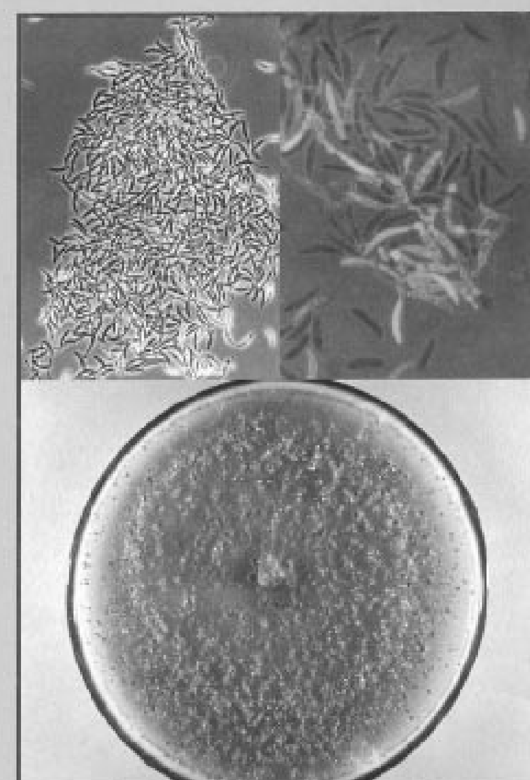
Red rot of Sugarcane



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Dedicated to
all
who bravely faced
the challenges of
Red rot

PREFACE

In the history of Plant Pathology, only a few diseases like rust of wheat, late blight of potato, ergot of cereals, rust of coffee, brown spot of rice and red rot of sugarcane have left longstanding impact on food availability which changed the face of human society. The potato murrain of 1842-45 in Ireland not only decimated over a million Irish people but also forced a mass exodus of another million or so to America.

In the Indian context, apart from the rusts of wheat and brown spot of rice, red rot of sugarcane is the most important disease that affected Indian agriculture badly with far reaching consequences. It is needless to say that red rot epidemic of 1938-39, along with brown spot epidemic of rice in 1942-43 augured the development of Plant Pathology as a subject in the curriculum of Indian Universities.

Red rot is the key menace of sugarcane in India, and the onus of its containment also squarely rests on the Indians. In spite of the best efforts, red rot is still posing challenges in stabilising sugarcane and sugar production. Through the concerted efforts of breeders and pathologists, the disease has been contained to a manageable level and thus, the frequency and magnitude of red rot epidemic has been reduced to a great extent in recent years. In fact, entire sugarcane breeding in India is now geared around red rot and no sugarcane variety is released for general cultivation without resistance to the prevalent pathotypes of red rot.

In spite of its immense importance in the sugarcane agriculture, hardly any compilation has been produced so far on red rot except some odd reviews. This book is a humble effort in this regard to bridge the longstanding gap. Moreover, this book has been developed as a photo essay on red rot covering many facets of the host, disease and the pathogen. Errors and failings doubtless remain, and for these I owe the sole responsibility.

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Lucknow

July 2008

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I wish to acknowledge my gratefulness to Dr. Kishan Singh, the then Director of IISR, Lucknow, who initiated me in the realm of red rot in 1986 and reposed full faith to carry forward researches on this disease, in spite of my postgraduate specialization in bacterial plant pathology. My grateful thanks are also due to my teachers, who inspired and instilled in me the confidence to become a 'Plant pathologist' and not to get restricted in the boundaries of pathogens.

I am grateful to Dr. R. L. Yadav, the present Director of IISR, Lucknow for kindly agreeing to publish this effort in the form of a book. His inspiration and constant encouragement gave me the desired impetus to complete this book in time.

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It is my pleasant duty to acknowledge the support provided by my wife in writing the book. She not only read the book from a layman's perspective but also gave critical suggestions in improving the readability and clarity of the presentation.

Finally, I wish to acknowledge my gratitude to my parents who inculcated in me the confidence to follow the arduous path of truth '*it is truth and only the truth that prevails; one has to have patience and courage to attain the goal*'.

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INTRODUCTION

The sugarcane crop in India

The importance of sugar in human diet needs no introduction; it has become a part and parcel of daily life. Sugar is produced mainly from sugarcane and sugarbeet and more than 75 per cent of the world sugar comes from sugarcane. Throughout ages, sugarcane remained an important commercial crop of agriculture and trade in India contributing substantial revenue to the exchequer by way of tax and duties. Today, it is fast transforming into the most sought after renewable energy crop, as the demand for ethanol is increasing as an alternative green fuel for the automobile. This has become far more important in the backdrop of dwindling oil reserves. Currently Brazil is diverting 50 per cent of its sugarcane for the production of ethanol and blending ethanol with petrol to the tune of 25%. India is hoping to blend ethanol at least up to 10% in the near future. It has been estimated that India will need 495 million tonnes of sugarcane by 2025 AD to meet both sugar and energy demands (Yadav and Duttamajumder, 2007).

Sugarcane is a native of India and has been in use for '*gur*' making since prehistoric times. '*Gur*' was known as early as 3000 BC. The name of the raw sugar '*gur*' has originated from the word '*Gaura*' - a well-known dynasty that ruled Bengal. The raw sugar produced in Java (now Indonesia) is called '*Goela*' indicating that the process of *gur* making had travelled eastward from Bengal to Java. It is estimated that by 250 BC sugarcane had reached China from India and by 1 AD it reached the islands of Java (Indonesia). The westward journey of sugarcane started from India in 327-325 BC with the invasion of Alexander, the Great. The Greeks termed sugarcane as '*honey yielding reed*' as they were unaware of the existence of sugarcane and it was their prized catch. By the 8th century, sugarcane established itself in southern Europe (Italy, France and Spain).

The story of POJ 2878

Early sugarcane breeders realized that the resultant F_1 progeny of *S. officinarum* and *S. spontaneum* was distinctively different from either of the parents. When *S. officinarum* clones were used as the female parent, progeny tended to be taller stalked, higher in sucrose level, and generally more vigorous than when *S. spontaneum* clones were used as the female parent. Reciprocal difference in vigour was attributed to '2n+n' transmission in *S. officinarum* (female) x *S. spontaneum* (male) cross combination. This phenomenon of irregular transmission was termed as **'female restitution'** by Bremer. The present day successes of sugarcane dates back to the early part of the 20th century when active use of this species with noble cane (*S. officinarum*) yielded the much-desired tolerant hybrids at the two main centres of cane breeding i.e., Java (Proefstation Oost Java) and India (Coimbatore). In fact, these spectacular hybrids provided desired fillip to the sugarcane and sugar industry worldwide (POJ 2878 in Java, Co 205, Co 213 in India, POJ 213 in USA), which were plagued with '*Sereh*', red rot and mosaic. No doubt, in the history of agriculture these events remained path-breaking achievements (use of wild species in the improvement of crop plant, harnessing of hybrid vigour, a quantum jump in acreage of the crop and yield) and ushered the *'first green revolution'*, both in India and elsewhere in the early part of 20th century. Due to the development of POJ 2878 alone, Java became a sugar surplus country in no time. In the history of sugarcane breeding, the Javan variety POJ 2878 stands out as a major achievement. Both as a commercial variety in its own merit and as a parent of many commercial varieties, it has established a remarkable record (Mangelsdorf, 1959). The genotype POJ 2878 was first raised in 1921 and by 1929, it established itself as the ruling variety of Java, surpassing all previous records. The breeding history of POJ 2878 is highlighted here as an example of utilisation of wild species in the development of disease resistant cane genotypes. The '*Sereh*' which was plaguing sugar industry of Java was a boon in disguise; it forced a comprehensive study of sugarcane–its breeding, diseases and the various management options. To name a few, like rediscovery of fertile seed of sugarcane, heat treatment of cane, discovery of diseases like red rot and leaf scald, development of hybrids and use of other related species in breeding are the most significant. The development started with the collection of Kassoer (a natural hybrid of unknown noble cane and unknown Glagh, *S. spontaneum*) from near the base of Tjerimai volcano in Java in about 1890. Similarly, POJ 100 was developed from an open pollinated tassel by Wakker

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SYMPTOMS

The pathogen, *Colletotrichum falcatum* Went, can attack any part of the sugarcane plant; be it stalk, leaf, buds or roots. *C. falcatum* completes its life cycle on the sugarcane leaf and usually the damage to leaf does not pose a serious threat to cane or cause much harm to the plant. The most damaging phase of this disease occurs when the pathogen attacks the stalk. Depending on the age of the stalk, time of infection and susceptibility of the cane genotype, it produces different types of symptoms. The typical stalk symptoms i.e., presence of white spots in otherwise rotten (dull red) internodal tissues and nodal rotting appear when the crop is at the fag end of the grand growth phase during August-September in subtropical India. In the early stages of infection, it is difficult to recognise the presence of the disease in the field, as the plant does not display any external symptom or distress. At a later stage, some discolouration of rind often becomes apparent when internal tissues have been badly damaged and are fully rotten. This is more pronounced in the stalk of light coloured genotypes **(Plates 9a, 38 b, c)**. At the end, affected plant dies. At the field level, this may be observed as the death of a few plants or clumps to the failure of entire crop **(Plate 2)**.

The infected cane setts carry the primary infection to the field. Depending on the nature of infection and availability of favourable environment, pathogen starts taking toll by killing the bud. This affects the germination and initial establishment of the crop. Poor germination leads to a gappy crop stand and reduction in yield. If, at all, the buds of the infected setts are able to sprout and grow, then above ground symptoms appear. The type of symptoms varies depending on the prevailing weather conditions. At first, symptoms appear as the death of young and emerging shoots **(Plate 3a, b)** without any conspicuous identifiable symptom (in March-April-May in north Indian condition, spring

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HISTORICAL BACKGROUND

"A historical knowledge of one's own branch of science, however profound, is no guarantee against the making of new mistakes in a new situation. But history also shows that it is only too easy to make the old mistakes in a recurrent situation that a thorough acquaintance with past work could have taught us to recognize." - *Garrett, S.D., 1959.*

The prophetic remark of Garrett aptly outlines the chequered history of red rot. Over and over again, pathologists of different genre have made similar mistakes in similar situations due to a lack of proper understanding of the disease. This situation was partly due to the non-availability of seminal literature at one's disposal (at one place), and partly due to the haste in having a quick-fix solution. Red rot, though prevalent in India since time immemorial, drew the attention of the scientific community only when FAFC Went chance encountered this malady in Java. He was deputed to Java (now Indonesia), to investigate the notorious '*Sereh*' disease, which was threatening the sugar industry of Java in 1880's. In search of the cause and cure of '*Sereh'*, Went stumbled upon a situation of cane dying in 1892 at the Tjomal estate in Java, and in the following year (1893) he published an account of this problem of stalk rotting of sugarcane plants and thus, this sugarcane disease came to light in the scientific world.

He studied the malady, and described the causal fungus as *Colletotrichum falcatum* Went and named the disease as "*het rood snot*", meaning **red smut**. Obviously, the name '**red smut**' is not a happy one as 'smut' is caused by an entirely different group of fungi and produce remarkably different type of symptoms. The species is named *'falcatum'* due to its typical falcate/sickle shaped conidia **(Plates 19, 21)**. The accepted name, **'Red rot'** was given to this cane disease in 1906 by Sir E. J. Butler, the celebrated Imperial Mycologist of India, who was then working at Pusa, Bihar. He wrote the first major account of this disease in 1906. It was by sheer chance that just after three years of Went's

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THE FUNGUS

Description of *Colletotrichum falcatum* Went

(= *Glomerella tucumanensis* von Arx and Muller*)*

The fungus causing red rot of sugarcane is commonly known by its imperfect state, i.e., *Colletotrichum falcatum* Went. The disease was first described to the scientific world in 1893 by Went (Went, 1893) from Java (now Indonesia). Incidentally, the perfect state (*Physalospora tucumanensis* Speg.) was first recorded by Spegazzini from Argentina in 1896 (Spegazzini, 1896) but it took almost half a century to establish the association between *Colletotrichum falcatum* Went and *Physalospora tucumanensis* Speg. (Carvajal and Edgerton, 1944). Ten years later von Arx and Muller from Germany transferred this fungus *Physalospora tucumanensis* to the genus *Glomerella* and renamed it as *Glomerella tucumanensis* (Speg.) von Arx and Muller (von Arx and Muller, 1954). The identifying characteristics of the imperfect state, the disease-causing phase, as described by different authors (Abbott, 1938; Sivanesan and Waller, 1986; Abbott and Hughes, 1961; Sutton 1980) are given below :

Colony greyish white with sparse aerial mycelium, occasional small dense felty patches, reverse white to grey, conidial masses **(Plate 18a, b)** salmon pink (light race); some cultures have abundant greyish white aerial mycelium with poor sporulation and no distinct acervuli (dark race). Sclerotia not produced by both the races, setae sparse, conidia falcate (but not markedly so), fusoid, apices obtuse, 15.5 (25-26.5) 48 µm x 4 (5-6) 8 µm **(Plates 19, 20, 21, 22)** and contents are granular and sometime contain oil globules. The central white area, when stained with appropriate stains indicates the presence of nuclear material. A conidium usually contains single central nucleus **(Plate 20)**. The occurrence of more than one nucleus in the conidium is often observed **(Plate 21)**. With the

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VARIABILITY AND RACE IDENTIFICATION

Variability is the rule of nature

Variability is the rule of nature; it is the seminal mechanism available to an organism to maintain the diversity. Buxton (1959) wrote :

"For evidence of variability in plant pathogenic fungi there is no need to look further than the many examples of crop varieties that have been bred for resistance to a given disease only to succumb to that disease after they have been brought into general cultivation."

The red rot pathogen is no exception to this general situation; rather it is more potent in changing the virulence pattern depending on the particular need to match the introduced host resistance. Both sexual and asexual mechanisms are operative for this purpose. However, one moot point that needs to be pondered at is the generation of variability in *C. falcatum*. What is the basis of frequent appearance of new virulences (races) that are able to start new outbreaks of disease both in new localities and in previously resistant host varieties?

Mechanism of variation

The variations in the asexual state of the fungus (*Colletotrichum* state) may originate through (a) heterokaryosis, (b) by recombination through parasexual mechanism, and (c) by the universal mechanism of mutation, selection and adaptation in response to the changes in the host environment. Heterokaryosis is the mechanism through which the fungus collects and consolidates two or more genetically different nuclei in the hypha and derives the benefit of the introduced genetic material. These newly gathered nuclei also multiply in tandem with the native nuclei. Anastomosis of hyphal cell is common **(Plate 32)** in *Colletorichum falcatum* (Duttamajumder *et al.*, 1990; Singh and Payak, 1968; Carvalho, 1968). Anastomoses may take place between hypha, hypha and conidium, conidium and

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EPIDEMIOLOGY

Life cycle and disease cycle

Manifestation of red rot varies depending on the nature of infection, time of the season and the prevailing environment. If sufficient inoculum is present in/on the sett in active form and if adequate moisture is available, it causes both pre- and post-emergence mortality of the sprouts during April-May in the subtropical region. With the advent of pre-monsoon showers, symptoms of the disease start appearing, and with the onset of the monsoon when the weather is most suitable, full manifestation of the disease takes place. Later on, infected plants turn yellow and finally dry up. At the grand growth phase, when sufficient stalk and sugar have formed, typical stalk rot phase (red rot phase) appears. Red rot is not easily identified from the external appearance of the cane unless it has caused irreparable damage by rotting the internal tissues. In such cases, the rind loses its natural bright colour, turns violet or displays dark reddish tinge, and becomes dull in appearance. The affected plants appear sick; usually yellowing of the 3rd or 4th leaf, these being the prominent leaves of the crown, draw the attention of the observer from a distance (The yellowing may start from any leaf of the crown - there is no hard and fast rule). Yellowing of leaf starts from the tip and proceeds down along the leaf margin. *C. falcatum* proliferates within the cane stalk happily, and usually comes out through the root primordia for secondary spread when sufficient rotting of the internal tissues has taken place. The fungus totally transforms the root primordia into black acervuli bearing abundant conidia (the black colour is due to the dark setae). At this stage, if the cane is split open longitudinally, typical symptoms of red rot, viz., reddening of the internal tissues with interrupted red and white patches (white spots) in the affected internodes, rotten or damaged node, and the presence of typical sourly alcoholic smell may

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EPILOGUE

No doubt, impressive work has been done on many aspects of the red rot disease in India, yet there are many facets in the epidemiology and management of the disease that need critical studies.

In epidemiology, the role of perfect stage remains a pathologist's enigma. How is the sexual reproduction in *C. falcatum* contributing to the generation of new variability or development of new pathotypes and in the aerial spread of the pathogen covering a large area ? Where does the inoculum come from to cause the mid-rib infection in the pre-monsoon period? In addition, how does this mid-rib infection contribute in the generation of new virulence in the pathogen? No satisfactory answer is available even today. The exact role played by the environmental factors like temperature, humidity, rainfall, light intensity, wind, etc. need to be worked out in detail. The contribution of alternative hosts in perpetuating virulent races of red rot pathogen is yet to be determined. Dispersal mechanisms of infective propagules like conidia, ascospores, chlamydospores, acervuli, etc. formed on cane plant during and after the rainy season need critical evaluation. Available information is inadequate to explain the reason of large-scale flare up of red rot covering hundreds of acres. The exact role of perpetuating structures of the fungus like chlamydospores/ appressoria, thick walled hyphae and setae in the epidemiology of the red rot, and the part played by *Fusarium* and other associated microorganisms in accentuating the damage need immediate attention of researchers. The role of associated bacteria needs critical study, as *C. falcatum* when grown in sucrose medium or cane juice does not produce typical sourly alcoholic smell, which is quite characteristically observed in diseased canes of susceptible genotypes.

The nature of variability and existence of physiological races in *C. falcatum vis a vis* sources of resistance in host have to be catalogued using modern molecular