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RESEARCH ARTICLE

SUDDEN OCCURRENCE OF WILT AND *POKKAHBOENG* IN SUGARCANE AND STATUS OF RESISTANCE IN THE PARENTAL CLONES IN NATIONAL HYBRIDIZATION GARDEN TO THESE DISEASES

R. Viswanathan[1*], P. Malathi[1], A. Annadurai[2], C. Naveen Prasanth[1], M. Scindiya[1]

Abstract

Wilt and *pokkahboeng* (PB) caused by *Fusarium sacchari* and *F. verticilloides* in sugarcane are considered to be minor diseases at Coimbatore, Tamil Nadu State, south India. However, during the 2009-10 season sudden outbreaks of these diseases appeared in National Hybridization Garden (NHG) where 608 sugarcane hybrid clones were maintained to perform hybridization. Detailed investigations were carried out on the associated pathogens, disease epidemiology, disease resistance in the clones and their management. During 5-6 months stage, initial symptoms of PB were noticed and gradually PB severity increased from chronic to acute phase along with top rot symptoms. As the days progressed affected canes exhibited wilting along with or without top rot symptoms. When the disease severity increased from resistant to susceptible, the affected clones exhibited combined infections of PB and wilt symptoms. Among the different phenotypes of PB and wilt in the clones, top rot and wilt caused more damage in the clones. However, some of the clones exhibited severe wilt alone with more crop loss and severe wilting, causing extensive damage to some of the established varieties and parents. Fusaria isolated from the affected tissues were found to be pathogenic in artificial testing. The parental clones in the NHG exhibited a clear variation in their resistance to PB, top rot and wilt. The clones originating from the subtropical region, except those from Uchani, were found to have more number resistant types as compared to those from the tropical states. Although the disease situation was precarious, it was successfully managed by an integrated approach involving chemicals and biocontrol agents in the subsequent seasons. Further studies are in progress to characterize the associated pathogens and their interrelationship in causing PB, top rot and wilt diseases in sugarcane.

Key words: Sugarcane, wilt, *pokkahboeng, Fusarium sacchari*, *F. verticilloides*

Introduction

More than 200 diseases were recorded worldwide in sugarcane (Rott et al. 2000) and about 55 diseases of economic importance were recognized in India (Viswanathan and Rao 2011). Among the major diseases reported in the country, wilt is a serious disease affecting cane production and productivity in different varieties (Viswanathan 2013a). The disease was responsible for the elimination of many commercial varieties from cultivation in disease endemic regions in the country. The disease severity was confined to the east coastal regions, south Gujarat and subtropical plains whereas upland regions in the states of Tamil Nadu, Karnataka, Maharashtra and Andhra Pradesh were relatively free from disease severity or showed sporadic incidence (Viswanathan et al. 2006; Viswanathan 2013b). Occasionally, aggravating factors like

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drought, waterlogging, root borer and scale infestations favoured its severity in these regions. Although there were conflicting views on the etiological agent, recent studies clearly proved *Fusarium sacchari* as the causative fungus (Poongothai et al. 2014a,b; Viswanathan et al. 2011, 2012). *Pokkahboeng* (PB), another *Fusarium* disease reported from all sugarcane-growing regions, was considered a minor disease. The causative fungus *F. verticillioides* (*F. moniliforme*) infects young leaves and causes malformation with twisted top during monsoon season in the country and usually the affected varieties recover from the disease after the monsoon (Viswanathan 2012a). However, in the recent years PB appeared in severe form in different states causing serious damage to cane cultivation (Viswanathan 2012b, 2013a,b).

It is now well known that *Fusarium* causes two different diseases, one in stalk and the other in leaves/spindle, and two different species, namely *F. saccahri* and *F. verticilloides*, respectively were associated with these diseases. 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. *Fusarium 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 and crop stage. Similarly, *F. verticilloides* 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 the fungus through infected canes. During 2009-10 season, we encountered a serious outbreak of PB and wilt in the National Hybridization Garden (NHG) of the Sugarcane Breeding Institute, Coimbatore, where more than 600 parental populations were maintained for effecting hybridization during October-December. Though Coimbatore is a non-endemic region for both the diseases, it was puzzling to discover the sudden occurrence of these diseases. Apart from independent infections of PB and wilt in certain varieties, there were combined infections of PB, top rot (TR) and wilt in many of the clones and such clones suffered greater damage than those that harbored separate infections. Hence, we conducted detailed investigations on epidemiology of these diseases in NHG clones, characterization of the associated pathogens, simulation of the disease and their management.

Materials and methods

Status of PB, top rot and wilt in NHG clones

Since the first disease symptoms noticed in June 2010, parent materials maintained in NHG was surveyed for the onset, severity and phases ofPB, top rot and wilt symptoms at weekly intervals. For each clone, infection due to PB, top rot, top rot + wilt and wilt was recorded on clump basis to generate data on disease status. Based on the expression of PB, top rot, wilt and their combinations, the clones were categorized as disease free, less than 5% disease, 5-10%, 10-20% and more than 20% anddesignated as resistant (R), moderately resistant (MR), moderately susceptible (MS), susceptible (S) and highly susceptible (HS), respectively. Finally, the disease status was assigned to each of the 619 clones which were then grouped according to symptoms and source of origin of the clones.

Pathogenicity studies

The affected cane tissues such as stalk, root, PB infected leaves, top rot affected spindle, etc.were subjected to pathogen isolation following standard mycological techniques. In addition, top rot affected tissues were subjected to bacterial isolation on Nutrient Agar medium. The recovered *Fusarium* isolates were purified and maintained for further pathogenicity studies. For pathogenicity studies the pathogenic isolates recovered from spindles of Co 8371 and Co 99004 were multiplied on oatmeal agar and conidial suspension was prepared. The suspension (1 ml) was poured into the axils of 5 to 6 crown leaves of potted plants (cvCoC 671)and incubated in a high humidity disease testing chamber during 2011-12 season. Phenotypic expression of disease symptoms was recorded periodically in the inoculated plants.

Planting of setts in wilt sick soil

The infectivity of wilt infected soil and stubbles after the harvest of the cloneswas assessed in a field experiment. Soil and stubbles from wilt affected sugarcane plots of varieties Co 62198, Co 85002 and Co 86002 in NHG were applied in 30 cm deep pits (100 cm diameter) of 15 cm height during 2011-12 and 2012-13 seasons. Two budded setts of seven varieties were planted in the pits and observations on germination and disease development were monitored regularly. After the disease development, the diseased cane tissues were subjected to pathogen isolation and confirmation. Inoculum-free control pits were also maintained in the experiment.

Biological control

Trichoderma harzianum isolates (Th1 and Th2) from sugarcane rhizosphere available in the collection of Plant Pathology lab and *Trichoderma* isolates obtained from infected canes, viz. Co 740, Co 86002, Co 89003 and Co 98017 were screened against four *Fusarium* isolates from stalks of Co 86002 (FS1) and Co 95020 (FS2), and spindle leaf of Co 99004 (FS3) and Co 8371 (FS4), isolated during the investigation by conventional dual plate assays. Based on the assays, two *T. harzianum* isolates, namely Th1and Th2 were selected for mass multiplication and field application. *Trichoderma* for field application was prepared by incorporating talc formulation with FYM (1:10) that was incubated under shade with optimum moisture for 3 months.

Results

Disease epidemiology

Initially, symptoms of PB with foliage discolouration and drying were noticed during third week of June. Subsequently detailed epidemiological investigations were carried out.

Pokkahboeng

Both chronic and acute phases of the disease were recorded in the affected clones from 5 to 6 months after planting. In the chronic phase, typical twisted top with varying levels of leaf deformities was found (Fig. 1). Recovery of the disease was observed in the newly emerging spindle leaves in many of the canes (Fig. 2). On close examination, the malformed leaves showed extensive lesions on leaf base causing white patches, blackening of affected laminar region and extensive black veinal necrosis (Fig. 3). These

Fig. 1. Typical twisted top with varying levels of leaf deformities in *pokkahboeng* affected sugarcane

Fig. 2. *Pokkahboeng* infected sugarcane plant showing recovery of symptoms in newly emerging spindle leaves

Fig. 3. Severe *pokkahboeng* infected plant showing severe malformed leaves

Fig. 4. Impact of *pokkahboeng* on sugarcane: severe internodal shortening (infected cane seen with a healthy cane in the field)

phenotypic symptoms remained static in tolerant varieties but in susceptible varieties severely necrotized leaves showed paleness, yellowing and drying. Due to severe PB infection, internodal elongation was drastically reduced in the affected stalk region. Depending on the severity of PB, up to five or six internodes showed shortened internodes (Fig. 4). However, in a variety like Co 8371, axillary buds also sprouted due to PB infection and in many varieties including CoC 671 the severely affected leaves turned yellowish and such discolouration spread to other leaves in the canopy leading to subsequent drying of such leaves. Stalks remained healthy in such canes without apparent wilt and most of the affected clones showed this type of leaf yellowing/drying.

Top rot

The acute phase of the disease resulted in top rot phase, whichwas characterized by rotting of growing point / spindle leaves. Most of the PB-infected canes generally recovered from the symptoms, but top rot phase never recovered from the damage. Top rot phase was an intriguing phase in PB/wilt epidemiology in sugarcane. Many of the top rot affected clones showed death of emerging leaves inside the crown and formed a whip-like dried spindle (Fig.5). Sometimes partial blight symptoms similar

Fig. 5.Top rot affected clones showing death of emerging leaves inside the crown and forming a whip-like dried spindle

to leaf scald blights on the lamina were also seen. Such blights of 2.0-2.5 cm width ran parallel to mid rib in the affected leaves and such symptoms were also seen in the axillary bud sprouts. Bud sprouting was very common in most of the top rot affected canes unless the disease turned to wilt phase.

Wilt

Although expression of wilt is expected during maturity stages of the crop, we recorded the disease during 5-6 months crop age. The infected canes were conspicuous due to stunted growth, paleness and withering/ drying of the canes in a background of healthy canes / resistant varieties. The varieties such as Co 86002, Co 89003 and C79218 exhibited typical wilt and ~80% of the canes of Co 86002 and C79218 showed wilt with typical paleness of rind, pith formation/discolouration inside the canes (Fig. 6a, b). All the affected canes showed death of spindle in these varieties. Although Co 89003

Fig. 6a. Sugarcane cv Co 86002 exhibiting severe wilt with typical rind paleness and cane drying in the field (healthy canes with vigorous growth seen in the foreground)

Fig. 6b. Internal symptoms of wilt affected sugarcane exhibiting tissue discolouration and cavity formation

showed typical wilt, it was not severe as in the other two varieties. Initially, drying or chlorotic discolouration of the youngest leaf in the whorl occurred and unlike top rot, there was no rotting of spindle tissues found in case of wilt. In the affected canes, the weakened spindle did not grow further and without new leaves, the canes became stunted with withering of leaves.The wilted canes were free from root borer infestation and this indicated no association with other biotic agents in aggravating wilt infection. Wilted canes never recovered and that led to extensive foliage drying. In severe cases, dried canes showed cut-off symptoms atnodes (Fig. 7a,b). Occasionally the wilt-infected canes of cv Co 86032 remained partially turgid with discoloured foliage till harvest. In such plants, the crown remained green and other leaves showed prominent yellowing/drying. Leaves of many wilt-infected plants exhibited a distinct pinkish red or pinkish purple discolouration on yellow background (Fig. 8).

Fig. 7a.Severely wilt affected clone exhibiting extensive foliage discolouration and drying

Fig. 7b. Extensive loss to cane stand due to wilt (dead canes are cut-off at nodes to show remaining few internodes)

Fig. 8. Distinct pinkish red discoloration of lamina tissue in wilt-affected canes

Spread of PB, top rot and wilt among the parental clones

Overall, 198 (32%) of 619 parental clones were free from these diseases and could be considered as resistant (R); ~105 (17%) clones maintained very low disease of 0.1-5.0% (MR) and 134 (20%) recorded the diseases to the tune of 5.1-10.0% (MS) (Table 1). The clones recorded more than 10% disease incidence suffered severely. Nearly 18% of the clones recorded disease incidence of 10-20%

Table 1. Distribution of resistance to wilt and *pokkahboeng* diseases in parental clones of sugarcane

Sl. No	Group / Name	Number of clones	Disease-free / resistance	Disease incidence (%)			
				<5	5.1 to 10.0	10.1 to 20.0	>20
1	Coimbatore (Co)	152	36	21	37	34	24
2	Andhra Pradesh						
	Anakapalle (CoA/A)	33	2	6	7	8	10
	Vuyyuru (CoV/V)	9	0	1	3	5	0
	Tirupati (CoT)	1	0	0	0	1	0
	Rudrur (R)	15	2	2	4	6	1
3	Assam (CoBln)	18	1	1	6	8	2
4	Bihar (BO/CoP)	27	20	4	2	0	1
5	Gujarat (CoN)	7	3	1	2	1	0
6	Haryana (CoH)	23	3	7	5	5	3
7	Karnataka (CoSnk)	5	4	0	0	1	0
8	Kerala (CoTl)*	11	1	1	1	4	4
9	Madhya Pradesh (CoJaw/CoJn)	9	3	3	1	1	1
10	Maharashtra (CoM)	12	1	2	4	5	0
11	Punjab (CoJ)	26	13	3	4	2	4
12	Tamil Nadu						
	Cuddalore (C/CoC)	24	3	4	5	6	6
	Sirugamani (CoSi)	4	0	1	3	0	0
13	Uttarakhand (CoPant)	21	9	7	2	3	0
14	Uttar Pradesh						
	Lucknow (CoLk)	24	13	6	3	2	0
	Lucknow (LG)	38	24	6	3	4	1
	Shajahanpur (CoS)	51	28	10	8	4	1
	Seorahi (CoSe)	13	5	4	3	0	1
15	Other clones						
	UP	6	4	1	0	0	1

Table 1 Continued

Sl. No	Group / Name	Number of clones	Disease-free / resistance	Disease incidence (%)			
				<5	5.1 to 10.0	10.1 to 20.0	>20
	CoL	2	0	2	0	0	0
	ISH	43	9	6	9	6	13
	HR	2	1	0	0	1	0
16	Foreign hybrids						
	CP	6	2	2	1	1	0
	Q	5	2	1	1	1	0
	POJ	2	1	0	1	0	0
	SP	3	0	1	2	0	0
17	Miscellaneous**	16	5	2	2	3	4
		608	195	105	119	112	77

*- Also contained few named parents

**- Comprises seedlings, selections, MS, KHS, KMS, Barbados, etc.

(S) and 13% of the clones recorded incidence of more than 20.1% (HS).

The varieties Co 98013 and CoA 07322 recorded more than 50% infection of wilt and PB and suffered in the same manner as Co 86002. Apart from these, many other popular varieties/ promising clones such as Co 740, Co 62198, Co 7527, Co 8371, Co 85019, Co 94012, Co 93009, Co 94008, Co 0121, Co 0122, CoA 89081, CoA 99081, CoC 671, CoC 771, CoC 8201, CoC 86062, BO 106, CoJ 83, CoJ 86, CoBln 03176,CoBln 05502, etc. suffered in the field mainly due to combined infections of PB and wilt. The clones from Thiruvalla, Kerala, such as CoTl 85120, Mathurima and Madhumathi were also severely affected. The recent introductions such as CoS 07233, CoA 07321, CoA 07322, LG 95037, Co 2000-10, Co 98010, Co 98007, CoA 96081, etc. also succumbed to the diseases. Five clones (MS 68/47, ISH 7, ISH 11, CoA 99081 and Co 62399) exhibited severe PB but had no wilt infection. Although disease severity was high in these clones with damage to vegetative growth and death of growing meristems, they escaped total crop loss due to wilt.

Most of the clones from Buralikson, Assam, and Vuyyuru, Andhra Pradesh, succumbed to these diseases. Although all the advanced varieties are screened for wilt resistance at Navsari in Gujarat, three of the eight clones in the NHG succumbed to the diseases. About 55 clones from four research stations of Andhra Pradesh are maintained at NHG and of them only four were R and more than 50% of the clones were S to the disease. Nearly 10 of them like CoA 89081, 91A37, 74A96, CoA 96081, 70A2, CoA 07321 and CoA 07322 recorded very severe disease in the field. Similarly, among the 28

clones originating from Cuddalore and Sirugamani stations in Tamil Nadu, only three were R and 15 others were S. Six of them, namely C79218, CoC 671, CoC 771, CoC 8201, CoC 86062 and CoC 774 were severely affected and found to be HS.

Nature of wilt and PB infections in sugarcane clones

Comparison of *Fusarium* infected clones for various symptoms such as PB, top rot, wilt, top rot + wilt and PB + top rot + wilt indicated a proportionate increase in percent infected clones in all these categories with the increase in severity of the disease(s) (Table 2). Increase in disease incidence among the clones was greater and drastic for wilt than for PB . In less than 5% category (MR), only 31.7% of the clones were wilt infected and the infected clones increased to 60, 79 and 90% when severity categories of MS, S and HS, respectively. Similarly, in case of top rot alsothe infected clones increased from 62 to 92%. Although there was an increase in PB incidence in the clones during severity, number of affected clones was less as compared to top rot and wilt in HS types. Nearly most of the caneswere infected by top rot or wilt in HS types, indicating that more damage is caused by these phases of disease development.

When specifically examined for separate infections of PB, TR and wilt phases of the diseases across MR, MS, S and HS types, it was clear that combined infections were less in MR/MStypes and it proportionately increased in S and HS types. For example, in HS category, only C 79218, Co 740,Co 86002 and CoH 7802 exhibited wilt whereas in MR category about 15 clones, namelyCo 8208, Co 8316, Co 91018, Co 93020, Co 0120,CoC 92061, CoJn 86272, CoL 29, CoLk 8001, CoPant 01215,CoSi 776, CP 52-256, Q 68 ISH 43 and Thirumarutham recorded only wilt without symptoms of PB or TR. Similarly, in case of PB, none of the clones in HS category was infected with PB alone whereas in MR category about 17 clones recorded PB only butremained free from TR or wilt. In addition, combined infections of TR + wilt and PB+TR+wilt also increased proportionately from MR to HS categories. This information clearly indicated that the susceptible clones succumb to infection of Fusaria through root, stalk, leaves and crown, i.e. they are vulnerable to soil borne and air borne inocula of Fusaria.

Table 2. Status of combined infections of wilt and *pokkahboeng* in sugarcane parental clones

Disease severity	Number of clones	PB-1	PB-2	Top rot	Top rot + wilt	wilt	PB + TR + Wilt
< 5 %	107	25	12	67	5	34	4
5-10%	121	29	12	95	17	73	10
10-20%	111	39	23	95	42	88	33
> 20 %	75	37	19	69	45	68	27

Resistance sources of sugarcane clones in NHG to *Fusarium* diseases

Among the 619 parental clones maintained in the NHG, 'Co' varieties constituted the maximum of ~25%, followed by the clones of CoS, ISH, BO, CoBln, CoJ, CoLk, LG, CoPant, CoH, CoC,CoA, CoV, Rudrur, etc. (Table 3). Comparison of resistance to wilt and PB in NHG clones revealed that 'BO' varieties such as BO 32, BO 43, BO 47, BO 54, BO 68, BO 89, BO 91, BO 92, BO 94, BO 96, BO 99, BO 102, BO 108, BO109, BO 110, BO 120, BO 128 and BO 130 were found to be resistant (R) and they contributed maximum proportion R types. About 20 of the 27 BO varieties were found to be resistant to wilt and PB. Similarly, LG (63%), CoS (54%), CoLk (54%), CoJ (50%), CoPant (42%) and CoSe (38%) clones constituted higher proportions of R types. However, only 23% of Co vareities such as Co 356, Co 976, Co 1053, Co 7204, Co 7717, Co 7910, Co 8210, CoJ 82315, Co 8318, Co 8339, Co 8341, Co 8353, CoS 86218, Co 87267, Co 87268, Co 87269, Co 87270, Co 87271, Co 87272, Co 87273, Co 88039, Co 89029, CoBln 90006, Co 91010, Co 91019, Co 95024, Co 98016, Co 0117, Co 0118, Co 0124, Co 0237, Co 0327, Co 0331, Co 0239, Co 05009 and Co 05011 were diseaee-free and behaved resistant to wilt and PB. Most of the ISH clones and derivatives of inter-specific hybrids, expected to have high levels of resistance were, however, susceptible to the diseases. Although only few foreign hybrids were maintained, many of them such as B38192, CP 44-101, CP 52-68, POJ 2802, Q 63 and Q 65 were found to be resistant.

None of the commecial vareities identified from Cuddalore in Tamil Nadu were resistant. However, three of their clones, namely C 79113, C 84028 and C 84070 were found to be resistant. A similar trend was noticed with the clones from Anakapalle, Andhra Pradesh, most of which were found to be susceptible except CoA 88081 and CoA 89085. Almost all the parental clones from Assam except CoBln 04131 have picked up the diseases indicating their high susceptibility.

The following parental clones from Punjab, namely CoJ 46, CoJ 64, CoJ 72, CoJ 75, CoJ 76, CoJ 79, CoJ 82, CoJ 86, CoJ 89, CoJ 82191, CoJ 83536, CoJ 84191, CoJ99192 (CoJ 88), Haryana, CoH 1, CoH 12, CoH 13, Madhya Pradesh CoJn 80141, CoJn 86141, 8 86310, Lucknow, CoLk 7810, CoLk 8002, CoLk 8102, CoLk 8901, CoLk 99001, CoLk 9116, CoLk 9414, CoLk 94184, CoLk 97009, CoLk 97022, CoLk 97023, and CoLk 97169, from Uttarakhand, namely CoPant 8202, CoPant 84211, CoPant 84214, CoPant 90223, CoPant 90224, CoPant 94213, CoPant 96219 and CoPant 99214, from Shahjahanpur, namely CoJ 67, CoS 109, CoS 510, CoS 687, CoS 767, CoS 796, CoS 802, CoS7927, CoS 8408, CoS 8436, CoS 8315, CoS 8407, CoS 87231, CoS 88216, CoS 90269, CoS 91269, CoS 93278, CoS 94192, CoS 94270, CoS 96268, CoS 96275, CoS 00221, CoS 02264, CoS 96258, CoS 96260, CoS 96265, CoS 96269 and CoS 07282, from Seorahi, namely CoSe 92423, CoSe 95422, CoSe 92423, CoSe 95423 and CoSe 97248, from Sankeshwar, namely CoSnk 03061, CoSnk 03707 and CoSnk 05103, Navsari, CoN 95132, CoN 03132, CoN 05072 and CoN 91132, from Pusa, namely CoP 9301 and CoP 9302 and CoTl 85118 (Thiruvalla) and CoM 95132 (Padegaon) were resistant. Other clones such as UP 1, UP 5, UP 6, UP 22, UP 39, UP 48, UP 40, UP 0097 and UP 9530 from Shahjahanpur

Table 3. Status of wilt and *pokkahboeng* incidence in sugarcane parental materials maintained at National Hybridization Garden

Disease-free	<5%	5-10%	10-20%	>20%
BO 32, BO 43, BO 47, BO 54, BO 68, BO 89, BO 91, BO 92, BO 94, BO 96, BO 99, BO 102, BO 108, BO 109, BO 110, BO 120, BO 128, BO 130, Co 356, Co 976, Co 1053, Co 7204, Co 7717, Co 7910, Co 8210, CoJ 82315, Co 8318, Co 8339, Co 8341, Co 8353, CoS 86218, Co 87267, Co 87268, Co 87269, Co87270, Co87271, Co87272, Co 87273, Co 88039, Co89029, Co 90006, Co91010, Co91019, Co 95024, Co98016, Co0117, Co 0118, Co0124, Co0237, Co 0327, Co 0331, Co 0239, Co 05009, Co 05011, C 79113, C 84028, C 84070, CoA 88081, CoA 89085, CoBln 04131, Co H 1, CoH 12, CoH 13, CoJ 46, CoJ 64, CoJ 75, CoJ 76, CoJ 79, CoJ 82, CoJ 86, CoJ 89, CoJ 82191, CoJ 83536, CoJ 84191, CoJ88, CoJn80141, Co	64A 30, 67A4, 85R186, BO78, BO 97, Co 0120, Co 06035, Co 06037, Co 419, Co 6806, Co 7219, Co 8208, Co 8213, Co 8214, Co 8316, Co 85033, Co 86011, Co 87012, Co 88013, Co 91018, Co 92008, Co 93020, Co 95005, Co 97015, Co 99006, CoA 8201, CoA 92082, CoA 95081, CoA 99082, CoBln 9104, CoC 777, CoC 8001, CoC 85061, CoC 92061, CoH 102, CoH 104, CoH 106, CoH 110, CoH 112, CoH 56, CoH 70, CoJ 68, CoJ 78, CoJ 87, CoJn 80-151, CoJn 82600, CoJn 862035, CoJn 862072, CoL 29, CoL 9, CoLk 8001, CoLk 91238, CoLk 9412, CoLk 9618, CoLk 97011, CoLk 99164, CoM 0265, CoM 7219, CoN 92133, CoP 9206, CoP 9401, CoPant 1215, CoPant84212, CoPant	70A5, 80 R 41, 83R23, 97 R 401,98 R 278, BO 17, C79180,Co0238,Co 0240, Co 05010, Co 06033, Co1148,Co312, Co 6304, Co 6904, Co 7201, Co 7314,Co7424, Co7915, Co8013,Co83 47,Co86036,Co86250, Co87002,Co87045,Co 87023, Co 87263, Co 88028, Co 89003, Co 89010, Co 90018, Co 92001, Co 92006, Co 92020, Co 94003, Co 94005, Co 97009, Co 98015, Co 98017, Co 99004, Co 99016, CoA 2000-082,CoA71,CoA 7602, CoA 7701, CoA 93081, CoBln 03172, CoBln 03175, CoBln 04172,CoBln 05501,Co Bln 9102, CoBln 94063,	2000 V 59, 2002 V 48, 69 A 5, 69 A 591, 79 A 271, 79 R 207, 79A28, 87R40, 88 R 13, 93 A 21, 93 V 297, 97 R 383, C 79017, C 81615, C81129, C 84206, Co 0116, Co 0241,Co06032,Co06036, Co 1158, Co 1307, Co 1305, Co 617, Co 62174, Co 6415, Co 7704, Co 7706, CoC 771, Co 775, Co 8209, Co 8338, Co 85002, Co 85246, Co 86010, Co 86032, Co 86249, Co 87021, Co 87025, Co 87252, Co 89036, Co 91002, Co 92002, Co 92013, Co 94012,Co95021,Co975, Co 98006, Co 98007, Co 98008, CoA 8401, CoA 8402, CoA 93082, CoBln 03-171, CoBln 03173,	70 A 2, 72 A 66, 74 A 96, 91 A 37, BO 106, C 79218, Co 453, Co 740, Co 7224,Co 7527, Co 0121, Co 0122, Co 2000-10, Co 62198, Co 62399, Co 8370,Co 8371, Co 85019, Co 85036, Co 86002, Co 87004, Co 88025, Co 93009, Co 94007, Co 94008, Co 98003, Co 98010, Co 98013, CoA 07321, CoA 07322, CoA 89081,CoA 92081, CoA 96081,CoA 99081, CoBln 03176, CoBln 05502, CoC 671, CoC 771, CoC 774, CoC 8201, CoC 86062,CoH 107, CoH 15, CoH 7802, CoJ 70, CoJ 72, CoJ 80, CoJ 83,CoJaw 70, CoS 07233, CoSe

Table 3 Continued

Disease-free	<5%	5-10%	10-20%	>20%
Jn 86141 , CoJn 86310, CoLk 7810, CoLk 8002, CoLk 8102, CoLk 8901, CoLk 9116, CoLk 9414,CoLk94184, CoLk97009, CoLk 97022, CoLk 97023, Co Lk 97154, CoLk 97169, CoM 95132, CoN03132 ,CoN05072, CoN91132, CoP9301, CoP9302, CoPant 8202,CoPant 84211,Co Pant 84214, CoPant 90223, Co Pant 90224, CoPant 94213,Co Pant 96219, CoPant99214, CoJ 67, CoS109, CoS510, CoS 687, CoS767, CoS796,CoS802,CoS 7927, CoS 8408,CoS 8436,CoS 8315,CoS 8407,CoS87231,CoS 88216, CoS 90269, CoS 91269, CoS 93278, CoS 94192, CoS 94270, CoS 96268, CoS 96275, CoS 00221, CoS 02264, CoS 96258, CoS 96260, CoS 96265, CoS 96269, CoS 07282, CoSe 92423, CoSe 95422, CoSe 92423, CoSe 95423, CoSe	84213, CoPant 88219, CoPant 88220, CoPant 97222, CoPant 98224, CoR 8001, CoS 06247, CoS 87216, CoS 92254, CoS 92263, CoS 94257, CoS 95255, CoS 97261, CoS 97264, CoS 99259, CoSe 93232, CoSe 95427, CoSe 96234, CoSe 96436, CoV 92103, CP 52-1, CP 52-256, ISH 147, ISH 192, ISH 28, ISH 43, ISH 52, ISH 9, LG 00116, LG 01030, LG 94114, LG 99112, LG 99114, LG 99190, Q 68, S 4491/03, Sel 943/98, SP 80-185, Thirumathuram	CoC773,CoC775,CoC 779, CoC 90063, CoH 114, CoH 119, CoH 14, CoH 35,CoH7803,CoJ 58,CoJ61,CoJ65,CoJ 73,CoJaw270,CoLk 9110, CoLk 97154, CoLk96029,CoM6806, CoM7712,CoN 04131, CoM88121,CoN05071, CoN 85134, CoP 9502, CoPant 92227, CoPant 99213, CoS 07231, CoS 770, CoS778,CoS8119, CoS 8432, CoS 91230, CoS 95270, CoS 98247, CoSe01268,CoSe95436, CoSe98231,CoSi6,CoSi 776, CoSi 86071, CoTl 85116, CoV92101,CoV 94101, CoV94102,CoV 98101, CP 63-326, ISH 118, ISH 128, ISH 139, ISH 175, ISH 229, ISH	CoBln 03-174, CoBln 04174, CoBln 90006, CoBln 9101,CoBln9103, CoBln 9605, CoC 772, CoC778,CoH76,CoH1, CoH92,CoH98,CoH99, CoJ 84291,CoJ 85,CoJn 86572, CoLk 9229, CoLk 95147, CoM 6615, CoM 7704, CoM 9206, CoM 9217, CoM 9220, CoN 08072, CoPant 1216, CoPant 92226, CoPant 94211, CoS 07234, CoS 611, CoS 633, CoS 673, CoSnk 03-44, CoT 8201, CoTl 85117, CoTl 85119, CoTl 85441, CoTl 85442, CoV 92102, CoV 09356, CP 61-23, HR 83-65, ISH 100, ISH 127, ISH 136, ISH 2, ISH 280, ISH-111, KMS 1142, KMS 881, LG 01016, LG 01170, LG	01256,CoTl 85120,ISH 7, ISH 11, ISH 18,ISH 20, ISH 58, ISH 101, ISH 110, ISH 131,ISH 156, ISH 157, ISH 170, ISH 301,ISH 307,KHS 3196, KMS 1185, LG 95037, Madhumathi, Mathurima, MS 68/47, MS 901, UP 9529

Table 3 Continued

Disease-free	<5%	5-10%	10-20%	>20%
97248, CoSnk 03061, CoSnk 03707, CoSnk 05103, CoTl 85118, CoV 07356, CP 44-101, CP 52-68, HR 83-144, ISH 12, ISH 153, ISH 176, ISH 41,ISH 23, ISH 65, ISH 135, ISH 228, ISH 287, PoJ 2802, Q 63, Q 65, NCo 310, UP 1, UP 5, UP 6, UP 22, UP 39, UP 48, UP 40, UP 0097, UP 9530, 88R58, 97R129, B38192, Akipura, S 4393/ 03, S 4509/ 03, LG 01118, LG 02100, LG 1002, LG 1009, LG 641, LG 7230, LG 94126, LG 94164, LG 97050, LG 95056, LG 95123, LG 96001, LG 96029, LG 96115, LG 97112, LG 97147, LG 9901, LG 99001, LG 99017, LG 9902, LG 991, LG 99122, LG 99017, LG 99183		267, ISH 306, ISH 69, KMS 2095, KMS 2422, LG 01014, LG72120, LG 99118, POJ 2878, Q 70, Sel 922/98, SP 80 - 3250, SP 83-5073, UP 49	72115, LG 95053, NB 94-545, Q58	

and LG 01118, LG 02100, LG 1002, LG 1009, LG 641, LG 7230, LG 94126, LG 94164, LG 97050, LG 95056, LG 95123, LG 96001, LG 96029, LG 96115, LG 97112, LG 97147, LG 97154, LG 99001, LG 99017, LG 9902, LG 991, LG 99122, LG 9917 and LG 99183 from Lucknow were also resistant to the diseases. Comparison of wilt status in the field very clearly revealed that most of the parental clones contributed by the subtropical stations such as Pusa, Shahjahanapur, Jalandhar, Seorahi and Pantnagar were resistant. However, contributions from Uchani in Haryana and Buralikson, Assam, were mostly susceptible to wilt and PB.

Pathogen isolation

Isolations were made from wilt-infected stalks (Co 86002, Co 740, Co 89003, Co 95020 and CoC 671) and top rot affected green (Co 8371) and yellow (Co 99004) spindles. Culture tests revealed typical *Fusarium* growth from tissue segments of affected leaves and roots of wilted canes. The pathogen recovery was 100% from spindle/leaf tissues and 20-30% from stalk tissues. *Fusarium* was invariably isolated from top rot or PB affected leaves and in wilted stalks *Trichoderma* was obtained occasionally along with *Fusarium* indicating invasion of saprophytic / antagonistic fungi in the wilted canes. *Fusarium* associated with PB, top rot and wilt were found to be *F. sacchari* and *F. verticilloides*. Since different bacteria were associated with top rots in sugarcane, efforts were made to isolate bacterial pathogens from top rot affected spindle tissues and we found none of the affected varieties showed association of bacterium with top rot.

Pathogenicity

When *Fusarium* isolates from affected leaves of Co 8371 and Co 99004 were inoculated on canes of CoC 671 by whorl method, typical yellowing of leaves in the crown was observed as in the field (Fig. 9). Unlike in the field conditions we did not observe typical PB symptoms of the disease.

Fig. 9. Reproduction of partial symptoms of *pokkahboeng* in CoC 671 showing typical yellowing of leaves in the crown after pathogen inoculation by whorl method. Left: field symptoms; right: induced symptoms

However, the inoculated canes imitated the typical yellowing of the leaves as observed in the field. In the second trial where infected soil and crop residues were applied in the field pits, almost all the susceptible varieties such as Co 6806, Co 62198, Co 85002, Co 86002, Co 86032, etc. recorded good germination as in the uninoculated controls. However, 60 days after germination the growing plants showed progressive yellowing of the foliage and subsequently exhibited wilting and drying of the plants in all these susceptible varieties (Fig. 10). The wilt resistant BO 91 remained healthy in the experimental field.

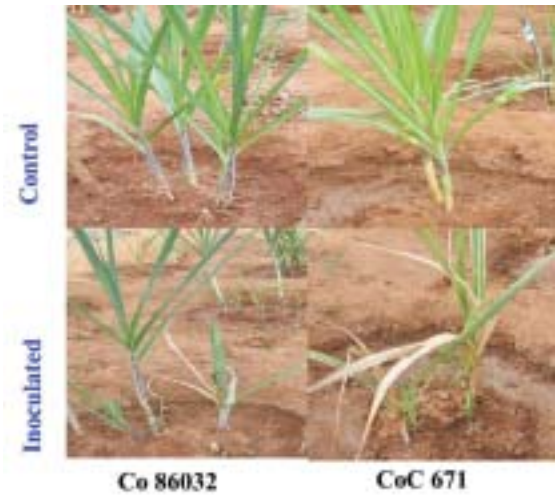

Fig. 10. Simulation of wilt in sugarcane by inoculating wilt-sick soil in the pits along with crop debris: wilt susceptible varieties Co 86032 and CoC 671 show progressive yellowing of the foliage and wilting 60 days after planting

Biological control

In bioassays, *T. harzianum* isolates Th1 and Th2 exhibited hyperparasitism against all the *Fusarium* isolates by 12th day. Hence, these two isolates were mass multiplied on FYM for field application. Application of *Trichoderma* formulation in the rows @ 1kg / 3 m row at the time of planting significantly reduced wilt infection in the field.

Discussion

Sugarcane flowers profusely and sets seed under Coimbatore conditions. In view of this a National Hybridization Garden was established at the Institute to extend hybridization facility to 23 State Sugarcane Research Stations in the country. Every year, over 500 bi-parental crosses, several polycrosses and general collections are carried out and fluff (true seed) is supplied under this programme to the stations. A large number of seedlings are raised from the fluff supplied by the Institute at different State Sugarcane Research Stations in the country. These are evaluated at different stages and potential clones which are adapted to the prevailing conditions in the states are identified for release (Nair 2008). The NHG Programme operated by the Institute has resulted in the development of a large number of prominent varieties such as CoA 89085, CoA 92081, CoC 671, CoC 85061, CoC 92061, CoG 93076, CoH 7801, CoH 8402, CoJ 46, CoJ 58, CoJ 64, CoJ 67, CoJ 76, CoLk 8001, CoLk 8102, CoM 88121, CoM 0265, CoPant 84211, CoPant 84212, CoPant 90223, CoS 510, CoS 611, CoS 633, CoS 687, CoS 767, CoS 802, CoS 8432, CoS 8436, CoS 88230, CoS 94257, CoS 95222, CoSe 92423, CoSe 92423, CoSi 86071, CoSi 95071, CoSi 96071, CoSi 6, CoTl 88322, 83 R 23, CoV 92102, CoV 94101, CoV 94102, CoV 09356, CoVc 89222, etc. in different states.

Since it is a national sugarcane improvement programme, the parental populations for this activity are being maintained with utmost care in NHG. Occurrence of PB and wilt in NHG to this scale was unexpected and the situation clearly gave an indication of the emergence of minor diseases as major diseases in sugarcane. During the same year, alarmingly high levels of PB incidence were recorded from different parts of the country (Viswanathan 2012b). We are not sure whether the severe occurrence of PB at Coimbatore has any significance to the national scenario. Incidentally, during the same period, severe occurrence of PB in Co 0323, Co 95020 and Si 2002-02 in other districts of Tamil Nadu was recorded (Viswanathan 2012a). Despite the fact that no severe wilt outbreaks were

recorded in the country during that time,we could still see high incidence of wilt in some of the susceptible clones in NHG. Another interesting observation made during the years was confinement of the disease complex to NHG and adjacent photoperiodic experimental field of Physiology Section. This indicates that the diseases are confined to the sugarcane fields with high genetic variability. Probably the clones with high susceptibility in these fields may have acted as an influencing factor in aggravating the disease. Among the different regions of sugarcane cultivation in India, viz. subtropical and tropical regions, relatively higher number of parental clones from the states of Bihar (Pusa), Punjab (Jalandhar), Uttar Pradesh (Shahjahanpur, Seorahi and Lucknow) and Uttarakhand (Pantnagar) in the subtropical region were resistant to the diseases. However, majority of the clones from the tropical states like Tamil Nadu (Cuddalore) and Andhra Pradesh (Anakapalle and Vuyyuru) were susceptible. This may be due to the presence of more *Saccharum spontaneum* genome in the subtropical selections (Selvi et al. 2005) which is considered the repository of genes for tolerance to pests and diseases (Nair 2012).

Artificial inoculation of the isolated pathogen from PB infected tissues caused partial symptoms of PB in susceptible variety (Fig. 9). Although stalk inoculation with wilt causing isolates did not reproduce the disease (R. Viswanathan, unpubl. data), the disease was simulated when wilt sick soil along with crop residues was placed in the field. Most of the susceptible varieties such as Co 6806, Co 62198, Co 85002, Co 86002 and Co 86032 picked up wilt inoculum from soil and disease infection progressed with ageing of the crop. This finding very clearly demonstrated the association of Fusaria with PB and wilt in sugarcane.

Among the clones, the popularly utilized parent Co 86002 suffered more severely and recorded >90% wilt. Although the variety is being cultivated in major areas in Gujarat, which is endemic to wilt, the variety remains free from the disease infection (R. Viswanathan, unpubl. data). Further, the variety did not exhibit any PB symptoms suggesting that wilt phase is predominant at Coimbatore in this variety. This situation suggests either the pathogenic strain prevailing at Coimbatore and Gujarat may be different or the variety would have succumbed to wilt without apparent PB symptoms. In addition to two stages of PB, Patil et al. (2007) additionally described a third phase of the disease termed 'knife-cut phase' based on the disease occurrence in Maharashtra. The symptoms include one to two or even more transverse angular cuts on the top portion of stalks as though the tissues are removed with a sharp knife. We have observed such severe symptoms in few clones indicating the severity of the disease and it is due to the partial damage of the growing meristem. Patil et al. (2007) opined aggressive strains of the pathogen would cause knife-cut infection while the non-aggressive strains cause chlorotic phase infection. Overall, exhibition of various stages of the disease depends on the strains of the pathogen, cane variety and prevailing weather conditions. Severe knife-cut phase of the disease was reported from Java, Florida and Brazil (Whittle and Eravan 2000). 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. Although the disease

was considered a minor disease, in the recent years the disease occurs in severe form throughout the country (Viswanathan 2012a). The disease has the potential to arrest the crop growth temporarily and if the disease results in top rot phase, crop growth would ceaseleadings to loss of millable canes. Furthermore, it is suspected that severe PB could result in wilting of canes and in such a situation complete loss to the crop occurs. Hence, severity of disease incidence varies depending on the phase or symptom expression of the crop.

In most of the cane growing countries where the disease has been reported, the disease was of little effect in economic importance. In the past, considerable cane yield loss (38%) in the cv POJ 2878 occurred in Java due to high susceptibility to the disease as dry weather condition followed by a wet season favoured the disease (Martin et al, 1961). Patil et al. (2007) reported such favourable climate prevailed in Maharashtra, where infection was initiated after pre-monsoon showers and the disease prevailed up to September. Although there is temporary reduction in cane growth, there will be substantial reduction in cane yield due to severe reduction in growth of internodes across the field (Fig. 4). Due to the severe occurrence of the disease in different regions, farmers were forced to take up fungicidal sprays to save the crop in parts of Tamil Nadu. It was also suspected that the *pokkahboeng* infection may lead to wilt development in certain varieties (Viswanathan 2012a,b).

Initial symptoms of PB are seen as chlorotic patches on the leaf lamina close to leaf sheath and they may extend further or show veinal infection as vascular streaks on the lamina (Viswanathan 2012a). During our investigation, we found profuse discolouration of entire leaves in PB-affected sugarcane varieties. Although such extensive discolouration of the leaves in the crown is associated with wilt, many PB affected varieties exhibited such symptoms and artificial inoculation of the recovered pathogen induced similar symptoms (Fig. 9). This situation further strengthens the opinion of the same fungus causing both PB and wilt depending upon the prevailing environment (R. Viswanathan, unpubl. data). *Fusarium sacchari* associated with wilt in sugarcane has also been reported to cause PB in Malaysia (Siddique 2007).

The source of inoculum for the sudden appearance of the diseases in NHG clones was hard to explain. One speculation was that a large plot of Co 99004 exhibiting 100% PB was grown very close to the NHG. The pathogen inoculum from the adjoining Co 99004 field was continuously available from planting onwards to the NHG that has wide genetic diversity of sugarcane varieties / genotypes. Hence, prolonged exposure of the pathogen inoculum to the young crop would have favoured disease development. However, Co 99004 rarely exhibits acute phase of PB and infections are confined to chronic phase. Further, this variety never shows wilt in the field. Since the region is far away from contiguous sugarcane area and close to urban locality and we suspect the possibility of Fusaria from Co 99004 causing severe disease infections in parental clones of sugarcane. We speculate that the inoculum causing PB in Co 99004 could have found more susceptible hosts among the NHG clones and hence serious manifestation of the disease was found across the clones. Probably the inoculum settled in the soil could have induced wilt through root

infections. Examination of wilt affected canes showed typical wilt in mother setts in Co 86002, C 79218 and Co 89003 (data not shown). Here the source of infection in mother setts could have come from sick soil or planting of wilt-affected setts derived from sugarcane stalks with quiescent infections of wilt.

Whatever may have been the source(s) of Fusaria, the damage caused to certain NHG clones was significant and these varieties never exhibited such a serious disease before. In contrast, some of the known susceptible varieties like Co 89003 recorded typical wilt in few clumps only. Since the disease severity was alarming in some varieties the healthy canes in them were separated, quarantined and carried forwardfor planting in the next season. Timely action taken to contain the diseases through an integrated approach of sett treatment with systemic fungicide before planting and application of *Trichoderma* formulated in organic substrate reduced the disease build up in the subsequent seasons. At the end of the fourth season (2012-13), the disease incidence was negligible in NHG (data not shown) and this could beattributed to the systematic seed cane selection and integrated disease management strategy.

Our earlier studies have shown existence of many non-pathogenic *Fusarium* in sugarcane that probably may have an excellent endophytic association with sugarcane (Viswanathan et al. 2011, 2012). The sugarcane associated *Fusarium* may move between pathogenic and non-pathogenic phases probably due to changes in environment. The changed environment favours the endophytic organism to switch from mutualistic to pathogenic mode. Endophytic association of *F. sacchari* in sugarcane was reported from South Africa (McFarlane and Rutherford 2005). Earlier work of Ma et al. (2010) on transposable elements points out that mobile chromosomes convert non-pathogenic strain into a pathogenic one. Further, they reported that LS regions of *F. oxysporum* f. sp. *lycopersici* causing tomato wilt are enriched for genes related to host–pathogen interactions and the mobilization of these chromosomes could, in a single event, transfer an entire suite of genes required for host compatibility to a new genetic lineage. If the recipient lineage had an environmental adaptation different from the donor, transfer could increase the overall incidence of disease in the host by introducing pathogenicity in a genetic background pre-adapted to a local environment. Such mechanism underlines rapid pathogen adaptation and onset of disease to an unexpected level.

Under field conditions, there could be a cocktail of Fusaria in the stalks and sugarcane rhizospheric soil.Vegetative propagation also favours maintenance of such varied fungal population in the plant / cropping system. Prevailing environment and predominance of a specific strain may influence disease development in the field. Further studies are required on identifying diversity of Fusaria in the sugarcane ecosystem and their pathogenicity potential. Additionally, environmental parameters influencing disease onset and development need detailed investigation. Although the disease outbreak was unexpected, during this investigation few positive aspects emerged. Firstly, we have identified resistant sources among the parents used in varietal development programme in the country to PB, top rot and wilt. Secondly, development of an integrated disease management strategy to counter the wilt

and PB outbreakswas another major outcome of the work. Further, wilt was artificially simulated using sick soil inoculation technique and partial symptoms of PB were induced through whorl method of inoculation. In future, possible recurrence of the diseases need to be monitored critically and impending issues on minor diseases becoming major diseases due to climate changes need detailed investigation.

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