susceptible.

Disease management

Currently, the disease management option for control of the disease caused by *Fusarium* is the application of fungicides but the consistent application of fungicides will affect the environment and also it may produce unintended consequences on non-target organisms (Benitez *et al.,* 2004). Recent studies involved biocontrol and biosurfactant based methods to suppress the pathogen. Goswami *et al.* (2014) found inhibitory effect of rhamnolipid biosurfactant produced by *Pseudomonas aeruginosa* against *F. sacchari* to control PB and therefore seems to be a good biocontrol agent to control PB of sugarcane. The disease at times becomes severe during vulnerable phase of the crop coincides with conducive climate. Hence fungicide sprays become inevitable to reduce the disease intensity. Spraying of contact fungicide Mancozeb @ 0.25 % or systemic fungicide Carbendazim @ 0.1% was effective to reduce the disease severity under field conditions. If the disease is expected in the crop season, sett treatment with Carbendazim 0.1% followed by foliar spray of the same fungicide is recommended.

Relation between Fusariums causing wilt and PB in sugarcane

The diseases of sugarcane in which species of *Fusarium* are involved include those listed as PB, stalk rots or wilt and seed-cane rots, however, the strains involved might be different (Martin *et al.,* 1961). It is well known that *Fusarium* causes two different diseases and two different fungal species *F. sacchari* and *F. verticilloides* were associated with these diseases, respectively. Except the work done by our group, only limited information is available on the wilt associated pathogen(s) in sugarcane. Other than *F. verticilloides* and *F. sacchari* many *Fusarium* spp. were reported to cause PB as mentioned earlier. However, information on whether the root/stalk pathogen *F. sacchari* that is infecting the stalks systemically causes foliar infections or *F. verticilliodes* infecting leaf/spindle tissues enters inside the stalk and causes wilt systemically is not available. *F. sacchari* infected setts serve as the primary source of pathogen inoculum and it remains inside the canes for months before breaking out as a disease depending on the prevailing weather, growth stage and variety. Similarly, PB pathogen spreads mostly through aerial route, lands on the foliage during monsoon season and initiates the disease (Whittle and Eravan, 2000) and there are chances for dissemination of *F. verticillioides* through infected canes.

Recently the senior author has witnessed wilt development after PB infection in some of the sugarcane varieties. In this situation, initially typical symptoms of PB like shortening of leaves in the crown with deformation and chlorotic patches with irregular black streaks on the leaf lamina were observed. Later, the affected and other matured leaves show yellowing and this leads to systemic yellowing and drying of canes with typical wilt symptoms inside the canes (Viswanathan, 2013a). Since *Fusarium* spp are associated with both the diseases, it is likely that the same pathogenic strain may cause two different diseases in sugarcane. Probably *F. sacchari* causing wilt may find infection through aerial route or the systemically infected sugarcane may initially cause PB and later wilt. The studies conducted at the Institute revealed that apart from *F. verticilloides, F. sacchari* also recovered from PB infected cane samples (Viswanathan, 2013a).

Earlier studies of Govender *et al.* (2010) in South Africa revealed that PB associated *F. sacchari* caused characteristic bend symptoms in sugarcane stalks. Similarly findings of Khani *et al*. (2013) revealed that *Fusarium* spp isolated from PB-affected leaves caused mild to moderate symptoms on the stalks as reddish purple discolouration in Iran.

Nordahliawate *et al*. (2008) found that only isolates of *F. sacchari* caused PB in sugarcane in pathogenicity studies in Malaysia and isolates of *F. proliferation* and *F. subglutinans* were not pathogenic. They also found that *F. sacchari* inoculation caused death of pathogen-inoculated plants. In these studies, although they did not explore the possibility of wilt development in sugarcane by the PB associated *Fusarium,* the results showed damages caused by the fungal isolates on the stalk tissues. Probably here also the plants may have apparent systemic infection caused by *F. sacchari* as seen under Indian conditions systemic infections. Viswanathan et al. (2017) recently discovered that same sugarcane plant exhibits both the diseases together, inflicted by the same pathogenic strain of *F. sacchari*. Probably, the widespread occurrences of these diseases in different states in the country during the last 7-8 years may be due to the cause of the two diseases by the same fungal pathogen.

Conclusion

Wilt of sugarcane is a major disease affecting cane productivity in India and its impact to sugarcane cultivation was recorded nearly 100 years ago. The disease is widespread in the country. Although the disease was reported a century ago, no serious effort was made in the country, to critically study the etiological agent. Our recent studies found variation in virulence among the isolates and Koch postulate was proved using specific molecular markers. However, existing variability of the pathogen from different regions need to be critically documented and detailed studies are required on pathogenicity of the isolates. Also there is a need to develop specific molecular marker to identify *F. sacchari* isolates which are pathogenic on sugarcane.

The disease could not be reproduced at ease under field conditions using a standard inoculation technique. Artificial inoculation experiments under disease endemic and non-endemic regions clearly revealed that a specific soil/environment are required for disease reproduction in sugarcane. Hence there is a need to develop a region-specific disease-simulation technique for the disease. It was clear from the field observation that the disease could be managed successfully in disease susceptible cultivars by reducing the associated biotic factor, root borer. However, this opportunity is restricted to those varieties susceptible to both the agents.

During the recent years sudden outbreak of PB across the country was noticed on several varieties (Viswanathan, 2012b). It was found that the same *F. sacchari* isolate causes both PB and wilt in certain sugarcane varieties (Viswanathan *et al.,* 2017) and further studies are needed on

epidemiology of the same pathogen causing two diseases in sugarcane. Past experiences indicate that occurrence of wilt and PB in sugarcane is rapid and unpredictable due to changes in environmental conditions and this area needs special attention to identify specific edaphic and environmental factors influencing disease development. Sugarcane wilt fits to be an ideal candidate to study the impact of climate changes on disease buildup and development of epidemics in the future. Recent studies indicate that *F. sacchari* and other *Fusarium* spp. infecting sugarcane also infect other crops. Hence critical monitoring of sugarcane associated *Fusarium* spp. in sugarcane ecosystem is needed to establish epidemiology of wilt and PB in sugarcane and to device efficient management system for these diseases.

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References

Abbott EV (1932). Seed rots of sugarcane in Louisiana. *Proc. Inter. Soc. Sugar Cane Technologists*, 4th Congress Bulletin, 48: 1-2.

Agnihotri VP (1983). *Diseases of Sugarcane*, Oxford and IBH publishing Co., New Delhi, India, p. 363.

Agnihotri VP and Rao GP (2002). A century status of sugarcane wilt in India, pp 145-160 in *Sugarcane Crop Management*, edited by S.B. Singh, G.P. Rao and S. Eswaramoorthy, Houston, USA: SciTech Publishing LLC.

Agnihotri VP and Singh RP (1989). Sugarcane wilt current status, pp 227-237 in *Prospective in Plant Pathology,* edited by V.P. Agnihotri, N. Singh, H.S. Chaube, U.S. Singh and T.S. Dwivedi. New Delhi: Today and Tomorrow's printers and publishers.

Barnes AC (1974). The Sugarcane, 2nd edition, Leonard Hill, Ltd, London.

Bhatti DS and Chohan JS (1970). Antagonism of certain microorganisms to *Cephalosporium sacchari. J. Res.* (PAU) 7: 631-635.

Bolle PC (1927). An investigation into the cause of pokkah boeng and top rot. Archief Suikerindustrie Nederlands-Indie 111, 35: 589-609.

Bourne BA (1922). Researches on the root diseases of sugarcane. Department of Agriculture, Barbados, 1-17.

Bourne BA (1953). Studies on dissemination of sugarcane diseases. *The Sugar Journal* 16: 19.

Butler EJ (1906). Fungus diseases of sugarcane in Bengal. *Memoirs Depart. Agri. (India)*. Botany Series 1: 2-24.

Butler EJ and Khan AH (1913). Some new sugarcane diseases. Part I, Wilt, *Memoirs of Department of Agriculture*, India, Botany Series, 6: 180-190.

Das IK, Talwar HS and Rakshit S. 2011. Pokkah boeng disease of sorghum. *Jowar Samachar* 8: 3-4.

Das IK, Rakshit S and Patil JV (2015). Assessment of artificial inoculation methods for development of sorghum pokkah boeng caused by *Fusarium subglutinans*. *Crop Protec.* 77: 94-101.

Dohare S, Mishra MM and Kumar B (2003). Effect of wilt on juice quality of sugarcane. *Ann. Biol.* 19: 183-186.

Edgerton CW and Moreland CC (1920). Effect of fungi on the germination of sugarcane. *Louisiana Agric. Exper. Stat. Bull.* 169: 1-40.

Gams W (1971). *Cephalosporium*-artige Schimmelpilze (Hyphomycetes). Stuttgart, Germany: Gustav Fischer Verlag.

Ganguly A (1964). Wilt, pp 131-135, in *Sugarcane Diseases of the World Volume II*, edited by C.G. Hughes, E.V. Abbott and C.A. Wismer, Amsterdam: Elsevier Publishing Co.

Ganguly A and Khanna KL (1955). Annual report of the scheme for investigation and control of wilt disease of sugarcane. Annual report (1954-55) Sugarcane Research Station, Pusa, Bihar (India), 53 pp

Gerlach W and Nirenberg HI (1982). The genus *Fusarium* - A Pictorial Atlas. Mitt Bio Bundesanstalt fur Land- u Forstw (Berlin-Dahlem) 209: 1-406.

Ghannam IAY, Roaiah HF, Hanna MM, El-Nakkady SS and Cox RJ (2014). Identification, crystal structure and antitumor activity of Fusaric acid from the sugarcane fungal pathogen, *Fusarium sacchari*. *Intern. J. Pharm. & Technol.* 6: 6528-6535.

Giatgong P (1980). Host index of plant diseases in Thailand (2nd edn), Dept. of Agriculture, Ministry of Agriculture and Cooperatives, Bangkok, Thailand.

Goswami D, Handique PJ and Deka S (2014). Rhamnolipid biosurfactant against *Fusarium sacchari*-the causal organism of pokkah boeng disease of sugarcane. *J. Basic Microbiol.* 54: 548-557.

Govender P, McFarlane SA and Rutherford RS (2010). *Fusarium* species causing pokkah boeng and their effect on *Eldana saccharina* Walker (Lepidoptera: *Pyralidae*). *Proc. South African Sugar Technol. Assoc.* 83: 267 - 270

Gupta MR and Gupta SC (1976). Assessment of losses caused by wilt disease of sugarcane in western U.P. *Indian Sugar* 26: 151-152.

Han Y, Sun B, Hu X, Zhang H, Jiang B, Spranger MI and Zhao Y (2007). Transformation of bioactive compounds by *Fusarium sacchari* fungus isolated from the soil-cultivated ginseng. *J. Agri. Food Chem.* 55: 9373-9379.

Khambalkar PB, Bharose AA, Gawande ND and Manape TK (2014). Identification, characterization and molecular validation of pokkah boeng disease causal organism (*Fusarium moniliforme*) of *Sorghum bicolor* in Maharashtra. *Trends Biosci.* 7: 2578-2582

Khani KT, Alizadeh A, Nejad RF and Tehrani AS (2013). Pathogenicity of *Fusarium proliferatum*, a new causal agent of pokkah boeng in sugarcane. *Proc. Intern. Soc. Sugar Cane Technol.* 28: 1–5.

Khanna KL and Chacravarti AS (1949). Studies on storage of gur. *Curr. Sci.* 18: 127-128.

King NJ, Mungomery RW and Hughes CG (1953). Manual of cane-growing. Sydney:Angus, 349pp.

Kirtikar, Singh GP and Shukla R (1972). Role of seed material in carryover of wilt disease of sugarcane. *Indian Sugar* 22: 89-90.

Kulshreshtha, JPA and Avasthy PN (1959). An estimate of damage caused to sugarcane crop by the stalk borer *Chilo traeaauricilia* Dudg. *Proc. All India Conf. Sugarcane Res. Deve. Workers* 3: 27-28.

Leslie JF (1993). Fungal vegetative compatibility. *Annu. Rev. Phytopathol.* 31: 127-150.

Leslie JF and Summerell BA (2006). The *Fusarium* Laboratory Manual. UK: Blackwell Publishing Ltd, 388 pp.

Leslie JF, Summerell BA, Bullock S and Doe DJ (2005). Description of *Gibberella sacchari* and neotypification of its anamorph *Fusarium sacchari*. *Mycologia* 97: 718-724.

Lin Z, Xu S, Que Y, Wang J, Comstock JC, Wei J, McCord PH, Chen B, Chen R and Zhang M (2014). Species-specific detection and identification of *Fusarium* species complex, the causal agent of sugarcane pokkah boeng in China. *PLoS ONE* 9: e104195. doi:10.1371/journal.pone.0104195

Lyrene PM, Dean JL and James NI (1977). Inheritance of resistance to pokkah boeng in sugarcane crosses. *Phytopathology* 67: 689-692.

Martin J, Handojo H and Wismer C (1989). Pokkah boeng. 157-168, in *Diseases of sugarcane: Major diseases*. New York: Elsevier.

Martin JP, Hong HL and Wismar CA (1961). Pokkah boeng. pp. 247-257 in *Sugarcane Diseases of the World Volume 1*. New York: Elsevier Publ. Co.

McFarlane S and Rutherford R (2005). *Fusarium* species isolated from sugarcane in KwaZulu-Natal and their effect on *Eldana saccharina* (Lepidoptera: Pyralidae) development *in vitro*. *Proc. South African Sugar Technol. Assoc.* 79: 120-123.

McFarlane SA, Govender P and Rutherford RS (2009). Interactions between *Fusarium* species from sugarcane and the stalk borer, *Eldana saccharina* (Lepidoptera: Pyralidae). *Ann. Appl. Biol.* 155: 349-359.

Mohammadi A, Nejad RF and Mofrad NN (2012). *Fusarium verticillioides* from sugarcane, vegetative compatibility groups and pathogenicity. *Plant Protec. Sci.* 48: 80-84.

Mohanraj D and Alexander KC (1984). A qualitative numerical index for rating severity in sugarcane wilt. *Indian Sugar Crops J.* 10: 19-20.

Munawar A. Marshall JW, Cox RJ, Bailey AM and Lazarus CM (2013). Isolation and characterization of a ferrirhodin synthetase gene from the sugarcane pathogen *Fusarium sacchari*. *Chembiochem* 14: 388-394.

Nirenberg H (1976). Mitt. Biol, Bundesanstalt. Land-U. Forstw. Berling-Dahlem, 169:117.

Nordahliawate MSS, Izzati M, Azmi A and Salleh B (2008). Distribution, morphological characterization and pathogenicity of *Fusarium sacchari* associated with pokkah boeng disease of sugarcane in Peninsular Malaysia. *Pertanika J. Trop. Agric. Sci.* 31: 279-286.

Oren L, Ezrati S, Cohen D and Sharon A (2003). Early events in the *Fusarium verticillioides*-maize interaction characterized by using a green fluorescent protein expressing transgenic isolate. *Appl. Environ. Microbiol.* 69: 1695-1701.

Patil AS, Singh H, Sharma SR and Rao GP (2007). Morphology and pathogenicity of isolates of *Fusarium moniliformae* causing Pokkah boeng of sugarcane in Maharashtra, pp. 234-263, in *Microbial Diversity: Modern Trends*, edited by R.C. Ram and A Singh. New Delhi: Daya Publishers.

Petrovic T, Burgess LW, Cowie I, Warren RA and Harvey PR (2013). Diversity and fertility of *Fusarium sacchari* from wild rice (*Oryza australiensis*) in Northern Australia, and pathogenicity tests with wild rice, rice, sorghum and maize. *Euro. J. Plant Pathol.* 136: 773-788.

Poongothai M, Viswanathan R, Malathi P and Ramesh Sundar A (2014a). Sugarcane wilt: Pathogen recovery from different tissues and variation in cultural characters. *Sugar Tech* 16: 50-66.

Poongothai M, Viswanathan R, Malathi P and Ramesh Sundar A (2014b). *Fusarium sacchari* causing sugarcane wilt: variation in morphological characteristics of the pathogen. *Intern. Sugar J.* 116: 54-63.

Poongothai M, Viswanathan R, Malathi P, Ramesh Sundar A, Naveen Prasanth C and Balaji CG (2015). Genetic relatedness among *Fusarium* populations associated with sugarcane wilt in India: bridging molecular variability and phylogenetic diversity. *J. Sugarcane Res.* 5: 33-48.

Raid RN and Lentini RS (1991). Pokkah boeng. in *Florida Sugarcane Handbook* edited by R.A. Gilbert (Online). (Accessed 28th December 2015). http://edis.ifas.ufl.edu/SC004

Rands RD and Abbott EV (1938). Sugarcane diseases in the United States. *Proc. Intern. Soc. Sugar Cane Technol.* 6: 202-212.

Rao GP and Agnihotri VP (2000). Wilt, pp 193-197, in *A guide to sugarcane diseases*, edited by P. Rott, R. Bailey, J.C. Comstock, B. Croft and S. Saumtally. Montpellier: CIRAD/ISSCT.

Rashid A and Singh N (2002). Role of plant parasitic nematodes on the incidence and severity of sugarcane wilt. *Sugar Tech* 4 (l&2): 61 - 62

Rosas-Guevara V, Hernández-Arenas M, Miranda-Marini R, Bravo-Mosqueda E and Berriozabal-Onofre A (2014). Identification and morphological variability of pokkah boeng (*Fusarium* spp) on sugar cane, in Mexico. *Investigación Agropecuaria* 11: 119-126.

Sardhana HR, Singh N and Tripathi BK (2000). Investigation on the relationship between root borer and wilt disease of sugarcane. *Indian J. Entomol.* 62: 11-17.

Sarma MN (1976). Wilt disease of sugarcane. *Sugarcane Pathol. Newsl.* 15/16: 30-33.

Scindiya M (2013). Molecular characterization of *Fusarium* species associated with wilt and pokkah boeng/top rot diseases of sugarcane. M. Phil thesis, Bharathiar University, Coimbatore, 105pp.

Sharma DDK, Bharti YP, Singh PK, Shukla DN and Kumar A (2014). Studies on prevalence and identification of new races of *Fusarium moniliforme* Sheldon incitant of pokkah boeng disease from Uttar Pradesh. *Global J. Biol. Agri. Health Sci.* 3: 53-61

Sheldon JL (1904). A corn mold (*Fusarium moniliforme*). *New Agr Exp Sta Ann Report*, 17: 23-32.

Simasalit S (1996). Effects of metabolites from sugarcane red rot wilt pathogens on disease induction and influence of nitrogen fertilizers on growth and population of *Fusarium moniliforme* Sheld. *AGRIS record:* 53-61.

Singh A, Chauhan SS, Singh A and Singh SB (2006). Deterioration in sugarcane due to pokkah boeng disease. *Sugar Tech.* 8: 187-190.

Singh K and Singh RP (1974). Involvement and pathogenicity of *Acremonium* in wilt syndrome of sugarcane. *Sugarcane Pathol. Newsl.* 11/12: 24-25.

Singh K, Singh N, Singh RP and Mishra SR (1980). Development of wilt disease in sugarcane through sett and soil inoculations. *Sugarcane Pathol. Newsl.* 24: 23-25.

Singh O and Waraitch KS (1981). Effect of wilt and red rot induced stress on quality deterioration of sugarcane. *Sugarcane Pathol. Newsl.* 27: 25-30.

Singh OP (1973). Wilt assumes a serious problem in the sugarcane riverine belt of Saraswati Sugar Mills Area, Yamunanagar, Ambala. *Indian Sugar* 23: 129-130.

Singh RP and Singh N (1975a). An observation on the association of *Fusarium moniliforme* with sugarcane wilt. *Indian Phytopath.* 28: 271-272.

Singh RP and Singh K (1975b). Pathogenic potential of wilt pathogens of sugarcane and their ecological relationship with *Colletotrichum falcatum*. *Proc. Deccan Sugar Technol. Assoc.* 9: 69-75.

Singh V and Joshi BB (2007). Mass multiplication of *Trichoderma harzianum* on sugarcane press mud. *Indian Phytopath.* 60: 530-531.

Subba Raja KT and Natarajan S (1972). Sugarcane wilt caused by *Cephalosporium sacchari* and *Fusarium moniliforme* in India. *Sugarcane Pathol. Newsl.* 8: 21-23.

Vishwakarma SK, Kumar P, Nigam A, Singh A and Kumar A (2013). Pokkah Boeng: An emerging disease of sugarcane. *J. Plant Patho. Microbiol.* 4: 1000170

Viswanathan R (2010). Plant Disease: Red rot of Sugarcane. New Delhi: Anmol Publishers, 301 pp.

Viswanathan R (2012a). *Sugarcane Diseases and Their Management*, Coimbatore: Sugarcane Breeding Institute, 140pp.

Viswanathan R (2012b). Need for a paradigm shift in sugarcane disease management, pp 171-206, in *Perspectives in Sugarcane Agriculture*, edited by N.V. Nair, D. Puthira Pratap, R. Viswanathan, J. Srikanth, A. Bhaskaran and Bakshi Ram. Coimbatore: Society for Sugarcane Research and Development.

Viswanathan R (2013a). Status of sugarcane wilt: one hundred years after its occurrence in India. *J. Sugarcane Res.* 3 (2): 86-106.

Viswanathan R (2013b). Sustainable ecofriendly disease management systems in sugarcane production under the changing climate – A review. *J. Mycol. Plant Pathol.* 43: 12-27.

Viswanathan R, Balaji CG, Selvakumar R, Malathi P, Ramesh Sundar A, Prasanth CN, Chhabra ML and Parameswari B (2017). Epidemiology of *Fusarium* diseases in sugarcane: a new discovery of same *Fusarium sacchari* causing two distinct diseases, wilt and pokkah boeng. *Sugar Tech* 19: 638-646

Viswanathan R, Malathi P, Ramesh Sundar A, Poongothai M and Singh N (2006). Current status of sugarcane wilt in India, *Sugar Cane Intern.* 24: 1-7.

Viswanathan R, Malathi P, Annadurai A, Naveen Prasanth C and Scindiya M (2014). Sudden occurrence of wilt and *pokkah boeng* in sugarcane and status of resistance in the parental clones in national hybridization garden to these diseases. *J. Sugarcane Res.* 4: 62-81.

Viswanathan R and Padmanaban P (2008). *Hand Book on Sugarcane Diseases and their Management.* Coimbatore: Sugarcane Breeding Institute, 80pp.

Viswanathan R, Poongothai M and Malathi P (2011). Pathogenic and molecular confirmation of *Fusarium sacchari* causing wilt in sugarcane. *Sugar Tech* 13: 68-76.

Viswanathan R, Poongothai M, Malathi P and Naveen Prasanth C (2015). Sugarcane wilt: Simulation of pathogenicity through different methods and environments. *Intern.l Sugar J.* 117: 286-293.

Viswanathan R, Poongothai M, Malathi P and Ramesh Sundar A (2012). Sugarcane wilt: New insights into the pathogen identity, variability and pathogenicity, pp 30-39, in *Functional Plant Science and Biotechnology 6 Special Issue 2*, edited by R. Viswanathan and A. R. Sundar, Ikenobe, Japan: Global Science Books.

Viswanathan R and Rao GP (2006). Developing disease resistant varieties and methods of disease screening in sugarcane, pp 759-800, in *Sugarcane: Crop Production and Improvement,* edited by S.B. Singh, G.P. Rao, S. Solomon and P. Gopalasundaram. Houston, USA: Studium Press LLC.

Viswanathan R and Rao GP (2011). Disease scenario and management of major sugarcane diseases in India. *Sugar Tech* 13: 336-353.

Walker JH and Went FAFC (1896). Overview of the diseases of sugarcane in Java. *Arch. Suikerind. Ned. Indie IV*, 427-435.

Wang JH, Peng XD, Lin SH and Wu AB (2015). First report of Fusarium head blight of wheat caused by *Fusarium sacchari* in China. *Plant Dis.* 99: 160

Waraitch KS (1981). Wilt disease in Co 1148 in Punjab and assessment of losses caused by it. *Indian Sugar* 31: 37-40

Whittle PJL and Irawan (2000). Pokkah boeng, pp 136-140, in *A guide to sugarcane diseases*, edited by P. Rott, R.A. Bailey, J.C. Comstock, B.J. Croft and A.S. Saumtally. Montpellier, France: CIRAD / ISSCT.