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REVIEW

Changing dynamics of sugarcane red rot pathogen *Colletotrichum falcatum* since the historical disease outbreaks

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Red rot caused by *Colletotrichum falcatum* Went was reported more than 120 years ago in India and it continues to be a major threat to sugarcane cultivation in India and other Asian countries. The red rot epidemics were responsible for the elimination of many elite sugarcane varieties and the epidemics caused huge economic damages to sugarcane farmers and sugar industry. The failure of the varieties occurred due to 'varietal breakdown', by which the new pathogenic variants overcome the host resistance and made the varieties susceptible. Hence detailed studies were conducted on the pathogenic variation from 1940s onwards at different centres in the country. Earlier, the variants were categorized into 'light' highly sporulating and virulent and 'dark' isolates, less sporulating and less virulent. Large number of *C. falcatum* isolates were characterized for pathogenic variability on a set of host differentials and 13 pathotypes named as CF01 to CF13 with distinct pathogenicity profile were designated in the country during the last 30 years. The newly designated pathotypes were found to be virulent and represent the prevailing pathogenic population in the ecosystem. The newly designated pathotypes are used to screen new varieties before their release for commercial cultivation. The recently designated *C. falcatum* pathotype CF12 in the tropical region was found to be highly virulent over the erstwhile predominant pathotype CF06. Investigation on the current red rot epidemics and breakdown of the popular cv Co 0238 in the subtropical India revealed that isolates from the variety maintained a discrete pathogenicity pattern to infect the host cv Co 0238, whereas, the designated pathotypes from the region exhibited resistant reactions on the variety. In addition to characterizing pathogenicity patterns in *C. falcatum,* specific pathogenicity domains in the pathogen were identified after complete genome and transcriptomes studies.

Keywords : *Colletotrichum falcatum*, pathogenic virulence, pathotypes, red rot, sugarcane, varietal break down

INTRODUCTION

Red rot of sugarcane caused by the fungal pathogen *Colletotrichum falcatum* was first reported as a disease in sugarcane in Java in 1893 (Went, 1893). Within a few years after the first report, its economic damages to sugar mills in Java, its occurrence was reported in several other parts of the world.

These reports revealed that the disease was widely spread before it was accepted as a new disease of sugarcane in different countries like India, Australia, USA including Hawaii and mainland, Brazil, Mauritius, Philippines, South Africa, West Indies, etc (Singh and Singh, 1989, Viswanathan, 2010, 2021a).

In India, Dr C.A. Barber, the Imperial Botanist, Madras Presidency did foundation work on the disease when it struck for the first time in the Presidency (Barber, 1901, 1906). Later, Butler (1906), the Imperial mycologist, at the Imperial Agricultural Research Institute, Pusa, Bihar (India) conducted detailed studies of the disease on the causative fungus, its portals of entry into the cane stalk and he referred the disease as 'red rot' based on the most distinctive feature of rotting of internal stalk tissues with reddish tissue discolouration. Both Barber and Butler recognized the importance of the disease and devised management strategies of healthy seed and

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avoidance of waterlogging to reduce the crop damages in India. Severe red rot epiphytotics in the Godavari delta and North Indian plains caused extensive damages to sugarcane, however, this scenario resulted in the establishment of Sugarcane Breeding Institute (SBI) at Coimbatore, India in 1912 by Dr Barber to develop red rot resistant varieties through interspecific hybridization. Development of inter specific hybrids involving *Saccharum officinarum* and *S. spontaneum* from Coimbatore started from Co 205 in 1918a later hundreds of 'Co' varieties were released for commercial cultivation and adopted in India and in many other countries. The monumental work done by Dr Barber was instrumental in developing many such inter specific hybrids and notably his contributions to sugarcane has been recognized by naming one of the species of *Saccharum* native to India as *S. barberi* (Viswanathan, 2018, 2021a, b).

Economic impact to sugarcane

The disease caused extensive damage to *S. officinarum* clones under cultivation in the tropics and subtropics during 1890s and early decades of the 19th century. After the release of hybrid varieties starting from Co 205 and the varieties failed one after another after initial success under field conditions. During the 1938-39 season, a red rot epidemic of exceptional severity occurred in the subtropical region predominantly in Uttar Pradesh (UP) and Bihar, the major sugarcane region in India. This devastation resulted in failure of the major commercial variety Co 213, in which thousands of hectares were devastated. Due to the poor supply of canes, the sugar mills in the eastern UP crushed only one third of their normal canes during 1938-39 and half during 1939-40 (Chona, 1941). Severe infections of red rot can cause loss of nearly two third of cane stalks produced in subtropical India (Chona, 1980). In Pakistan, 28.5% losses in cane weight was reported at initial infection by the red rot pathogen and it reaches 82.7% when the disease intensity increased to 75% in the cvs L 116 and B 4360 *(*Ahmad *et al.* 1986). Sucrose content, the main economic produce is reduced in the range of 31 - 75% at different infection levels (Munir *et al.* 1986; Khan *et al.* 2011). Severe red rot epidemics in Peninsular India during 1990s caused losses of 30-50% in cane yield in the varieties like Co 6304, CoC 671, CoC 85061, CoC 86062, CoC 92061, CoSi 86071 etc. Yield losses of upto 100% were found in ratoon crops in different factory regions (Alexander and Viswanathan, 1996, 2002; Viswanathan *et al*. 1997; 2002). The recent red rot epidemics due to varietal breakdown of the popular variety Co 0238 has caused a catastrophic effect on sugar economy in the country and overall losses were estimated to about Rs 10,000 annually during the last 4 to 5 seasons (Fig. 1) (Viswanathan, 2021b; Viswanathan *et al*. 2022, 2024). Affected canes show complete rotting of cane tissues with reddish discolouration and characteristic white spots hence the canes become unsuitable for industrial use (Fig. 2).

Fig.1: Complete drying of red rot affected canes (cv Co 0238) in West Champaran Dt, Bihar

Fig 2: *C. falcatum* infection causes complete rotting of internal tissues and drying of canes

Pathogen

The anamorphic stage of the pathogen *Colletotrichum falcatum* F.A. Went is recorded

under field condtions. *Glomerella tucumanensis* (Spegazzini) von Arx & Muller, the Ascomycete fungus, is the teleomorph of *C. falcatum*. The fungus comes under Phylum: Ascomycota, Subphylum: Pezizomycotina, Class: Sordariomycetes and Family: Glomerellaceae. After describing the disease in detail with the symptoms, Went (1893), demonstrated the pathogenicity of the fungus he isolated from the diseased tissues, calling it *Colletotrichum falcatum* Went and further carried out life-history studies.

Variation in C. falcatum

Cultural characteristics

In USA, Abbott (1938) described *C. falcatum* colonies on oatmeal agar that consist of abundant aerial mycelium, broadly spreading, sometimes zonate, densely woven into compact, velvety turf in some isolates, or a cottony, floccose one in others, referred as dark and light races, respectively. The fungus colour ranges from almost white through light ashy grey to dark grey, growing darker with age (pale olive grey to pearl grey) with no colour or pigmentation in reverse or in the medium. Hyphae are densely interwoven anastamosing in definite ropes. Conidia develop in a pink or salmon coloured, water soluble, mucilaginous mass and when produced rapidly on the upper portion of the acervulus is covered with a shining droplet. The isolates show enormous phenotypic variation on the plates and broadly they are grouped as light and dark pathotypes; however there are intermediate types with variation for sporulation and colony characters (Viswanathan, 2021a). Several of the isolates studied at Coimbatore were able to produce acervuli in culture with diameter ranging from 0.639 to 1.54 mm and setae are found in all the isolates and in few cases it was rare. Number of setae ranged from 3 to 20 per acervulus and length range from 90 to 220 μm. Conidiophore length ranged from 120 to 330 μm. In the past, several authors have reported emergence of new isolates with variation in morphological and cultural characters in *C. falcatum.* However, the dimensions of all the isolates may fall within the broad range for conidial, acevuli, conidiophore and setae. Abbott (1938) observed the dark type isolates in synthetic medium that produce chlamydospores in much greater abundance than the light ones. Certain isolates of *C. falcatum* are reported to produce black hard structures i.e., stromatic bodies on culture media that consist of fertile hyphae, some of which perform true function as conidiophores and bear typical falcate conidia of *C. falcatum* at their tips. Earlier, a stroma forming strain of *C. falcatum* was reported from infected sugarcane varieties from Karnal, India (Chona *et al*. 1961a). It is presumed that they may play an important role in perpetuation of *C. falcatum*; however, the conditions which favour development of stroma in the field are unknown.Cultural instability of *C. falcatum* with the formation of sectors has been reported. Usually, the cultures grow less luxuriantly or become mycelial with low sporulation after a prolonged period of artificial cultivation. The successive batches of cultures of an isolate obtained at different times varied in sporulation. Abbott (1938) further reported that the isolates are morphologically stable without significant changes in virulence as a result of long continued cultivation on oatmeal medium. However, the author has recorded instability of the fungal cultures in the long run and they lost their virulence after repeated sub-cultures on oatmeal agar. Hence, many sugarcane pathologists pass the cultures through their respective host cultivars regularly, re-isolated and maintain in culture collections. Sometimes the poorly defined 'sectors' or 'patch' variants appear in the plates, but transfer of these sectors to fresh plates produce colonies apparently identical with the original. Numerous reports on physiology of *C. falcatum* including media, nutrients, pH, temperature, conidial germination etc were reported earlier in detail (Duttamajumder, 2002; Viswanathan, 2010) In the review, more emphasis is given on pathogenicity, pathogenic and molecular variation.

Although asexual phase of the pathogen is commonly recovered from the infected tissues, Spegazzini (1896) and Carvajal and Edgerton (1944) described and characterized the perfect state of the pathogen. In India also, occurrence of perfect state of the fungus was recorded both under field conditions and in culture (Chona and Bajaj, 1953; Chona and Srivastava, 1952; Chona

et al. 1961b). Duttamajumder (2002) made a detailed account of perfect state of the fungus in India. He felt that the red rot pathogen is basically a leaf parasite similar to *Colletotrichum* infecting sorghum and it completes its life cycle on sugarcane leaf. He also opined that so long as *C. falcatum* restricts itself on the mid-rib, it does not cause any significant harm to the cane crop. It was emphasised that most of the mid rib population survive on the mid-rib without causing much harm to sugarcane and only a few of them adopt to invade the stalk. However, studies conducted at Coimbatore revealed that most of the midrib isolates of *C. falcatum* from susceptible varieties are found to be more virulent than stalk isolates, which counters the previous notions (Viswanathan *et al.* 2003a). The recent studies of the author clearly established critical role of the mid rib isolates in causing severe epiphytotics in Uttar Pradesh and other subtropical states.

Pathogenic variation

During 1936-39, the historic red rot epiphytotic on the popular cv Co 213 in subtropical India led to emergence of light-coloured isolates with highly sporulating *C. falcatum* phenotype, which caused severe devastation on the variety (Chona and Padwick, 1942). For the first time in the country, the researchers studied in detail on the pathogen side and till that time the research was focussed only on deploying high quality and high yielding sugarcane varieties. Many researchers attributed to the highly virulent strains of the pathogen for successive failures of the varieties such as Co 301, Co 312, Co 313, Co 385, Co 421, Co 453, Co 513, BO 11, CoS 443 etc (Rafay, 1950, Rafay and Singh, 1957). Hence, after 1940, the researchers have initiated screening for red rot resistance in the varieties and germplasm (Chona, 1980).

The pathogenic isolates of *C. falcatum* were maintained in different names in different centres. Historic failures of the popular variety Co 1148 in the 1970s and other varieties later prompted the scientists to characterize the new virulent pathogenic strains on a set of differentials. In 1980s, a clear report on occurrence of three distinct pathotypes of *C. falcatum* from the subtropical region was reported based on the pathogenicity on a set of host varieties (Beniwal et al. 1989). Later, to characterize the pathogenic variation in *C. falcatum,* detailed studies were conducted in tropical and subtropical locations to identify suitable host differentials from 48 *Saccharum* spp. and hybrid cultivars (Padmanaban *et al*. 1996). A set of 13 host differentials were identified and established occurrence six pathotypes CF01, CF02, CF03, C04, CF05 and CF06 originated from the popular varieties that succumbed to *C. falcatum* viz. Co 1148, Co 7717, CoJ 64, Co 419, Co 997 and CoC 671, respectively (Viswanathan *et al*. 2003b). Later another three pathotypes CF07 (CoJ 64), CF08 (CoJ 64/ CoJ 85) and CF09 (CoS 767) were designated from the subtropical region. Subsequently, prevalence of a new pathotype (CF10) from the cv 85A261 along with three identified pathotypes was reported in coastal Andhra Pradesh and Odisha (Rao and Patro, 2005). Characterization of the new isolates by phenotyping on the host differentials continued and about 11 were as designated from four agro-climatic zones in the country till 2010 and used for screening of sugarcane progenies in the respective zones (Viswanathan, 2010). Another pathotype CF12 was designated from the tropical regions (Viswanathan, 2017). Recently a highly virulent pathotype CF13 responsible for breakdown of resistance in the popular cv Co 0238 was characterized from the subtropical India (Viswanathan *et al*. 2022).

Molecular variation

Earlier RAPD was used to group *C. falcatum* isolates and it was reported that the isolates had a general agreement with their pathogenicity on different sugarcane varieties (Mohanraj et al. 2002, Suman *et al*. 2006). Work of ITS nucleotide sequence analysis of 15 *C. falcatum* isolates of Sangdit *et al.* (2014) in Thailand showed 95.32–100% identity among themselves and 96.30–97.74% related to other *C. falcatum* isolates. Nine isolates from red rot affected locations possessed a high degree of pathogenicity on the susceptible cvs E-Heaw and K93-236, whereas those from non-epidemic were nonpathogenic. Also, in the same study the isolates from endemic areas had a light type of colony character and it was

concluded that in Thailand *C. falcatum* is differentiated into two distinct races (pathogenic and nonpathogenic) based on their pathogenicity on sugarcane stalks.

Malathi *et al*. (2010) characterized nine major *C. falcatum* pathotypes used for disease screening based on sequencing of 5.8 - internal spacer region of rDNA into two phylogenetic groups. Further, by using nitrate non-utilizing (nit) mutants heterokaryon formation was demonstrated and vegetative compatibility grouping (VCG) in *C. falcatum*. The VCG grouped the mutants into five categories and through this different isolates of the same pathotype were recognized. Grouping of the pathotypes based on VCG, pathogenicity and ITS was similar with certain deviation, however, these tools distinguished the two contrasting pathotypes Cf1148 and Cf7717 as reported earlier by RAPD (Malathi *et al*. 2010; Viswanathan *et al*. 2003b). Additionally, serological reactions between antisera of the pathotypes and the antigen in diffusion agar plates gave a clear relationship between the pathotypes (Alexander and Jothi, 1995; Viswanathan *et al.* 2003b). Subsequent molecular grouping of the *C. falcatum* isolates revealed that the Indian isolates fell into three separate assemblages as Group I, II, and III. Among them the latter had a distinct phenotype of dark coloured, least virulent and non-sporulating had a similarity to other country isolates whereas, the first two subgroups had overlapping phenotypic and pathogenic features (Malathi *et al*. 2011). Apart from ITS spacer region, sequencing other conserved genes calmodulin, actin and GPDH were used to further refine molecular grouping which revealed presence of one major group of virulent isolates and a minor group of least virulent isolates, which was established by definite nucleotide variation and further confirmed by RAPD and ISSR. Some of the primers were able to differentiate the virulent isolates with specific markers with the least virulent isolates. In addition, *C. falcatum* proteomes were established between virulent and least virulent isolates and identified distinctive and differentially expressed proteins related to virulence. Pathogenicity related genes identified from *Colletotrichum* spp and other fungi like glutathione S-transferase, DJ-1/PfpI family protein and serine protease were identified from *C. falcatum*. Utilizing the NGS data, Prasanth *et al.* (2021) identified virulent strain specific simple sequence repeat (SSR) marker from the genome of *C. falcatum*.

Diversity in pathogenic virulence

C. falcatum occurs in nature with enormous diversity for pathogenicity. Either the virulence is influenced by the varietal diversity or spread of a

Fig 3: Comparative virulence of *C. falcatum* isolates from the red rot affected cv CoS 8436 on the susceptible test variety CoC 671. The isolates originated from different places of Uttar Pradesh and Haryana

ruling variety over large areas. The new variants may be evolving from the already prevailing pathotype(s) in the field with a significant deviation in their pathogenicity and virulence due to the adaptation to the new host varieties or to a new environment for the pathogen carried through seed canes.

Diversity in the new *C. falcatum* isolates was carried out in the respective locations in India and no study was made to assess pathogenic diversity in huge collection of the isolates simultaneously in the country. The author has made an elaborate study to document pathogenic behaviour for nearly 117 isolates originated from tropical and subtropical locations in two locations, Coimbatore (tropical) and Karnal (subtropical) on a highly susceptible cv CoC 671 for five seasons by following plug method of evaluation under field conditions (Viswanathan *et al.* 2017). The isolates exhibited a huge variation in their behaviour from

season to season for red rot reactions at both the locations (Fig. 3). At subtropical condition, more number of less and least virulence reactions were recorded whereas, more highly virulent categories also recorded there and it may probably be due to weather conditions prevalent after pathogen inoculation. Though both the locations showed similar behaviour for moderate virulence, at the subtropical location it ranged from 5.22 to 21.15% during the five seasons with a mean of 11.94% whereas in the tropical location it ranged from 10.08% to 16.36% with a mean of 12.73%, indicating a stable behaviour in the latter than the former. Some of the isolates from the tropical region maintained a higher virulence in both the locations, whereas, many of the subtropical isolates and pathotypes exhibited less virulence in both the locations. These findings also revealed probable influence of weather factors in the expression of the pathogenic virulence. Further, over the years, the virulence is not static in the locations for many of the isolates due to their stable and unstable behaviour and place of phenotyping, source of host variety and interaction with weather conditions. Assessing more than 100 *C. falcatum* isolates simultaneously brought out existence of a vast diversity in *C. falcatum* isolates for their pathogenicity in India. Such a high pathogenic variation is probably attributed for frequent varietal breakdowns in sugarcane in the country for more than 100 years.

Factors governing pathogenicity in C. falcatum

It is well established that *C. falcatum* pathotypes vary in their virulence and this specific trait decides the pathogenicity of a particular pathotype. The pathogen produces secondary metabolites and hydrolytic enzymes, considered as bioweapons that probably contribute aggressiveness/virulence during pathogenesis and spread in the host tissue. Several workers studied production of hydrolytic enzymes viz. pectolytic and cellulolytic by *C. falcatum.* It was reported that virulent isolates of the pathogen produce more hydrolytic enzymes than weakly pathogenic ones (Manocha and Vasudeva, 1964; Singh and Husain, 1963, 1964). *C. falcatum* produces a toxic metabolite initially reported as anthroquinone compound, which may facilitate pathogen infection and spread in the stalk. The phytotoxin is soluble in water and in many organic solvents, capable of producing symptoms of red rot in the stalks except white spot (Olufolaji and Bamgboye, 1986; Mohanraj *et al*. 1995). Malathi and Viswanathan (2004) further studied in detail *C. falcatum* pathogenesis and established a correlation between disease expression and production of phytotoxins and hydrolytic enzymes by the pathogen. *C. falcatum* is a typical hemibiotroph, initiate infections as a biotroph and turn to necrotrophic phase for colonization and ramification inside the host tissue to complete life cycle. The phytotoxins and hydrolytic enzymes produced during necrotrophic phase of the pathogen determine the pathogen colonization and tissue damage. When pathogenicity is interfered by co-inoculating *Trichoderma harzianum,* production of these metabolites were reduced with no symptoms production on the host. Further, less virulent pathotype produces low levels of phytotoxins and hydrolytic enzymes compared to a virulent pathotype CF06 (Malathi and Viswanathan, 2007).When major *C. falcatum* pathotypes were studied for the relation between toxin production and symptom production on leaves, it was found that the virulent pathotypes caused more severe symptoms along with more loss of electrolyte leakage. Further, the virulent isolates produced higher levels of pectinolytic and cellulolytic enzymes especially exo-polygalacturanase and melanin production (Malathi and Viswanathan, 2012a).

The biochemical studies clearly established a positive association on the production of secondary metabolites like toxins and melanin and hydrolytic enzymes in *C. falcatum* virulence and disease expression. Later, molecular basis of the pathogenicity mechanism was studied using both genomic and proteomic tools. A study of 28 pathogenicity gene homologues in two distinct pathotypes vary in their virulence revealed a specific role of some of these pathogenicity genes in *C. falcatum* pathogenesis with a clear differential expression (Scindiya *et al*. 2017). Tricyclazole interferes in germination of the conidia and appressoria production and its melanisation in many fungal pathogens. Using tricyclazole, the melanin inhibitor, the role of

melanin in *C. falcatum* pathogenesis has been proved. Role of melanin was further established by studying the expression of melanin biosynthesis genes such as PKS1, SCD1 and THR1 (Kaverinathan *et al*. 2017). Subsequently knockdown mutants of *C. falcatum* were developed through RNA silencing strategy and Agrobacterium mediated transformation to functionally analyse polyketide synthase1 (PKS1), the major gene which regulates dihydroxy naphthalene melanin production. The loss of function mutants for PKS1 showed reduced pathogenicity in leaf- and stalk-tissues and established a clear role for melanin in pathogenic virulence in *C. falcatum* (Scindiya *et al*. 2019). Further studies of knocking down glucose transporter and sucrose non-fermenting genes affected pathogenicity in *C. falcatum* and revealed the role of these genes in pathogenicity (Scindiya *et al*. 2021).

Proteomics-based investigations lead to characterization of cellular and extracellular virulence and pathogenicity factors produced by pathogens as well as to identify changes in protein levels in host plant upon infection by pathogenic organisms and symbiotic counterparts (Ashwin *et al.* 2017a). *In vitro* secretome of *C. falcatum* cultured under light and dark conditions using 2DE coupled with MALDI TOF/TOF MS was analysed. The study has identified nine differentially expressed proteins and revealed a major portion of alterations occurred in low molecular weight (LMW) proteins of <30kDa. In dark cultures, the LMW proteins were either less abundant or absent, except a ceratoplatanin protein called eliciting plant response like protein 1 (CfEPL1), very high abundant LMW protein. While, in light cultures, a novel protein named as 'plant defense inducing protein 1' (CfPDIP1) was highly abundant (Ashwin et al. 2017b**).** Further studies functionally characterized distinct domains of CfEPL1 and CfPDIP1 by *in vitro* expression and purification; which indicated that CfEPL1ŠN1-92 and CfPDIP1ŠN1-21 induce hypersensitive reaction in tobacco and systemic resistance in sugarcane against *C. falcatum*. These studies have identified proteins that putatively contribute to *C. falcatum* virulence and demonstrated the potential role of PAMPs/effectors of *C. falcatum* inducing PAMP-triggered immunity (PTI)/effector-triggered immunity (ETI) in sugarcane (Ashwin *et al*. 2018).

Genomic and transcriptomic studies inC. falcatum

Viswanathan *et al*. (2016) reported complete genome of *C. falcatum* for the first time. The genome was 48.16 MB in size with 12,270 genes. Subsequent transcriptomic studies (*in vitro*) revealed its 31 MB size with 23,136 predicted CDS (Prasanth et al 2017). Mining of *C. falcatum* genome and transcriptome data yielded putative 768 and 884 small secreted proteins (SSPs), respectively. The predicted secretory proteins were further divided into classical and non-classical proteins and discovered that signal peptides have an apparent role during pathogenesis by stabilizing fungal secretory proteins in the host environment. The SSPs contained a large number of esterase, proteinase, CAZy families, cytochrome P450, peptidases, secondary metabolites, transporters and transcription factors. In *in planta* transcriptomic studies, these SSPs were recognised as major pathogenic determinants (Prasanth *et al.* 2019).

Recently, whole genome and consecutive transcriptome sequencing along with phenotypic and genotypic studies involving three pathotypes varying in virulence viz *C. falcatum* pathotype *Cf*671 MTCC accession number-12142), ROC (isolated from the sugarcane variety ROC) and R-1 (isolate recovered from CoC 671) were conducted. The studies identified the infection process, mating type and population structure using HMG (High Mobility group) proteins. This finding showed that *C. falcatum* is a definite stalk intriguing pathogen which establishes itself as precursor in attributing gene families for its virulence. During interaction with sugarcane, *C. falcatum* was found to express 2/3 of CAZy genes during biotrophic and necrotrophic phases (Prasanth et al 2022).

Infection process and life cycle

Recently, modern tools were used to reveal the infection process of the fungus with more clarity. On contact, the conidia germinate with germ tubes, produce appressoria, attach firmly to the sugarcane tissue, and endure stably for varying periods. This may support the fungus to

perpetuate when the infective mycelium is unable to proliferate further. The main portals of the entry for the pathogen are leaf scar, root primordia and buds in the nodal region (Fig. 4). After penetration, the fungus makes inter- and intracellular colonization. Later acervuli produced profusely around the infected tissues. Using *C. falcatum* isolate expressing green fluorescent protein (GFP) markers and other sensitive histological assays infection, colonization and fructification of the pathogen on the leaf and stalk tissues were clearly established.

Fig 4: *C. falcatum* causes infection in sugarcane stalks through portals of entry like bud, root eyes and leaf scar in the nodes

On sugarcane leaf tissues, by 12 h post inoculation (hpi) *C. falcatum* conidia germinate and form appressorial structures; by 24 hpi, after formation of primary hyphae the fungus enter into the host cell; by 48 hpi spread to nearby cells through secondary hyphae and this phase mark the end of biotrophic phase, in which the fungus does not kill the cells. Later, necrotrophic phase of pathogenesis begins, in which the secondary hyphae continue to damage the cell structure with widespread colonization during kill the colonized the cells to make rapid proliferation. The pathogen makes both intra and intercellular colonization and emerge outside through the stomatal pores with sporulating acervuli structures with setae by 72 hpi. On stalk tissue also, the pathogen shows similar infection cycle, unlike in the leaf tissue, in stalks the pathogen continue to make colonization due to availability of host tissue and make both upward and downward progress. In due course, it occupies entire core tissues in the internodes and nodes. By 30-45 days, the pathogen macerates entire tissues and causes complete destruction of the stalk tissues. The pathogen also grows inside and fills the pith cavities with greyish black mycelia. Presence of aervuli throughout rind tissues and typical sporulation can also be seen on the root eyes, leaf scar and rind (Mohanraj *et al*. 1997; Viswanathan, 2010; Ashwin *et al*. 2020; Nandakumar *et al.* 2020).The pathogen infection progresses considerably in the susceptible varieties in the same season or it may remain as dormant incipient infections. In highly susceptible varieties, the conidia landed in the whorl directly infect and penetrate the growing point to reach the stalk. Although *C. falcatum* may infect almost any part of the sugarcane plant its importance is limited largely to its occurrence on the leaf midribs, the internal stalk tissues, and the stubbles of the ratoons (Fig. 4). Infection of the roots may occur, however red rot is not important as a root disease.

Fig 5: New shoots in a ratoon crop show drying after *C. falcatum* infection (Affected shoots are indicated with arrows)

Origin of new variants of C. falcatum and their adaptation to sugarcane varieties

C. falcatum variants continuously emerge in the field and the new variants pose challenge to host resistance in the new varieties. However, limited studieswere conducted on the adaptation of *C. falcatum* to the newly released varieties under field conditions. Srinivasan (1962) studied in detail on the role of host varieties on emergence of new isolates. He has shown that some sugarcane varieties induce rapid development and

dominance in infected tissues of the dark, avirulent type of variant, while others appear to favour the dominance of the virulent parental clone. Sometimes a more virulent isolate than its parental clone has also been appeared. Later, Srinivasan (1965) also opined adaptive changes in cultivated *C. falcatum* in relation to the host varieties, with subsequent alterations in the virulence patterns of the fungus. He also observed that the pathogenic isolates are often unstable in their pathogenicity and have a tendency to pass irrevocably into an avirulent phase. Earlier studies at ICAR-SBI explained how a less virulent isolate gain virulence after several rounds of repeated inoculation and isolation on an incompatible host variety (Malathi *et al*. 2006). After repeated inoculations, the dark isolates at initial phases become light with increased sporulation on their adapted hosts. Development of light isolates and reduced latent period for symptom expression by repeated inoculations on incompatible host varieties indicated gain of virulence or pathogenicity of that pathotype for adaptation on a particular cultivar. The adapted cultures were able to tolerate to the new cytoplasm as suggested by Srinivasan (1962).

The author has conducted a detailed study with 12 *C. falcatum* pathotypes and 20 varieties varying in disease resistance for 10 years to identify how the pathotypes change their behaviour on the host varieties under tropical conditions (Viswanathan et al. 2020a). The pathotypes always exhibited their virulence on the highly susceptible varieties but not on the varieties with susceptible or intermediate reactions when they were inoculated by plug method under field conditions. Also the isolates originated from susceptible or moderately susceptible (MS) varieties had expressed their virulence similarly, indicating that all the new pathotypes completely evolved with potential virulence to cause knockdown effect on the host. The study revealed that although the resistant varieties remained free from infections from the pathotypes, on few occasions, the isolates have broken barriers of incompatibility and caused disease. On the susceptible host varieties, the expression of the virulence was in the range of 62.9-97.9% whereas on MS hosts it was 21.3-40%, clearly revealing that the pathogen has to evolve further to completely adapt on the latter group of varieties. Such an evolution happens under field conditions after varietal introduction and the existing pathotype(s) adapt to the host variety and slowly gain its virulence to cause knockdown effect. This study also demonstrated on the gain of virulence by the new pathotypes over the old pathotypes that were isolated 30 years ago, CF04 (*Cf*419), CF05 (*Cf*997) and CF06 (*Cf*671) and used for the resistance screening in the tropical states. Meanwhile, in the last two to three decades, new varieties were deployed and correspondingly new pathotypes have evolved from the new varieties suggesting the gain of virulence from the new hosts.

In another interesting study, the author has again demonstrated on the continuous evolution of *C. falcatum* in tune with the new varieties deployed in the field. He used the case study with *C. falcatum* pathotype CF06, the predominant pathotype of the tropical region in India used for varietal screening and assessed how it has given way for new pathotype CF12 with 32 host varieties under field conditions for seven years (Viswanathan, 2017). The then popular cv CoC 671 which was cultivated extensively during 1980s and 1990s in the tropical region had a devastating red rot epiphytotics and that led to the emergence of highly virulent pathotype CF06 that time (Viswanathan et al., 1997, 2003b, Viswanathan, 2010, 2021b). However, many varieties like Co 86002, Co 87012, Co 92012, Co 92020, Co 94003, Co 99006, Co 2001-13, Co 06022, Co 06027, CoC 24, CoSi 6, 81V93, 89V44, CoV 92102, CoV 09356, PI 96-843, PI 1110, Si 7, Si 8 etc deployed for cultivation from 1990s onwards succumbed to the pathogen though they were rated as resistant at the time of their introduction to the field. It was suspected that the pathotype CF06 either lost its virulence or new pathotype(s) with matching virulence have emerged in the field. Hence, the pathotypes CF06 and a highly virulent pathotype Cf94012 were compared for their comparative behaviour on a set of 32 varieties varying in red rot resistance. On three R varieties, both the pathotypes behaved similarly, whereas on another six R varieties the latter caused MS or S reactions. Further, on the S and MS varieties the pathotype Cf94012 (CF12) exhibited a very high virulence during the seven seasons

indicating acquired virulence of the new pathotype, reflecting on the virulent isolates arose in the field on the above varieties. The pathotype CF12 is regularly used for varietal screening in the tropical region. Even though the study has clearly demonstrated higher virulence of the pathotype CF06, it could not exhibit virulence as like the new pathotype CF12 on the new varieties developed in the recent years. Reason could be that the pathotype CF06 no longer has contact with the host in the field to gain virulence and the new pathotype CF12 emerged after the year 2000, an adapted one from CF06 to attack the new varieties by gaining virulence. This specific adaptation in the new pathotype matched to the new varieties hence they failed in the field. This is how new pathotypes adopt to the new varieties in the field. A role of high sugar in sugarcane varieties in acquisition of high virulence by *C. falcatum* was demonstrated with a set of species clones from *Saccharum* and *Erianthus* (Malathi and Viswanathan, 2012b). The pathogen inoculum present in the soil plays major role in origin of new variants by adapting to the new varieties introduced for cultivation (Viswanathan et al. 2020b). Further, the resistant varieties succumb to the new variants and gradually the pathogen is able to cause 'varietal breakdown' in sugarcane. This was demonstrated by comparing disease development in the plug method of inoculation and soil inoculum applied at the time of planting with a set of sugarcane varieties varying in disease resistance and *C. falcatum* isolates vary in their virulence (Viswanathan and Selvakumar, 2020).

Gain of virulence by the red rot pathogen

Over the decades, it was found that *C. falcatum* gained virulence and the gain of virulence is directed by the host varieties deployed for cultivation in the field. Report of light coloured *C. falcatum* cultures with highly sporulating phenotype after severe epiphytotics in the cv Co 213 during 1936-39 was first report on virulence in *C. falcatum* (Chona and Padwick 1942). Later, another disease outbreak in the cv Co 313 led to emergence of 'D' strain (isolate No 244) of the fungal pathogen (Rafay, 1953). Although *C. falcatum* caused destruction in the field in the tropical region was known, major gain of virulence in the pathogen was found after failure of the popular high sugar variety CoC 671 in the states of Tamil Nadu, Kerala, Andhra Pradesh, Puducherry and Gujarat. For the first time, origin of highly virulent pathotype Cf671, later designated as CF06 was recorded in the tropical region. It was also found to be the highly virulent pathotype of all the major isolates available in the country that time. It had surpassed other isolates and pathotypes like CF04, CF05, Cf658, Cf6304, Cf8001 etc in virulence (Viswanathan et al. 1997). It has maintained the virulence for more than 20 years in the tropical states. Another pathotype CF12 was recently designated from the tropical region with high virulence than CF06 (Viswanathan, 2017).

It is reported earlier that in Indian scenario especially in the Indo-gangetic plains of UP and Bihar, red rot epiphytotics followed a 'boom' and 'bust' cycle in the last 100 years (Viswanathan, 2021b). Every time when the popular varieties were grown over large areas, the pathogen gained virulence substantially with something like super-virulent strains after failures of the varieties like Co 213, Co 312, Co 453, Co 1148, CoJ 64 in the previous century. Recently the popular variety Co 0238 was spread unscrupulously to entire command area that resulted in evolution of another super-virulent strain in the region. In the state of UP, the variety was grown in 2.2 M ha (82.21% of total cane area) and in Bihar 0.16 M ha (64.12% of total cane area) in 2019-20 cropping season (Ram, 2020). This monoculture of single variety over a large areas has favoured evolution of highly aggressive strain of the pathogen (Vertifolia effect) within three seasons. In this situation, the pathogen has evolved very quickly and caused varietal breakdown within a few years. First incidence of the disease was recorded during 2016-17 season in few districts and this historical epiphytotic engulfed nearly 0.3 to 0.4 M ha area in the subtropical region in the last five seasons. The new pathotype CF13 has evolved on Co 0238 and gained virulence to knock down host resistance. Although the variety still exhibits resistance to all the pathotypes in the subtropical region viz. CF01, CF02, CF03, CF07, CF08, CF09 and CF11, it is susceptible to CF13 selectively evolved by adapting to the variety with rapid changes in the pathogen virulence. Here

monoculture of the variety triggered gain of higher virulence (Viswanathan *et al*. 2022). In the past, such gain of virulence was witnessed after severe epiphytotics in the cv CoC 671 in the tropical region with the evolution of CF06 pathotype (Viswanathan *et al*. 1997). Although emergence of highly aggressive strains of the pathogen has caused havoc in the country, the breeders have always undermined the pathogen attack by promoting single variety over several thousands of hectares. Greedy sugar millers also spread the variety in an unscientific manner for a short term gain and finally lose the battle with the pathogen. The loss is not ending here, due to the high virulence and aggression, the new pathotype causes varietal breakdown quickly and this poses a difficulty in finding alternate varieties. Hence origin of new aggressive strains of the pathogens solely dictated by the host side as suggested by Srinivasan (1962).

CONCLUSION

Red rot of sugarcane has been a major constraint for sugarcane production for more than 100 years. The disease is being managed time to time by releasing disease resistant varieties and the susceptible varieties are replaced with resistant varieties. The susceptible varieties were once resistant ones, became susceptible due to varietal breakdown caused by the new variants of *C. falcatum*. The new variants of *C. falcatum* represents the current pathogenic flora spread in the command areas. Hence the new variants of the fungal pathogen are being characterized on a set of differentials and the widely occurring isolate with a higher virulence and stability is designated as a new pathotype and used to screen sugarcane progenies/ varieties to manage the disease in the field. The challenges caused by the pathogen were overcome by the efforts of dedicated sugarcane breeding in India by developing disease resistant varieties time to time. However, in the varietal development, combining high yield, high sugar and red rot resistance in sugarcane is a huge challenge and rarely we get wonder varieties like Co 213, Co 1148, CoC 671, CoJ 64, Co 86032, Co 0238 etc with such a desirable combination. However, poor varietal management, monoculture and lack of proper disease monitoring shortened the field life of many elite varieties and we could not effectively harness the genetic gain. Also, each and every red rot epidemic comes with huge economic losses to the farmers and industry. In the long run, red rot of sugarcane can be effectively managed by addressing the pathogenic variation and proper varietal management.

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DECLARATION

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