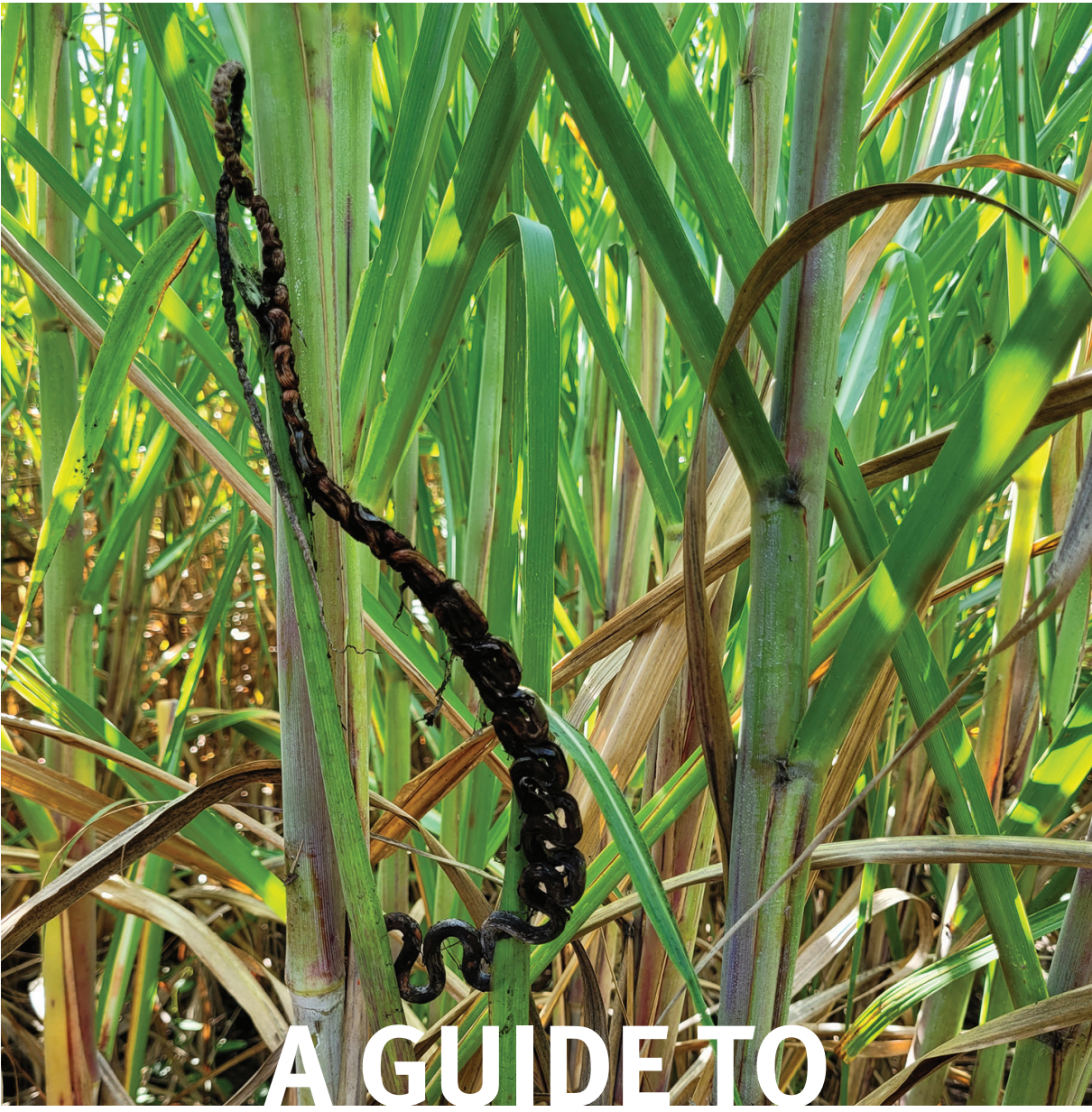

A GUIDE TO SUGARCANE DISEASES

P. Rott, J.-C. Girard, J.H. Daugrois, eds

2nd edition

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A guide to sugarcane diseases

Philippe Rott, Jean-Claude Girard,
Jean Heinrich Daugrois, eds

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Cover: Serpentine-shaped whip produced by *Sporisorium scitamineum*,
the causal agent of smut. Photo credit: Philippe Rott.

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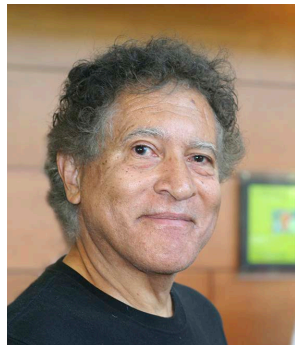

for their outstanding contributions to sugarcane pathology.

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Foreword

From its centre of origin in New Guinea some 10,000 years ago, sugarcane has spread across diverse geographical zones largely through manmade activities. Today, it is grown on approximately 26 million hectares in 120 countries. Among all the cultivated crops, sugarcane has the largest biomass and is regarded as one of the most efficient natural biofactories. It accounts for 80% of the world's annual sugar production of some 186 million tons and plays an increasing vital role in the production of bioelectricity, biofuel, bioplastics and a growing range of sucrochemicals. It has various attributes to address the societal issues such as food production, climate change, renewable energy, decarbonisation, etc.

If sugarcane has reached such an importance, it is mostly through the result of research and development, innovation and technological advancement. It is worth noting that sugarcane was among one of the first crops to have benefitted from organised research with the British and the Dutch in Barbados and Java in 1886, followed by the creation of research centres in Mauritius, in Hawaii and in Australia in the 1890s. The pioneering initiative stimulated similar activities in other sugarcane growing areas.

Early research efforts focused primarily on the creation of new high yielding varieties, resistant to diseases and pests with improved sucrose content. Disease identification, and disease management strategies ranked high as priorities on the agenda of the research centres. The creation of the International Society of Sugar Cane Technologists (ISSCT) in Hawaii in 1924 marked a turning point, federating global research impetus on sugarcane diseases. The ISSCT created various standing committees, including one devoted to sugarcane diseases. This committee contributed significantly to advancing knowledge on sugarcane pathology and quarantine (referred as biosecurity in the modern context). At each of the 32 ISSCT congresses held to date, sugarcane diseases have been an important component on the agenda.

It is to be recalled that sugarcane diseases have been a serious yield-reducing factor with ratoon stunt as an insidious disease, epidemics of mosaic in the 1920s in the USA, Fiji leaf gall in Australia and gumming in the Mascarenes in the 1960s, smut and brown rust in the Caribbean in the 1970s, yellow leaf in the 1990s, orange rust in Australia in year 2000, to name some occurrences. In addition, new pathogens such as sugarcane bacilliform virus and sugarcane yellow leaf virus have been identified, while several known ones including *Puccinia kuehnii* and sugarcane streak mosaic virus have spread to previously unaffected regions.

Over the years, the Proceedings of ISSCT congresses have served as a rich source of information on sugarcane diseases. To further disseminate knowledge, the ISSCT supported the publications of three books by Elsevier Publishing Co., Amsterdam, The Netherlands, written by sugarcane pathologists. They are *Sugar-Cane Diseases of the World, Volume I* (J.P. Martin, E.V. Abbott and C.G. Hughes, Eds., 1961), *Sugar-Cane Diseases of the World, Volume II* (C.G. Hughes, E.V. Abbott and C.A. Wismer, Eds., 1964) and *Diseases of Sugarcane – Major Diseases* (C. Ricaud, B.T. Egan, A.G. Gillespie Jr and C.G. Hughes, Eds., 1989).

In 1991, ISSCT in collaboration with the FAO and the International Board for Plant Genetic Resources (IBPGR) published *Technical Guidelines for the Safe Movement of Sugarcane Germplasm*. Additionally, from September 1968 to November 1982, the ISSCT published 29 issues of the Sugarcane Pathologists' Newsletter.

In 2000, a practical guide entitled *A Guide to Sugarcane Diseases*, under the helm of P. Rott, Chairman of the Editorial Committee, was published by CIRAD within its series REPERES. This guide provided a very useful resource for sugarcane pathologists in the identification, understanding and practical management of diseases.

Since then, new diseases have emerged in several sugarcane producing areas such as tawny rust caused by the fungus *Macruropyxis fulva* in Africa. In addition, the agents of long known diseases like chlorotic streak described in the 1920s and Ramu stunt in the 1980s have seen major advancement. The causal agent of chlorotic streak was identified as *Phytocercomonas venanatans*, a cercozoan, while that of Ramu stunt is a virus, specifically a *Mechlorovirus*. In addition, novel viruses belonging to various genera such as *Mastrevirus*, *Polerovirus*, *Mechlorovirus*, and *Umbravirus* were identified. In parallel, the application of molecular biology has revolutionized disease diagnostics, with tools such as polymerase chain reaction (PCR), reverse transcriptase (RT)-PCR, nested-PCR, multiplex PCR, multiplex RT-PCR, and loop-mediated isothermal amplification (LAMP) now commonly used.

These new developments have prompted a new edition of the *Guide*, led by Philippe Rott, Jean-Claude Girard, and Jean Heinrich Daugrois of CIRAD together with a team of dedicated collaborators. It offers a comprehensive and updated description of some 80 diseases, their cause, geographical distribution, symptoms, diagnosis, strain variation, transmission, host range, epidemiology, economic importance, control, and essential references. The inclusion of photographs of symptoms, pathogens, and vectors enhances its practical value.

I commend the Editors and authors of the chapters for producing this new Edition which will undoubtedly become a vital resource for sugarcane pathologists, researchers and industry professionals worldwide.

Jean Claude Autrey
Sugarcane pathologist and former Director
of the Mauritius Sugar Industry Research Institute (MSIRI)
General Secretary of the International Society
of Sugar Cane Technologists (ISSCT)

Introduction

Philippe Rott, Jean-Claude Girard, Jean Heinrich Daugrois

Since the release of the first edition of the Guide to sugarcane diseases in 2000, new diseases developed in several sugarcane producing countries. For example, tawny rust caused by the fungus *Macruropyxis fulva* was reported for the first time in South Africa. Additionally, the cause of diseases such as chlorotic streak and Ramu stunt was discovered. New viruses infecting sugarcane were uncovered thanks to the progress of molecular methods such as high-throughput sequencing. Several diseases that were limited to a given location increased their geographical distribution. For example, orange rust that was limited to the Asian-Oceania region was reported for the first time in Florida in 2007 and, in subsequent years, the disease spread through most of the American continent. Similarly, sugarcane streak mosaic virus, that was limited to Asia for decades, was found for the first time in the late 2010s in Africa. These examples illustrate the ever-changing nature of sugarcane diseases and consequently the information available regarding the pathogens affecting the sugarcane crop and disease management needs to be updated. A lot of progress has also been made in the areas of disease diagnosis and detection, resulting in the development of various highly performant molecular assays that are being continuously improved. Nevertheless, although diseases can be better diagnosed than ever, their control remains challenging. Thus, the control of sugarcane diseases involving the production and use of resistant varieties, the use of healthy planting material to avoid pathogen transmission, pesticide applications, and movement of germplasm using quarantine facilities continues to evolve.

The objective of this new edition of "A guide to sugarcane diseases" was therefore to provide a new reference book with updated and recent data. More than 80 diseases are described. Each description includes name of the disease, cause, geographical distribution, symptoms, diagnosis, strains of the pathogen, transmission, host range, epidemiology, economic importance, and control. Disease symptoms, their causal agents or pathogens' vectors are illustrated by numerous photographs. Updated procedures for the safe movement of sugarcane germplasm, common names of sugarcane diseases and their causal agent(s), as well as sugarcane producing countries/locations and their diseases are also included. Each book chapter has been written by one or two experts in the field of sugarcane pathology. We hope that this new book will be a useful new resource for sugarcane and plant pathologists in general, the sugarcane community at large, and any person interested in this subject.

1

Sugarcane morphology, anatomy and physiology

Jean-Yves Hoarau

This chapter has been updated and rewritten based on the original chapter entitled "Sugarcane morphology, anatomy and physiology" and authored by G. Claude Soopramanien in 2000.

Sugarcane (*Saccharum* spp.) is a monocotyledon belonging to the *Poaceae* family (*Gramineae*) and the *Andropogoneae* tribe.

Sugarcane is a member of the *Saccharum* genus which evolved in South-East Asia from continental areas to Melanesia. The genus is usually divided into six species or horticultural groups all of which are high-order polyploids (≥5x) (Grivet *et al.*, 2006). Two of them, *S. spontaneum* and *S. robustum*, are wild species with very little juice and sucrose and a high fiber content. They are morphologically well differentiated. *S. spontaneum* (2n = 40–128) has thin stalks, and is diverse in size, ranging from short bushy types to tall types. It has an extensive distribution from the Mediterranean region and Africa to Indonesia and Japan. *S. robustum* (2n = 60–80) has thicker long stalks and is most likely native to Southeast Asia, southeast of Sulawesi. *S. officinarum* (2n = 80), also known as 'the noble cane', is the sugar-producing species. It was domesticated from *S. robustum* around 8000 BC by Melanesian people in Papua New Guinea for its thick, juicy and sweet stalks which were grown for chewing. Two groups of old cultivars, *S. barberi* (2n = 81–124) and *S. sinense* (2n = 116–120), formerly grown in India and China, respectively, have an interspecific origin between *S. officinarum* and *S. spontaneum*. *S. edule* (2n = 60–122), cultivated for its edible aborted inflorescences in subsistence gardens from New Guinea to Fiji, represents a marginal group derived from *S. robustum*.

Modern sugarcane varieties (2n = 100–130) currently grown worldwide are artificial interspecific hybrids between the 'noble' species *S. officinarum* and the wild species *S. spontaneum*. They are the result of breeding schemes involving interspecific crosses followed by several generations of backcrosses on the 'noble' species or on hybrids, and subsequent crosses between advanced backcross products. As a result, the genome of modern interspecific varieties comes from *S. officinarum* (75–85%) and *S. spontaneum* (15–25%). The two ancestral progenitors of modern interspecific varieties differ markedly in many morphological and agronomic traits (Figure 1):

– *S spontaneum* is a highly vigorous species which develops numerous tillers from a large underground stool with a deep, dense root system capable of producing rhizomes.

It has fibrous, thin stalks (5 to 10 mm in diameter) with a hard rind surrounding a small dry pith zone frequently hollow in its central part. The leaves consist of narrow strips, often reduced to the central midrib. This species often has a high level of resistance to many diseases but can exhibit some variation for susceptibility. It is composed of highly polymorphic clones, enabling it to adapt to a wide variety of environments and abiotic stresses (some of its clones are found growing under drought stress in deserts, water-logged conditions in marshes and under a wide range of temperatures from the hottest tropics to the coldest subtropics).
– *S. officinarum* produces relatively few stalks (6 to 10 per clump) with large diameters (4 to 8 cm), completely filled with a juicy, sweet, low-fiber pith. The leaves are broad and droop under their own weight, making it easy to detach from the stalk. The species has a relatively shallow root system compared to *S. spontaneum*. It requires a lot of water and sunshine to grow and is strictly tropical. The species is susceptible to most major sugarcane diseases.

Cultivation of old *S. officinarum*, *S. barberi* and *S. sinense* varieties stopped at the beginning of the 20th century after severe disease epidemics including mosaic and sereh diseases. They were replaced by modern interspecific varieties (*S. officinarum* × *S. spontaneum*) which were first bred for disease resistance. These have also proven to be far superior in terms of cane and sugar yields. *S. officinarum* transmitted its high sucrose content and good milling characteristics, while *S. spontaneum* contributed physiological traits that allowed modern hybrids to be grown in diverse, frequently unfavorable, environments with multiple harvests. The ability of modern varieties to adapt to a wider range of environments has contributed to the worldwide expansion of sugarcane production areas over the last hundred years.

Sugarcane has specialized anatomical and physiological features that enable it to accumulate large quantities of sucrose. The features described in this chapter are those of modern interspecific varieties (*Saccharum* spp.). This review describes the anatomy of sugarcane organs and tissues in relation to their functional roles and the physiology of the plant through its successive stages of development from tillering, canopy development, growth and elongation to flowering and ripening.

The diseases described in this book can affect one or several organs of sugarcane (leaves, stems or roots) during the plant's life cycle. Severity of symptoms observed for each organ will depend on the pathways of infection and colonization patterns of pathogens within the host plant, environmental conditions, and the level of disease resistance of a sugarcane variety. In order to recognize sugarcane plants affected by a disease (if possible, at an early stage of infection), and take appropriate control measures, knowledge of the morphology, anatomy and physiology of sugarcane is very important. This knowledge will help (i) to analyze field observations associated with a phytosanitary issue, (ii) to investigate the interactions between sugarcane and a pathogen, (iii) to study and understand the epidemiological context, and (iv) to better assess the disease impact on sugarcane organs/tissues and predict yield losses. This introduction chapter therefore aims to provide a general description of the sugarcane morphology, anatomy and physiology. For further information, the reader is referred to the publications in the references list.

Morphology and anatomy

The general morphology of sugarcane is that of a large perennial bunch grass. Plant anatomy resembles that of other C_4 grasses such as maize or sorghum. As with other grasses, the stalk is composed of repeating phytomeric units containing a node, an attached



Figure 1. Morphology comparison of *Saccharum officinarum*, *S. spontaneum*, and a modern interspecific hybrid. *S. spontaneum* produces numerous thin and highly fibrous stalks containing little or no sucrose, while *S. officinarum* develops a smaller number of thick, juicy and sweet stalks. Modern interspecific varieties have intermediate morphological traits between their two ancestral progenitors (J.-Y. Hoarau).

leaf, and an internode with the youngest units at the top of the stalk. As internodes grow and age, they progressively accumulate more sucrose. When there is a sufficient number of mature internodes with high sucrose content, the cane is harvested by cutting the stalks at the base and removing the immature top. After each periodic harvest, a new crop cycle begins, with new shoots sprouting from buds in the below-ground parts of the harvested stalks. This process is known as ratooning. Sugarcane is grown on a semi-perennial basis over several successive cycles, each lasting from 12 to 18 months, depending on the growing environment and the cropping system. Sugarcane is distinguished from many common grasses by its long, indeterminant vegetative growth period, which translates into a large plant size and high biomass per unit area.

The root system

Sugarcane is usually propagated from stalk cuttings consisting of one or several nodes. A first root system develops from primordia in the root band region of the stalk nodes. These sett roots are thin and highly branched, and absorb water and nutrients to feed the young, developing shoot (Figure 2A). They are present in the first weeks of development of the shoots and gradually die back. Their function is taken over by shoot roots emerging from primordia within the root bands of underground nodes of young shoots (Figure 2B). Shoot roots are more robust and thicker (five to ten times) than sett roots. They represent the persistent root system that supports the plant's development and growth throughout its life cycle. Cane harvest at the end of the cycle causes a partial die-back of this root system. As new shoots emerge from the clump's underground nodes, new roots develop at their base. Partial survival of the former root system provides protection against drought during the early stages of growth of the ratoon crop.

The anatomical organization of young sugarcane roots resembles that of other grasses. In a cross-section, concentric cylinders of tissue are made of different cell types.

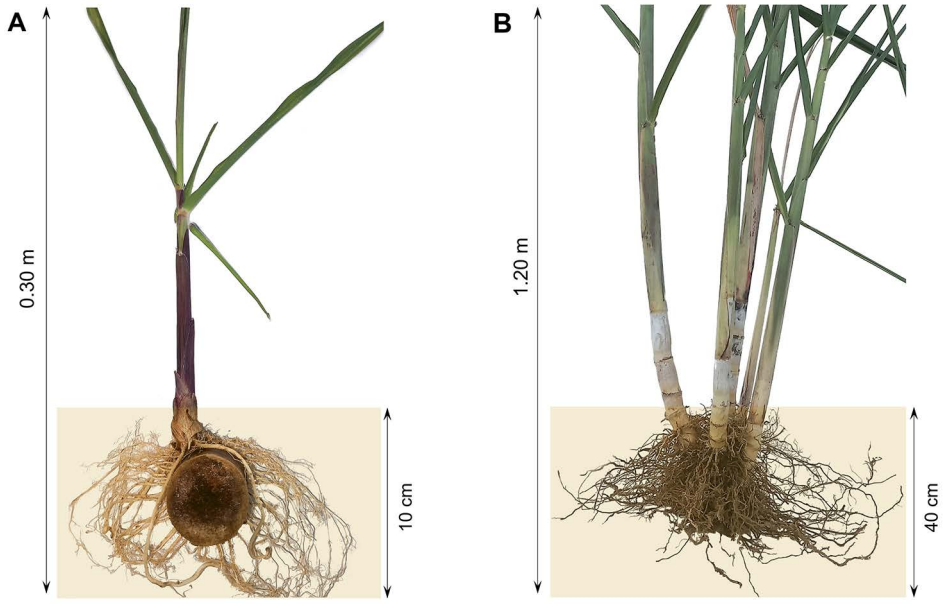

Figure 2. The root system of sugarcane variety R579 after soil washout. (A) The fine sett roots three weeks after the germination of a single-eye stalk cutting in a small pot. (B) The thick, dense shoot roots in a five-month-old sugarcane clump removed from the field (J.-Y. Hoarau).

Beneath the epidermis lies the cortical parenchyma made up of several layers of cells, surrounding the endodermis and the inner cylinder of the stele, which consists of the pericycle, the vascular system and the pith (Figure 3A) (Artschwager, 1925). In mature roots, part of the thin parenchyma cells of the cortex is disintegrated, leaving large air cavities that protect the central vascular part of the root, which remains alive and active (Figure 3B) (Pierre *et al*., 2018).

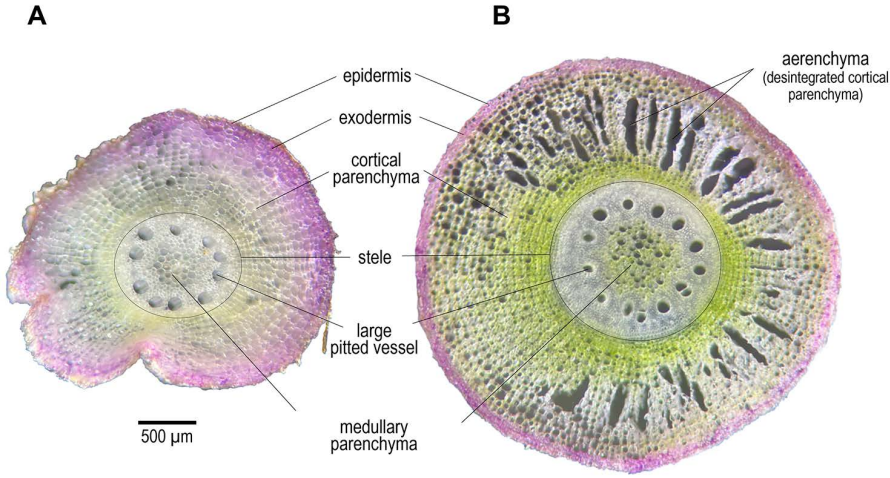

Figure 3. Transverse sections of a young root growing close to the soil surface (stereoscopic views of an unstained free-hand section of fresh material on a dark support highlighting the lumen of the cells and air cavities). (A) In a distal elongating zone near the root tip. (B) In an older proximal zone (J. Dijoux).

Three classes of roots are distinguished in the persistent root system of the sugarcane stool: superficial roots, buttress roots and rope systems (Van Dillewjin, 1952) (Figure 4). The superficial roots which tend to develop horizontally are located in the top layers of the soil. They are fine and highly branched in their distal part. Branch rootlets are densely covered with root hairs and are considered the main absorption roots. Buttress roots develop vertically and downwards. Thicker and not highly branched, their primary role is plant anchorage. Under drought conditions, water is not tapped by superficial roots, but by the rope system. Formed by the agglomeration of several roots, this type of root can grow down to a depth of several meters. Nevertheless, on vertical soil profiles, it is not always easy to distinguish superficial roots from buttress roots, and rope systems are rare (Blackburn, 1984). These three types of structural roots that grow from the stool also bear fine lateral roots (less than 1 mm in diameter) and very fine roots (less than 0.05 mm) barely visible to the naked eye. These fine roots are responsible for most of the sugarcane plant's nutrient and water absorption processes. In terms of total root length, they account for the vast majority of the root system (Chopart *et al*., 2010; Pierre *et al*., 2018).

Sugarcane's root system is highly plastic and can be easily modified according to the soil and its environment (Smith *et al*., 2005). The majority of roots are usually found close to the surface, and their density declines with increasing depth. Typically, about 50% of the root biomass is found in the first 20 centimeters of soil, and 85% in the first 60 centimeters. Differences in root development are observed between modern interspecific varieties in terms of density and depth. These differences could stem from differences in the contribution of the *S. spontaneum* species to their genomes.

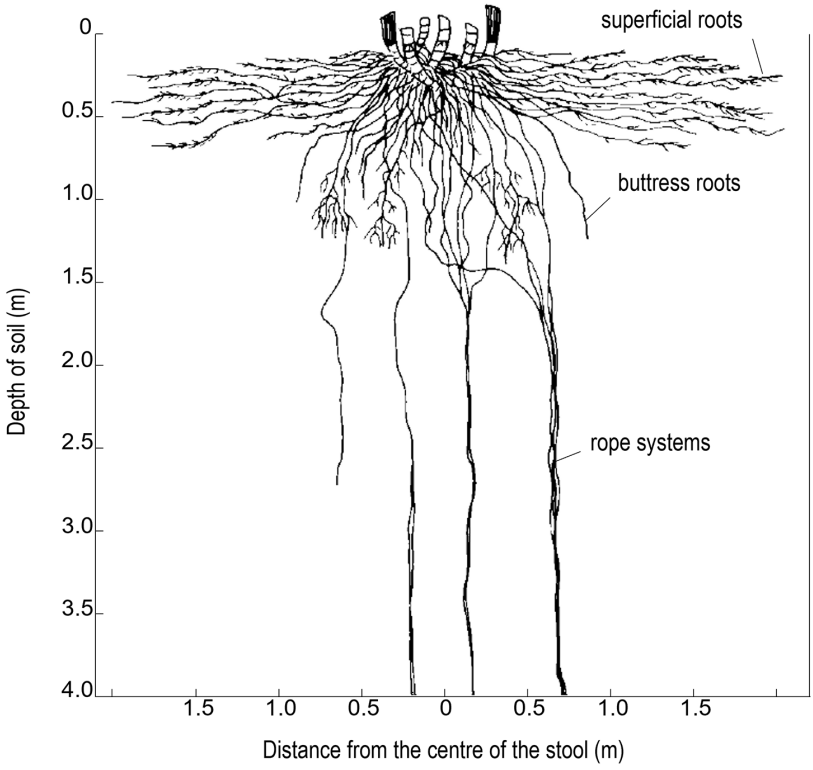

Figure 4. Schematic representation of different root types of sugarcane (After Evans, 1936 cited in Van Dillewijn,1952).

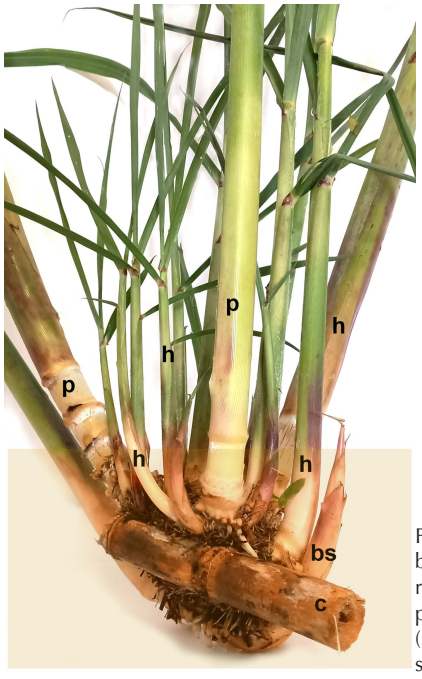

Figure 5. Tillering process from underground branching of a stool of sugarcane variety R579 after removal of the roots. The stalk cutting (c) produces primary shoots (p) and successive branches of higher (h) orders from bud sprouting (bs) to form a complex structure (J.-Y. Hoarau).

The shoots

In the plant crop, sugarcane shoots arise from the sprouting of nodal buds on seed cane stalk cuttings, and in subsequent ratoon crop cycles (after each cane harvest), from sprouting of nodal buds in the below-ground structure of the sugarcane stool. Tillering, the process by which new shoots emerge, is the result of successive underground branching from primary tillers (Figure 5). Branches of higher order result from sprouting of axillary buds on underground nodes at the base of older tillers. The repeated pattern of bud sprouting at the lowermost node of erect shoots results in a complex branched structure. The number of tillers is influenced by growing conditions (fertilization practices, soil water status and temperature) and can vary from one variety to another. The tillering ability of modern interspecific varieties has been mainly provided by *S. spontaneum*, which has a greater propensity to produce tillers than *S. officinarum*. The wild sugarcane species produces a greater level of branching from its primary stalk, and it has the ability to produce rhizomes while the noble species cannot (Matsuoka and Garcia, 2011; Glassop *et al.*, 2021). Modern varieties have an intermediate branching pattern between their two ancestral progenitors. High tillering capacity is generally associated with a good ratooning ability.

The stalk

Stalk growth

The tillers gradually grow in circumference and length to give rise to stalks. In continuing good conditions, stalk internodes tend to increase in length from the base toward the apex (acropetally) but become shorter again at the top of the stalk where elongation is active (Figure 6A). Within each phytomeric unit, cell elongation and maturation take place in a basipetal manner with a wave of development moving down the unit (Rae *et al.*, 2014). Thus, the leaf apex matures first, then the base elongates to reach full size, then the sheath and finally the internode downwards. As for other *Poaceae*, the last region to expand is the base of the internode. A residual meristematic zone, termed the intercalary meristem, is

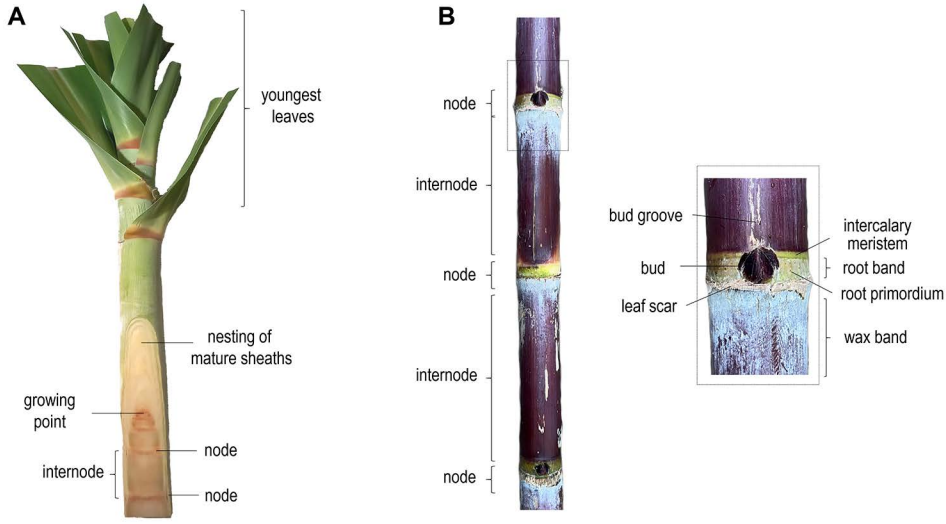

Figure 6. Detailed anatomy of the sugarcane stalk. (A) Split top part. (B) Mature portion seen from a perpendicular angle.

located at the base of mature internodes (Figure 6B). The intercalary meristem is important for the resumption of growth to maintain stem erection in response to gravity after winds and other physical phenomena cause the stem to tilt or fall.

Stalk anatomy

The mature cane, cylindrical in shape, is clearly differentiated into joints each comprised of a node and an internode above it (Figure 6B). The node consists of a lateral bud located in the axil of the leaf sheath or of its scar, a band containing root primordia, and a ring of residual intercalary meristem.

When the leaf sheaths are detached, the buds are located just above the leaf scar. They include the lateral meristem covered by leaf-like scales that protect the meristem from desiccation, physical damage, and from pathogen attack. The meristem of buds is an embryonic shoot structure containing preformed nodes and internodes. Their development is blocked by the apical dominance of the vegetative meristem at the apex of the stalk. Germination of buds on the whole stalk may happen following stalk damage caused by insects (the buds germinate below the tunnels of sugarcane borers), diseases (development of older buds promoted by leaf scald infections). Some morphological details of the bud (size, shape, color and hairs on either side of its external scale) constitute useful criteria for variety identification. Root primordia also represent dormant meristematic zones. Within a few days of planting in a humid soil, the root primordia develop and elongate to give fine, branched sett roots associated with the development of the young shoot.

The sugarcane stalk diameter ranges usually from 2.5–5.0 cm, depending on the variety and the environmental conditions. The general organization of the internal anatomy of a sugarcane stalk is typical of that of a monocot stem (Figure 7). A transverse section of a mature internode exhibits vascular bundles scattered radially throughout the ground tissue made of a whole mass of parenchyma cells with no differentiation of endodermis, cortex, pericycle and pith. Xylem and phloem vessels are conjoint, collateral, and without cambium between them or any secondary growth in girth by cell division.

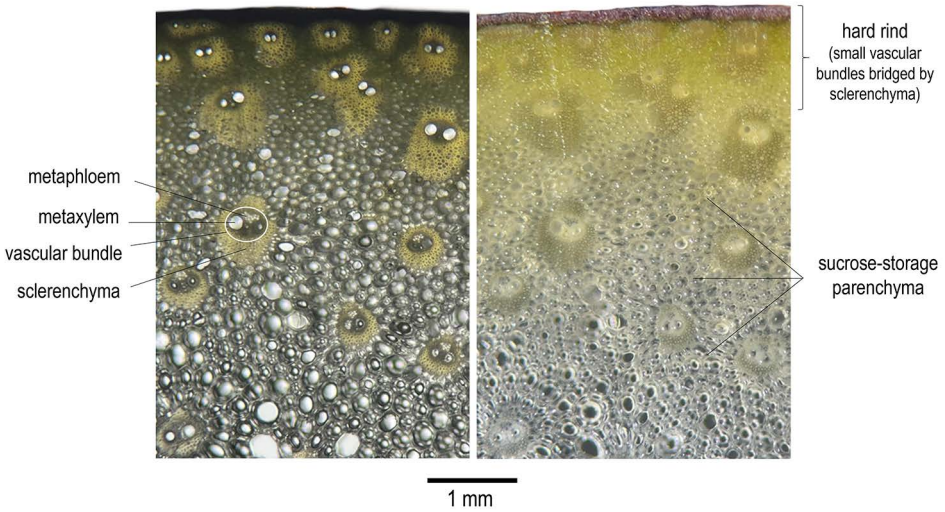

Figure 7. Distribution of the vascular bundles in a transverse section of a mature sucrose-filled internode of sugarcane variety R579 (stereoscopic views of an unstained free-hand section of fresh material). Left: with under-lighting. Right: with overhead lighting on a black support. (J. Dijoux).

Each vascular bundle is surrounded by a few layers of sclerenchyma cells. Within each bundle, the phloem is oriented toward the stalk periphery and the xylem toward the inside of the stalk.

The stalk of modern interspecific varieties resembles that of the 'noble' sugar-producing *S officinarum* species. Its specialized ability to transfer and store large quantities of sucrose is related to the anatomical features of its tissues. In contrast to most plant stems, sugarcane stalks have the capacity to store exceptionally high amounts of soluble sucrose, rather than insoluble polysaccharides such as starch or cellulose, in large parenchyma cells. This storage parenchyma is the predominant tissue of the stalk (Figure 7). At maturity, sucrose usually represents 12–14% of stalk fresh weight up to 16% in the most efficient sugar-producing varieties when stalks are grown in very favorable environments. Size and distribution of parenchyma and vascular bundles are not uniform across the stalk. Fibrovascular bundles are more dispersed in the parenchyma in the central zone of the stalk where thin-walled parenchyma cells become larger. The latter are somewhat circular in shape in a cross section and rectangular in a longitudinal view. They are largely vacuolated. In the stalk periphery, there is no parenchyma but numerous small vascular bundles bridged by enlarged polar caps of sclerenchyma that forms a hard rind. These bundles in the periphery are not involved in long-distance transfer of solutes in comparison to large vessels located in the central zone. A thick-celled wax-covered epidermis covers the peripheral rind.

Vascular bundles in the internode are nearly parallel to each other. However, their organization becomes complex when approaching the node. Many vascular bundles become branched, some of which continue to the next internode, while others pass into the leaf sheath or connect with the bud and root primordia. Nodal tissues are more dense in this area of extensive branching.

The leaf

The leaves of sugarcane are attached alternately to the nodes of the stalk. They are arranged in two vertical ranks on opposite sides of the stalks which lie approximately in the same plane, like most *Poaceae* (alternate, distichous phyllotaxis). The leaf comprises a thin and long blade with variable width supported by a thickened midrib, a sheath which wraps around the internode and a collar between them (Figure 8). Externally, the collar has two deltoid areas, called dewlaps, that almost meet on the back surface of the midrib. These two hinge areas give mobility to the blade.

The leaf blade and leaf sheath have parallel venations typical of monocots. Both leaf segments have an abaxial surface with similar appearance. However, the anatomical organization of their tissues is not identical regarding their differences of functions.

The leaf lamina

Numerous parallel venations of the blade correspond to vascular bundles starting at an acute angle from the midrib. A transverse section of the lamina exhibits bundles of different sizes alternating from one type to another. Small bundles are regularly interspersed between alternating large- and medium-sized bundles across the leaf lamina (Figure 9). The internal anatomy of the sugarcane leaf is characteristic of C_4 grasses. Each vascular bundle is encircled by a sheath of thick-walled chlorophyll-bearing cells which, in turn, is surrounded by chlorophyll-rich mesophyll cells. Mesophyll cells adjacent to the bundle sheath are thin-walled, much branched and loosely arranged with intercellular spaces to allow gas circulation (Rae *et al.*, 2014). Large vascular bundles

usually enclose two large xylem vessels (metaxylem) connected with smaller ones, and the phloem is oriented toward the lower surface of the leaf and surrounded at its base by sclerified cells (phloem fiber). The phloem is composed of thin-walled sieve tubes and companion cells ensuring the export of sugars produced in the leaf. Longitudinal bundles are interconnected by numerous, small transverse bundles.

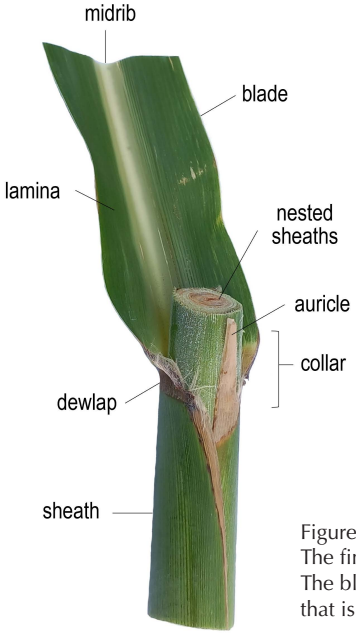

Figure 8. Leaf blade and leaf sheath in the top part of a stalk. The fine leaf lamina is supported by a stiff midrib. The blade is connected to the stalk through its cylindric sheath that is attached at the base of a node (J.-Y. Hoarau).

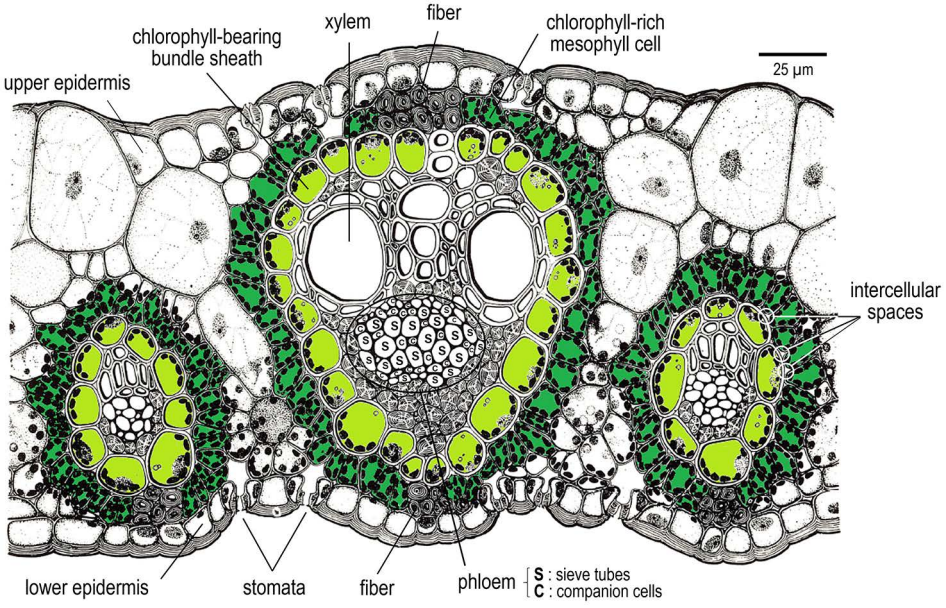

Figure 9. Schematic representation of the anatomy of a sugarcane leaf lamina showing one large vascular bundle flanked by two small ones drawn by Martin (1961) from a brightfield microscopy view. The bundle sheath cells and chlorophyll-rich mesophyll cells have been colored in light and dark green, respectively, for better differentiation.

Together, the layers of mesophyll cells and bundle sheath cells (single-cell layers in the case of the sugarcane leaf as shown in Figure 9) are termed the 'Kranz' anatomy given their resemblance to a wreath-like structure, 'kranz' meaning wreath in the original German language (Sage *et al*., 2014). This anatomy is typical of plants with the C_4 plant photosynthetic pathway in which CO_2 is initially assimilated into a four-carbon (C_4) organic acid. An intimate linkage exists between the 'Kranz' spatial configuration of the two chlorenchymatous cell types and C_4 photosynthesis related to a functional partition of labor (Dengler and Nelson, 1999). The mesophyll cells are specialized for initial primary carbon assimilation and CO_2 transportation to the bundle sheath in which a second photosynthetic reassimilation of CO_2 takes place. The 'Kranz' anatomy together with the partitioning of biochemical components between cell types is highly efficient for carbon capture (Rae *et al*., 2014). The mesophyll cells are toward the exterior of the leaf in contact with intercellular airspace, and the inner bundle sheath cells which produce advanced elaborate photo-assimilates are closer to phloem vessels. Sugarcane has the two leaf characteristics of C_4 plants related to a short diffusion pathlength for metabolites between mesophyll and bundle sheath allowing high rates of photosynthesis: (i) a relatively low ratio of mesophyll to bundle sheath cells (near two, whereas it is over four in C_3 species) and (ii) a relatively high vein density (short interveinal distance of about 100 µm on average) (Dengler and Neslon, 1999; Sage *et al*., 2014).

Each vascular bundle has an abaxial cap of sclerenchyma cells adjacent to the lower epidermis of the leaf blade. These cells with highly lignified, thick walls are long and slender. They form strands of fiber which extend longitudinally giving the blade rigidity with a minimum amount of tissue. These subepidermal sclerenchyma strands are strongly developed on the lower side of the midrib of the leaf to support the blade (Figure 10C). The fiber strands may also flank large vascular bundles on their upper side. Large bundles occupy almost the entire thickness of the blade, while small bundles are only located on the abaxial side of the leaf opposite to bulliform cells. These large thin-walled cells appear on the upper surface of the leaf as longitudinal bands of nearly square cells. Bulliform cells contain a large vacuole and are thought to assist leaf unrolling and rolling in response to the water status of the plant. The lower and upper surfaces of the leaf have an epidermis made of longitudinal brick-shaped cells. These are thick, lignified cells covered by wax. This structure gives rigidity to the leaf, limits water loss, and protects underlying softer tissues against pathogens and insects. Stomata of sugarcane are typical of *Poaceae* (two dumbbell-shaped guard cells flanked by two lateral subsidiary cells) and appear in longitudinal bands along both sides of the leaf. They are more abundant in the lower epidermis of the leaf.

The leaf midrib

The midrib protrudes from the underside of the leaf blade and has a groove on its upper side, giving it a half-moon shape in a transverse section (Figure 10A). Its anatomical organization is simpler than that of the leaf lamina (Martin, 1961; Rae *et al*., 2014). The vascular bundles of the midrib lack the photosynthetic rings of bundle sheath and rich-chlorophyll mesophyll cells typical of the leaf lamina (Moore, 1987a). The midrib has no bulliform cells in its upper surface and a higher proportion of thick-walled cells in both its upper and lower parts. This gives the midrib a rigid structure with no capacity for movement and provides a support function for the lamina. The upper epidermis is formed of compact brick-shaped cells with a thick, lignified wall which overlays two to three layers of collenchymatous cells (Rae *et al*., 2014) (Figure 10B). Beneath this solid upper region is the core of the midrib consisting of numerous layers of large parenchyma cells which appear somewhat hexagonal in cross section. These thin-walled,

colorless cells have little structural differentiation and are spongy in nature (Figure 10A). The absence of chlorophyll in this core region gives the whitish appearance of the upper surface of the midrib. The vascular bundles are confined to the lower rib region. They are overlaid with collenchyma cells (Moore, 1987a) and supported below by sclerenchyma fibers much more strongly developed than below bundles of the leaf lamina. This sclerenchyma of thick-walled cells, which abut directly on the lower epidermis, give rigidity, strength, and protection to the midrib (Figure 10C). Groups of small chlorophyll mesophyll cells are confined to the lower edge of the leaf rib below the abaxial epidermis in an alternate position with sclerenchyma fibers. These cells provide the green color of the midrib on its external surface.

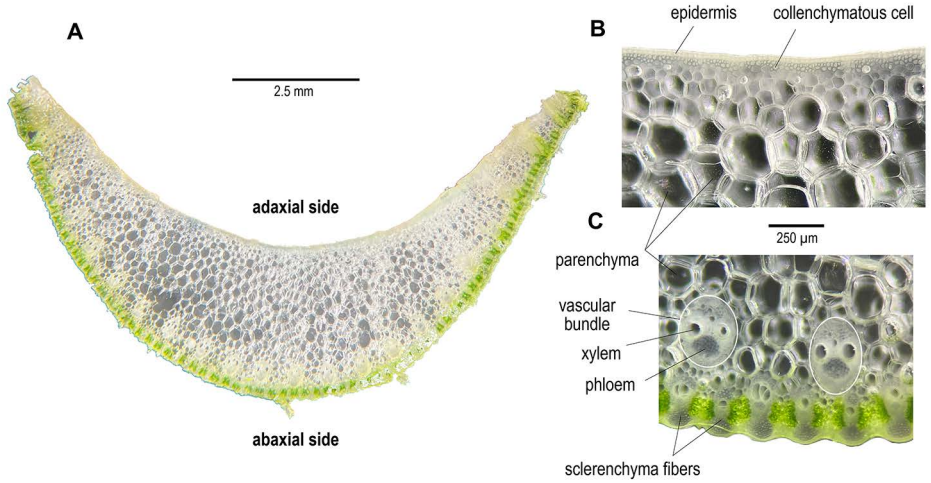

Figure 10. Transverse sections of a leaf midrib from the lower third of its blade (stereoscopic views of an unstained free-hand section of fresh material on a dark support highlighting the lumen of the cells). (A) Overview of the midrib. (B) Detailed anatomy in an enlarged view of its upper edge and (C) of its lower edge (J. Dijoux).

The leaf sheath

The leaf sheath is attached to the stalk at a node all around the stalk. The leaf sheath is usually about four times shorter than the leaf blade, except in the top part of flowering stalks where the blade is shortened and the sheath elongates to reverse their relative sizes.

The leaf sheath has fewer functions than the leaf blade and is anatomically simpler (Figure 11). Its role to support and strengthen the blade in full continuity of midrib and collar is provided by longitudinal pads of sclerenchymatous fibers below its outer epidermis. Its conducting vascular bundles are organized in radial rows. Radial files of bundles alternate with large areas of parenchyma often perforated by air cavities in the middle of the sheath, midway between the sheath base and the blade. Typical chlorophyll-bearing cells of the bundle sheaths of the leaf are frequently replaced by sclerenchyma cells. This surrounding sclerenchyma jacket is a sheath continuation of the stalk bundles. Vascular bundles and their phloem vessels are on average larger than in the leaf, reflecting the major photosynthate export function of the leaf sheath. The bundles located in the middle of the sheath are the largest in diameter. These are the bundles that penetrate into the central area of the stalk and may extend to a depth of several internodes. Other radial files of smaller bundles penetrate less deeply into the stalk and are shorter vertically.

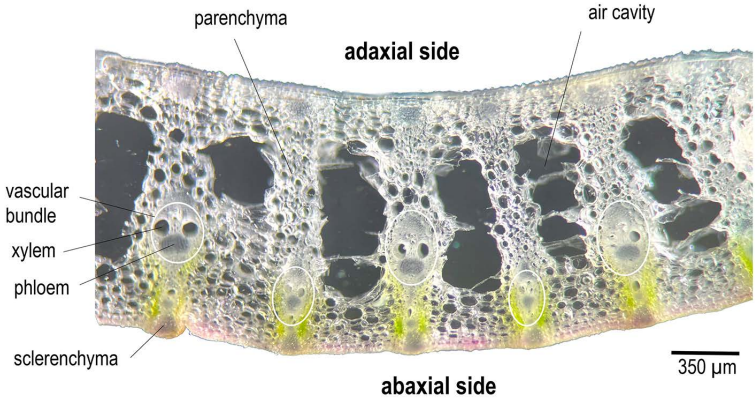

Figure 11. Anatomy of a portion of a leaf sheath of sugarcane variety R579 in a transverse section midway between the sheath base and the blade joint (stereoscopic view of an unstained free-hand section of fresh material on a dark support highlighting the lumen of the cells and the air cavities) (J. Dijoux).

The inflorescence

When environmental conditions are favorable, sugarcane stalks may flower, depending on the variety. The apical meristem ceases forming leaf primordia and produces a large branched inflorescence (Figure 12A). The sugarcane inflorescence is an open panicle. Its morphology (shape, degree of branching and size) is typical for species and variety. The main axis (rachis) carries rachilla which bear numerous spikelets (single florets) (Figure 12B). The anatomy of the spikelet is typical of grass spikelets. The sugarcane spikelet consists of an outer glume and an inner glume, a gynoecium with one ovule, a pistil with two stigmas, and an androecium of three stamens bearing bilobed anthers. It is surrounded at its base by long bristles of silky appearance.

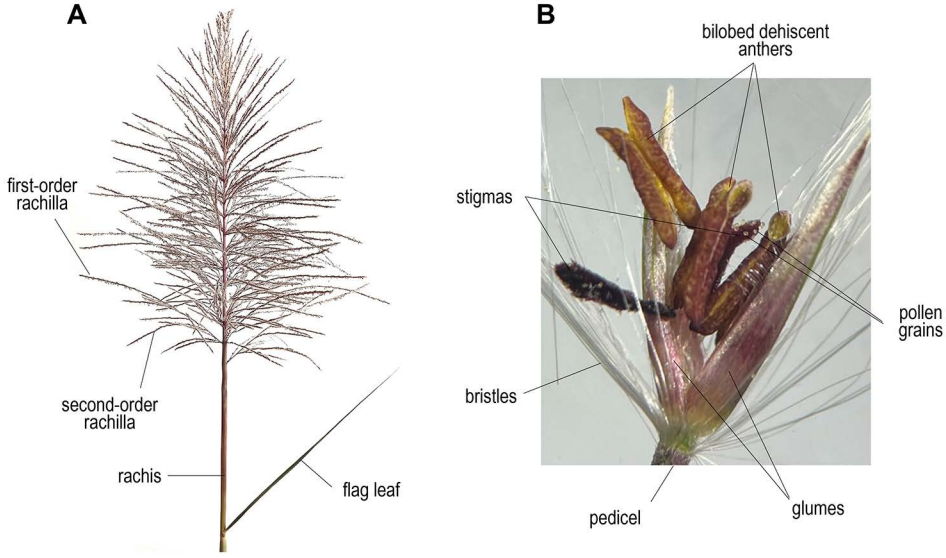

Figure 12. Anatomy of a sugarcane inflorescence (also called tassel) composed of several thousands of tiny florets (spikelets). (A) Open-branched panicle up to 60 cm long. (B) Close view of a spikelet (4 mm) (J.-Y. Hoarau).

Physiology of growth and development

Sugarcane is cultivated in humid tropical and subtropical regions of more than 100 countries around the world. Like all cultivated C_4 species except two (maize and *Miscanthus*), sugarcane is largely absent from temperate climates due its long crop cycle and high sensitivity to cold. The four main growth and development stages of sugarcane include (i) germination and establishment, (ii) tillering and canopy development, (iii) growth and stalk elongation and cane ripening, and (iv) flowering. The duration of the different phases is influenced by factors such as the environment and the cultivation practices, and to a lesser extent by the sugarcane variety.

Germination and establishment

Several environmental factors influence germination of buds on seed cane stalk cuttings (Van Dillewijn, 1952). Temperature is one of the most important factors. Slight differences may exist between varieties in response to temperature. Medium, persistent soil moisture content around the cutting is also a factor of great importance. In addition, high nitrogen levels favor stalk bud germination. Primary tiller development is favored by high light intensity, medium-moist soil and relatively high temperatures. Generally, a base temperature around 15–17°C is required for initial tiller appearance with optimum production at 30–35°C. The process of germination is characterized by a considerable increase in respiration and consequently soil aeration is an important factor.

Tillering and canopy development

After germination, tillering from underground branching of primary tillers is the next step that provides a high stalk population conducive to a good yield. The most important climatic factors influencing tillering are light (intensity and day-length) and temperature which needs to be relatively high (tillering gradually increases until a maximum around 30°C). These two factors may be limiting ones in subtropical locations during the cool season (Van Dillewijn, 1952). Tillering is also favored by nitrogen fertilization. Under specific environmental conditions, the number of tillers is a varietal characteristic. As a result, seed cane density at planting can be slightly modulated according to the variety grown, to achieve good tillering during the plant cane crop. However, some competition occurs between tillers. When the canopy completely covers the ground, a number of smaller tillers die in the shade while larger tillers continue to elongate. At canopy closure, the tiller population in a field (for a given row-spacing) reflects an equilibrium mainly determined by available soil resources, the variety grown and, to a lesser extent, initial seed cane density.

Growth and stalk elongation

The growth and stalk elongation phase occurs after tillering, and this phase has a considerable influence on total stalk biomass that will be harvested at the end of the crop cycle. Growth and stalk elongation is often referred to as the 'boom or grand growth stage' due to a sharp increase in stalk elongation and weight, as well as a high rate of leaf appearance. It takes place between the 4th and the 7th month of the crop cycle, depending on the variety, environment and cultivation practices. Growth and stalk elongation is strongly linked to temperature with a minimum base temperature of 16–18°C and an optimum temperature around 35°C. The beginning of stalk elongation is believed to be determined by canopy closure or by the accumulation of thermal time until a specific threshold is reached. Leaf appearance rate is a function of thermal time (cumulative

mean daily temperature minus a base temperature). Differences in leaf appearance rates are observed between varieties, and this rate declines slightly with crop age (Bonnet, 1998; Singels *et al*., 2005). Moisture, nutrition and temperature are the most important factors likely to affect the boom stage. A delay in growth during periods of moisture stress is usually reflected in a few shorter internodes along the stalk. Under non-limiting conditions of growth (temperature, water and nutrient supplies), nodes formed during the boom stage are generally spaced at intervals of about 15–25 cm, depending on the variety. Under tropical conditions, with daytime temperatures above 25°C and nighttime temperatures above 15°C, sufficient soil moisture and adequate radiation levels, stalk elongation rate can easily reach 1–2 cm per day.

During the boom stage, sugarcane leaf blades are about one meter long and 4–8 cm wide, depending on the genotype. At full growth, most varieties permanently have at least 10 green leaves, as the lower leaves of the stalk senesce and new ones appear at the top. Hence, the ratio of green leaf area to ground area (gLAI) of sugarcane can reach relatively high values under usual cropping practices (planting density, fertilization). High gLAI values combined with its C_4 photosynthetic type make sugarcane a particularly efficient plant for converting solar radiation to biomass. While the world's average cane yield (millable stalks) currently stands at about 70 t/ha/year, yields in excess of 100 t/ha/year are common in many producing regions of the world.

Sucrose accumulation and cane ripening

During plant development, sucrose ($C_{12}H_{12}O_{11}$) synthetized in sugarcane leaves is translocated in both mature and immature internodes up to the apical meristem. Sucrose accumulation in the plant is a continuous process throughout the plant's life cycle. Sucrose accumulation results from a dynamic balance between leaf photosynthesis, plant respiration, cell biosynthesis for growth and sucrose storage (Wang *et al*., 2013). This balance depends on the stage of plant development and the season as well as the age of the internode. In young, growing, immature internodes, photo-assimilates are mainly used to build cell walls, while in mature internodes they are mainly stored as sucrose. At the whole-plant level, sucrose as a percentage of the dry matter is less than 10% in young stalks but gradually increases to about 50% at harvest. Sucrose is accumulated in the stalk with an increasing gradient from top to base. Sucrose accumulation in the basal internodes continues after they have reached their maximum length, until their parenchyma cells are completely filled. As the harvest season approaches, the temperature often drops and the water deficit increases. Since plant growth is more sensitive than photosynthesis to these two abiotic stresses, fewer photoassimilates are mobilized for growth and, consequently, sucrose storage is gradually favored. Accumulation is faster at the end of the cycle when cane growth is reduced or stopped (when temperature drops below a minimum base of 16–18°C). Optimum maturity of sugarcane in terms of sucrose percent of cane weight is favored by a relatively dry season (cane moisture content ideally dropping from about 85% to 70%) and by increasing temperature differences between day and night at harvest season (limited sucrose loss by dark respiration at night).

Flowering

Flowering of the cane stalk may appear under favorable environmental conditions in varieties having the ability to flower. The process of flower initiation is controlled by a photoperiod stimulus (Moore, 1987b). Flower initiation occurs during a 3-week period during the waning daylight season, when the average day length for this period is about 12 hours and 15 minutes. Inflorescences appear months thereafter. Stalks of varieties

that could have the ability to flower but that are still at a juvenile stage of development at the time of this decreasing daylength will not perceive the flower stimulus and will therefore not flower. Moreover, water stress or minimum temperature below 18°C during the inductive signal period and the differentiation period of the meristem may result in reversion to the vegetative condition or may delay or stop inflorescence development (Julien *et al*., 1989). Since flowering is synonymous with a cessation of stalk elongation and cane tonnage increase, the cultivation of varieties with little or no flowering is preferred. Flowering intensity decreases at elevations above 400 to 500 meters, or in high latitudes (in subtropical regions) or during crop years characterized by a period of water deficit in the weeks preceding flower initiation (rainfall and/or irrigation regime below the plant's water requirements).

Disease control

Sugarcane is subject to a large number of diseases as reported in this book. Economic importance varies between diseases and for a particular disease from one region to another, usually because of varying epidemiological contexts. Diseases can affect various physiological processes in sugarcane, such as photosynthesis, plant growth, or sucrose production and storage.

When a sugarcane industry is confronted with disease epidemics or outbreak risks, various ad-hoc preventive or curative control measures can be taken in the short term. These measures may include the safe germplasm movement through quarantine units, healthy seed cane nurseries for planting, eradication of old diseased fields, regular inspection of growing fields and possibly removal of plants with disease symptoms, and pesticide applications.

Nevertheless, the best long-term measure to control most sugarcane diseases is to plant varieties bred for a good resistance or tolerance level. This is the most economical and safe measure that can often provide the best sustainable yields. Selecting and growing varieties carrying resistance genes is an efficient strategy to control most damaging diseases. The most famous major resistance gene identified so far in the sugarcane genome is *Bru*1 that confers resistance to brown rust (Healey *et al*., 2024). Primers to detect this gene have been developed and are currently used in several breeding programs to screen for resistance to this disease. Based on numerous breeding and pathology studies, sources of resistance to many sugarcane diseases are found in the wild species *S. spontaneum*. Useful sources of resistance from this key ancestral species have been recently characterized in genomic studies for orange rust and yellow leaf diseases, with the objective to incorporate them into breeding germplasm and to produce new resistant varieties (Dijoux *et al*., 2024; Hoarau *et al*., 2025).

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II

Diseases caused by bacteria

Bacterial chlorotic streak

Muqing Zhang, Meilin Li

Cause

Xanthomonas sacchari Vauterin *et al*. 1995, bacterium.

Xanthomonas sacchari is a Gram-negative bacterium that was first described based on a bacterial isolate obtained from diseased sugarcane in Guadeloupe (Vauterin *et al*., 1995). Subsequently, this bacterial species was also reported in China (Fang *et al*., 2015; Sun *et al*., 2017) and Argentina (Mielnichuk *et al.,* 2021).

Geographical distribution

Argentina, China, Guadeloupe, Philippines.

Symptoms

Sugarcane leaves of cultivar ROC22 infected with *X. sacchari* develop chlorotic streaks that appear as pale-yellow lines along the leaves (Figure 1). The streaks resemble the 'pencil lines' characteristic of leaf scald caused by *Xanthomonas albilineans* extending longitudinally from the leaf base to the tip (Figure 2) (Sun *et al*., 2017). As the disease progresses, infected plants may exhibit wilting and yellowing, especially under dry conditions (Figure 3), and leaves may eventually turn brown.

Diagnosis

Diagnosis is initially based on visual symptoms but can be confirmed through bacterial isolation on selective XAS medium (Davis *et al.,* 1994; Li *et al.,* 2022; Sun *et al.,* 2017) and PCR targeting specific genes, including *16S* rDNA (GenBank Accession No. KX925202.1), *16S-23S* rDNA internal transcribed spacer (ITS) (GenBank Accession No. KX925203.1), and gyrase B (*gyrB*) gene (GenBank Accession No. KY432568.1) (Gonçalves and Rosato, 2002). Colonies of *X. sacchari* are circular, convex, glossy, translucent, non-mucoid, and faintly yellow (Sun *et al.,* 2017).

X. sacchari is a capsular bacterium characterized by bullet-shaped cells measuring 0.6–1.0 × 0.2–0.3 µm, and it is motile with a single polar flagellum. The optimal growth temperature ranges from 25°C to 28°C (Sun *et al.,* 2017).

The genome of *X. sacchari* strain DD13 consists of a 4.88 Mb circular chromosome with a GC content of 69.61%, encoding approximately 4,022 genes, including carbohydrate-active enzymes (CAZymes) that break down plant cell walls to facilitate infection. The genome also contains six prophages, three genomic islands, and transposable elements contributing to bacterial virulence and adaptability (Li *et al.,* 2022; Pieretti *et al.,* 2015; Studholme *et al.,* 2011).



Figure 1. Sugarcane leaf with a pale, large yellow streak parallel to the midrib (M. Zhang).



Figure 2. 'Pencil line' streaks extending from the base to the tip of the leaf (M. Zhang).



Figure 3. Bleaching, necrosis, and wilting of sugarcane leaves (M. Zhang).

Strains of the pathogen

Strains of *X. sacchari* have been isolated from various sugarcane-producing regions, but their pathogenic variability remains unknown.

Transmission

X. sacchari thrives in warm, humid environments and primarily infects sugarcane by entering through wounds or natural openings like stomata. It is primarily transmitted by contaminated planting materials, irrigation water, and farm tools. Rain splash and wind can also spread the bacteria locally.

Host range

While sugarcane is the primary host, *X. sacchari* has been found in other plants, including common beans, oranges, and rice, and was even identified in an insect from

a banana plant (Studholme *et al.,* 2011). This broad host range raises concerns about its potential to infect other crops (An *et al.,* 2020).

Epidemiology

The incidence and severity of bacterial chlorotic streak is influenced by environmental factors, particularly rainfall and temperature typical to humid tropical and subtropical climates. Irrigation systems and contaminated equipment spread the pathogen that persists in plant debris, thus increasing the risk of reinfection in subsequent crops (An *et al.,* 2020).

Economic importance

Bacterial chlorotic streak can cause significant yield losses in sugarcane, with reductions of up to 30–50% when disease incidence and severity are high. The disease adversely affects stalk sugar content and quality, leading to not only yield reductions but problems in sugar extraction and bioethanol production.

Control

Effective management of *X. sacchari* requires an integrated disease control strategy. The use of resistant sugarcane varieties, certified disease-free planting material, and strict field sanitation are essential preventive measures. Implementing crop rotation and avoiding continuous monoculture can help reduce pathogen buildup in the soil. Chemical control using copper-based bactericides is an option but should be applied judiciously to mitigate the risk of pathogen resistance. Early detection and timely intervention are crucial to minimizing disease impact.

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Bacterial leaf wilt

San-Ji Gao

Cause

Pantoea stewartii (Smith 1898) Mergaert *et al*. 1993 (Basonym = *Pseudomonas stewartii* Smith 1898), bacterium.

Geographical distribution

China.

Symptoms

Leaf blade bleaching occurs at the disease initiation stage (Figure 1). Subsequently, blight and necrotic lesions develop on whole sugarcane leaves of young or older plants (Figure 2). These symptoms resemble those of leaf scald but white to yellow, narrow 'pencil-line' streaks characteristic of this latter disease are not present. These lesions expand and coalesce resulting in leaf death. The pathogen can colonize the stalks which do not display distinct symptoms.

Diagnosis

The bacterial pathogen is isolated from diseased leaf samples by surface sterilization with 75% alcohol followed by tissue homogenization in sterile water. Upon plating of macerated leaf tissue on nutrient agar medium and incubation at 28°C for 24–36 hours, yellow bacterial colonies will grow. These colonies with translucent edges are circular, convex, and smooth (Figure 3). Colonies of *P. stewartii* are formed by straight rods and nonencapsulated cells as observed by transmission electron microscopy (Figure 4). Identification of the pathogen is usually performed by PCR and sequencing of the bacterial 16S rRNA gene using universal primer pair 63F and 1387R[1] that produces a 1,362 bp DNA sequence (Marchesi *et al.*, 1998).

The disease can also be diagnosed using a PCR assay with primers PANST_rpoB_fwd and PANST_rpoB_rev[2] that specifically target two subspecies of *P. stewartii*, namely *P. stewartii* subsp. *stewartii* (*Pss*) and *P. stewartii* subsp. *indologenes* (*Psi*). A positive reaction of this assay results in the production of a 262 bp fragment with both *Pss* and *Psi* (Kini *et al.*, 2021). Two other primer sets, 3614galE/3614galEc and DC283galE/DC283galEc, can be used to differentiate *Psi* and *Pss*, respectively (Gehring *et al.*, 2014). Amplification with these primers[3] results in a 250-bp fragment (Shin *et al.*, 2022).

1. 63F: 5′-CAGGCCTAACACATGCAAGTC-3′; 1387R: 5′-GGGCGGWGTGTACAAGGC-3′.
2. PANST_rpoB_fwd: 5′-CACCGGTGAACTGATTATCG-3′; PANST_rpoB_rev: 5′-GTCCTGAGGCATCAATGTGT-3′.
3. 3614galE: 5′-CGACCTGTTTGCCTCTCACC-3′; 3614galEc: 5′-CATCAGCTTGGAGGTGCCG-3′; DC283galE: 5′-CGACCTGTTTGCCTCTCACT-3′; DC283galEc: 5′-CATCAGCTTGGAGGTGCCA-3′.



Figure 1. Leaf blade bleaching and necrosis on the spindle leaves of a young sugarcane plant (S.-J. Gao).



Figure 2. Leaf blight and necrotic lesions on the spindle leaves of a young sugarcane shoot (S.-J. Gao).



Figure 3. Colonies of *Pantoea stewartii* growing on nutrient agar (S.-J. Gao).

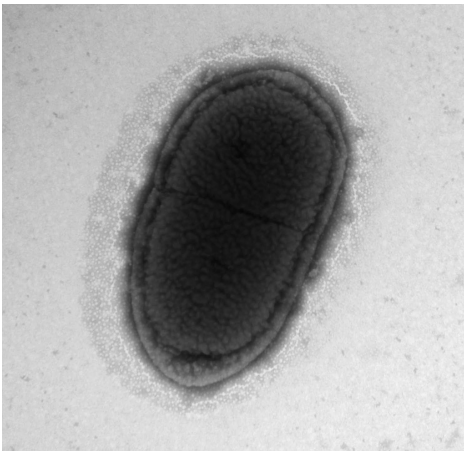

Figure 4. *Pantoea stewartii* strain PSCN1 observed by transmission electron microscopy (S.-J. Gao).

For further identification and characterization of the causal agent of bacterial leaf blight, the complete genome can be sequenced using second-generation (such as Illumina Hiseq) and third-generation platforms such as Oxford Nanopore. Sequences of the PCR products or the genome sequence should be blasted against the NCBI data base for comparison with strain PSCN1 of *P. stewartii* to confirm pathogen identity[4]. This pathogenic strain has been proposed to belong to the subspecies *Pss* based on its 16S rDNA sequence (Cui *et al.*, 2021), but this strain could be assigned to the subspecies *Psi* using the pan-genome sequence (Chu *et al.*, 2024).

4. https://www.ncbi.nlm.nih.gov/nuccore/CP046585

Strains of the pathogen

Two pathovars of *Psi* were proposed i.e. *P. stewartii* subsp. *indologenes* pv. *cepacicola* pv. nov. and *P. stewartii* subsp. *indologenes* pv. *setariae* pv. nov. (Koirala *et al*., 2021). The first pathovar causes symptoms on *Allium* spp. (leek, onion, chive, and Japanese bunching onion) and on foxtail millet (*Setaria italica*), pearl millet (*Pennisetum americanum*), and oat (*Avena sativa*). The second pathovar infects only members of the *Poaceae* family (foxtail millet, pearl millet, and oat). Strain variation in sugarcane has not been reported.

Transmission

In maize, *Pss* is thought to be mainly spread over long distances by infected seed. The corn flea beetle (*Chaetocnema pulicaria*) plays an important role in the pathogen's transmission in the field (Esker and Nutter, 2003). This insect is the only known overwintering niche for *P. stewartii* and is the most important factor for survival, dissemination, and infection of the pathogen. Alternative grass hosts also serve as reservoirs of *P. stewartii* (Esker and Nutter, 2003).

Host range

Strains of *P. stewartii infect* maize (*Zea mays*), sugarcane and other grasses, and beetles (Chu *et al*., 2024; Palmer and Coutinho, 2024). The type strain of *Psi* was isolated from foxtail millet. Additional strains were isolated from pearl millet, pineapple (*Ananas comosus*), cluster bean (*Cyamopsis tetragonolobus*), onion (*Allium cepa*), rice (*Oryza sativa*), and *Psi* is also an endophyte in maize seed. The original strain of *Pss* was isolated from a maize plant displaying symptoms of vascular wilt (Stewart's wilt). *Pss* is vectored by the corn flea beetle (*C. pulicaria*) and isolates of this subspecies were also obtained from maize plants and from the insect vector.

Epidemiology

The pathogen overwinters in plant residues and in the sugarcane stubble which are the primary sources of inoculum for new crops. *P. stewartii* can also survive in irrigation water. The pathogen usually invades the host through wounds of the leaves, stems, and roots. In maize, wounds caused by sandstorms, hailstones, storms, and strong winds are all conducive to infection. Disease severity is increased in potassium-starved maize seedlings, most likely because of high nitrate/nitrogen levels within the vascular tissue. Excess nitrogen fertilization may also increase disease severity. High soil moisture, high temperatures and abundant rainfall seem to accentuate disease severity (Pepper, 1967).

Economic importance

No data regarding losses due to bacterial leaf wilt are available for sugarcane. Direct yield losses of 10–20% caused by *P. stewartii* have been reported in sweet corn (Esker and Nutter, 2003).

Control

Breeding and selecting resistant sugarcane hybrids is an effective means for disease control and prevention. Sugarcane varieties exhibit different levels of disease in the field. Eliminating secondary plant hosts and insect vectors significantly contributed to disease control in maize.

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Bacterial mottle

Barry J. Croft

Cause

Dickeya poaceiphila Hugouvieux-Cotte-Pattat et al. (2020), bacterium. Formerly, *Pectobacterium carotovorum* var. *graminarum*, *Erwinia chrysanthemi*.

Geographical distribution

Australia.

Symptoms

Bacterial mottle initially produces creamy-white regular stripes, 1–2 mm wide, from the base of leaves towards the leaf tip running parallel to the vascular bundles (Figures 1 and 2). The streaks often cease at irregular intervals along the leaf but can extend along the full length of the leaf. As the leaf ages, the streaks develop rusty red areas. The disease progresses from this initial infection into a systemic stage with varying degrees of chlorotic mottling or complete chlorosis. Chlorotic leaves develop rusty red flecks and streaks as they age. During warm, humid weather, white globules of bacterial exudate form on the underside of the leaf (Figure 3), and water that accumulates in the spindles and dewlaps of leaves turns milky with bacterial exudate (Steindl, 1964).

Infected shoots are severely stunted, and the leaves wither and die prematurely (Figure 4). Diseased plants have excessive tillering, and the tillers show the characteristic mottling. Infected stalks often produce stem galls and multiple buds. Internally, diseased stalks show brown necrotic areas around the nodes and growing points. These necrotic areas can occasionally form small cavities filled with gummy substance.

Diagnosis

Dickeya poaceiphila is easily isolated on a range of simple nutrient agar media such as Wilbrink's medium (sucrose 2%, peptone 0.5%, K_2HPO_4 0.05%, $MgSO_4$ 0.025%, agar 1.5–2%). Colonies form in one-two days and are white, opaque, slightly raised, glistening and butyrous, tending to turn grey on ageing. Bacterial cells are 0.4–0.6 × 1.5–3.0 µm with 4–6 peritrichous flagella. The identity of the bacterium can be confirmed by comparing DNA sequences from the bacterium to those published by Hugouvieux-Cotte-Pattat *et al.* (2020).

The early stages of disease development could be confused with downy mildew, and the chlorosis and wilting could be confused with leaf scald but the mottling is characteristic. The highly motile and relatively large bacterial cells can be seen oozing from the infected tissue when a small piece of tissue is placed in water on a microscope slide and examined under a high-power microscope (x400–1,000 magnification).



Figure 1. Creamy-white leaf stripes (B.J. Croft).



Figure 2. Chlorotic leaf mottling (P. Rott).



Figure 3. White globules of bacterial exudate on the underside of the leaf (B.J. Croft).



Figure 4. Severely stunted shoots with withered leaves (B.J. Croft).

Strains of the pathogen

Dowson and Hayward (1960) suggested strains that infect grasses may be distinct from those that infect sugarcane. Hugouvieux-Cotte-Pattat *et al*. (2020) found some differences in gene sequences of *gap A* and 16S rRNA genes between an isolate from sugarcane and an isolate from Guinea grass but further studies would be required to determine if these differences justify designating them as distinct strains.

Transmission

Bacterial mottle is associated with fields that have been flooded. The bacteria ooze from stomata and are carried to other plants in flood water. The bacteria possibly enter the plant through wounds caused by the flood water. Field observations suggest that wind-blown rain is of less importance in spread of the disease. Most infection takes place during warmer months which coincide with the wet season.

The bacteria have been artificially transmitted by vacuum infiltration into the cut end of cane setts and by injecting a suspension into the spindle leaves just above the growing point. Injecting bacteria into mature leaves caused a local lesion but did not produce systemic infections.

Infected cane stalk cuttings will produce infected plants but these are not important in disease spread as infected cuttings often fail to germinate or die soon after germination.

Host range

Bacterial mottle is common in Guinea grass (*Megathyrsus maximus* syn. *Panicum maximum*), para grass (*Brachiaria mutica*) and elephant/Napier grass (*Cenchrus purpureus* syn. *Pennisetum purpureum*) in fields near to and far away from sugarcane in Queensland. Symptoms in these grasses are similar to those described for sugarcane.

Systemic infections have been produced in a range of grasses, including maize and sorghum, by artificial inoculation but not in dicotyledonous plants.

Epidemiology

Flooding is the main factor associated with bacterial mottle. The disease is more prevalent in years of heavy flooding. Usually, the disease only affects a small number of plants in a field and does not spread rapidly.

Economic importance

Bacterial mottle does not cause significant yield losses because of the restricted spread of the disease.

Control

No active control measures are practised for bacterial mottle. Some varieties are resistant to the disease. Destruction of infected plants and grasses around sugarcane fields could limit spread but these practices have not been justified.

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False red stripe

Eder A. Giglioti, Sizuo Matsuoka,
revised by Philippe Rott and Sizuo Matsuoka

Cause

Xanthomonas sp., bacterium.

No significant differences were found between the pathogens causing false red stripe (FRS) and gumming disease (caused by *Xanthomonas axonopodis* pv. *vasculorum*) in cultural, biochemical, physiological and serological traits (Carvalho, 1991; Giglioti *et al.*, 1999). For this reason, both diseases were thought to be caused by the same organism (Almeida, 1994; Almeida *et al.*, 1989; Carvalho, 1991). However, only 35% genetic similarity of DNA restriction patterns was found between the two pathogenic bacteria. Additionally, the genetic profiles obtained for isolates of the FRS pathogen did not resemble those of other *Xanthomonas* species attacking sugarcane and other Poaceae (Giglioti *et al.*, 1999). The FRS symptoms are also different from those of the other sugarcane diseases caused by xanthomonads. Consequently, the FRS bacterium appears to be a unique pathogen whose taxonomic position remains to be investigated.

Geographical distribution

Brazil. Possibly also Argentina (Monachesi *et al.*, 2024).

Symptoms

FRS is characterized by the presence of narrow stripes (1 mm) parallel to the leaf midrib (Figures 1 and 2). These stripes progressively extend from the tip towards the middle of the leaf and have frequently a mixture of reddish and yellowish colors (Figures 2 and 3). The stripes can reach the base of the leaf but have not been observed on the leaf sheath. The apical meristem is not affected, and top rot symptoms have not been associated with FRS. When viewed against sunlight, the stripes appear partially translucent, and the yellow color becomes more evident. In highly susceptible sugarcane varieties, the stripes frequently coalesce and are several millimeters wide (Figures 3 and 4). They have a more intense and predominant reddish color. Whitish flecks corresponding to bacterial exudates can be seen on the leaf surface of severely affected leaves (Figure 1). Shortening of the stalk internodes has also been found in highly susceptible varieties. In contrast, fewer stripes, usually only one or two, are observed on the leaves of resistant varieties. Medium aged and adult plants appear to be more susceptible to the disease and symptoms are more severe in older leaves.

For many years, FRS was mistakenly identified as red stripe disease caused by *Acidovorax avenae* subsp. *avenae* (Figure 5). Variation in varietal response was thought to be responsible for the discrepancy with the characteristic red stripe symptoms. This misjudgment explains the name 'false red stripe' given to the disease (CCA/UFSCar, 1997;



Figure 1. Brownish leaf stripes parallel to the midrib. The whitish round flecks on the leaf surface are bacterial exudates (S. Matsuoka).

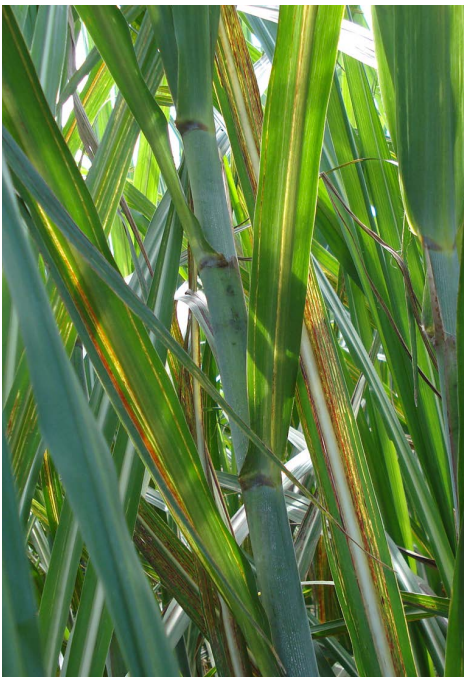

Figure 2. Leaves with stripes of various length on the blade (R. Chapola).



Figure 3. Coalescing leaf stripes on a sugarcane leaf (R. Chapola).



Figure 4. Sugarcane plant with foliage severely affected by false red stripe (R. Chapola).

Giglioti *et al*., 1998 and 1999). Differences in symptoms between FRS and red stripe can contribute to correct identification of each of these two diseases. In contrast to red stripe, the leaf lesions of FRS start near the leaf tips and progress towards the base of the leaf. FRS is mostly seen in mature plants whereas red stripe affects young leaves of young and less mature plants. FRS does not exhibit a top rot phase that is sometimes prevalent for red stripe. Plants affected by red stripe tend to be distributed in clusters and disease incidence may vary from 15–50% in intermediate and susceptible varieties when conditions are conducive for disease occurrence. In contrast, in a field affected by FRS, all plants generally show at least one leaf stripe.

Diagnosis

First diagnosis can be based on distinctive symptoms before use of serological assays such as dot-blot and ELISA. Serological variability was observed among strains of the pathogen and this must be taken into consideration for serological testing. Isolation is a useful approach because the pathogen grows readily on relatively simple culture media. The medium most frequently used is a modification of the DYGS medium that contains dextrose (2 g), peptone (1.5 g), yeast extract (2 g), K_2HPO_4 (0.50 g), $MgSO_47H_2O$ (0.50 g), glutamic acid (1.5 g), agar (18 g), distilled water (1 L) as described by Rodrigues Neto *et al*. (1986). Isolates can be readily obtained from leaf streaks. Confluent growth at 28°C on modified DYGS medium becomes visible after 1.5 days, and individual colonies appear after 3–4 days. Colonies are bright-lemon yellow, circular with entire margins, convex and viscous. Upon ageing, the bacteria cause a slight darkening of the medium. In addition to colony color and growth rate, serological micro-precipitation assays using microscope glass slides can be performed to distinguish rapidly the FRS pathogen from other yellow contaminant colonies that grow on the isolation medium.



Figure 5. Leaf with symptoms of false red stripe (on the left) and leaf with symptoms of red stripe (on the right) (S. Matsuoka).

Strains of the pathogen

Differences in pathogenicity have not been observed among isolates of the pathogen obtained from different sugarcane varieties, during different years or from different locations (Giglioti *et al.*, 1999).

Transmission

The natural modes of transmission of the FRS bacterium are so far unknown. However, spread by aerial means is supported by the generalized distribution of diseased plants and the greater severity of symptoms at the edges of sugarcane fields. Because stripes usually start from the leaf tip, plant infection most likely occurs through small lesions caused by contact of the leaf edges. The FRS bacterium can easily be transmitted experimentally by injection into spindle leaves (5–10 cm above the apical growing point) and by leaf trimming with contaminated shears (Giglioti *et al.*, 1999). Spread of the pathogen through infected cuttings is unknown.

Host range

The FRS pathogen has only been isolated from sugarcane. However, the bacterium can survive and incite symptoms in maize, oat and sorghum following artificial inoculation. These plants might possibly serve as alternative sources of inoculum.

Epidemiology

Besides symptomatology, epidemiological characteristics are important features that can be used to differentiate FRS from red stripe (Giglioti *et al.*, 1996a and 1996b). Disease distribution in the field follows a widespread infection pattern with each plant showing at least one striped leaf. Usually, disease severity is highest at the edges of the fields. This may be due to the stronger movement of air creating wounds by leaf contact.

The disease is favored by high humidity and soil fertility that are common features in sugarcane fields in lowland areas and in river valleys of Brazil. Symptoms become more intense in the spring and summer because of rain and higher temperatures. During these periods, most of the sugarcane plants are 8 to 12-month-old and they appear to be more predisposed to the disease. Remission of symptoms has been observed in symptomatic plants in autumn.

Economic importance

Reduced functional leaf area might reduce yields in susceptible sugarcane varieties. A high frequency (22–23%) of varieties that are moderately or highly susceptible to FRS have been recorded in the middle stages of selection in breeding programs in Brazil.

Control

Use of resistant varieties is the most effective means of control. The planting of susceptible varieties should be avoided in locations with high relative humidity, fertility and mild temperatures that favor the disease. Routine evaluations and/or tests are necessary to screen sugarcane clones for FRS resistance and thus to increase the frequency of resistant varieties in variety improvement programs.

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Grassy shoot/White leaf

Govind Pratap Rao, Rasappa Viswanathan

This chapter has been updated and rewritten based on the original chapters entitled "Grassy shoot" and "White leaf" authored by Rasappa Viswanathan, and C.T. Chen and Anusorn Kusalwong, respectively, in 2000.

Cause

Candidatus Phytoplasma sacchari, mollicute.

Geographical distribution

Bangladesh, China, India, Iran, Japan, Laos, Malaysia, Myanmar, Nepal, Pakistan, Sri Lanka, Sudan, Taiwan, Thailand, Vietnam.

Symptoms

The characteristic symptoms of the disease include chlorotic-white, creamy or green leaves and premature proliferation of lateral buds forming numerous lanky tillers with severe stunting (Figures 1 to 6). This phenotype gives a typical grassy appearance to the sugarcane clumps, hence the name "sugarcane grassy shoot" or SCGS given to this disease. SCGS is alternatively known as "sugarcane white leaf" or SCWL because infected plant shows partial chlorotic or complete white leaves, especially the leaves adjacent to the central leaf whorl. Young (2–3-week-old) sugarcane plants exhibit proliferative growth of axillary tillers, appear yellowish or whitish, and usually die after 3–4 months of growth. Comparatively, older (6–20-week-old) plants produce excessive tillers, short internodes, premature proliferation of axillary buds, and few or no millable stalks, thus exhibiting a typical grassy appearance of shoots with severe stunting. These symptoms occur at all growth stages during germination, tillering, formative, grand growth and maturity phases of the crop (Viswanathan and Rao, 2011; Rao *et al*., 2012; Wongkaew, 2012).

The symptoms of SCGS/SCWL are frequently masked at low temperatures, but reappear as temperatures rise, with chlorosis of the spindle leaves. Death of the diseased stalks regardless of size in the field is observed in plant crops and even more in ratoon crops during hot and dry seasons. The incidence of disease symptoms is always high in ratoon crops.

Symptoms of SCGS/SCWL show phenotypic similarity with sugarcane plants affected by iron deficiency. This can result in confusion in differentiating symptoms of SCGS/SCWL and nutrient (iron) deficiency. However, the phytoplasma disease causes excess tillering with axillary bud sprouting, whereas nutrient deficiency does not cause these symptoms.

Diagnosis

Usually, phytoplasma infection is detected by visual observation of chlorotic leaf and grassy (bushy) shoot symptoms of the affected clumps, but this diagnosis needs to be



Figure 1. Sugarcane grassy shoot/white leaf affected plant cane crop showing proliferative growth of axillary tillers (R. Viswanathan).



Figure 3. Sugarcane white leaf symptoms in an infected ratoon crop of sugarcane (W. Saengsai).



Figure 5. Partial chlorosis and chlorotic streaks on sugarcane leaves infected by the sugarcane white leaf phytoplasma (G.P. Rao).



Figure 2. Primary sugarcane white leaf symptoms during germination in plant cane crop (A. Sumedha Thushari).



Figure 4. Typical close-up view of white leaf and grassy shoot symptoms in severely infected sugarcane clumps showing a smaller number of fertile tillers (K. Nithya).



Figure 6. Sugarcane clump severely affected by grassy shoot in a mature sugarcane field (P. Rott).

confirmed by laboratory investigations. Dienes' stain (light microscopy) and DAPI stain (fluorescence microscopy) of infected leaf tissues can be used to reveal the presence of phytoplasmas in phloem tissues. Occurrence of pleomorphic phytoplasma-like bodies in the phloem sieve tissues can be seen by electron microscopy examination of ultra-thin sections of infected leaves (Rao *et al*., 2012; Wongkaew, 2012).

Serological techniques can also be applied to detect the phytoplasmas associated with the disease. The direct and indirect ELISA techniques are effective for the detection of the pathogen (Viswanathan, 1997). The plate-trapped antigen Enzyme-linked immunosorbent assay (PTA-ELISA), dot immunoblotting assay (DIBA), and tissue print immunoassay (TPIA) are used for SCGS/SCWL phytoplasma detection (Viswanathan, 1997; Biabani *et al*., 2009). Molecular biology techniques (DNA probes, DNA amplification by nested-PCR assays) have been developed for quick detection of the phytoplasmas in infected SCGS/SCWL tissues (Klingkong and Seemuller, 1993; Nakashima *et al*., 1994; Rao *et al*., 2014). Today, phytoplasma-specific primers are routinely used in nested-PCR assays for the rapid detection of the phytoplasmas associated with SCGS/SCWL (Rao *et al*., 2018; Nithya *et al*., 2023a and 2023b). Phylogenetic and RFLP analyses based on 16S rRNA and other multilocus gene sequences are further used to identify and classify the phytoplasma strains associated with SCGS/SCWL up to the group and subgroup levels (Rao *et al*., 2018; Nithya *et al*., 2023a). To determine the phytoplasma titre in SCGS/SCWL infected tissue, SYBR green-based qPCR can be used, and this assay can also be useful to detect phytoplasma cells in asymptomatic tissue (Manimekalai *et al*., 2011; Kirdat *et al*., 2021). Loop mediated isothermal amplification (LAMP) and recombinase polymerase amplification (RPA) assays have also been developed for detection of *Candidatus* Phytoplasma sacchari (Nithya *et al*., 2025). Recent whole-genome sequencing provides opportunities to develop additional and new diagnostic assays for specific diagnosis of the causal agent of SCGS/SCWL (Verma *et al*., 2026).

Strains of the pathogen

The genetic diversity of the SCGS/SCWL phytoplasma has been studied extensively in the last decade. These studies have been primarily based on the 16S rRNA gene and whole genome sequences. All investigated isolates of the SCGS/SCWL phytoplasma from different countries have 99–100% sequence identity (Kirdat *et al*., 2020 and 2021). The SCGS/SCWL isolates belong to "*Candidatus* P. sacchari" and are conventionally grouped under the 16SrXI/RYD phytoplasma group (Kirdat *et al*., 2021).

Transmission

Because sugarcane is a vegetatively propagated crop, the primary phytoplasma transmission occurs through infected planting material. This means of propagation further results in increasing incidence of SCGS in successive ratoon crops. The harvesting blades used during sugarcane harvest are responsible for mechanical spread of the SCGS/SCWL phytoplasma (Viswanathan and Rao, 2011). Secondary spread occurs via several species of leafhoppers in a persistent manner. The leafhoppers *Deltocephalus vulgaris* and *D. menoni* transmit the SCGS phytoplasma in India and Sri Lanka (Srivastava *et al*., 2006; Senavirathne, 2008). *Matsumuratettix hiroglyphicus* (Matsumura) and *Yamatotettix flavovittatus* (Deltocephalinae) are reported as the natural potential vectors of the SCWL phytoplasma in Thailand, Sri Lanka and Vietnam. The phytoplasmas are transmitted transovarially to the new insect generations (Hanboonsong *et al*., 2002 and 2006; Hoat *et al*., 2012). Three additional species of leafhoppers, namely *Cofana unimaculata* and

Maiestas portico from India (Tiwari *et al*., 2017) and *Tettigoniella viridis* from the Yunnan province in China (Li *et al*., 2022) are also reported as natural vectors of the SCGS/SCWL phytoplasma from sugarcane to sugarcane.

Host range

Besides modern sugarcane hybrids, the SCGS/SCWL causal agent can infect the *Saccharum officinarum* clone Otaheite, *S. robustum* clone 28NG251 and *S. edule* clone Talur. *S. officinarum* clone Badila, *S. robustum* clone 28NG219 and *S. sinense* are resistant to the disease (Chen, 1983). Apart from sugarcane, the SCGS/SCWL related phytoplasma strains have been found in India in commercial crops such as areca nut, coconut, rice, and wheat (Rao, 2021). However, *Cannabis sativa* and *Sorghum bicolor* are the natural hosts of the 16SrXI-B subgroup strains associated with SCGS/SCWL disease (Maurya *et al*., 2020; Nithya *et al*., 2023b).

Epidemiology

Cutting transmission plays a major role in the natural spread of the disease. Crops planted with healthy cuttings are free from the disease for several months. However, crops planted with cuttings from infected seed cane sources show high disease incidence within 2–3 months. Disease levels in the field usually increase during a crop cycle and leaving infected plants scattered throughout fields are important sources of inoculum for the spread of the pathogen. The incidence of SCGS/SCWL is also correlated with the population dynamics of the insect vector. The females of the leafhoppers *M. hiroglyphicus* and *Y. flavovittatus* usually prefer to lay their eggs in sandy soil, and this may be one of the reasons why the disease often occurs more severely in sugarcane grown in sandy soils in Thailand and Sri Lanka (Nithya *et al*., 2023a). The association of dry and warm climate with infertile sandy soil favors high disease incidence.

Economic importance

Very high yield losses due to SCGS/SCWL have been reported in numerous Asian countries, particularly when the planting material was obtained from infected sources, or when disease transmission occured during the early stages of crop growth. High infection levels resulted in significant reductions of millable cane, especially in ratoon crops. SCGS/SCWL has been reported to cause 5–70% losses in plant crops and up to 100% losses in ratoon crops (Nasare *et al*., 2007; Tiwari *et al*., 2012; Viswanathan *et al*., 2011; Yadav, 2013). SCWL caused losses of over 20 million US$ to the Thailand sugarcane industry (Wongkaew *et al*., 1997).

Control

Use of resistant varieties to control SCGS/SCWL is limited due to the lack of varieties combining high yield and disease resistance. Planting disease-free cuttings, roguing of diseased plants and the prohibition of ratooning in infected fields are recommended to control the disease (Nithya *et al*., 2023a).

Attempts to use thermotherapy to control the disease have not been completely successful. Hot air treatment (HAT) at 54°C for 8 h and a hot water treatment (HWT) at 54°C for 50 min kills the causal agent, but the germination of the buds of cuttings is also reduced (Nithya *et al*., 2023a). Aerated steam therapy (AST) was recommended

for 1 h at 50°C for inactivation of the phytoplasmas (Edison and Ramakrishnan, 1972; Viswanathan, 2001). Recent findings have shown that HWTs for 2–3 hours at 50°C or 1 h at 52°C were highly effective in the elimination of the phytoplasmas in infected cuttings. Plants grown from this heat-treated sugarcane are symptomless for up to 12 months and even longer in ratoon crops. Moist hot air treatment (MHAT) for 1.5–2 h at 54°C in 90–95% relative humidity was also effective in inactivating the SCGS/SCWL phytoplasma (Shukla and Singh, 1990).

In Thailand, different combinations of hot water and chemicals are used to manage SCGS/SCWL. This includes a HWT followed by soaking of cuttings in a cold tetracycline bath. The hot water and tetracycline treatments reduced the incidence of the SCGS/SCWL phytoplasma up to 28% as compared to the untreated control. However, the germination percentage of this planting material was reduced drastically (Kaewmanee and Hanboonsong, 2011). The neonicotinoid insecticide dinotefuran proved to be effective in controlling the vector of SCWL (*M. hiroglyphicus*) in sugarcane fields in Thailand (Hanboonsong and Kobori, 2017).

Integrated disease management through a three-tier seedcane nursery program is very effective in India. This program involves production of disease-free seedcane material using heat therapy. The program consists of three stages: (1) foundation seedcane, (2) certified seedcane and (3) commercial seedcane. The seedcane material employed for raising foundation seedcane is obtained from a nucleus seedcane with 100% genetic purity. The seedcane materials are subjected to AST or MHAT and a foundation nursery is raised. The crop raised from the foundation seedcane provides cuttings for raising certified seedcane. Good quality certified seedcane is used for commercial seedcane production. No heat treatment is given while raising certified/commercial seedcane. The field nurseries are all inspected at regular intervals and diseased clumps are eliminated to keep these nurseries disease-free. This commercial seedcane is distributed to farmers for commercial planting. This system ensures a continuous supply of disease-free planting material to farmers in a sugar factory zone, and this program is very successful in managing the disease in commercial sugarcane production.

Growing apical meristems of lateral buds from infected plants in tissue culture resulted in disease-free plantlets. The recovery rate of disease-free plantlets is higher when apices of less than 1 mm length are used (Bhansali and Singh, 1991).

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Green grassy shoot

Robert C. Magarey, Suchirat Sakuanrungsirikul

This chapter has been updated and rewritten based on the original chapter entitled "Green grassy shoot" and authored by Upsorn Pliansinchai and Siripatr Prammanee in 2000.

Cause

Phytoplasma, mollicute (Pliansinchai and Prammanee, 1995).

Geographical distribution

Thailand, Vietnam.

Symptoms

Sugarcane green grassy shoot (SCGGS) is characterized by grassy clump-like symptoms without leaf chlorosis (Figure 1). In plant crops, diseased plants produce normal tillers, but small and late profuse tillering often develops at the base of the affected stools. When the disease is severe, profuse tillering may occur two months after planting. Disease development leads to a reduced number and size of mature stalks leading to very significant yield losses. Symptom expression and severity increase in ratoon crops and plants may die. Many varieties in Thailand are susceptible to this disease (Figure 2).



Figure 1. Sugarcane stool affected by green grassy shoot in Vietnam (L. Burgess). Note the many small, green tillers associated with a few well-developed shoots within the same stool and that none of these show any chlorosis.



Figure 2. Sugarcane field severely affected by green grassy shoot at Khamphaeng Phet province, central Thailand in 2013 (S. Sakuanrungsirikul).

Diagnosis

SCGGS can be differentiated from sugarcane grassy shoot (SCGS) and sugarcane white leaf (SCWL) diseases based on the absence of leaf chlorosis in SCGGS affected plants (Rishi and Chen, 1989). In severely affected plants, prolific side shooting can be observed either at the top or the base of stalks.

Initial research into SCGGS in Thailand revealed typical phytoplasma profiles in the sieve tubes of phloem cells as seen in ultra-thin sections of infected leaves (Pliansinchai and Prammanee, 1995). No cross-reactivity with the SCGGS pathogen was found in serological assays using SCWL phytoplasma antiserum, which indicated that the two phytoplasmas have different antigens (Sarindu and Pliansinchai, 1996). Dot-blot hybridization using a specific DNA probe, PCR-RFLP and sequencing data confirmed that the SCWL and the SCGGS causal agents were different (Prammanee *et al*., 1996; Sdoodee *et al*., 1999; Wongkaew *et al*., 1997). In contrast, the phytoplasmas causing SCWL and SCGS have a close serological and genetic relationship (Sarindu and Clark, 1993; Viswanathan, 1997; Wongkaew *et al*., 1997). In a phylogenetic analysis of the three phytoplasma causal agents (SCWL, SCGS and SCGGS) using sequences of partial 16S-23S rRNA, secA and immunodominant membrane protein (Imp) genes, the SCGGS phytoplasma differed from the phytoplasmas causing SCWL and SCGS. The latter two, however, showed a close relationship (Sakuanrungsirikul *et al*., 2012, 2013, and 2021a).

Mixed infections of sugarcane yellow leaf virus (SCYLV) and sugarcane phytoplasmas may cause confusion as to the cause of field-based symptoms. However, a simple, fast and low cost, multiplex PCR with a primer set targeting the 16S rDNA phytoplasma gene is available for the simultaneous detection and differentiation of the three sugarcane phytoplasmas (Saengsai *et al*., 2025b). This multiplex assay includes three primer sets, each amplifying a DNA fragment with a specific size: primers SCWL-F and SCWL-R[5] yield an amplificon of 340 bp from the phytoplasma causing SCWL; primers SCGS-F and SCGS-R[6] produce an amplification product of 230 bp for the SCGS pathogen, and primers SCGGS-F and SCGGS-R[7] yield a 120 bp amplicon for the phytoplasma causing SCGGS.

To date, almost all phytoplasma detection approaches are nucleic acid-based molecular techniques targeting one or multiple genes (Hanboonsong *et al*., 2006; Saengsai *et al*., 2023; Sakuanrungsirikul, 2021; Sakuanrungsirikul *et al*., 2015, 2020, 2021a and 2021b). Nested-PCR stands out as a highly sensitive method for pathogen detection

5. SCWL-F: 5′-TGCCCCTTATGATCTGGGCTACAA-3′ and SCWL-R: 5′-TATTTCCTTAGAAAGGAGGTGATC-3′.
6. SCGS-F: 5′-AAGAGTAGCTGGAACGCAAGTT-3′ and SCGS-R: 5′-ACGACTTAACCCCAATCATCGAT-3′.
7. SCGGS-F: 5′-GTTCTCAGTTCGGATCGCAGTC-3′ and SCGGS-R: 5′-GCTTCGGGTGTTGCAAACTCTC-3′.

and its sensitivity is usually much higher (up to 1000 times) than standard PCR. Loop-mediated isothermal amplification (LAMP) also provides approximately 100 times higher sensitivity than standard PCR with the advantage of multiple detection sites of the primers in the target gene. The currently available LAMP assay targets gene *groEL* using outer primers F3 and B3[8], inner primers FIP and BIP[9], and loop primers LoopF and LoopR[10]. Nested-PCR can be used for the screening of plant material for tissue culture propagation while LAMP is best used for screening of planting material to establish mother plots. It is usually recommended to use standard PCR (including real-time PCR) for general disease screening.

Strains of the pathogen

No nucleotide variation was detected in the partial 16S-23S rDNA sequence (1770–1772 bp) of 10 sugarcane samples with SCGGS symptoms collected from fields in the lower northern (Sukothai), central (Chonburi, Uttraradit) and western (Prachinburi) regions in Thailand during 2009-2011. Minor nucleotide variations were found in partial *sec*A gene (275–377 bp) of these samples (Sakuanrungsirikul *et al*., 2012 and 2015). No variation in pathogenicity is known to occur within the causal agent of SCGGS.

Transmission

The SCGGS phytoplasma can be spread via infected sugarcane cuttings but not by mechanical means (Pliansinchai and Suchato, 1995). Propagation by stalk cuttings plays a major role in the spread of the pathogen but there is evidence for other transmission means (Pliansinchai *et al*., 1997). Disease incidence in a naturally infected field increased from 13.3% in plant cane crop to 56.0% in the first ratoon crop. Up to 30% infection was detected in healthy cane rows planted alternately with infected cane rows in an experimental plot. During a SCGGS outbreak in Thailand, 16 potential planthopper and leafhopper vector species were identified (Pliansinchai *et al*.,1998). Nevertheless, all investigated species tested negative for phytoplasma using a specific DNA probe. An insect vector of SCGGS is also suspected to be present in Vietnam but it has not been identified yet (Trin Xuan Hoat, Plant Protection Research Institute, Vietnam, personal communication).

Host range

There are no other reported hosts besides sugarcane (Pliansinchai *et al*.,1998).

Epidemiology

Little information is available on the patterns of disease spread. A severe epidemic occurred in Vietnam in the Quy Hop sugar factory area in the 2008-2012 period. Rapid spread into newly established cane was observed in areas severely affected by the disease. In Thailand, symptom development was associated with rapidly growing crops during the rainy season (May to September). The disease can be observed in ratoon crops soon after shoot initiation, if the source material is heavily infected.

8. F3: 5′-GCAATTGATGCAGGAGCT-3′ and B3: 5′-CATTAATAACTCCATCCTTACCT-3′.
9. FIP: 5′-TCTTGGGCGTCTACTTTTTTAGATT-TAGTAAAAGAAGGAATTGAGTTAGC-3′
and BIP: 5′-ATTCAAAATGTGGCTTCTGTTTCAT-ACTTTTTGCATCGCTTGG-3′.
10. LoopF: 5′-TCTTGGGCGTCTACTTTTTTAGATT-3′ and LoopR: 5′-ATTCAAAATGTGGCTTCTGTTTCAT-3′.

Economic importance

Yield losses caused by SCGGS can be as high as 40%, and a CCS (commercial cane sugar) reduction of 0.5–0.8 has been observed (Pliansinchai *et al*., 1995). The impact of the disease is greater in ratoon crops due to tillers failing to develop into millable stalks. Ratoon crop failure is common where disease pressure is intense. The first outbreak of SCGGS occurred around 1995 in the susceptible variety U-thong 1 in Western Thailand, and the disease spread to other adjacent regions via infected seed cane. Losses in Thailand are now lower due to the replacement of older susceptible varieties with new varieties such as Khon Kaen 3 (KK3) that originated from SCGGS-free regions since 2010. Currently, the affected areas in Thailand are confined to small areas in the west of the country (Kanchaburi) where some older susceptible varieties are grown.

In Vietnam, a severe epidemic occurred in the 2008-2012 period associated with the cropping of susceptible cultivars. Losses were excessive with many stunted crops. Crop production at Quy Hop Sugar factory (Nghe An Province) in Vietnam decreased from 1.2 million tons in 2007-2008 to 0.6 million tons in 2008-2009, and much of this decrease was due to severe SCGGS. In more recent times, the planting of variety KK3 has very significantly reduced disease incidence (Tu Ngo Van, Quy Hop Sugar Factory, personal communication), and SCGGS has assumed more minor importance.

Control

Roguing of infected plants is highly recommended for keeping plant sources disease-free. It is important that all diseased or infected plants are eradicated from intended cropping areas before establishing a new planting. Planting with disease-free material is imperative. Incidence of the disease is significantly reduced when cuttings or whole stalks are hot water treated at 50ºC for 2 h. Other treatments of 0.5 h or 1 hr at 52ºC, 1 h at 50ºC and a 4 h hot air treatment at 54ºC are less effective (Pliansinchai *et al*., 1998). Disease-free planting material obtained from tissue culture also assists with the establishment of disease-free crops.

The use of resistant varieties is the most effective means for disease control. However, limited resistance is available in recent commercial varieties. Some old commercial varieties (Alunan, B34664, Badila, Bo14, C323-68, CB47-15, Co 775, F136, My55-14, NCo387, Phil54-52, CP57-603) showed no disease under natural field infestation. An efficient method to screen for disease resistance has yet to be developed and is an obvious need. Combining resistance with high yield potential is difficult to achieve (Pliansinchai *et al*., 1998).

In Vietnam, there has been a step change in cultivars to maintain the cane production area while limiting SCGGS incidence. The main varieties currently cropped are LK92-11 and KK3, which both have origins in Thailand. LK92-11 now accounts for 70.3% of the production area while KK3 accounts for 28.9% (Tu Ngo Van, Quy Hop Sugar Factory, personal communication). Symptoms of all three phytoplasma diseases (SCWL, SCGS and SCGGS) appeared sporadically in 2022 and 2023 but their incidence has increased in 2024 (Tu Ngo Van Quy Hop Sugar Factory, personal communication).

Recently, reports on the application of bacterial antagonists in Thailand has provided a potential means for sustainably controlling phytoplasma diseases under field conditions. Hot water treatment of seed cane prior to planting in combination with the spraying of a *Bacillus subtilis* isolate at planting/during cane growth has provided effective control of phytoplasma diseases in sugarcane (Saengsai *et al*., 2024a, 2024b, 2025a).

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Gumming

A. Salem Saumtally, Asha Dookun-Saumtally

Cause

Xanthomonas axonopodis pv. *vasculorum* (Cobb 1893) Vauterin et al. 1995 and *X. vasicola* pv. *vasculorum* Studholme et al. 2020.

After its association with sugarcane gumming in 1893, the causal agent of this disease was named *Bacillus vasculorum* (Ricaud and Autrey, 1989). In 1978, after several taxonomic reclassifications, the name *X. campestris* pv. *vasculorum* was attributed to the gumming pathogen. Because strains of this pathogen were heterogeneous based on protein profiles, fatty acid analysis and DNA hybridization, they were subdivided into two groups: *X. campestris* pv. *vasculorum* type A and *X. campestris* pv. *vasculorum* type B (Vauterin *et al*., 1992). In 1995, strains of type A were renamed *X. axonopodis* pv. *vasculorum* whereas strains of type B were changed to *X. vasicola* pv. *vasculorum* (Vauterin *et al*., 1995). Although this later name was considered invalid for over two decades, because of the absence of a type strain (Young *et al*., 1996), it was only officially described in 2020 (Studholme *et al*., 2020).

Since the occurrence in 2014 of bacterial leaf streak disease of corn (maize) caused by *X. vasicola* pv. *vasculorum* in the United States, this pathovar has been generally considered in the literature as the unique causal agent of sugarcane gumming. This conclusion was drawn based of the genetic proximity of strains from maize and sugarcane and their pathogenicity in both plant species (Lang *et al*., 2017; Ortiz-Castro *et al*., 2020; Studholme *et al*., 2020). These studies did, however, not consider the full diversity of the causal agent of sugarcane gumming, especially strains belonging to the taxon *X. axonopodis* pv. *vasculorum*. Consequently, comparative molecular and pathogenicity investigations are still warranted to unravel the complex taxonomic history of the pathogen(s) causing gumming. The future studies need to include isolates from different hosts and countries, particularly from the Mascarene islands and Southern Africa where the disease has been prevalent on sugarcane.

Geographical distribution

Antigua and Barbuda, Argentina, Belize, Colombia, Cuba, Dominica, Dominican Republic, Eswatini, French Guiana, Ghana, India, Jamaica, Madagascar, Madeira, Malawi, Mauritius, Mexico, Mozambique, Panama, Puerto Rico, Réunion, St Kitts and Nevis, South Africa, Trinidad and Tobago, USA, Zimbabwe.

The disease has been considered eradicated from Australia (Ryley, 2024). Gumming was also reported in Barbados, Guadeloupe, and Martinique in the first half of the 20th century, but it has not been seen in these locations for several decades. The disease was formerly present in Brazil, Fiji, St Lucia, and St Vincent but is now reported absent in these countries (CABI Compendium, 2021). The presence of gumming in Papua New Guinea reported in the 1910-1920s was never confirmed.

Symptoms

Gumming is a vascular disease, and infection occurs when the serrated edges of the sugarcane leaves rub against each other, resulting in microscopic lesions, or when the leaves become damaged, mainly under windy conditions. There are two distinct phases of the disease: foliar and systemic. In the foliar phase, infection usually starts at the edge of the leaf and more typically towards the leaf tip, as distinct cream or yellow stripes, 4–5 mm wide, often with reddish dots (Figure 1). As the stripe elongates, the older part becomes necrotic (Figure 2). Several stripes may be present on the same leaf, but not all the leaves of a stalk show symptoms. A distinctly red foliar stripe, as opposed to yellow, has also been described (Ricaud and Autrey, 1989).



Figure 1. Necrotic and creamy stripe preceded by reddish dots on a sugarcane leaf blade (P. Rott).



Figure 2. Cream/pale yellow leaf stripes starting from the leaf tip and the leaf edge and progressing towards the base. The older part of the stripe turns necrotic (H. Mungur).



Figure 3. Very long stripes in a variety highly susceptible to gumming, extending towards the base of the leaf, which can result in systemic infection (H. Mungur).



Figure 4. Partial leaf chlorosis following the systemic phase of gumming disease (H. Mungur).



Figure 6. Death of the stalk upon infection of the growing point by the gumming pathogen (H. Mungur).



Figure 5. Yellow gum exudation from a systemic infected stalk cut transversely (H. Mungur).

In highly susceptible varieties, stripes run almost the whole length of the leaf (Figure 3), and can penetrate the leaf sheath reaching the stalk to induce the systemic phase. On new leaves of mature cane, partial chlorosis (Figure 4), similar to the chlorotic symptom produced by leaf scald (caused by *Xanthomonas albilineans*), is observed. However, chlorosis is not necessarily observed in all cases of systemic infection. Chlorosis can also be present in young ratoons after harvest, following knife transmission of the bacterium. Chlorotic leaves may revert to their normal color if conditions favorable to growth are present, although the bacterium remains latent. Stalk deformation, such as flattening of one side and bulging on the other, may also occur. Knife-cut lesions due to transverse splits in young elongating tissue may be observed externally. Internal symptoms include a reddish discoloration, particularly at the nodes, and the presence of gum pockets in nodes, internodal tissues, and the growing point. In highly infected sugarcane, gum exudation can be observed when the stalk is cut transversely, and this phenomenon is more readily observed in some varieties of *Saccharum officinarum* (Figure 5). Death of the mature stalk can occur if the growing point is affected (Figure 6).

Diagnosis

Gumming leaf stripes may not be easily distinguishable from those induced by the leaf scald pathogen when the latter is aerially transmitted. A piece of the gumming striped tissue, mounted in a drop of water on a microscope slide, releases a milky bacterial exudate visible to the naked eye after a few minutes. This feature distinguishes it from the leaf infections of leaf scald and chlorotic streak (caused by *Phytocercomonas venanatans*). The gumming bacterium is readily isolated on a sucrose peptone agar from an infected stalk or leaf stripe. For the latter, after surface sterilization, the trimmed stripe is directly streaked onto the medium. Isolation from the stalk is carried out by slicing the nodal part thinly, allowing it to diffuse in sterile distilled water and streaking a loopful on the medium. Colonies appear within 3–4 days of incubation at 28°C and are yellow, smooth, and circular, with a convex elevation. They can reach a diameter of 5–8 mm and produce abundant exopolysaccharides (xanthan gum). The bacterium is Gram-negative, rod-shaped (0.4–0.5 × 1.0–1.5 µm) with a single flagellum (Ricaud and Autrey, 1989). It can be diagnosed by serological assays such as immunofluorescence microscopy.

PCR assays and diagnostic primers to detect *X. vasicola* pv. *vasculorum* have been developed using the rich genome sequence data from the xanthomonad members (Lang *et al*., 2017). Four primer sets can be used to specifically amplify pathogen sequences from isolated DNA, heat-killed cells, or infected, macerated tissues. Methods for detection of a specific subset of *X. vasicola* pv. *vasculorum* from maize are also available (Stulberg *et al*., 2020). A loop-mediated isothermal amplification (LAMP) assay targeting the *pelL* gene, which is unique to *X. axonopodis* pv. *vasculorum* strains, has also been developed for rapid and precise diagnosis (Marabella *et al*., 2025).

Strains of the pathogen

Significant heterogeneity exists within the populations of the gumming bacterium. Strains that show a differential host reaction to sugarcane varieties have been reported, and three races were described in Mauritius in the early 1980s (MSIRI, 1983). Race 1 attacked the noble canes (*S. officinarum*) while races 2 and 3 were more prevalent on hybrid varieties. Race 3 has no longer been observed in Mauritius since the late 1990s. The races could be distinguished on Kelman's tetrazolium medium (Ricaud and Autrey, 1989), by ELISA using monoclonal antibodies (Dookun *et al*., 1996), by DNA-based techniques including restriction enzyme analysis and restriction fragment length polymorphism (RFLP) (Saumtally, 1996), and fatty acids profiling (Dookun *et al*., 2000).

Two geographical and different genetic groups (Southern Africa and Eastern Africa) of the pathogen were distinguished in Africa based on RFLP investigations (Qhobela and Claflin, 1992). Similarly, isolates of the causal agent of sugarcane gumming from different countries and hosts were also distributed in five different groups by RAPD and ERIC-PCR (Saumtally, 1996).

Strains of *X. campestris* pv. *vasculorum* from Mauritius and other countries were distributed in five different groups based on fatty acid analyses i.e., the cellular lipid composition of the bacterial cells (Dookun *et al*., 2000). Group A corresponded to strains from *Thysanolaena maxima* that were weakly pathogenic to sugarcane. Group B mainly consisted of strains belonging to race 1 isolated from sugarcane and a few strains from *T. maxima* and palm that corresponded to *X. axonopodis* pv. *vasculorum*. Group C comprised strains isolated from various countries, including South Africa, Zimbabwe, and Madagascar that were

related to *X. vasicola* pv. *vasculorum* (Vauterin *et al*., 1995). Group D was formed mainly of strains of the races 2 and 3 from Mauritius and strains from Réunion, while group E was associated with the red stripe symptoms of gumming disease.

Transmission

The gumming pathogen is transmitted by wind-blown rain, infected setts, and cutting implements. Violent winds and rains during stormy conditions facilitate its spread over wide areas. High humidity associated with warm temperatures is also favorable for disease transmission as under such conditions, the bacterium exudes from the infected leaf. Dispersal by infected setts is another important means of transmission, spreading the bacterium from one locality to another and from country to country through latent infections. Knife transmission may occur either during the preparation of cuttings or at harvest. Mechanical harvesters can efficiently spread the pathogen through the cutting action of the blade but also by the blowers disseminating infective juice, which may land over the freshly cut surfaces of the stubble.

Host range

Gumming is mainly a disease of sugarcane. Its occurrence in hosts other than sugarcane does not appear important, though the role of these hosts as reservoirs of the disease has not been clearly established. Natural hosts of the gumming pathogen include hurricane palm (*Dictyosperma album*), royal palm (*Roystonea regia*), areca nut palm (*Areca catechu*), Guatemala grass (*Tripsacum laxum*), maize (*Zea mays*), and broom bamboo (*Thysanolaena maxima,* synonym *T. latifolia)* (Peros *et al*., 1994). A host jump of the gumming pathogen from sugarcane to *Eucalyptus grandis* was reported in South Africa (Coutinho *et al*., 2015).

The causal agent of bacterial leaf streak in maize (*X. vasicola* pv. *vasculorum*) was able to infect experimentally 10 different species of the Poaceae family including barley, oat, rice, and various grasses (Longhi *et al*., 2023). Most of these plants (including sugarcane) did, however, not exhibit disease symptoms after inoculation. Furthermore, only small reddish brown leaf lesions and no systemic infections (that are typical of gumming in sugarcane) were reported after inoculation of sugarcane with *X. vasicola* pv. *vasculorum* from maize (Karamura *et al*., 2015). No natural transmission of *X. vasicola* pv. *vasculorum* between maize and sugarcane has been reported so far.

Epidemiology

Epidemics of sugarcane gumming are associated with intense windy conditions during cyclonic periods followed by a dry and cool season. The effect of the disease is more pronounced when the nearly matured plant is stressed under conditions of low temperatures and drought. The incidence of the disease gradually declines over the years if turbulent climatic conditions do not persist.

Economic importance

With the cultivation of resistant hybrid varieties, gumming disease has become less important. However, it remains potentially a serious problem under cyclonic weather conditions that favor the build-up of the pathogen's inoculum. In controlled experiments, the disease caused a decrease in the length and diameter of stalks, as well as a

reduction in cane and sugar yields. Systemic infection can result in cane losses of 45% (Autrey *et al*., 1986). Severe infections interfere also with the manufacturing of sugar as xanthan gum, abundantly produced by the pathogen in infected stalks, renders juice clarification difficult and thus hinders the recovery of sugar.

Control

The most effective method for the control of gumming is through the cultivation of resistant varieties. No completely effective therapy is available, but it is recommended to treat cuttings in hot water at 50°C for 30 min followed by 2–3 h at 50°C the next day. This treatment reduces infection in cuttings. The hot water treatment is recommended for the establishment of nurseries in conjunction with the roguing of diseased plants. Other measures required to control the disease include the elimination of alternative hosts and knife disinfection during harvest and preparation of cuttings. Disease-free plantlets can be produced by tissue culture and are recommended for the exchange of germplasm. The occurrence of strains highlights the importance of quarantine for this disease.

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Leaf scald

Philippe Rott, Michael J. Davis,
revised by Philippe Rott and Jean Heinrich Daugrois

Cause

Xanthomonas albilineans (Ashby 1929) Dowson 1943, bacterium.

Geographical distribution

Argentina, Australia, Barbados, Belize, Benin, Brazil, Burkina Faso, Burundi, Cameroon, Chad, China, Colombia, Congo, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominica, Dominican Republic, Ecuador, Eswatini (formerly Swaziland), Fiji, French Guiana, French Polynesia (Tahiti), Gabon, Ghana, Grenada, Guadeloupe, Guatemala, Guyana, Hawaii, India, Indonesia, Iraq, Jamaica, Japan, Kenya, Madagascar, Malawi, Malaysia, Martinique, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nigeria, Pakistan, Panama, Papua New Guinea, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, St Vincent, South Africa, Sri Lanka, Surinam, Tanzania, Taiwan, Thailand, Trinidad and Tobago, Uruguay, USA, Venezuela, Vietnam, Zambia, Zimbabwe.

Symptoms

There are two different forms of leaf scald symptoms, chronic and acute, and two distinct phases, latency and eclipse (Martin and Robinson, 1961; Ricaud and Ryan, 1989).

The chronic form is characterized by chlorotic streaks on leaves parallel to the main veins. These are generally white to yellow, narrow 'pencil-line' streaks (Figure 1), but they can also be several millimeters wide (Figure 2). Their color often becomes reddish with age. They are the only external symptom which develops on resistant cultivars. As the disease progresses, a necrosis of the leaf tissue around the chlorotic streaks may be observed and this extends progressively from the tip towards the base of the leaf (Figure 3). The streaks also tend to widen and become more diffuse on leaves reaching maturity. The fine central line which is characteristic of the disease can, however, always be seen in the centre of the lesion. The widening of the lines coincides with the chlorosis or bleaching of the leaf tissue (Figure 4). Chlorosis or bleaching may affect all the leaves, and this discoloration is followed by desiccation of leaf extremities which curl inwards, giving the shoot or the stalk a tapered aspect (Figure 5). The stalks can be stunted and the leaves wilted, brown and bent at the ends (Figure 6). This process, which looks like a scalding, explains the name given to the disease. A common symptom in mature cane arises from the abnormal development of side shoots on stalks, with the basal side shoots generally being more developed than those higher up, in contrast to the opposite situation commonly observed in healthy stalks (Figure 7). These diseased side shoots may show similar symptoms to those on the main stalk (Figure 8). Longitudinal sections of diseased stalks show reddening of the

vessels near the nodes and sometimes in the internodes (Figure 9). Lysigenous cavities may be observed in severely diseased canes (Kong *et al.*, 2025). In susceptible cultivars, the whole stalk may die (Figure 10).



Figure 1. Sugarcane leaf blade showing white and reddish "pencil line" streaks parallel to the midrib (P. Rott).



Figure 2. Sugarcane leaf blade with narrow red "pencil lines" streaks and wide white streaks (P. Rott).



Figure 3. Reddish "pencil line" streaks and necrosis on a sugarcane leaf infected by *Xanthomonas albilineans* (P. Rott).



Figure 4. Bleaching of sugarcane leaves affected by leaf scald (P. Rott).



Figure 5. Drying of leaves with leaf tips curling inward (P. Rott).



Figure 6. Stunted sugarcane plant with brown and wilted leaves (P. Rott).



Figure 7. Mature sugarcane stalk with numerous side shoots (P. Rott).



Figure 8. Stalk side shoots showing leaf scald symptoms (P. Rott).



Figure 9. Cut stalk showing red vascular bundles colonized by *Xanthomonas albilineans* (P. Rott).

The acute form is characterized by a sudden wilting of mature stalks, often without the earlier development of symptoms associated with the chronic form. Previously symptomless sugarcane dies as if it had been killed by drought. The acute form often occurs after a period of rain followed by a period of prolonged dry weather and is limited to highly susceptible cultivars.

Leaf scald is also characterized by a latency phase. Plants can tolerate the pathogen for several weeks or even months without exhibiting any symptoms at all, or the symptoms are so inconspicuous as to escape detection. This latency phase comes to an end for reasons which are as yet unknown, but the commonly given explanation is that of stress, particularly climatic or nutritional. An eclipse phase can occur at the same time as latency. Indeed, the chronic form is most often observed at the beginning of a ratoon or just after the appearance of shoots after planting the cuttings. A few stalks die, others survive with a few symptoms, and others seem to overcome the disease. However, the symptoms can reappear on seemingly cured stalks at harvest, or because the environmental conditions favor the disease, or on the shoots produced by cuttings sampled from these stalks.

Diagnosis

The frequent occurrence of latent infections greatly limits the usefulness of diagnosis based on visual symptoms. To overcome this problem, serological methods (immunofluorescence, ELISA, etc.) and polymerase chain reaction (PCR) procedures have been developed for detection and identification of the pathogen (Comstock and Irey, 1992; Davis *et al.*, 1998; Kong *et al.*, 2025; Pan *et al.*, 1999; Shi *et al.*, 2021) (Figure 11). Because at least three serovars of *X. albilineans* exist, serological variability of the pathogen must be taken into consideration (Rott *et al.*, 1994).

Isolation of the pathogen can also be a useful approach (Duarte Dias *et al.*, 2018). To aid in isolation, selective media were developed (Davis *et al.*, 1994). One of the media of choice consists of a modification of Wilbrink's medium (sucrose 10 g, peptone 5 g, yeast extract 5 g, K_2HPO_4 0.50 g, $MgSO_4 \cdot 7H_2O$ 0.25 g, $Na_2SO_3 \cdot 7H_2O$ 0.05 g, agar 15 g, distilled water 1 l) supplemented with 5 g/l of KBr, 50 mg/l of cycloheximide, 2 mg/l of benomyl, 25 mg/l of cephalexin, 30 mg/l of novobiocin and 50 mg/l of kasugamycin. Isolates of *X. albilineans* can be obtained from all infested parts of the sugarcane plant (leaves, stalks and roots) but the bacteria are most frequently and reliably isolated from the pencil-line leaf streaks. The leaf scald pathogen is a slow growing bacterium. Confluent growth on Wilbrink's medium becomes visible after 3 days, and minute colonies appear after 4–5 days at 28°C. Colonies are circular, convex, smooth, shining, translucent, non-mucoid, and produce a honey-yellow pigment which imparts a darker coloration upon ageing (Figure 12). As a species, *X. albilineans* can be readily distinguished from other xanthomonads (Van den Mooter and Swings, 1990). PCR products can be sequenced and blasted against the NCBI data base to confirm the identification of the pathogen[11].

Strains of the pathogen

Breakdown of varietal resistance to leaf scald has sometimes been attributed to the development or introduction of new strains but has never been unequivocally proven. Because of the latent phase, symptoms can go unseen in the field until the acute form

11. https://www.ncbi.nlm.nih.gov/datasets/taxonomy/29447/names/



Figure 10. Stalk killed by leaf scald and showing symptomatic side shoots (P. Rott).

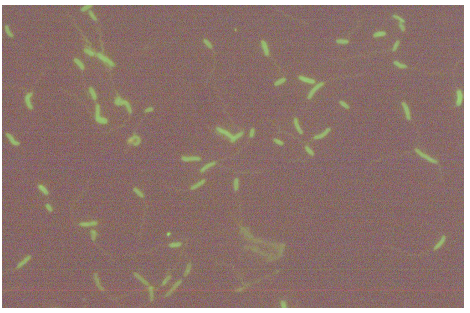

Figure 11. Cells of *Xanthomonas albilineans* observed by immunofluorescence (P. Rott).

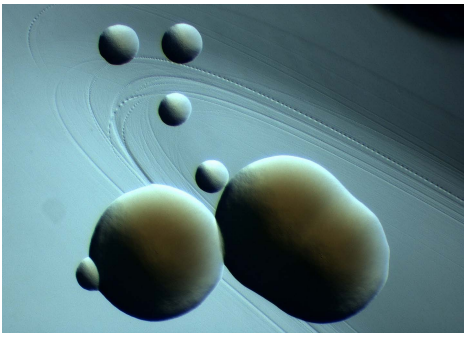

Figure 12. Small and large colonies of *Xanthomonas albilineans* growing on Wilbrink's medium (P. Champoiseau).

of the disease occurs. This may leave the impression that a sudden epidemic took place, possibly due to the appearance of a more virulent strain. However, variation in virulence among strains of *X. albilineans* is known to occur, and published data support the possible existence of different races in Mauritius (Autrey *et al.*, 1995). Several genetic variants exist among isolates of *X. albilineans*, and the outbreak of leaf scald in the 1990s in Florida was closely associated with the appearance of a genetically new strain of the pathogen (Davis *et al.*, 1997). However, no correlation between the genome organization of *X. albilineans* and its variation in pathogenicity has been established so far (Li *et al.*, 2022; Miranda *et al.*, 2023).

Transmission

The leaf scald pathogen is thought to be mainly spread by infected cuttings and transmitted by cutting implements such as knives and harvesting machines (Ricaud and Ryan, 1989). Aerial transmission of *X. albilineans* has been reported in Florida, Guadeloupe and Mauritius (Daugrois *et al.*, 2012). Leaf symptoms that develop after aerial transmission are yellow stripes turning necrotic that resemble those of gumming disease (Figure 13). The spittlebug *Mahanarva fimbriolata* (Hemiptera: Cercopidae) is an insect vector for the causal agent of sugarcane leaf scald in Brazil (Bini *et al.*, 2023).



Figure 13. Necrotic leaf stripes after aerial transmission of *Xanthomonas albilineans* (J.H. Daugrois).

Host range

Sugarcane is the main host of *X. albilineans* but maize and several grasses (*Brachiaria piligera*, *Imperata cylindrica*, *Panicum maximum*, *Paspalum* sp., *Pennisetum purpureum*, *Pennisetum* sp., *Rottboellia cochinchinensis*) have been reported as natural hosts of the pathogen (Martin and Robinson, 1961; Meng et al., 2019; Ricaud and Ryan, 1989). In particular, the bacteria can survive in *I. cylindrica* for a long period of time and, therefore, this species could be a long-term source of infection. In general, when these grasses are diseased, they exhibit only narrow leaf stripes or the more characteristic white, pencil-line symptom.

Epidemiology

Leaf scald is generally of lesser importance in areas with mild oceanic climates. It seems to be more severe in locations with continental climates and significant variations in temperature and humidity. In particular, the disease is favored by strong precipitation during cyclonic periods. Symptoms appear especially at the start of rains after a dry period. Low temperatures also increase the severity of the disease (Ricaud and Ryan, 1989). Cumulative rainfalls >1300 mm during the rainy season favor aerial transmission of *X. albilineans* in Guadeloupe (Daugrois *et al.*, 2012).

Economic importance

Leaf scald can cause large yield losses and can totally destroy plantations of susceptible cultivars within a few months or years. Spectacular drops in cane yields have been noticed in association with the acute form of the disease (Martin and Robinson, 1961; Ricaud and Ryan, 1989).

Control

Use of resistant varieties is the most effective means of control and, therefore, susceptible cultivars should be eliminated (Ricaud and Ryan, 1989).

The behavior of varieties that are only moderately susceptible can be improved by planting healthy, i.e. disease-free material, produced in nurseries. Cuttings used to plant the mother nurseries can be disinfected by a long hot water treatment: soaking of cuttings for 2 days in running water at 18–25°C and then in water at 50°C for 3 h (Steindl,

1972). Treatments at higher temperature and shorter times result in germination failure (Dayasena et al., 2024). The long hot water treatment can easily be applied in quarantine or in other situations with a limited number of selected materials. Its application on a large scale can be problematic because it requires a strategy for treatment, which among other considerations takes into account the possible reduction in germination due to treatment.

Further prophylactic measures are also required to control the disease, especially in nurseries: destruction of wild sugarcane shoots and diseased stools, disinfection of cutting tools by brushing them or soaking them in bactericides, and control of weeds. Additionally, as leaf scald is a latent disease, phytosanitary measures (quarantine) prior to introducing new plant material are indispensable.

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Leaf yellows

Asha Dookun-Saumtally, Nawshad Joomun

This chapter has been updated and rewritten based on the original chapter entitled "Yellow leaf syndrome" and authored by Ben E. Lockhart and C. Pieter R. Cronjé in 2000.

Cause

Sugarcane leaf yellows phytoplasma (SCLYP), phytoplasma.

Geographical distribution

Australia, Brazil, Cuba, Egypt, Hawaii, India, Kenya, Malawi, Mauritius, Mexico, Mozambique, Réunion, South Africa, Uganda, USA, Vietnam, Zambia, Zimbabwe.

Symptoms

Symptoms of leaf yellows are characterized by distinct yellowing of the lower surface of the midrib, gradually extending to the leaf blade that may become necrotic over the whole leaf. In some varieties, the midrib can become reddish (Figures 1 to 4). Affected plants of some varieties, especially those older than six months, can show stunted growth with leaves taking a fan-like appearance. These symptoms are often undistinguishable from those caused by sugarcane yellow leaf virus (SCYLV), the causal agent of yellow leaf of sugarcane.

When unusual leaf yellowing of sugarcane was first described in the late 1980's in Hawaii, it was referred to as yellow leaf syndrome or YLS (Schenck, 1990). A phytoplasma in YLS-affected sugarcane was first reported by Cronjé *et al*. (1998) in South Africa. Subsequently, this phytoplasma and SCYLV were associated with YLS symptoms in several sugarcane-producing locations. More severe yellowing was also reported in varieties co-infected by SCYLV and SCLYP as compared to single infections (Elsayed *et al*., 2016). Nevertheless, varieties infected by SCLYP, SCYLV or both are often symptomless.

To avoid confusion and to differentiate the diseases caused by each pathogen, two new disease names were proposed by the pathology section committee of the International Society of Sugar Cane Technologists: yellow leaf for SCYLV and leaf yellows for SCLYP (Rott *et al*., 2005).

Diagnosis

Phytoplasmas are pleomorphic, single membrane, wall-less bacteria that can cause diseases in a wide variety of plant species. They cannot be cultured, although axenic culture of plant pathogenic phytoplasmas has been reported (Contaldo *et al*., 2012).



Figure 1. Symptoms of sugarcane leaf yellows characterized by intense yellowing of the lower side of the midrib (N. Joomun).



Figure 2. Yellowing of the lower side of the midrib extending into the leaf blade (N. Joomun).



Figure 3. Sugarcane stalk with leaves showing different intensities of yellowing (N. Joomun).



Figure 4. Yellow to reddish discoloration of the midrib of leaves affected by sugarcane leaf yellows (N. Joomun).

Hence, their detection and identification are mainly based on molecular tools and DNA sequencing, while electron microscopy can be used to observe their morphological characteristics.

Detection of SCLYP in sugarcane is challenging due to low titres of the phytoplasma in the phloem tissue. Thus, detection is primarily through nested PCR, targeting the 16S rDNA and 16S-23S spacer regions, with different primer sets offering variable detection sensitivity. Restriction fragment length polymorphism (RFLP) analysis and sequencing is then used to confirm the phytoplasma group and to differentiate strains.

For nested-PCR, universal primers fP1/rP7 (Deng and Hiruki, 1991; Schneider *et al*., 1995) or P6/SN910601 (Namba *et al*., 1993) targeting the 16S rRNA gene, can be used for the first round of amplification. These primers are highly effective in detecting phytoplasmas associated with various sugarcane diseases. This first PCR step is followed by a second round of amplification targeting an internal fragment of the 16S rRNA of SCLYP using primers R16F2n/R16R2 (Lee *et al*., 1993; Gunderson and Lee, 1996). Additionally, quantitative PCR (qPCR) can be used to quantify the titre of the phytoplasma in infected plants (Christensen *et al*., 2004). However, when titres of SCLYP are very low in sugarcane, nested-PCR seems to be more effective for phytoplasma detection than a one-step qPCR (Soufi *et al*., 2013).

Strains of the pathogen

Initially, symptoms of leaf yellows of sugarcane (previously YLS) in eight African countries were associated with the Western X group phytoplasma (Cronjé *et al*., 1998). Subsequently, additional strains of phytoplasma were found in sugarcane affected with this disease: phytoplasma group III related to Western X phytoplasma in Mauritius (Aljanabi *et al*., 2001); aster yellow group phytoplasma, subgroup 16SrI-A in Cuba (Arocha *et al*., 1999); phytoplasma sub-group 16SrI-B in the São Paulo region (Silva *et al*., 2009); 16SrVII phytoplasma (Ash yellows group) in Northern Brazil (Oliveira *et al*., 2020); *Candidatus* Phytoplasma asteris in Mexico (Pérez-López *et al*., 2019); range of phytoplasmas in Australia, with the tomato big bud phytoplasma being the most prevalent (Tran-Nguyen *et al*., 2000); 16SrI group phytoplasmas in Egypt (Elsayed and Boulila, 2014); phytoplasma group 16SrXII and phytoplasma subgroup 16SrI-B in India (Gaur *et al*., 2008; Kumar *et al*., 2015).

In Hawaii, strains 16SrI (Aster yellow group) phytoplasma and 16SrXI (Rice yellow dwarf) phytoplasmas were reported in asymptomatic plants (Soufi and Komor, 2014). In this sugarcane-producing location, the 16SrI phytoplasma was prevalent for several years before being replaced by the 16SrXI phytoplasma. This latter phytoplasma was detected in commercial crops and in abandoned fields on Maui, whereas the 16SrI phytoplasma was primarily found in abandoned fields on Kauai and the Big Island of Hawaii.

Transmission

SCLYP is propagated by infected stalk cuttings. In a field, transmission rates of the phytoplasma vary according to sugarcane producing regions, largely because of variations in vector populations, climatic conditions, and range of host plants. SCLYP is primarily spread from plant to plant by insect vectors such as the leafhopper *Saccharosydne saccharivora* (Arocha *et al*., 2005). In Mauritius, *Perkinsiella saccharicida* was considered a key vector of SCLYP following the detection of phytoplasma-related DNA sequences in this delphacid planthopper (Joomun *et al*., 2007). These sequences belonged to two distinct phytoplasma groups: the 16SrIII-Western X group and the aster yellows 16SrI group.

Host range

Sugarcane is the primary host for sugarcane-associated phytoplasmas. Nevertheless, wild plant species such as weeds growing in close proximity may serve as alternative hosts, thus facilitating spread of the pathogen. In Eastern Uttar Pradesh (India), 16SrI-B group phytoplasmas were found in the common weed *Spilanthes acmella*, which may be a natural host of SCLYP (Tiwari *et al*., 2022). In Mauritius, DNA fragments related to the Western X group were amplified from the wild Sudan grass, (*Sorghum verticilliflorum*) collected from sugarcane fields (Joomun *et al*., 2007).

Epidemiology

Development and progress of leaf yellows symptoms are influenced by several factors, including sugarcane varieties, plant age, vector distribution, environmental conditions, and agricultural practices. Tropical climates favor vector populations, thus increasing the rate of transmission. In Hawaii, symptoms of leaf yellows/YLS appear in the summer months along with water stress, while in Brazil and Australia, the disease is most prevalent during cooler months. Stress conditions seem to aggravate expression of leaf yellowing. SCLYP co-exists with SCYLV in many countries, and interactions between the two pathogens most likely also play a role in disease severity.

Economic importance

Limited information is available regarding the economic importance of leaf yellows because of (i) the difficulty to compare healthy versus phytoplasma-infected plants, (ii) occurrence of a diverse array of phytoplasma strains, (iii) absence of sensitive assays to determine phytoplasma titres in the sugarcane phloem tissue, and (iv) mixed infections of SCLYP and SCYLV. Nevertheless, phytoplasma infections lead to physiological changes in sugarcane, including reduced photosynthesis, stunted growth, smaller and thinner stems, and a decrease in juice quality. These effects result in a reduction of sucrose content, lower juice yield, and ultimately, less sugar production.

Severe cases of YLS associated with SCYLV and/or SCLYP can cause up to 50% reduction in sugar yield, a substantial economic loss for growers (Rassaby *et al*., 2003; Gonçalves *et al*., 2005; Viswanathan *et al*., 2014), but these losses were not attributed directly to SCLYP.

Managing phytoplasma diseases adds to production costs due to the need for frequent monitoring, replanting with resistant varieties and treatment of cuttings before planting.

Control

Controlling leaf yellows in sugarcane requires a multi-faceted approach as single methods do not provide complete protection because of the persistent nature of phytoplasmas and their efficient vector transmission. Hence, integrated pest management (IPM) strategies that address pathogen detection, vector control, resistant varieties, and biosecurity are required. Continued investment in breeding for resistant genotypes and effective diagnostic tools are essential to keep pace with evolving phytoplasma strains. Moreover, countries importing germplasm for breeding purpose or for selection need to have in place proper biosecurity measures to mitigate introduction of different strains of phytoplasma causing leaf yellows disease.

Rigorous surveillance programs are vital for identifying early signs of phytoplasma infection and monitoring pathogen spread. Molecular diagnostic tools, such as nested-PCR and qPCR, play a key role for detection of phytoplasmas, especially in asymptomatic plants. Early detection enables prompt management interventions, which can reduce the disease impact on yields.

Control efforts also include thermotherapy with moderately high temperature of 50°C for 2 h (Soufi and Komor, 2014), as well as tissue culture techniques to ensure that clean seed is produced and planted. Soaking stalk cuttings in hot water, usually around 50°C for 1 h or longer but not exceeding 3 h, has been employed to reduce phytoplasma loads in planting material. However, this method has limitations as it may not completely eradicate phytoplasmas, which can persist at low titers after such a treatment (Soufi and Komor, 2014). Callus culture has been successful in eliminating SCLYP from 19 sugarcane varieties (Parmessur *et al*., 2002).

Insect vectors, particularly leafhoppers, are key drivers of phytoplasma spread. These vectors could be controlled through insecticide applications, but chemical control is not applied in many sugarcane growing regions. IPM using biological controls, such as natural predators and parasitoids, along with cultural practices are often preferred to minimize vector populations. Additionally, the removal of alternative host plants near sugarcane fields, like weeds that harbor phytoplasmas, can limit pathogen reservoirs.

Selecting resistant sugarcane varieties remains crucial for long-term management of leaf yellows. Additionally, strict quarantine regulations to ensure that only disease-free sugarcane materials are imported, transported, and planted in new areas are essential.

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Mottled stripe

A. Salem Saumtally

Cause

Herbaspirillum rubrisubalbicans (Christopher & Edgerton 1930) Baldani *et al*. 1996, bacterium.

Geographical distribution

Angola, Australia, Barbados, Benin, Brazil, Burkina Faso, Burundi, Central African Republic, China, Colombia, Côte d'Ivoire, Cuba, Fiji, Guadeloupe, Indonesia, Jamaica, Japan, Madagascar, Malawi, Martinique, Mauritius, New Zealand, Nicaragua, Nigeria, Panama, Peru, Puerto Rico, Réunion, South Africa, Sri Lanka, Tanzania, Togo, USA (Florida, Louisiana, Texas), Thailand, Venezuela (CABI, 2011).

Symptoms

Mottled stripe is characterized by very fine leaf stripes (streaks) that run on both sides to the midrib and commonly converge towards the base of the leaf. The color of the stripe is not consistent over the whole length, varying initially from cream (Figure 1) and becoming reddish with age (Figure 2). The red color becomes more prominent with age, and the symptoms become more conspicuous. A heavily infected leaf has a distinct reddish appearance (Figure 3). At an early stage of infection, only one or a few stripes may be present on the leaf. As the infection progresses, several stripes become conspicuous, and they tend to coalesce near the base of the leaf, forming mottled red and white bands. The stripes can also be present on the midrib and are more abundant on older leaves. The stripes vary in length, with some being short (5–10 cm) and others extending over 50 cm. The width of the stripes remains consistently narrow, usually less than 2 mm, and they maintain sharp, well-defined borders throughout their length. Symptoms can be observed in young regrowth as well as in adult plants.

Diagnosis

First diagnosis is based on disease symptoms although symptoms of mottled stripe can resemble those of red stripe caused by *Acidovorax avenae* subsp. *avenae*. However, unlike red stripe, the color of mottled stripe is not bright red throughout the stripes' length. Moreover, mottled stripe does not exhibit the bacterial exudation seen for red stripe where a pellicle is visible on the infected leaf surface, and mottled stripe does not result in top rot. Leaf stripes of mottle stripe have some resemblance to the white pencil lines observed for leaf scald caused by *Xanthomonas albilineans*, but with notable differences. The stripe in leaf scald is initially white and later turns slightly reddish, and it has a slightly broader width than that of mottled stripe. Sugarcane variety B4362 is very susceptible to the disease and can be used for testing the pathogenicity of *H. rubrisubalbicans*.



Figure 1. Initial stage of infection: fine, cream-colored stripes on one side of the midrib (A.S. Saumtally).



Figure 3. Numerous fine stripes fused on both sides of the midrib, particularly at the base of the leaf in a susceptible variety, producing bands with a reddish appearance (A.S. Saumtally).



Figure 2. Cream-colored, fine stripes becoming more reddish with a tendency to turn brick-red in part where the leaf is torn (A.S. Saumtally).

The bacterium was transferred from the genus *Pseudomonas* to the genus *Herbaspirillum* based on its habitat, DNA-rRNA and DNA-DNA hybridization data, as well as its substrate utilization (Baldani *et al*., 1996). It is Gram-negative, rod-shaped (0.8 × 2.5 µm), with 4–6 polar flagella in a lophotrichous arrangement. Colonies on sucrose peptone agar are light buff, translucent, circular, non-mucoid, 2–3 mm in diameter, and have an entire margin. Gelatin is not liquefied by *H. rubrisubalbicans* (Bradbury, 1967). In contrast to *A. avenae* subsp. *avenae*, malonate is utilized and lipid (Tween 80) is not broken down by *H. rubrisubalbicans*. The mottled stripe bacterium can be isolated from sugarcane leaves, roots, and stalks using malic acid as a carbon source formulated in a semi-solid JNFb medium that does not contain nitrogen (Matteoli *et al*., 2020). When a vial containing JNFb medium is inoculated with *H. rubrisubalbicans*, a thin white pellicle or film of bacterial cells initially grows in the centre of the medium. The pellicle then migrates to the surface and a white haze is apparent below it. After 7 days, the white pellicle thickens, and the color of the medium changes from pale green to pale blue.

Oligonucleotide probes[12] (probe HS and probe HR), based on the partial sequences of the 23S rRNA of *H. rubrisubalbicans* have been designed and used in hybridization experiments with rRNA to distinguish the latter from other species of *Herbaspirillum* (Baldani *et al*., 1996).

Species-specific PCR primers for diazotrophic plant growth-promoting bacteria, including *H. rubrisubalbicans*, were also designed (Da Silva *et al*., 2023). These primers target specific repetitive regions of the pathogen's genome. Primer pair Hr103C1870 was the most suitable for detection and quantification of the mottled stripe pathogen, producing an amplicon of 112 bp. This primer pair exhibited specificity for *H. rubrisubalbicans* in conventional and real-time PCR assays, and the bacterium could be quantified using total DNA isolated from sugarcane.

Strains of the pathogen

No strains of pathogenic significance have been reported.

Transmission

The major route of transmission of the bacterium is by wind and rain. Though present in the stalk, there has been no report on sett transmission.

Host range

Sugarcane and sorghum are the main hosts of *H. rubrisubalbicans*. In the latter host, the disease is referred to as the bacterial leaf stripe of sorghum (Hale and Wilkie, 1972).

Epidemiology

The pathogen develops under mild temperatures and humid conditions, and abundance of foliar stripes can be observed during the summer months (Steindl and Edgerton, 1964). The bacterium is considered to be an endophytic diazotroph (nitrogen-fixing) and it colonizes the roots, the stalk, the leaf sheath and predominantly the leaf blades of sugarcane in which its populations are highest (Moutia, 2018; Olivares *et al*., 1996). Populations of *H. rubrisubalbicans* decline rapidly in inoculated soil but, in the presence of sorghum, the bacteria colonize the roots and multiply within the plant tissues (Olivares *et al*., 1996).

12. Probe HS, 5′-GTC CCG GTT TTT GCA TCG A-3′ and probe HR: 5′-TAG TCG GTT TTT GCA TCG A-3′.

Economic importance

Mottled stripe is considered as a mild pathogen of sugarcane, and no economic losses have been reported. In Mauritius, the pathogen has been isolated from different varieties, M 1658/78 being the most commonly infected, without any apparent negative impact on yield. The use of *H. rubrisubalbicans* in biological nitrogen-fixation has been discussed by Döbereiner (1992 and 1997), Muthukumarasamy *et al*. (1994), and Moutia (2018).

Control

No control measure has been advocated for the disease.

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Ratoon stunt (Ratoon stunting)

Michael J. Davis, Roger A. Bailey, revised by Chuong N. Ngo

Cause

Leifsonia xyli subsp. *xyli* (Davis *et al*. 1984) Evtushenko *et al*. 2000, bacterium. Formerly known as *Clavibacter xyli* subsp. *xyli* Davis *et al*. 1984.

Geographical distribution

Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Bolivia, Brazil, Burkina Faso, Cameroon, China, Colombia, Comoros, Congo, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Djibouti, Dominican Republic, Ecuador, Egypt, El Salvador, Eswatini (former Swaziland), Ethiopia, Fiji, French Guiana, Guadeloupe, Guyana, Hawaii, India, Indonesia, Iran, Jamaica, Japan, Kenya, Madagascar, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Mozambique, Myanmar, Nicaragua, Nigeria, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, Seychelles, Somalia, South Africa, Spain, Sri Lanka, Sudan, Taiwan, Tanzania, Thailand, Trinidad and Tobago, Uganda, Uruguay, USA (Florida, Louisiana, Texas), Venezuela, Zambia, Zimbabwe.

Symptoms

Ratoon stunt also known as ratoon stunting disease (RSD) produces no reliable or characteristic external symptoms (Steindl, 1961; Gillaspie and Teakle, 1989). Stress, especially moisture stress, may enhance the disease. Yield reduction is caused by the slower growth of diseased crops with a general reduction in height and number of stalks (Figure 1). However, assessing this is difficult. The non-uniform distribution of disease in a field may have an 'up-and-down' appearance due to infected (with possibly differing levels of stunting) and uninfected stools being adjacent to one another. Yield losses can be more pronounced in ratoon crops, where diseased plants may be slower to initiate growth and production of thinner stalks more prevalent. Death of individual plants of extremely susceptible varieties may also occur when the disease is severe. Some highly susceptible varieties may show wilting under moisture stress and develop a necrosis of leaves at the tips and margins. The size of the root system of diseased cane may be reduced.

Diseased stalks of some varieties may exhibit an internal discoloration of vascular bundles at the lower portion of nodes (Figures 2 and 3). They appear as reddish-brown dots, commas, or short lines when viewed by slicing longitudinally through nodes. The discoloration does not extend into the internode unlike similar symptoms due to other diseases. Juvenile stalk symptoms may be observed in some varieties appearing as orange to reddish-orange discoloration of the vascular tissues just below the apical meristem. However, both symptoms are not reliable indicators.



Figure 1. RSD demonstration plot showing healthy cane (left) beside infected cane (right) (D. Luzuriaga).



Figure 2. Reddish discoloration of vascular bundles at the nodes of RSD infected stalk (J.-C. Girard).



Figure 3. Infected stalk with short red lines at the node (left) and healthy stalk (right) (S. Matsuoka).

Diagnosis

The lack of reliable visible symptoms makes in-field diagnosis of RSD difficult (Chakraborty *et al*., 2024). A reliable diagnosis of commercial fields where infection is often non-uniform can be problematic. Sampling from a large stalk population cannot guarantee detection of the disease, especially when disease incidence is low (Magarey *et al*., 2025). Consequently, in commercial crops, a negative diagnosis does not equate to a disease-free status of that crop. *Leifsonia xyli* subsp. *xyli* (Lxx) is a small coryneform bacterium that inhabits xylem, mesophyll and bundle sheath cells surrounding the vascular system (Quecine *et al*., 2016). Lxx populations vary among varieties and are greatest in the basal portion of mature stalks (Bailey, 1977). High Lxx populations are known to be present in vascular sap extracts and allow for potential detection using phase-contrast or dark-field microscopy (x1000) or by immunofluorescence (Figures 4 and 5). The effectiveness of microscopy examination depends largely on pathogen population size of the chosen material to sample. While positive detections can confirm disease, negative detections should not be relied on, due to the lack of sensitivity of this method.



Figure 4. RSD bacteria (Lxx) observed under an electron microscope (R.H. Lawson).

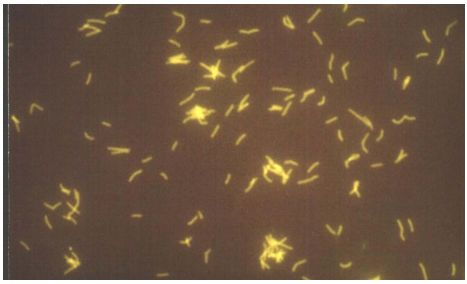

Figure 5. RSD bacteria (Lxx) observed by immunofluorescence (P. Rott).

Lxx is extremely fastidious in its nutritional requirements and can only be grown in axenic culture on special media such as the SC or S8 media (Davis *et al.*, 1980) (Figures 6 and 7), with addition of methionine (Monteiro-Vitorello *et al.*, 2004). Due to slow growth, exacting nutritional requirements and lack of a selective medium for isolation, diagnosis based on isolation of the pathogen in culture is rarely used. If isolation is required, pure cultures can be obtained more rapidly by filtering infected vascular sap through a 0.22 µm syringe filter into liquid media without antibiotics (Anthony Young, personal communication).



Figure 6. Four-week-old culture of *Leifsonia xyli* subsp. *xyli* on SC medium (R. Kanaabi).

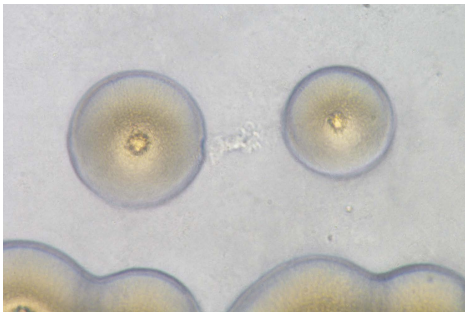

Figure 7. Two colonies (each approx. 100 µm in diameter) of *Leifsonia xyli* subsp. *xyli* on SC medium (R. Kanaabi).

The bacteria are more reliably detected using other methods. Various immunochemical tests offer a lower tech requirement compared to molecular methods. Two procedures that permit multiple samples to be analysed simultaneously are the tissue-blot enzyme immunoassay (TB-EIA) (Davis *et al.*, 1994) and the evaporative binding enzyme immunoassay (EB-EIA) (Croft *et al.*, 1994). TB-EIA enables detection and enumeration of colonised vascular bundles in stalk cross-sections. EB-EIA is a modified ELISA procedure for analysis of vascular sap extracts.

Molecular diagnostic methods offer better sensitivity and specificity. However, improved sensitivity may lead to false positive results. Due to often high Lxx levels in vascular sap samples care must be taken to avoid cross contamination during field sampling or laboratory procedures. In areas where there is a high prevalence of RSD, decontamination of equipment with sodium hypochlorite solutions should be incorporated into the field sampling protocol. Detection is possible for all molecular methods using unmodified vascular sap extracts or leaf sheath biopsies (LSB) (Young *et al.*, 2014) and offers a cost and time saving over DNA extractions. Evidence from work using the LSB

quantitative polymerase chain reaction (qPCR) test has demonstrated detection is possible 16 and 28 weeks after planting inoculated cuttings from susceptible and resistant varieties respectively, with maximal Lxx populations reached in sheaths at this age. In the following ratoon plants, populations have reached maximum levels eight weeks after harvest or earlier (Magarey *et al*., 2022). Detection from leaf tissue is possible but is not as reliable compared to other tissues mentioned.

Several iterations of polymerase chain reaction (PCR) assays based on detection of the 16S ribosomal RNA gene of the pathogen have also been developed for detection and identification (Sun *et al*., 2019). The qPCR assay requires expensive instruments but offers improved sensitivity (Grisham *et al*., 2007; Carvalho *et al*., 2016; Young *et al*., 2016). Loop-mediated isothermal amplification (LAMP) offers a more rapid result using less sophisticated and more portable equipment while giving similar or improved sensitivity over qPCR (Liu *et al*., 2013; Ghai *et al*., 2014; Wu *et al*., 2018).

Strains of the pathogen

Current data suggest there is only a single pathogenic clone of Lxx (Young *et al*., 2006).

Transmission

Lxx has been found in nature only in sugarcane, has no known insect vector(s) and is not true seed transmitted. It is mainly mechanically transmitted, through wounds, on the blades of equipment used to cultivate and harvest crops and is spread by propagation with infected cuttings including infection through shared fungicide dips. Lxx can remain viable and infectious for several months, apparently in either moribund plant debris or the soil itself (Bailey and Tough, 1992). It is unclear if Lxx can actually survive in the soil, however genome sequencing has indicated that Lxx has, or at least at one time had, the capacity to live as a free-living organism (Monteiro-Vitorello *et al*., 2004). Volunteer regrowth due to incomplete fallows of formerly infected crops provides a common source of infection of new plantings.

Host range

Sugarcane hybrids (*Saccharum* spp.) are the known natural hosts of Lxx. Other members of the *Saccharum* germplasm, including *S. barberi*, *S. edule*, *S. officinarum, S. robustum*, *S. sinense*, and *S. spontaneum*, can be naturally infected by the pathogen. Numerous grasses have also been determined to be hosts after experimental inoculation, including *Brachiaria mutica*, *Brachiaria miliiformis*, *Chloris gayana*, *Cynodon dactylon*, *Echinochloa colonum*, *Erianthus arundinaceus*, *Imperata cylindrica*, *Panicum maximum*, *Pennisetum purpureum*, *Rhynchelytrum repens, Sorghum* spp. and *Zea mays* (Gillaspie and Teakle, 1989; Roach, 1992).

Epidemiology

The rate of disease spread and extent of colonization of infected plants are directly related to varietal susceptibility. Yield reductions are sometimes greater in successive ratoon crops, possibly due to increased disease incidence or changes in the variety's physiological response. Moisture stress, from either drought or waterlogging, can increase yield reduction. A synergy between sugarcane mosaic disease and RSD can exist and greater losses are incurred when sugarcane is infected with both pathogens simultaneously than when infected by either pathogen separately.

Economic importance

RSD probably causes greater economic loss to cane sugar industries worldwide than any other disease. Yield losses have frequently been estimated at 5 to 10% overall. Losses may be negligible in some years but in others they may be 30% or greater. Losses are largely dependent on varietal susceptibility, growing conditions and disease incidence. In Florida for example, RSD yield loss was estimated at US$36.8 million for the 1988-89 crop (Dean and Davis, 1990). This estimate was based on a relatively small average yield reduction of 5% applied essentially to the entire crop in Florida. In South Africa, losses in a normal season are currently estimated at 1% of production compared with 5% in 1979, but losses in some other African cane industries are estimated to amount to 10–20% of annual production (Bailey and McFarlane, 1999). In Queensland, annual loss estimates for the study areas was A$25 million for the 2019 crop (Magarey *et al*., 2021).

Control

RSD management and control relies on planting Lxx-free seed cane, equipment sanitation and planting into fallow ground devoid of volunteers. Continued vigilance in these management practices over several years can result in a reduction in the incidence of RSD in both plantings for seed cane production and commercial crops. Breeding for resistance is also suggested as an option for assisting with control.

Thermotherapies (hot water, hot air and aerated steam) can effectively reduce the presence of Lxx in seed cane (Gillaspie and Teakle, 1989; Steindl, 1961). Hot water treatment at 50°C for 2 h is the most widely used method, with the Australian industry using 50°C for 3 h. Apart from expense, major issues limiting the effectiveness of these heat treatments are reductions in germination and lack of complete elimination. Nevertheless, thermotherapy is often used to establish 'pathogen-free' nurseries to supply planting material for commercial fields. Treatment of material with 'pathogen-free' status in successive seasons can further reduce disease incidence in nursery fields. Tissue culture programs can also assist the establishment of healthy nursery fields. Diagnostic techniques should be used for quality control monitoring.

Sanitation is important to keep healthy cane from becoming infected. Handheld cutting implements, harvesting and planting equipment can be disinfected by thorough washing and application of an appropriate disinfectant such as 5–15% Lysol (a neutralized cresylic acid solution), 1% Dettol (a mixture including pine oil and para-chlorometaxylenol), 0.1% benzalkonium chloride, 50–70% ethanol, 0.1% Mirrol or Roccal or 1% Steri-maX (solutions of quaternary ammonium compounds). If chemical dips are used, regular replacement of solutions should be performed, especially when the disease is present at significant levels in a region or when suspect cane has passed through the system. All surfaces contacting the solution should also be cleaned appropriately.

Immunity to infection is not known to occur in sugarcane. Lxx populations are known to be lower and higher in 'resistant' and 'susceptible' varieties respectively. Screening for resistance to the disease has previously been performed in Florida (Davis *et al*., 1994) and Australia (Croft *et al*., 1994). It has been suggested that control of the disease might best be obtained by disregarding the effects of the disease on variety yields and concentrating on lowering the potential for epidemics by selection of clones that are less susceptible to infection and produce less inoculum when infected (Davis *et al*., 1994). However, at present there are no known breeding programs actively trying to

produce resistant varieties. This approach has challenges while still requiring management practices (Bhuiyan *et al*., 2021). There is, however, practical value in work on generating information on levels of RSD susceptibility in existing varieties that can be used by growers to mitigate risk.

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Red streak

Heshmatolah Rahimian

Cause

Pseudomonas syringae pv. *syringae* van Hall 1902, bacterium.

Geographical distribution

Iran, Japan.

Symptoms

The initial symptoms of red streak are yellowish-red narrow lines of varying length running parallel to the leaf veins. The lesions are commonly 1–2 mm in width and range from 2 to over 200 mm in length on expanded leaves. Occasionally, longer streaks developing on the entire length of the leaves appear on some varieties. The lesions turn red to reddish-brown and may develop whitish to straw-colored centres (Figure 1). These bleached centres are quite prominent on some varieties. In severe infections, lesions coalesce, thus forming large discolored necrotic blotches resulting in death of the leaf portions distal to the affected areas. Older leaves harboring many discrete streaks tend to split longitudinally and characteristically assume a shredded appearance.

Diagnosis

The characteristic red to reddish-brown streaks, with the bleached centres, are useful for field diagnosis of red streak on most sugarcane varieties. Positive identification, however, requires isolation of *P. syringae* pv. *syringae* on appropriate culture media and proof of pathogenicity. Widespread existence of epiphytic, and frequently non-pathogenic populations of *P. syringae* pv. *syringae* on sugarcane foliage, and the absence of any other reliable method to differentiate the strains inciting red streak, makes pathogenicity testing inevitable. Isolation from suspect specimens can be made on sucrose nutrient agar (sucrose 50 g, nutrient agar 23 g, distilled water 1 l). Colonies are greyish-white, circular, smooth, convex, mucoid and measure 3–4 mm in diameter after 4 days of growth at 25–28°C. The red streak inciting strains are rather slow to produce levan, as compared to the strains causing canker and blight on stone fruits and wheat, respectively (Rahimian, 1995). Alternatively, PCR and loop-mediated isothermal amplification can be used for diagnosis of *P. syringae* pv. *syringae* (Goudarzi and Mortazavi, 2020; Guilbaud *et al*., 2016; Khezri and Mohammadi, 2018).

Strains of the pathogen

Strains of *P. syringae* pv. *syringae* isolated from red streak affected sugarcane leaves in Iran and Japan produce syringomycin (Fukuchi *et al*., 1990; Rahimian, 1995). No

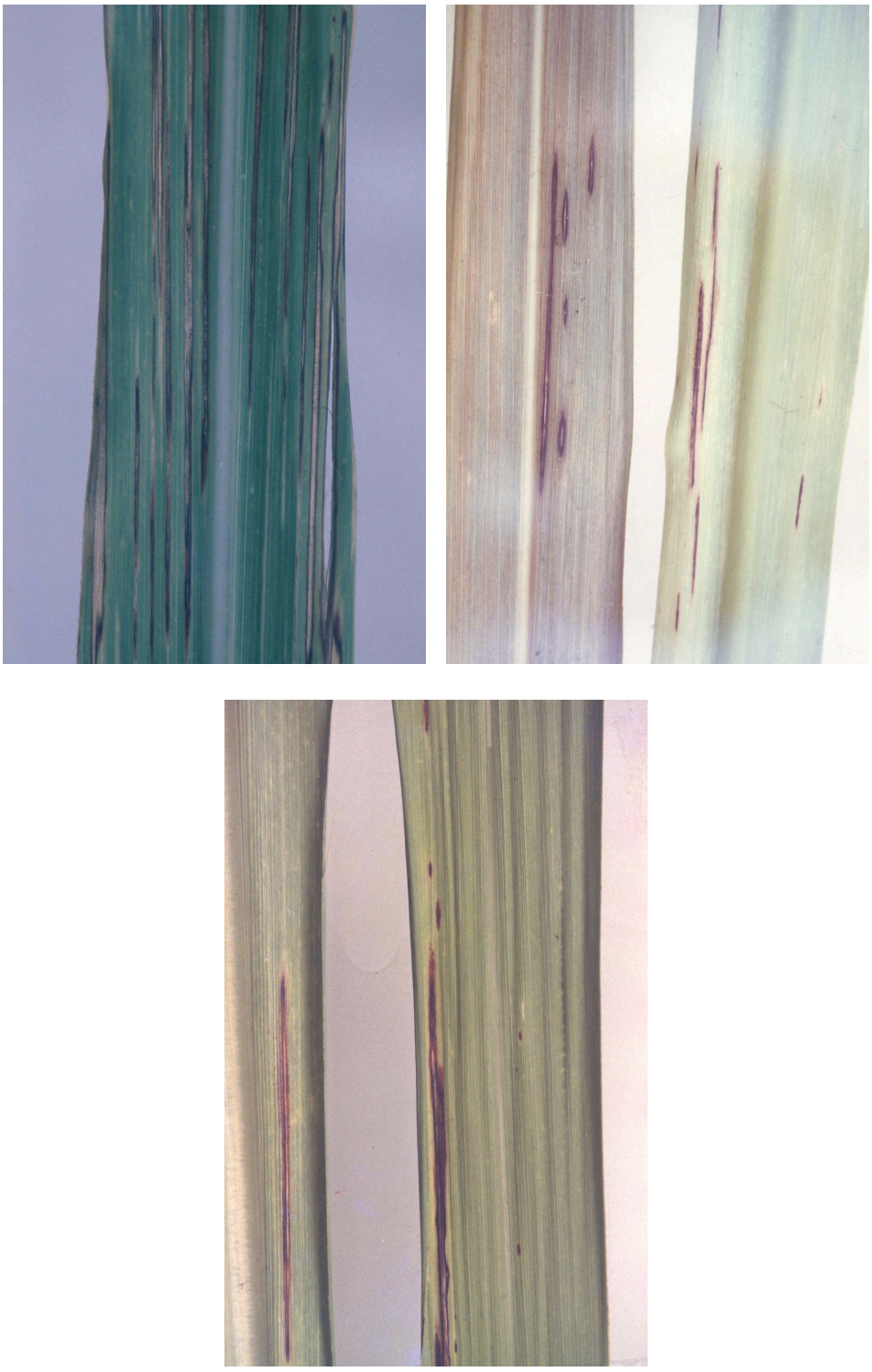

Figure 1. Leaves with reddish-brown streaks (H. Rahimian).

toxin deficient variant capable of inciting the disease in sugarcane has, thus far, been isolated. Differences in electrophoretic profiles of cell proteins and rep-PCR fingerprints occur among strains isolated from sugarcane in the same and different areas of Iran (Mousivand *et al*., 2009). Despite this variation, no noticeable difference in pathogenicity or virulence appears to exist between strains of *P. syringae* pv. *syringae* from sugarcane. The strains causing red streak form a group distinct from those inciting canker in stone fruit trees or blight in barley and wheat (Rahimian, 1995). Based on rep-PCR fingerprints and 16S DNA, ITS and *gyr*B nucleotide sequences, the sugarcane isolates are most closely related to *P. syringae* pv. *syringae* isolates causing leaf spot on *Malvaviscus penduliflorus* (Khodaygan *et al*., 2012 and 2013).

Transmission

The red streak bacterium is spread mainly by wind, splashing water and externally contaminated cuttings. The pathogen is not usually carried systemically within cuttings. Other possible transmission means have not yet been determined.

Host range

No extensive host range studies have been carried out with sugarcane strains of *P. syringae* pv. *syringae*. The pathogen has not been isolated from wheat or barley plants growing next to infected sugarcane, and it did not infect these plant species or fruit tree species under experimental conditions (Rahimian, 1995).

Epidemiology

Red streak has, thus far, been recorded in areas with humid subtropical climates. Infection occurs most frequently during cool wet springs and autumns and especially during periods of high or frequent rainfalls. The bacterium thrives as an epiphyte on several weed species, including *Cynodon dactylon*, *Phalaris minor*, *Setaria viridis*, and *Sorghum halepense*, as well as on *Zea mays* throughout the year in northern Iran (Khani *et al*., 2007). Hot and dry weather prevents disease progress and spread of the pathogen in the field as well as under glasshouse conditions.

Economic importance

Yield loss estimates due to red streak are not available.

Control

Cultivation of resistant or tolerant varieties is the most economical and promising means for the control of red streak. Reducing plant density through proper row spacing also tends to reduce the incidence and severity of the disease (Rahimian, 1995 and unpublished). Application of some bactericides, including streptomycin, has given some levels of control (Yano *et al*., 1986; Rahimian, unpublished). Use of disease-free cuttings and control of weeds that harbor epiphytic populations of the bacterium (such as *C. dactylon*, *P. minor*, *S. viridis*, and *S. halepense*) is also recommended.

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Red stripe (Top rot)

Philippe Rott, Michael J. Davis
revised by María Francisca Perera and Romina Priscila Bertani

Cause

Acidovorax avenae subsp. *avenae* (Manns 1909) Willems *et al.* 1992, bacterium.

The taxonomic position of the pathogen has been controversial. Along time, it was named *Bacterium rubrilineans*, *Phytomonas rubrilineans*, *Xanthomonas rubrilineans, Pseudomonas rubrilineans*, and *Pseudomonas avenae*. Most recently, based on morphological and biochemical characteristics, the causal agent of sugarcane red stripe was reclassified as *Acidovorax avenae* subsp. *avenae* (*Aaa*) (Willems and Gillis, 2015).

Geographical distribution

Red stripe of sugarcane was first observed in Hawaii in 1890 (Huang and Li, 2011). The disease has currently been reported in more than 60 sugarcane growing countries or locations including Argentina, Australia, Bangladesh, Barbados, Bolivia, Brazil, Cambodia, Cameroon, China, Colombia, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, El Salvador, Ethiopia, Fiji, Gabon, Guadeloupe, Guam, Guatemala, Guyana, Hawaii, Honduras, India, Indonesia, Iran, Iraq, Italy, Jamaica, Japan, Kenya, Madagascar, Malaysia, Martinique, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Nigeria, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, South Africa, Sri Lanka, Surinam, Taiwan, Tanzania, Trinidad and Tobago, Uganda, Uruguay, USA, Venezuela, Vietnam.

Symptoms

The disease is characterized by two forms: leaf stripe (Figures 1 and 2) and top rot (Figures 3 to 5). These can occur individually or simultaneously (Martin and Wismer, 1961 and 1989), mainly under relatively high atmospheric humidity (>80%) and high temperature (>28°C) (Yonzone and Devi, 2018).

Leaf stripes are often more prevalent in young, 4–6-month-old cane (seedlings and immature plants) in both plant and ratoon crops. In some countries, young ratoons are more susceptible to infection than plant canes whereas in others the opposite occurs (Yonzone and Devi, 2018).

Symptoms appear usually midway up young leaves and near the midrib, but may in some instances be concentrated toward the leaf base. They begin as water-soaked, green stripes that subsequently elongate up and down the leaf, turn reddish in color, and finally become maroon or dark red. They are 0.5 to 4 mm wide and from several centimeters long to the entire length of the leaf blade. Adjacent veins clearly delineate their borders. The stripe may extend in certain varieties into the sheath. Dried bacterial exudates may form small whitish flakes on the surface of the stripe (Figure 1).



Figure 1. Leaf showing red stripes with dried bacterial exudates (whitish flakes) (left: R.P. Bertani and right: J.-C. Girard).

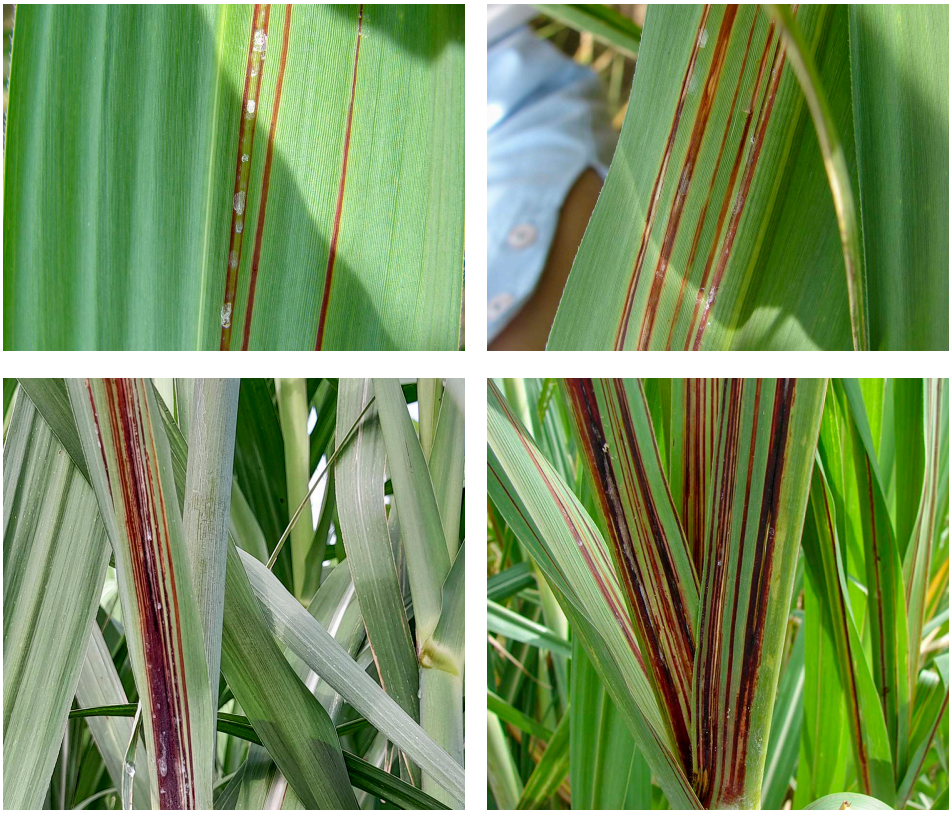

Figure 2. Varying intensity of red stripe symptoms on sugarcane leaves (R.P. Bertani).

Top rot can arise from leaf infections (Figure 3) or directly from stem and bud infection (Figure 4). The pathogen may also attack the unfolded leaves causing rotting of the leaf spindle. Spindle leaf infection may progress into the apical meristem resulting in top rot. Stem and bud infections may give rise to top rot without leaf symptoms being exhibited. Plants affected by top rot may exhibit chlorosis and wilting of the older leaves, as well as red stripe symptoms. Affected internodes may exhibit sunken lesions which are at first water-soaked in appearance and later brown to red in color. Reddish-brown discoloration may also develop within internodes and as the rotting progresses, large cavities may form. Upper internodes are usually affected (Figure 5). In the advanced stages, young spindle leaves die and are easily pulled out of the top of the stalk. The rotted spindle has a characteristic unpleasant odor, which is often discernable from the edge of the field. Side shoots sometimes develop, and their leaves may show red leaf stripes. Stalks affected by top rot are retarded in growth and usually die.

Unusual symptoms caused by *Aaa* have been described in specific varieties. In Central America and Mexico, red-colored blotches were observed on stalks of variety B4362 in the region of the root primordia (Fors, 1978). These lesions extended towards the internodes as thick red lines. In Australia, variety Trojan was affected by



Figure 3. Top rot and brown leaf stripes (R.P. Bertani).



Figure 4. Top rot of the spindle leaves (R.P. Bertani).

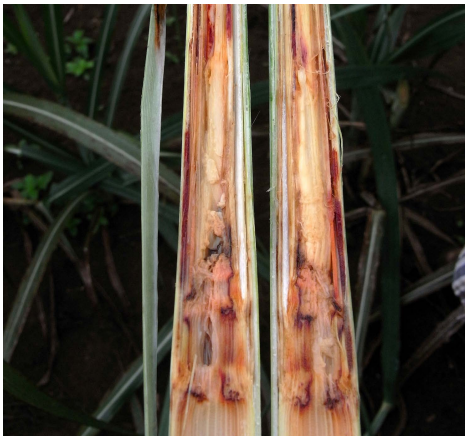

Figure 5. Longitudinally cut stalk showing internal symptoms of top rot (J.-C. Girard).

a rot that originated some distance from the growing point, and that subsequently extended in both directions (Croft *et al*., 1979). In Louisiana, a form of top rot in which the rot extended right through the stalk was reported (Edgerton, 1955, cited by Martin and Wismer, 1989).

Symptoms of reddish streaks (1–3 cm) with needle-like yellow extremities were also attributed in the early 2000s to red stripe disease of sugarcane in Japan (Figure 6). Initially considered to be caused by *Aaa*, these unusual symptoms were recently associated with a new *Acidovorax* species named *A. sacchari* (Sawada *et al.,* 2025). Further investigations are needed to determine if red stripe can indeed be caused by two different species of *Acidovorax* or if the symptoms observed in Japan should be attributed to a new disease of sugarcane.

Diagnosis

The diagnosis based on visual symptoms is the most used, but *Aaa* can be detected by PCR with specific primers in leaves without symptoms (Li *et al*., 2018).

Isolates of *Aaa* can be obtained from different infested parts of the sugarcane plant (leaves, stalks and trash). However, the bacteria are most frequently isolated from the leaf stripes, while no bacteria were isolated from root samples (Bertani *et al*., 2021).

Aaa grows on various culture media, such as the modified Wilbrink's medium used to isolate *Xanthomonas albilineans*, the causal agent of sugarcane leaf scald. It is a Gram-negative bacterium, rod shaped (0.7 × 1.6 µm), and motile with a single polar flagellum (Willems and Gillis, 2015). On yeast extract-dextrose-calcium carbonate (YDC) medium, bacterial colonies from sugarcane are convex, smooth, and 2–3 mm in diameter after 3 days at 30°C. Variation in colony color has been reported, especially when *Aaa* is grown on different agar media: off-white on potato dextrose agar (PDA) and yellow or beige or white-cream with tan to brown centers on YDC (Fontana *et al*., 2013; Schaad *et al*., 2001; Zia-ul-Hussnain *et al*., 2011); yellow-cream on yeast-peptone-glucose-agar (YPGA); white-cream (Fontana *et al*., 2013; Li *et al*., 2018; Bertani *et al*., 2021) or yellow (Zhao *et al*., 2023) on nutrient agar (NA) (Figure 7); or grayish-white (Hernández-Juárez *et al*., 2021) on Wilbrink's agar medium.

Old colonies of *Aaa* are firm and adhere to the agar. Optimum temperature for growth is 36°C (Saddler, 1994). The absence of production of fluorescent pigments on King's medium B (KB) as well as the accumulation of poly-β-hydroxybutyrate (PHB) granules intracellularly when grown on a high carbon/low nitrogen media are important features of the species. Detailed descriptions of the morphological, cultural and physiological characteristics of the pathogen have been published by several authors (Martin and Wismer, 1989; Saddler, 1994; Willems *et al*., 1992).

Aaa can also be identified by PCR assays and sequencing using specific primers targeting the 16S rDNA region (Song *et al*., 1997). Different primer pairs targeting the 16S–23S rDNA internal transcribed spacer (ITS) were designed for specific detection (Fontana *et al*., 2019; Hernández-Juárez *et al*., 2021; Klijn *et al*., 1991; Li *et al*., 2018; Shan *et al*., 2017; Zhao *et al*., 2023).

The cell size of the *A. sacchari* bacterium is 1.8 × 0.9 µm. On standard agar plates, the colonies of this new species are white/cream in color, opaque/translucent, round with entire margins, slightly convex, smooth and glistening, with a diameter of about 1 mm after 48 h of growth at 28°C. The *A. sacchari* strains are positive for oxidase activity, catalase activity, tobacco hypersensitive reaction and poly-β-hydroxybutyrate accumulation.



Figure 6. Leaf showing reddish streaks (1–3 cm) with needle-like yellow extremities in Okinawa, Japan (H. Shinohara, reproduced with the permission of the authors of Sawada et al., 2025).



Figure 7. Cultures of *Acidovorax avenae* subsp. *avenae* on nutrient agar medium: white-cream (on the left) or yellow (on the right) (M.A. Monachesi).

They are negative for fluorescent pigment production on KB medium, potato soft rot, yellow insoluble pigment production on NA and precipitate reaction in nutrient broth (Sawada *et al.*, 2025).

Strains of the pathogen

Occurrence of two colored types of *Aaa* in some infected plants in China revealed that red stripe can be caused by a mixture of strains (Zhao *et al.*, 2023). Symptom expression of sugarcane varieties can vary with the isolate of the pathogen (Almeida *et al.*, 1988). Aggressiveness in a resistant sugarcane variety also varied among *Aaa* isolates in Argentina (Bertani *et al.*, 2021). In Mexico, different strains caused a range of symptoms such as necrosis and wilting of the youngest leaves, death of inoculated leaves, and stem rot (Hernández-Juárez *et al.*, 2021). In China, two isolates of the pathogen varied in pathogenicity based on the area under the disease progress curve (Zhao *et al.*, 2023).

Five major groups of *Aaa* strains were found in China by restriction fragment length polymorphism (RFLP) analysis (Li *et al.*, 2018). At least 10 genetic groups were reported in Argentina using repetitive element polymorphism-based polymerase chain reaction (rep-PCR) (Bertani *et al.*, 2021). No relationship was found between the RFLP profiles and the geographical origin, host variety, and colony color (Zhao *et al.*, 2023). Similarly, no correlation was found between the geographical origin and the sampling year of strains of *Aaa* from Argentina using multi-locus sequence typing or MLST (Fontana *et al.*, 2019). Genetic diversity of *Aaa* identified by rep-PCR was not related either with sugarcane genotype, ratoon cycle, tissue type, field fertilization, or year of sampling (Bertani *et al.*, 2021).

Aaa strains appear to have clonal homogeneity, as well as geographical and host specificity to a certain extent (Bertani *et al*., 2021; Fontana *et al*., 2019; Yan *et al*., 2017; Zhao *et al*., 2023). It has also been hypothesized that dispersal rates of *Aaa* are too high to maintain spatially structured populations within a limited geographic distance and in a single host (Zeng *et al*., 2017). However, isolates of the pathogen from China shared high identity (>96.7%) with isolates from Argentina based on five housekeeping genes (*ugp*B, *pil*T, *lep*A, *trp*B, and *glt*A) (Zhao *et al*., 2023).

Data for strain variation in *A. sacchari* are not available as only two strains were investigated so far using nucleotide identity and digital DNA–DNA hybridization (Sawada *et al*., 2025).

Transmission

Inoculum arises mostly from bacterial exudates on the surface of leaf lesions developing during periods of moist warm weather. The bacteria are readily transmitted from plant to plant in wind-blown rain. Leaf infection is favored by injuries caused by the marginal spines of one leaf scraping another; however, leaves may also become infected through stomatal cavities on both their upper and lower surfaces. All parts of the plant can be infected through injury, especially the youngest stalk internodes and leaves.

The pathogen is rarely transmitted by mechanical equipment, cane knifes, work animals or stalk cuttings. *Aaa* is not thought to survive well in soil (Saddler, 1994). Nevertheless, the pathogen survives in affected leaves for at least one month and can colonize healthy single bud cuttings giving rise to typical symptoms of red stripe disease (Monachesi *et al*., 2023).

The disease is seed borne (fuzz) and mature plants surviving infection at the seedling stage appear to harbour latent infections (Saddler, 1994). In Argentina, no red stripe symptoms were observed when stalks exhibiting leaf stripe or top rot symptoms were used as seed cane (Monachesi *et al*., 2023). No disease symptoms were observed either in plant cane when stalks exhibiting mild and severe red stripe symptoms were used as seed cane in Louisiana. However, disease symptoms were recorded in the first and second ratoon crops (Johnson *et al*., 2016).

The red stripe bacterium can easily be transmitted experimentally by spraying inoculum on the sugarcane leaves until run-off (Bertani *et al*., 2020).

Host range

Aaa has a wide host range in the *Poaceae* family, including barley, creeping bentgrass, maize, millets, oats, rice, rye, sorghum, sugarcane and Vasey grass, but also plants such as tea and the white bird of paradise (Li *et al*., 2018).

Epidemiology

High relative humidity due to high rainfall and high temperature are generally considered ideal for red stripe development. In Queensland, variations of the top-rot stage of the disease are often related to variations in the water-holding capacity of the soil: the lower the capacity, the greater the amount of disease (Egan and Hughes, 1958, cited by Martin and Wismer, 1989). An abnormal dry spring and early summer before the wet season also favors disease development in Queensland (Martin and Wismer, 1989).

Incidence of red stripe is positively correlated with several soil properties (phosphorus, potassium, zinc, and calcium contents) and nitrogen rate, with the greatest effects in heavy soil (Johnson *et al*., 2016).

Economic importance

The disease can cause significant economic losses when susceptible varieties are grown and affected. Therefore, sugarcane varieties that are highly susceptible to the disease should be eliminated during the varietal selection process. The loss of stems and the use of diseased stalks for grinding or milling results in reduction of juice quality and yields. In Argentina, an outbreak of red stripe caused 30% yield losses (Fontana *et al*., 2013).

Top rot is the most economically damaging form of the disease. In Pakistan, cane weight was reduced by 12% and 38% at the leaf stripe and top rot phases, respectively (Ahmed *et al*., 1993). Using stalks exhibiting severe symptoms of red stripe as a seed cane source significantly decreases shoot emergence, stalk population, and subsequent cane and sugar yields. However, when stalks only exhibit the milder symptoms, shoot emergence is not affected (Johnson *et al*., 2016).

Control

Use of resistant varieties is the most effective means of control. Consequently, susceptible varieties should be eliminated in breeding programs. Top rot incidence can be reduced considerably by changing the planting date of moderately resistant varieties to avoid periods with high probability of young plant infection.

A biostimulant based on the Elicitor AsES has proven to be effective for controlling red stripe in research trials but its efficacy in commercial production remains to be determined (Chalfoun *et al*., 2018). Recently, a copper-based fungicide and bactericide (Difere®) has been registered for the control of red stripe in Brazil[13].

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Spindle rot

A.S. Patil, revised by Chandramani Raj and Rasappa Viswanathan

Cause

Reported as *Acidovorax avenae* subsp. *avenae* (Manns 1909) Willems *et al*. 1992, bacterium, but needs further clarification.

Initially, the name *Pseudomonas rubrilineans* pv. *spindulifoliens* was tentatively given to the causal agent of spindle rot by Patil and Hapase (1994), and a culture of this bacterium was deposited at the International Collection of Microorganisms from Plants (ICMP), DSIR, New Zealand under accession No. ICMP 9562. It was later given the same name as the causal agent of sugarcane red stripe when the taxonomic position of this latter pathogen was further clarified based on DNA/DNA similarities and rDNA sequencing (Fontana *et al*., 2013; Schaad *et al*., 2008; Willems *et al*., 1992).

Geographical distribution

India.

Symptoms

Spindle rot was first described as a distinct disease of sugarcane by Patil and Hapase (1994). The symptoms were primarily observed in the Maharashtra region and were reported during the early stages of the crop's growth.

The disease affects the spindle, which consists of the youngest, central unfolding leaves at the growing point of the cane. Initially, the symptoms appear as light brown to dark brown elongated lesions on the spindle leaves (Figure 1). These lesions can reach several centimeters in length and are usually seen on the uppermost, emerging leaves. As the infection progresses, parts of the affected tissue soften and begin to rot (Figure 2). At a more advanced stage, the rotting becomes more prominent, with the affected leaf tissue taking a deep brown or chocolate color. The spindle gradually withers and dries, starting from the tip and extending downward toward the base of the affected area (Figure 3). One of the key features of spindle rot is that the infection is typically confined to the youngest leaves in the spindle. These leaves often become firmly attached and cannot be pulled out easily from the growing point due to softening and internal decay (Figure 4). In contrast to red stripe caused by *Acidovorax avenae* subsp. *avenae* (*Aaa*), the rotting tissue of spindle rot does not produce any unpleasant smell (Ryan, 1989). Overall, the symptoms of spindle rot are not typical of those observed for red stripe or top rot in the Maharashtra region. Another difference between the two diseases is the absence in spindle rot of sprouting from the upper axillary buds, which is commonly observed in red stripe as a plant response to systemic stress. However, this aspect requires further investigation before drawing firm conclusions.



Figure 1. Light to dark-brown elongated streaks on the spindle leaves (R. Viswanathan).



Figure 2. Softening of spindle leaf tissue and subsequent rotting of spindle area (R. Viswanathan).



Figure 3. Drying of entire plant affected with spindle rot (R. Viswanathan).



Figure 4. Spindle rot, mainly confined to the youngest leaves that cannot be easily pulled out from the growing point, at an advanced stage (6–7 months) of the crop (R. Viswanathan).

Fusarium species have been occasionally isolated from spindle rot-affected tissues. The role of these fungi in the disease is not yet fully understood as they might be involved in a disease complex, either as a co-pathogen or contributing to symptom severity.

Diagnosis

A bacterial ooze from the cut ends of affected spindle leaves of sugarcane can be observed under the light microscope at a magnification of x100 or x400. The pathogen can be isolated on King's B medium (2% proteose peptone, 1% glycerol, 0.15%

$MgSO_4 7H_2O$, 0.15% K_2HPO_4, 2% bacto agar, pH 7.2) (King *et al*., 1954). Alternatively, modified YSMGK medium (1% yeast extract, 2% sucrose, 0.05% K_2HPO_4, 0.01% DL-methionine, 0.01% L-glutamic acid, 2% bacto agar, pH 6.8–7.0) can be used. Single colonies of *Aaa* appear within 72–96 h of growth at 30°C. They are translucent, smooth, raised with an entire margin and produce a diffuse pigmentation. The morphological, cultural and biochemical characteristics of the pathogen are similar to those described for *Aaa* (previously known as *Pseudomonas rubrilineans*; Ryan, 1989).

The bacterium is Gram-negative, aerobic, formed by rod-shaped cells measuring 1.6 × 0.7 µm, and motile with a single polar flagellum. Gelatin is liquefied by *Aaa*, and nitrate is reduced to nitrite. Acid, but not gas, is produced from glucose, fructose, glycerol and galactose. No acid is produced from lactose, sucrose, raffinose, maltose or cellobiose. The citrate test is negative and hydrolysis of starch is weak. Abundant growth occurs at 30°C on King's B agar medium within a pH range of 5.5–7.2. Growth does not occur at 40°C. Nutrient agar does not support good growth of the pathogen.

A pathogenicity test can be performed using an artificial inoculation method (Bertani *et al*., 2020; Chinea *et al*., 1978). Typical symptoms of spindle rot develop 45 days post inoculation, but only on the sugarcane spindle leaves.

Strains of the pathogen

Spindle rot has been regularly observed under tropical conditions in India. Although symptoms of this disease resemble those of red stripe, there are some clear differences suggesting variation within the pathogens causing the two diseases. Unlike classical red stripe, which shows reddish streaks on mature leaves, spindle rot only displays localized dry rot in the spindle, often with limited ooze and minimal systemic spread. These distinct symptoms suggest involvement of a particular biovar or strain of *Aaa*. Molecular and pathogenicity studies comparing the causal agents of spindle rot and red rot are needed to clarify the etiology of the spindle rot's pathogen, its variability, and role across different agroclimatic zones.

Transmission

The spindle rot pathogen is transmitted primarily by aerial means. Transmission through cuttings has not been observed.

Host range

Sugarcane appears to be the main host as no alternative hosts are known. The disease has been observed on the major commercial varieties in India such as CoC 671, Co 740, Co 7219, Co 8014 and Co 86032.

Epidemiology

Spindle rot does not occur uniformly throughout the growing season. The disease tends to be more severe in areas experiencing sharp fluctuations in temperature and humidity. However, its occurrence is typically limited to specific sugarcane varieties, particularly within the command areas of certain sugar mills. Notably, in the variety CoC 26, the disease has been observed throughout the year, suggesting a possible varietal susceptibility or adaptation of the pathogen to this host.

Economic importance

Although no economic loss has been observed, occurrence of spindle rot in several areas in India indicates that the disease could become of economic importance, most likely because of climate change.

Control

Use of resistant varieties is the most effective way to manage the disease, hence susceptible planting material should be identified and eliminated during the varietal development process. Nevertheless, rotting is a serious concern and if a severe impact is expected in the field, spindle rot can be controlled by foliar sprays of streptocycline + tetracycline (60 g/ha/500 L of water) or plantomycin (250 g/ha/500 L of water). Antibiotic applications should start two months after planting and applied at 15 day-intervals until the symptoms subside.

Supplementary information: comparative analysis of sugarcane spindle rot and red stripe

Spindle rot and red stripe have traditionally been reported as separate diseases in sugarcane, particularly across distinct agroclimatic zones of India. However, recent field observations and literature suggest that they may be different manifestations of the same disease modulated by environmental factors, varietal response, crop growth stage, and pathogen strain variability.

In Maharashtra, Patil and Hapase (1994) reported spindle rot symptoms during the early stages of the crop's growth, characterized primarily by the rotting of the spindle leaves without the presence of the classical red stripes on unfolding and unfolded leaves. It was also suggested that the disease was non-vascular in nature, localized to the spindle region and not systemically expressed in the plant. These findings led to the classification of spindle rot as a separate, non-vascular disease entity. However, Viswanathan (unpublished data) observed systemic symptoms in sugarcane stalks, including vascular discoloration (Figure 5), which suggested a more widespread infection than previously thought. This observation challenges the earlier understanding of the disease as strictly non-vascular and suggests that the pathogen may exhibit different infection strategies under varying conditions. In contrary to the early-stage specificity initially reported, spindle rot has also been seen at later stages of the crop's growth (Figure 4). This highlights the possibility that the disease is not restricted to a particular development phase, and it may exhibit systemic progress under certain conditions.



Figure 5. Transversal section of a sugarcane stalk exhibiting red vascular streaks, indicating systemic spread of the pathogen causing spindle rot (R. Viswanathan).

Both spindle rot and the classical red stripe symptoms, such as reddish longitudinal lesions on the leaf blade and sheath, have been reported in Northern India. These concurrent disease expressions support the hypothesis of systemic spread of the pathogen. The variation in symptom expression between regions may be attributed to climatic differences, such as temperature and humidity, which are known to affect disease progression and host-pathogen interactions.

Moreover, the possibility of strain variation within *Aaa* cannot be ruled out. Different strains may vary in their virulence, tissue specificity, and ability to systemically colonize the host, thereby causing distinct symptom patterns in different regions. Strain diversity could therefore be a key factor contributing to the observed variability between spindle rot and red stripe symptoms. Consequently, studies involving pathogenicity assays, strain characterization, and molecular diagnostics are needed to clarify the etiology of spindle rot and/or red stripe in India, and to develop integrated management strategies.

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Stalk bacterial soft rot

Baoshan Chen, Chengwu Zou

Cause

Dickeya zeae Samson *et al*. 2005, bacterium.

Geographical distribution

China.

Stalk bacterial soft rot of sugarcane was first discovered in Beihai City and Chongzuo City in Guangxi, China, in 2019 (Yang *et al*., 2020). The disease was sporadically found in sugarcane growing areas in Guangxi but so far not reported in any other country.

Symptoms

Stalk bacterial soft rot is a vascular disease whose symptoms are similar to those of gumming disease of sugarcane caused by *Xanthomonas axonopodis* pv. *vasculorum*. Initially, yellowing and wilting appear at the leaf tips (Figure 1). As the disease progresses, the stem tissue becomes soft and rotten, the stalk breaks easily and bacterial exudates emerge from the broken stalk (Figures 2 and 3). The affected area emits a sour smell and infected plants generally wither and die within a few days.



Figure 1. Early field symptoms of stalk bacterial soft rot of sugarcane (C. Zou).



Figure 2. Late field symptoms of stalk bacterial soft rot of sugarcane (C. Zou).



Figure 3. Rotten stalk tissue of sugarcane caused by *Dickeya zeae* (C. Zou).

Diagnosis

One obvious difference between stalk bacterial soft rot and gumming is the strong sour smell of decaying tissue infected by *D. zeae*. Furthermore, if the leaves infected by this pathogen wither, they do not exhibit red spots or stripes as it can be the case for sugarcane infected by *Xanthomonas axonopodis* pv. *vasculorum*. Rotten sugarcane stems affected by stalk bacterial soft rot are also very easily broken.

D. zeae can be identified using traditional methods of plant bacteriology, including isolation of the pathogen on culture media and determination of bacterial morphology and biochemical characteristics. After 48 hours of growth on nutrient agar medium, the colonies of strain BH9 are grayish with irregular edges and slightly raised centers (Figure 4). Bacterial cells are rod-shaped (Figure 5), about 0.8~1.0×2.0~2.8 µm in size, with peritrichous flagella. Gram staining of the bacteria is negative (Figure 6).



Figure 4. Colonies of *Dickeya zeae* strain BH9 on nutrient agar medium (Y. Yang).



Figure 5. Cell of *Dickeya zeae* strain BH9 observed by scanning electron microscopy (Y. Yang).

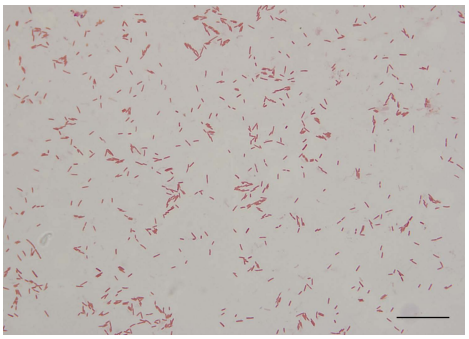

Figure 6. Gram-negative stain of *Dickeya zeae* strain BH9 from sugarcane (Y. Yang).

Molecular identification can be performed by BlastN searches and phylogenetic analyses after amplification and sequencing of various genes such as the 16S rRNA gene (with primer pair fD2/rP1), *dnaX* (with primer pair dnaXf/dnaXr), *gyrB* (with primer pair gyrBf1/gyrBr1), and *recA* (with primer pair recAf/recAr) (Zhang *et al.*, 2014).

Strains of the pathogen

Stalk bacterial soft rot of sugarcane is caused in China by strain BH9 of *D. zeae* (Yang *et al.*, 2020). The genome of this strain has a size of 4,927,334 bp and contains 4,489 predicted protein-coding genes. This is the largest genome among the sequenced strains of the *D. zeae* species. In a phylogenetic tree constructed with the concatenated sequences of three genes (*dnaX*, *gyrB* and *recA*), 32 strains of *Dickeya* were distributed in eight different clades. Strain BH9 clustered with all *D. zeae* strains in the same evolutionary group. Genetic divergence and variation in virulence occurs among strains of *D. zeae* isolated from different plants (Hu *et al.*, 2018; Zhang *et al.*, 2020). However, the degree of variation among isolates from sugarcane is unknown.

Transmission

The causal agent of sugarcane bacterial stalk rot is mainly transmitted through wind-driven rainwater, cutting tools, infected seed cane, and infected plant residues. Wind and rain associated with high temperatures are conducive to the spread of the pathogen. When sugarcane plants are mechanically damaged or weakened, they are more susceptible to bacterial infection.

Host Range

Besides sugarcane, isolates of *D. zeae* are able to infect both monocot and dicot plants, including maize (*Zea mays*; Martinez-Cisneros *et al.*, 2014), rice (*Oryza sativa*; Pu *et al.*, 2012), banana (*Musa nana* also known as *M. acuminata*; Lin *et al.*, 2010; Zhang *et al.*, 2014), clivia (*Clivia miniata*; Hu *et al.*, 2018), and potato (*Solanum tuberosum*; Toth *et al.*, 2011).

Epidemiology

The disease is prone to occur and develop under warm and humid conditions, especially in the sugarcane areas of Beihai and Chongzuo cities in the Guangxi province. These locations are characterized by continuous high temperatures after strong convective weather during summer.

Economic importance

Most sugarcane varieties currently in production have strong resistance to the disease, and bacterial stalk rot is not common in most sugarcane growing areas in China. However, some sugarcane varieties are susceptible, and therefore, the disease remains a potential problem when suitable climatic conditions prevail.

Control

The most effective method to control bacterial stalk rot is to plant resistant sugarcane varieties and pathogen-free seed cane. Other measures include strict quarantine and enhanced field management i.e., disinfection of knives during planting and harvesting, and prompt removal of diseased plants and plant residues at the early stages of the disease, with the objective to limit the spread of the disease.

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III

Diseases caused by fungi and fungus-like organisms

Australian basal stem, root and sheath rot

Barry J. Croft

Cause

Unidentified basidiomycete fungus.

Geographical distribution

Australia.

Symptoms

Australian basal stem, root and sheath rot causes a rot of the base of the leaf sheaths in young plants or less frequently in young ratoon crops (Figure 1). Unlike Marasmius basal stem, root and sheath rot, the leaf sheaths are not cemented together but can be easily pulled away from the stem (Steindl and Egan, 1964). Brown lesions are found at the base of stalks of affected plants, particularly around the root primordia and buds, but also extending into the internode region (Figure 2). The lesions are sunken, pale brown and initially water-soaked, later becoming dry and dark brown to red. The affected tissue generally extends only a few millimeters below the rind. The majority of roots are rotted and the plant can be easily pulled from the ground. The underground stems of diseased plants are enveloped in the white mycelium of the fungus.

Affected shoots are stunted with short stiff erect leaves, giving the shoots a fan-like appearance. Leaves of some varieties produce narrow white stripes along the vascular bundles (Figure 3). The stripes have regular margins, are variable in length and can turn reddish towards the base in older leaves.

Severely affected plants have few tillers and aerial roots form from undamaged nodes above ground level giving a 'buttress' effect.

Diagnosis

The fungus, which causes this disease, has not been observed to produce fruiting bodies but does produce hyphal clamp connections. The mycelium of the fungus has a distinctive mushroom and formic acid odor. Diagnosis is by symptoms and by inoculation of susceptible varieties such as Q63.

Strains of the pathogen

No information is available.



Figure 1. Diseased stalk (B.J. Croft).

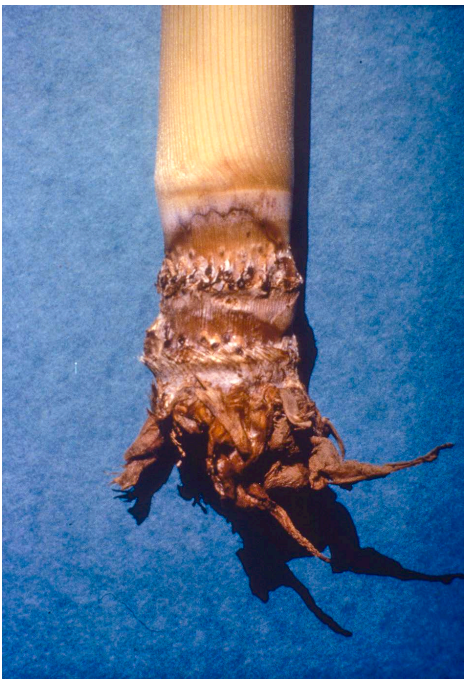

Figure 2. Brown lesions at the base of a stalk (B.J. Croft).

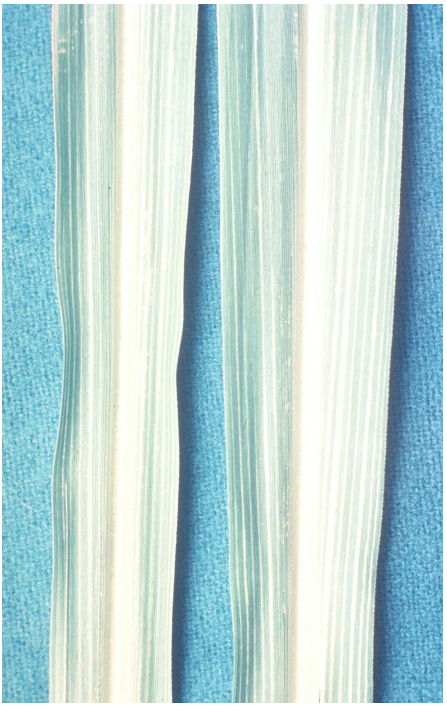

Figure 3. Narrow white leaf stripes along the vascular bundles (B.J. Croft).

Transmission

The fungus spreads in soil by mycelial strands. Movement of contaminated soil or organic matter could spread the disease.

Host range

Sugarcane is the only known host of Australian basal stem, root and sheath rot.

Epidemiology

The fungus which causes this disease can survive in soil as a saprophyte for long periods. The disease is favored by hot dry conditions and generally only affects young plant crops or less frequently young ratoon crops. Crops usually recover after rain or irrigation, especially if the soil is hilled-up around the new roots forming above the damaged areas of the stalks. The disease can kill stools, but it is uncommon for more than a few stools to be killed and most crops recover when growing conditions improve.

Economic importance

Australian basal stem, root and sheath rot is generally of minor importance but some isolated cases of severe damage can occur. On average, the disease is recorded in up to 500 ha/yr in Queensland, Australia, but incidence within fields is usually low (Magarey, 2006).

Control

The effects of the disease can be alleviated by hilling-up soil around the plants and, if possible, irrigating the crop. Varieties vary in susceptibility and susceptible varieties should be avoided in areas with a history of the disease.

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Banded sclerotial disease

Claude Ricaud, revised by Andre B. Gama

Cause

Rhizoctonia solani J.G. Kühn [Syn. *Thanatephorus sasakii* (Shirai) C.C. Tu & Kimbr. and *T. cucumeris* (A.B. Frank) Donk], basidiomycete fungus.

Geographical distribution

Algeria, Australia, Bangladesh, Burkina Faso, China, Cuba, Fiji, India, Indonesia, Japan, Madagascar, Malaysia, Nigeria, Panama, Papua New Guinea, Philippines, Puerto Rico, Samoa, Taiwan, Thailand, USA, Vietnam.

Symptoms

The disease is characterized by a series of irregularly shaped patches across the leaf blades which are confined to the older leaves (Figure 1). These bands are initially dirty green, then brownish and finally straw-colored with a well-defined reddish-brown border. Similar symptoms may occasionally occur on the sheaths (Figure 2). Pale brown fungal mycelium can often be seen on the surface of the leaf and small spherical to irregularly shaped sclerotia, 2–5 mm in diameter, beige colored when immature, but turning dark brown to black later, are frequently observed on the surface of diseased areas (Abbott and Takashi Matsumoto, 1964).



Figure 1. Irregularly shaped patches across the leaf blade (L. Putra).



Figure 2. Irregularly shaped patches across the blades and sheaths of older leaves (R. Viswanathan).

Diagnosis

Traditionally, the fungus is easily isolated from sclerotia plated directly to potato dextrose agar. Specific characteristics of the fungus have been described by Mordue (1974) and Sivanesan and Waller (1986). The pathogen can also be detected in symptomatic tissue, soil samples, and alternate hosts by PCR using primers Rs1F and Rs2R that target the ITS gene for the anastomosis group 1 (AG-1) (Sayler and Yang, 2007) and primers Rs2.1/8F and Rs2.1/8R for groups AG2-1 and AG8, also targeting the ITS region (Okubara *et al.*, 2008). For accurate diagnosis, the amplified DNA can be sequenced and pathogen identity confirmed by a BlastN search in GenBank. Real-time PCR can be used for quantification of the pathogen from soil and plant tissue samples (Okubara *et al.*, 2008).

Strains of the pathogen

Rhizoctonia solani is a cosmopolitan and complex species with variable parasitic patterns, morphological and physiological characteristics, host range and specificity. The pathogen causes disease in multiple hosts, and some biotypes of this pathogen are more severe in some crops. Fungal isolates from AG-1 are more severe in rice, while the isolates from AG-3 are more aggressive to solanaceous crops (Bernardes de Assis *et al.*, 2008; Okubara *et al.,* 2008).

Transmission

Rhizoctonia solani survives in the soil and infects host plants during contact of the leaves with the soil. Transmission also occurs by leaf contact between diseased weeds and healthy sugarcane. The pathogen is spread by windblown rain and soil particles containing the fungal spores or sclerotia.

Host range

Species within the *Rhizoctonia* genus have a wide host range, infecting plants in the Anacardiaceae, Apiaceae, Apocynaceae, Arecaceae, Betulaceae, Brassicaceae, Caryophyllaceae, Chenopodiaceae, Cucurbitaceae, Ericaceae, Fabaceae, Lauraceae, Liliaceae, Malvaceae, Myrtaceae, Pinaceae, Poaceae, Rosaceae, Rubiaceae, Rutaceae, Simaroubaceae, Solanaceae, Taxaceae, and Vitaceae families. Among cultivated species, the most important include avocado, blueberry, cabbage, carrot, cauliflower, common bean, oat, okra, pistachio, radish, soybean, and watermelon. *Rhizoctonia solani* also infects common weeds in sugarcane fields, such as Johnson grass (*Sorghum halepense*) and Bermuda grass (*Cynodon dactylon*) (Baruah and Lal, 1981; CABI, 2021).

Epidemiology

Rhizoctonia solani survives as sclerotia in the soil, on host residues and on grass weeds. High relative humidity and rainfall spreads the pathogen to sugarcane plants (Abbott and Takashi Matsumoto, 1964). The fungus can survive for long periods in different soil conditions, although survival rates may vary depending on soil temperature and water content (Feng *et al.*, 2016).

Economic importance

Banded sclerotial disease is considered a disease of minor importance. The disease is most commonly observed on leaves in the lower section of the sugarcane plants. These leaves are not as photosynthetically active as the ones located in the upper part of the plants. However, the disease may cause considerable yield losses in susceptible varieties (Singh *et al.*, 1994).

Control

No specific control measure is warranted except that fuzz seedlings with obvious susceptibility should be discarded early in a variety selection program.

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Black leaf spot (Tar spot)

Claude Ricaud, revised by Andre B. Gama

Cause

Phyllachora sacchari Henn., ascomycete fungus.

Geographical distribution

Argentina, Australia, Bangladesh, Borneo, Brunei, Cambodia, China, Hong Kong, India, Indonesia, Italy (Sicily), Japan, Malaysia, Myanmar, New Guinea, Nigeria, Pakistan, Philippines, Sierra Leone, Singapore, Solomon Islands, Taiwan, Thailand.

Symptoms

The initial symptoms appear as small, raised black spots (tar spots) on the lower leaf blades (Figure 1). Over time, necrotic areas form around these spots, which can merge, thus resulting in extensive necrotic and straw-colored regions on the leaf lamina. In severe tar spot epidemics, infections may develop on younger leaves and older leaves senesce and dry up prematurely (Koike, 1988).

Diagnosis

The fruiting bodies, perithecia, asci, and ascospores of *P. sacchari* have distinct characteristics that facilitate identification of this fungus. Stroma contain up to five perithecia measuring 90–155 × 150–900 µm. Asci are cylindrical, contain eight ascospores, are unitunicate and paraphysate, measuring 80–130 × 13–20 µm. The ascospores measure 16–25 × 8–11 µm. Additional characteristics have been described by Anahosur and

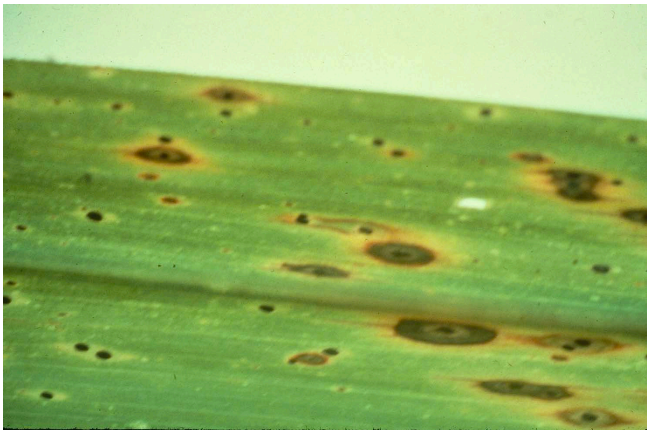

Figure 1. Leaf with tar spots and necrotic areas (H. Koike).

Sivanesan (1978). Although no diagnostic primers were specifically developed for *P. sacchari*, amplification and sequencing of the nrSSU, ITS, RPB2, and TEF1 genes can be used for species identification within the order *Phyllachorales* (Mardones *et al.*, 2017). Nevertheless, identification of the genus is easily performed based on the morphological characteristics of the fungus.

Host range and strains of the pathogen

Phyllachora sacchari or other fungal species from the same genus infect grass weeds such as *Polytoca macrophylla*, *Rottboellia exaltata*, and several *Sorghum* species (*S. almum*, *S. bicolor*, *S. halepense*, *S. sudanense*, *S. vulgare*, and *S. vulgare* var. *saccharatum*) (Anahosur and Sivanesan, 1978). Recently, severe outbreaks of tar spot caused by *P. maydis* were reported on *Zea mays* in North America, raising concerns to other host species regarding *Phyllachora* spp. as potentially aggressive pathogens (Ruhl et al., 2016).

Transmission

Ascospores present in crop debris are suspected to be a possible source of primary infection; they may be disseminated by wind or rain (Anahosur and Sivanesan, 1978).

Epidemiology

Humid tropical environments are favorable to infection.

Economic importance and control

The disease is of minor economic importance in sugarcane, not requiring the deployment of disease management strategies. However, disease monitoring, especially during breeding, is advisable.

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Brown rust

Sushma Sood, Philippe Rott

This chapter has been updated and rewritten based on the original chapter entitled "Common rust" and authored by Richard N. Raid and Jack C. Comstock in 2000.

Cause

Puccinia melanocephala Syd. & P. Syd., basidiomycete fungus (Cummins, 1971).

Geographical distribution

Angola, Antigua and Barbuda, Argentina, Australia, Barbados, Belize, Benin, Bolivia, Brazil, Burundi, Cameroon, China, Colombia, Congo, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, Eswatini, Ethiopia, Guadeloupe, Guatemala, Guyana, Haiti, Hawaii, Honduras, India, Indonesia, Jamaica, Japan, Kenya, Madagascar, Malawi, Martinique, Mauritius, Mexico, Mozambique, Nepal, Nicaragua, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, South Africa, Taiwan, Tanzania, Thailand, Trinidad and Tobago, Uganda, USA, Venezuela, Vietnam, Zambia, Zimbabwe.

Symptoms

The early symptoms of brown rust infection are small, elongate yellow flecks visible on both leaf surfaces. The flecks become reddish-brown on susceptible plants within 3–4 days, elongate, and become mature sporulating pustules (uredinia) within 10–14 days (Figures 1 and 2). Pustules of brown rust develop mainly on the lower side of the leaves (Ryan and Egan, 1989). These foliar lesions are parallel to the vascular bundles, and their size varies in length from 2–20 mm and width from 1–3 mm (Figures 3 to 5).

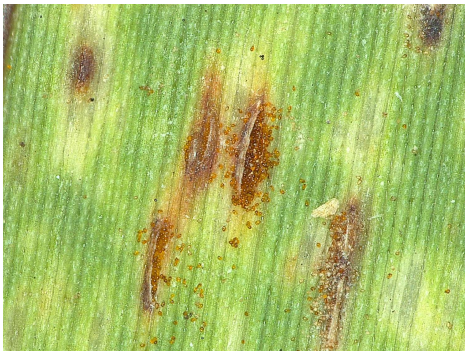

Figure 1. Mature sporulating pustules (uredinia) of *Puccinia melanocephala* (S. Sood).

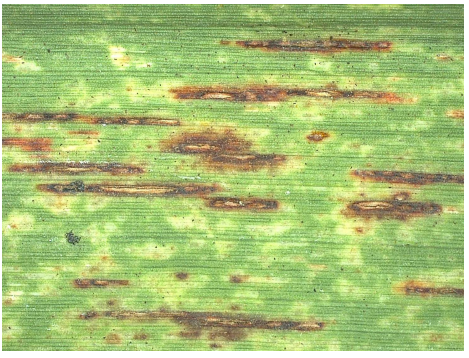

Figure 2. Old pustules (uredinia) after sporulation of *Puccinia melanocephala* (S. Sood).

When mature, pustules erupt and expose reddish-brown masses of urediniospores. In susceptible varieties with high levels of infection, these pustules merge, and necrosis develops over large leaf areas. The brown rust pustules look similar to those of orange rust caused by *P. kuehnii* and can, therefore, be mistaken for one another, especially when older lesions are examined. However, orange rust pustules are orangish-brown and not reddish-brown, and these color differences are more obvious when young pustules are observed.



Figure 3. Brown rust symptoms on the upper (left) and lower (right) sides of a leaf blade (P. Rott).



Figure 4. Numerous sporulating pustules on the lower side of a leaf affected by brown rust (P. Rott).



Figure 5. Leaf showing severe symptoms of brown rust (P. Rott).

Diagnosis

The occurrence of reddish-brown to brown sporulating pustules on the lower side of leaves is diagnostic of brown rust. The frequency of these pustules is usually higher in the upper third of the leaf, while orange rust pustules are present in the mid to lower half of the leaf and occur in groups. Urediniospores of *P. melanocephala* are darker with more prominent germ pores but are smaller (21–40 × 17–27 µm) than those of *P. kuehnii*

(25–57 × 17–34 µm) (Ryan and Egan, 1989). The spore wall in *P. melanocephala* urediniospores is thinner than in *P. kuehnii* and has no apical thickening (Figure 6). Also, the spines on the walls are closer, spaced 1–1.5 µm apart in a regular pattern (Figure 7), whereas those of *P. kuehnii* are 3–4 µm apart and are not as regularly spaced.

Several molecular assays are available for the identification of the brown rust pathogen, such as conventional and real-time PCR (Glynn *et al*., 2010; Wang *et al*., 2017; Wu *et al*., 2022). A loop-mediated isothermal amplification (LAMP) has also been developed for fast, specific, and sensitive detection of *P. melanocephala* (Wu *et al*., 2024). RGB images of infected leaves generated with object-based image analysis segmentation can be used in the field to identify and differentiate the brown and orange rusts automatically (Ordine Pires da Silva Simões *et al*., 2023).

Strains of the pathogen

Races of *P. melanocephala* were reported in India and Florida. At least six races of the pathogen were characterized in India based on differential reactions of sugarcane varieties (Srinivasan and Muthaiyan, 1965). Two races were identified in 1983 in Florida following changes in varietal resistance (Dean and Purdy, 1984). In 1988, the development of severe symptoms of brown rust on previously resistant cultivar CP78-1247 in Florida also suggested the appearance of a new race of the pathogen (Raid, 1989). Existence of four races of *P. melanocephala* in Florida in 1991-1992 was reported based on inoculation data using six sugarcane varieties (Shine Jr *et al*., 2005). No differences in virulence among isolates of the brown rust pathogen were observed in Australia based on a detached leaf bioassay.

Transmission

The spread of *P. melanocephala* primarily occurs through wind-disseminated urediniospores. These spores are released from the brown rust pustules that form on the underside of the leaves. Urediniospores are passively released during early daylight hours when relative humidity decreases. The spores become airborne after dislodging



Figure 6. Urediniospores and paraphyses of *Puccinia melanocephala* observed under a microscope (S. Sood).

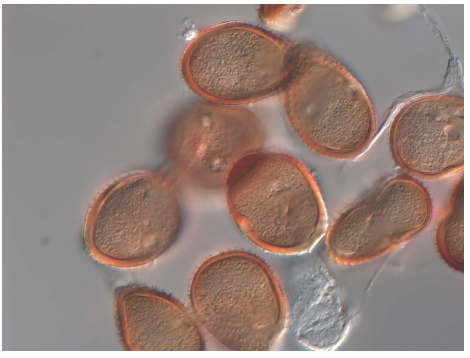

Figure 7. Urediniospores of *Puccinia melanocephala* with spines on the walls that are spaced apart in a regular pattern (L. Castlebury).

from the pustule by gravity, wind currents, or foliar movement. Once into the air, spores of brown rust may travel a few centimeters to hundreds of kilometers. Thanks to its wind-dissemination, the pathogen can travel long distances in relatively short periods. Sugarcane rust spores were most likely transmitted from Africa to the Americas by trans-oceanic high-altitude air currents (Purdy *et al.*, 1985).

Urediniospores can also be disseminated by rain splash and water but only over short distances, from a few millimeters to several meters. Brown rust spores are not disseminated by stalk cuttings, as *P. melanocephala* does not colonize the sugarcane stalk.

Host range

The main hosts of *P. melanocephala* are species of *Saccharum* but pustules of brown rust have also been seen on related species such as *Erianthus fulvus* and *Narenga porphyrocoma* (Rutherford, 2018; Ryan and Egan, 1989). Red flecks corresponding to non-sporulating lesions of resistant varieties were reported on species of *Bambusa* and *Erianthus*.

Epidemiology

The critical environmental factors for the development of sugarcane brown rust are leaf wetness and temperature (Chaulagain *et al.*, 2021; Comstock and Raid, 1994). At least eight hours of leaf wetness or high relative humidity are required for the germination of urediniospores. Brown rust is considered a cool to moderate temperature disease. Urediniospores can germinate at 5 to 34ºC, but the formation of appressoria that is needed for infection is drastically reduced when temperatures exceed 30ºC (Purdy *et al.*, 1983; Sotomayor *et al.*, 1983). In Florida and Louisiana, high summer temperatures (>30–32°C) were associated with the decline of brown rust epidemics during the crop season (Sanjel *et al.*, 2019). Brown rust is often more severe in crops established at higher elevations because of temperature limitations. *Puccinia melanocephala* has a short reproductive cycle, and when conditions are favorable for the pathogen's multiplication, rust severity and epidemics progress rapidly. Infection and sporulation will occur within 14 days of urediniospore landing on a sugarcane leaf. A field of sugarcane variety susceptible to brown rust will appear reddish-brown in six weeks.

Brown rust development is closely related to the host genotype and the plant age. Resistance to the disease varies according to sugarcane varieties, whose reactions vary from the absence of symptoms to leaves covered with hundreds of sporulating pustules. Brown rust appears to be a disease of young sugarcane, and 4–6 months is the optimum age for disease development (Comstock and Ferreira, 1986; Victoria *et al.*, 1990). In Florida, brown rust is absent in the fall and winter in mature cane (> 8 months), although the temperatures are similar to those in spring when epidemics are severe (Sanjel *et al.*, 2019).

Several soil factors have been associated with brown rust severity, such as the soil's pH and its nutrient status (Anderson *et al.*, 1990). High nitrogen levels also appear to promote rust severity.

Economic importance

Before its introduction into the Western Hemisphere in 1978, brown rust was considered a minor disease of sugarcane (Purdy *et al.*, 1983). When *P. melanocephala* spread to the Americas, several varieties susceptible to brown rust, especially highly susceptible

variety B4362, were grown over large areas. These varieties suffered yield losses of up to 50% and had to be rapidly replaced by more resistant ones (Figure 8). In Florida, 10–40% yield losses were reported in susceptible varieties (Comstock *et al.*, 1992). Reductions in both millable stalks and stalk biomass are responsible for these losses (Hoy and Hollier, 2009).



Figure 8. Sugarcane variety highly susceptible to brown rust (foreground) and sugarcane variety resistant to the disease (background) (P. Rott).

Brown rust causes also indirect economic losses due to the extra cost of fungicides and their applications and the need to develop rust resistant varieties to control the disease. Several potentially high yielding varieties are discarded because of their susceptibility to brown rust. Additionally, variants within the rust pathogen populations require ongoing breeding and selection efforts to develop disease resistance.

Control

Growing resistant varieties is the best management strategy for brown rust (Comstock and Raid, 1994). Dominant brown rust (*Bru*1 and *Bru*2) and other resistance loci or alleles have been identified (Chaves *et al.*, 2022; Daugrois *et al.*, 1996; Healey *et al.*, 2024; Islam *et al.*, 2024; Raboin *et al.*, 2006). Primers to detect the *Bru*1 and *Bru*2 genes have been developed and used to screen for resistant varieties (Costet *et al.*, 2012; Glynn *et al.*, 2013). *Erianthus arundinaceus* germplasm is resistant to brown rust without possessing the *Bru*1 gene, thus offering novel resistance sources in breeding programs (Chen *et al.*, 2025).

Several fungicides have also been registered to control brown rust. Single or combinations of fungicides such as pyraclostrobin, fluxapyroxad + pyraclostrobin, pyraclostrobin + epoxiconazole, azoxystrobin + cyproconazole, azoxystrobin + tebuconazole, trifloxystrobin + tebuconazole, and fluxapyroxad can be used (Chaulagain *et al.*, 2019; Koch *et al.*, 2021). Additionally, silicon fertilization can play an important role in the management of sugarcane brown rust (Camargo et al., 2020).

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Brown spot

A. Salem Saumtally, Sydney Sullivan, revised by A. Salem Saumtally

Cause

Cercospora longipes E.J. Butler, deuteromycete and cercosporoid fungus (Braun *et al.*, 2015).

Geographical distribution

Argentina, Bangladesh, Belize, Brazil, Burundi, Cameroon, China, Colombia, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Eswatini, Ethiopia, Fiji, Gabon, Hawaii, India, Indonesia, Jamaica, Kenya, Madagascar, Malawi, Mauritius, Mexico, Morocco, Mozambique, Nepal, Nicaragua, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, South Africa, Sri Lanka, Taiwan, Tanzania, Thailand, Uganda, USA (Florida, Louisiana, Texas), Venezuela, Zimbabwe.

Symptoms

The pathogen causes reddish-brown to dark-brown spots on the sugarcane leaf. The lesions vary in size from barely visible to about 3 × 15 mm. The spots are oval, often surrounded by a yellow halo, and are equally visible on both sides of the leaf (Figures 1, 2, and 3). The central part of the lesion is dark brown. The long axis of the spot is usually parallel to the midrib. Under conducive conditions, leaf spots are generally well distributed over the entire surface, particularly on the older leaves. Under undefined conditions, probably due to a varietal reaction, some lesions and the associated halo may be reddish and as large as 10–15 × 30–40 mm (Figure 4). The percentage of the leaf area infected depends on the resistance level of the sugarcane variety. In susceptible varieties, the spots cover extensive areas of the leaf surface, they coalesce, and may be present on younger leaves. On some varieties, older leaves turn yellow and die prematurely. Late in the season, this produces a "fired" appearance of affected fields (Abbott, 1964). Lesions can also be observed on the leaf sheath (Edgerton, 1955; Nolla, 1965; Hsieh and Tseng, 1978). The disease can be present on young and actively growing cane as well as on adult plants.

Diagnosis

Although the symptoms of brown spot may be confused with those of other leaf spot diseases such as brown stripe, eye spot or ring spot, brown spot can be distinguished by its oval-shaped, reddish-brown lesions surrounded by a yellow halo. Lesions produced by the brown stripe fungus are elongated with the formation of stripes from both ends of the lesion. Eye spot is elliptical with stripes or runners extending from the lesions towards the leaf tip. Ring spot is irregular in shape and has a distinctly dark outer border with a necrotic centre. The brown spot fungus can be recognized by its conidia and conidiophores by examining a piece of infected leaf tissue under a low-power microscope. The spores of *C. longipes* are more abundant on the lower leaf surface.



Figure 1. Reddish brown to dark brown oval spots caused by *Cercospora longipes* (A.S. Saumtally).



Figure 2. Brown spot lesions are often surrounded by a yellow halo (A.S. Saumtally).



Figure 3. Close-up of brown spot lesions and the accompanying yellow halo (H. Mungur).



Figure 4. Normal small lesions and atypical very large reddish lesions of brown spot on the same sugarcane leaf (A.S. Saumtally).

C. longipes belongs to the subdivision Deuteromycotina and has been described by Mulder and Holliday (1974) and Braun *et al*. (2015). The conidiophores are olive-brown and up to about 200 × 4 µm, arising from a stroma, held in fascicles, geniculate, rarely branched, and septate with thickened conidial scars. Conidia vary in size from 20–200 × 3–8 µm and are hyaline, broader at the base, straight or curved at the tip, and distinctly 3 to 11 multi-septate (Figure 5).



Figure 5. Conidiophores and conidia of *Cercospora longipes* (A.S. Saumtally).

The pathogen can be isolated on cane leaf agar, Czapek yeast extract agar, potato dextrose agar, or V-8 juice medium. After incubation for 5–7 days on Czapek yeast extract agar at 28°C, a dark green (olive) colony producing a brown diffusible pigment is formed. On culture media, conidia production is greatest at 22°C (Hsieh and Tseng, 1978). Highest radial growth and sporulation of *C. longipes* was observed on 25% fresh sugarcane leaf extract + 20% V8 medium at 25°C (Borase *et al.*, 2026). Molecular characterization of the fungus can be performed by amplification and sequencing of the ITS, 18S rRNA, β-tubulin, and actin genes (Borase *et al.*, 2026).

Strains of the pathogen

A new race of *C. longipes* was identified in Taiwan based on its pathogenicity (Hsieh and Tseng, 1978).

Transmission

Spores of the pathogen are wind-borne. The fungus is present on infected leaves adhering to plant parts as well as on leaf trash (Abbott, 1964). Survival on young sugarcane regrowth has also been observed. The disease is less pronounced in seasons following winters severe enough to kill all above-ground parts (Edgerton, 1955).

Host range

Saccharum officinarum and *Saccharum* hybrids are the main hosts. However, the disease is also present on *S. spontaneum,* which is susceptible to brown spot (Abbott, 1964; Hsieh and Tseng, 1978). In a survey of over 500 wild germplasm clones at Kannur, India, where conditions are reported to be well-suited for the occurrence of foliar diseases, brown spot was present to various degrees in about 60% of *S. spontaneum* clones but not on *Erianthus* spp. (Gopi *et al.*, 2023).

Epidemiology

In most countries, the disease is generally present throughout the year, but infection is prevalent under humid conditions and mild temperatures of 20–22°C.

Economic importance

In general, brown spot is considered to be a minor disease. However, concerns have been expressed in some instances, such as in Louisiana where a 12% sugar loss due to the disease was reported (Abbott, 1951). Outbreaks of brown spot in Karnataka and Maharashtra (India) also severely affected crop productivity (Viswanathan and Ashwin, 2020).

Control

The disease is controlled using resistant varieties and screening methods have been developed for brown spot (Wismer and Koike, 1965; Wang and Lee, 1983). Nevertheless, most selection programs do not conduct formal trials to identify resistant varieties. Fungicides such as mancozeb, copper oxychloride, copper oxychloride + zineb are effective in controlling the disease (Muthusamy and Sithanantham, 1974), but in practice, no chemical treatment is applied against brown spot.

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Brown stripe

Peter J.L. Whittle, revised by Barry J. Croft

Cause

Bipolaris stenospila (Drechsler ex Faris) Shoemaker (Syn. *Cochliobolus stenospilus* T. Matsumoto & W. Yamam.). In the Yunnan province of China, two other species of *Bipolaris* cause sugarcane brown stripe: *B. secalis* Sisterna ex Y.P. Tan, Madrid, Crous & R.G. Shivas, and *B. setariae* Shoemaker (Li *et al.,* 2022 and 2025).

Geographical distribution

Australia, Bangladesh, Barbados, Belize, Brazil, China, Côte d'Ivoire, Cuba, Dominican Republic, Ethiopia, Fiji, French Guiana, Gabon, Guadeloupe, Guyana, Hawaii, India, Indonesia, Jamaica, Japan, Malawi, Malaysia, Mozambique, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, St Kitts and Nevis, Samoa, Senegal, South Africa, Taiwan, Thailand, Trinidad and Tobago, USA, Zambia.

Symptoms

Earliest symptoms are minute (0.5 mm), watery spots on the leaf blade. These turn reddish and elongate in both directions parallel to the veins. When mature, lesions are brownish-red, up to 2–4 mm wide and 10–30 mm (up to 75 mm) long (Figure 1). Lesions are surrounded by a yellow halo, which is only slightly wider than the lesion itself (Figure 2). In severe cases, lesions may coalesce and kill large areas of the leaf. Symptoms on different varieties may differ slightly in appearance (Figure 3).

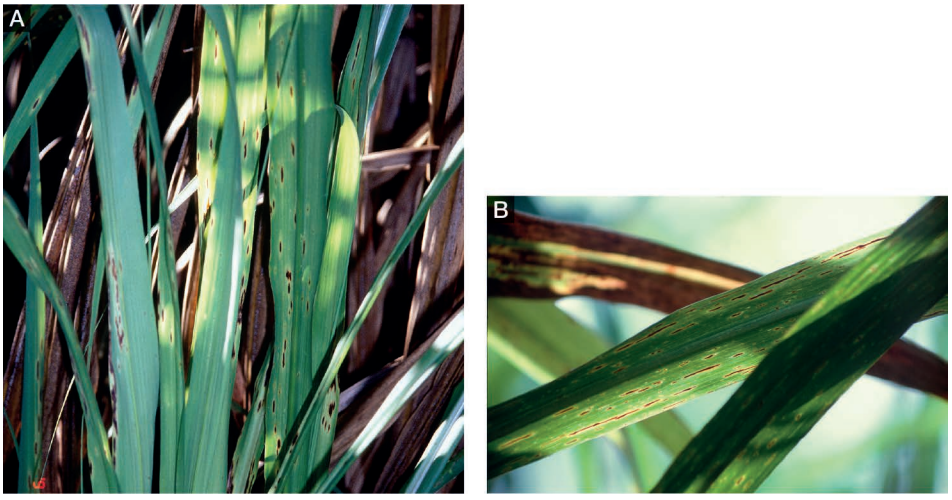

Figure 1. Brownish-red leaf lesions surrounded by a yellow halo (A: BSES; B: J.H. Daugrois).

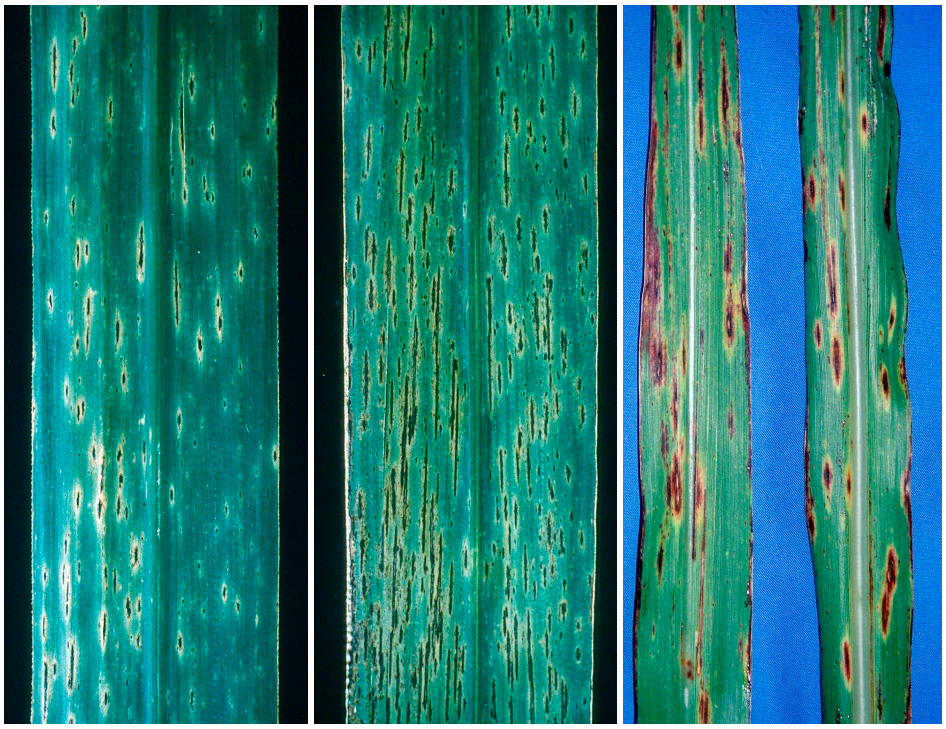

Figure 2. Brownish-red leaf stripes surrounded by a yellow halo (BSES).

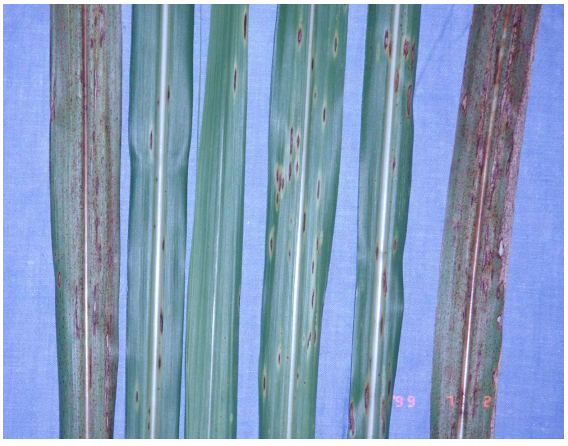

Figure 3. Leaves showing various degrees of brown stripe lesions (BSES).

Diagnosis

Leaf symptoms are characteristic. Lesions of brown stripe are similar to those of eyespot but are more elongated and less oval shaped, and they lack the chlorotic 'runner' extending up the leaf from the lesion. Brown stripe is normally found on crops growing in soils of low fertility and may be prevalent at any time of the year.

Bipolaris stenospila and *Bipolaris setariae* can be identified from their conidia (Ellis and Holliday, 1971; Manamgoda *et al.,* 2014). These can readily be induced to form by incubating diseased leaves in a humid chamber for a few days, or by isolating on a range of

standard growth media. The morphology of the different species of *Bipolaris* has been described by Manamgoda *et al.,* (2014), and a key for distinguishing *Bipolaris* species is given by Sivanesan (1987).

The species of *Bipolaris* can be identified by sequencing of the internal transcribed spacer region (ITS), GAPDH and EF-1α loci of isolates and the GAPDH gene is the best marker for *Bipolaris* species (Li *et al*., 2025; Manamgoda *et al.,* 2014).

Strains of the pathogen

Physiologic races of the pathogen have been reported, but this is not confirmed, because varieties may react differently to brown stripe under different nutrient regimes (Martin, 1989).

Transmission

Brown stripe is transmitted by conidia spread by wind and rain.

Host range

B. stenospila has been reported on *Brachiaria platyphylla*, *Cynodon dactylon*, *Imperata arundinacea*, *Pennisetum glaucum* and *Zea mays* (Manamgoda et al., 2014). *Bipolaris setariae* has a very wide host range on monocotyledons and dicotyledons (Manamgoda *et al.*, 2014).

Epidemiology

Brown stripe occurs on sugarcane suffering nutrient deficiencies growing on soils with low fertility. Conidia develop on dead leaves in large numbers and are spread by wind. The fungal spores germinate on leaves in the presence of free moisture. After penetration, infection proceeds regardless of atmospheric conditions.

Economic importance

Economic losses occur from brown stripe when susceptible varieties are grown on nutrient-deficient soils. It is difficult to separate the losses caused by brown stripe and those caused by poor nutrition. The incidence of brown stripe in China has recently increased with yield losses in the range of 18–35% (Wang *et al.,* 2021).

Control

Brown stripe can be controlled by fertilizing affected crops, especially with fertilizers containing potassium and/or phosphorus. Generally improving soil nutrition and nutrient retention by addition of organic fertilizers such as mill filter cake/mill mud can provide longer term control of brown stripe. Varieties vary in resistance to brown stripe and resistant varieties should be planted in areas where the disease is common. Varieties have been screened for resistance in field trials in China (Wang *et al.,* 2021) and genes associated with resistance to brown stripe have been identified (Cheng *et al.,* 2022).

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Downy mildew

Sidney Suma, Robert C. Magarey,
revised by Robert C. Magarey and Nicole Thompson

Cause

Peronosclerospora sacchari (T. Miyake) Shirai & Hara, *P. philippinensis* (W. Weston) C.G. Shaw, *P. spontanea* (W. Weston) C.G. Shaw (can also infect sugarcane), and undescribed *Peronosclerospora* species.

Geographical distribution

Indonesia, India, Fiji, Japan, Papua New Guinea, Philippines, Taiwan, Thailand.

The disease is restricted to the Pacific, South Asia and South East Asian regions.

Symptoms

The disease is characterized by leaf streaks, varying in color as the leaves and symptoms age. Streaks initially are pale to light yellow in color, but with increasing age change from greenish-yellow to a more definite yellow, and finally to a brick-red color (Figures 1 and 2). Streaks are generally 1–3 mm in width but can be much wider in some varieties. The streaks vary in length, run parallel to the leaf venation and have a well-defined straight margin, at least initially. There may be many streaks per leaf and over 20 is not uncommon. Streaks may occur on the midrib but are not normally seen on the leaf sheath. Narrow streaks may be seen on most canes in winter.

A characteristic symptom is the production of down on the underside of infected leaves on warm humid nights (Figures 3 and 4). When freshly produced, the down is soft and velvety in appearance but may appear grey and dusty after it has dried. The down results from the production of conidiophores and conidia by the fungus on the lower side of affected leaves.

Leaf shredding is also a symptom of infection by *Peronosclerospora* species (Figure 5); shredding has been associated with the primary *Peronosclerospo*ra species host (Magarey *et al.*, 2018b). Oospores are produced between the vascular elements and are so large that they push the adjacent elements apart; this leads to leaf shredding, an obvious symptom under certain conditions.

In colder months, the systemic disease may give rise to rapid elongation of certain diseased stalks leading to the symptom referred to as 'jump-up'. These stalks are usually much thinner than normal stalks and up to twice as long. Often they lodge, or break, and may die before harvest.



Figure 1. Yellow-green leaf streaks interspersed with normal green leaf tissue: early symptoms (R. Magarey).



Figure 2. Brick-red leaf streaks interspersed with normal green leaf tissue: older symptoms (N. Thompson).



Figure 3. Down produced on the underside of an infected young leaf (N. Thompson).

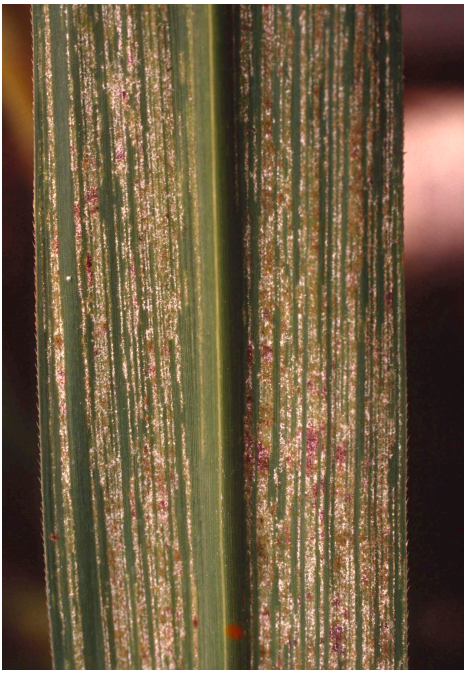

Figure 4. Down produced on the underside of leaf with severe symptoms of downy mildew (P. Rott).



Figure 5. Leaf shredding symptoms (N. Thompson).

Diagnosis

Visual diagnosis relies on the observation of the leaf streak symptoms and the distinctive down on the underside of leaves. Incubation of diseased leaves in a humid chamber overnight will readily induce down production, if none is present on the leaves showing symptoms. Molecular assays for *Peronosclerospora* species infecting sugarcane have been developed (Magarey *et al*., 2018b).

Strains (species) of the pathogen

Disease symptoms in the late 1980s suggested that at least two pathogen 'strains' were present on the Estate of Ramu Sugar Limited, Papua New Guinea (PNG), based on differential resistance observations in several varieties (Egan, 1991; Suma and Pais, 1996).

These observations were researched further with the collection of downy mildew infested leaves from a range of *Saccharum* species from sites around PNG. Morphological and molecular analysis (Cox1 and Cox2 assays) of pathogen oospores and infected leaves, coupled with transmission experiments involving different hosts (*S. edule, S. officinarum, S. robustum*, *Zea mays*), suggested that at least three *Peronosclerospora* species complexes are present in PNG (Magarey *et al*., 2018b). It was concluded that *P. sacchari* is likely present along with two other undescribed species (Magarey *et al*., 2018a and 2018b). No evidence was found to suggest the presence of *P. philippinensis* in PNG.

Within the large species complex, there are potentially 3 species: one infecting *Miscanthus* (a new species, genetically different from the *P. miscanthi*); one infecting *S. robustum* (from the lower Madang and Gusap Provinces); and a third undescribed species complex that can infect a wide range of hosts (Magarey *et al*., 2018b).

Further research is needed to describe strains/species within the identified species complexes.

Transmission

The disease is transmitted by movement of the air-borne conidia of the pathogen and the planting of systemically infected plant material. Conidia are produced on warm (maximum production between 22 and 25°C), humid (close to 100% relative humidity) nights and are discharged from about 23h30 onwards. They are susceptible to drying and lose viability within hours of daybreak. Wind currents may carry conidia up to 400 m from diseased crops. Infection of standing crops usually occurs via lateral buds on standing stalks, or in immature leaf tissue such as in the spindle.

Research has shown that oospores may act as primary inoculum (Magarey *et al*., 2018a). Spread between regions, or countries, is thought to have been through diseased planting material. The planting of diseased cane will usually lead to diseased crops. Sometimes diseased plants result from planting apparently healthy cane; here, latently infected planting material (with diseased lateral buds) may give rise to diseased shoots on germination.

Host range

Peronosclerospora species (P. sacchari, P. philippinensis and other species in PNG*)* may infect a number of other hosts including Johnson grass (*Sorghum halepense*), maize (*Zea mays*, which may be as susceptible as sugarcane), some sorghum lines, Sudan grass (*S. sudanense*), and teosinte (*Euchlaena mexicana*).

Epidemiology

The production of conidiophores and conidia occurs at night when conditions are suitable. Conidial production is optimal at 22–25°C and 100% relative humidity (Leu and Egan, 1989). Under natural conditions, conidia begin to discharge from 23h30 onwards and peak around 1h30–2h30 when about half the discharge occurs (Leu and Egan, 1989).

Sporulation is initiated in daylight hours; free water on the leaf surface or low light levels may hinder spore production. Sporulation does not occur on rainy days and nights.

The role of oospores in the disease epidemiology is uncertain but recent research suggests that they may play a role in disease causation via infection of germinating setts and subsequent conidia disease spread via wind.

Economic importance

Downy mildew causes serious economic damage in locations where susceptible varieties are grown. The losses can be two-fold: (1) direct yield losses resulting from disease infection in susceptible varieties (Figure 6), and (2) loss of potential yield from not growing susceptible, but otherwise agronomically superior, varieties (Leu and Egan, 1989). In PNG, direct yield losses of up to 15% of total tonnage harvested were reported (Suma and Pais, 1996). Estimated losses in commercial cane in the Philippines (due to *P. philippinensis*) reached 38% (tons of cane) and 58% (tons of sugar/ha) (Husmillo, 1982). More recent research at Gusap, PNG suggested losses of 58% tons cane and 75% tons sugar per hectare (Rauka *et al*., 2005).

Between 1979 and 1992, up to 36% of all newly introduced commercial varieties were discarded at Ramu Sugar due to downy mildew susceptibility (Suma and Pais, 1996). The disease remains one of the most important affecting commercial sugarcane production in PNG. Breeding programs must take resistance to downy mildew into consideration in regions where the disease is prevalent.



Figure 6. Sugarcane variety showing severe symptoms of downy mildew (P. Rott).

The disease has been eradicated in Australia. However, downy mildew remains a major quarantine threat to movement of germplasm between countries, particularly movement of germplasm out of PNG (Magarey, 1996).

Control

The disease is controlled through the use of resistant varieties and disease-free planting material. Material may be rendered disease-free by hot water treatment at 50°C for 2 h or by dipping in a solution of the fungicide metalaxyl (1.25 g a.i./l, 5-s immersion). The fungicide has also been applied through a mechanical planter, as well as to both the soil and foliage of infected crops. Metalaxyl has a prophylactic effect, maintaining the disease-free status of the crop for up to 3 months after application (Eastwood and Malein, 1998; James, 1983; Malein, 1993).

The use of disease-free seed cane has been shown to complement the use of partial resistance in varieties (Eastwood and Malein, 1998; Egan, 1986; James, 1983; Malein, 1993; Suma and Pais, 1996) and is an important aspect of the disease control strategy.

Control in ratoon crops has been achieved by soil application of granular metalaxyl at 1.8–2.0 kg a.i./ha and foliar applications (Eastwood and Malein, 1998; James, 1983; Malein, 1993).

Control of the disease on other host plants can be achieved through fungicide treatment of seed, as observed in PNG. This reduces the disease load in an area where sugarcane is planted with other hosts.

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Dry top rot

Jack C. Comstock, revised by Jack C. Comstock and Sushma Sood

Cause

Ligniera vasculorum (Matz) M.T. Cook, plasmodiophoromycete fungus.

Geographical distribution

Barbados, Colombia, Cuba, Guyana, Panama, Puerto Rico, USA (Florida).

Found in an herbarium sample (sugarcane variety Cristalina) from Venezuela collected in 1932 (Cornell Plant Pathology Herbarium, 2023).

Symptoms

The symptoms of dry top rot are like drought stress. Symptoms usually appear late in the season on mature plants (Abbott, 1964; Matz, 1920). Initial symptoms include the withering of spindle leaves and drying of leaf tips and edges of the leaf blades (Figures 1 and 2). Stalk growth is eventually stunted and the upper internodes are shortened and become withered. In severe cases, some or all the stalks may die. The dead stalks usually



Figure 1. Drying of the upper leaves of sugarcane plants (J.C. Comstock).



Figure 2. Drying of the upper leaves of a sugarcane stalk (J.C. Comstock).

remain standing and become more shriveled (Comstock *et al.*, 1994). Rind disease may also occur on the affected stalks. Leaves remain attached to the dead stalks until harvested or detached with only the stalk remaining (Figures 3 and 4). There is no side shooting on the affected stalks.



Figure 3. Drying of leaves and loss of some leaves (J.C. Comstock).



Figure 4. Withered and shrunken dead stalk (J.C. Comstock).

Diagnosis

A positive diagnosis is made by observation of the characteristic orange-brown spores (17–25 µm in diameter) of *L. vasculorum* in the xylem cells of the orange-pink vascular bundles located in the internodes at the base of dry top rot symptomatic plants (Comstock *et al.*, 1994; Matz, 1922) (Figure 5). The discolored vascular bundles are pale and not as red as those discolored by leaf scald and ratoon stunt (Comstock *et al.*, 1994). The discolored vascular bundles can be dissected from the stalk using a sharp scalpel (Figure 6). Spores are rarely found in non-discolored vascular bundles located in the same internode. Although the discolored vascular bundles and spores can be found in several internodes above the soil line, they are most prevalent in the older internodes. Furthermore, the xylem vessels of the successive internodes of the infected stalks may have a grey granular substance that is thought to be the plasmodium stage of the pathogen (Cook, 1937; Matz, 1922). In young plants (3–5 months old) that either germinated from dry top rot infected seed pieces or developed from ratooned stools, spores of the pathogen may be found at the base of the plant, usually below the soil line.

In stalks that have deteriorated because of death due to the disease, it may be impossible to observe spores of the dry top rot pathogen because of the invasion of other fungi.

Strains of the pathogen

No information is known.

Transmission

The dry top rot pathogen, *L. vasculorum*, can be transmitted by planting seed pieces cut from infected stalks but only stalks with slight symptoms that are not dead. Germination and survival of new plants from affected seed cane are greatly reduced. Healthy seed pieces planted in areas where the disease is present become infected, indicating that the pathogen is soil-borne.

Host range

No host other than sugarcane is known.

Epidemiology

Dry top rot was more prevalent in poorly drained wet areas in Puerto Rico. High water tables for at least a portion of the year favor the disease (Cook, 1929).

Economic importance

Dry top rot has not caused economic losses in Florida due to its limited distribution, primarily to a small area (one ha) on the USDA-ARS Sugarcane Field Station at Canal Point, Florida (Comstock *et al*., 1994). Dry top rot was discovered in a few varieties of Stage 3 of the Canal Point sugarcane cultivar development program planted on growers' farms, specifically in Eastgate in 2016 and Okeelanta in 2022. The limited disease spread to growers' farms was probably by planting of infected stalks collected from the affected field at Canal Point, FL. Dry top rot is, however, a potential threat because in the 1920s it was economically damaging in Puerto Rico (Matz, 1922). If the disease was widespread, it could cause yield losses (Figure 7).

Control

Control of dry top rot is achieved by planting healthy seed cane or resistant varieties, thus preventing the spread of the disease. Hot water treatments (52 °C for 45 min) gave partial control (Comstock *et al*., 1994).

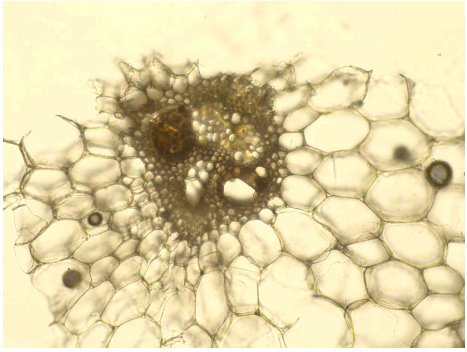

Figure 5. Spores of the pathogen in xylem cells of a vascular bundle (J.C. Comstock).

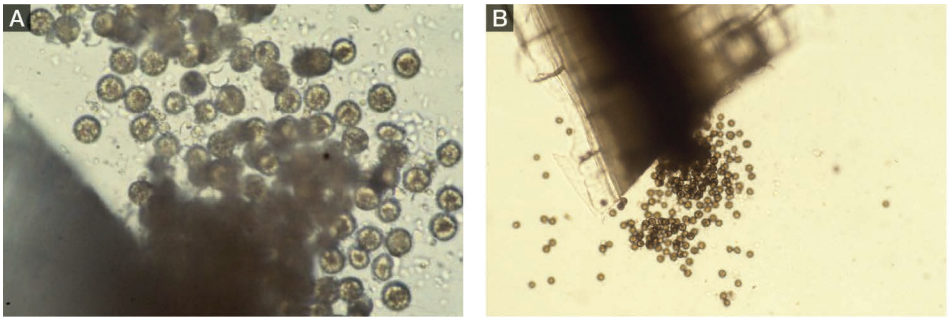

Figure 6. Spores of *Ligniera vasculorum* escaping from a cut vascular bundle (A: S. Sood; B: J.C. Comstock).



Figure 7. Sugarcane variety highly susceptible to dry top rot (J.C. Comstock).

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Exserohilum leaf spot

R. Selvakumar

Cause

Exserohilum rostratum (Drechsler) K.J. Leonard & Suggs, ascomycete fungus resembling *Bipolaris* and *Drechslera* species of the *Pleosporaceae* family.

Geographical distribution

India, Iran.

Symptoms

First symptoms on leaves appear as small, reddish brown colored spots. These spots enlarge as brown stripes and later merge with adjacent stripes, thus forming leaf blights with irregular margins (Figures 1 and 2). In later stages of the disease, the leaf surface becomes dry and the entire sugarcane plant shows dried up symptoms.



Figure 1. Reddish-brown spots and stripes on a sugarcane leaf (R. Selvakumar).



Figure 2. Leaf showing severe symptoms of *Exserohilum* leaf spot (R. Selvakumar).

Diagnosis

The pathogen can be isolated from infected leaves on potato dextrose agar (PDA), and the fungal colonies are grey to blackish-brown on the upper side while they are olivaceous-black on the lower side. Conidiophores are single, cylindrical, geniculate, dark brown, up to 200 µm long, 5–7 µm thick, with a swollen basal cell. Conidia are straight to slightly curved, ellipsoid to narrowly obclavate or rostrate, brown, 6–16 distoseptate, with a basal septum darker and thicker than the other septa (Figures 3 and 4). Their size varies from 15–200 × 7–29 µm and they have a distinct protruding basal hilum. Growth of *E. rostratum* is very slow at 8–40°C and optimum temperature is 28–32°C (Tsai *et al*., 2001). Germination of conidia is monopolar and bipolar.

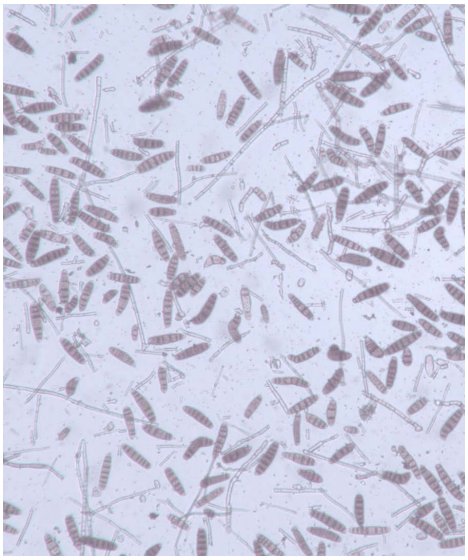

Figure 3. Numerous conidia of *Exserohilum rostratum* (R. Selvakumar).



Figure 4. Septate conidia and septate mycelia of *Exserohilum rostratum* (R. Selvakumar).

Molecular diagnosis of *E. rostratum* can be performed by PCR, sequencing, and sequence comparison with isolates of the pathogen from sugarcane and other plants such as banana, cherry tomatoes, *Datura inoxia*, ginger, maize, orchids and rice. Universal primer pairs ITS1 and ITS4 have been used to amplify the internal transcribed spacer (ITS) region of the rRNA gene (Ahmadpour *et al*., 2012; Selvakumar *et al*., 2023). Genes coding for the glyceraldehyde-3-phosphate dehydrogenase (GAPDH) and the translation elongation factor 1 alpha (TEF1-α) can also be targeted for pathogen identification (Kaboré *et al*., 2022).

Strains of the pathogen

No strains of the pathogen have been reported.

Transmission

The causal agent of sugarcane Exserohilum leaf spot is transmitted from plant to plant by air borne conidia.

Host range

E. rostratum causes disease in more than 30 plant species covering 28 genera and 11 families, including many economically important crops such as banana, maize, rice, sugarcane, sweet sorghum, tomato and wheat (Kaboré *et al*., 2022; Korra *et al*., 2023; Lin *et al*., 2011; Selvakumar *et al*., 2023; Xie *et al*., 2022). The fungus is also capable of causing leaf spots on weeds growing in or in the proximity of crop fields (Korra *et al*., 2023).

Epidemiology

Warm weather associated with seasonal rainfall favors disease development in tropical environments.

Economic importance

The economic impact of sugarcane leaf spot has not been determined yet. Besides being a plant pathogen, *E. rostratum* is also an important opportunistic human pathogen causing skin and corneal infection, as well as fungal meningitis and sinusitis (Adler *et al*., 2006; Alajmi *et al*., 2019; Larone and Walsh, 2013).

Control

Prophylactic spraying of benzimidazole and propiconazole fungicides are recommended under favorable conditions on highly susceptible varieties. Further research work is required to develop an integrated disease management strategy.

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Eye spot

Jack C. Comstock, revised by Jack C. Comstock and Sushma Sood

Cause

Bipolaris sacchari (E.J. Butler) Shoemaker, hyphomycete fungus. *Curvularia spicifera* (Bainier) Boedijn [syn. *Bipolaris tetramera* (McKinney) Shoemaker 1959 or *Helminthosporium tetramerum* McKinney 1925] causes also eye leaf spot of sugarcane in India (Sharma *et al.*, 2004).

Geographical distribution

Andaman Islands, Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Bolivia, Brazil, Cambodia, Cameroon, China, Colombia, Costa Rica, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, El Salvador, Fiji, Grenada, Guatemala, Guyana, Haiti, Hawaii, Honduras, India, Indonesia, Italy, Jamaica, Japan, Kenya, Madagascar, Madeira, Malawi, Malaysia, Martinique, Mauritius, Mexico, Mozambique, Nigeria, Pakistan, Papua New Guinea, Panama, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, St Thomas, Samoa, Senegal, Sierra Leone, Solomon Islands, South Africa, Sri Lanka, Surinam, Taiwan, Tanzania, Thailand, Trinidad and Tobago, Uganda, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

Minute water-soaked spots that usually occur on young leaves are the first observable eye spot symptom. Typical mature lesions are reddish-brown with yellowish-brown margins (Comstock and Steiner, 1989; Martin, 1961). The lesions are elliptical (0.5–6.0 mm long and 0.5–3.0 mm wide). Yellow to reddish-brown runners often extend toward the apex from the lesion and can be 3–6 mm wide and extend up to 90 cm (Figures 1 to 4). The runners result from tissue damage caused by toxins produced by the pathogen (Comstock and Steiner, 1989; Macko, 1983; Steiner and Strobel, 1971). If the leaf has multiple lesions, necrosis may develop and cover a large portion of the leaf (Figure 5).



Figure 1. Elliptical eye-shaped lesion with a chlorotic runner (S. McFarlane).



Figure 2. Elliptical eye-shaped lesion without a runner (R.P. Bertani).



Figure 3. Long yellow-reddish runners extending from elliptical eye-shaped lesions (S. McFarlane).



Figure 4. Leaf with various eye spot lesions (P. Rott).



Figure 5. Leaves showing severe eye spot symptoms (A: R.P. Bertani; B: P. Rott).

The typical elongate eye spot lesion with the characteristic reddish-brown runner extending toward the leaf margin is diagnostic. *Bipolaris sacchari* will sporulate within 3–4 days from young lesions that have been placed in moist chambers after being surfaced sterilized in a dilute sodium hypochlorite solution and rinsed in sterile water. The olive-brown conidia (22–110 × 9–21 µm) are elliptical and slightly curved with 3–10 segmentations.

Strains of the pathogen

No information is available.

Transmission

Eye spot is transmitted by wind and rain-dispersed conidia. Transmission by other means such as machinery, human and stalk setts are not important.

Host range

B. sacchari is primarily confined to *Saccharum* species. However, the eye spot pathogen has also been isolated from elephant grass (*Pennisetum purpureum* or *Cenchrus purpureus*) and lemon grass (*Cymbopogon citratus*), but only the isolates from lemon grass were pathogenic to sugarcane. Pathogenic isolates of *B. sacchari* were obtained from two noxious weeds in Florida, namely the Japanese climbing fern (*Lygodium japonicum*) and the Old World climbing fern (*L. microphyllum*) (Elliott and Rayamajhi, 2005). The pathogen also caused leaf spots on bananas in Brazil (Silva *et al.*, 2008). In India, lemongrass (*Cymbopogon flexuosus*) and citronella (*Cymbopogon winterianus*) naturally infected by *B. sacchari* exhibited leaf spots parallel to veins and streaks extending towards the leaf tips (Mane *et al*., 2020),

Epidemiology

B. sacchari infection is favored by cool and wet weather conditions. Wind and rain help to disperse the pathogen's conidia (Martin, 1961). Cool weather is also required for susceptible plants to be sensitive to the host-specific toxin produced by *B. sacchari*. Eye spot susceptible plants grown at high temperatures are toxin insensitive and do not develop the typical runners associated with the disease (Byther and Steiner, 1975 and 1976).

Economic importance

Eye spot is considered a minor disease. Most cultivars have adequate field resistance. Occasionally, a susceptible cultivar is released and disease outbreaks occur (Figure 6). In areas that are conducive for the disease, variety development programs screen clones and eliminate susceptible varieties in their selection program. This not only increases the cost of variety development programs but also eliminates some potentially high yielding varieties because of their susceptibility (Comstock and Steiner, 1989).

Control

Resistant cultivars are the only practical means of control (Osada and Flores, 1968). Screening programs for eye spot resistance have been based on inoculation with spores of the pathogen and by using a toxin produced by the pathogen (Dean and Miller, 1975).



Figure 6. Sugarcane field grown with a variety susceptible to eye spot (S. McFarlane).

Control by fungicides is not economical. Essential oil of *Citrus sinensis* and aquatic extract of garlic bulb completely inhibit mycelial growth of *Curvularia spicifera in vitro* but their efficacy under field conditions remains to be investigated (Sharma *et al*., 2004).

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Floral smuts and ergot

Claude Ricaud, revised by Andre B. Gama

Cause

The fungi listed below are associated with the floral smuts and ergot that infect sugarcane (Bachchhav, 1978; Robinson, 1964; Sivanesan and Waller, 1986).

Covered smut: *Sphacelotheca macrospora* W.Y. Yen & C.S. Wang, basidiomycete fungus.

Floral smuts: *Sporisorium cruentum* (J.G. Kühn) Vánky [Syn. *Sphacelotheca cruenta* (J.G. Kühn) Potter and *Ustilago cruenta* J.G. Kühn], *Sphacelotheca erianthi* (Syd. & P. Syd.) Mundk. [Syn. *Ustilago erianthi* Syd. & P. Syd.], and *Sporisorium schweinfurthianum* (Thüm.) Karatygin [Syn. *Sphacelotheca schweinfurthiana* (Thüm.) Sacc.], all basidiomycete fungi.

False floral smut: *Claviceps* sp., ascomycete fungus.

Ergot: *Claviceps purpurea* (Fr.) Tul. and *C. pusilla* (Ces.), ascomycete fungi.

Geographical distribution

Covered smut: Taiwan.

Floral smuts: Cuba, India.

False floral smut: Australia, Colombia, India, Thailand.

Ergot: Australia, Colombia, India, Panama and probably Philippines.

Symptoms

In the case of floral smuts, the fruiting bodies of the fungus (sori) develop in the floral organs, usually the ovaries, which are turned into a dusty spore mass around a central columella made up of host tissue and covered by a definite whitish grey pseudomembrane (Figure 1). The latter, which is composed largely or entirely of sterile fungus cells, later flakes away.



Figure 1. Floral organs covered with a dusty spore mass (ISSCT).

Infection of *Sporisorium cruentum* has been reported to cause shortening of internodes with profuse tillering, excessive sprouting of lateral buds, and nearly all inflorescences are affected (Chona and Munjal, 1951). However, the evidence so far is that floral smuts infect inflorescences, and systemic infection has not been proved. *Sporisorium schweinfurthianum* induces early flowering (Robinson, 1964).

With *Claviceps* infection, the ovaries are turned into a fungal mass, the sclerotia. Frequently there is an upset of the enzyme mechanism in the floral parts resulting in the exudation of a sugary liquid "honeydew". This is associated with the presence of the imperfect sphacelial (conidial) stage of the fungus. Secondary infection by the saprophytic fungus *Epicoccum andropogonis* often occurs and the spikelets turn dull grey and eventually black, being covered with the black fruiting bodies of the saprophyte which give them a smutted appearance (Robinson, 1964).

Diagnosis

The floral smuts can be recognized by the sori of the floral organs as described above. The spores of *Sphacelotheca* and *Sporisorium* are free, spherical to ovoid for *Sporisorium cruentum*, globose or sub-globose for other species. Spore sizes are: *Sporisorium cruentum*, 5–9 µm; *Sphacelotheca erianthi,* 4.8–8.6 µm; *Sporisorium schweinfurthianum,* 10–12 µm.

Claviceps can be recognized by the presence of the sclerotia, the ascomycete fruiting bodies and the conidial stages. For *C. purpurea*, ascopores are 100 × 1 µm and conidia 6–9 × 3–4 µm. For *C. pusilla*, sclerotia measure 8 × 2 mm, ascospores 160 × 1 µm and conidia 10–15.5 × 5.0–7.5 µm.

More precise descriptions are given by Mundkur and Thirumalachar (1952), Robinson (1964), and Sivanesan and Waller (1986).

Transmission

The diseases are probably transmitted by wind-blown spores and infection in true seed can help to spread them from one region to another.

Host range and strains of the pathogen

Sphacelotheca, *Sporisorium*, and *Claviceps* infect sugarcane and its related wild *Saccharum* species as well as a number of cultivated and weed grasses including *Erianthus* (Holliday, 1980; Robinson, 1964). These fungi are known to have several races but there is no information on strain variation and host specialization for the sugarcane pathogens.

Epidemiology

False floral smut is influenced by conducive weather conditions. Mild to low temperatures not exceeding 28°C with high relative humidity and long periods of wetness during flowering increases the severity of false floral smut epidemics (Gopi *et al*., 2023).

Economic importance and control

Infections of the inflorescence by floral smut fungi are not of economic importance in cultivated sugarcane. Exceptionally *Sporisorium cruentum* has been found to affect sugarcane growth (Chona and Munjal, 1951). However, these pathogens present a serious drawback in a breeding program.

No specific control measure is required in cane fields but movement of true seed (fuzz) for a breeding program must be subject to strict quarantine and fungicide treatment. Although not much information is available regarding varietal susceptibility to false floral smut, with the exception of some hybrids in India, most modern sugarcane varieties and related germplasm species (*S. barberi, S. officinarum, S. robustum, S. sinense* and *S. spontaneum*) are resistant to false floral smut (Gopi *et al*., 2023).

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Fusarium diseases: Pokkah boeng and Fusarium sett and stem rot

Barry J. Croft

Note that wilt disease caused by *Fusarium sacchari* is detailed in a specific chapter of the book.

This chapter has been updated and rewritten based on the original chapters entitled "Fusarium sett and stem rot" and "Pokkah boeng" authored by B.J. Croft, and P.J.L. Whittle and Irawan, respectively, in 2000.

Cause

Fusarium fujikuroi species complex (FFSC), ascomycete fungi (Costa *et al*., 2022).

Molecular studies of the *F. fujikuroi* complex associated with pokkah boeng and Fusarium sett and stem rot of sugarcane resulted in the identification of different *Fusarium* species depending on the geographical location:
– In Asia: *F. andiyazi* Marasas, Rheeder, Lampr., K.A. Zeller & J.F. Leslie, *F. fujikuroi* Nirenberg, *F. incarnatum* (Desm.) Sacc., *F. proliferatum* (Matsush.) Nirenberg ex Gerlach & Nirenberg, *F. sacchari* (E.J. Butler) W. Gams, *F. subglutinans* (Wollenw. & Reinking) P.E. Nelson, Toussoun & Marasas and *F. verticillioides* (Sacc.) Kuntze (Bao *et al*., 2023; Hilton *et al*., 2017; Lin *et al*., 2014; Siti Nordahliawate *et al*., 2008; Srimali *et al*., 2022; Viswanathan *et al*., 2026).
– In South Africa: *F. andiyazi* Marasas, Rheeder, Lampr., K.A. Zeller & J.F. Leslie, *F. proliferatum* and *F. sacchari* (Govender *et al*., 2010; McFarlane *et al*., 2009).
– In Brazil: *F. sacchari*, *F. proliferatum* and an unidentified *Fusarium* species (Costa *et al.,* 2019).

Fusarium tricinctum (Corda) Sacc. has also been isolated from sugarcane showing stem rot symptoms (Koike, 1978). *F. tricinctum* is generally not a pathogen but it can attack sugarcane that has suffered from frost damage.

Geographical distribution

Pokkah boeng

Andaman Islands, Angola, Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Benin, Brazil, Burkina Faso, Burundi, Cambodia, Cameroon, Central African Republic, Chad, China, Colombia, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Ecuador, Egypt, Eswatini (formerly Swaziland), Ethiopia, Fiji, French Guiana, Gabon, Ghana, Guadeloupe, Guam, Guyana, Haiti, Hawaii,

Honduras, India, Indonesia, Iran, Iraq, Italy, Jamaica, Japan, Kenya, Madagascar, Madeira, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nicaragua, Niger, Nigeria, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, St Thomas, Samoa, Senegal, Sierra Leone, South Africa, Sri Lanka, Sudan, Surinam, Taiwan, Tanzania, Thailand, Togo, Trinidad and Tobago, Uganda, Uruguay, USA, Venezuela, Vietnam, Zimbabwe.

Sett and stem rot

Argentina, Australia, Barbados, Brazil, Burkina Faso, Côte d'Ivoire, Cuba, Dominican Republic, Fiji, Guadeloupe, Guyana, Hawaii, India, Indonesia, Madagascar, Malaysia, Mauritius, Mexico, Morocco, Nicaragua, Pakistan, Panama, Peru, Philippines, Puerto Rico, Senegal, South Africa, Thailand, Uganda, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

Pokkah boeng

The most obvious symptom of pokkah boeng is a malformed or twisted top, which gives this disease its name from a Javanese language (Martin *et al.,*1961) (Figures 1 and 2). Symptoms develop during rainy periods when growth is rapid. Initially, young leaves are chlorotic at their base and patchy elsewhere on the blade (Figure 3). Chlorosis is most obvious on the lower surface of the leaf, where white mycelium may be evident. Affected leaves tend to be narrow at the base (Figures 4 and 5). Development of further symptoms is dependent on the susceptibility of the variety and on environmental conditions conducive to the pathogen. Young leaves may become infected in the spindle, resulting in pronounced wrinkling, twisting and shortening of the leaves. As leaves mature, irregular reddish stripes and specks develop within the chlorotic areas. Ladder-like, necrotic lesions may develop on leaves, leaf sheaths and midribs. Infection in the spindle may reach the growing point and continue into the stalk (Figure 6). Dark, reddish streaks and ladder-like, necrotic lesions may form either inside the stalk or on the rind. In some cases, lesions may penetrate across much of the stalk to give the appearance of a horizontal knife-cut (Figures 7 and 8).



Figure 1. Twisted top of a sugarcane stalk (P. Rott).



Figure 2. Twisted and chlorotic leaves at the top of a sugarcane stalk (G.P. Rao).



Figure 3. Chlorosis and necrosis of leaves affected by pokkah boeng (P. Rott).



Figure 4. Narrow and deformed chlorotic leaf base (P. Rott).



Figure 5. Deformed and necrotic leaf base (P. Rott).



Figure 6. Stalk malformation caused by pokkah boeng (P. Rott).



Figure 7. Stalks with necrotic lesions on the rind and horizontal knife-cut lesions (P. Rott).



Figure 8. Stalk broken at knife-cut lesion caused by the pathogen (P. Rott).

Most infected shoots grow away from the symptoms and recover, but sometimes the growing point is killed (top rot), resulting in shooting of the lateral buds.

Sett rot

Fusarium sett rot causes a purplish red discoloration of the internal tissues of sugarcane setts starting at the cut ends or from points of damage on the rind of the setts (Bourne, 1961) (Figure 9). The infection spreads more rapidly in the vascular bundles which are a brighter purple-red than the surrounding tissues. The buds of infected setts fail to germinate and the roots turn brown-red and rapidly die. Fusarium sett rot can be differentiated from pineapple sett rot (formerly pineapple disease) by the lack of the characteristic odor of over-ripe pineapples produced by pineapple sett rot and the more intense red coloring of the tissues.

Stem rot

In mature stalks of standing cane, infection is often associated with damage from borers or other stalk injury. A purple-red discoloration of the internal tissues similar to that seen in setts occurs and the disease extends through the nodes in the vascular bundles (Figure 9). Leaves on infected stalks wilt, turn yellow and die (Figure 10). This form of the disease has been associated with the disease known as wilt in India (Viswanathan *et al*., 2023).

Diagnosis

The diseases are diagnosed by examination for the symptoms and isolation of *Fusarium* species. The fungi can be easily isolated on simple nutrient agars such as potato dextrose agar or corn meal agar. Species of the FFSC exhibit great variation in morphology and cultural characteristics and cannot be identified based on these features.

Fusarium sacchari produces microconidia in the aerial mycelium only on polyphialides and are 0–1-septate, measuring 5–12.5 × 2.5–5 µm. Macroconidia are produced in the sporodochia and are mostly 3-septate or occasionally 5-septate (measuring 20–42.5 × 2.5–5 µm) with the presence of a poorly developed foot cell (Costa *et al.,* 2019). *Fusarium sacchari* produces black perithecia, which are ovoid and warty, measuring 290–380 × 250–380 µm and the ascospore cirrhi are orange in color. Asci are clavate, with eight ascospores per ascus. Ascospores are ellipsoid, somewhat irregular, smooth, with one to three septa and measuring 15–20 × 5–7.5 µm. Other species within the complex produce spores similar to *Fusarium sacchari* which is presented here as an example.

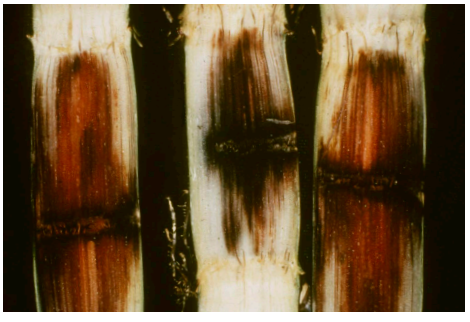

Figure 9. Longitudinally cut sugarcane sett showing purplish-red discoloration of the internal tissues due to sett rot (ISSCT).



Figure 10. Sugarcane field severely affected by stem rot (Y. Huang).

PCR assays and DNA sequencing have been used to identify the various *Fusarium* species associated with sugarcane (Costa *et al.,* 2019; Govender *et al*., 2010; Lin *et al*., 2014; Siti Nordahliawate *et al*., 2008). The translation elongation factor-1a gene (EF-1a) can be amplified with primer pairs EF-1/EF-2 or EF-3/EF-22 (O'Donnell, 2000; O'Donnell *et al*., 1998 and 2008). Fragments of the second largest subunit of RNA polymerase II (RPB2) can be obtained with primers 5F2 (Reeb *et al*., 2004) and 7cR (Liu *et al*., 1999).

Strains of the pathogen

The *F. fujikuroi* complex includes at least 50 phylogenetic variants or species that are in some cases sexually compatible forming hybrids. At least seven species within the complex have been reported from sugarcane.

Transmission

Fusarium species can grow saprophytically on decaying plant debris and spores are spread by wind and rain. The fungi can be carried on the outside of stalk pieces or within the stalk and in contaminated soil. Volatile emissions from *F. verticillioides* attract caterpillars of the sugarcane borer *Diatraea saccharalis* and the fungus is transmitted vertically to the insect's offspring (Franco *et al*., 2021). Consequently, when *D. saccharalis* continues its cycle, it disseminates *F. verticillioides* to healthy plants. Several *Fusarium* isolates infecting sugarcane were found beneficial to the stalk borer *Eldana saccharina* but their putative spread by this borer species remains to be investigated (McFarlane *et al*., 2009).

Host range

Fusarium species have a wide host range on both monocotyledons and dicotyledons. They cause a range of diseases in these hosts (Booth, 1971).

Epidemiology

Pokkah boeng

Pokkah boeng is favored by warm, humid growing conditions, with symptom development typically beginning early in the rainy season during rapid and vigorous growth periods (Vishwakarma *et al*., 2019). Conidia are carried by water to the base of the spindle where they germinate (van Dillewijn, 1950). The fungus penetrates the soft cuticle of the young leaves and gradually breaks down the leaf tissue. It grows through the vascular bundles from the leaves to the stem, then moves to the surrounding tissues. Cane is most susceptible to infection when 3 to 7-months-old (van Dillewijn, 1950). In India, the disease incidence can be aggravated by *Phenacoccus saccharifolii*, the crown mealybug (Siva *et al*., 2025).

Sett and stem rot

Fusarium stem rot is associated with standing cane suffering stress, such as waterlogging or drought, or damage by boring insects. The sett rot is associated with insect or mechanically damaged setts planted under adverse conditions. The damage caused by chopper harvesters, when they are used to cut setts for planting, can predispose the setts to infection from Fusarium sett rot.

Economic importance

Pokkah boeng

While outbreaks of pokkah boeng can appear spectacular, they usually are of little economic importance. Usually, not all stalks in a stool are affected and the affected stalks recover and resume normal growth with little effect on final yield. In severe cases, some stalks may die but unaffected stalks in the stool compensate for any losses from the dead stalks. Serious epidemics have taken place in very susceptible varieties in some countries (Figure 11). In Java, up to 38% of affected stalks of POJ 2878 died (Martin *et al*., 1989). Pokkah boeng has been reported to have increased in severity in India in recent years (Vishwakarma *et al*., 2019; Viswanathan *et al*., 2023). Pokkah boeng (twisted top) in association with the crown mealy bug, *Phenacoccus saccharifolii,* has been reported to cause total crop loss in some cases in India from 2020 to 2022 (Viswanathan *et al*., 2023).



Figure 11. Sugarcane field severely affected by pokkah boeng: numerous stalks broken at knife-cut lesion at the stalk bottom (P. Rott).

Sett and stem rot

Fusarium sett and stem rot has been considered to cause significant yield losses in the past but there have been few recent reports (Egan *et al*., 1997). The disease has been associated with extensive damage in association with red rot caused by *Colletotrichum falcatum* Went (Syn. *Glomerella tucumanensis*) in Thailand (Anonymous, 1991).

Control

Pokkah boeng

Resistant varieties have been used to control pokkah boeng. Clones can be screened for resistance to pokkah boeng by injecting a conidial suspension of the fungus into the leaf spindle 10 cm below the highest visible leaf joint (Han Lioe Hong, 1956). Resistance is highly heritable (Lyrene *et al*., 1977). Varieties were rated for resistance to pokkah boeng in replicated field trials by recording incidence and severity over a crop cycle in China (Xu *et al*., 2023).

Applications of pesticides such as carbendazim and imidacloprid can reduce yield losses caused by pokkah boeng and the crown mealy bug (Viswanathan *et al*., 2023).

Endophytic fungi with biocontrol effects were associated with varieties resistant to pokkah boeng in China (Xiao *et al.*, 2024). Mycoviruses infecting *F. sacchari* and *F. andiyazi* causing pokkah boeng in China were identified as potential biocontrol agents (Yao *et al.*, 2020).

Sett and stem rot

The most effective control of Fusarium stem rot is to avoid stress by improving drainage and irrigation and to reduce damage from stem borers (Bourne, 1961). Where this is not possible, resistant varieties should be grown. The impact of Fusarium sett rot can be reduced by selecting well-grown planting material with no insect damage. Mechanical equipment used to cut and handle setts for planting should be well maintained and set to cut setts with at least two eyes and with minimal damage. Mechanical harvesters used solely by planting contractors can reduce damage by using rubber coated feed rollers. Mercurial fungicides were reported to control Fusarium sett rot but there has been no study of the effectiveness of more recently developed fungicides.

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Leaf blight

W.H. Hsieh, J.C. Comstock, revised by Sushma Sood

Cause

Saccharicola taiwanensis (W.Y. Yen & C.C. Chi) O.E. Erikss. & D. Hawksw; synonym = *Leptosphaeria taiwanensis* W.Y. Yen & C.C. Chi (Eriksson and Hawksworth, 2003). Anamorph stage known as *Stagonospora tainanensis* W.H. Hsieh.

Geographical distribution

China, India, Japan, Philippines, Taiwan.

Symptoms

Mature leaf blight lesions are elongate, 1–50 mm long, and 1–3 mm wide (Figures 1 and 2). Lesion size decreases as the number of lesions on the leaf increases. The color of the lesions varies from bright red, reddish-brown, light yellow, and light yellow with a reddish tint depending on the variety. Often, multiple lesions coalesce, forming wide bands of infected tissue. When necrotic tissues dry out, they appear reddish-brown from a distance (Figure 3). Lesions may also occur on leaf sheaths (Yen, 1964; Yen and Chi, 1953).



Figure 1. Elongated leaf blight lesions (J.C. Comstock).



Figure 2. Leaf with numerous elongated lesions (J.C. Comstock).



Figure 3. Sugarcane field severely affected by leaf blight (ISSCT).

Diagnosis

Leaf blight is diagnosed by its symptoms and the presence of either perithecia of *Saccharicola taiwanensis* or pycnidia of *Stagonospora tainanensis* (Hsieh, 1979). Perithecia are scattered, hypophyllous or amphigenous, spherical to ovoid, dark brown, glabrous, 80–178 × 80–146 µm. Asci are hyaline to light brown, ovoid, straight or slightly curved, 62–115 × 21–33 µm, 8-spored. Ascospores are oblong-fusiform, slightly curved, usually 3-septate, constricted at septa, dark brown at maturity, 39–46 × 6.6–12.5 µm. Paraphyses are unbranched, hyaline, filiform, and similar in length to asci. Pycnidia are globose to sub-globose, ostiolate, membranous, 88–192 × 70–120 µm. A pycnidium can contain two forms of conidia. The first is a *"Stagonospora"* form, hyaline, straight to slightly curved, with 1–3 septa, 28–40 × 11.6–16.0 µm, containing 3–8 cytoplasmic oil droplets. The second is a *"Phoma"* form that is single-celled, hyaline, elliptical to cylindrical, straight to slightly curved, 4.4–13.0 × 2.5 µm, containing 1–2 oil droplets (Hsieh, 1979). The pathogen can also be identified by amplification and sequencing of the ITS and TEF-1α regions of the fungus (Huang *et al*., 2023). The genome sequence of *Stagonospora tainanensis* became recently available, thus providing a useful resource for the development of new and specific molecular diagnostic assays (Xu *et al*., 2022).

Strains of the pathogen

No information is available.

Transmission

Leaf blight is spread primarily by conidia and to a lesser extent, by asci.

Host range

The main hosts are *Saccharum* species and *Saccharum hybrids* (Leu *et al*., 1974). *Miscanthus* may be a host since *Miscanthus* blight described in the USA has either the same or a closely related causal agent (O'Neill and Farr, 1996).

Epidemiology

Leaf blight is favored by humid and wet conditions. In Taiwan, the disease occurred all year round in the high rainfall east coast area but only at times of high rainfall in southern Taiwan (Yen, 1964; Leu and Hsieh, 1970).

Economic importance

Leaf blight has been an economic concern in Taiwan, causing a loss of cane tonnage and sucrose in susceptible varieties.

Control

Planting resistant varieties is recommended to control leaf blight in sugarcane. Varieties can be screened in the field and the greenhouse using pycniospores (Leu *et al*., 1976; Leu and Hsieh, 1971). Ratings for both tests were similar to results obtained under natural infection. Six quantitative trait loci (QTLs) have been associated with leaf blight resistance, and seven pathogen receptor genes were proposed as candidate genes for resistance to the disease (Wang *et a*l., 2021 and 2022a). Candidate genetic markers for resistance to leaf blight in sugarcane were also identified using a bulk-segregant analysis based on a sequencing strategy (Wang *et al*., 2022b)

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Leaf scorch

Ching-Shiou Lee, Yaw-Guang Liang, revised by Andre B. Gama

Cause

Two different pathogens cause similar symptoms: *Stagonospora sacchari* T.T. Lo & L. Ling, deuteromycete fungus, and *Stagonospora bicolor* (D. Hawksw., W.J. Kaiser & Ndimande) Kaz. Tanaka & K. Hiray. (Syn. *Leptosphaeria bicolor* D. Hawksw., W.J. Kaiser & Ndimande), ascomycete fungus. However, there is controversy regarding the classification of *Stagonospora sacchari* based on the morphological structures described by Sivanesan (1983), and this fungal species possibly belongs to the genus *Parastagonospora* (Zhao *et al*., 2021).

Geographical distribution

Argentina, Bangladesh, Cuba, Fiji, Guatemala, India, Indonesia, Japan, Kenya, Nigeria, Panama, Philippines, South Africa, Taiwan, Thailand, Venezuela, Vietnam.

Stagonospora sacchari caused epidemics in the Philippines and in Taiwan. *Stagonospora bicolor* has only been reported in Kenya (Kaiser *et al*., 1979).

Symptoms

Symptoms occur mostly on the leaf blades below the second or third unrolled top leaves (Figures 1 to 3). Young lesions are spindle-shaped, very small, red or reddish-brown spots with a definite yellowish halo. Gradually, these lesions elongate along the veins, coalesce and eventually become straw-colored bordered by a reddish margin. Mature lesions are 5–20 cm long and 0.5–1 cm wide, extending towards the tip of the leaf. In severe cases, the entire leaf surface, except the midrib which remains intact, is blighted. On the older lesions, pycnidia of *Stagonospora* appear in the dead leaf tissues. Variety and environmental conditions both influence symptom development. Streaks take from 3 to 5 weeks to develop fully. Under field conditions, scorching is more pronounced on the lower older leaves where two or more lesions coalesce to form large patches of dead tissues. Infected leaves dry prematurely appearing similar to plants suffering from drought.

Diagnosis

Diagnosis requires observation and accurate identification of either the pycnidia of *S. sacchari* or the perithecia of *S. bicolor*. Pycnidia of *S. sacchari* are immersed mainly on the upper surface of leaves and are either epiphyllous, hypophyllous or amphigenous. They are sub-spherical to spherical, dark brown, 150–228 µm in diameter, with a membranous wall and 13.7–17.1 µm thick (Lo, 1961). The ostiole is slightly raised and protruding. Conidia are hyaline, ellipsoid, with tapered apex, and round or somewhat truncate base

(Figure 4). They are straight or slightly curved measuring 38.5–51.5 × 9.8–22.1 µm. They are generally triseptate, rarely 1- or 4-septate, and constricted at the septa. Mature conidia contain 1–2 oil drops in each cell (Lo and Leu, 1989).

Strains of the pathogen

The existence of strains is suspected in *S. sacchari* because six fungal isolates collected from distinct regions in the Philippines had different virulence patterns in tested varieties (Sampang and Reyes, 1980).

Transmission

Stagonospora sacchari is transmitted by rain or dew accompanied by wind. There is no transmission through cuttings.



Figure 1. Elongated leaf scorch lesions (B.J. Croft).



Figure 3. Drying of leaves due to leaf scorch (L. Putra).



Figure 2. Leaf with different sizes of leaf scorch lesions (B.J. Croft).



Figure 4. Conidia of *Stagonospora sacchari* (C.-S. Lee).

Host range

Besides sugarcane, *S. sacchari* occurs naturally on *Miscanthus sinensis* and *M. floridulus* (= *M. japonicus*) in Taiwan, and on *Saccharum spontaneum* in the Philippines (Lo and Leu, 1989). In artificial inoculation tests, *Sorghum bicolor* (= *S. vulgare*), *Andropogon sorghum*, *A. sorghum* var. *vulgaris*, *Imperata cylindrica*, *Sorghum halepense* and *Pennisetum purpureum* were infected. Although *M. sinensis* is not a natural host of leaf scorch in the Philippines, it could be infected by artificial inoculation.

Epidemiology

The occurrence of leaf scorch disease is closely correlated to rain and drought. In Taiwan, the disease spreads rapidly after rain, especially during the summer season when high temperatures and heavy rains accelerate the dissemination and favor the development of the pathogen. In the drier autumn, infected plants show typical and severe scorch symptoms.

Economic importance

Sugarcane losses due to leaf scorch vary according to weather conditions and varieties (Lin, 1952). In a comparative study, diseased plants with only four green leaves of variety Co 290 showed a 17% loss in tonnage and a 13% reduction in sugar yield compared to healthy plants. The sugar content of an H37-1933 crop in the Philippines was substantially reduced, and sugar losses were estimated at 10–30% (Exconde, 1963). The highly susceptible variety Phil6111 in the Philippines suffered approximately 25% losses in tonnage and sugar/hectare (Sampang and Reyes, 1980). In Indonesia, leaf scorch caused 36.5% and 16.8% sugar losses in SP70-1284 when the plants were infected at the age of 5 and 8 months, respectively (Suranto and Harsanto, 1989). The economic impact of leaf scorch disease to sugarcane may differ between years. In Guatemala, leaf scorch was unimportant until recent years, but the disease hampers varietal screening in the breeding program and significantly affects released varieties (Ovalle and Viswanathan 2020).

Control

Varietal resistance is the primary method of leaf scorch control. Fungicide application has proved to reduce the disease, but has not been used in commercial fields. Removal/management of alternate hosts may be effective. Methods for screening varieties for resistance to *S. sacchari* have been developed and are still being used in Taiwan.

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Marasmius basal stem, root and sheath rot

Barry J. Croft

Cause

Marasmius sacchari Wakker and *Collybiopsis stenophylla* (Mont.) R.H. Petersen (syn. *Marasmiellus stenophyllus*), basidiomycete fungi.

These two fungal species are also involved in a disease called Marasmius sheath rot and shoot blight (see "Marasmius sheath rot and shoot blight; root rot", p. 294).

Geographical distribution

Antigua and Barbuda, Australia, Barbados, Brazil, Colombia, Cuba, Dominican Republic, Fiji, Grenada, Guadeloupe, Guyana, Haiti, Hawaii, India, Indonesia, Jamaica, Japan, Martinique, Mauritius, Mexico, Myanmar, Panama, Paraguay, Peru, Philippines, Puerto Rico, St Kitts and Nevis, St Lucia, St Vincent, South Africa, Taiwan, Thailand, Trinidad and Tobago, Uganda, USA, Zimbabwe.

Symptoms

Marasmius basal stem, root and sheath rot is usually associated with sugarcane plants growing poorly because of environmental or nutritional stresses, or are affected by other pathogens (Cook, 1932). The symptoms of the other disorders can be confused with those caused by this weak pathogen. Marasmius rot is characterized by the growth of white mycelium at the base of the sheaths (Figure 1), and between the sheath and the stalk. The fungus glues the sheaths to each other and to the stalk. Affected shoots can develop red freckling on the leaves. Above-ground sections of the stalk are not affected. Parts of the stalks and shoots below the soil can develop brown cankers at the nodes. The cankers do not penetrate much below the rind. The growth of the roots at these nodes is hindered.



Figure 1. Growth of white mycelium at the base of sheaths (J. Hoy).

When there is high humidity, the fruiting bodies or basidiocarp of the fungus develop at the base of the stalks at soil level (Figure 2).



Figure 2. Fruiting bodies of the pathogen at the base of stalks (A: J. Hoy; B: S. Matsuoka).

Diagnosis

The disease is diagnosed by visual symptoms and the presence of the mushroom fruiting structures at the base of the affected leaf sheaths at soil level. The spores of *M. sacchari* are irregularly elongate, slightly curved, larger at one end, and 4–5 × 16–20 µm. Spores of *M. stenospilus* are elongate 5–6 × 7–9 µm (Rands and Abbott, 1964; Sivanesan and Waller, 1986).

Strains of the pathogen

Other *Marasmius* species and subspecies have been reported associated with this disease (Cook, 1932).

Transmission

Transmission is by mycelial strands and soil-borne spores.

Host range

Marasmius stalk and root rot has been reported on maize (*Zea mays*) in Australia (Pont 1973).

Epidemiology

Marasmius spp. live as saprophytes on dead organic matter and only attack plants which are weakened by disease or adverse environmental conditions.

Economic importance

The disease was thought to be important in the noble canes, *Saccharum officinarum*, but the disease was often associated with other conditions, which were more likely to be the primary cause of the poor growth. Since the culture of resistant interspecific hybrids, the disease is of no economic importance.

Control

This disease can be controlled by correcting environmental and nutritional stresses and controlling other diseases.

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Orange rust

Robert C. Magarey, revised by Robert C. Magarey and Sushma Sood

Cause

Puccinia kuehnii (W. Krüger) E.J. Butler, basidiomycete fungus.

Geographical distribution

American Samoa, Argentina, Australia, Belize, Bolivia, Brazil, Cameroon, China, Colombia, Costa Rica, Côte d'Ivoire, Cuba, Dominican Republic, Ecuador, El Salvador, Fiji, French Polynesia (Tahiti), Gabon, Guam, Guatemala, Guyana, India, Indonesia, Jamaica, Japan, Kenya, Malaysia, Mauritius, Mexico, Myanmar, New Caledonia, Nicaragua, Pakistan, Panama, Papua New Guinea, Philippines, Réunion, Samoa, Solomon Islands, Sri Lanka, South Africa, Taiwan, Thailand, USA (Louisiana, Florida, Texas), Vietnam.

Symptoms

Rust diseases are characterized by leaf lesions called uredinia or pustules that, at maturity, break through the leaf epidermis, giving the surface a rough appearance. The two most frequently encountered and widely distributed sugarcane rust diseases, orange rust and brown rust (caused by *Puccinia melanocephala*) may be readily distinguished. The third one, tawny rust caused by *Macruropyxis fulva*, has only been reported in the southern part of Africa (Martin *et al*., 2017). Brown rust pustules are reddish-brown to dark brown, orange rust pustules are orange, and tawny rust pustules are bright orange to orangish-red (Rutherford, 2018).

The initial symptoms of orange rust are minute, elongated yellow spots which take on a pale yellow-green halo as they increase in size (Ryan and Egan, 1989). As the lesion grows, an orange to orange-brown color develops depending on the cane variety (Figures 1 to 5). Unlike brown rust, orange rust lesions are never dark brown. Pustules of orange rust tend to occur 'in groups' on the affected leaf surface, with most pustules on the lower surface (Figure 6). While leaf area affected by brown rust pustules is greater in the mid- and distal portions of the leaf, orange rust pustules may occur towards the base of the leaf as well – more so than with brown rust. Symptoms of orange rust are more prevalent in semi-mature to mature sugarcane, and the disease is favored by hot humid weather.

Diagnosis

Orange rust is identified either by symptomatology or by characteristics of the urediniospores and teliospores (Figures 7 to 10). RGB images of infected leaves generated by object-based image analysis segmentation can be used to identify the brown and orange rusts symptoms automatically (Ordine Pires da Silva Simões *et al*., 2023). Pustule color, lesion aggregation (or not) and urediniospore shape and color are the defining



Figure 1. Leaves showing whitish teliospore lesions (left) and orange urediniospore lesions (right) (W.H. Hsieh).



Figure 4. Sugarcane leaves severely affected by orange rust (P. Rott).



Figure 2. Orange rust lesions on the upper side of a sugarcane leaf (P. Rott).



Figure 3. Leaf with numerous orange rust pustules (uredinia) on the lower side of a leaf, including the midrib (P. Rott).



Figure 5. Inside view of a sugarcane field affected by orange rust (P. Rott).



Figure 6. Clusters of oranges rust lesions on the mid to lower part of sugarcane leaves (P. Rott).

parameters. The urediniospores of *P. kuehnii* have an apical thickening and are paler in color, with generally few paraphyses. Teliospores are not abundant. Urediniospores of *P. melanocephala* are smaller, with a brown coloration, more prominent pores with no apical thickening, abundant paraphyses and teliospores are common. The surface features of the urediniospores are also different from those of *P. melanocephala* having regularly placed spines 1.0–1.5 µm apart, while in *P. kuehnii* the spines are spaced further apart and are irregularly spaced (Rutherford, 2018).



Figure 7. Leaf showing teliosori containing teliospores (W.H. Hsieh).



Figure 8. Teliospores of *Puccinia kuehnii* (W.H. Hsieh).

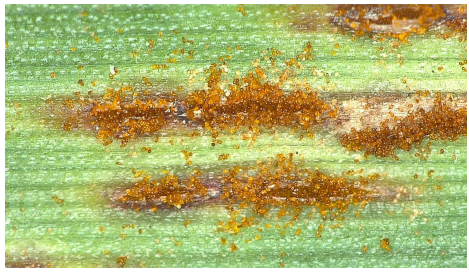
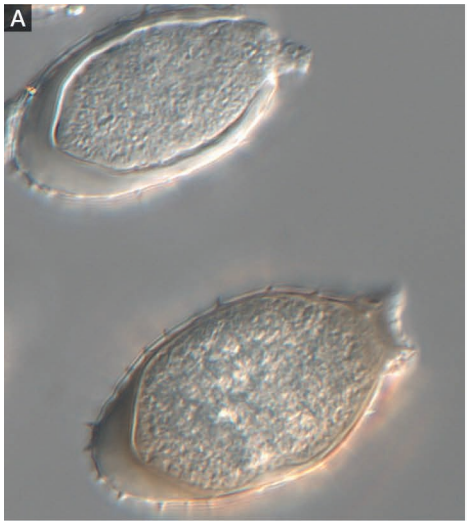


Figure 9. Leaf showing pustules (uredinia) releasing urediniospores (S. Sood).



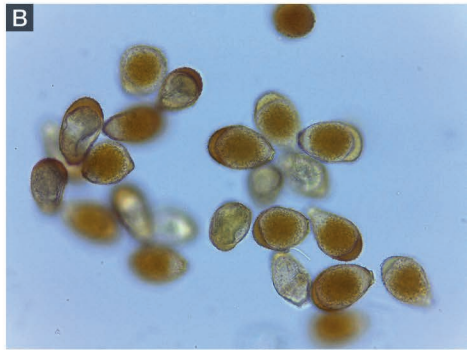


Figure 10. Urediniospores of *Puccinia kuehnii* showing thickened apices (A: L. Castlebury; B: E.A. Rincon).

Orange rust can be diagnosed by conventional polymerase chain reaction (PCR) and real-time PCR (qPCR) using different primers amplifying the internal transcribed spacer (ITS) region of the ribosomal DNA (Glynn *et al*., 2010). PCR with primer pair Pk1F/Pk1R results in the production of an amplicon of 527 bp. A 142 bp product is obtained by qPCR with the Pk2F/Pk2R primer pair while PIRA-PCR produces a 136 bp amplicon using primers pair Pk-PIRAF/Pk-PIRAR. DNA products from conventional PCR and qPCR must be sequenced to confirm the identity of *P. kuehnii* and its isolates (Cárdenas *et al*., 2024). Primer pair PkPmF/PkPmR can be used to detect brown and orange rust pathogens. These primers produce a 585 bp fragment for brown rust and a 606 bp fragment for orange rust under conventional PCR conditions (Glynn *et al*., 2010).

Strains of the pathogen

The development of a new strain of *P. kuehnii* is believed to be the reason for the very significant orange rust epidemic in Australia in 2000 and the subsequent epidemics in North, Central and South America, Africa and other countries. Several varieties that had been resistant to the disease, and grown very extensively, became susceptible within a single season, with large yield losses resulting.

DNA sequence data were obtained from three rDNA regions to determine the genetic relationships amongst several groups of *P. kuehnii* isolates (Braithwaite *et al*., 2009). The first collection comprised recent Australian field crop isolates, and limited sequence variation was detected within this population. In the second study, Australian isolates were compared with isolates from Papua New Guinea, Indonesia, China, and historical herbarium collections. Phylogenetic analyses grouped the isolates into three clades. All isolates from commercial cane fields, including the recent Australian commercial crop isolates and the Australian historical isolate from 1898, clustered together in one clade. The other two clades included orange rust isolates from wild and garden canes in Indonesia and Papua New Guinea. All specimens appeared morphologically similar. Those characterized from wild and garden canes potentially pose a risk to currently resistant varieties in countries around the world.

Several commercial sugarcane varieties were susceptible to orange rust in Florida in 2007 when the disease was first reported in the Western Hemisphere. However, variety CP89-2143 was resistant to orange rust until 2011-2012, thus indicating a resistance breakdown. The occurrence of at least two races of *P. kuehnii* in Florida was demonstrated based on inoculation studies and variation in virulence of two isolates of the pathogen collected from CL85-1040 and CP89-2143 (Sanjel *et al*., 2021).

Transmission

Rust fungi are usually transmitted by wind and water splash, and orange rust is no exception. Sreeramulu and Vittal (1970) monitored urediniospores above an orange rust susceptible variety and concluded that spore numbers were greatest on dry rather than wet days, and incidence peaked in the middle of the day (10h00–14h00).

In the Australian orange rust epidemic of 2000-2001, masses of urediniospores were produced on the susceptible variety Q124, so much so that machinery moving through crops became covered in an orange film (spores) while clothes on those walking through diseased crops and even house verandas also turned orange.

Host range

Puccinia kuehnii has been reported to infect *Saccharum spontaneum*, *S. officinarum*, *S. robustum*, *S. edule*, commercial hybrid varieties, *Erianthus arundinaceus* and *Sclerostachya fuscum* by Butler (1918). Research in Australia showed that although some major sugarcane varieties became susceptible after the pathogen strain change, most commercial varieties were resistant to the disease.

Epidemiology

Wind and large air masses readily spread the pathogen. It only took around 18 months for the Queensland sugarcane industry to be affected by the new strain of *P. kuehnii* after it was first detected in the central (Mackay) cane-growing region. Environmental conditions favoring disease development in Australia include warm humid summer, and cool autumn (fall), conditions (Staier *et al*., 2004). In Florida, orange rust-conducive conditions include high humidity at night and temperatures between 20 and 22°C. Accumulation of hours with these temperatures between 20h00 and 8h00 is a key predictor for rust severity (Chaulagain *et al*., 2020).

Economic importance

The disease caused one of the greatest single-year yield losses of any disease in the history of the Australian sugarcane industry. It wasn't until crop variety transition occurred that losses were minimised. With adequate screening of new clones for disease resistance in the plant improvement program, yield losses in the Australian industry have been minimised.

Orange rust also significantly reduced commercial yields in many other countries around the world following the Australian epidemic, especially after its spread to the Americas.

Control

The main control measure for the disease is varietal resistance. There is generally a significant level of resistance amongst commercial Australian and the USA hybrid varieties; commercial management of the disease is relatively straightforward through appropriate variety selection. However, the genetics of the orange rust resistance have not been fully explored. Several studies have identified candidate genes and quantitative trait loci associated with resistance to orange rust (Dijoux *et al*., 2024; Yang *et al*., 2018 and 2019) and shown that certain resistant alleles are dominant in *Saccharum spontaneum* (Dijoux *et al*., 2024). The molecular marker G1, linked to an orange rust resistance gene, has been used in breeding programs predicting resistance with 73% accuracy (Fier *et al*., 2020).

Fungicide management has been researched extensively in Australia (Magarey *et al*., 2001; Staier *et al*., 2003) and in the USA (Chaulagain *et al*., 2019). Australian research aimed to provide an economic option for farmers to transition from susceptible to more resistant varieties. The approach was of doubtful economic gain, given the cost of fungicides and the yield responses elicited. Efficacious fungicides were identified (cyproconazole, tebuconazole, mancozeb) which provided reasonable control with two strategically timed applications (Staier *et al*., 2003). Early crop fungicide applications provided the greatest benefit. Disease modelling coupled with weather predictions were used to provide farmers with a guide as to when to spray (Staier *et al*., 2004).

More extensive research into fungicides has been conducted in Florida (Chaulagain *et al*., 2019; Raid, 2010) due to the breakdown of resistance in some commercial varieties (thought to be due to strain development). Three to four consecutive fungicide applications separated by three to four weeks with strobilurins (pyraclostrobin and azoxystrobin) are most effective in controlling orange rust, followed by triazoles (metconazole and propiconazole). They found that early crop applications (when the first visible dewlap leaf area affected was <10%) provided effective disease management and sound economic returns. In 2023, orange rust resistant varieties occupied most of the sugarcane acreage in Florida. The release of sugarcane varieties resistant to orange rust contributes to reducing fungicide applications in commercial sugarcane production in Florida (Cárdenas *et al*., 2024).

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Pachymetra root rot

Barry J. Croft, Alison S. Jensen

This chapter has been updated and rewritten based on the original chapter entitled "Pachymetra root rot" and authored by Barry J. Croft and Robert C. Magarey in 2000.

Cause

Pachymetra chaunorhiza B.J. Croft & M.W. Dick (Dick *et al*., 1989), oomycete. *P. chaunorhiza* is a member of the Verrucalvaceae in the order Saprolegniales.

Geographical distribution

Australia.

The disease is unique to Australian cane fields and has never been observed elsewhere in the world (Magarey *et al*., 2013). It has been detected at low levels in a few native forest areas and may have originated as a minor pathogen of plant species within this vegetation type (Jensen, 2020). In Queensland, the disease is widespread in northern, Herbert, central, Bundaberg and Maryborough districts and affects sugarcane growing on some soil types in the Burdekin and New South Wales.

Symptoms

Pachymetra root rot is characterized by extensive rotting of the larger roots of the sugarcane plant, leading to poor root system development and stunted plant growth.

The initial symptoms of Pachymetra root rot are a water-soaking of the affected root, usually just behind the root tip in primary and larger secondary roots (Magarey, 1986) (Figure 1). This symptom rapidly develops into a soft flaccid rot. In advanced stages, affected roots are held intact only by the root epidermis, all other root structures having disintegrated (Figure 2). Root reddening, up to 2.0 mm long encircling the root, often accompanies Pachymetra root rot infection. In many instances, this discoloration indicates the limit of infection, though on occasions, infection progresses beyond this discoloration and along the whole root length. Where high prevalence of the disease occurs in susceptible varieties, 80–90% of the roots may be rotted leaving a very poor and debilitated root system (Figure 3). Such symptoms result in reduced yield, a loss in stool anchorage with consequent stool tipping, failed ratooning and increased soil levels in the cane supplied to the sugar factory.

Symptoms differ from Pythium root rot in that Pachymetra mainly affects the larger roots, is favored by higher soil temperatures (25–30°C), has ornamented large oogonia and does not produce sporangia/zoospores.

Reduced root function leads to reduced plant height, a susceptibility to water stress and general unthrifty growth.



Figure 1. Early symptoms of Pachymetra root rot (R. Magarey).



Figure 2. Complete rotting of the root tip region (R. Magarey).



Figure 3. Pachymetra root rot symptoms: roots mostly rotted with few healthy (white) primary roots (R. Magarey).



Figure 4. Oogonia of the pathogen (up to 63 µm) with characteristic large blunt projections (R. Magarey).

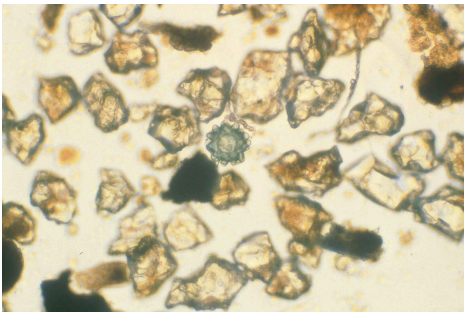

Figure 5. Blue-stained oospore of the fungus in soil extract (R. Magarey).

Diagnosis

Diagnosis of the disease relies on observing the characteristic rotted primary roots and the observation, under a microscope, of the distinctive oospores located in these roots (Croft and Magarey, 1984). Oospores are relatively large (25–65 µm in diameter) and have large blunt projections on the oogonial wall, making them larger and distinctive compared to the various *Pythium* species that infect sugarcane (Figure 4). Staining of oospores within roots, with stains such as lactophenol cotton blue, is helpful, but not normally required for diagnosis.

An assay has been developed for assessing *P. chaunorhiza* inoculum density in field soil (Magarey, 1989a and 1989b). Wet sieving extraction of the oospores from soil, bleaching of the collected material and semi-selective staining of the spores is the basis for the assay. Oospores are counted under a microscope at x60 magnification (Figure 5).

The internal transcribed spacer (ITS) sequence has been obtained for 10 *P. chaunorhiza* isolates originating from representative sugarcane districts affected by the root disease (Magarey and McKay, 2018). DNA detection assays were developed and primers specific to *P. chaunorhiza* were designed, including a forward primer, a reverse primer, and a TaqMan MGB Probe[14]. All tested isolates of the pathogen were detected by qPCR with Ct (cycle threshold) values ranging between 15.7 and 17.9.

Strains of the pathogen

Some evidence suggests there is slight pathogen variation across Queensland. Molecular research highlighted slight differences between two groups of isolates: one group is largely from the central and southern parts of Queensland while the other comes mostly from northern Queensland (though not exclusively). Pathogenicity testing failed to identify a significant variation in varietal reaction between the two isolate groups, but small differences in the resistance of a few varieties have been noted. No sequence variation of the ITS region of the genome was observed amongst the 10 isolates used to develop molecular diagnostic assays (Magarey and McKay, 2018).

Transmission

Pachymetra chaunorhiza does not produce zoospores and therefore has no motile spore stage (Magarey, 1991). Disease incidence in previously disease-free fields relies either on the build-up of inoculum from a very low natural level (native vegetation) or the transfer of soil on implements and machinery. Build-up of the disease in fields occurs through the production and deposition of oospores produced in rotted roots immediately beneath the sugarcane stool. Inoculum may rise quickly, particularly in the planting line beneath diseased stools, when susceptible varieties are grown (Magarey and Mewing, 1994).

Host range

Several potential native plant grass species that may be infected artificially by *P. chaunorhiza* have been identified (Jensen, 2020). These include *Bothriochloa macra* (red grass), *Themeda australis* (kangaroo grass) and *Imperata cylindrica* (blady grass).

14. Forward primer: 5′-TTTATGAGGCTTGTGTCTAGTTCTCTTC-3′;
reverse primer: 5′-TGACTGCTCGTTTCAATCAGTAATTATT-3′;
TaqMan MGB Probe: FAM 5′-CGAAACCTTCTGCTAG-3′ (reverse complementary orientation).

It is likely that *P. chaunorhiza* is a pathogen of a native Australian grass which is also able to infect the introduced sugarcane (Jensen, 2020). *P. chaunorhiza* is weakly pathogenic on sorghum, *Saccharum spontaneum* and *Erianthus* spp. (Magarey and Croft, 1996).

Epidemiology

Pachymetra root rot is favored by high rainfall and a relationship between rainfall and inoculum density was found across a rainfall gradient in two varieties differing in resistance to the disease. Disease severity and soil types appear also to be correlated in some regions, with higher disease levels in lighter soils (Magarey and Soper, 1992; Magarey *et al*., 2013).

One of the most important factors governing disease severity is crop varietal resistance. The Australian commercial sugarcane germplasm contains clones with a wide range of resistance to Pachymetra root rot (Croft, 1989). Varietal resistance greatly influences soil inoculum density (Magarey, 1989a), and there is a strong relationship between root rot and inoculum density (Magarey and Mewing, 1994).

When sugarcane land was fallowed, it took five years for oospore populations to decline to 40% of the original (Magarey and Mewing, 1994). This highlights the importance of preventing the development of the disease in commercial fields. Temperature is also important and the disease is favored by soil temperatures between 25 and 30°C.

Economic importance

Pachymetra root rot may lead to major yield losses. In experiments conducted in northern Queensland, a 40% yield reduction was attributable to Pachymetra root rot in a susceptible sugarcane variety (Magarey, 1994). Smaller but significant yield losses were found in varieties of intermediate resistance (Magarey *et al*., 2003; Jensen, 2020).

Yield losses in sugarcane breeding trials have been assessed (Magarey and Bull, 2003). In selection trials at sites severely affected by Pachymetra root rot, it was found that the mean yield of the resistant clones was higher than the mean yield of the susceptible ones, reinforcing that the disease causes significant yield losses in commercial fields. Where disease levels were lower, there was no relationship between the resistance of sugarcane clones and yield.

Control

Control is centred on varietal resistance. All commercial varieties are screened for disease resistance during the Australian sugarcane plant breeding selection program. Susceptible clones are discarded based on their glasshouse-assigned resistance in screening trials (Croft, 1989; Croft and Magarey, 1989; Magarey and Croft, 1996). The crossing of parents takes into account their individual resistance ratings with a threshold set for the mean rating of the two parents; this minimises the likelihood of producing highly susceptible seedlings.

Severity of the disease in each region has been assessed in district-wide surveys using an assay based on quantification of soil oospores (Magarey, 1989b). This has highlighted regional disease priorities. The soil assay has been offered on a commercial basis for more than 25 years enabling individual farmers to assess inoculum levels (predicted disease severity) in each of their commercial fields. This information is used at planting time to select varieties with appropriate disease resistance.

Soil fumigants such as methyl bromide and metham sodium give excellent control but are not economically viable (Croft and Magarey, 1989). Biocides effective against many oomycetes do not control Pachymetra root rot. Minimum till planting of cane in the interspace gave moderate temporary yield improvement by avoiding the high soil inoculum pressure immediately beneath the previous crop planting line. This has not been adopted commercially for other agronomic reasons.

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Pineapple sett rot
(Previously known as Pineapple disease)

Jean-Claude Girard, Philippe Rott

Cause

Ceratocystis paradoxa (Dade) C. Moreau [Syn. *Thielaviopsis paradoxa* (De Seynes) Höhn.] and *Thielaviopsis ethacetica* Went [Syn. *Ceratocystis ethacetica* (Went) Mbenoun & Z.W. De Beer], ascomycete fungi.

Currently, *C. paradoxa* is considered a complex of different fungi and the taxonomic position of isolates infecting sugarcane remains to be resolved (de Beer *et al.,* 2014; Mbenoun *et al.,* 2014). As mentioned by Mbenoun *et al.* (2014): "*C. paradoxa* s. str. is far less common than previously considered. It most commonly has been confused with *C. ethacetica* which better fits the description of widespread distribution and broad host range generally associated with *C. paradoxa* s. lat. (Morgan-Jones, 1967; Anonymous, 1987)".

Geographical distribution

Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Brazil, Burkina Faso, China, Colombia, Congo, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, Eswatini, Fiji, French Polynesia (Tahiti), Ghana, Guadeloupe, Guyana, Haiti, Hawaii, India, Indonesia, Iran, Jamaica, Japan, Madagascar, Madeira, Malawi, Malaysia, Mauritius, Mexico, Mozambique, Nicaragua, Nigeria, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, Senegal, South Africa, Sri Lanka, Taiwan, Thailand, Trinidad and Tobago, Uganda, Uruguay, USA, Venezuela, Vietnam, Zimbabwe.

The disease is expected to occur in almost all countries where sugarcane is grown.

Symptoms

Pineapple sett rot is primarily a disease that causes rotting of sugarcane seed pieces or setts (Figures 1 to 3). Poor germination and/or wilting of the young sugarcane shoots at emergence in the field may indicate the presence of the disease (Figure 4). When the setts are cut longitudinally at an early stage of the disease, the internodal tissues are red in color. The cuttings may smell like very ripe pineapple, a characteristic feature that gives the disease its name. At a later stage, the centre of the cutting breaks down and turns black because of the dust-like chlamydospores which are distributed between the vascular bundles (Figures 1 and 2). Pineapple sett rot may also occur in stalks of growing sugarcane if they have been physically damaged by rats, borers, mechanical means or generally debilitated by insect attacks or drought (Wismer, 1961; Wismer and Bailey, 1989).



Figure 1. Black and brown-colored internodal tissues of sugarcane setts (R. Viswanathan).



Figure 2. Black-colored internodal tissues of sugarcane setts (ISSCT).



Figure 3. Red and black discoloration of internodal tissues of sugarcane setts (G.P. Rao).



Figure 4. Poor germination of sugarcane affected by pineapple sett rot (S. Kaur).

In Brazil, a stalk internal dry rot disorder of standing cane was recently associated with simultaneous infection of *Colletotrichum falcatum* (causal agent of sugarcane red rot) and *Thielaviopsis paradoxa* (Silva *et al.*, 2023).

Diagnosis

A close observation of the internal symptoms in sugarcane setts split longitudinally generally helps to diagnose pineapple disease, and to discard other sett rot-inducing diseases (Fusarium sett rot, red rot, black rot). Special care should be taken not to mistake pineapple sett rot for black rot of sugarcane, caused by *Ceratocystis adiposa*, although the latter is rather uncommon (see "Minor fungal diseases", p. 290). Confirmatory diagnosis is provided by the microscopic examination of the spores (conidia and chlamydospores, also called microspores and macrospores respectively) of the imperfect stage of the fungus (Wismer and Bailey, 1989). Conidia are cylindrical to somewhat oval, hyaline to mid-brown, measure 6–24 (mean 13 µm) × 2–5.5 µm, and emerge in chains from the open end of the conidiophores (Figures 5 and 6). Chlamydospores are obovate to oval, thick-walled, brown, measure 10–25 × 7.5–20 µm, and are also usually produced in chains. Additional information on the pathogen can be found in the literature (de Beer *et al.,* 2014; Mbenoun *et al.,* 2014; Morgan-Jones, 1967; Sivanesan and Waller, 1986; Wismer, 1961; Wismer and Bailey, 1989).



Figure 5. Macrospores of *Thielaviopsis ethacetica* observed by light microscopy (S. Kaur).

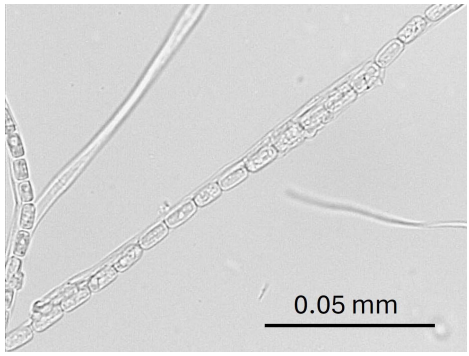

Figure 6. Microspores of *Thielaviopsis ethacetica* observed by light microscopy (S. Kaur).

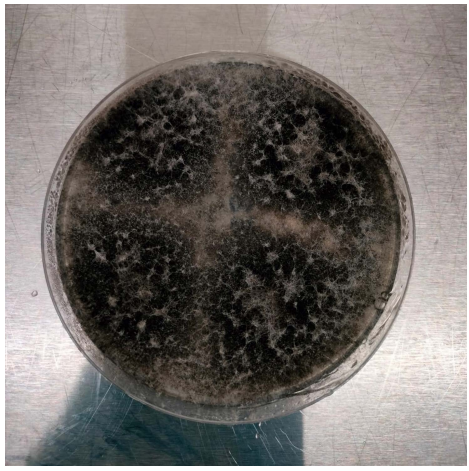

Figure 7. *Thielaviopsis ethacetica* growing on potato dextrose agar supplemented with antibiotics (S. Kaur).

Ceratocystis/Thielaviopsis species can be isolated on potato dextrose agar supplemented with neomycin, penicillin, and streptomycin (Kaur *et al*., 2026) (Figure 7). Morphological characteristics are, however, not very useful to differentiate the different fungi of the *C. paradoxa* species complex. For accurate species identification, amplification and sequencing of genome regions such as the ITS, β-tubulin and TEF-1α loci, followed by phylogenetic analyses, is necessary (Ferreira Borges *et al*., 2019; Kaur *et al*., 2026; Mbenoun *et al*., 2014).

Strains of the pathogen

Limited information is available on variation of the pathogen. Little difference in virulence was found among isolates of *C. paradoxa* collected from sugarcane in Hawaii (Byther and Steiner, 1974, cited by Wismer and Bailey, 1989). When sugarcane setts were inoculated with an isolate of *C. paradoxa* from pineapple and with an isolate from sugarcane, the isolate from sugarcane was less pathogenic (Liu and Marcano, 1973, cited by Wismer and Bailey, 1989). Three isolates of *C. paradoxa* from arecanut, coconut and sugarcane, respectively, proved to be cross-pathogenic (Sastry *et al*., 1989). However, these isolates formed two different groups based on the proportion of macrospores to microspores produced *in vitro* and the symptoms on the three hosts. One group was formed by the arecanut and coconut isolates, whereas the other group comprised the sugarcane isolate.

Transmission

Pineapple sett rot is mainly soil-borne: the conidia and chlamydospores of the fungus survive in the upper layer of the soil and ensure the contamination of cuttings. The period of survival of the pathogen in soil may exceed 15 months in sugarcane residues (Milanes Virelles and Herrera Isla, 1994). Physically damaged stalks of standing cane or stalks debilitated by drought can occasionally be infected by wind-blown spores (Wismer, 1961).

Host range

C. paradoxa s. str. has a limited host range (Cocoa, oil palm) (Mbenoun *et al.,* 2014). *T. ethacetica* can infect a variety of plants including sugarcane, pineapple, cocoa, coconut, *Phoenix canariensis* and oil palm (Morgan-Jones, 1967; Wismer, 1961).

Epidemiology

Factors which slow down germination of cuttings favor infection by the pathogen, such as cool temperatures, excessive soil moisture, drought or an inability of buds to germinate readily. Hot water treatments render the cuttings more susceptible to pineapple sett rot if they are not associated with a fungicide treatment (Wismer, 1961).

In Brazil, implementation of new cultural practices such as mechanical harvesting of green cane resulted in the build-up of large amounts of crop residues. These residues form a field biomass favorable for survival and multiplication of pathogens and pests. Consequently, incidence of three stalk-related disorders increased: shoot stunting induced by the spittlebug *Mahanarva fimbriolata*, red rot caused by *Colletotrichum falcatum*, and stalk internal dry rot caused by a simultaneous infection of *Colletotrichum falcatum* and *T. paradoxa* (Silva *et al*., 2023).

Economic importance

Pineapple sett rot can cause considerable damage at emergence of sugarcane if the conditions are favorable to disease development and if no appropriate control measures are taken. In such cases, cane growers may have to partially or completely re-plant the diseased fields (Wismer and Bailey, 1989).

Control

Although potentially highly destructive, pineapple sett rot can be efficiently avoided or controlled if a range of precautions or control measures are taken. A general recommendation is to use healthy setts of an appropriate physiological age to ensure rapid germination, setts with at least three nodes to increase the likelihood that the buds towards the centre will germinate before the fungus invades all the tissues, and crop management practices that promote germination and rooting (drainage, irrigation, etc.) (Wismer and Bailey, 1989).

Fungicide treatments at planting may be necessary, particularly after hot water treatment. The organomercury compounds are no longer recommended, and are no longer allowed in most, if not all of the countries, because of their toxicity and the environmental risks. Benzimidazoles like benomyl, or triazoles like propiconazole, can be used as fungicide sprays in the furrow at planting or, even better, as a fungicide bath for the setts before planting (Wismer and Bailey, 1989). A single application of the fungicide flutriafol protected sugarcane setts from pineapple sett rot, but also from smut caused by *Sporisorium scitamineum* (Bhuiyan *et al.*, 2014). In Florida, it was suggested that the treatment of sugarcane setts with a fungicide may enable Florida sugarcane growers to reduce planting density while maintaining stalk population and yield of susceptible cultivar CP74-2005 (Raid *et al.*, 1991). Physical protection of the sugarcane setts by polyethylene coating of short, hot water treated planting material significantly improved the control of pineapple sett rot, especially when the setts were also treated with a fungicide (Croft, 1998).

Some promising experimental results were obtained when different species of *Trichoderma* and two species of *Gliocladium* were used as biological control agents for sugarcane sett rot (Guevarra, 1990; Sampang, 1991). The efficiency of these fungi on a larger scale has, however, yet to be proven. Several *Pseudomonas* strains produce volatile and organic bacterial compounds that induce DNA damage and harmful ultrastructural changes to *T. ethacetica* (Freitas *et al.*, 2021). Twelve commercial microbial fungicide formulations composed of mixtures of antagonistic *Bacillus* species were labelled in Brazil for the management of stalk diseases caused by *C. falcatum* or *T. paradoxa* (Silva *et al.*, 2023).

Disease resistance has rarely been considered to control pineapple sett rot but should be further investigated. Significant levels of resistance to pineapple sett rot were found among 19 sugarcane varieties in Florida (Gama and Raid, 2016). A single-bud inoculation protocol was recently developed for greenhouse-based screening of sugarcane varieties against *T. ethacetica* (Kaur *et al.*, 2026).

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Pythium root rot

Jeffrey W. Hoy

Cause

Pythium arrhenomanes Drechsler and *Pythium* spp., oomycete.

Pythium arrhenomanes is the only highly pathogenic species (Hoy and Schneider, 1988a; Rands, 1961; Rands and Dopp, 1938). Numerous other *Pythium* species have been isolated from sugarcane roots. However, these species are weakly to mildly pathogenic, with the exception of *Pythium myriotylum* Drechsler, which is intermediate in pathogenicity between the other species and *P. arrhenomanes* (Croft, 1988; Magarey, 1984). Another species, *P. graminicola* Subraman., closely resembles *P. arrhenomanes* in morphology and ecology, and confusion over identification of isolates exists in the literature. The two have been demonstrated to be distinct species and, in pathogenicity tests, *P. graminicola* was only weakly pathogenic to sugarcane.

Geographical distribution

Australia, Barbados, Belize, Brazil, Colombia, Cuba, Dominican Republic, El Salvador, Fiji, Hawaii, India, Indonesia, Mauritius, Mexico, Nicaragua, Pakistan, Panama, Philippines, Puerto Rico, South Africa, Taiwan, Thailand, USA (Florida, Louisiana).

Symptoms

Above-ground symptoms of Pythium root rot are usually not obvious in the modern interspecific-hybrid varieties. Leaf chlorosis, curling and necrosis, wilting and plant death are rarely observed (Figures 1 and 2). However, root rot affected plants may produce fewer tillers and have lower stalk weights. Rotting of nodal roots can inhibit bud germination and initial shoot growth and thereby contribute to poor stand establishment. *Pythium* is a pathogen of immature root tissues. Primary root tips may be rotted, but most of the injury occurs in the lateral, secondary and tertiary roots (Figures 3 to 5). Rotted fine roots quickly deteriorate, and diseased plants have deficient root systems. Infected root tips are water-soaked and flaccid, and the margin of infection consists of a zone of tissue that is reddish-brown in color (Hoy and Schneider, 1988a; Magarey, 1984) (Figure 6). Lesions on primary roots where lateral roots once attached are elongate (1–3 × 1–2 mm) and red-black in color. Primary roots also may show general reddish-black discoloration (Figures 7 and 8).



Figure 1. Field severely affected by Pythium root rot with numerous wilted and dead young plants (A. Gama).



Figure 2. Young sugarcane plant killed by *Pythium arrhenomanes* soon after germination (A. Gama).



Figure 3. Root systems of sugarcane plants grown in soil infested with *Pythium arrhenomanes* (right) or in steam-treated soil (left) showing damage to roots resulting from infection and reduction in number of shoots produced (J. Hoy).



Figure 4. Sugarcane root system severely affected by Pythium root rot. Note the reduced size of the root system and the black discoloration of the primary roots (A. Gama).



Figure 5. Individual root showing rotting of the primary root tip and lateral roots, and lesions on the primary root where lateral roots were once attached (J. Hoy).



Figure 6. Root tip rotted by *Pythium arrhenomanes* showing water-soaked, flaccid tip with red-brown advancing lesion margin (J. Hoy).



Figure 7. Primary root rotted by *Pythium arrhenomanes* showing black discoloration (A. Gama).

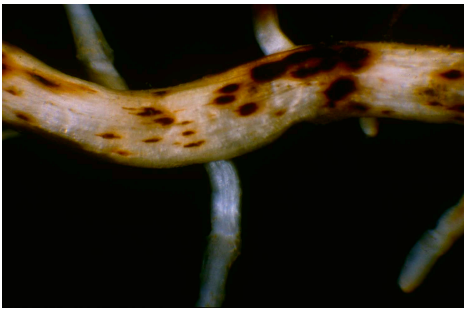

Figure 8. Close-up view of a sugarcane root showing black lesions of Pythium root rot (R. Magarey).

Diagnosis

Certain diagnosis of Pythium root rot and assessment of disease severity are difficult. Infection is limited to the root system which is not readily accessible in the soil. Rotted roots decay and often are lost when roots are removed from soil. Infections also can be ephemeral. *Pythium* species-specific sequences occur in the internally transcribed spacer (ITS) region of ribosomal DNA (Lévesque and de Cock, 2004). Specific pathogen identification and detection can be achieved by polymerase chain reaction (PCR) based assays (Fukuta *et al.*, 2014; Schroeder *et al.*, 2013; Wang *et al.*, 2003). Isolation of *Pythium* is most easily achieved on selective agar medium (Morita and Tojo, 2007); however, water agar also may be used. Roots should be washed and blotted dry to minimize bacterial contamination then plated on the surface or immersed in the agar medium. *Pythium* grows rapidly and should emerge into the agar within 24–48 h. Upon transfer, *P. arrhenomanes* produces a colony composed of branched, coenocytic hyphae exhibiting a radiate growth pattern with little aerial growth. The growth rate of an uncontaminated colony at room temperature will usually exceed 20 mm/day.

Strains of the pathogen

Strains of *P. arrhenomanes* have not been described. However, variability in virulence has been observed among isolates, and host genotype adaptation at the variety level has been reported (Adair, 1972).

Transmission

The pathogen occurs in the soil and may be moved in soil or water. *Pythium arrhenomanes* produces motile zoospores capable of swimming short distances. Zoospores show a chemotatic attraction to roots. Oospores also are produced that can serve as long-term

survival structures (Figure 9). Primary inoculum may consist of oospores, zoospores or mycelium. Infected roots of sugarcane plants or alternative hosts are possible sources of inoculum. Abundant asexual sporulation may occur in infected tissues, and high quantities of zoospores can be produced under wet soil conditions.

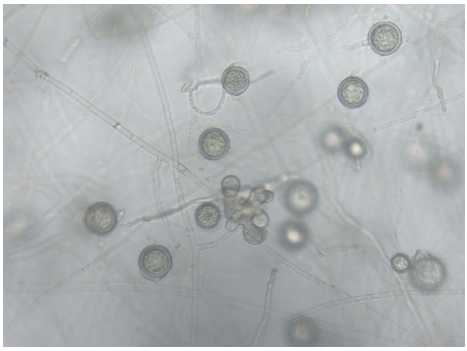

Figure 9. Oospores of *Pythium arrhenomanes* (J.H. Daugrois).

Host range

The host range of *P. arrhenomanes* is limited to graminaceous plants. However, there are many possibilities for alternative weed hosts. Comparisons of grass weeds found in Louisiana as hosts for *P. arrhenomanes* have revealed variability in host suitability among different weeds (Dissanyake *et al.*, 1997). Roots of johnsongrass (*Sorghum halepense*) and itchgrass (*Rottboellia cochinchinensis*) are extensively colonized by *P. arrhenomanes*, whereas browntop panicum (*Panicum fasciculatum*), goosegrass (*Eleusine indica*) and Italian ryegrass (*Lolium multiflorum*) are not. Barnyardgrass (*Echinochloa crus-galli*), Bermuda grass (*Cynodon dactylon*), broadleaf signalgrass (*Brachiaria platyphylla*), and large crabgrass (*Digitaria sanguinalis*) are intermediate in host suitability.

Epidemiology

Zoospore production and dispersal are favored by wet soil conditions. The sugarcane plant needs warm to hot temperatures for optimal growth, whereas *P. arrhenomanes* can grow over a wide range of temperatures. As a result, root rot is more severe under cool to mild temperatures and wet conditions that are favorable for growth and sporulation of the pathogen but unfavorable for plant growth. Root rot damage is more severe in poorly drained or heavy clay soils that retain soil moisture. Furthermore, Pythium root rot tends to be a problem mainly in more temperate growing areas, such as Louisiana.

Interactions have been documented between Pythium root rot and nematodes and red rot of planted cane. Severity of many sugarcane diseases is greater in stressed plants. Pythium root rot adversely affects the plant root system and thus enhances the likelihood of occurrence or duration of plant stress due to drought. Pythium root rot also can play a role in the complex problem known as yield decline.

Economic importance

The amount of damage caused by Pythium root rot depends on variety susceptibility, growth conditions, and the inoculum potential of the pathogen. Nevertheless, it is difficult to assess the economic impact of a soilborne disease, such as Pythium root rot.

Reductions in tillering and stalk weight due to this disease can lead to unrecognized but significant yield losses. Yield increases as great as 20% were obtained with treatments of the fungicide metalaxyl in Louisiana, but overall, treatment results were erratic (Hoy and Schneider, 1988b). The total body of evidence suggests that in regions where sugarcane must persist through periods with conditions unfavorable for growth, Pythium root rot will damage root systems and reduce growth during those periods. However, sugarcane is an indeterminate growth plant, and the effect of root rot early in the season on final yield is uncertain. The economic importance of Pythium root rot is probably underestimated due to the lack of readily discernable symptoms and because some adverse effects are manifested through interactions with stress factors and other diseases.

Control

Variety resistance and field drainage are the two most important means by which Pythium root rot is controlled. Resistance varies among *Saccharum* species and interspecific hybrid clones, and strategies for selecting resistant varieties have been developed (Koike, 1965). *Saccharum officinarum* is highly susceptible, and the industry based on the noble canes was nearly eliminated during the 1920s in Louisiana by a combination of diseases, including Pythium root rot. *Saccharum spontaneum* is a good source of resistance. However, few if any breeding programs are currently screening for disease resistance. Instead, most industries are relying on the generally improved resistance of the interspecific sugarcane hybrids. Knowledge about the heritability of resistance and efficiency of selection are unknown.

The prospects for successful chemical or biological control of Pythium root rot are not good. Control of soilborne diseases even with effective chemicals is often erratic. Soil fumigation often results in large increases in growth; however, the use of fumigants is not economically feasible in sugarcane.

Pythium root rot severity is limited by interactions with other microorganisms in the soil (Srinivasan, 1968). It may be possible to provide natural biological control of root rot with soil amendments of organic materials. Materials, such as sugar mill filterpress cake, sewage sludge and composts prepared from agricultural wastes have shown the potential to reduce root rot severity and increase plant growth. However, results from field experiments with organic material soil amendments have been erratic.

Good soil drainage reduces the frequency and duration of conditions conducive to disease. In Louisiana, extensive surface drainage systems are routinely employed. In the absence of better control measures, growing interspecific hybrid varieties under the best possible drainage conditions will minimize the damage from Pythium root rot.

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Ramu orange leaf

Robert C. Magarey, Sidney Suma, revised by Robert C. Magarey

Cause

An unidentified fungus (belonging to the order Exobasidiales).

Geographical distribution

Papua New Guinea.

The disease has only been observed in Papua New Guinea. Very little recent research has been undertaken because of its minor effect on crop yield.

Symptoms

Disease symptoms are first apparent as the spindle leaf unfurls, appearing as a uniform pale-green band around the base of the leaf. As the leaf expands, the symptoms progress up the leaf. At the margin of the pale-green band, symptoms grade into thin streaks (0.25–3 mm) of variable length (Magarey *et al.*, 1995; Magarey, 1996) (Figure 1). At first glance, affected leaves appear similar to those affected by downy mildew, though the streaks are thinner. The margins of affected leaves remain green.



Figure 1. Very fine leaf stripes running parallel to the leaf venation (R. Magarey).

With time, symptoms change from pale-green to yellow and finally to yellow-orange (Figures 2 to 4). Symptoms are more pronounced on the lower leaf surface, especially once the pathogen has sporulated. The orange-colored spores are produced on erumpent hyphal masses running parallel to the leaf venation. Red flecking develops within the diseased tissue and these flecks coalesce to produce thin red streaks (1–2 mm wide) of variable length and distribution (Figure 5).



Figure 2. Pale-green leaf stripes (P. Rott).



Figure 3. Yellow-orange leaf stripes (P. Rott).



Figure 4. Orange leaf stripes and leaf discoloration (P. Rott).



Figure 5. Yellow stripes and red flecking on the leaf blade (P. Rott).

With further development, older leaves die, and later, the whole stool. Not all shoots may be diseased within a stool. There is a tendency to grassiness with the disease (Figure 6).



Figure 6.
Grassy aspect of a diseased stool (P. Rott).

Diagnosis

Diagnosis relies on the recognition of leaf symptoms (Magarey, 1996).

Strains of the pathogen

There has been no evidence to suggest the existence of strains.

Transmission

The mode of transmission has not been studied. The disease does not appear to be systemic as cuttings planted from severely infected stalks have never been diseased; some have produced apparently disease-free shoots. It is also not uncommon to find infected shoots amongst vigorously growing healthy shoots in the same stool. Localized spread could be through spore dispersal.

Host range

The disease has been observed on noble cane (*Saccharum officinarum*), wild canes (*S. robustum* and *S. spontaneum*), *Saccharum edule* and interspecific sugarcane hybrids.

Epidemiology

The disease only occurs in ratoons, both in young and older crops. Disease levels are higher immediately after the dry season and in ratoons of late harvested crops. Moisture stress could initiate and/or facilitate the development and expression of symptoms. Disease incidence increases in the first 3 months after harvesting, then declines with the progressive death of infected shoots. In highly susceptible varieties, infected suckers can be found though the entire life of the crop.

Economic importance

Disease levels of 920 stools/ha have been observed on susceptible varieties in the field in Papua New Guinea (Suma and Pais, 1996). However, the disease is not considered economically important.

Control

No control measures are implemented in commercial fields. In seed cane nurseries, infected stools are rogued and burned.

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Red leaf spot (Purple spot)

Barry J. Croft

Cause

*Dimeriella sacchari (*Breda de Haan) Hansf. ex E.V. Abbott, ascomycete fungus.

Geographical distribution

Australia, Bangladesh, Colombia, Cuba, Fiji, Guatemala, India, Indonesia, Japan, Mauritius, Mexico, Nepal, Panama, Papua New Guinea, Philippines, South Africa, Taiwan, Tanzania, Thailand, Trinidad and Tobago, USA, Vietnam.

Symptoms

Red leaf spot first appears as small red dots up to 2 mm in diameter which can occur singularly or in clusters of dots (Abbott, 1964). The spots are generally on the upper surface of leaves but can also occur on lower leaf surfaces (Figures 1 and 2). In some cases, the clusters of red lesions have a distinctly rhizoid appearance (Figure 3).



Figure 1. Red leaf spots and patches on the upper side of a sugarcane leaf (P. Rott).



Figure 3. Red rhizoid patches on the lower side of a sugarcane leaf (P. Rott).

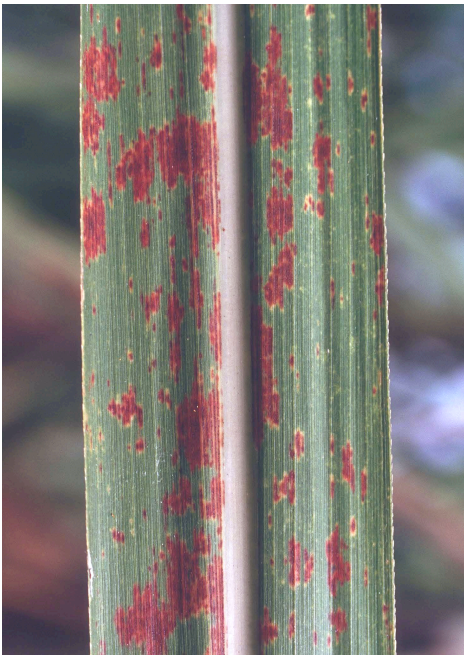

Figure 2. Red leaf spots and patches on the sugarcane leaf blade (P. Rott).

The lesions are more common on older leaves and in severe cases the lesions can coalesce to form red patches. Lesions tend to be more concentrated on the top third of leaves. Red leaf spot can be difficult to distinguish from yellow spot, which can be a red color on ageing. However, yellow spot lesions penetrate the leaf and form lesions on both the upper and lower leaf surfaces, whereas red leaf spot lesions do not penetrate the leaf and are seen only on the leaf surface facing the sun. In severe infestations, the crop canopy can have a general red appearance.

Diagnosis

Diagnosis of red leaf spot is by symptoms and the presence of the globose perithecia within the red lesions on the leaf surface (Figure 4). The perithecia are 39–72 µm (mean = 58 µm) in diameter. Ascospores are elongate to elliptic, uniseptate and 12–18 × 4–7 µm (mean = 15 × 6 µm).

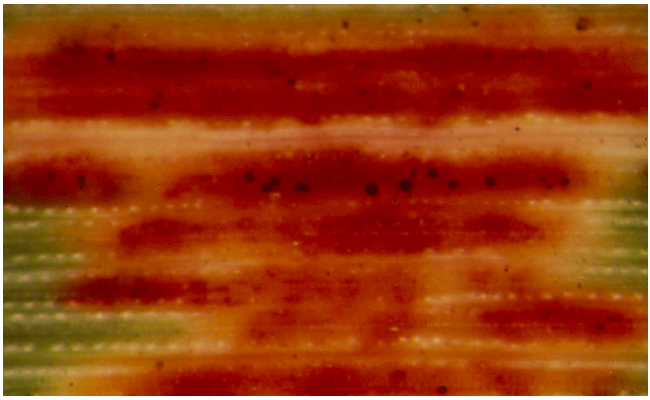

Figure 4. Red leaf lesion showing globose perithecia of the pathogen (B.J. Croft).

Strains of the pathogen

No information is available.

Transmission

Spread of the fungus is presumably by spores carried by wind and wind-blown rain.

Host range

Sugarcane is the only known host of *D. sacchari*.

Epidemiology

Red leaf spot generally occurs on older leaves during the warm, wet season months. The lesions only occur on the leaf surface facing the sun.

Economic importance

Loss of leaf area on older leaves of up to 20% has been reported but the disease is generally thought to cause no economic losses (Ryan and Birch, 1980).

Control

No control is warranted. Varieties differ in susceptibility.

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Red rot

R.P. Singh, Sunita Lal, revised by Rasappa Viswanathan and Chandramani Raj

Cause

Colletotrichum falcatum Went [syn. *Glomerella tucumanensis* (Speg.) Arx & E. Müll.], ascomycete fungus.

Geographical distribution

Afghanistan, Angola, Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Benin, Bolivia, Brazil, Burkina Faso, Burundi, Cambodia, Central African Republic, Chad, China, Colombia, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, El Salvador, Eswatini, Fiji, Gabon, Ghana, Guadeloupe, Guam, Guatemala, Guyana, Haiti, Hawaii, India, Indonesia, Iraq, Jamaica, Japan, Kenya, Madagascar, Madeira, Malawi, Malaysia, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Nigeria, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, Samoa, Solomon Islands, South Africa, Sri Lanka, Sudan, Taiwan, Tanzania, Thailand, Togo, Trinidad and Tobago, Uganda, Uruguay, USA, Vanuatu, Vietnam, Zimbabwe.

Currently the disease is widespread in South and South East Asian countries.

Symptoms

The red rot pathogen can infect seed canes or cuttings (setts), standing stalks, leaf sheaths, lamina and midribs, but rotting of the stalk is of primary importance (Abbott and Hughes, 1961; Singh and Singh, 1989; Viswanathan, 2021). Young plants show yellowing of leaves followed by total drying with or without midrib symptoms on the spindle leaves (Figure 1). During tillering and later during grand growth or maturity stages, disease symptoms are seen as distinct yellowing and drying of foliage (Figure 2). However, classic diagnostic symptoms are observed when diseased canes are split longitudinally. In split stalks of affected canes, internodal tissues show a red discoloration interrupted by whitish patches (spots) perpendicular to the axis of the stalk (Figures 3 and 4). These stalks emit a slightly acidic and starchy odor. The white patches or spots vary in size and appearance. Sometimes the affected tissues appear mottled. The number and nature of the white spots vary with cane varieties. During advanced stages of tissue rotting, greyish fungal mycelium fills the pith cavities (Figure 5). The infection may progress from bottom upwards or from the spindle downwards causing damages to both the nodal and the internodal tissues. Internal tissue rotting is manifested as discolored patches on the rind (Figure 6). Under favorable conditions, the acervulus production is seen all over the rind and nodal tissues as blackish specks and salmon colored spore masses on the rind and root eyes or entire nodes.



Figure 1. Characteristic foliage discoloration of a red rot-affected young sugarcane plant (R. Viswanathan).



Figure 2. Typical yellowing and drying of leaves in red rot-affected sugarcane (R. Viswanathan).



Figure 3. Characteristic reddish discoloration of internodal tissue with white spots (R. Viswanathan).



Figure 4. Longitudinal section of a stalk internode showing characteristic white spots (R. Viswanathan).



Figure 5. Greyish mycelia of *Colletotrichum falcatum* fill pith cavities of affected stalk internodes (R. Viswanathan).



Figure 6. Red rot affected canes with characteristic discolored patches on the rind. Spore masses and acervuli of *Colletotrichum falcatum* are seen on the nodal region (R. Viswanathan).



Figure 7. Complete devastation of a sugarcane field caused by red rot (R. Viswanathan).

Patches of a few clumps show drying of canes in the field with moderate infection levels. In contrast, when the infection rate is high, entire fields or areas are devastated, thus causing complete failure of the crop (Figure 7).

On the leaves, the pathogen produces elongated red spots on the lower or upper surface of the midribs. They can remain small or become more elongate along the midrib. Initially, the spots are dark red, then become straw-colored with purple margins that may be covered with small black flecks which are the fruiting bodies of the fungus (acervuli) (Figure 8). The spots coalesce to form elongated lesions covering the entire midrib of a leaf or many leaves in the spindle (Figure 9). In rare cases, the pathogen produces small dark red spots on the lamina. The pathogen also attacks leaf sheaths producing reddish patches.

Diagnosis

Went (1893) first observed red rot and described the causal organism as *Colletotrichum falcatum*. The sexual stage of *C. falcatum* was later reported by Spegazzini (1896) in Argentina who named it *Physalospora tucumanensis*. Later, the causal organism of red rot was reclassified by von Arx and Müller (1954) and included in the genus *Glomerella* as *G. tucumanensis*. The fungus produces falcate conidia, either in specialized fruiting structures, acervuli, or on the hyphal tips. The spore masses produced in the acervuli are in a mucilaginous matrix having a pinkish appearance. Conidia measure 16–40 µm in length and 4–8 µm in width. Septate setae present in the acervuli vary in number and size between fungal isolates. In general, they are bulbous at the base, tapering towards the tip, and measure 100–200 × 4 µm. Germinating conidia produce appressoria and sometimes appressoria are produced on hyphal tips. These appressoria are smooth but thick-walled and cinnamon-buff in color. The perithecia, when produced,



Figure 8. Dark red coloration of the leaf midrib covered with small black flecks (acervuli of the pathogen) (J.-C. Girard).



Figure 9. Spindle leaves exhibiting midrib lesions of red rot along the entire lamina (R. Viswanathan).

are completely embedded in leaf tissues except for the protruding ostioles. They are 100–200 µm in width and 85–250 µm in height, containing clavate asci and paraphyses. Ascospores are hyaline, straight to fusoid, single celled and 18–22 × 7–8 µm in size.

Polyclonal antisera against *C. falcatum* and several immunological assays have been developed for the detection of the red rot pathogen (Viswanathan *et al.*, 1998). A loop-mediated isothermal amplification (LAMP) assay is also available for molecular identification of the pathogen in pure culture or in infected plant tissue (Chandra *et al.*, 2015). The pathogen can be identified by PCR and sequencing of the β-tubulin and actin genes as well as the internal transcribed spacer (ITS) region (Ahmad *et al.*, 2023). Genome sequences specific to *C. falcatum* were also used to develop a PCR-based assay for detection of the red rot causal agent (Nithya *et al.*, 2012).

Strains of the pathogen

Pathogenic variants to different varieties may arise independently from genetically different groups. However, the pathogenic grouping and the genomic grouping of isolates are different (Malathi *et al.*, 2010). Pathogenic variation among the *C. falcatum* isolates is being assessed regularly on a set of differential hosts and pathotypes prevailing under field conditions are designated. So far, 13 pathotypes (CF01–CF13) were characterized and the prominent ones in different agro-climatic regions are used to screen sugarcane clones for disease resistance (Viswanathan, 2017 and 2021; Viswanathan *et al.*, 2022). In general, the pathogen slowly adapts to the new varieties by acquiring aggressiveness and causes breakdown of resistance in the host varieties. Variants of

the pathogen arise from the existing isolates/pathotypes under host selection pressure, thus reflecting a dynamic variation under field conditions (Viswanathan *et al.*, 2017 and 2022). Although midrib isolates are reported weakly pathogenic, the recent red rot epidemics on cultivar Co 0238 suggested that midrib infections were involved in catastrophic damages in the subtropical India (Viswanathan, 2023).

Transmission

Red rot is spread primarily through planting of infected seed cane and crop debris. Crop debris comprising diseased stalks, stubble pieces and trash left over after harvesting provide a large amount of inoculum to infect the new crops. These inoculum sources are not only important within the particular crop but may also help to initiate infection in young and developing crops. Though the fungus is not soil-borne, it remains viable for one to two months and causes infection in the cuttings planted during that period. However, the fungal inoculum remains viable on the crop debris for up to one year. This phenomenon is common in disease endemic regions.

In addition to the planting of infected seed cane, inoculum is disseminated by rain, heavy dews, irrigation water and wind. The conidia produced on the midrib or the stalk rind can easily be washed down and then spread by water. The occurrence of the disease during dry periods and infection of the upper nodes and young midribs indicate that wind-borne infection is common. Wind-borne inoculum may be either dried, mucilaginous conidia matrix, and/or ascospores.

Host range

Besides sugarcane, the fungus has been isolated from sorghum and Johnson grass leaves (Viswanathan, 2021). The perithecia were found on grasses from natural flora (*Anacentrus sp.* and *Leptochloa filiformis*), and on *Miscanthus* in Taiwan. However, the role of non-sugarcane hosts in the epidemiology of the disease is secondary.

Epidemiology

The pathogen mainly infects stalks through the nodal region, leaf scar, growth ring, root primordia and buds. Environmental factors, particularly temperature and relative humidity, play an important role in the establishment of infection and further disease development (Viswanathan, 2021). Red rot is most destructive during the rainy season from July to September in tropical and subtropical regions. Under low temperature and less humid conditions, progress of the disease is slow under field conditions, but this situation results in the development of the dormant state of nodal infections. In the case of the planted cuttings, initiation of the disease occurs from both cuttings and soil inoculum surviving in infected debris. The disease onset and progress are favored by both high and low soil moisture contents. At times, the attack by secondary infections of the wilt pathogen (*Fusarium sacchari*) enhances the tissue damage and the magnitude of losses (Figure 10).

Economic importance

Red rot dictates sugarcane cultivation in India and many other Asian countries. It has caused severe damages in tropical and subtropical areas that resulted in complete crop losses (Figure 6). Many outstanding varieties were removed from cultivation due to varietal breakdown in the past decades and the recent disease epidemics on the popular cultivar Co 0238 caused billions of US$ losses in India (Viswanathan, 2021).



Figure 10. Combined infections of the red rot and the wilt pathogens in a sugarcane stalk. Red rot affected internodes exhibit abundant greyish mycelial growth in the pith cavities while wilt affected internodes show pith cavities with white mycelial growth and pink/purple discoloration (R. Viswanathan).

Control

Red rot can be effectively controlled through the use of resistant varieties. Red rot is one of the diseases responsible for the disappearance of the noble canes (*Saccharum officinarum*). *S. robustum* and *S. sinense* are also susceptible to the disease but resistance genes were found in *S. spontaneum* and in some clones of *S. barberi*. Resistance is controlled by one or a few genes from *S. spontaneum* whose effects can be counter balanced by an inhibitor gene of *S. officinarum* (Azab and Chilton, 1952).

Sugarcane varieties are screened for resistance by artificial inoculation with the pathogen under field as well as controlled conditions. Designated pathotypes should be used for disease screening. Six to eight-month-old canes are inoculated at the nodes (nodal swabbing method) or onto internodal tissues (plug method) of standing canes and disease ratings are assessed after two months (Viswanathan *et al*., 2018).

Fungicides failed so far in controlling seed-borne infections due to the lack of efficient application protocols. However, a mechanized sett treatment device was recently developed and successfully applied to diffuse the chemicals inside the cuttings by vacuum infiltration (Malathi *et al.*, 2017). This methodology prevents infections from both cutting- and soil-borne inocula for 3 to 4 months. Once the primary infections are reduced, disease development during grand growth phases is significantly contained. However, in endemic areas, two foliar sprays with effective fungicides are suggested to reduce secondary spread of the disease during the monsoon season (July to September)

in India. Thermotherapy of the seed canes produced inconsistent results for elimination of stalk-borne infections. Overall, use of healthy seed cane produced from healthy seed nurseries coupled with effective treatment of cuttings has proven quite effective in managing red rot.

Besides the use of healthy seed cane and resistant varieties, red rot incidence may be reduced through the use of some agronomic practices. These include timely roguing and burning of diseased plants, avoiding ratoons in the affected fields, and performing crop rotation. All these measures will prevent secondary spread of the disease in the field.

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Red spot of the leaf sheath

Claude Ricaud, revised by Andre B. Gama

Cause

Passalora vaginae (W. Krüger) U. Braun & Crous [Syn. *Mycovellosiella vaginae* (W. Krüger) Deighton], deuteromycete fungus.

Geographical distribution

Afghanistan, Argentina, Australia, Barbados, Belize, Benin, Brazil, Burkina Faso, China, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Ecuador, Ghana, Guyana, Haiti, Hawaii, Honduras, India, Indonesia, Jamaica, Japan, Madagascar, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Mozambique, Nicaragua, Peru, Philippines, Puerto Rico, Réunion, Senegal, South Africa, Taiwan, Thailand, Togo, Trinidad and Tobago, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

Ellipsoid bright red spots are present on the leaf sheaths, sharply delimited from the normal green color of the surrounding tissues (Figures 1 and 2). The spots enlarge and coalesce to form irregular reddish lesions which penetrate through to the inner sheaths (Figure 3). In later stages, a greyish-green mold may develop on the affected areas, more abundantly on the inside of the sheath (Abbott, 1964).

Diagnosis

Conidiophores and conidia of the fungus are readily visible on microscopic examination of the mold. Conidiophores are olivaceous brown, branched, 1–5 septate, 40–120 × 3–5 µm. Conidia are hyaline to straw-colored, 1–3 septate, 30–55 × 4.0–4.5 µm. Additional characteristics have been described by Kirk (1982) and Sivanesan and Waller (1986).

Strains of the pathogen

No information is available.

Transmission

The disease spreads by air-borne conidia.

Host range

Only sugarcane was reported as a host for *Passalora vaginae*.





Figure 1. Ellipsoid bright red spots on leaf sheaths (J.-C. Girard).



Figure 2. Close-up view of an ellipsoid bright red spot on a leaf sheath (P. Rott).

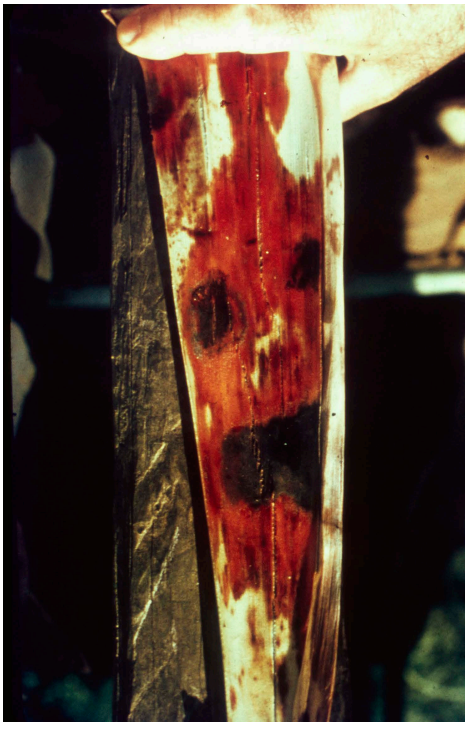

Figure 3. Reddish lesions on the inner leaf sheath (ISSCT).

Epidemiology

The disease is favored by warm, moist weather.

Economic importance

Although widespread in sugarcane, the disease is usually of minor economic importance. Occasionally some varieties may be affected because of high susceptibility.

Control

The disease does not warrant any control measure except the elimination of rare cases of high susceptibility during selection.

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Rind disease and sour rot

Jack C. Comstock, Roger Bailey, revised by Sushma Sood

Cause

Phaeocytostroma sacchari (Ellis & Everh.) B. Sutton, coelomycete fungus.

Geographical distribution

Andaman Islands, Angola, Antigua and Barbuda, Australia, Barbados, Belize, Bolivia, Brazil, China, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Egypt, Fiji, French Polynesia (Tahiti), Guadeloupe, Guyana, Haiti, Hawaii, Honduras, India, Indonesia, Iraq, Jamaica, Japan, Madagascar, Malawi, Malaysia, Mauritius, Mexico, Mozambique, Myanmar, New Caledonia, Nicaragua, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Portugal, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, South Africa, Sri Lanka, Taiwan, Tanzania, Thailand, Trinidad and Tobago, Uganda, USA, Vietnam, Zimbabwe.

The disease occurs probably in almost all countries where sugarcane is grown.

Symptoms

Infected stalks exhibit rind discoloration varying from green to yellow, turning into reddish brown. The reddish color in the nodal region may extend to leaves (Viswanathan *et al*., 2003). Later, numerous black, coiled, thread-like spore masses extrude from conspicuous black pustules that break through the stalk rind, often most evident near the nodes (Figures 1 to 3). The pustules can also occur on the leaf sheaths and midribs. Severely affected stalks appear straw-colored and shriveled. Internally, an orange-brown rotting occurs, initially at the nodes and later extending through the internode tissues, and may turn dark later (Figures 4 to 6). When severe, entire stalks may be rotted (Figure 7). Affected stalks often have a distinctive sour odor (Goodall *et al*., 1999). Leaf yellowing and premature desiccation may occur (Abbott *et al*., 1964).

Diagnosis

The diagnostic features of the infected stalks include black, coiled, threadlike spore masses extruding from the black pustules (Figures 1 to 3). Colonies of *P. sacchari* grown on potato dextrose agar medium for 15 days appear yellowish-white. The edge of colonies has dense mycelia with dark pycnidia measuring 150.5–577 × 72.5–303.5 µm (Melo *et al*., 2023). Conidia are pale brown, single-celled, ellipsoidal to ovoid, smooth-walled, with an obtuse apex and truncate base, measuring 6.5–15 × 3–5 µm (Abbott *et al*., 1964; Melo *et al*., 2023; Sivanesan and Waller, 1986; Sutton and Waterston, 1966). Paraphyses are hyaline, aseptate, occasionally branched, and flexuous (Saucedo Carabez *et al*., 2014).



Figure 1. Long thread-like coiled tendrils of the pathogen spore masses extending from a stalk (S. Matsuoka).

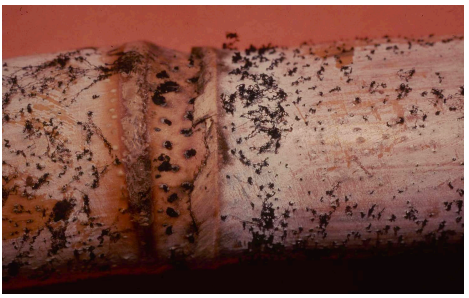

Figure 2. Sugarcane stalk showing short spore masses on the rind (J.C. Comstock).

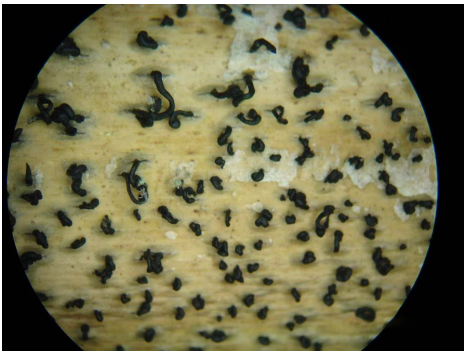

Figure 3. Spore masses of the pathogen erupting from the rind of an infected stalk (S. Matsuoka).



Figure 4. Severe sour rot causing stalks to collapse (S. McFarlane).



Figure 5. Internal and external symptoms of sour rot (S. McFarlane).



Figure 6. Internal orange-red discoloration of the stalk tissue (S. McFarlane).



Figure 7. Severe internal rotting of sugarcane affected by sour rot (S. McFarlane).

Molecular identification assays with primer pairs ITS1/ITS4 and ITS5/ITS4 for the internal transcriber spacer region (ITS-5.8S-ITS2) result in 536 bp and 493 bp amplification products, respectively (Saucedo Carabez *et al.*, 2014). Primers LROR and LR7 for the large subunit (LSU) produce a 379 bp amplicon by polymerase chain reaction (PCR) (Gopi *et al.*, 2022). A rapid PCR procedure has also been developed for the detection and identification of the pathogen using two sets of primers that amplify translation elongation factor 1-alpha (TEF1-α) (Melo *et al.*, 2024). PCR products can be sequenced and blasted against the NCBI database to confirm the identity of the pathogen.

Strains of the pathogen

The existence of strains of the pathogen is not known.

Transmission

Rind disease is spread by conidia dispersed by wind and rain. Infection usually takes place at the nodes through wounds.

Host range

Rind disease and sour rot is only known to occur on *Saccharum* spp.

Epidemiology

P. sacchari, a common but a weak pathogen, damages plants that are weakened by drought stress or are overly mature. It does not normally cause damage to actively growing sugarcane. After affected crops are harvested, the infection does not usually persist into subsequent ratoons. The pustules of rind disease can often be found on seed cane pieces that deteriorate after planting.

Economic importance

P. sacchari is a weak pathogen that seldom causes direct economic damage, but it aggravates crop deterioration by reducing the juice quality of stalks that are weakened for other reasons. Severe damage can result if mature crops are subjected to prolonged drought (Viswanathan *et al.*, 2003). In an outbreak in South Africa in 1998, 45% of

fields in one area were affected, and more than 50% of the internodes in most stalks in some fields were rotted, resulting in cane consignments being rejected by the mills (Goodall *et al*., 1999). The recent emergence of the disease in Brazil caused average death of one plant per meter (Melo *et al*., 2023).

Control

Control practices are not usually required, since plants grown under normal cultural practices are unlikely to develop rind disease. In situations where sugarcane is grown over long seasons and where seasonal droughts are likely, crops should be harvested before becoming over-mature.

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Ring spot

Barry J. Croft

Cause

Epicoccum sorghinum (Sacc.) Aveskamp, Gruyter & Verkley [Syn. *Leptosphaeria sacchari* Breda de Haan; *Phoma sorghina* (Sacc.) Boerema, Dorenb. & Kesteren], ascomycete fungus (Laurel *et al.*, 2021). Ring spot is reported to be caused by *Curvularia ischaemi* McKenzie in Guangdong province of China (Chen *et al.*, 2023), and by *C. guangxiensis* in the region of Guangxi Zhuang (Wei *et al.*, 2026).

E. sorghinum is also the causal agent of twisted leaf (see "Twisted leaf", p. 264).

Geographical distribution

Andaman Islands, Angola, Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Benin, Bolivia, Brazil, Burkina Faso, Burundi, Cambodia, Cameroon, Central African Republic, Chad, China, Colombia, Côte d'Ivoire, Costa Rica, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, El Salvador, Fiji, French Guiana, Ghana, Guadeloupe, Guyana, Hawaii, Honduras, India, Indonesia, Jamaica, Japan, Kenya, Madagascar, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, Samoa, Senegal, Sierra Leone, Solomon Islands, South Africa, Sri Lanka, Sudan, Surinam, Taiwan, Tanzania, Thailand, Togo, Trinidad and Tobago, Uganda, Uruguay, USA, Vanuatu, Venezuela, Vietnam, Zambia, Zimbabwe.

Symptoms

Ring spot initially forms as dark green to brown, elongated oval-shaped lesions with a yellow halo. The lesions enlarge and the centre of the lesion usually becomes straw-colored with a well-defined red-brown margin (Figures 1 to 5). In some cases, the lesions will remain red-brown. The lesions occur mainly on the leaf blade but can occur on the leaf sheath and are very variable in size, 4–18 × 1–5 mm. Ring spot is more common on older leaves but can also affect young leaves (Figure 6). On older spots, the small black fruiting bodies of *E. sorghinum* are usually present (Figure 6).

Diagnosis

The characteristic ring-shaped lesions and the presence of the fruiting bodies of *E. sorghinum* are usually sufficient for diagnosis of the disease. The perithecia of *E. sorghinum* are spherical, brown, approximately 140 µm in diameter and contain eight 4-celled ascospores (20–24 × 3 µm) per ascus (Abbott, 1964; Sivanesan and Waller, 1986).



Figure 1. Various leaf lesions of ring spot (P. Rott).



Figure 2. Leaf with young lesions of ring spot (P. Rott).



Figure 3. Leaf with old lesions of ring spot (P. Rott).



Figure 4. Leaf with young and old lesions of ring spot (J.-C. Girard).



Figure 5. Leaf severely affected by ring spot (P. Rott).

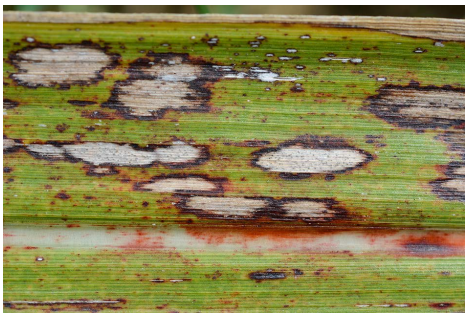

Figure 6. Old ring spot lesions showing small black fruiting bodies of *Epicoccum sorghinum* (P. Rott).

E. sorghinum produces pycnidia with straight necks, 68–118 × 45–90 µm. Conidia are unicellular, oval shaped or curved, 3.6–5.3 × 1.8–2.8 µm. Sequences of internal transcribed spacer (ITS-rDNA), beta-tubulin (TUB2) and actin (ACT) genes of *E. sorghinum* have been reported and could be used for PCR assays and sequencing (Laurel *et al.*, 2021; Shi *et al.*, 2025). ITS sequences were used to identify *C. ischaemi* in China (Chen *et al.*, 2023).

The early stages of ring spot can be confused with other leaf spots such as eye spot and brown stripe. The lesions caused by fine droplets of the common herbicide, paraquat, can resemble ring spot.

Strains of the pathogen

No information is available

Transmission

Ring spot is spread by wind or rain-borne spores.

Host range

E. sorghinum causes ring spot on sugarcane and leaf spots on rice and sorghum (Liu *et al.*, 2020). *C. ischaemi* has been reported to cause disease on sugarcane in China (Chen *et al.*, 2023) and on Batiki bluegrass (*Ischaemum indicum*) in Fiji and the Solomon Islands (McKenzie, 1981). *E. sorghinum* has been isolated from numerous plant species worldwide, including berries, brassicas, cereals, legumes, and ornamental plants (Shi *et al.*, 2025).

Epidemiology

Ring spot can be seen throughout the year but is not common on young cane. In India, disease severity of *Saccharum officinarum* accessions was highly correlated with rainfall, evening relative humidity and rainy days, whereas a negative correlation was observed with maximum temperature (Gopi *et al*., 2021).

Economic importance

Ring spot is not considered to be economically important.

Control

No control is warranted. Varieties differ in susceptibility to the disease.

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Root and basal stem rot

Jing-Gwo Fang, Ching-Shiou Lee, revised by Lilik K. Putra

Cause

The disease is caused by two species of *Xylaria* (ascomycete fungi): *Xylaria warburgii* Henn., reported in Taiwan (Fang *et al*., 1986a) and Indonesia (Sitepu *et al*., 2010), and *X. arbuscula* Sacc., reported in the USA and Puerto Rico (Anonymous, 1960) and Indonesia (Maryono *et al*., 2020a). A third species of *Xylaria*, namely *X. bambusicola* Y.M. Ju & J.D. Rogers, was recently obtained from stroma in sugarcane with symptoms of root and basal stem rot in Indonesia, and its genome was sequenced (Selvaraj *et al*., 2024). However, pathogenicity of this third *Xylaria* species and its involvement in the disease remains to be demonstrated.

Geographical distribution

Indonesia, Puerto Rico, Taiwan, USA.

Symptoms

The characteristic symptoms of root and basal stem rot in the field are the yellowing and wilting of mature sugarcane. In plant crop, the symptoms are rarely found in young sugarcane. Meanwhile, the drying leaves can be seen on young canes in ratoon crop due to a continuation of previous infection that occurred in plant crop. Root and basal stem tissues of diseased plants are rotted, and basal stem tissues appear light brown and reddish (Figures 1 to 3) (Hsieh, 1980; Sitepu *et al.*, 2010). White mycelium masses (Figure 4) and black lines (Figure 5) are usually observed in longitudinal sections of infected stalks, and a carbonaceous, sterile base later develops. White-tipped, upright, sterile stroma (less than 8 cm in length) bearing abundant conidia are produced on the surface of diseased stems during the spring rainy season (Figure 6). Black, unbranched or occasionally branched, cylindrical to clavate ascostroma also are found in the fields during April to June (Fang *et al*., 1986a). Many ratoon plants die as a result of the disease. If the pathogen infects the canes at the later stage of growth, the diseased ratoon canes are retarded in growth (Figure 7). The stunted plants contain fewer stalks that are shorter and thinner than normal. Eventually, diseased plants die (Sitepu *et al*., 2010).

Diagnosis

The yellowing and wilting symptoms occurring in the late growth stage of sugarcane are characteristic of the disease. Diseased plants can easily be pulled from the soil because the roots are rotted. The typical white rot symptoms are easily found in a longitudinal section of the stem (Figures 2 and 3). The aggregate distribution of the diseased plants and the presence of stroma on diseased plants are important characteristics of diagnosis of root and basal stem rot in sugarcane plantations. Cultural characteristics



Figure 1. Initial symptoms of basal stem rot (T. Maryono).



Figure 3. White rot symptom in the basal part of harvested sugarcane stems (R. Sitepu).



Figure 5. Black lines formed in the basal stem (T. Maryono).



Figure 2. Infection on the new shoot (R. Sitepu).



Figure 4. White mycelium masses in the infected stem (R. Sitepu).



Figure 6. White-tipped stroma formed on ratoon cane stubble (A. Kristini).



Figure 7. Poor germination of ratoon cane caused by *Xylaria arbuscula* (A. Kristini).

of the pathogen on common fungal growth media, such as potato dextrose agar and oatmeal agar, can also be used to diagnose the disease. A typical culture of the pathogen would consist of a mosaic-like, black and white mycelial mat with a number of thin rhizomorphs (Fang and Lee, 1995; Hsieh, 1980; Martin, 1967; Maryono *et al*., 2017; Rogers, 1979; Sitepu *et al*., 2010). Although no specific PCR detection assays have been published so far, the pathogen can be taxonomically identified based on ITS and LSU rRNA sequence analyses (Selvaraj *et al*., 2024).

Strains of the pathogen

No strains of the pathogen have been reported so far. However, vegetative compatibility groups of the pathogen have been found in cane fields in Taiwan (Fang and Lee, 1995). Pathogen isolates obtained from different areas belonged to different vegetative compatibility groups.

Transmission

The pathogen is primarily transmitted from the remaining stubble of diseased plants to healthy plants in the soil. The pathogen can persist in diseased tissues from one year to the next (Fang *et al*., 1986b; Sitepu *et al*., 2010). A large number of common wild grasses

that occur in sugarcane fields are potential hosts of the pathogen. Therefore, weeds may promote the spread and propagation of inoculum in cane fields and facilitate the survival of the pathogen. Grass hosts other than sugarcane can be important sources of inoculum, particularly when the cane stubble is not available (Fang and Lee, 1994). The rapid degeneration of germination tubes indicates that conidia are probably not the major inoculum source. Rather, the primary inoculum source seems to be the ascospores.

Host range

Sugarcane is the main host of *X. warburgii*; however, all *Saccharum* and *Miscanthus* species can be infected by the pathogen. Four common cane field weeds were found to be natural hosts of this pathogen: barbate windmillgrass (*Chloris barbata*), Bermuda grass (*Cynodon dactylon*), satintail (*Imperata cylindrica*) and nutgrass (*Cyperus rotundus*). Maize (*Zea mays*) and sorghum (*Sorghum bicolor*) also are highly susceptible to the pathogen (Fang and Lee, 1994). Besides being pathogenic to sugarcane, *X. arbuscula* can infect Macadamia trees (Ko and Kunimoto, 1991) and soybean (Allen *et al*., 2017).

Epidemiology

Root and basal stem rot of sugarcane is a major production constraint of sugarcane in some regions of Taiwan and Indonesia. In Taiwan, the disease was first reported by Hsieh (1980) and was limited to the Yuching cane growing area until 1984, however, it has later on become epidemic (Fang *et al*., 1994). The disease was found in cane fields at 8 of 21 sugarcane factories. In Indonesia, the disease is only found on Sumatra Island, particularly in Lampung and South Sumatra (Sitepu *et al*., 2010; Maryono *et al*., 2017). In South Sumatra, the disease spread rapidly and, within seven years, the number of affected cane fields increased from 214 to 1220. Ratoon plants were more affected than plant cane (Widowati *et al*., 2022).

Xylaria can survive in the sugarcane stools buried in the soil at a depth of 0–50 cm and subsist for more than seven months as a saprophyte. It will re-infect the sugarcane roots or basal stems if ascospores are available as the primary inoculum (Sitepu, unpublished data). The disease is more severe in fields with sandy or sandy loam soil.

The disease progress follows monomolecular and logistic models depending on the sugarcane variety. The infection rate in susceptible varieties is higher than the one observed in more resistant varieties. Rainfall and relative humidity have a positive effect on disease development and incidence, whereas temperature and duration of irradiation have a negative effect. Potassium availability and soil permeability are positively correlated, whereas total nitrogen availability and iron are negatively correlated with disease incidence (Maryono *et al*., 2020b).

Economic importance

Root and basal stem rot is an extremely important disease of sugarcane in certain localities of Taiwan and Indonesia. More than 700 ha of sugarcane are infected by the pathogen in Taiwan every year. The disease not only affects plant cane yield, but also the germination and yield of ratoon crops. Losses of 5% have been estimated in plant cane but may be 30% or more in ratoon cane (Fang *et al*., 1994). A 12 and 15% decrease of cane weight and sugar yield, respectively, was observed in a plant crop experiment conducted in Indonesia using artificial inoculation (Sitepu *et al*., 2010).

Control

The breeding and selection of resistant varieties is the only practical method of controlling the disease. Resistance of varieties, such as ROC7 and ROC9, has been used successfully in Taiwan. Conversely, resistant variety have not yet been obtained in Indonesia (Yulianti, 2017). In Taiwan, application of green manures and phorate granules in the soil, and growth of crotalaria and alfalfa failed to suppress disease incidence in the field (Fang *et al*., 1986b). Meanwhile, in Indonesia, minimum tillage followed by adding organic materials and planting cover crops (*Mucuna cochinchinensis*) reduced disease incidence in the field (Sitepu, unpublished). Other control methods are based mainly on cultural practices. In highly susceptible varieties, early cane harvesting results in a substantial yield reduction. The planting of sugarcane in spring is recommended to reduce disease incidence. Field sanitation must be undertaken, and plant residues must be collected and buried to limit the dispersal of the pathogen. Isolates of *Trichoderma* spp. have been tested in the laboratory and have the potential to be biological control agents of the disease (Dirmawati *et al*., 2018; Jati *et al*., 2022).

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Sclerophthora stunt
(Previously known as Sclerophthora disease)

Claude Ricaud, revised by Andre B. Gama

Cause

Sclerophthora macrospora (Sacc.) Thirum., C.G. Shaw & Naras., oomycete.

Geographical distribution

Australia, India, Mauritius, Peru, South Africa, USA.

Symptoms

Sclerophthora stunt induces severe dwarfing and profuse tillering of the whole or part of a stool (Figure 1), giving it the appearance of a clump of grass (grassy shoot). Infected shoots usually fail to develop millable stalks; when they do, stem galls of varying size and shape are found on them (Figure 2).



Figure 1. Dwarfing and profuse tillering of a stool (ISSCT).



Figure 2. Proliferation of shoots sprouting from buds at the upper nodes (left) and stem galls (right) (ISSCT).

Leaves of affected plants are much reduced in size and are coarse and brittle in texture. They are chlorotic due to the presence on part or the whole of the lamina of irregular yellowish-white streaks of varying lengths between the veins and of chlorotic blotches of varying shapes and sizes (Figure 3). The streaks may turn reddish. When growth conditions are poor, the leaves are stiff and erect, but under favorable conditions they can be flexible and drooping with wavy edges. The tips and edges of leaves dry-out prematurely causing shredding of the tissues, thus giving the edges a ragged appearance. Leaf tips may curl inwards into loops.



Figure 3. Leaves with irregular yellowish-white streaks (BSES).

When infection develops late on a stalk there may be a proliferation of shoots sprouting from buds at the upper nodes (Figure 2).

Diagnosis

Microscopic examination of disease leaf tissues can reveal the characteristic fruiting bodies of the different stages of the fungus. Diseased material may be incubated either in a moist chamber or in sterile water overnight before examination.

Oospores of the fungus are seen in the chlorotic streaks in mesophyll tissue around vascular bundles. Their size varies in different countries. Measurements from Louisiana were 49–72 µm (average 60 µm) and from Australia and Mauritius they were 35–63 µm (average 51 and 47µm, respectively). They are thick-walled, 4–10 µm. Sporangia are thin-walled, lemon-shaped, and measure 49–95 × 29–65 µm. Additional characteristics of the fungus have been described by Roth (1967), Sivanesan and Waller (1986) and Steindl and Steib (1961).

Strains of the pathogen

The variability in size of oospores from different countries has suggested the existence of different strains of the pathogen (Steindl and Steib, 1961). However, there has been no supporting evidence based on pathogenicity tests.

Transmission

The disease is readily transmitted through seed pieces taken from infected stalks. It may also be transmitted by zoospores of the fungus in free water flowing through cane fields.

Host range

Sclerophthora macrospora has a wide range of both tropical and temperate graminaceous hosts. Besides sugarcane, it infects barley, maize, oat, rice, sorghum, and wheat, and more than 40 grasses, including *Axonopus, Brachiaria, Coix, Digitaria, Echinochloa, Eleusine, Panicum, Pennisetum, Polypogon, Setaria, Sorghum* (Holliday, 1980; Steindl and Steib, 1961).

Epidemiology

The disease is usually prevalent in flooded or poorly drained soils in low-lying areas subject to flooding. Several grasses in or around cane fields may be a source of infection in disease areas.

Economic importance

Because of its sporadic occurrence and limitation to very specific conditions, the disease is considered of minor importance. However, when infection does occur, crop loss can be severe.

Control

Care must be taken to avoid the use of infected planting material. Therefore, in areas where the disease is known to occur, nurseries should not be established in low-lying areas liable to flooding. Use of resistant varieties cannot be resorted to because their assessment is not practicable on account of the sporadic nature of the disease.

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Sclerotium sheath rot
(Previously known as Red rot of the leaf sheath)

Claude Ricaud, revised by Andre B. Gama

Cause

Agroathelia rolfsii (Sacc.) Redhead & Mullineux [Syn. *Athelia rolfsii* (Sacc.) C.C. Tu & Kimbr.; *Sclerotium rolfsii* Sacc., and *Corticium rolfsii* (Sacc.) Curzi], basidiomycete fungus.

Geographical distribution

Australia, Belize, Brazil, Burkina Faso, Central African Republic, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Fiji, Guam, India, Indonesia, Iran, Japan, Madagascar, Malaysia, Martinique, Mauritius, Mexico, Myanmar, Nigeria, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, South Africa, Sri Lanka, Taiwan, Thailand, Trinidad and Tobago, Uganda, USA, Vietnam, Zimbabwe.

Symptoms

Typical sheath rot symptoms initially appear on the lower leaf sheaths causing orange-red discoloration in irregularly shaped areas that are sharply different from healthy tissue (Figure 1). As symptoms progress, the pathogen penetrates and spreads to the inner leaf sheaths, and slightly binds them together. Fungal mycelium grows between the leaf sheaths in the infected regions, forming small, round, brownish sclerotia approximately 1 mm in diameter.

The rind beneath the infected sheath areas may also be invaded, resulting in light brown discoloration with distinct margins like those on the sheaths. As the stalks mature, affected cells become necrotic and dry, leading to shallow cankers. In advanced stages, the leaf sheaths become necrotic, and tissue disintegration between the veins may occur, leaving the vascular bundles exposed. Additionally, leaf blades may exhibit slight discoloration and premature drying (Takashi Matsumoto and Abbott, 1964).

Diagnosis

Agroathelia rolfsii can be recognized by the sclerotia and absence of typical fructifications of other sheath-invading fungi such as *Cytospora sacchari* and *Colletotrichum falcatum* (Syn. *Glomerella tucumanensis*). The fungus can be isolated on potato dextrose agar. Cultural and physiological characteristics have been described by Mordue (1974). Specific PCR primers were developed to identify the pathogen affecting another host, *Amorphophallus paeoniifolius* (Jeeva *et al.*, 2010).



Figure 1. Delineated orange-red discoloration of the sugarcane leaf sheath (ISSCT).

Strains of the pathogen and host range

Agroathelia rolfsii is able to infect *Arachis*, *Amorphophallus*, *Phaseolus*, and numerous other legumes, as well as species of *Beta*, *Brassica*, *Cannabis*, *Cinchona*, *Citrus*, *Cucumis*, *Ficus*, *Lycopersicon*, *Musa*, *Oryza*, *Solanum*, and a diverse range of other crops, ornamental plants, and weeds (CABI, 2021).

Transmission and epidemiology

The fungus is a facultative parasite and survives in the soil, on crop residues and weed and other crop hosts, since it affects a wide range of plant species. The disease is favored by warm, wet weather.

Economic importance

The disease is not of economic importance on sugarcane.

Control

No control measure is warranted.

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Sett and seedling rots

Shamsul A. Bhuiyan

Cause

Agroathelia rolfsii (Sacc.) Redhead & Mullineux [Syn. *Athelia rolfsii* (Sacc.) C.C. Tu & Kimbr.; *Corticium rolfsii* (Sacc.) Curzi; *Sclerotium rolfsii* Sacc.], basidiomycete fungus.

Geographical distribution

Australia, India.

The disease was first reported on sugarcane in Australia in 2019 (Bhuiyan *et al.*, 2019) and later in India in 2022 (Balamurugan *et al.*, 2022; Gopi *et al.*, 2023). However, the fungus *A. rolfsii* (previously called *Corticium rolfsii* or *Sclerotium rolfsii*) is also the causal agent of a disease in sugarcane named Sclerotium sheath rot (previously called Red rot of the leaf sheath) that occurs in many countries and that is considered to be a minor disease (see "Sclerotium sheath rot", p. 242).

Symptoms

General symptoms include germination failure of planting materials and poor crop establishment (Figure 1), discoloration of the internal stalk tissues, ranging from pale red to red, especially in the vascular bundles and central pith, the presence of white mycelium in the pithy internal stalk tissues and at cut ends (Figure 2), water-soaked, light brown lesions located at the base of young plants and lower leaf sheaths, the appearance of white mycelia and sclerotia on infected tissues and in the soil near diseased plants (Figures 3 and 4), and ultimately death of young plants. In the initial stage, typical collar rot symptoms are observed on infected young plants, including wilting of the lower leaves. Later, grey or dark-brown sclerotia may be present at the bottom and across the infected leaf sheath (Figure 4).

Diagnosis

The fungus can be easily isolated on common culture media such as potato dextrose agar (PDA) or corn meal agar (CMA). Sometimes, a massive white, coarse mycelial growth can be observed on sugarcane trash or at the bottom of affected plants. Small (0.5 to 2.5 mm), whitish to dark brown sclerotia may be present within the mycelial mat (Figure 5). Growth on culture media or on the organic substrates are fan-shaped, projected outwards (Figures 5 and 6). The fungal hyphae are thin-walled, hyaline with sparse septation, and often clamp connections are observed on one or both side of the hyphal septae (Figure 7). Molecular diagnosis can be performed by amplification, sequencing and phylogenetic analysis of various loci of the pathogen's genome such as the ITS region or the 18S rDNA and 28S rDNA (Mascarenhas and Quesada-Ocampo, 2024; Terrones-Salgado *et al.*, 2023; You *et al.*, 2024).



Figure 1. Germination failure and poor crop establishment in a field trial site: inoculated row (front) and uninoculated row (back row) (S.A. Bhuiyan).

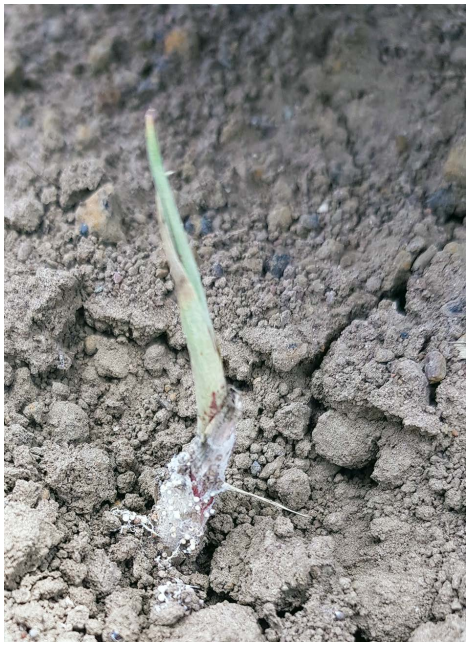

Figure 3. Field infected seedling showing lesions on the leaf sheath and white fungus with small grain-like sclerotia at the bottom (S.A. Bhuiyan).



Figure 2. Internal symptoms in sugarcane sett tissues after inoculation with *Agroathelia rolfsii*. Note the whitish mycelial growth from degraded pithy tissue (red arrows) (S.A. Bhuiyan).



Figure 4. Young sugarcane plant infected by *Agroathelia rolfsii* showing lesions on the leaf blade and the leaf sheath. Small whitish sclerotia are visible across the leaf sheath (S.A. Bhuiyan).



Figure 5. White coarse thread-like fungal mycelia of *Agroathelia rolfsii* on potting media with sugarcane seedlings (S.A. Bhuiyan).

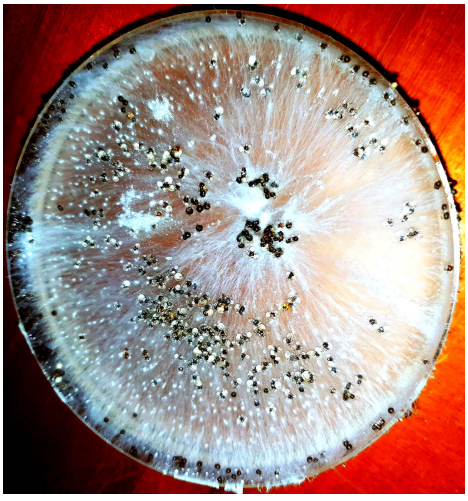

Figure 6. *Agroathelia rolfsii* growing on potato dextrose agar (PDA). Note the white and dark brown sclerotia on the fungal mycelial mat growing toward the edge of the plate (S.A. Bhuiyan).

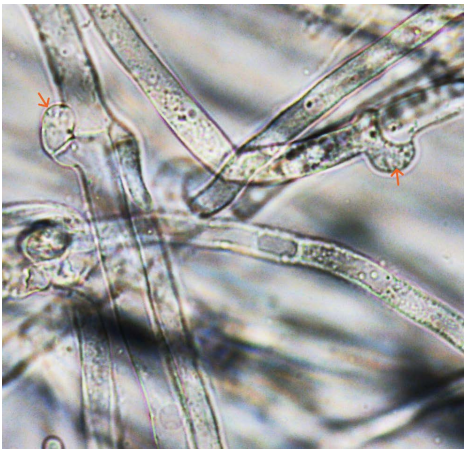

Figure 7. *Agroathelia rolfsii* mycelia observed with a microscope. Note the clamp-connections (red arrows), a characteristic feature of basidiomycete fungi (S.A. Bhuiyan).

Strains of the pathogen

No new strains have been reported in sugarcane.

Transmission

The pathogen can be transmitted through contaminated soil or plant materials infected with sclerotia or mycelia. Additionally, irrigation or floodwater can disseminate sclerotia from one field to another.

Host range

A. rolfsii has a wide host range and infects at least 500 species of plants from 100 families, including legumes, crucifers, and cucurbits (Punja, 1985). Related grass species,

such as maize, Bermuda grass, and several turf grasses, are known hosts of *A. rolfsii* (Aycock, 1966; Kator *et al.*, 2015). No information has been available on the pathogenicity of the fungus on wild relatives of sugarcane.

Epidemiology

Limited information is available on the epidemiology of *A. rolfsii* in sugarcane. Two modes of infection were described (Bhuiyan *et al.*, 2019): (a) the pathogen infects through the cut end of sugarcane setts or possibly through cracks and injuries, and (b) it infects the soft tissues of seedlings or young plants, leading to wilting and seedling death. Information from other crops suggests that the disease can establish and spread rapidly in warm and humid conditions.

Economic importance

Limited information is available on the importance of this disease. In Australia, reduction of sett germination was estimated up to 60 and 70% in glasshouse and field conditions, respectively (Bhuiyan *et al.*, 2019). In India, incidence of the disease ranged from 17–100% in germplasm clones (Gopi *et al.*, 2023).

Control

No control measures are currently in place for this disease.

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Sheath rot

A. Salem Saumtally

Cause

Cytospora sacchari E.J. Butler, deuteromycete fungus.

Geographical distribution

Argentina, Australia, Bangladesh, Belize, Bolivia, Borneo, Brazil, Cuba, Dominican Republic, Fiji, Guyana, Hawaii, India, Indonesia, Iran, Japan, Malawi, Mauritius, Mexico, Mozambique, Nicaragua, Pakistan, Panama, Peru, Philippines, Puerto Rico, South Africa, Sri Lanka, Taiwan, Tanzania, Thailand, Türkiye, USA, Venezuela, Zimbabwe (CABI, 2005).

Symptoms

The pathogen is more commonly encountered as an infection of the leaf sheath, but it can also attack cuttings, stubble pieces, young shoots, and stalks. Brick-red patches on the outside and inside of the sheath are characteristic symptoms of the disease (Figure 1). When the infection is severe, the entire leaf sheath turns reddish-brown (Figure 2) and eventually becomes necrotic (Figure 3). Leaves dry out from the tip, droop downwards but remain attached to the stalk. Following necrosis of the sheath, pycnidia, which are the fungus' fruiting bodies emerge from the plant tissue (Figure 4). They are visible to the naked eye as black spines over the surface of the sheath. These structures are hard and make the sheath rough to touch. Under humid conditions, droplets containing masses of spores of the pathogen exude from the pycnidia, and the spores are dispersed. Under dry conditions, pycnidia production is sparse (Caceres and Forbes, 1965). Sheath rot may be widespread in a particular variety, and severe infection causes the stalks to die out (Ahmed *et al*., 1979).

Infected setts develop a brownish-black coloration of the rind from where pycnidia erupt. On infected stubble, the pathogen can be observed on the above-ground parts. Internal tissues turn greyish while a dull brown color is present on the outside. The disease tends to be more pronounced on older ratoon stubbles than on younger ones (Abbott, 1938; Caceres and Forbes, 1965). As is the case for the leaf sheath and cuttings, black pycnidia eventually develop on the necrotic tissue of the stubble. The disease can attack the lower nodes of stalks (Abbott, 1938), kill young shoots, and cause a reduction in the number of suckers (Caceres and Forbes, 1965). Whole stools or only a few canes per stool may become infected (Luthra *et al*., 1938). Pycnidia can be readily observed on the different parts of the stalk, particularly when the cane has completely shriveled.

Diagnosis

The pathogen is usually isolated on culture media from symptomatic material but it also has been isolated from leaf scars and dormant buds. The pathogen can be grown



Figure 1. First symptoms of sheath rot: reddish-brown patches on the sugarcane leaf sheath (A.S. Saumtally).



Figure 2. Reddish-brown patches covering the entire leaf sheath (A.S. Saumtally).



Figure 3. Severe symptoms of sheath rot with dry and necrotic lesions (A.S. Saumtally).



Figure 4. Black, protruding pycnidia, characteristic of sheath rot on the dead leaf sheath of sugarcane (H. Mungur).

on vegetable juice agar or potato dextrose agar at 28–30°C (Caceres and Forbes, 1965). Growth is best in the dark and pycnidia are more readily produced under illumination. The fungus belongs to the subdivision Deuteromycotina and has been described by Sivanesan (1983). The hyphal structure bearing the conidia is stromatic, separate, sub-epidermal, dark brown to black, and protrudes above the surface. Locules converge to a single ostiole. The conidiophores are hyaline, septated, and branched with dimensions 15–27 × 3–5 µm. Conidia are aseptate, smooth with rounded ends, straight or curved, about 2.5–3.5 × 1.5 µm (Figure 5).

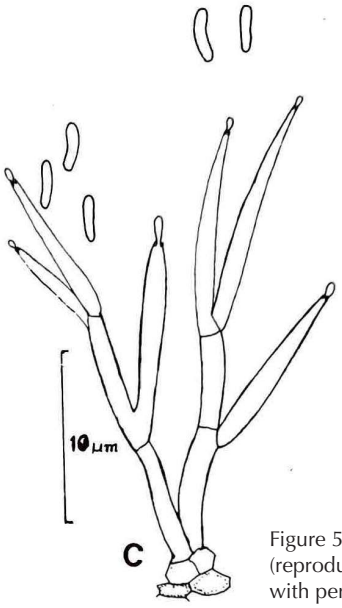

Figure 5. Conidiophores and conidia of *Cytospora sacchari* (reproduced from Sivanesan A., 1983; with permission from CAB International, Wallingford, UK).

As the rind disease fungus (*Phaeocytostroma sacchari*) also attacks the same plant parts *as C. sacchari*, and producing similar fruiting bodies, the two pathogens have at times been confused. Conidiophores of *P. sacchari* are smaller (5–20 × 1.5–2.0 µm), while conidia are larger (11–14.5 × 3.5–5.0 µm) than those of *C. sacchari*.

The identity of the pathogen can be confirmed by amplification and sequencing of genomic loci conserved within the genus *Cytospora*. *C. sacchari* formed clade 168 in a phylogenetic tree constructed with a combined sequence of ITS, *act1*, *rpb2*, *tef1-α*, and *tub2* genes of 399 isolates of *Cytospora* from 32 countries (Lin *et al.*, 2024).

Strains of the pathogen

No strains of pathogenic significance have been reported.

Transmission

The conidia of the fungus are spread by water. The pathogen can overwinter on dead sheath and stalk tissues in the soil, and plants can be inoculated by burying infected sheaths in the soil (Caceres and Forbes, 1965). Inoculum, in the form of fungal growth on sterile barley seeds added to soil, also caused infection.

Host range

The disease has been reported mainly on *Saccharum officinarum* and *S. spontaneum*. It is postulated that the susceptibility of some *Saccharum* hybrids comes from *S. spontaneum* (Abbott, 1964). Sheath rot has also been observed on *S. barberi, S. sinense* and *Sorghum vulgare*.

Epidemiology

Cytospora sacchari is considered to be a weak pathogen, being able to invade only damaged plant tissue or sheaths becoming senescent. Attenuated virulence might be related to a putative partitivirus infecting *C. sacchari* (Peyambari *et al*., 2014). Stress such as cold temperatures, poor drainage, moisture deficit, and the presence of other diseases like red rot and root rot favor the development of sheath rot (Edgerton, 1955).

Economic importance

The disease is not considered economically important. A 5% yield reduction due to mortality of tillers and poor growth of affected stalks has been reported in Louisiana (Abbott, 1938).

Control

Nitrogen fertilization applied either in normal or excessive amounts reduced infection (Abbott, 1938). In one instance, the disease was reduced by as much as 24% in plots that were heavily fertilized compared to normally fertilized cane. Sheath rot can be controlled using resistant varieties. However, the disease has not been sufficiently important to apply any selection pressure for resistance.

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Smut

Shamsul A. Bhuiyan

This chapter has been updated and rewritten based on the original chapter entitled "Smut" and authored by Jack C. Comstock in 2000.

Cause

Sporisorium scitamineum (Syd.) M. Piepenbr., M. Stoll & Oberw (Syn. *Ustilago scitaminea* Syd.).

Geographical distribution

Afghanistan, Antigua and Barbuda, Argentina, Australia, Bangladesh, Barbados, Belize, Bolivia, Brazil, Burkina Faso, Burundi, Cambodia, Cameroon, Chad, China, Colombia, Congo, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominica, Dominican Republic, Ecuador, Egypt, El Salvador, Eswatini (former Swaziland), Ethiopia, Gabon, Ghana, Guadeloupe, Guatemala, Guyana, Haiti, Hawaii, Honduras, Hong Kong, India, Indonesia, Iran, Iraq, Jamaica, Japan, Kazakhstan, Kenya, Madagascar, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Nigeria, Oman, Pakistan, Panama, Papua New Guinea, Paraguay, Peru (?), Philippines, Portugal, Puerto Rico, Réunion, St Kitts and Nevis, Senegal, Somalia, South Africa, Sri Lanka, Sudan, Taiwan, Tanzania, Thailand, Turkmenistan, Trinidad and Tobago, Uganda, Uruguay, USA, Venezuela, Vietnam, Zambia, Zimbabwe.

Symptoms

The most noticeable sign of *S. scitamineum* infection is the long, whip-like sorus that emerges from the growing point and often extends above the tops of the infected plant (Figure 1) (Marques *et al*., 2017). These whips are sometimes serpentine-shaped as illustrated on the cover page of this book. A sorus is an elongated internode that can grow up to 3 m. It consists of central pithy tissues surrounded by a thin layer of teliospores (Figure 2). Less obvious are sori produced from side shoots originating from lateral buds of mature plants (Figure 3). Sori originated from side-shoots, are smaller, and often hidden within crop canopy. In susceptible varieties, plants may produce pencil-like grassy shoots, with up to 25 individual shoots (Figure 4). In rare cases, *S. scitamineum* can cause unusual symptoms such as stem galls, mass proliferation of side shoots, smut 'whips' on flowering panicles, and phyllody or vegetative reversion of floral panicles (Bhuiyan *et al*., 2021) (Figure 5).

Diagnosis

Sugarcane smut can easily be diagnosed by the presence of typical whip-like sori that are produced from the apex of the plant or side shoots originating from lateral buds.



Figure 1. Whip-like sori of sugarcane smut that emerged from the growing points extended above the tops of the infected plants (S.A. Bhuiyan).



Figure 2. A typical smut sorus, an extension of sugarcane internode covered by brown or black-brown teliospores (S.A. Bhuiyan).



Figure 3. Smut sorus developed from a lateral or side shoot (S.A. Bhuiyan).



Figure 4. Grassy shoots produced from infected sugarcane plants as manifested by the proliferation of pencil-like tillers (S.A. Bhuiyan).



Figure 5. Unusual symptoms of sugarcane smut: smut on flowering panicles (left), and phyllody or vegetative reversion of floral panicles (right) (S.A. Bhuiyan).

However, it can take six weeks to more than 12 months to develop a characteristic whip-like sorus. Teliospores are either identified directly under a microscope or form the base material for DNA assays. Teliospores of *S. scitamineum* are small (diameter of 5.5–7.5 µm; Comstock, 2000) dark-brown, circular and microscopically punctate;

microscopic observation of these teliospores is usually sufficient to confirm the presence of the disease. Molecular-based detection methods such as PCR, qPCR and loop-mediated isothermal amplification (LAMP) were reported for the early detection of the whip smut pathogen in the plant tissues (Dalvi *et al*., 2012; Su *et al*., 2016).

Strains of the pathogen

Smut races have been reported in various sugarcane-growing regions such as Hawaii, Pakistan, the Philippines, Brazil, Nigeria, Taiwan, China, Kenya, and Australia (Bhuiyan *et al*., 2015; Ferreira and Comstock, 1989). The prevalence of races is a contentious issue (Ferreira and Comstock, 1989). Variable smut reactions have been reported for the same cultivars in different countries, and some of those may be related to different inoculation methods and environmental conditions. The presence of pathogenic races could complicate the development of resistant cultivars. However, the biological fitness, or the ability of the potential new races of the sugarcane fungus to survive or maintain virulence, is unclear. Most smut fungi can readily hybridize between races, making it difficult to maintain the same level of pathogenicity (Bakkeren and Kronstad, 1996).

Transmission

Sugarcane smut primarily spreads through two mechanisms: a) airborne teliospores are carried by wind currents, and b) movement of systematically infected vegetative canes used as planting materials. Both of these main mechanisms allow the pathogen to spread internationally and rapidly across infested regions and countries. Other minor methods of transmission include surface-contaminated plant materials, soil, farm machinery, and clothing (Bhuiyan *et al*., 2021).

Host range

Most commercial sugarcane varieties are natural hosts of *S. scitamineum*. Several species of *Saccharum* and related genera have been found to be infected when artificially inoculated with *S. scitamineum* teliospores. Although some clones of *S. officinarum* and *S. spontaneum* were reported to be highly susceptible to smut, Artschweiger and Brandes (1958) considered *S. officinarum* as resistant, susceptibility to smut resulting from admixtures of *S. spontaneum* aiming at controlling mosaic disease. However, other wild relatives of sugarcane such as *Saccharum barberi*, *S. sinense*, *S. robustum*, *Erianthus saccharoides* (*Bothriochloa saccharoides*), and *Rottboellia cochinchinensis* showed varying degrees of resistance. Apart from sugarcane and its relatives, there are no known hosts of *S. scitamineum* (Bhuiyan, 2024).

Epidemiology

Sugarcane smut can spread rapidly via teliospores carried in atmospheric air movement. The main factors favoring disease developments are the presence of susceptible cultivars and favorable weather conditions (Swaraj *et al*., 2026). Susceptible varieties permit the pathogen to proliferate, allowing it to release millions of teliospores into the environment. Very high infection pressure from nearby susceptible varieties can cause intermediate or resistant varieties to have a high degree of disease incidence (Bhuiyan *et al*., 2021). Hot and dry weather favors disease development and spread. Other factors, such as ratooning and abiotic stress, can accelerate disease incidence.

Economic importance

Loss of sugarcane productivity depends on environmental conditions and cultivar susceptibility, which may range from 0 to >60%. In Louisiana, a yield loss of up to 0.7% was estimated for every 1% increase in disease incidence (Hoy *et al*., 1986). In Australia, the loss of raw sugar or cane production was estimated to be greater than 42% for susceptible varieties and less than 6% for resistant varieties (Bhuiyan *et al*., 2022).

Control

Sugarcane smut can be managed effectively using resistant varieties. In most sugarcane industries, the primary smut management tool is resistant varieties. However, risk of smut infestation and associated yield loss can be significantly minimized by implementing a well-planned integrated disease management (IDM) strategy. The main components of IDM are a) resistant varieties, b) removal of heavily infested sugarcane crops, c) planting disease-free seed cane, d) treating planting materials with hot water and fungicides before planting, and e) roguing of diseased plants and planting materials. For quarantine purposes, hot water treatment at 52°C for 45 minutes, followed by dipping in triazole fungicides (e.g., flutriafol and propiconazole) will eradicate the smut fungus from planting materials (Bhuiyan *et al*., 2012).

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Sooty mould

Claude Ricaud, revised by Andre B. Gama

Cause

Capnodium spp. and *Fumago sacchari* Speg., deuteromycete fungi; both are saprophytic fungi growing on honeydew associated with sugarcane-feeding insects such as aphids, leaf hoppers, mealybugs and scale insects.

Geographical distribution

Argentina, Australia, Bangladesh, Brazil, Cuba, Ecuador, Eswatini, Guadeloupe, Guyana, Hawaii, India, Indonesia, Jamaica, Japan, Madagascar, Malaysia, Martinique, Mauritius, Mozambique, Myanmar, Nicaragua, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Solomon Islands, South Africa, Taiwan, Thailand, Trinidad and Tobago, Uganda, USA, Vietnam, Zimbabwe.

The disease is probably present in most sugarcane growing countries in the world where honeydew-producing insects are present. *Fumago sacchari* was first detected in Tucuman, Argentina.

Symptoms

The leaves and stalks of affected plants develop a black film or crust resembling soot, which can be easily scraped off the leaves (Figures 1 to 3). Chlorotic patches may appear on the leaves beneath the covered areas. The disease is associated with a sticky secretion produced by the insects. After rain, the films are typically washed off the leaves (Abbott, 1964; Martin, 1938).

Diagnosis

Microscopic examination reveals the presence of fungal mycelium. Black growth can be easily peeled off the leaves by scraping their surface.

Strains of the fungus, transmission and host range

No information is available.

Epidemiology

Factors favoring the build-up of populations of the insects involved are conducive to sooty mould development. Sooty mould is usually most pronounced during periods of dry weather. Increasing populations of insects that produce honeydew will lead to higher incidences of sooty mould in the field.



Figure 1. Lower leaves covered with a black film (P. Rott).



Figure 2. Sugarcane plants severely affected by sooty mould (P. Rott).

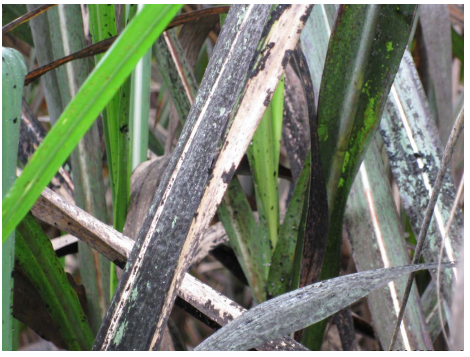

Figure 3. Sugarcane leaf covered by sooty mould (Y. Huang).

Economic importance and control

Although the fungi are not parasitic to the plant, they can slightly hinder its growth by covering sugarcane leaves and interfering with photosynthesis. However, these attacks are typically sporadic and not economically significant. Consequently, treatment is generally not necessary, except in multiplication plots where controlling insect infestations may be required.

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Tawny rust

Sharon McFarlane

Cause

Macruropyxis fulva L.A. Martin, S.A. McFarl. & L.A. Castl., basidiomycete fungus.

Geographical distribution

Eswatini, Mozambique, South Africa, Zimbabwe.

Symptoms

Symptoms of tawny rust are characterized by uredinia (pustules) that develop on the sugarcane leaf blade. Uredinia are brown to dark red-brown and similar in color to those of brown rust caused by *Puccinia melanocephala* (Martin *et al*., 2017a). Individual uredinia are linear, up to 3 mm in length, usually coalescing to form longer lesions (Figures 1 and 2). They are most common on the abaxial leaf surface but also occur on the adaxial surface, particularly when infections are severe (Figures 3 and 4). Uredinia tend to be grouped towards the tip of the leaf and cause severely infected leaves to die prematurely. Sporulation is profuse, with bright orange urediniospores erupting from uredinia when infections are fresh (Figure 5). Purpling of the leaf around lesions may be observed on some varieties (Figure 6). Like *Puccinia kuehnii*, the causal agent of orange rust, *M. fulva* infects sugarcane of any age (Figures 7 and 8).



Figure 1. Uredinia (pustules) of tawny rust on a sugarcane leaf blade (S. McFarlane).



Figure 2. Single uredinium (pustule) of tawny rust on a sugarcane leaf blade (S. McFarlane).



Figure 3. Tawny rust symptoms on the upper leaf surface (S. McFarlane).



Figure 6. Purpling of the leaf infected by *Macruropyxis fulva* (S. McFarlane).



Figure 8. Symptoms of tawny rust in a field with mature sugarcane (S. McFarlane).

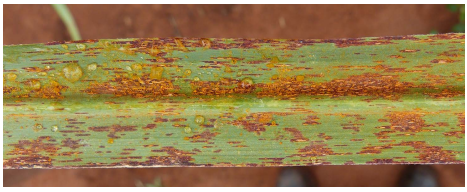

Figure 4. Tawny rust symptoms on the lower leaf surface (S. McFarlane).



Figure 5. Profuse sporulation of *Macruropyxis fulva* on the lower side of a sugarcane leaf (S. McFarlane).



Figure 7. Symptoms of tawny rust in a field with young sugarcane (S. McFarlane).

Diagnosis

Tawny rust can be diagnosed by symptomatology, particularly when infections are fresh. As the uredinia age and urediniospores disperse, symptoms may resemble brown rust (Figure 9). Urediniospores are usually bright orange, but may be orange-red, red-brown to hyaline, typically measuring 25–32 × 20–26 µm (Martin *et al*., 2017a). They are mostly ellipsoid–obovoid, echinulate with abundant, evenly-spaced spines (Figure 10 to 12). The cell wall is uniform and between 1.5 and 4.8 µm thick. Apical thickening is rare. Germ pores, if present, are sparse in number and randomly positioned. No sterile paraphyses have been observed and telia have not been recorded.



Figure 9. Old tawny rust uredinia that resemble those of brown rust (S. McFarlane).

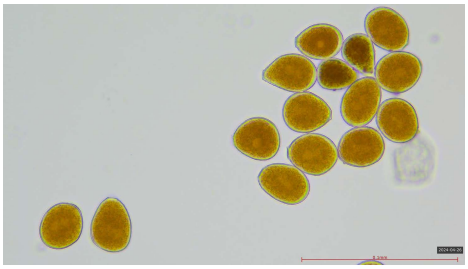

Figure 10. Urediniospores of *Macruropyxis fulva* observed by light microscopy (S. McFarlane).

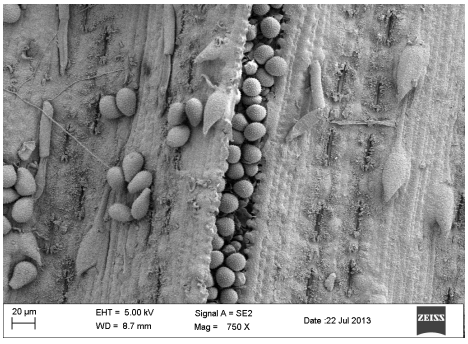

Figure 11. Uredinia and urediniospores of *Macruropyxis fulva* observed by scanning electron microscopy (L. Martin).

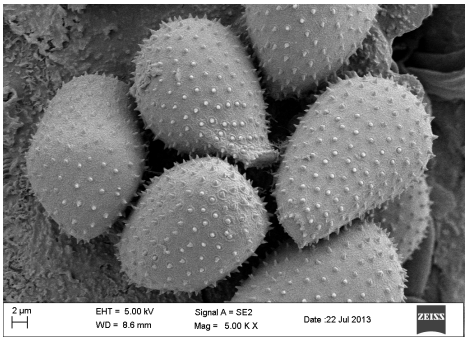

Figure 12. Urediniospores of *Macruropyxis fulva* observed by scanning electron microscopy (L. Martin).

A touchdown polymerase chain reaction assay was developed to rapidly and accurately identify *M. fulva* and discriminate between the three rusts infecting sugarcane (Martin *et al*., 2017b).

Strains of the pathogen

No information is available regarding genetic or pathogenic variation in *M. fulva*.

Transmission

As with brown and orange rust, tawny rust is spread by wind and water splash.

Host range

Sugarcane hybrids (*Saccharum* spp.) and *Miscanthidium capensis* are hosts of *M. fulva* (Martin *et al*., 2017a) (Figure 13). It is thought that *M. capensis* represents a native original host of the pathogen (Rutherford, 2018).



Figure 13. Symptoms of tawny rust on *Miscanthidium capensis* (S. McFarlane).

Epidemiology

Tawny rust tends to occur in autumn, spring and early summer. The disease is rarely seen in late summer. Based on laboratory studies, conditions for the germination of *M. fulva* urediniospores are optimal in the presence of free moisture, and at temperatures between 18 and 22°C (Martin *et al*., 2015).

Economic importance

Yield losses of 10% have been recorded for tawny rust (Martin *et al*., 2015), but growers have reported losses of up to 40% when infections are severe. Disease severity, persistence and associated yield losses vary from year to year depending on the climatic conditions and the sugarcane varieties being grown.

Control

Varietal resistance is the most effective means of managing tawny rust (McFarlane *et al*., 2024). Strobilurin-triazole fungicide formulations, applied twice with a spray interval of 28–30 days, have been registered against tawny rust. These are most effective when applied to young, actively growing sugarcane at the early stages of the disease outbreak.

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Twisted leaf

Muqing Zhang, Yixue Bao

Cause

Epicoccum sorghinum (Sacc.) Aveskamp, Gruyter & Verkley [Syn. *Leptosphaeria sacchari* Breda de Haan; *Phoma sorghina* (Sacc.) Boerema, Dorenb. & Kesteren], and *Phoma* sp., ascomycete fungi.

P. sorghina var. *saccharum*[15] (Current name = *E. sorghinum*) has been identified as the causal agent of twisted leaf of sugarcane that was first reported in sugarcane in mainland China in 2012 (Lin *et al*., 2014 and 2015). Symptoms similar to those described below were previously attributed to *Phoma* sp. in other geographical locations but the species involved remains to be determined (Lin *et al*., 2014; Hsien, 1980; Laurel *et al*., 2021).

Geographical distribution

China, Philippines.

E. sorghinum (Synonym *L. sacchari*) is also the causal agent of another sugarcane disease called ring spot (see "Ring spot", p. 229) that is widely distributed in sugarcane growing locations around the world (Laurel *et al*., 2021).

Symptoms

In the early stages of infection, sugarcane leaves exhibit mild yellowing and a wrinkled appearance (Figure 1). As the disease progresses, leaves undergo twisting and curling, leading to shredding of the leaf tissue (Figure 2). In advanced stages, the entire plant develops intense chlorosis, with leaves twisting into a compact mass, significantly reducing the photosynthetic surface area. This damage can result in severe leaf necrosis and ultimately leaf death (Figure 3).

Diagnosis

The diagnosis of twisted leaf disease in sugarcane relies on a combination of symptom identification, pathogen isolation, and molecular analysis. When cultured on potato dextrose agar (PDA), *E. sorghinum* forms moderately fast-growing colonies, reaching 3–5 cm in diameter within 7–10 days. The colony texture transitions from fluffy to velvety or granular as it matures, with the reverse side appearing brown to black (Figure 4). Mature pycnidia release hyaline, ovoid conidia, which are essential for fungal reproduction (Lin *et al*., 2015).

15. Note by the editors: *P. sorghina* var. *saccharum* or *P. sorghina* var. *sacchari* are not recognized as valid taxa names in the Index Fungorum database (https://www.indexfungorum.org/names/names.asp).



Figure 1. Symptoms of twisted leaf disease in sugarcane: mild yellowing and wrinkling of the leaves at early stage (M.Q. Zhang).



Figure 2. Symptoms of twisted leaf disease in sugarcane: progressive leaf twisting, curling, and shredding (M.Q. Zhang).



Figure 3. Severe manifestation of twisted leaf disease in sugarcane, characterized by leaf yellowing, necrosis, and extensive shredding (M.Q. Zhang).

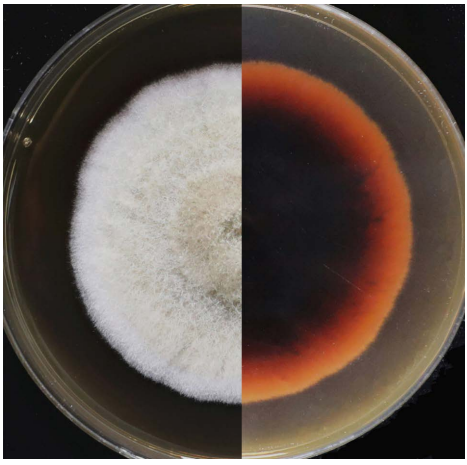

Figure 4. Colony of *Epicoccum sorghinum* darkening to gray or black on PDA medium (M.Q. Zhang).

The pathogen can be accurately identified through PCR amplification, sequencing, and phylogenetic analyses (Bao *et al*., 2020 and 2024; Lin *et al*., 2015; Shi *et al*., 2025). Molecular characterization includes genetic markers, including 18S nrDNA (SSU), 28S nrDNA (LSU), β-tubulin (TUB), translation elongation factor 1-alpha (TEF-1α), and internal transcribed spacer (ITS) regions (ITS 18S, 5.8S, and 28S nrDNA).

The complete genome of *E. sorghinum* (strain BS2-1, previously classified as *P. sorghina* var. *saccharum*) from sugarcane has been sequenced (Bao *et al*., 2020). The genome assembly consists of 22 contigs with an N50 contig length of 1.92 Mb, a total size of 33.12 Mb, and a GC content of 52.12%. It is predicted to encode approximately 8,000 genes, including 147 transcription factor families. Among them, homeobox transcription factors have been identified as playing crucial roles in fungal growth, development, conidiation, secondary metabolite regulation, and pathogenicity (Bao *et al*., 2020 and 2024).

These combined diagnostic approaches ensure accurate detection and identification of *E. sorghinum*, facilitating effective disease management in sugarcane.

Strains of the pathogen

Strain BS2-1 has been successfully isolated from multiple sugarcane varieties cultivated in Guangxi, China (Lin *et al*., 2014). However, genetic diversity among *E. sorghinum* isolates from sugarcane appears to be low, and no variations in pathogenicity have been reported to date. A closely related but unidentified *Phoma* species has been detected in sugarcane caryopses in Brazil, though it exhibited weak pathogenicity in sugarcane (Sanguino and Tokeshi, 1980). Differences between pathogen strains causing ring spot (VNOC62a and VNR26) and twisted leaf (BS2-1) disease remain to be further investigated.

Transmission

The primary mode of transmission for the pathogen is through conidial spores, which are dispersed by wind, rain, and soil movement. Rainwater facilitates spore transport to the growing points of sugarcane, where they germinate and initiate infection under warm and humid environmental conditions. Additionally, the pathogen can persist in plant debris, enabling its survival in the soil and contributing to future infections.

Host range

E. sorghinum (previously classified as *P. sorghina* and *Leptosphaeria sacchari*) infects a wide range of wild and cultivated plant species across multiple genera (Shi *et al*., 2025). It has been identified as a pathogen in major crops, including maize, rice, sorghum, sugarcane, and wheat. However, the pathogenicity of isolates from non-sugarcane hosts remains to be fully characterized in sugarcane.

Epidemiology

E. sorghinum is a widespread fungal pathogen capable of thriving in diverse environments, including soil, plant debris, and water. It primarily spreads through airborne conidia, with transmission favored by warm, humid conditions. Additionally, the fungus can be disseminated *via* rainwater and soil, further facilitating infection. *E. sorghinum*

persists in plant residues, enabling its survival between growing seasons and contributing to recurring infections. Its ability to co-infect with other fungal pathogens further amplifies its impact on plant health.

Economic importance

Twisted leaf disease has a significant impact on sugarcane production by causing leaf deformation and reducing ratooning ability, which leads to decreased yields and lower sugar content. This reduction in both crop quality and quantity directly affects the economic value of sugarcane as a source of sugar and bioenergy. Consequently, the disease results in substantial financial losses for farmers and the sugar industry due to declined productivity and increased management costs.

Control

The cultivation of resistant sugarcane varieties remains the most effective strategy for controlling twisted leaf disease. Additionally, balanced fertilization and strategic crop rotation play crucial roles in eliminating inoculum sources and improving field ventilation, particularly during the early and middle growth stages of sugarcane. Effective disease prevention and management measures include soaking stalk cuttings in a 50% carbendazim solution before planting and applying targeted fungicidal treatments at the onset of infection. Furthermore, the use of unmanned aerial vehicles (UAVs) for precision spraying at optimal flying heights and with recommended liquid volumes enhances application efficiency, contributing to more effective disease control.

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Veneer blotch

Sidney Suma, revised by Philippe Rott

Cause

Deightoniella papuana D.E. Shaw (1959), *Magnaporthaceae*.

Geographical distribution

Indonesia, Papua New Guinea, Philippines, Solomon Islands.

Symptoms

The first sign of the disease is the development of small oval spots of a light green or straw color surrounded by a thin reddish-brown margin, occurring on any part of the lamina (Koike, 1988). The lesion then develops, usually symmetrically, flanked by two longitudinally elongated lesions resembling wings on each end of the primary lesion. Further secondary lesions develop one after another like wings, each pair progressively larger than the preceding pair. The original lesion can be surrounded by up to 12 of these wings and the entire group of lesions can measure up to 60 cm long and 1–1.5 cm wide (Shaw, 1964) (Figure 1). Sometimes asymmetrical wings can occur, as can secondary chains parallel to the main lesion. The whole lesion has a beautiful veneer pattern, particularly on the upper surface, hence the name.

The upper surface of the wings is outlined with a dark reddish border, 0.5–1 mm wide, while the interior of the lesion is initially light green, later turning to greeny-grey or straw-colored and finally to light brown (Kohler *et al*., 1997). On the lower surface, the conidiophores form a dense black pile in older lesions. Occasionally conidiophores also form on the upper surface but never on newly formed wings on the apex and base of the lesion. Spores can be found with the conidiophores, although detached from them.

Diagnosis

The diagnosis of the disease is based on visual symptoms. The causal agent has not been successfully cultured on artificial media. Light microscopy observation of the conidia shows that the mature conidia are globose to nearly ovoid, aseptate, minutely echinulate, very pale olivaceous-brown in color, 15–20 × 15–18 µm, and have no basal scar. The spores germinate rarely in water, but readily if leaf pieces are added to the spore suspension. Germinating spores retain their circular shape and produce one germ tube, or rarely two per spore.

Strains of the pathogen

There is no information on the presence of strains of *D. papuana*.



Figure 1. Group of elongated leaf lesions surrounded by a thin reddish-brown margin (P. Rott).

Transmission

The disease is transmitted via the movement of air-borne conidia of the pathogen. The movement of infected leaves attached to "tops" used as planting material is a possible mode of spread over long distances. The practice is common amongst natives of Papua New Guinea in relation to the cultivation of noble canes (*Saccharum officinarum*).

Host range

The hosts of the disease include *S. edule, S. officinarum, S. robustum, S. spontaneum*, as well as interspecific hybrids of *Saccharum*.

Epidemiology

The disease occurs in various ecological zones depending on the host. In Papua New Guinea, it occurs on *S. spontaneum* on sandy beach soil and grassland at sea level, and in abundance on *S. robustum* along river banks and valleys in the highlands at 2500–3000 m above sea level (Shaw, 1984).

Economic importance

Veneer blotch is a minor disease and of no economic importance.

Control

No control regimes have been administered to control this disease.

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White speck (White rash)

Claude Ricaud, revised by Andre B. Gama

Cause

Elsinoe sacchari T.C. Lo (Syn. *Sphacelocema sacchari* T.C. Lo), ascomycete fungus.

Geographical distribution

Brazil, Cuba, Federated States of Micronesia, French Polynesia (Tahiti), French Guiana, Guam, Hawaii, Japan, Malaysia, Marshall Islands, Philippines, Puerto Rico, Taiwan, Thailand, USA, Vietnam.

Symptoms

Leaf lesions occur usually on the blades or midrib and occasionally on the sheaths. They are initially minute, yellow or purple, round to elliptic spots 1–4 × 0.4–1 mm on average, with the long axis parallel to the veins (Figure 1). They later turn light brown and finally whitish-grey, sometimes with a reddish margin (Figures 2 and 3). They may coalesce to form elongate narrow streaks. Examination with a stereoscopic microscope reveals the presence of erumpent and powdery fruiting bodies (Koike, 1988; Lo, 1964).

Diagnosis

Microscopic examination reveals the ascomycete fructifications. Asci measure 10.3–13 × 9.6–10.6 µm, and ascospores 8.6–10 × 3–3.3 µm. Additional characteristics of the fungus have been described by Lo (1964) and Sivanesan and Waller (1986). Molecular diagnosis can be performed by amplification, sequencing, and BLASTn analysis of several fungal housekeeping genes (Shi *et al.*, 2026).

Strains of the pathogen

No information is available on strain variation.

Transmission and epidemiology

The disease is probably spread by wind but there is no report to that effect or on its epidemiology.

Host range

No other host of the fungus has been reported.

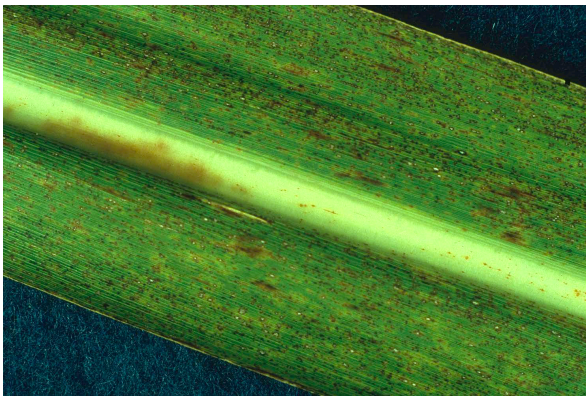

Figure 1. Initial leaf symptom: minute, round to elliptic spots (S. Matsuoka).



Figure 3. Whitish-grey elliptic spots on the leaf blade that takes a brown discoloration (J.H. Daugrois).



Figure 2. Whitish-grey elliptic spots on the leaf blade (A. S. Saumtally).

Economic importance and control

The disease is of minor economic importance and does not warrant any control measures. A 10-year survey conducted in Cuba revealed that although 47% of their varieties showed high severity of white rash, height and diameter of stalks were not affected (Pelegrín *et al.*, 2012).

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Wilt

Govind Pratap Rao, Virendra P. Agnihotri,
revised by Rasappa Viswanathan and Govind Pratap Rao

Cause

Fusarium sacchari (E.J. Butler) W. Gams, ascomycete fungus.

Butler and Khan (1913) described the causal organism of wilt as *Cephalosporium sacchari* E.J. Butler. Subsequently, other scientists reported *Fusarium moniliforme* var. *subglutinans* as the pathogen of stalk rot and wilt. Booth (1971) and Gams (1971) reviewed the taxonomy of *C. sacchari* and considered it as *F. moniliforme* var. *subglutinans*. Since the name *C. sacchari* was reported in 1913 and *F. moniliforme* var. *subglutinans* in 1925 for the same taxon, Gams (1971) renamed it *F. sacchari* (E.J. Butler) W. Gams, and both fungi were made synonyms. Although other *Fusarium* species were associated with sugarcane wilt, only *F. sacchari* was confirmed as the causal agent of this disease in sugarcane (Viswanathan *et al*., 2011 and 2026). This pathogen can also cause both wilt and pokkah boeng in a same cane, and the pokkah boeng phase usually precedes wilt (Viswanathan *et al*., 2017 and 2023).

Geographical distribution

Argentina, Australia, Bangladesh, Barbados, Belize, Benin, Brazil, Burkina Faso, Central African Republic, China, Colombia, Cuba, El Salvador, Guadeloupe, Guatemala, India, Iran, Malaysia, Mauritius, Mexico, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Pakistan, Panama, Philippines, Réunion, St Kitts and Nevis, Senegal, South Africa, Sudan, Thailand, Togo, Trinidad and Tobago, Uganda, USA, Vietnam, Zimbabwe.

Symptoms

Symptoms appear from planting onwards throughout the entire crop cycle. Diseased setts fail to germinate or young shoots die (Figure 1). During tillering and grand growth phase, leaves of affected stalks show yellow discoloration and desiccation (Figures 2 to 4). Sugarcane stalks lose their firm attachment to the soil and the stalk rind becomes pale (Figure 5). Eventually, infected clumps completely dry and die, thus leaving hollow stalks that result in a total milling loss (Figure 6). Typical internal symptoms can be observed after splitting the diseased stalks longitudinally (Figures 7 to 9). The internal pith tissue, particularly the one of the lower internodal tissues, takes a light to dark purplish-brown color. The tissue discoloration is more prominent at the internodes. The vascular tissues appear as dark reddish-brown streaks that pass through the internodal tissues from one internode to another. The wilt affected stalks do not emit a foul odor, and there are no white spots in internodal tissues that are typical of red rot affected sugarcane. However, shrinkage of internodal tissues is common due to the loss of sap movement resulting in the formation of longitudinal, spindle-shaped hollow cavities in the middle of the internodes.



Figure 1. Typical wilt symptoms in the diseased sett and death of the germinated shoots (R. Viswanathan).



Figure 2. Wilt affected canes showing discoloration of the spindle leaves (R. Viswanathan).



Figure 3. Yellow discoloration of spindle leaves in wilt affected sugarcane (R. Viswanathan).



Figure 4. Yellowing and desiccation of leaves caused by wilt (R. Viswanathan).



Figure 5. Weakening of canes and rind discoloration after a wilt attack. Affected canes are marked with red arrows (R. Viswanathan).



Figure 6. Complete drying and withering of stalks due to wilt (R. Viswanathan).



Figure 7. Variation in internal symptoms of stalks affected by wilt (J.-C. Girard).

The mycelium and the spores of the pathogen sometimes fill these cavities. Tissue discoloration and cavity formation are relatively more prevalent in the basal portion of affected stalks. Occasionally, the nodal tissues alone exhibit pinkish patches with partial pith cavity formation. In the initial phase of the disease, the roots of affected plants do not display distinct symptoms. Infected roots die later on, after the vascular bundles of the shoot become brownish as the stalks die (Figure 10). Diseased stalks are completely dry with no millable cane, thus causing a 100% crop loss.



Figure 8. Characteristic pith cavities and tissue discoloration in wilt affected stalks (R. Viswanathan).



Figure 9. Longitudinal section of a stalk showing light to dark purplish-brown color of the internodal tissue (R. Viswanathan).



Figure 10. Reduced root mass and root death in wilt affected sugarcane;
Left: healthy, right: diseased (R. Viswanathan).

Diagnosis

Externally, sugarcane wilt can be diagnosed by the drying of the stalks and leaves. Affected stalks display typical internal wilt symptoms. However, reddening of the internodal tissue in wilt affected canes creates confusion with red rot and borers. Paleness of the rind tissues and loose attachment of the stalks in the soil are additional indicators for wilt diagnosis. The disease is confirmed by isolation of *F. sacchari* from the nodes, internodes, or roots (Poongothai *et al*., 2014a). Potato dextrose agar medium supports luxuriant growth of the fungus (morphology of the pathogen's spores has been described in the chapter on Fusarium diseases, p. 165). Several standardized PCR assays are available to detect *F. sacchari* and to confirm its presence in infected tissues. Partial gene of translation elongation factor (TEF-1α) of the pathogen is commonly targeted in PCR assays, and species identification is confirmed after DNA sequencing (Viswanathan *et al*., 2017).

Strains of the pathogen

F. sacchari exhibits extensive variability in cultural and morphological characteristics of micro and macroconidia (Poongothai *et al*., 2014a and b). Great variation was also recorded in pathogenicity of strains of the fungus after inoculation of differential sugarcane varieties (Viswanathan *et al*., 2017 and 2022a).

Transmission

F. sacchari is primarily transmitted by infected stalk cuttings. Secondary spread occurs through wind, rain and irrigation water. The fungus can survive in sugarcane debris in the soil and transmission occurred in soils with a pH of 7.0–8.0 (Srinivasan, 1964).

Host range

F. sacchari causes disease in sugarcane but this fungus can also infect other plants such as maize, rice and sorghum (Viswanathan, 2020).

Epidemiology

Wilt is favored by neutral to alkaline soils (pH 7.0–8.0) and low moisture regimes. The wilt fungus can survive in the soil for 2.5–3 years (Viswanathan, 2013 and 2020). A high C/N ratio in the soil also favors disease development. Moisture stress during summer months with high daily temperatures and low humidity increases incidence of wilt (Viswanathan and Rao, 2011). Wilt is more dangerous and causes enormous damage to the crop when associated with red rot, pineapple sett rot, stalk borers or scale insects (Viswanathan *et al*., 2006). These disease/insect complexes have become a threat to sugarcane growers (Agnihotri, 1983; Viswanathan 2018). Although root borer infestation is often believed to play a role in wilt severity, there is no evidence for that (Viswanathan *et al*., 2022a). *F. sacchari* can cause wilt and pokkah boeng in the same sugarcane clump and usually the wilt phase is initiated after the pokkah boeng phase. During the pokkah boeng phase, infections by the pathogen are initially confined to the foliage but, as the disease progresses, *F. sacchari* can enter the stalks through upper nodes and cause top rot and knife-cut symptoms, or become systemic in the entire stalk. These systemic infections cause wilt in the same season or they are carried over to the next crop through seed cane (Viswanathan *et al*., 2017 and 2023).

Economic importance

Wilt has caused significant losses in India where several epidemics occurred in the past decades (Agnihotri, 1983; Viswanathan, 2013 and 2018). The disease adversely affects germination, causes withering of young tillers as well as stalk death. Drying of shoots takes place when sugarcane is 4–5-month-old. During the monsoon period, considerable mortality of affected shoots occurs and occasionally all the shoots in a clump dry out, resulting in a crop stand with numerous gaps. Wilt incidence is always higher in ratoon crops compared to plant crops. Besides yield reduction, wilt also causes 14.6–25.8% reduction in juice extraction and 3–20% losses in sugar recovery. Juice quality is deteriorated due to a decrease in sucrose content and an increase in reducing sugars, gums, titratable acidity, flavonoids and soluble salts (Singh and Waraitch, 1981). These changes adversely affect the processing of white sugar in the mill. Complete crop losses are common during severe epidemics in India, especially in ratoon crops (Figure 11).



Figure 11. Total destruction of a sugarcane crop due to wilt (R. Viswanathan).

Control

No single method is known to control wilt in sugarcane but the use of resistant varieties is the best management strategy. The planting of healthy cuttings, obtained through sanitary selection, is indispensable. Ratooning of wilt affected crops should be avoided to prevent the build-up of inoculum and severe crop losses. Proper irrigation should be applied as drought conditions favor wilt development. A 3-year crop rotation has also been recommended for *F. sacchari* contaminated soils. Wilt incidence can be reduced appreciably under field conditions by applying 40 ppm boron or manganese to wilt contaminated soils. Similarly, sett treatment with boron (40 ppm) before planting also reduced wilt incidence (Ganguly, 1964). Soil amendment with boric acid (15 kg/ha) and sett treatment with aretan (0.1%) followed by soil drenching with 0.2% carbendazim considerably reduced wilt incidence as well as the population densities of the pathogen (Agnihotri and Singh, 1989). However, when the use of fungicides is needed to control sugarcane wilt, environment-friendly fungicides should be used as aretan has hazardous effects.

The antagonistic *Trichoderma* formulated on pressmud, a by-product of sugar mills, is effective in managing wilt in endemic locations (Viswanathan *et al*., 2012). Integrated control of wilt and pokkah boeng includes mechanized sett treatment with propiconazole, soil application of *Trichoderma harzianum* (strains Th1 and Th2), and foliar applications of propiconazole (Viswanathan *et al*., 2014). Selective use of resistant parents in breeding programs will increase the production and distribution of wilt resistant varieties. Two screening assays (artificial inoculation of stalks and

soil inoculation) were developed to assess resistance to wilt in sugarcane. These assays are currently implemented by different sugarcane research centres under the All India coordinated project on sugarcane, a national network cane improvement program in India (Viswanathan *et al*., 2022b).

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Yellow spot

Jean Claude Autrey, A. Salem Saumtally

Cause

Passalora koepkei (W. Krüger) U. Braun & Crous [previously *Mycovellosiella koepkei* (Krüger) Deighton], ascomycete fungus.

Geographical distribution

Argentina, Australia, Bangladesh, Barbados, Borneo, Brazil, Burundi, Brunei, Cambodia, Cameroon, Chad, China, Colombia, Congo, Costa Rica, Côte d'Ivoire, Cuba, El Salvador, Fiji, Gabon, Ghana, Guadeloupe, Guatemala, Guyana, Honduras, India, Indonesia, Japan, Kenya, Madagascar, Malaysia, Martinique, Mauritius, Mexico, Myanmar, New Caledonia, Pakistan, Panama, Papua New Guinea, Philippines, Puerto Rico, Réunion, Samoa, Singapore, Solomon Islands, South Africa, Sri Lanka, Taiwan, Tanzania, Thailand, Trinidad and Tobago, Uganda, USA, Vietnam, Venezuela.

Symptoms

The symptoms of yellow spot disease may appear on the second to third youngest leaf as minute chlorotic dots in highly susceptible varieties, otherwise the spots are usually present on the older leaves (Figure 1). As infection progresses, the spots increase in size, are irregular in shape, coalesce if they are abundant (Figure 2) and may reach 10 mm wide on mature leaves (Ricaud and Autrey, 1989). The spots tend to be yellow in color and later turn slightly reddish. However, in some varieties, especially those with red stalks, the spots are brick red to reddish-brown (Figure 3). Spots are visible on both sides of the leaf lamina, including the lower surface of the midrib. Symptoms are rarely present on the leaf sheath, while other parts of the stalk are not infected. Severe infection leads to necrosis (Figure 4) from the tip to the base of the leaf and ultimately defoliation occurs, particularly in flowering stalks of highly susceptible varieties. In non-flowering stalks, as the conditions favoring infection subside, a flush of green leaves appears.

Diagnosis

The spots are different in size, shape, and color from those induced by other foliar pathogens such as the causal agents of brown stripe, brown spot, eye spot, red leaf spot (purple spot) or ring spot, but they can be confused with them. Under moist conditions and heavy infection, *P. koepkei* typically produces a dirty grey mass of conidiophores, conidia, and hyphae on the lower side of the lamina. This mycelium mass is localized and not as profuse and widespread as the down associated with downy mildew. Examination of the infected tissues under the light microscope reveals the presence of

a primary and secondary mycelium, septate conidiophores 50 µm long by 4.5–6.0 µm wide, borne in fascicles of up to 15. The conidia are fusiform to obclavate-fusiform, straight, and 3-septate, not constricted with an obtuse apex (Figure 5) (Deighton, 1979; Mulder and Holliday, 1974). The dimensions of the conidiophores and conidia vary (Ricaud and Autrey, 1989).



Figure 1. Distinct irregular yellow spots on sugarcane leaves (H. Mungur).



Figure 2. Coalescing leaf spots as yellow spot infection progresses (H. Mungur).



Figure 3. Appearance on some varieties of reddish lesions instead of yellow lesions (H. Mungur).



Figure 4. Necrosis of older leaves as a result of severe yellow spot infection (H. Mungur).

The fungus can be isolated on Czapek medium with difficulty (Hughes and Ocfemia, 1961). The colonies are yeast-like in appearance but give a fluffy, cottony grey mycelium with slow growth and low sporulation on potato dextrose agar (Figure 6). However, growth and sporulation are enhanced in culture on cane leaf agar, especially with extracts of leaves from susceptible varieties (Autrey and Saumtally, 1986).



Figure 5. Conidiophores and conidia (blue) of *Passalora koepkei* (J.C. Autrey).

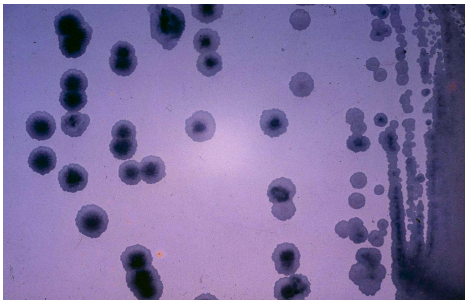

Figure 6. Yeast-like colonies of *Passalora koepkei* on Czapek medium (J.C. Autrey).

Strains of the pathogen

Changes in varietal reaction have been observed in Australia (Egan, 1970 and 1972) and in Mauritius (Ricaud *et al.*, 1978; MSIRI, 2008), but there is no definite proof of strain variation in the yellow spot pathogen. No variation of the ITS region was found among 29 isolates of *P. koepkei* from Mauritius (Ranghoo-Sanmukhiya and Dookhun-Saumtally, 2007).

Transmission

Wet and warm weather is conducive to yellow spot infection. In Mauritius, infection significantly declines as the temperature drops at the onset of winter and despite continuing high humidity. Conidia are splashed by rain and wind-blown to other leaves within the same field or adjacent ones, thus establishing new disease symptoms. Yellow spot is not transmitted by sugarcane stalk cuttings. Seasonal carry-over takes place by the inoculum on leaves of bull shoots and volunteer growth (Ricaud and Autrey, 1989).

Host range

Wild canes such as *Saccharum robustum, S. spontaneum* and *S. edule* (Dignadice and Orillo, 1953; Roach, 1975), *Miscanthus sinensis* and *M. floridulus* (Deighton, 1979) are affected by the disease. Apart from wild canes, the role of the other hosts in disease carry-over is unclear.

Epidemiology

Rainfall and the resulting high relative humidity within the canopy, together with warm temperatures, are the most determinant environmental factors in the severity of the disease. Spore germination of *P. koepkei* is optimum when relative humidity is high (>99%) and temperatures reach 20–30°C (Magarey, 2003). These conditions correlate with the period of high disease incidence in the wet tropical coast of Queensland, Australia and the superhumid zone in Mauritius. Epidemics occur when susceptible varieties are extensively grown in areas prone to infection.

Economic importance

Epidemics of yellow spot have been reported in Australia, India, Indonesia, and Mauritius. Losses have been assessed in Mauritius in variety Saipan 17 (S17) in which about 8,000t of sugar were lost in 1978 and an estimated 20,000t from 1976 to 1980 (Ricaud *et al.*, 1980). Under epidemic conditions, the disease is particularly damaging in profusely flowering varieties in which the reduction of sucrose content can range from 10 to 25% (Ricaud *et al.*, 1980; Autrey *et al.*, 1983). In Australia, based on fungicide field assays, yellow spot was estimated to reduce the commercial cane sugar (CCS) of susceptible varieties by up to 2 units early in the harvest season, while in the 2001 season, cane and sugar yield losses reached up to 20% (Magarey, 2003).

Control

Only resistant or slightly susceptible varieties should be cultivated in high-rainfall areas. A screening method for resistance to yellow spot has been developed (Ricaud, 1974). Infection in both flowered and non-flowered stalks should be assessed. Breeding for resistance has been rewarding in Australia (Hughes and Ocfemia, 1961) and in Mauritius (Ricaud *et al.*, 1983). However, the development of resistant varieties to replace susceptible ones has been challenging both in Australia and Mauritius (Magarey, 2003). A major quantitative trait locus (QTL) has been identified for yellow spot resistance (Aljanabi *et al*., 2007; Parmessur and Dookun-Saumtally, 2016). Delaying harvest in non-flowering, late-maturing varieties was found to be a good strategy to allow the cane to recover after the peak of infection (Ricaud *et al.*, 1983). Chemical control with benomyl was resorted to in variety S17 before the removal of the variety from high rainfall areas of Mauritius (Autrey *et al.*, 1983). However, fungicide application is considered a palliative since the ultimate objective is the use of resistant varieties.

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Zonate leaf spot

Jack C. Comstock, revised by Jack C. Comstock and Sushma Sood

Cause

Gloeocercospora sorghi D.C. Bain & Edgerton ex Deighton, hyphomycete fungus.

Geographical distribution

Papua New Guinea, Samoa, Solomon Islands, USA.

Symptoms

Initial symptoms include small necrotic lesions that grow to 5–6 cm in size, forming concentric rings of alternating dark and light brown zones of necrotic tissue (Figure 1). These lesions are frequently found on older leaves (Bain and Edgerton, 1943; Luttrell, 1950).

Diagnosis

The fruiting structures of *Gloeocercospora sorghi* usually develop on leaf tissue placed in moist chambers within 24 to 48 h. As described on sorghum, the fungus forms salmon-colored sporodochia over stomatal openings, bearing conidia in a pinkish-slimy

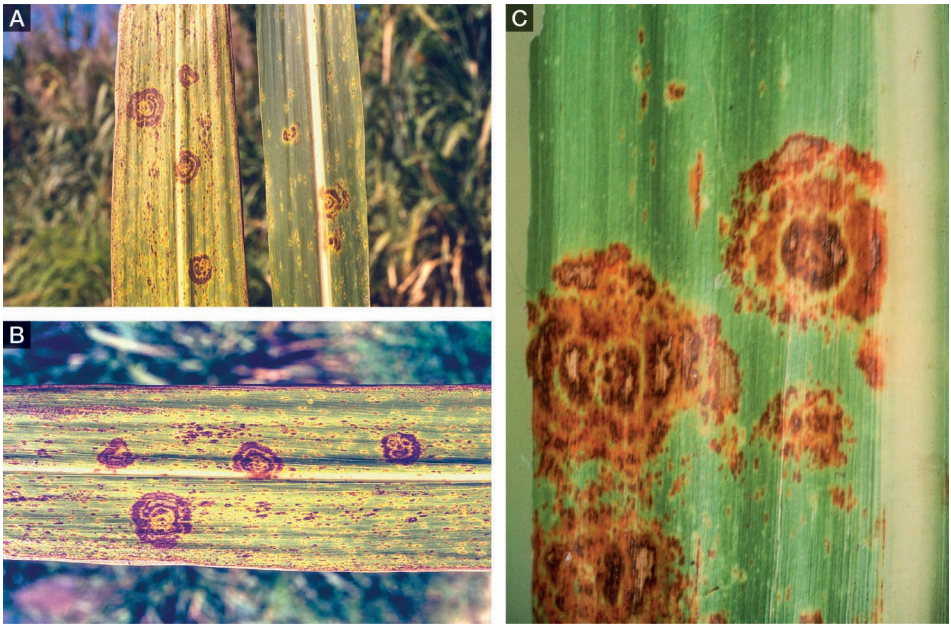

Figure 1. Necrotic leaf lesions made up of series of concentric rings (A and B: P. Rott; C: H. Koike).

matrix. The conidia are 20–195 µm long and 1.5 to 3.2 µm wide, hyaline, straight to curved, wider in the middle, smooth, and have one to seven septations (Deighton, 1971). *G. sorghi* produces black hemispherical sclerotia beneath the lower epidermis, measuring approximately 115–135 × 70–100 µm (Luttrell,1950). Molecular diagnosis can be performed by amplification and sequencing of the internal transcribed spacer (ITS) region of rDNA using primers ITS1/ITS4 (Jiang *et al*., 2018).

Strains of the pathogen

No information is available.

Transmission

Wind and rain-blown conidia are thought to spread the disease in sugarcane.

Host range

Besides sorghum and sugarcane, the host range of *G. sorghi* extends to bent Grass, Bermuda grass, Johnson grass, maize, Napier grass, pearl millet, Sudan grass (*Sorghum vulgare* var. *sudanense*), and possibly other grasses (Bain and Edgerton, 1943; Dean, 1965; Jiang *et al*., 2018; Kucharek, 1992).

Epidemiology

The disease is favored by hot humid weather.

Economic importance

Zonate leaf spot in sugarcane is considered of minor economic importance.

Control

Control is not necessary because of insignificant losses due to the disease.

References

Bain D.C., Edgerton C.W., 1943. The zonate leaf spot, a new disease of sorghum. *Phytopathology*, 33: 220–226.

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Deighton F.C., 1971. Validation of the generic name *Gloeocerospora* and the specific names *G. sorghi* and *G. inconspicua*. *Transactions British Mycological Society*, 57: 358–360. https://doi.org/10.1016/s0007-1536(71)80022-0

Jiang Y., Xu J., *et al*., 2018. First report of sorghum zonate leaf spot caused by *Gloeocercospora sorghi* in China. *Plant Disease*, 102:1033. https://doi.org/10.1094/pdis-08-17-1217-pdn

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Luttrell E.S., 1950. Grain sorghum diseases in Georgia – 1949. *Plant Disease Reporter*, 34: 45–52.

Minor fungal diseases

Philippe Rott, Jack Comstock, revised by Philippe Rott

Alternaria leaf blight

Cause: *Alternaria tenuissima* (Kunze) Wiltshire.

Geographical distribution: China.



Figure 1. Alternaria leaf blight: irregular straw-yellow blight lesions (J. Li).



Figure 3. Alternaria leaf blight: young sugarcane plant affected by Alternaria leaf blight (J. Li).



Figure 2. Alternaria leaf blight: numerous small black conidia on dead leaf tissue (J. Li).

Symptoms: initial symptom is plant wilting similar to the one caused by water stress. When the disease progresses, irregular straw-yellow and blighted lesions appear along the entire leaf blade. Under humid conditions, small black conidia can be seen in the dead leaf tissue (Figures 1 to 4).

Diagnosis: isolation of the pathogen (Figures 5 to 7) followed by amplification, sequencing, and BLASTn analysis of the internal transcribed spacers (ITS), histone 3 genes, and plasma membrane ATPase (Li *et al.*, 2021).



Figure 4. Alternaria leaf blight: diseased sugarcane field (J. Li).

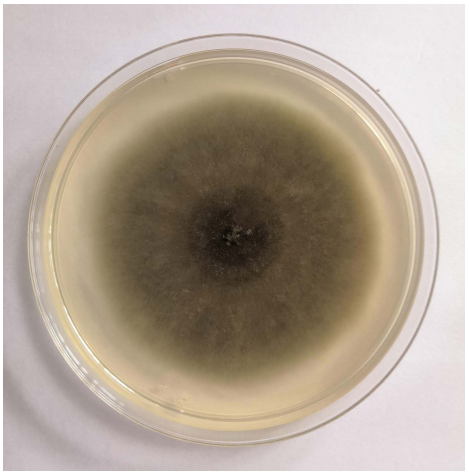

Figure 5. Alternaria leaf blight: colony of *Alternaria tenuissima* on potato carrot agar (J. Li).

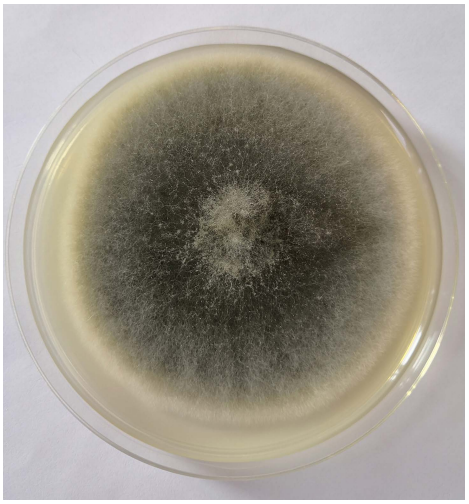

Figure 6. Alternaria leaf blight: colony of *Alternaria tenuissima* on potato dextrose agar (J. Li).

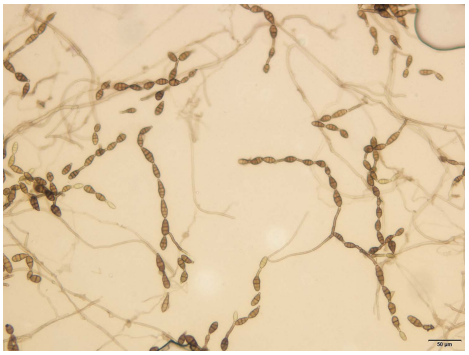

Figure 7. Alternaria leaf blight: conidia of *Alternaria tenuissima* on potato carrot agar (J. Li).

Reference:

Li J., Zhang R.Y., *et al.*, 2021. First report of *Alternaria tenuissima* causing leaf blight on sugarcane in China. *Plant Disease*, 105(4): 1222. https://doi.org/10.1094/pdis-07-20-1507-pdn

Apiospora mold

Cause: *Apiospora arundinis* (Corda) Pintos & P. Alvarado.

Geographical distribution: China.

Symptoms: sugarcane stems are moldy and the internal stalk tissues are grayish-white, red, or reddish-brown. Stalks can smell musty.

Diagnosis: amplification, sequencing, and BLASTn analysis of several genes such as the internal transcribed spacer region (ITS rDNA), the translation elongation factor 1-α gene (TEF1), or the β-tubulin gene (TUB2).

References:

Liao J., Jiang W., *et al*., 2022. First report of Apiospora mold on sugarcane in China caused by *Apiospora arundinis* (*Arthrinium arundinis*). *Plant Disease*, 106: 1058. https://doi.org/10.1094/pdis-02-21-0386-pdn

Liao J., Liang X., *et al*., 2023. Biocontrol ability of *Bacillus velezensis* T9 against *Apiospora arundinis* causing Apiospora mold in sugarcane. *Frontiers in Microbiology*, 14: 1314887. https://doi.org/10.3389/fmicb.2023.1314887

Basal stem rot

Cause: *Agroathelia rolfsii* (Sacc.) Redhead & Mullineux [Syn. *Athelia rolfsii* (Sacc.) C.C. Tu & Kimbr.; *Corticium rolfsii* (Sacc.) Curzi; *Sclerotium rolfsii* Sacc.], basidiomycete fungus. *Agroathelia rolfsii* is also the causal agent of Sclerotium sheath rot (page 242) and Sett and seedling rots (page 244).

Geographical distribution: China.

Symptoms: red rotting at the base of the stalk and the leaf sheath; severe symptoms include decomposition of the epidermis and the aerial roots. These symptoms can also be observed at the upper part of the stalk.

Diagnosis: amplification, sequencing, and BLASTn analysis of several genes such as the internal transcribed spacer region (ITS), the mitochondrial small subunit (SSU), and the nuclear ribosomal large subunit (LSU).

Reference:

Lao S.-C., Luo Z.-X., *et al*., 2024. First report of basal stem rot on sugarcane (var. Badila) caused by *Sclerotium rolfsii* in China. *Plant Disease*, 108: 3652. https://doi.org/10.1094/pdis-07-24-1460-pdn

Black rot

Cause: *Catunica adiposa* (E.J. Butler) C. Mayers & T.C. Harr. [Syn. *Ceratocystis adiposa* (E.J. Butler) C. Moreau].

Geographical distribution: Australia, Brazil, China, Dominican Republic, Hawaii, India, Indonesia, Panama, Peru, Taiwan, USA.

Symptoms: purplish to black, soft watery rot of sugarcane seed cuttings; rot often confined to the end of internodes; production of a mass of black, cottony fungal growth on the cut ends; symptoms resemble those of pineapple sett rot (Figure 8).

References:

Abbott E.V., 1964. Black rot. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds)2. Amsterdam, The Netherlands, Elsevier Publishing Company, 99–10.

Byther R. S., 1971. Black rot of sugarcane cuttings in Hawaii. *Plant Disease Reporter*, 55: 7–9.

Sivanesan A., Waller, J.M., 1986. *Ceratocystis* Ell. & Halsted. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 14–16. Slough, UK, CAB International.



Figure 8. Black rot: longitudinal section of a sugarcane cutting showing purplish-black rotting of the internal tissue (Y.-K. Huang).

Black stripe

Cause: *Pseudocercospora atrofiliformis* (J.M. Yen, T.C. Lo & C.C. Chi) J.M. Yen.

Geographical distribution: Taiwan.

Symptoms: narrow, dark brown to black streaks 5–36 mm long by 0.5–1.2 mm wide on the leaf blades, between veins.

References:

Sivanesan A., Waller, J.M., 1986. *Pseudocercospora* Speg. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 49–51. Slough, UK, CAB International.

Yen W.Y., Lo T.C., Chi C.C., 1964. Black stripe. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 21–23.

Charcoal rot

Cause: *Macrophomina phaseolina* (Tassi) Goid.

Geographical distribution: Mexico.

Symptoms: firm and dark brown stem rot and root rot associated with chlorotic foliage and plant death. Development of black microsclerotia and pycnidia at the stem base.

Diagnosis: amplification, sequencing, and BLASTn analysis of the internal transcribed spacer region (ITS).

Reference:

Leyva-Mir S.G., Velázquez-Martínez G.C., *et al*., 2015. First report of charcoal rot of sugarcane caused by *Macrophomina phaseolina* in Mexico. *Plant Disease*, 99: 553. https://doi.org/10.1094/pdis-06-14-0652-pdn

Collar rot

Cause: *Hendersonia sacchari* Speg.

Geographical distribution: Argentina, Bangladesh, Brazil, India, Mauritius, Pakistan, Philippines, Sri Lanka.

Symptoms: wilting and internal rot (external symptoms similar to those of true red rot); withering of top leaves from the tips along the edges, with the midrib only remaining green; dry and pithy tissues in the upper internodes; tissues watery and brown with red streaks in tissues in the lower internodes; blackened and rotted roots arising from the basal nodes.

References:

Abbott E.V., 1964. Collar rot. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 105–107.

Sivanesan A., Waller, J.M., 1986. *Hendersonia* E. Butler. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 53–54. Slough, UK, CAB International.

Dry rot (see also "Red leaf midrib" caused by the same pathogen)

Cause: *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl.

Geographical distribution: Barbados, Brazil, Cameroon, Cuba, Dominican Republic, Guyana, India, Indonesia, Jamaica, Malaysia, Mexico, Myanmar, Philippines, Puerto Rico, Sri Lanka, Trinidad.

Symptoms: shrunken and shrivelled canes; reddened and dark colored internal stalk tissues; eruption of fruiting bodies of the pathogen in rows of elongate blisters on the rind (similar to those of rind disease); primarily on overripened cane.

Reference:

Abbott E.V., 1964. Dry rot. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 108–109.

Iliau

Cause: *Clypeoporthe iliau* (Lyon) M.E. Barr.

Geographical distribution: thought to be widely distributed but rarely reported (Australia, Brazil, Cuba, Hawaii, Mauritius, Philippines, USA).

Symptoms: binding of the leaf sheaths into a firm and tight case surrounding the growing point; distortion of emerging stems in moderately affected cane; frequent breakage of weakened young plants; death of the outer leaves, sheaths and stalks of severely affected cane (Figure 9).

References:

Martin J.P., 1964. Iliau. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 114–118.

Sivanesan A., Waller J.M., 1986. *Clypeoporthe* Höhnel. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 18–19. Slough, UK, CAB International.



Figure 9. Iliau: binding of leaf sheaths and distortion of emerging stem (S. Matsuoka).

Leaf blast

Cause: *Didymosphaeria taiwanensis* W.Y. Yen & C.C. Chi.

Geographical distribution: Taiwan.

Symptoms: yellowish, elongate and narrow spots parallel to the leaf blade veins; purplish-red lesions, 3–25 mm long by 0.5–1.0 mm wide on both leaf surfaces; entire leaf may appear purplish-red by numerous coalescing lesions; withering and death of affected leaves from the tip downwards.

Reference:

Yen W.Y., Chi C.C., 1964. Leaf blast. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 29–31.

Leaf splitting

Cause: *Peronosclerospora miscanthi* (T. Miyake) C.G. Shaw; *P. northii*, *Peronosclerospora* sp., *Mycosphaerella striatiformans* N.A. Cobb.

Geographical distribution: *Peronosclerospora miscanthi* in Papua New Guinea (?), Philippines and Taiwan; *P. northii* in Fiji; *Peronosclerospora sp.* in India; *Mycosphaerella striatiformans* in Fiji, Hawaii, Indonesia and Papua New Guinea.

Symptoms: narrow yellow to dark red leaf streaks, often extending the entire leaf; parenchyma tissue in streaks disintegrate between vascular bundles splitting leaves into numerous partially connected thread-like vascular bundles, appearing like long-tangled coarse hair; some stunted plants may die (Figure 10).

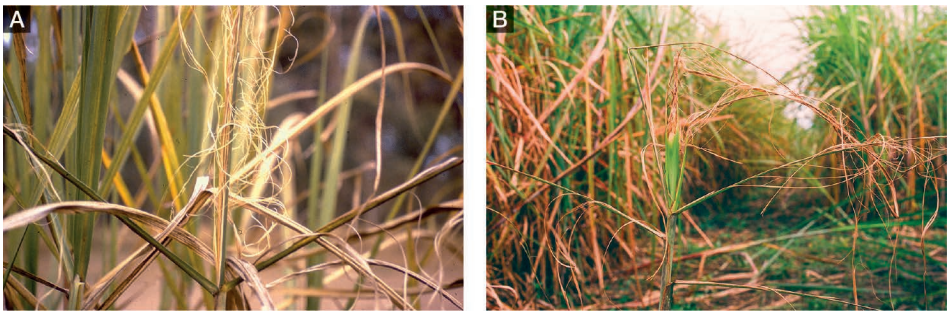

Figure 10. Leaf splitting: split leaves looking like long-tangled coarse hair (A: J.C. Comstock; B: P. Rott).

References:

Chu H.T., 1964. Leaf-splitting disease. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 37–39.

Sivanesan A., Waller, J.M., 1986. *Peronosclerospora* (Ito) Shirai & K. Hara. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 58–61. Slough, UK, CAB International.

Marasmius sheath rot and shoot blight; root rot

Cause: *Collybiopsis stenophylla* (Mont.) R.H. Petersen; *Marasmius sacchari* Wakker; *Rhizoctonia* sp.; unidentified oomycete.

C. stenophylla and *M. sacchari* are also the causal agents of a disease named Marasmius basal stem, root and sheath rot (see "Marasmius basal stem, root and sheath rot", p. 181).

Geographical distribution: thought to be widely distributed (Barbados, Indonesia, Mauritius, Peru, Philippines, Puerto Rico, USA).

Symptoms: white mycelium present between the leaf sheath and the stalk; rotting of the leaf sheaths near the base; death of the stem at or below the soil surface.

References:

Rands R.D., Abbott E. V., 1964. Basal stem, root and sheath rots. Part 1. *In*: *Sugar-Cane Diseases of the World, Vol 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 89–93.

Steindl D.R.L., Egan B.T., 1964. Basal stem, root and sheath rots. Part 2. *In*: *Sugar-Cane Diseases of the World, Vol 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 94–98.

Sivanesan A., Waller J.M., 1986. *Marasmius* Fr. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 11. Slough, UK, CAB International.

Myriogenospora leaf binding

Cause: *Myriogenospora aciculispora* Vizioli.

Geographical distribution: Argentina, Australia, Brazil, USA (Louisiana).

Symptoms: severe stunting and adherence of the tips of the unfolding leaves to the adjacent older leaves by a black stroma of the pathogen; the adhering older and emerging leaves form a circular hoop that may look like a whip; the shoot may die (Figure 11).



Figure 11. Myriogenospora leaf binding: adhering leaves (BSES).

References:

Abbott E.V., 1964. *Myriogenospora* leaf binding. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 40–42.

Sivanesan A., Waller, J.M., 1986. *Myriogenospora* Atk. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 35. Slough, UK, CAB International.

Phyllosticta leaf spot

Cause: *Phyllosticta hawaiiensis* Caum., *Phyllosticta* sp.

Geographical distribution: Colombia, Cuba, Hawaii, India, Malawi, Nepal, Panama, Philippines, Puerto Rico, South Africa, USA, Zambia, Zimbabwe.

Symptoms: small, irregular, light-brown straw-colored spots on the leaf sheath located close to where leaf attaches; fungus associated with older spots of ring spot. On the leaf blade, small and irregular yellow spots that become brown-reddish when ageing (Figure 12).

References:

Abbott E.V., 1964. Ring spot. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 52–58.

Martin J.P., 1938. *Phyllosticta* spot. *In*: *Sugar cane diseases in Hawaii*. Honolulu, Experiment Station of the Hawaiian Sugar Planters' Association, 84–85.



Figure 12. Phyllosticta leaf spot: leaf blade with numerous small and irregular brown-reddish spots (P. Rott).

Phytophthora sett rot
(formerly Phytophthora rot of cuttings)

Cause: *Phytophthora* spp.; *Phytophthora megasperma* Drechsler.

Geographical distribution: USA.

Symptoms: water-soaked rot of cuttings; orange-red streaking through interior of cuttings (Figure 13).



Figure 13. Phytophthora sett rot: orange-red streaking inside cuttings (J. Hoy).

References:

Steib R.J., 1964. *Phytophthora* seed piece rot. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 120–123.

Sivanesan A., Waller, J.M., 1986. *Phytophthora* de Bary. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 62–64. Slough, UK, CAB International.

Red leaf midrib
(see also "Dry rot" caused by the same pathogen)

Cause: *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl.

Geographical distribution: China.

Symptoms: initial symptoms are red spots on the leaf midrib that subsequently form elongated red lesions. Severe lesions cause leaf blades to break and hang down (Figures 14 to 17).



Figure 14. Red spots on the sugarcane leaf midrib (J. Li).



Figure 15. Elongated red lesions on the sugarcane leaf midrib (J. Li).



Figure 16. Broken sugarcane leaf blade (J. Li).



Figure 17. Numerous leaves affected by red leaf midrib in a sugarcane field (J. Li).

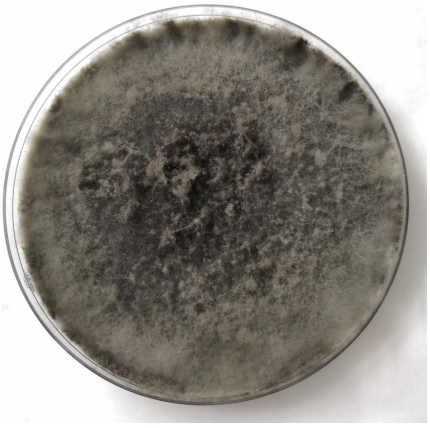

Figure 18. Colony of *Lasiodiplodia theobromae* on potato dextrose agar (J. Li).

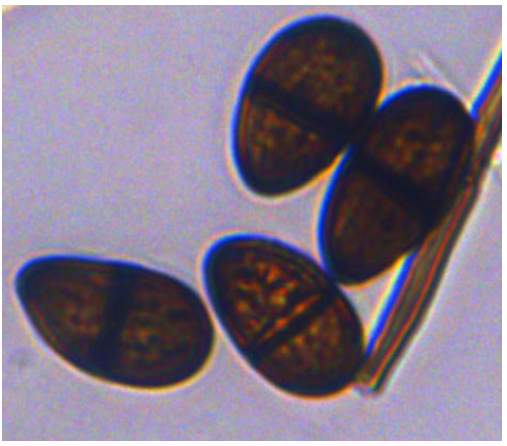

Figure 19. Mature conidia of *Lasiodiplodia theobromae* (J. Li).

Diagnosis: isolation of the pathogen (Figures 18 and 19) followed by morphological and molecular identification (Li *et al.*, 2023).

Reference:

Li J., Zhang R., *et al.*, 2023. First report of red leaf midrib lesions on sugarcane caused by *Lasiodiplodia theobromae* in China. *Plant Disease*, 107(6): 1952.

Seedling foliage blights

Cause: *Alternaria alternata* (Fr.) Keissl.; *Curvularia hawaiiensis* (Bugnic. ex M.B. Ellis) Manamgoda, L. Cai & K.D. Hyde; *Curvularia lunata* (Wakker) Boedijn; *Curvularia senegalensis* (Speg.) Subram.; *Bipolaris sacchari* (E.J. Butler) Shoemaker; *Exserohilum rostratum* (Drechsler) K.J. Leonard & E.G. Suggs.

Geographical distribution: Cuba, India, Taiwan (*Alternaria*); Argentina, Hawaii, India (*Curvularia*); widespread (*Bipolaris*); India, Puerto Rico, USA (*Exserohilum*).

Symptoms: blight of seedlings (*Alternaria* and *Bipolaris*). Circular to oval, scattered, reddish-brown leaf spots; the spots may enlarge into irregular patches and turn dark brown with age; the entire leaf blade may turn yellow and dry up (*Curvularia*). Narrow, reddish, elongated broken stripes that become dark brown with age; stunted seedlings (*Exserohilum*).

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Byther R.S., Steiner G.W., 1972. Four sugarcane seedling diseases in Hawaii: Causal agents, control and a selective medium for isolation. *Phytopathology*, 62: 120–124. https://doi.org/10.1094/phyto-62-120

Sivanesan A., Waller, J.M., 1986. *Alternaria* Nees. *In*: Sugarcane Diseases. *Phytopathological Paper*, 29, 39–40. Slough, UK, CAB International.

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Sivanesan A., Waller, J.M., 1986. *Drechslera* Ito. In: Sugarcane Diseases. *Phytopathological Paper*, 29, 43–47. Slough, UK, CAB International.

Sivanesan A., Waller, J.M., 1986. *Setosphaeria* Leonard & Suggs. In: Sugarcane Diseases. *Phytopathological Paper*, 29, 37–38. Slough, UK, CAB International.

Target blotch

Cause: *Helminthosporium* sp. Priode. In China, a leaf spot disease whose symptoms are identical to those of target blotch were attributed to *Bipolaris spicifera* (Bainier) Subram. (Lin *et al.*, 2012). *B. spicifera*, currently named *Curvularia spicifera* (Bainier) Boedijn, is also a synonym of *Helminthosporium spiciferum* (Bainier) Nicot.

Geographical distribution: China, Cuba, India, Indonesia, Japan, Papua New Guinea, South Africa, Thailand, USA, Zimbabwe.

Symptoms: minute, reddish-brown spots; straw-colored to brownish necrotic areas with irregular, concentric rings roughly resembling a target (Figures 20 and 21).

Diagnosis: isolation of the pathogen (Figures 22 and 23) followed by amplification, sequencing, and BLASTn analysis of the rDNA-ITS (internal transcribed spacer) (Lin *et al.*, 2012).

Reference:

Lin S.H., Huang C.H., *et al.*, 2012. First report of a leaf spot disease on sugarcane caused by *Bipolaris spicifera* in China. *Australasian Plant Dis*. Notes, 7: 51-53. https://doi.org/10.1007/s13314-012-0046-x

Todd E.H., 1964. Target blotch. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 74–77.

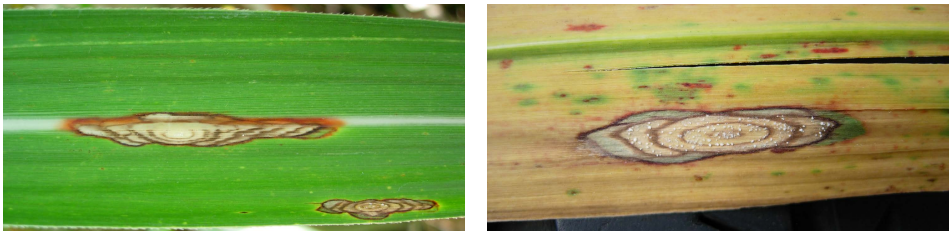

Figure 20. Concentric necrotic rings on a sugarcane leaf (L. Putra).



Figure 21. Leaf spots caused by *Curvularia spicifera* in China (S.H. Lin).



Figure 22. Conidia of *Curvularia spicifera* (S.H. Lin).

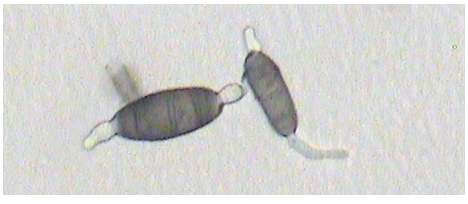

Figure 23. Bipolar-germinating conidia of *Curvularia spicifera* (S.H. Lin).

IV

Diseases caused by protozoa

Chlorotic streak

Robert C. Magarey, Brian T. Egan, revised by Chuong N. Ngo

Cause

Phytocercomonas venanatans (Ngo et al., 2018), protozoan.

Geographical distribution

American Samoa, Argentina, Australia, Brazil, Cambodia, China, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Egypt, Fiji, French Guiana, Grenada, Guadeloupe, Guyana, Haiti, Hawaii, Honduras, Indonesia, Jamaica, Kenya, Madagascar, Malawi, Mali, Martinique, Mauritius, Mexico, Mozambique, Nicaragua, Pakistan, Panama, Papua New Guinea, Peru, Philippines, Puerto Rico, Réunion, St Lucia, Samoa, South Africa, Surinam, Taiwan, Thailand, Trinidad and Tobago, Türkiye, Uganda, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

Chlorotic streak (CS) is characterized by yellow to creamy-white leaf streaks with broken, wavy, irregular margins (Figure 1). The streaks can be distinguished from similar symptoms occurring on sugarcane leaves from other causes (e.g. leaf scald and insect damage) by their broken, wavy, irregular margins. Streaks can appear on a leaf at any stage of development and there may be one to multiple streaks on the same leaf. Leaf streaks vary in width and length, ranging from very narrow to 12 mm in width and they may be short or can be the entire length of the leaf. Streaks appear similar on both the upper and lower leaf surfaces and are not often found on the leaf sheath and midrib. Newly developed leaf-streaks are irregular, often fragmented, short and faint in color. As the symptoms progress, streaks generally elongate and become more pronounced. However, they may also not enlarge much from their original size. Older streaks take on a yellow color, and in some instances, but not always, the central parts of the streak become necrotic (within as few as seven days) either in sections or along the full length of the streak (Figure 2). These necrotic tissues are ash-grey-tan and may have a red-brown margin at their edge. Leaf tips and margins may also show necrosis, especially where streaks meet the edge of the leaf. Streaks may be present on one or all leaves on a stalk and present on one or all stalks in a stool. Sometimes infected plants appear symptomless because symptomatic leaves are senescent or shedded.

Internal stalk symptoms include discolored vascular bundles at the nodes. The coloration is usually red and occurs right through the node and sometimes a short distance into the internode. The intensity of these symptoms is usually greater in stalks where the leaves are exhibiting abundant symptoms, though this may not always be the case; some symptomless stalks may also show internal symptoms. The apical meristem of affected cane may also show internal discoloration following slicing (Earsman and Young, 2024).



Figure 1. Classic chlorotic streaking of leaves (C. Ngo).



Figure 2. Necrotic areas within streaks (C. Ngo).

Symptom expression is environmentally dependent, mainly on high soil moisture and temperature, leading to transient symptoms. Soil temperature affects the expression of symptoms with 30°C being the optimum (Sturgess, 1962). Dry conditions do not favor symptom development.

Diagnosis

Identification of the disease is performed by the recognition of leaf symptoms and when classic symptoms are expressed; this is a reliable diagnosis. Difficulties arise when there is transient symptom expression or when the intensity of symptoms is such that it is difficult to separate CS from other scalding type effects. Symptoms are often more easily observed in varieties with a wide leaf blade characteristic. The protozoa have been visualised using light microscopy (x200 to x400) in vascular sap extracts, however, the pathogen is not in high enough abundance to reliably use this method for diagnosis. An inverted microscope is recommended for examination of *P. venanatans* grown in cell culture flasks in potato dextrose broth medium (Braithwaite *et al*., 2018). Cells are round to ovoid (6–15 µm long), generally with two flagella directed anteriorly and posteriorly (dividing cells have four) (Figure 3). Flagella extend equally in both directions and are typically 1.5 to 3x cell length. Cells may be stationary or gliding at variable speeds.

Molecular diagnostic methods (PCR and qPCR) are available for this pathogen (Braithwaite *et al*., 2018). These tests can be performed on water samples, vascular extracts, and most plant tissues. Although the disease is systemic, there is no apparent trend in abundance along the stalk. There is evidence that asymptomatic stalks can test positive, while symptomatic stalks can test negative. This is possibly due to the non-uniform distribution of the pathogen in the plant and the related transient nature of symptoms. The use of vascular extracts and leaf sheath biopsy (LSB) (Young *et al*., 2014) samples removes the requirement for DNA extractions, but vascular extracts are the most reliable sample for detection. A required modification for the LSB incubation step is 28°C for 48–72 h. The vascular extract can also be used to test for ratoon stunting and leaf scald.

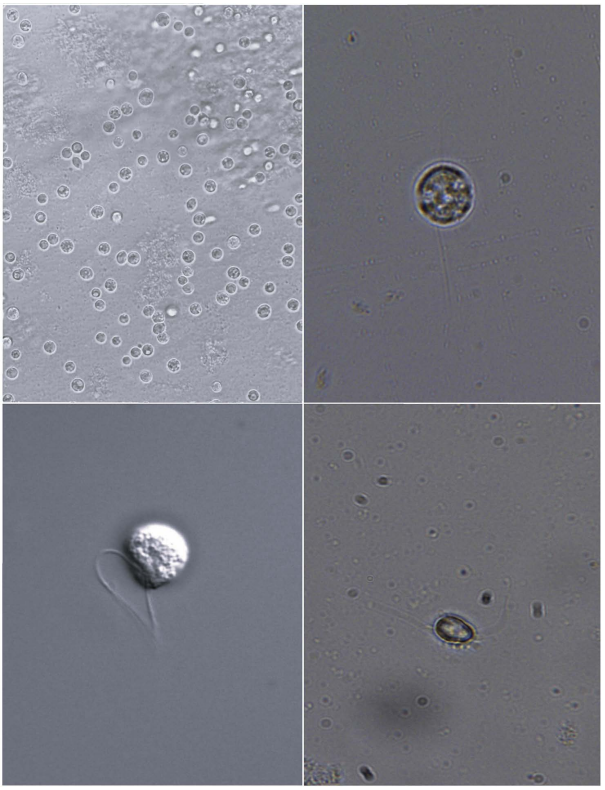

Figure 3. *Phytocercomonas venanatans* at X400 (images are not to scale). Clockwise from top left: inverted microscopy of cells in culture; typical round shaped cell; ovoid shaped cell; Nomarski microscopy showing subapically inserted flagella (C. Ngo).

Strains of the pathogen

There is currently no evidence for the existence of strains of the pathogen.

Transmission

Much work has been undertaken on the transmission of CS (Antoine, 1957; Bird *et al*., 1958; Egan, 1961 and 1963; Sturgess 1961, 1963 and 1965). More recently, it was demonstrated that *P. venanatans* can be transmitted to plants via shoot roots, stalks and leaf whorls (Braithwaite *et al*., 2018). The disease is not transmitted mechanically by harvesting or planting equipment. The disease is readily spread by infected planting material, in flood waters, normal surface water and in drainage water flowing from diseased to healthy fields. In addition, diseased fields will remain infective in the absence of sugarcane (or another host) for up to nine months (Sturgess, 1964). Disease transmission is favored by ratooning under wet conditions. Glasshouse trials showed that inoculation within three days of ratooning favored disease development. The minimum time from inoculation to symptom expression is 12 days.

Host range

Sugarcane hybrids (*Saccharum* spp.) are the known natural hosts of *P. venanatans*. This protozoan also appears to infect other members of the *Saccharum* complex, including *S. edule*, *S. officinarum, S. robustum*, *S. sinense*, and *S. spontaneum*. The disease occurs

naturally in several grasses in at least four countries, and these can act as sources of inoculum for commercial crops. These grasses include *Arundo donax, Erianthus arundinaceus*, *E. maximus, Panicum maximum, Paspalum paniculatum* and *Pennisetum purpureum*. Other species into which the pathogen has been transmitted include *Brachiaria mutica*, *E. procerus*, *Imperata cylindrica, Miscanthus floridulus*, *M. violaceus*, *Sorghum almum*, *S. bicolor* and *S. verticilliflorum* (Egan, 1965). Confirmation with molecular diagnostics is needed for this list.

Epidemiology

The disease is favored by soil temperatures between 25 and 30°C (Sturgess, 1962) and particularly by poorly drained, flood-prone conditions. In such areas, the planting of healthy seed cane does not adequately control the disease because re-infection can result from drainage water flowing from diseased fields or from residual infectivity in the soil. Dry conditions and cooler temperatures will lead to much lower levels of symptomatic disease, even in favored areas, and the disease may appear to lose its importance in certain areas. However, the onset of conducive environmental conditions, such as high rainfall or floods, soon leads to high levels of disease in susceptible varieties and the need for control. The disease is also spread in recycled irrigation water.

Economic importance

Chlorotic streak can be of major economic importance in areas favoring the disease. Significant yield losses were reported in various countries, including Australia, Guyana, Hawaii, Mauritius and Puerto Rico (Egan, 1989). Components of yield loss are generally accepted as being attributed to germination losses, poor ratooning, decreases in stalk length and number (harvestable biomass), however CCS (commercial cane sugar) is usually not affected (Figure 4).



Figure 4. Reduced growth of sugarcane plants affected by chlorotic streak (three rows on the left) as compared to disease-free plants (three rows on the right) (P. Rott).

In Australia, CS is possibly the most widely recorded sugarcane disease and has been estimated to cost the Australian Sugar Industry $8–$10 million annually (Braithwaite and Croft, 2013; Magarey 2005). Yield losses (tons cane per hectare) of up to 40% in one trial and 60% in another were recorded in susceptible varieties (Magarey and Neilsen, 2002). Correlation of yields in healthy and diseased plots in the Herbert River

district of Australia suggested a 0.24% yield loss for every 1% stalks showing symptoms (Nielsen *et al*., 1986). These results were obtained during a period of below average rainfall but still indicated a maximum yield loss of 24% in a completely diseased crop. Germination reductions of 10, 16, and 22% were caused by CS in varieties Q66, Q67 and Pindar, respectively (Egan, 1962). Yield losses in another trial with these same varieties over plant and ratoon crops were 21, 25 and 19% respectively (not including germination effects). In Taiwan, 4–14% yield reductions were found in six varieties over a plant and first ratoon crop, with no effect on sugar content (Wang and Jiang, 1982). Significant reductions in stalk numbers, stalk length and diameter, but not in Brix were also reported from field trials in Mauritius (Wiehe, 1955).

Control

Control of the disease relies on an integrated approach. Three key parts to this approach are: improved drainage, disease-free planting material, and the use of varieties with sufficient resistance. The elimination of poorly drained fields through levelling, and provision of adequate field drains, will assist in lowering disease levels in many cases. Disease-free planting material is readily obtained by thermotherapy consisting of hot water treatments at 50°C for 30 min or 52°C for 20 min. The longer treatment duration may be preferred for thicker stalks. Even though the chance of reinfection may be present, disease-free material leads to better germination. It is important that propagation plots are grown in well drained, disease-free sites, where re-infection is unlikely. In disease-prone areas it is difficult to avoid some level of infection pressure, so it is also important that varieties with sufficient resistance are grown. In most countries, there are no routine resistance screening trials for CS and the selection of suitable canes relies on field observations of varietal reaction. Trials have been performed in Australia and relied on natural flooding events or hydroponic systems with a diversity of resistance reactions being noted in commercial canes. There are currently new attempts to deliver a rapid screening method in Australia.

Other aspects to be considered in control are the presence of diseased volunteers in newly planted crops, infected grasses along headlands or in surrounding fields, and ratooning under wet conditions. The latency of symptoms under certain conditions makes the visual selection of disease-free planting material uncertain especially under particular environmental conditions.

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V

Diseases caused by viruses

Fiji leaf gall
(Previously known as Fiji disease)

Barry J. Croft, Robert C. Magarey

This chapter has been updated and rewritten based on the original chapter entitled "Fiji disease" and authored by Grant Smith in 2000.

Cause

Fiji leaf gall is caused by Fiji disease virus or FDV (species name = *Fijivirus fijiense*; family *Spinareoviridae*).

Geographical distribution

Australia, Fiji, Indonesia (Halmahera Island), Malaysia, New Caledonia, Papua New Guinea, Philippines, Samoa, Solomon Islands, Thailand, Vanuatu.

In Australia, Fiji leaf gall has never occurred in areas north of Proserpine. Major epidemics occurred in the Mackay and the Bundaberg regions (Magarey *et al.*, 2019) but these areas are now considered free of the disease with the last recorded infections 30 years ago. In areas south of Bundaberg, the disease persisted for longer but there have been no recent reports of the disease. The disease is maintained for research purposes in a research station near Brisbane, Australia, in isolation from commercial crops. Madagascar is considered free of FDV with the disease last reported in 1971.

Symptoms

The diagnostic symptom of Fiji leaf gall is the raised galls on the underside of the leaf blade and midrib (Figure 1). Galls vary in size from 1 to >200 mm in length by <1 mm to 3–4 mm in width and 1 to 2 mm in height. The galls vary in color from white to green.

Fiji leaf gall causes severe stunting, profuse tillering and in some cases death of plants. The severe stunting is often the first symptom that attracts further investigation. Leaves are often shorter than normal and develop ragged edges with transverse cuts and twisted, distorted tips, giving the appearance that an animal has bitten the top off the leaf (Figure 2). Stunted leaves are often darker green than normal leaves. Occasionally the growing point dies, leading to side-shooting. Stunting is particularly severe when diseased stalks are planted and when infected plants are ratooned (Figure 3). These symptoms are not definitive and examination of the leaves for the diagnostic galls is essential. In some tolerant or resistant varieties, the only symptom present is galls, and they are often small and difficult to see in these varieties.

In fields of susceptible varieties with a high incidence of Fiji leaf gall, large patches of stunted and dead plants give the fields a very uneven appearance.







Figure 1. Galls on the dorsal surface of the leaf blade and midrib (A and C: P. Rott; B: B.J. Croft).





Figure 2. "Bitten off" aspect of the sugarcane top (A: B.J. Croft; B: P. Rott).



Figure 3.
Stunting of sugarcane caused by Fiji leaf gall disease (J.H. Daugrois).

The time between infection and the appearance of the first symptom varies from 15 days to six (or more) months depending on the variety, age of the plant, level of infection and environmental conditions. Ratooning of a diseased but symptomless plant will often induce symptom development in the following ratoon growth.

Diagnosis

In the field, Fiji leaf gall is diagnosed by the presence of the galls. These galls are the result of proliferation of the phloem and xylem cells in the vascular bundles. A transverse section through the gall, and observation of the associated cell structures can be used to distinguish Fiji leaf gall from other conditions. Leaf galls and stunting in maize and sugarcane can be induced by toxins produced by feeding of maize leafhoppers in the genus *Cicadulina* (Tokuda and Matsumura, 2005). These galls often occur in chains on either side of the vascular bundle with the vascular bundle still clearly seen running down the centre of the gall. They can be distinguished from those of Fiji leaf gall as they are formed from proliferation of the parenchyma and bundle sheath cells, not the phloem and xylem cells.

The most reliable diagnostic assay for confirmation of the presence of FDV is the RT-PCR assay developed by Smith and Van de Velde (1994). This RT-PCR assay was developed using primers FDV7F and FDV7R and is routinely used in Australia for screening clones moving from regions where Fiji leaf gall has been present to virus-free regions and in post-entry quarantine (Thompson *et al.*, 2022).

Virions of FDV are double-shelled, icosahedral in shape and approximately 70 nm in diameter, with spherical, short surface spikes (A spikes) on each of the 12 vertices of the icosahedron (Hatta and Francki, 1977; Figure 4). The outer shell is very fragile, leaving the inner shell with 12 B spikes. Examination of leaf galls by electron microscopy for the distinctive virions of FDV can be used for diagnosis but equipment and expertise are limited in most laboratories. The viral genome contains 10 segmented double-stranded RNAs. The full genome of FDV has been sequenced (Harding *et al.*, 2006). FDV is most closely related to maize rough dwarf fijivirus (species name = *Fijivirus zeae*) and rice black streaked dwarf fijivirus (species name = *Fijivirus alporyzae*) (Harding *et al.*, 2006; McMahon *et al.*, 1999).

Antisera and serological assays for FDV have been produced (Francki *et al.*, 1986; Soo *et al.*, 1998; Wagih and Adkins, 1996), but these assays are less sensitive and/or specific than the RT-PCR assay.

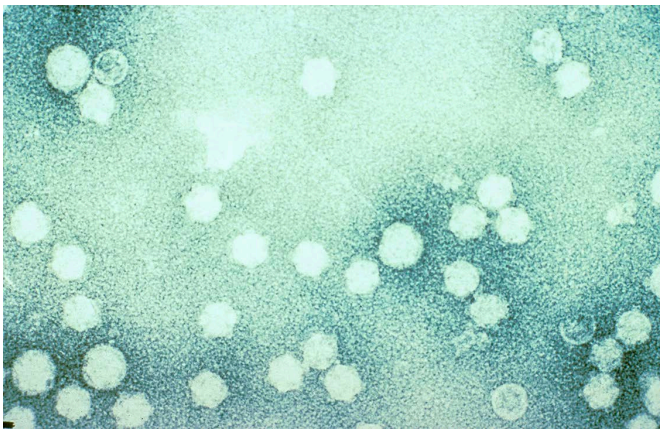

Figure 4. Purified particles of Fiji disease virus (diameter 50–70 nm) observed by transmission electron microscopy (R. Harding).

Strains of the pathogen

Up to 15% variation was found in partial nucleotide sequences of segments 3 and 9 of the FDV genome in 25 isolates from Australia, Papua New Guinea and Malaysia (Jiang *et al.*, 2008). The Malaysian isolates formed one group that showed the highest genetic divergence from the isolates from Australia and Papua New Guinea. Isolates from within Australia showed limited sequence divergence and were similar to 3 of the 10 isolates from Papua New Guinea. Within Papua New Guinea, there was substantial divergence between isolates from different locations and within isolates from the same location.

Transmission

FDV is transmitted by species of the delphacid planthopper genus, *Perkinsiella,* in a persistent-propagative manner (Figure 5). Species known to transmit FDV are *P. saccharicida, P. vastatrix* and *P. vitiensis* (Egan *et al.*, 1989; Zhang *et al.*, 2021).

P. saccharicida lays its eggs in the midrib of the leaf and when the eggs hatch, there are five instars of 4 to 5 days duration each before development into adults. Adults can either be macropterous (long-winged) or brachypterous (short-winged). Brachypterous males are rare and the proportion of macropterous to brachypterous females varies depending on environmental and population factors. FDV is only acquired by first and second instars of *P. saccharicida* (Daniels *et al.*, 1969; Croft, unpublished data). The insects transmit the virus when they feed on disease-free plants. FDV incidence in insects bred on an infected-susceptible variety varies greatly from 20–80% (Dhileepan *et al.*, 2006; Hughes *et al.*, 2008; Ridley *et al.*, 2006 and 2008) but only 30% or less of insects in which the virus is present can successfully infect plants of a susceptible variety. The proportion of insects that acquire the virus when they are bred on infected-resistant and/or tolerant varieties is much lower than if they are bred on infected-susceptible varieties. The virus titre within infected insects is related to their transmission competence (Hughes *et al.*, 2008). No difference was found between the virus incidence in macropterous males and females, brachypterous females and older instars (Ridley *et al.*, 2008). There was very little variation in the ITS2 sequence of *P. saccharicida* collections from Australia, USA, South Africa, and Reunion (Ridley *et al.*, 2006). *P. saccharicida* collected from North Queensland, where FDV has never been recorded, were able to acquire and transmit the disease (Ridley *et al.*, 2006).



Figure 5. Adult planthoppers of *Perkinsiella saccharicida*, vector of Fiji leaf gall in Australia. Note the egg punctures and associated reddening in the leaf midrib (B.J. Croft).

Some evidence exists for vector transovarial transmission (transmission through the egg) of FDV (Chang, 1977). The successful eradication of FDV in Madagascar and large areas of Australia would suggest that transovarial transmission does not occur or does not occur at a level sufficient to maintain the virus in a population of the insect in the absence of diseased plants.

Infected planting material (stalks, cuttings or setts) is an important means of spreading the disease between fields, farms, regions or countries. Almost all plants developing from infected planting material will be infected.

FDV is not mechanically transmissible.

Host range

Fiji leaf gall occurs naturally in *Saccharum officinarum, S. edule* (common names: pit pit, duruka), and commercial sugarcane hybrids (Egan *et al.*, 1989). *S. maximum* (syn. *Erianthus maximus,* a natural *Saccharum* × *Miscanthus* interspecific hybrid) is a host on Pacific islands. Other *Saccharum sp.* and some grasses can be infected by forcing infected planthoppers to feed on them in cages but do not appear to play any significant role in the field. In Papua New Guinea and Fiji the aborted inflorescences of *S. edule* are prized as a vegetable and this species can act as a source of spread to *S. officinarum* in native gardens and commercial crops (Magarey *et al.*, 2002; Tamanikaiyaroi and Johnson, 1996).

Epidemiology

Spread of Fiji leaf gall in planting material establishes a source for further spread by planthoppers. *P. saccharicida* feeds and breeds almost exclusively on sugarcane and populations in Australia peak in late summer to early autumn. During the

epidemic in Bundaberg in the 1970s, extremely high populations of planthoppers of 200–300 insects per stalk were recorded (Bull, 1977). The highest populations were in the variety NCo310, which was highly favorable to the planthopper and susceptible to Fiji leaf gall. NCo310 was planted over 95% of the area in this region in the 1970s. The combination of high planthopper populations and a susceptible variety resulted in rapid spread of the disease. When populations are high, the planthoppers form swarms that move long distances.

P. saccharicida has several natural predators that can reduce populations of the planthopper and slow the spread of the disease. These include the egg predator *Tytthus mundulus,* ladybirds, spiders and wasps (Egan *et al.*, 1989).

Economic importance

Fiji leaf gall has caused major epidemics in the past in Australia, Fiji, and Malaysia. These epidemics were so severe that they threatened the viability of these industries (Egan *et al.*, 1989). Susceptible varieties can suffer losses of 50% or more. The plough-out of infected crops after first or second ratoon, production of disease-free planting material and field workers to conduct inspections and roguing greatly increases production costs. The disease has a significant impact on breeding programs through the loss of potentially high yielding, but susceptible varieties, and restrictions on the use of susceptible parent varieties. Currently, the disease is not causing any significant losses in commercial crops.

Control

Fiji leaf gall has been successfully controlled through planting of resistant varieties. In Australia, the highly resistant variety Q110 was widely planted in southern Queensland at the peak of the epidemic, when disease pressure was extreme, and was the primary factor contributing to successful disease management. Plants are more susceptible when they are young. Moderately susceptible varieties can be planted in late autumn, so they are older in summer when planthopper populations peak. Transgenic varieties with partial resistance to Fiji leaf gall have been produced but these have not been grown commercially (McQualter *et al.*, 2004).

Clones in the breeding program can be screened for resistance by planting test clones between rows of infected plants and allowing natural planthopper populations to spread the disease (Croft *et al.*, 2004). When planthopper populations are low, this method becomes unreliable, and glasshouse or insectary methods have been used (Daniels *et al.*, 1969; Croft *et al.*, 2004).

Quarantine is essential to prevent the disease spread to disease-free regions or countries. Varieties in quarantine should be grown in an isolated area, inspected for symptoms of Fiji leaf gall, and leaf samples assayed for the virus using an RT-PCR assay before they are released from quarantine. Tissue culture from apical and axillary meristems eliminated FDV from 70–80% of the plants that were successfully regenerated from an infected variety (Cheong *et al.*, 2012).

Removal of infected plants and/or ploughing out infested fields can reduce losses and slow the spread of the disease (Egan *et al.*, 1989). Fields should be replanted with disease-free planting material from inspected sugarcane nurseries. Insecticides have not been used commercially to control the planthoppers.

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Leaf fleck

Rasappa Viswanathan, Shweta Singh

This chapter has been updated and rewritten based on the original chapter entitled "Sugarcane bacilliform virus" and authored by Ben E. Lockhart and Jean Claude Autrey in 2000.

Cause

Leaf fleck of sugarcane is caused by several pararetroviruses known as sugarcane bacilliform viruses or SCBVs: sugarcane bacilliform Guadeloupe A virus or SCBGAV (species name = *Badnavirus alphasacchari*), sugarcane bacilliform Guadeloupe D virus or SCBGDV (species name = *Badnavirus betasacchari*), sugarcane bacilliform MO virus or SCBMOV (species name = *Badnavirus deltasacchari*), and sugarcane bacilliform IM virus or SCBIMV (species name = *Badnavirus gammasacchari*).

From a taxonomic point of view, all these viruses belong to the genus *Badnavirus* of the *Caulimoviridae* family. SCBVs are a genetically diverse group of viruses within the badnaviruses that infect sugarcane and cause leaf fleck. Virus particles are non-enveloped, bacilliform, 30 × 130 to 150 nm in size, and contain a genome consisting of a circular, relaxed, 7.5 to 8.0 kb dsDNA molecule (Hull *et al.*, 2005) (Figure 1). The genome is not covalently closed because each strand is interrupted by site-specific discontinuities. It is replicated via a virus-encoded reverse transcriptase (RT). SCBV maintains the typical badnavirus genome organization, consisting of three open reading frames (ORFs) on the positive strand.

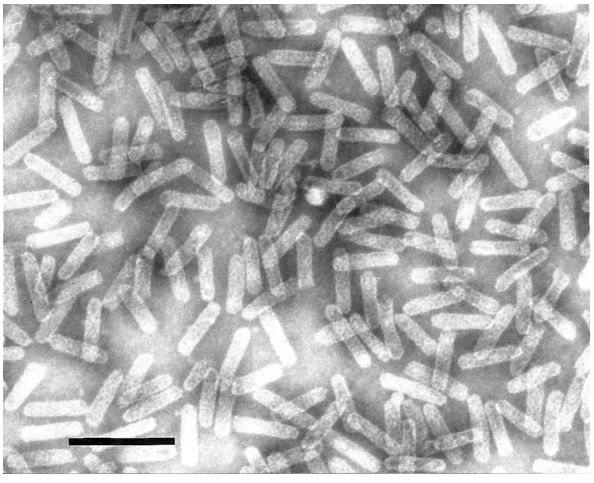

Figure 1. Virions of sugarcane bacilliform virus observed by negative-stain transmission electron microscopy (scale bar = 200 nm) (B.E. Lockhart).

Geographical distribution

Argentina, Australia, Barbados, Brazil, Burundi, Cape Verde, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Ethiopia, Guadeloupe, Hawaii, India, Indonesia, Madagascar, Madeira, Malawi, Mauritius, Mexico, Morocco, Papua New Guinea, Philippines, Puerto Rico, Réunion, South Africa, Taiwan, Thailand, USA.

Leaf fleck was first identified in Cuba in 1985 in infected sugarcane cultivar B34-104 and subsequently in numerous sugarcane-growing regions worldwide (Autrey *et al*., 1995; Lockhart *et al*., 1996). Reports of the disease emerged around the same time in Florida and Texas (Comstock and Lockhart, 1990), India (Viswanathan *et al*., 1996), Mauritius (Autrey *et al*., 1990), Morocco and Hawaii (Lockhart and Autrey, 1988), and Taiwan (Liang *et al*., 1995).

Symptoms

The characteristic symptoms are chlorotic leaf flecks and stripes which lead to chlorotic mottling on the entire lamina of mature leaves (Figure 2).

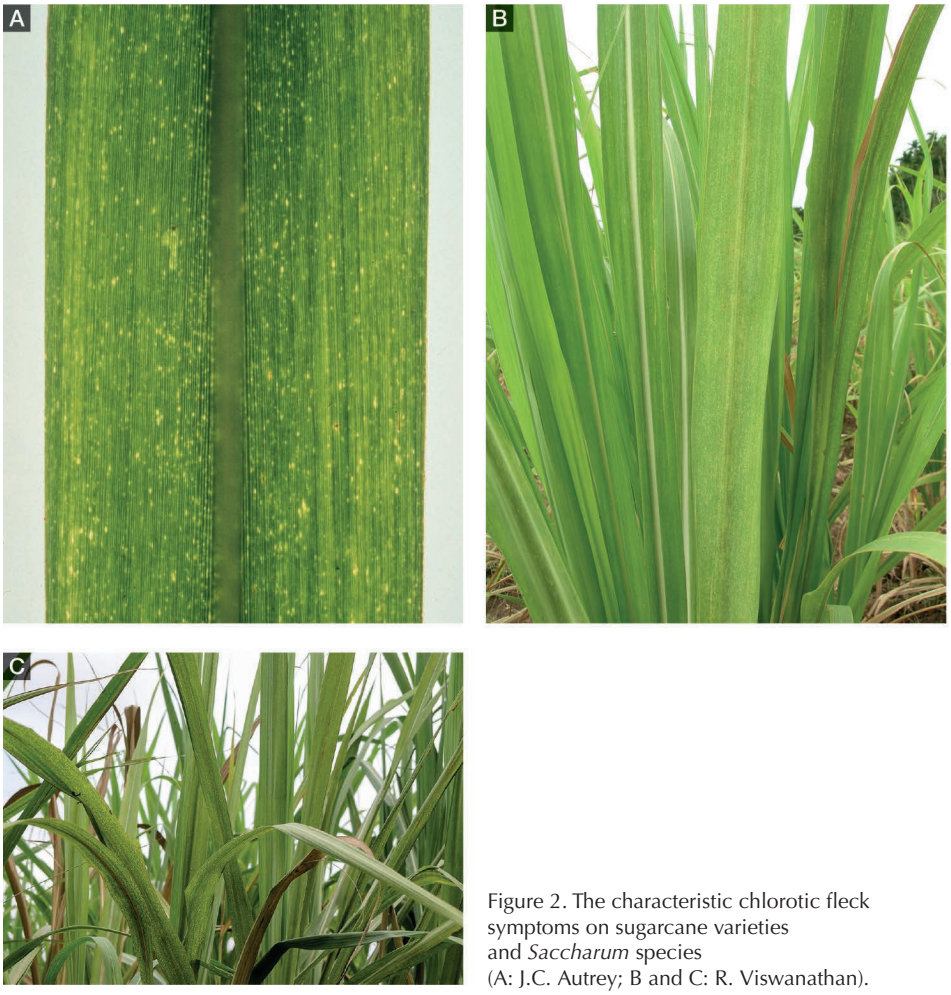

Figure 2. The characteristic chlorotic fleck symptoms on sugarcane varieties and *Saccharum* species (A: J.C. Autrey; B and C: R. Viswanathan).

In several *S. officinarum* genotypes such as Iscambine, Iscambine Rayée, Boetata Bilatoe, Castilla, and Guam A, the symptoms appear as chlorotic leaf stripes that merge together, thus resulting in interveinal chlorosis (Figure 3). These symptoms are more common on older distal leaves while the younger leaves, especially those from the crown, remain asymptomatic in most cases. The distal section of the leaf lamina may also exhibit yellowish streaks that turn into yellow blotches, progressively occupying the entire leaf surface (Figure 4). Gradually, yellow flecks/streaks turn reddish yellow, more prominently on the distal region and finally the entire leaf lamina takes a reddish hue in varieties CoV 92102 and CoV 09356 (Figure 5). Although the disease symptoms are witnessed from germination onwards, leaf fleck severity increases with age, and its appearance is predominant in tillering and maturity phases. Severe disease symptoms are expressed as pale canopy throughout commercial field plantations (Figure 6). Intense flecks or extensive yellowish and subsequent reddish discoloration of the canopy lead to premature drying of the leaves (Figure 7). Overall, entire fields can be symptomatic and associated with stunted sugarcane growth and premature drying of mature leaves. In India, disease incidences of up to 100% were reported in certain fields where it was difficult to find disease-free plants or plants expressing only mild symptoms. Ratoon crops of susceptible varieties often exhibit increased disease severity as compared to plant cane crops. Leaf fleck symptoms as well as those of mosaic (caused by sugarcane mosaic virus or SCMV) may also be exacerbated in varieties simultaneously infected by SCBV and SCMV (Vamsi Krishna *et al.,* 2023). In the growing regions except India, sugarcane infected solely by SCBV usually does not produce strong symptoms and infected commercial hybrids are usually symptomless (Lockhart *et al*., 1996).



Figure 3. Chlorotic stripes and interveinal chlorosis in *Saccharum* spp. clones (R. Viswanathan).



Figure 4. Chlorotic flecks turning into yellowish or yellow blotches (R. Viswanathan).



Figure 5. Leaf fleck affected leaf lamina becoming reddish (R. Viswanathan).



Figure 6. Severe expression of leaf fleck in commercial plantations: pale reddish canopy with poor growth (R. Viswanathan).



Figure 7. Severely affected crop exhibiting discoloration of the canopy and premature drying of the leaves (R. Viswanathan).

Diagnosis

During the initial days of virus identification, electron microscopy and enzyme-linked immunosorbent assays (ELISA) were employed to confirm the presence of SCBV (Viswanathan and Alexander, 1995). Protocols for direct antigen coated (DAC)-ELISA, double antibody sandwich (DAS)-ELISA, and triple antibody sandwich (TAS)-ELISA were standardized and used for sensitive detection of SCBV. The virus has also been detected with high sensitivity in partially purified extracts using immunosorbent electron microscopy (ISEM) or immunocapture (IC)-PCR (Lockhart *et al.*, 1996). Bacilliform virus particles can be observed in lyophilized leaf extracts using ISEM. All these methods have paved the way for more sensitive molecular diagnostic assays such as PCR. SCBV detection based on PCR and qPCR has been carried out by targeting the three ORFs

of the virus genome as well as BADNA-specific primers (Balan *et al*., 2022). A robust diagnostic approach based on recombinase polymerase amplification (RPA) has recently been developed for sensitive and quick detection of SCBV in crude sugarcane sap, thus overcoming the limitations of the PCR assays (Parameswari *et al*., 2023).

Strains of the pathogen

Four *Badnavirus* species infecting sugarcane have so far been officially recognized by the ICTV[16], while 25 phylogenetic groups (genotypes) of SCBV have been reported in various plant species, ranging from SCBV-A to SCBV-Y. Consequently, significant genetic diversity occurs within this virus group (Janiga *et al*., 2023). At least five different genotypes were found in sugarcane germplasm collections and varieties cultivated in India based on partial and complete genome sequences (Karuppaiah *et al*., 2013). Among those, three could be new sugarcane virus species: sugarcane bacilliform BB virus (SCBBBV), sugarcane bacilliform BO virus (SCBBOV), and sugarcane bacilliform black Reunion virus (SCBBRV). These viruses from India share 69–85% identity at the complete genome level and have potential recombination sites that could result in development of additional genetic variants. Strain variation based on pathogenicity has not been investigated so far.

Transmission

SCBVs are naturally transmitted by mealybugs (Figure 8), notably the pink mealybug (*Saccharicoccus sacchari*) and the grey sugarcane mealybug (*Dysmicoccus boninsis*), which are efficient vectors that most likely transmit these viruses in a semi-persistent manner (Lockhart *et al*., 1996). *Planococcus citri*, the citrus mealybug, can also transmit the SCBVs but this insect does not typically colonize sugarcane. SCBVs were transmitted mechanically to rice, banana and sorghum using partially purified virus extracts (Bouhida *et al*., 1993; Lockhart *et al*., 1996). Interestingly, these viruses are not only vegetatively transmitted by stalk cuttings but also by true seeds/fluffs generated from crosses between parents of interest (Balan *et al*., 2022; Lockhart *et al*., 1996; Viswanathan *et al*., 1996). These latter observations suggested that SCBVs are not only transmitted horizontally but also vertically, e.g. from parents to progenies. SCBVs are not easily transmitted mechanically from diseased to healthy plants using harvesting tools.



Figure 8. The pink sugarcane mealybug, *Saccharicoccus sacchari*, a vector of sugarcane bacilliform virus (P. Rott).

16. ICTV (International Committee on Taxonomy of Viruses), https://ictv.global/report/chapter/caulimoviridae/taxonomy/caulimoviridae.

Host range

In addition to commercial sugarcane hybrids (*Saccharum* spp.), SCBVs infect all species of the *Saccharum* germplasm such as *S. barberi*, *S. officinarum*, *S. robustum*, *S. sinense*, and *S. spontaneum* (Janiga *et al*., 2023; Viswanathan and Premachandran, 1998; Viswanathan *et al*., 1996 and 2019). Nevertheless, the natural host range of SCBVs extends beyond sugarcane as these viruses were detected in other plant genera and species. *Brachiaria* sp., *Panicum maximum*, *Rottboellia exaltata,* and *Sorghum halepense* were found naturally infected by SCBVs (Lockhart *et al*., 1996). These viruses were also artificially transmitted to *Musa* sp., *Oryza sativa*, and *Sorghum vulgare* (Bouhida *et al*., 1993; Lockhart *et al*., 1996; Viswanathan *et al*., 1996). SCBVs are closely related to banana streak virus (BSV) (Lockhart and Autrey, 1988), and the symptoms induced by SCBV infection in banana are indistinguishable from those caused by BSV. However, although SCBV can colonize banana after transmission from infected sugarcane by the mealybug *S. sacchari*, there is currently no evidence that this transmission occurs naturally.

Epidemiology

The high incidence of leaf fleck in the Indian sub-continent can be attributed to several factors such as extensive cultivation, ratooning practices, weather conditions favorable to the vector populations, and specific crop management practices. Consequently, disease occurrence and severity vary according to crop stage, sugarcane variety, prevailing agroclimatic conditions, and management practices. Notably, disease progress tends to increase during summer months in India (Balan *et al*., 2020). Lack of virus management and continuous cropping also contribute to the build-up of leaf fleck in the fields.

Economic importance

Few studies have been conducted to assess the economic impact of leaf fleck on sugarcane production. In susceptible varieties, severe infection of SCBV can result in 25–35% reduction in biomass production (Comstock and Lockhart, 1996). In India, leaf fleck incidence varied from 12–51% and 28–56% in the states of Kerala and Tamil Nadu, respectively. Frequency of flecking and yellow mosaic mottling symptoms ranged from 5–100% in Maharashtra and Uttar Pradesh. Maximum incidence of the disease was noted on varieties Co 7219 and CoS 510 (Singh *et al*., 2003). Besides their putative direct effect on sugarcane production, SCBVs are economically important pathogens as they have serious implications in sugarcane germplasm exchange across the globe.

Control

The most economical and sustainable approach of leaf fleck management involves the use of resistant or tolerant sugarcane varieties. Identification and deployment of resistant sources as donors in breeding programs have given promising results. SCBV was detected in 60 of 126 clones of a *Saccharum* germplasm collection in India, therefore virus-free clones can be used in sugarcane breeding (Janiga *et al*., 2023). Eight additional sources of germplasm resistant not only to SCBV but also to sugarcane streak mosaic virus (SCSMV) and SCMV were identified in both plant and ratoon crops in Andhra Pradesh, namely 2017A 553, 2017A 416, 2017A 517, 2016A 379, Co 7717, CoS 767, Baragua and Co 13034 (Vamsi Krishna *et al*., 2023).

While breeding for virus resistance is a long-term strategy, other control methods have been explored to mitigate the impact of leaf fleck. Attempts to control SCBV using hot water treatments and antiviral chemicals were unsuccessful in eliminating the virus in infected planting material. The production of virus-free planting material is essential for reducing the spread of viruses, particularly when transmission occurs through both vector(s) and true seed. Although mechanical transmission of SCBV appears to be rare, proper disinfection of harvesting tools is recommended to limit its spread in nurseries. Meristem tip culture is usually highly efficient to produce virus-free sugarcane plants (>90%) but this technique appears to be less efficient for SCBV with only 16–30% virus elimination (Abide *et al.*, 2022). Meristem tip culture combined with thermotherapy, chemotherapy, and cryotherapy may improve SCBV elimination in infected sugarcane. Furthermore, performance of cleaned material needs further evaluation in older plants to ascertain its long term efficacy.

The exchange of genetic resources across international borders has inadvertently introduced viruses into new regions, and SCBV is frequently detected during quarantine inspections. Therefore, application of strict quarantine procedures and accurate disease testing is also imperative for the causal agent(s) of leaf fleck.

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Mild chlorotic streak

Philippe Rott, Jean Heinrich Daugrois

Cause

Maize striate mosaic virus or MSMV (species name = *Mastrevirus striatis*, family *Geminiviridae*).

Geographical distribution

Brazil. The virus has also been found in Argentina on maize (Posse *et al.*, 2023).

Symptoms

Sugarcane leaves infected by MSMV in Brazil display mild chlorotic streaks whereas maize leaves infected by this virus exhibit striate mosaic symptoms (Batista *et al.*, 2021) (Figure 1).



Figure 1. Leaf of forage Guinea grass cultivar BRS Zuri infected by maize striate mosaic virus (C. Lacorte).

Diagnosis

A PCR assay based on abutting primers was developed to amplify the entire genome of MSMV (Fontenele *et al.*, 2018). Use of these primers[17] results in the amplification of a DNA fragment of about 2,700 kb. Specific primers for detection of MSMV were also designed[18], and these primers yield an amplicon of 664 bp (Vilanova *et al.*, 2022).

Strains of the pathogen

Variation in virulence among MSMV isolates is unknown. The full genome identity between virus isolates from sugarcane and maize is ≥99% (Batista *et al.*, 2021; Posse *et al.*, 2023).

Transmission

Although not demonstrated yet, MSMV is most likely transmitted by vegetative propagation of sugarcane (stalk cuttings). The virus has been transmitted from MSMV-infected to healthy

17. J455-F: 5′-ACCCTTCTTAACTTCCACCACGGCAGAA-3′; J455-R: 5′-GGTAATTGTCTGATGGTTACCTCCTACA-3′.
18. MSMVF1: 5′-CCGAACGGAGTGGAAGAACA-3′; MSMVR1: 5′-TCGGGCCACACACAACTACTTC-3′.

maize and sugarcane plants by the corn leafhopper (*Dalbulus maidis*) but not by the corn aphid (*Rhopalosiphum maidis*) or by mechanical inoculation (Vilanova and Lopes, 2026; Vilanova *et al*., 2022). Because *D. maidis* is specialized on maize, other leafhoppers may spread MSMV from maize to sugarcane or from sugarcane to sugarcane. However, transmission of the virus from maize to sugarcane by the corn leafhopper cannot be excluded as sugarcane is a shelter (feeding) host of *D. maidis* in Brazil (Oliveira *et al*., 2020).

Host range

Sugarcane and maize are cultivated hosts of MSMV (Batista *et al*., 2021; Fontenele *et al*., 2018), but the virus has also been found in the forage Guinea grass (*Megathyrsus maximus*) in Brazil (Vidal *et al*., 2026).

Epidemiology

No epidemiological data are currently available. Nevertheless, presence of infected maize or sugarcane plants, as well as high vector populations, are most likely conditions that will favor incidence of the virus in sugarcane and the development of the disease.

Economic importance

Prevalence of MSMV was approximately 50% in sugarcane and maize crops in surveys conducted in Brazil in 2016 and 2018 (Batista *et al*., 2021). The potential impact of the virus on sugarcane production remains to be determined. Height reduction was observed in infected maize test plants exhibiting mottling symptoms including mild chlorotic streaks (Vilanova *et al*., 2022).

Control

Mild chlorotic streak can be controlled using resistant sugarcane varieties and virus-free seed cane.

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Mild mosaic

Ben E. Lockhart, Jean Claude Autrey, revised by Dimitre Mollov

Cause

Sugarcane mild mosaic virus or SCMMV (species name = *Ampelovirus sacchari*, family *Closteroviridae*) (Figure 1).

Geographical distribution

Argentina, Australia, Barbados, Brazil, Chad, China, Colombia, Costa Rica, Côte d'Ivoire, Dominican Republic, Egypt, Guadeloupe, Hawaii, Malawi, Mauritius, Philippines, Réunion, South Africa, Sudan, Thailand, USA (Florida, Louisiana).

Symptoms

Mild mosaic is associated with non-uniform coloration of the leaf blade, with intermingled normal, yellowish and light green small patches (Figure 2). SCMMV has been found naturally in sugarcane only in mixed infections with sugarcane bacilliform virus (SCBV), sugarcane yellow leaf virus, sorghum mosaic virus, and sugarcane mosaic virus. In some *Saccharum officinarum* clones, like Selemi Bali, striate mosaic symptoms were observed. However, both SCBV and SCMMV were detected in Selemi Bali, which makes it harder to discern symptoms caused by one or both viruses. In most varieties, including commercial sugarcane hybrids, mixed infection by SCMMV and other viruses listed above are not associated with any distinct foliar symptoms.

Diagnosis

At first, SCMMV was detected only by immunosorbent electron microscopy using partially purified leaf extracts (Lockhart *et al*., 1992 and 1996). Antisera to SCMMV were prepared from plants co-infected with SCBV, and these antisera do, therefore, not discriminate between SCMMV and SCBV in serological assays.

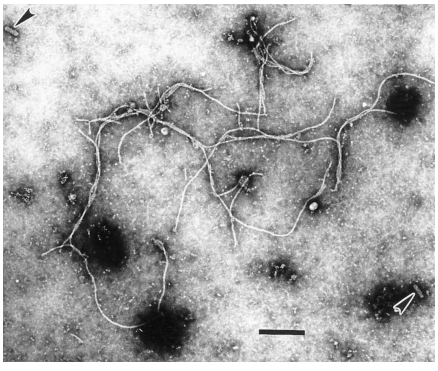

Figure 1. Particles of SCMMV in a partially purified preparation of variety B41227 from Malawi. This variety was also infected with SCBV (indicated by arrowheads); scale bar = 200 nm (B.E. Lockhart).



Figure 2. Mild mosaic leaf symptoms in sugarcane (A: Y. Huang; B: MSIRI).

Recent use of high throughput sequencing (HTS) targeting virion-associated nucleic acids or whole genome sequencing resulted in successful detection of SCMMV in sugarcane. Based on SCMMV genome data, an RT-PCR assay targeting the *hsp70* gene was developed (Mollov *et al.*, 2023). Additionally, a more sensitive immunocapture RT-PCR technique was also developed using the original antiserum produced by B.E.L. Lockhart and recently obtained genome sequences. An RT-PCR assay using specific primers SCMMV-TF and SCMMV-TR, resulting in the amplification of a 264-bp fragment of the *P24* gene, became also recently available for the detection of the causal agent of mild mosaic (Yin *et al.*, 2025).

Strains of the pathogen

Three distinct groups of SCMMV isolates named A, B, and C were identified by comparing whole genome sequences and a survey by RT-PCR of infected varieties from various origins. An example of distinct genetic signature is the P6 peptide, with group C isolates having a longer P6 peptide than those of isolates of groups A and B. Additionally, group C isolates have a shorter ORF1a protein than isolates of groups A and B (Mollov *et al.*, 2023).

Transmission

SCMMV is spread in infected cuttings and can be transmitted by the pink sugarcane mealybug, *Saccharicoccus sacchari* (Figure 3). Transmission tests have not been carried out with other mealybug species such as *Dysmicoccus boninsis*, the grey sugarcane mealybug. Mealybug-transmitted clostero-like viruses also occur in pineapple

(Gunasinghe and German, 1989) and grapevine (Rosciglione *et al*., 1983). No clostero-viruses or clostero-like viruses are known to be transmitted by seed or pollen (Bar-Joseph and Murant, 1982). It is also highly unlikely that SCMMV can be transmitted by mechanical contact with cutting tools or machinery.



Figure 3. The pink sugarcane mealybug, *Saccharicoccus sacchari*, a vector of sugarcane mild mosaic virus (P. Rott).

Host range

SCMMV occurs naturally in *S. barberi*, *S. officinarum*, *S. sinense*, and *Saccharum* hybrids including M27-16, N14, NCo376, B41-227, Pindar and Waya (Ganoo *et al*., 1998). The virus has been transmitted by mechanical inoculation and by *S. sacchari* to rice (*Oryza sativa*) and Johnson grass (*Sorghum halepense*), in which mild mosaic symptoms occur, resembling those induced in sugarcane.

Epidemiology

SCMMV is widely prevalent and occurs in all continents where sugarcane is grown but conditions for its dissemination and for disease development are so far unknown. Detailed occurrence and frequency of infection is not readily available.

Economic importance

There is no information on the actual or potential economic importance of SCMMV. The virus was identified in multiple sugarcane varieties throughout the world. Due to mixed infections with other viruses, the role of SCMMV in the disease etiology cannot be determined. It is possible that the coinfection of SCMMV and other sugarcane viruses may predispose sugarcane to damage by other stress factors or diseases.

Control

SCMMV is not eliminated from sugarcane setts by standard hot water treatments. For germplasm exchange or field propagation, plant material can be indexed and certified virus free by RT-PCR and HTS testing.

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Mosaic

Michael P. Grisham, revised by Philippe Rott

Cause

Sugarcane mosaic virus or SCMV (species name = *Potyvirus sacchari*, family *Potyviridae*) and sorghum mosaic virus or SrMV (species name = *Potyvirus sorghitessellati*, family *Potyviridae*) (Figure 1).

Note: Strain F of SCMV has been renamed sugarcane streak mosaic virus (SCSMV) and is now the causal agent of sugarcane streak mosaic (Daugrois *et al.*, 2024).

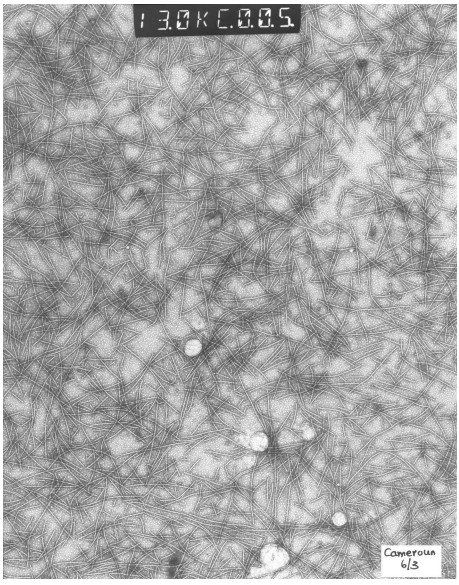

Figure 1. Particles of sugarcane mosaic virus (about 750 nm in length) in a purified preparation of sugarcane leaves (D. Gargani).

Geographical distribution

Andaman Islands, Angola, Argentina, Australia, Bangladesh, Barbados, Belize, Bolivia, Brazil, Burundi, Cambodia, Cameroon, Cape Verde, China, Colombia, Côte d'Ivoire, Congo, Costa Rica, Cuba, Democratic Republic of the Congo, Dominican Republic, Ecuador, Egypt, El Salvador, Eswatini, Ethiopia, Fiji, Gabon, Ghana, Guatemala, Haiti, Hawaii, Honduras, India, Indonesia, Iran, Iraq, Italy, Jamaica, Japan, Kenya, Laos, Madagascar, Malawi, Malaysia, Mexico, Morocco, Myanmar, Nepal, Nicaragua, Nigeria, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Sierra Leone, St Kitts and Nevis, St Thomas, South Africa, Spain, Sri Lanka, Surinam, Tanzania, Taiwan, Thailand, Trinidad and Tobago, Türkiye, Uganda, Uruguay, USA, Venezuela, Vietnam, Zambia, Zimbabwe.

The disease was reported but has not been seen for many years in Barbados, Guadeloupe, Madagascar, Martinique, and Réunion. The disease was never detected in Mauritius, even at a time when the neighboring island Réunion was highly affected by this disease during the 20th century.

Symptoms

A multicolor drawing representing an unusual striping on sugarcane leaves was first disclosed in 1893 with a note by Arendsen Hein (Figure 2). This striping was named "gele strepenziekte" or yellow-stripe disease, the former name of mosaic disease (van Musschenbroek, 1893). The general symptom of mosaic is a pattern of contrasting shades of green resulting from varying levels of chlorophyll concentration on the leaf blade (Figures 3 and 4) (Lu et al., 2021). The lighter green or yellowish chlorotic areas usually have diffuse margins, but the response of some varieties to certain strains may result in sharply defined chlorotic areas accompanied by reddening or necrosis. A red or reddish-brown discoloration of the midrib may also be associated with certain variety and virus strain combinations (Koike and Gillaspie, 1989).



Figure 2. Multicolor drawing representing the first description of mosaic symptoms in 1893 [Reproduced from Arendsen Hein S.A. (1893), with the authorization of the Indonesian Sugar Research Institute].

Symptoms of mosaic may vary in intensity with variety, growing conditions, temperature and strain of the virus. The chlorotic areas are most easily seen in young, rapidly growing leaves, particularly near the basal portion of the leaf. The proportion of the leaf that is covered by the chlorotic areas may vary from scattered, short yellowish stripes to chlorotic areas that predominate over the leaf blade with islands of normal green. On older leaves, the symptoms tend to fade.

Chlorotic symptoms may extend to the leaf sheath and stalk, particularly in noble canes (*Saccharum officinarum*). Stalk symptoms are rare among interspecific hybrid varieties. With a longitudinal section of the stalk, long necrotic areas are sometimes visible, and the nodal region may have a reddish hue.



Figure 3. Various leaf symptoms of sugarcane mosaic caused by sugarcane mosaic virus (P. Rott).

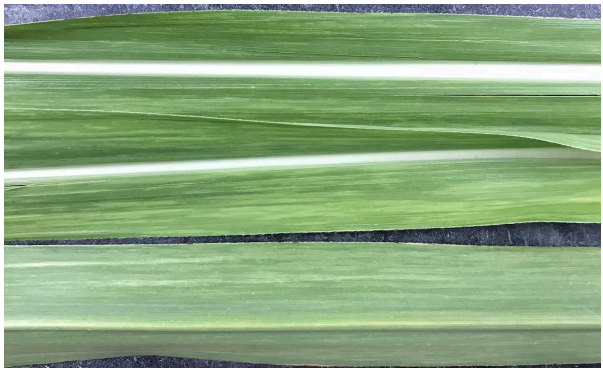

Figure 4. Leaf symptoms of sugarcane mosaic caused by sorghum mosaic virus (K. Warnke).

Diagnosis

Visual observation of symptoms is the primary method of diagnosis. The time after aphid transmission or mechanical inoculation until symptoms are expressed varies in response to age and variety of sugarcane, growing conditions, and strain of the virus. Symptoms appear earlier in young, rapidly growing plants than in plants growing more slowly. Symptoms usually appear on the young, developing leaves in about 10 days, but may appear as early as 6–7 days or be delayed for 20–30 days. ELISA or RT-PCR protocols are rarely used for routine field diagnosis of mosaic (Smith and Van de Velde, 1994; Yang and Mirkov, 1997). However, these assays are very useful for diagnosis of mosaic on some clones of *S. spontaneum* where symptoms are difficult to see because the plant has very thin leaves or where symptoms are not produced. The immunological and nucleic acid-based assays can be used to confirm that quarantined germplasm or cane to be used for seed cane propagation is mosaic free but also for proper identification of the causal agent in a location where mosaic-like symptoms are caused by different virus species (He *et al.*, 2023; Marie-Jeanne *et al.*, 2000; Viswanathan *et al.*, 2018).

Strains of the pathogen

Until the late 1980s, most if not all viruses causing mosaic symptoms in Poaceae grasses were believed to be strains of sugarcane mosaic virus (SCMV). At least 14 pathogenic strains (A-N) were reported for this virus in sugarcane based on the symptoms produced on a set of differential host plants (Koike and Gillaspie, 1989). In 1989-1991, the SCMV complex was split into four different virus species using amino acid sequences of the coat protein and nucleic acid genome sequences, namely *Sugarcane mosaic virus*, *Sorghum mosaic virus* (former SCMV strains H, I, and M), *Johnson grass mosaic virus*, and *Maize dwarf mosaic virus* (McKern et al., 1991; Shukla et al., 1989). A few years later, SCMV strain F was also moved to a new virus species named *Sugarcane streak mosaic virus* (Hall *et al.*, 1998). Johnson grass mosaic virus (JGMV) does not naturally infect sugarcane. Maize dwarf mosaic virus (MDMV) strains B (SCVM-MDB), D, E, and F do not cause mosaic on sugarcane either, but there is a report of an unspecified strain of MDMV being recovered from sugarcane. In Florida, isolates of SCMV from Columbus grass, St Augustine grass, and sugarcane exhibit different virulence patterns (Hincapie *et al.*, 2021). ELISA can be used to distinguish strains of SCMV from strains of SrMV but not strains within the two viruses. An RT-PCR-based restriction fragment length polymorphism analysis can be used to identify strains without the use of differential host clones (Yang and Mirkov, 1997). Nevertheless, genome sequencing and phylogenetic analysis are currently the most appropriate and widely used methods to identify genetic variation with these viruses (Hincapie *et al.*, 2021; Mollov *et al.*, 2025; Xu *et al.*, 2023). SCMV and SrMV have been reported throughout the world, but SCMV strains have been more widely reported among sugarcane production areas.

Transmission

The primary means of plant-to-plant transmission of the mosaic viruses is by several aphid vectors including *Dactynotus ambrosiae, Hysteroneura setariae, Longiunguis sacchari, Rhopalosiphum maidis,* and *Toxoptera graminum*, and the primary spread of the disease from field to field is by planting infected seed cane (Lu et al., 2021). Plants can be mechanically inoculated with the viruses for field or laboratory experiments but spread in the field by farm implements including knives and harvesters is negligible (Koike and Gillaspie, 1989). There are no reports of transmission of SCMV or SrMV in sugarcane by true seed or "fuzz".

Host range

In addition to species of *Saccharum*, a number of wild and cultivated grasses have been reported as natural hosts of SCMV, including the genera *Arundinaria, Brachiaria, Cynodon, Dactyloctenium, Digitaria, Echinochloa, Eleusine, Elytrigia, Eragrostis, Erianthus (Ripidium), Panicum, Paspalum, Paspalidium, Pennisetum, Rhynchelytrum, Rottboellia, Setaria, Sorghum, Stenotaphrum, Tripsacum,* and *Zea* (Koike and Gillapsie, 1989; Rao *et al*., 1990). Sugarcane, sorghum and maize are the major crops affected by this virus worldwide. In China, SCMV can also naturally infect and cause symptoms in canna (*Canna* spp.) and in pumpkin plants (*Cucurbita moschata*) (Tang *et al*., 2018; Zhao *et al*., 2019). In Florida, the virus was also detected in red Maranta (*Maranta leuconeura* var. *erythroneura*) imported from Costa Rica (Baker *et al*., 2010).

The reported hosts susceptible to strains of SrMV are *Erianthus* spp., *Saccharum* spp., *Sclerostachya fusca,* and *Sorghum bicolor* (Grisham *et al.*, 1992; Koike, 1980; Koike and Gillaspie, 1989; Giorda *et al.*, 1986).

Epidemiology

Aphid transmission of the mosaic virus in the field involves interaction among the vector, plant, and virus. Spread of mosaic is generally more rapid and the incidence is higher in subtropical than in tropical areas. This is probably caused by a high population of aphids moving from dying weeds in the autumn and spring to young, rapidly growing plants at their most susceptible stage of growth.

Economic importance

Yield loss caused by mosaic varies greatly depending on the cultivar of sugarcane and strain of the virus. Cultivars susceptible to infection may be tolerant or intolerant to yield losses or, infrequently, may recover from infection. In a 14-year study in Louisiana among commercially released cultivars with different susceptibilities to mosaic, mosaic caused yield losses (sugar/ha) ranging from 7 to 21% over a 3-year crop cycle (Grisham *et al.*, unpublished). Mosaic in combination with other diseases often reduces growth and yield more than each disease separately. For example, mosaic and ratoon stunt, caused by the bacterium *Leifsonia xyli* subsp. *xyli*, together on cultivar NCo310 were found to cause greater yield loss than that caused by each disease separately. Also, additive and synergistic effects of mosaic and Pythium root rot on reducing growth and weight of several sugarcane cultivars have been reported (Koike and Gillaspie, 1989). Perhaps the greatest loss caused by mosaic has been the loss of germplasm beginning with susceptible noble-type canes in the early part of the 20th century and continuing with the loss of many cultivars and promising clones as new strains of the virus have appeared.

Control

Development and use of resistant cultivars is the most effective method of controlling mosaic (Lu et al., 2021). The diversity of germplasm used in breeding programs has been increased through the collection and use of clones of wild relatives of sugarcane. A major objective is to identify mosaic resistance among the clones of these wild relatives (Grisham *et al*., 1992; Koike, 1980). The recent advances in developing transgenic cultivars resistant to mosaic is a potential new approach to the control of mosaic.

When resistant cultivars are not available or where mosaic pressure is not intense, the use of mosaic-free seed cane may prove effective as a control measure. Methods to free infected cane of the virus include serial hot water treatments (Benda, 1972; Huang *et al.*, 2026) and apical meristem culture (Cheong *et al.*, 2012). Several tolerant cultivars continue to be used in Louisiana with the commercial production of mosaic-free seed cane. Once tissue free of the pathogenic virus is obtained, micropropagation is used to increase rapidly the number of mosaic-free plants. Chemical roguing is used to maintain mosaic-free seed cane plots from which seed cane is sold to farmers.

Timing of planting and harvest may help to reduce the incidence of mosaic infection of susceptible cultivars by ensuring that the early, rapid growth of the sugarcane does not coincide with high levels of vector activity.

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Ramu stunt

Dimitre Mollov, Robert C. Magarey

This chapter has been updated and rewritten based on the original chapter entitled "Ramu stunt" and authored by Sidney Suma and Phil Jones in 2000.

Cause

Ramu stunt virus or RmSV (species name = *Mechlorovirus ramuense*, family *Phenuiviridae*).

Geographical distribution

Papua New Guinea.

Symptoms

Ramu stunt is characterized by stunting and leaf symptoms; the growth reduction can occur suddenly and rapidly (Waller *et al*., 1987) (Figure 1). Additional symptoms are poor ratooning and leaf symptoms that vary according to the sugarcane variety and/or growing conditions. These leaf symptoms include a series of leaf streaks, flecks and leaf tip shrivelling (Figures 2 and 3). Initial leaf symptoms are irregular, short, pale to creamy green flecks or streaks. These streaks later increase in size, turn yellowish-green, and can be as long as the entire leaf blade. A grassy growth habit coupled with shorter leaves and internodes are also associated with the disease (Figure 4) (Braithwaite *et al*., 2019a). The root system is also reduced in size and becomes necrotic in severely affected plants that ultimately die.



Figure 1. Stunting and trashy appearance of a sugarcane crop affected by Ramu stunt (P. Jones).



Figure 2. Various colors and shapes of leaf streaks and flecking caused by Ramu stunt virus (P. Rott).



Figure 3. Irregular streaks on sugarcane leaves affected by Ramu stunt (D. Mollov).



Figure 4. Shortened and malformed internodes on variety Ragnar under greenhouse conditions (D. Mollov).

Diagnosis

Diagnosis via visual symptoms alone is not reliable due to the variable and sometimes non-classical stunting and leaf streaking. Molecular assays provide a very specific diagnosis based on detection of the viral causal agent. A phytoplasma was initially considered as the cause of Ramu stunt disease (Cronje *et al.*, 1999). Few years later, in samples exhibiting Ramu stunt, Braithwaite and collaborators detected viral proteins using ultracentrifugation and protein hybridization followed by protein sequencing (Braithwaite *et al.*, 2007). Nucleic acid sequencing primers were generated, and the virus polymerase gene was amplified, cloned, and sequenced. An RT-PCR detection method was developed and validated to confirm the cause of Ramu stunt as a virus. Later on, the

full genome sequence of RmSV was obtained using high throughput sequencing (HTS) technology (Mollov *et al*., 2016; Braithwaite *et al*., 2019b). RmSV has a multipartite genome consisting of nine RNA fragments and a diagnostic assay was developed based on primers detecting RNA1 and RNA6 segments of the virus (Abrahamian *et al*., 2026; Braithwaite *et al*., 2019a).

Strains of the pathogen

Distinct strains of the pathogen have not been formally identified as only six isolates have been fully sequenced. These genomic sequences were from different *S. officinarum* specimens obtained from Alotau, Asas, and Gusap in Papua New Guinea. Additionally, genomic sequences were obtained from three commercial varieties (R570, PN97-54, and Ragnar). While distinct strains have not been recognized, it is notable that the isolate from Alotau differed significantly in genomic sequence identity from the other five sequenced isolates (Braithwaite *et al*., 2019b). Alotau is in the southern part of Papua New Guinea and causal agent evolution in geographical isolation could explain the genetic differences between the isolates. This was supported by a phylogenetic analysis of the RNA6 from 34 RmSV samples with six from Alotau (Braithwaite *et al*., 2019a). All six Alotau virus isolates grouped separately and had only 80.6–82% nt identity with the other isolates from more northern locations. Another less discerning but notable separation was that all isolates from hybrid varieties grouped separately from those obtained from garden grown noble canes (*Saccharum officinarum*).

Transmission

RmSV can be spread by propagating infected setts and by the insect vector *Eumetopina flavipes*, which is common in sugarcane crops and garden canes in Papua New Guinea (Figure 5) (Kuniata *et al*., 1994). Insect transmission was the likely reason for the rapid spread across the Gusap Estate in the 1985-86 seasons. After RmSV was confirmed as the causal agent for Ramu stunt and a confirmatory diagnostic test became available, a controlled transmission study with *E. flavipes* was carried out. Symptoms started to appear about two months after viruliferous insects fed on healthy plants. The virus transmission by the vector *E. flavipes* was then confirmed using three RT-PCR primer pairs and by silver stained SDS-PAGE RmSV protein (Braithwaite *et al*., 2019a).

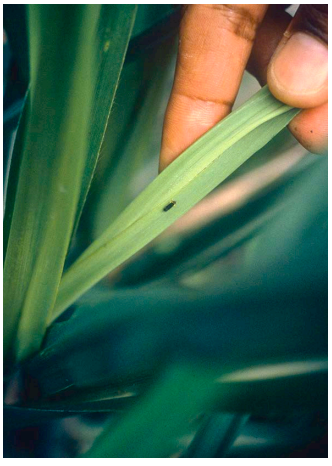

Figure 5. *Eumetopina flavipes*, the vector of Ramu stunt virus (R. Magarey).

Host range

Ramu stunt occurs through Papua New Guinea with symptoms assigned to the disease observed during a survey on *Saccharum officinarum*, *S. robustum*, as well as on hybrid sugarcane varieties (Magarey *et al.,* 2002). Visual symptoms were later confirmed by RT-PCR detection (Braithwaite *et al*., 2019a). No other hosts are known for Ramu stunt. Ramu stunt-like symptoms were also observed on *Imperata cylindrica* at a time when the disease was tentatively attributed to a phytoplasma, and the presence of RmSV in this plant species has not been confirmed by molecular assay.

Epidemiology

Ramu stunt can occur in Papua New Guinea in sugarcane grown at sea level up to 2000 m altitude. The disease may spread very quickly across crops of susceptible varieties, as evidenced in the 1985-86 Ramu stunt epidemic at Ramu Sugar, Gusap (Eastwood 1990; Waller *et al*., 1987). This was due to high vector populations coupled with a very susceptible variety. Populations of the insect vector and disease incidence were notably high during the wet season. With the initial epidemic brought under control, only minor occurrences of the disease have occurred at Gusap as the Estate now crops varieties either resistant or of intermediate resistance. The disease seen in commercial crops in recent times appears to be linked to diseased planting material and minor insect transmission.

Economic importance

The cropping of the susceptible variety Ragnar when the Estate was first established led to severe Ramu stunt and devastating economic losses. The disease almost caused Estate bankruptcy (Suma and Jones, 2000). With the transition to more resistant varieties, losses became minor. Ramu stunt only remains a threat to crop production in susceptible varieties. For this reason, new varieties are screened for RmSV resistance on introduction to the Estate. Almost one third of varieties introduced into Papua New Guinea were eliminated between 1979-1992 because they were susceptible to Ramu stunt (Suma and Pais, 1996).

Control

The most important management strategy for Ramu stunt is the cropping of resistant varieties (Kuniata *et al*., 2010), coupled with the use of disease-free planting material. Ramu stunt resistance screening trials include infection rows with diseased cane planted in alternate rows, thus relying on *E. flavipes* for virus transmission (Braithwaite *et al*., 2012; Kuniata *et al*., 2010). Hot water treatment does not eliminate the virus. For germplasm exchange, plant material can be indexed and certified virus free by RT-PCR and HTS testing.

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Red leaf mottle

Philippe Rott, Michèle Chatenet,
revised by Philippe Rott and Jean Heinrich Daugrois

Cause

Peanut clump virus (species name = *Pecluvirus arachidis*, family *Virgaviridae*) and Indian peanut clump virus (species name = *Pecluvirus indicum*, family *Virgaviridae*).

The virus occurring in Africa is referred to as peanut clump virus (PCV) whereas the virus present in the Indian subcontinent is referred to as Indian peanut clump virus (IPCV). The two viruses cause indistinguishable disease symptoms in sugarcane. Nevertheless, PCV and IPCV are two distinct *Pecluvirus* species differing in host range, serology and nucleotide identity (Pandey *et al.*, 2017).

Geographical distribution

Burkina Faso, Chad, Congo, Gabon, India, Niger, Senegal, Sudan.

Symptoms

Several types of symptoms can be observed on well-developed leaves depending on the variety (Baudin and Chatenet, 1988; Rott, 1996). Leaves of infected plants may exhibit chlorotic bands that are elongated over the entire leaf length, from 1 cm wide to as wide as the blade. These chlorotic bands can appear spotted, yellow or rust-colored (Figures 1 to 3). The entire leaf can become dark red with age (cultivar Co 1001, B70574, etc.). Leaves may also show numerous wine-red spots with diffuse edges and no clearly defined shape that develop on both sides of the blade in some varieties (cultivar Ragnar, etc.) (Figure 4). White stripes can also appear on the leaves of some varieties, resembling the stripes caused by leaf scald (caused by *Xanthomonas albilineans*) but they have wavy edges and develop on any part of the blade without being linked to the midrib (Figure 5). A few isolated leaves develop white diamond-shaped or partly diamond-shaped stripes on the surface of the leaves (cultivars B51129, B51410, B7134, etc.).

Diagnosis

The leaf dip technique can be used to observe the pathogen by electron microscopy in leaves or roots sampled from symptomatic plants (Baudin and Chatenet, 1988). The virus forms rigid rods of variable length in sugarcane leaf preparations. Particles' length ranges from 200 to 250–300 nm and the diameter is approximately 20 nm (Figure 6). The virus can also be detected in leaves, stalks and roots by various serological methods (ELISA, dot-blot, tissue-blot) using an antiserum prepared with purified virus (Baudin and Chatenet, 1988; Chatenet and Saeed, 1995). PCV and IPCV can also be detected and quantified by RT-PCR using primers targeting the highly conserved 3'-untranslated region of pecluviruses (Dieryck *et al.*, 2010).



Figure 1. Chlorotic elongated streaks or bands on the leaf with rust-colored spots (J.-C. Girard).



Figure 2. Red bands and streaks on the leaf blade (J.-C. Girard).



Figure 3. Wine-red leaf streaks that coalesce on the leaf blades (J.H. Daugrois).



Figure 4. Wine-red leaf spots with diffuse edges on both sides of the leaf blade (P. Rott).



Figure 5. White stripes on the leaves (P. Baudin).

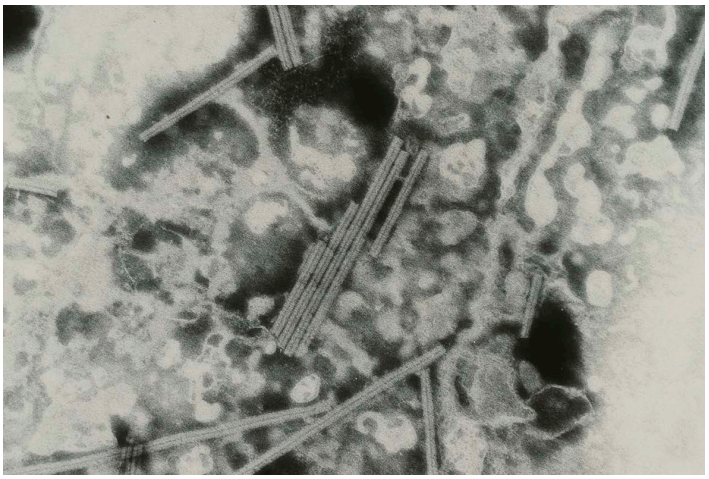

Figure 6. Virus particles (200–300 nm long) observed by transmission electron microscopy (M. Chatenet).

Strains of the pathogen

There is no information available regarding strain variation of PCV in sugarcane. However, two major strains were identified in groundnut: the original PCV strain that was found in Africa and a second strain that was identified in India and called Indian peanut clump virus (Thouvenel *et al*., 1976; Reddy *et al.*, 1983). The biological and physical properties of the two strains are identical but great variation was observed in their serological properties (Reddy *et al*., 1985). Additionally, characterization of PCV with monoclonal antibodies showed the existence of five serotypes of the virus (Huguenot *et al*., 1989).

Transmission

Red leaf mottle is a soil-borne disease. It has been proven experimentally that the disease can be transmitted through the soil, not only from sugarcane to sugarcane, but also from sugarcane to groundnut. The virus can be transmitted from sugarcane to cereal species and from groundnut to groundnut by an obligate parasite of plant roots, *Polymyxa graminis*, which is a Cercozoa of the *Plasmodiophoraceae* family (Dieryck *et al.*, 2011; Thouvenel and Fauquet, 1981; Xu *et al*., 2018). The disease is also transmitted by cuttings taken from symptomatic sugarcane stalks. Besides transmission by *P. graminis*, the virus infecting groundnut is also seed-transmitted (Delfosse, 2000). Seed (fuzz) transmission has not been reported in sugarcane.

Host range

Sorghum and groundnut are the major hosts of PCV, and the pathogen is only occasionally found in sugarcane. The virus has also been identified under natural conditions in *Sorghum arundinaceum* in Burkina Faso (Dollet *et al.*, 1976). In India, IPCV infects barley, cowpea, chili, groundnut, maize, pigeon pea, sorghum, sugarcane and wheat (Delfosse *et al.*, 1995; Pandey et *al*., 2017). Virus isolates obtained from sugarcane cannot be mechanically transmitted to sugarcane nor to maize. However, symptoms such as mosaic or necrotic spots were observed after artificial inoculation of several species of *Nicotiana, Chenopodium amaranticolor* and *C. quinoa* (Baudin and Chatenet, 1988; Rao and Singh, 1999).

Epidemiology

Sandy soils with pH of 7.0 and higher particularly favor transmission. In some fields, there are areas of infected sugarcane where the disease reappears after replanting, even if a different variety is grown. Rainfalls increase transmission of the virus and incidence of the disease as humid soils favor the free movement of the motile zoospores of the vector *P. graminis* (Pandey et *al*., 2017).

Economic importance

Red leaf mottle caused losses in yield trials in Senegal, but in plant cane only (Baudin *et al*., 1989). However, no yield losses due to red leaf mottle have been reported in commercial sugarcane fields so far.

Control

No efficient method has yet been reported to eliminate the virus from infected sugarcane cuttings. Soaking cuttings in water for 3 h at 50°C does not eradicate the virus. Stools that show symptoms should be eliminated in multiplication plots or nurseries. As red leaf mottle of sugarcane has been reported in only a few countries in Africa, great attention should be paid during germplasm exchange involving affected locations.

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Streak

Philippe Rott, Michel Peterschmitt,
revised by Jean Heinrich Daugrois and Philippe Rott

Cause

At least six viruses in the genus *Mastrevirus* cause streak symptoms in sugarcane:
1. Maize streak virus A (MSV-A; species name = *Mastrevirus storeyi*, family *Geminiviridae*) (Van Antwerpen *et al.*, 2011; Yahaya *et al.*, 2017).
2. Saccharum streak virus (SacSV; species name = *Mastrevirus sacchari*, family *Geminiviridae*) (Lawry *et al.*, 2009).
3. Sugarcane chlorotic streak virus (SCSV; species name = *Mastrevirus saccharumpallidi*, family *Geminiviridae*) (Yahaya *et al.*, 2017).
4. Sugarcane streak Egypt virus (SSEV; species name = *Mastrevirus saccharumegyptense*, family *Geminiviridae*) (Bigarré *et al.*, 1999).
5. Sugarcane streak Reunion virus (SSRV; species name = *Mastrevirus saccharumbourbonense*, family *Geminiviridae*) (Bigarré *et al.*, 1999).
6. Sugarcane streak virus (SSV; species name = *Mastrevirus saccharofficinari*, family *Geminiviridae*) (Hughes *et al.*, 1993).

These six viruses are known as the sugarcane infecting streak virus (SISV) group and are part of the African streak virus (AfSV) group that includes nine different species. The genome structure of mastreviruses comprises four coding sequences (CDS), a long intergenic region (LIR), and a short intergenic region (SIR). These CDS encode four proteins, namely the movement protein (MP), the coat protein (CP), the replication protein (Rep), and the replication-associated protein A (RepA).

Geographical distribution

Benin, Cameroon, Cape Verde, Côte d'Ivoire, Egypt, India, Kenya, Madeira, Malawi, Mauritius, Mozambique, Nigeria, Pakistan, Réunion, South Africa, Sudan, Uganda, Zimbabwe.

At least one SISV was found in sugarcane in the above-mentioned countries/locations.

Symptoms

Infected leaves are covered with numerous small spots or streaks which are translucent to white, narrow and parallel to the veins. After infection, the streaks are irregularly distributed and observed mostly on the lower part of the leaf blade. These symptoms, however, tend to be uniformly distributed on all leaves once the virus is established in the plant (Storey and Thomson, 1961). The streaks are usually 0.5–2 mm wide and vary in length from 0.5 mm to more than 2 cm (Figures 1 to 7). The longest streaks (several cm) were observed on plants infected by SCSV (Daugrois *et al.*, 2025), maize streak virus (MSV) (McFarlane *et al.*, 2008), and SacSV. The streaks observed

on young leaves that develop from an infected cutting can be relatively wide, they coalesce and sometimes form a pattern of mosaic (Storey and Thomson, 1961). For some sugarcane varieties, streak symptoms can also be observed on the rind of the stalk (Figure 8). Highly susceptible varieties are affected in growth and show internode stunting (Figure 9).



Figure 1. Sugarcane leaf with streak symptoms caused by maize streak virus A (S. McFarlane).



Figure 2. Leaf symptoms of sugarcane streak caused by maize streak virus A (G. Buchanan).



Figure 3. Sugarcane leaf spindle with streak symptoms caused by maize streak virus A (A. Yahaya).



Figure 4. Leaf symptoms of sugarcane streak caused by sugarcane chlorotic streak virus (J.H. Daugrois).



Figure 5. Leaf symptoms of sugarcane streak caused by sugarcane chlorotic streak virus (A. Yahaya).



Figure 6. Sugarcane streak symptoms caused by sugarcane streak Reunion virus on the upper side of the leaf (P. Rott).



Figure 7. Sugarcane streak symptoms caused by sugarcane streak Reunion virus on the lower side of the leaf (P. Rott).



Figure 8. Sugarcane streak symptoms caused by sugarcane streak Reunion virus on the stalk rind (P. Rott).



Figure 9. Stunting of stalk internodes caused by sugarcane chlorotic streak virus (A. Yahaya).

Diagnosis

Symptoms are the first criteria to diagnose a potential mastrevirus infection in sugarcane. Symptoms are best observed on younger leaves as the streaks tend to become more diffuse and less marked as the leaf ages (Bock and Bailey, 1989). However, the different SISVs cannot be differentiated based on streak symptoms.

PCR assays targeting a specific gene and rolling circle amplification, both followed by sequencing and sequence analysis, can be used for diagnosis of SISVs and accurate identification of the causal agent. Primers SSV_1732F and SSV_2176R[19] have been developed for simultaneous detection of MSV-A, SacSV, SSEV, SSRV, and SSV (Candresse *et al.*, 2014). These primers can also be used for detection of SCSV, although the target genome sequence of this virus has two nucleotide mismatches with forward primer SSV_1732F. Although more time-consuming and more costly, high-throughput sequencing using extracted plant DNA is an alternative approach for SISV identification, especially for new mastreviruses (Kraberger *et al.*, 2017).

Strains of the pathogen

SISVs belonging to six different virus species of the genus *Mastrevirus* were found in sugarcane exhibiting streak symptoms in Africa and the Mascarenes: SSV in South Africa, MSV-A in South Africa and Nigeria, SacSV in South Africa, SSRV in Réunion and Mauritius islands, SSEV in Egypt and SCSV in Cameroon and Nigeria.

Very few experiments were conducted so far to compare pathogenicity of SISVs. Nevertheless, Golden Bantam maize plants infected by SSRV (previously known as sugarcane streak Mauritius virus or SSMV) showed milder symptoms than plants infected by SSV and SSEV isolates (Bigarré *et al.*, 1999; Pinner *et al.*, 1988). After transmission of MSV/A from sugarcane to maize by *Cicadulina mbila*, plants exhibited a pattern of fine streaks resembling those caused by the pathogen in sugarcane (Pinner *et al.*, 1988). The symptoms were, however, milder than those caused by MSV/A isolates from maize.

19. SSV_1732F: 5′-CARTCVACRTTRTTYTGCCAGTA-3′; SSV_2176R: 5′-GARTACCTYTCHATGMTHCAGA-3′.

Transmission

The sugarcane streak mastreviruses cannot be transmitted mechanically from plant to plant or through true seed (Bock and Bailey, 1989). They are spread by cuttings taken from infected plants and by leafhopper species of the genus *Cicadulina* in a persistent circulative manner. These leafhopper species include *C. mbila*, *C. bipunctella* and others depending on identity of the SISV. For example, at least nine of the African *Cicadulina* species are vectors of MSV (Alegbejo *et al.*, 2002). The capsid protein of the virus is a key determinant for virus transmission by leafhoppers (Whitfield *et al.*, 2015).

Host range

Wild and cultivated Poaceae species can be infected by SISVs. In Réunion Island, SSV was found in *Cenchrus myosuroides*, SSRV in *Brachiaria umbellata*, *Dactyloctenium* sp., *Digitaria ciliaris*, *Setaria barbata* and *Sorghum arundinaceum*, MSV-A in *Cenchrus echinatus*, *Cyperus rotundus*, and *Zea mays* (Ben Chédida *et al.*, 2024). SSRV was also identified in *Paspalum conjugatum* in Zimbabwe and *Eleusine coracana* in Nigeria (Kraberger *et al.*, 2017). MSV was recently first reported in rice fields in Burkina Faso (Fouad *et al.*, 2025).

Saccharum officinarum and *S. sinense* are susceptible to streak, whereas *S. barberi* and *S. spontaneum* are relatively resistant. In Mauritius, *Cenchrus echinatus* and *Coix lacryma-jobi* were described as hosts of streak virus isolates (presumably SSRV isolates). In Réunion, however, *Coix lacryma-jobi* did not show streak symptoms after insect transmission of the virus, whereas *Cenchrus echinatus* did (Peterschmitt *et al.*, 1991).

Epidemiology

Streak incidence is closely related to the pathogen's vector population dynamics which, in turn, is influenced by rainfall, temperature, and availability of alternative hosts (Alegbejo and Banwo, 2005). Wild grasses infected by SISVs and growing in proximity of sugarcane fields are potential natural reservoirs for streak epidemics in sugarcane.

Economic importance

Streak had a considerable impact on sugarcane production in South Africa in the 1920s and 1930s when the Natal sugar industry relied almost entirely on the susceptible variety Uba (Bock and Bailey, 1989; Storey and Thomson,1961). The disease was shown to cause yield losses between 8 and 11% for several crops in experimental fields established with diseased planting material. Streak declined between 1935 and 1945 with the replacement of the Uba variety by resistant varieties. Similarly, the decline of the disease in Egypt coincided with the extensive cultivation of variety C9 (Bigarré *et al.*, 1999). Varieties grown in the countries where the disease is reported to occur have some resistance, and streak is presently considered of little importance.

Control

Use of resistant varieties is the most effective means of control and, therefore, susceptible varieties should be eliminated. Screening of sugarcane for resistance to streak can be performed in breeding programs by planting varieties between rows of diseased plants. When needed, as it is the case for germplasm movement in quarantine, SISV-free plants can be obtained from infected sugarcane by apical meristem tissue culture.

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Streak mosaic

Jean Heinrich Daugrois, Philippe Rott

Cause

Sugarcane streak mosaic virus or SCSMV (species name = *Poacevirus sacchari*, family *Potyviridae*) (Figure 1). Until 1998, this virus was known as sugarcane mosaic virus strain F (Hall *et al.,* 1998).

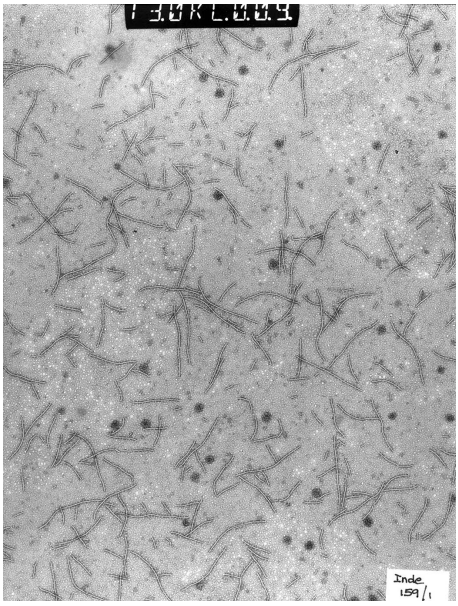

Figure 1. Virus particles of sugarcane streak mosaic virus (680 to 900 nm in length) observed by transmission electron microscopy (D. Gargani).

Geographical distribution

Bangladesh, China, Côte d'Ivoire, India, Indonesia, Iran, Japan, Myanmar, Pakistan, Sri Lanka, Thailand, Vietnam, Zambia.

SCSMV was also found in the USA (Louisiana) in one variety in the 1930s, as well as in 2018 in imported germplasm, but the virus did not spread to local varieties and has been eradicated in this geographic location (Daugrois *et al.,* 2024).

Symptoms

Symptoms of streak mosaic are patterns of green colors on the leaf lamina, which include longitudinal, yellowish-green streaks (3–40 mm long) and light to dark green islands (Figure 2). The symptoms are easy to recognize on the spindle leaves and on the top visible dewlap leaf, but they often disappear on older leaves (Putra *et al.,* 2015).

Symptom severity varies with the host genotype and infected plants without symptoms have been reported in India and Indonesia (Bagyalakshmi and Viswanathan, 2021; Hema *et al.,* 2001; Putra *et al*., 2015). Plants may develop symptoms of streak mosaic only several weeks or months after infection, thus suggesting occurrence of a latency phase under some conditions. Presence of galls on the leaf sheath have been associated in the field with the presence of a putative mite vector of SCSMV (Figures 3 to 6).



Figure 2. Symptoms of streak mosaic on sugarcane leaves (P. Rott).

Diagnosis

SCSMV can be detected in symptomatic and asymptomatic sugarcane plants using several immunological and molecular methods: Dot-blot immunoassay (DBIA; Thompson *et al*., 2016), enzyme linked immunosorbent assay (ELISA; Wisnanathan *et al*., 2013; Prabowo *et al*., 2014), reverse transcription-polymerase chain reaction (RT-PCR; Chatenet *et al*., 2005; Rott *et al*., 2023), immunocapture-RT-PCR (IC-RT-PCR; Neliana *et al*., 2025), loop-mediated isothermal amplification (LAMP; Anandakumar *et al*., 2020), recombinase polymerase amplification (RPA)-CRISPR/Cas12a (Gao *et al*., 2025), and RT-RPA (Srivastava *et al*., 2025). Highly sensitive real-time or quantitative RT-PCR assays were also developed to determine titers of SCSMV in plants (Bagyalakshmi *et al*., 2019; Fu *et al*., 2015). A recombinant coat protein antibody was produced to diagnose SCSMV directly in leaf extracts without RNA purification, as well as by immuno-capture (IC)-RT-PCR (Hema *et al*., 2003).



Figure 3. Mite lesions (galls) on the leaf sheath of a stalk infected by sugarcane streak mosaic virus (J.H. Daugrois).

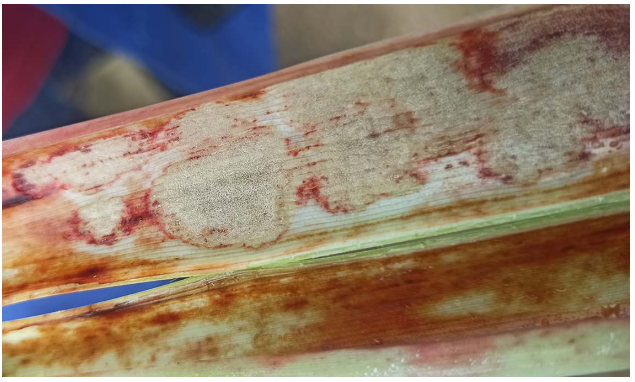

Figure 4. Colony of the mite *Aceria sacchari*, putative vector of sugarcane streak mosaic virus, within a sugarcane foliar sheath (J.H. Daugrois).



Figure 5. The mite *Aceria sacchari* observed by confocal microscopy (scale bar = 50 µm) (M.-S. Vernerey).

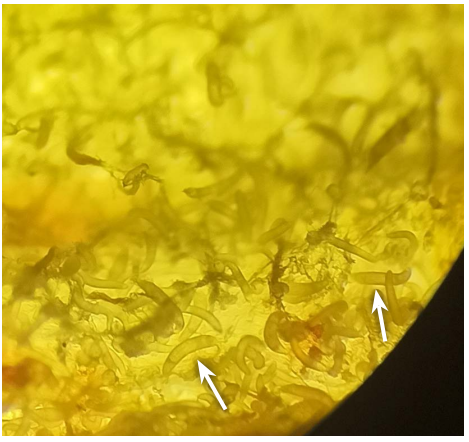

Figure 6. Colony of *Aceria sacchari* observed under the microscope. Worm shaped mites are 180–260 µm long and 30–60 µm thick. Two single mites are highlighted with a white arrow (J.H. Daugrois).

At least 10 different primer pairs were reported for RT-PCR diagnosis of SCSMV in extracted RNA. These primer sequences were polymorphic when aligned with 32 genome sequences of SCSMV available in GenBank (Daugrois *et al.*, 2024). Alignments of the forward primers revealed at least one to two nucleotide mismatches with these genome sequences. Forward primers with the least number of mismatches were SCSMVcpf

(Damayanti and Putra, 2011), SCMVF (Moradi *et al*., 2015) and ST2 (Rott *et al*., 2023). The sequences of three reverse primers matched 100% with all 32 genome sequences i.e., SCS-NIb-CP_R (Kasemsin *et al*., 2016), SCSMV-CPR2 (Fu *et al*., 2015), and ST5 (Rott *et al*., 2023).

Strains of the pathogen

The 33 isolates of SCSMV available in GenBank formed two separate lineages in a phylogenetic tree constructed with the full amino acid sequence of the virus coding-genome or polyprotein. One lineage comprised isolates from Côte d'Ivoire, China, Myanmar, Thailand, USA, and one isolate from India. The second lineage included isolates from India, Iran, and Pakistan (Daugrois *et al*., 2024). However, variation in virulence among isolates of SCSMV is not known so far.

Transmission

SCSMV can be transmitted at harvest from infected sugarcane plants to healthy ones by cutting implements (Putra *et al*., 2014). However, high levels of field contamination and disease clumping in several areas in Indonesia and Côte d'Ivoire also suggested that an arthropod vector is involved in virus spread (Magarey, 2019; Daugrois *et al.,* 2024). SCSMV was diagnosed by RT-PCR in arthropods feeding on sugarcane in Indonesia such as aphids (*Melanaphis sacchari*), lacewings (*Tingidae* sp.) and plant hoppers (*Eumetopina* sp. and *Perkinsiella* sp.), but none of them transmitted SCSMV from infected to healthy plants (Magarey, 2019). Transmissions of SCSMV by the aphids *Aphis craccivora, M. sacchari*, *Rhopalosiphum maidis* and *Ceratovacuna lanigera* were unsuccessful (Hema *et al*., 2001; Putra *et al*., 2014 and 2015).

The helper component protein (HC-Pro) of *Potyviridae* viruses is involved in vector transmission and only two vector types have been identified among the *Potyviridae* viruses possessing an HC-Pro i.e., aphids and eriophyid mites. Phylogenetic investigations based on the amino acid sequences of this HC-Pro revealed that potyviruses were associated with aphid vectors while the remaining viruses (ipomoviruses, poaceviruses, roymoviruses, rymoviruses, and tritimoviruses) were associated with eriophyid mites or unknow vectors (Daugrois *et al*., 2025). Furthermore, in the phylogenetic tree of the HC-Pro, SCSMV grouped with viruses transmitted by eriophyid mites of the genus *Aceria*.

At least 10 eriophyid mites are present on sugarcane, including *Aceria sacchari*. This mite species is absent in the Americas where the virus did not spread, and it was only found in countries where SCSMV is present in commercial sugarcane production fields. Consequently, *A. sacchari* is a good vector candidate for transmission of SCSMV (Daugrois *et al*., 2025).

Host range

SCSMV naturally infects cultivated sugarcane (*Saccharum* interspecific hybrids), noble cane (*S. officinarum*), and related species (*S. barberi*, *S. robustum*, *S. sinensis*, *and S. spontaneum*). Natural infections of SCSMV in *Sorghum bicolor* were also reported in India (Srinivas *et al*., 2010). Additional hosts of the Poaceae family can be inoculated mechanically with SCSMV and exhibit streak mosaic symptoms. They belong to several genera such as *Brachiaria*, *Dactyloctenium*, *Digitaria, Miscanthus*, *Panicum*, *Paspalum*, *Pennisetum*, *Rottboellia*, *Sorghum*, and *Zea*. In contrast, Egyptian crowfoot grass

(*Dactyloctenium aegyptium*) does not exhibit streak mosaic symptoms when infected by SCSMV (Moradi *et al*., 2018; Putra *et al*., 2014). Besides Poaceae species, SCSMV was not transmissible to plants belonging to other plant families (Putra *et al*., 2014).

Epidemiology

SCSMV is apparently transmitted very efficiently by an arthropod vector that remains to be identified. Like other systemic pathogens, the virus is also spread by infected seed cane (Putra *et al*., 2014 and 2015). Streak mosaic symptoms can sometimes be discreet and asymptomatic plants can also be infected by SCSMV. Latent infections favoring vegetative transmission of SCSMV have been reported in India and Indonesia (Hema *et al*., 2001; Putra *et al*., 2015). Cuttings taken from plants with latent infections contribute to the spread of streak mosaic to virus-free areas if no quarantine measures involving SCSMV detection are implemented.

Economic importance

SCSMV rarely causes death of infected plants and, consequently, the economic impact of streak mosaic is often minimized. Yield parameters like stalk weight and diameter, and sugar content, are affected by the disease (Ouattara *et al*., 2025; Putra *et al.,* 2015). In Indonesia, when incidence of SCSMV was >50%, cane and sugar yields of variety PS 864 were significantly reduced (Putra *et al*., 2014). Stalk juice yield and juice quality are also impacted by SCSMV infection (Bagyalakshmi *et al*., 2019). In Côte d'Ivoire, three years after the first observation of streak mosaic symptoms, 30% yield losses were observed in sugarcane fields planted in non-irrigated areas. Average yields of a highly susceptible variety with almost 100% symptomatic stalks were reduced by 70% in non-irrigated areas and 50% in irrigated areas (Daugrois *et al*., 2024). Extractable sugar yield losses were >40% in some varieties (Ouattara *et al*., 2025).

Control

Sugarcane diseases, including streak mosaic, are best controlled using resistant or tolerant varieties. However, only few varieties resistant to streak mosaic have been identified in breeding trials of locations contaminated by SCSMV. In Indonesia, three of 18 promising varieties had a low infection rate (4–7%) after inoculation with the virus (Putra *et al*., 2015). In another screening trial with promising material in Indonesia, four additional resistant accessions were identified (Suhara *et al.,* 2022). In China, only 24 of 71 inoculated elite varieties were moderately to highly resistant to SCSMV (Li *et al.,* 2018).

As breeding is a long-term goal to control streak mosaic, other measures have been evaluated to limit the impact of the disease. Hot water treatments (HWTs) are often used to eliminate sugarcane pathogens, especially fungi and bacteria, in infected planting materials. However, the thermal inactivation point of SCSMV is 55–60°C, which is higher than the sugarcane thermal death point (Damayanti *et al.,* 2010). HWTs of planting material for 10 to 30 min at 52–55°C did not eliminate SCSMV but a treatment at 53°C for 10 min drastically reduced disease severity in the plant cane crop with no impact on plant viability (Damayanti *et al.,* 2010; Putra *et al*., 2014).

Production of virus-free planting material is an essential step to limit the impact of the disease, especially in locations where vector spread of SCSMV is limited. Virus-free

material can be obtained using meristem tip (≤ 1 mm) culture (Cheong *et al.,* 2012; Mishra *et al*., 2010; Subba Reddy and Sreenivasulu, 2011). Six-month-old plants, regenerated from meristem tip culture, were symptomless and only 8% of leaves tested positive for SCSMV (Subba Reddy and Sreenivasulu, 2011). The efficacy of production of SCMV-free plants by meristem tip culture remains to be validated in older field-grown plants to make sure that disease development is not postponed, as it is the case for HWTs (Putra *et al*., 2014).

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Striate mosaic

Barry J. Croft, John W. Randles, revised by Barry J. Croft and Nicole Thompson

Cause

Sugarcane striate mosaic-associated virus or SCSMaV (species name = *Sustrivirus sacchari*) is associated with striate mosaic infected plants (Choi 1997; Choi *et al*., 1999).

Double-stranded RNA (dsRNA) approximately 9 kilobase pairs (kbp) in size was isolated from striate mosaic affected leaves of sugarcane (Choi and Randles, 1997). This dsRNA was subsequently sequenced and found to be an 8146 nt positive sense RNA genome (Thompson and Randles, 2001). A novel relatively rigid rod-shaped virus approximately 950 nm in length and 15 nm in diameter has been purified in small amounts from affected sugarcane. *S. sacchari* is the only member of the *Sustrivirus* genus in the *Betaflexiviridae* family[20], and is only distantly related to other members of this family. Only partial similarity in a small region of the genome to other viruses was found by a recent BlastN search and genome alignment (Thompson, unpublished data).

Geographical distribution

Australia.

Striate mosaic has only been reported in the Burdekin River district of Queensland.

Symptoms

Sugarcane plants infected by SCSMaV are severely stunted with a general yellow appearance. They often die or fail to ratoon leaving large bare patches in fields. Leaves show short chlorotic striations, 0.5 × 0.5 to 2 mm over the whole leaf surface and are best observed on the younger leaves (Figure 1) (Hughes, 1967). Symptoms are difficult to distinguish on older leaves and when temperatures exceed 30°C. In varieties with red stalks, striations can be clearly seen on the rind of stalks of infected plants (Figure 2). Affected stalks have shortened internodes and a reduced diameter (Choi, 1997). Infected stalks of variety Q96 have a marked restriction around the nodes giving the stalks a bulbous appearance.

Diagnosis

In the field, the fine chlorotic striations on leaves are the diagnostic symptom but this symptom is not always clear. RT-PCR assays specific for SCSMaV have been developed and full genome sequencing has been used for genomic analysis (Choi *et al*., 1999; C. Ngo, personal communication; Thompson, 2001).

20. https://ictv.global/taxonomy, accessed December 2025.

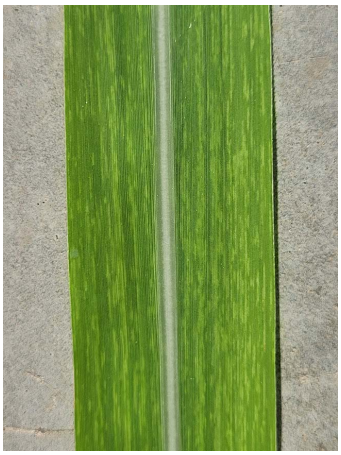


Figure 1. Short chlorotic striations on sugarcane leaves infected by sugarcane striate mosaic-associated virus (P. Rott).



Figure 2. Stalk striations associated with striate mosaic (N. Thompson).

Strains of the pathogen

Recent full genome sequence analysis of specimens from the Burdekin showed significant variation from the published sequences (C. Ngo, personal communication). Strain variation has not been investigated.

Transmission

Field observations suggest that striate mosaic is soil-borne and that SCSMaV probably infects plants through the agency of a soil-borne vector. No vector has been identified yet and no active spread from affected patches has been recorded.

All plants derived from infected planting material are infected (Hughes, 1961). There is no evidence that the disease establishes in the soil in previously unaffected fields when diseased planting material is planted in these fields. When the diseased plants are removed, the disease does not reoccur in these fields in the next crop. Experimentally, the disease has been transmitted by the Sein's needle-prick technique (Hughes, 1964) and by planting healthy cuttings in pots of soil from affected fields (C.C. Ryan, personal communication).

Host range

The disease has only been reported in sugarcane.

Epidemiology

Striate mosaic occurs in distinct patches within fields. The disease does not appear to spread significantly out of these patches and reappears whenever a susceptible variety is planted in the field. The patches can be up to one ha in area. The disease is associated with sandy or shallow soils in fields, which otherwise, have rich deep alluvial soils. The severely stunted plants in affected patches are in stark contrast to the rest of the fields, which are some of the highest yielding fields in Australia. Striate mosaic is usually not detected in plant crops but is first found in ratoon crops, except where diseased planting material has been used.

Economic importance

Striate mosaic is limited in distribution and, therefore, does not cause significant losses on an industry-wide basis. However, the disease can cause severe losses in individual fields (Figure 3). Severe stunting and death of plants in patches of up to one ha in area is not uncommon. Variety Q96, which was the major variety in the Burdekin district for many years, is highly susceptible to striate mosaic. The failure of patches, in otherwise highly productive fields, often leads to weed control problems and premature ploughing out of affected fields.

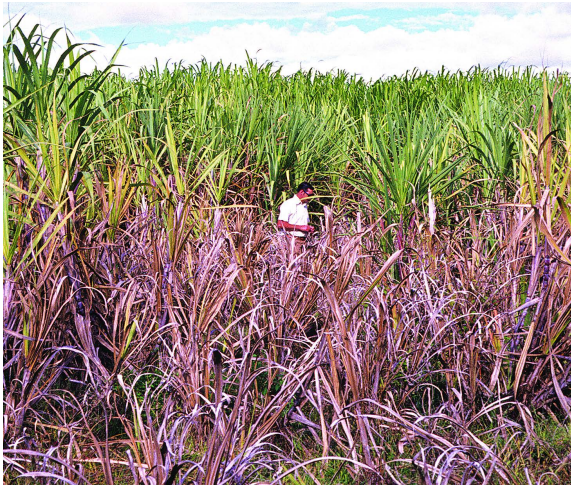

Figure 3. Sugarcane variety severely affected by striate mosaic (BSES).

Control

Varieties with resistance to striate mosaic have been used to control the disease. Varieties are rated for resistance by observing disease incidence when the varieties are planted in known infested fields.

When striate mosaic is found in a field, the affected area is recorded by the local Productivity Service staff and these records are used to advise growers to plant resistant varieties in these fields and to not use planting material from the field.

Methyl bromide fumigation of the soil in affected patches has eliminated the disease for at least one crop cycle but is not economically viable as a control method (C.C. Ryan, personal communication).

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Striation

Baoshan Chen, Ronghui Wen

Cause

Striation is caused by sugarcane striate virus or SStrV (species name = *Mastrevirus saccharumstriati*; family *Geminiviridae*).

Geographical distribution

China, Guadeloupe, India, Indonesia (Java), Iran, Papua New Guinea, Philippines, USA (Florida).

In the Western Hemisphere, SStrV has, however, only been found in germplasm collections and not in commercial fields.

Symptoms

SStrV is associated with yellow striations on the sugarcane leaves but numerous infected plants are asymptomatic (Figures 1 and 2). In China, less than 10% (44/531) samples of *S. officinarum* and *Saccharum* spp. local varieties collected in fields or germplasm collections exhibited yellow striation symptoms. Thirty-six (82%) of the 44 symptomatic leaves tested positive for SStrV by PCR. Coinfections of SStrV and other viruses are common in the field, thus resulting in various striate-looking symptoms.

Diagnosis

SStrV possesses twinned virions and a genomic organization characteristic of mastreviruses (Figure 3) (Boukari *et al.*, 2017). The single-stranded circular DNA genome of SStrV has a size of about 2.7 kb. The virus can rapidly be detected with conventional



Figure 1. Leaf showing striation symptoms on the upper side of a leaf of *Saccharum officinarum* (P. Rott).



Figure 2. Leaf showing striation symptoms on the lower side of a leaf of *Saccharum officinarum* (P. Rott).

PCR assays using leaf extracted DNA as a template. Virus-specific primers sets SCP and SRep[21] are based on the virus CP and Rep genes, respectively. PCR assays with these primers are commonly used in China for field detection of SStrV (Lin *et al.*, 2021).

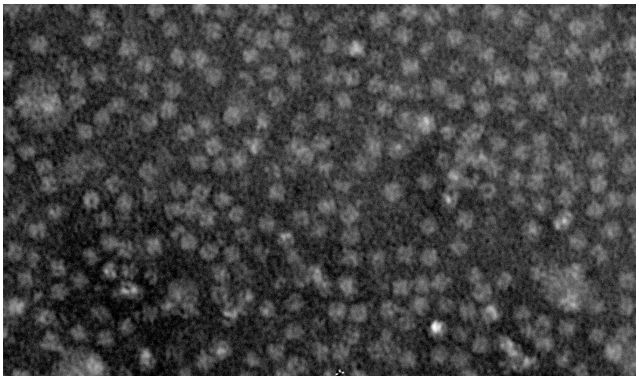

Figure 3. Particles of sugarcane striate virus negatively stained with uranyl acetate (Y. Lin).

The circular DNA of the SStrV genome can be efficiently amplified using the rolling circle amplification (RCA) method for virus detection and the Phi29 DNA polymerase. RCA products are usually restricted with enzyme *Bam*HI because the SStrV genome has only one *Bam*HI restriction site. Consequently, a single band of similar size to the viral genome is visualized when the restriction product is subjected to 1% agarose gel electrophoresis (Boukari *et al.*, 2017; Lin *et al.*, 2021). Cloning and sequencing this single band will yield the full-length sequence of the virus genome. While RCA has high efficiency in amplifying full-length virus genomes, it does not offer cost advantages compared to conventional PCR methods, making it unsuitable for large-scale detection of SStrV.

Polyclonal antibodies against the coat protein of SStrV were also produced and a specific 28.3 kDa protein band was observed by Western blot analysis of SStrV-infected samples (Lin *et al.*, 2021). However, no rapid immunological detection assay has been developed so far for the causal agent of striation.

Strains of the pathogen

One hundred seventy-five complete genome sequences of SStrV are currently available. According to the strain demarcation threshold of 94% for the genus *Mastrevirus* (https://ictv.global), these sequences can be distributed into four different strains: A (n = 165), B (n = 3), C (n = 3), and D (n = 4). Strain A isolates can further be divided into eight genetic subgroups (I – VIII).

SStrV has been found in the Eastern and the Western Hemispheres (Boukari *et al.*, 2017 and 2020; Filloux *et al.*, 2018; Lin *et al.*, 2021). Nevertheless, the four strains of the virus were only detected in germplasm collections in the Western Hemisphere (USA/Florida and Guadeloupe). Strain A is widespread in China in germplasm collections and in commercial fields, but strain B has only been found in germplasm materials.

SStrV strain A was dominant in *S. officinarum* and *Saccharum* spp. local varieties in China, and infected plants showed either no or only very mild symptoms. Infection of

21. SCP-F: 5′-CGCTCGGATAAGAGACCTAATC-3′ and SCP-R: 5′-CTCTTGAAATACAATCTGGTAGTGC-3′; SRep-F: 5′-CTGTTTCCGATAGTGGAAGAC-3′ and SRep-R: 5′-CGTGATCCTGCTTTCTTTGAT-3′.

S. officinarum with strain B was associated with yellow striation-like symptoms on the leaves. No obvious relationship was found between the genetic variation of SStrV and its host species, as SStrV was found in almost all *Saccharum* species collected in China, including *S. officinarum*, *S. robustum*, *S. sinense*, *S. spontaneum*, *Saccharum* spp. local varieties, and *Saccharum* spp. hybrids, as well as in two closely related *Erianthus* species, *E. arundinaceus,* and *E. rufipilus* (Lin *et al*., 2021). However, strains C and D from the United States were only found in *S. barberi* and *S. spontaneum*, respectively (Boukari *et al*., 2017), and most isolates of strain A from Guadeloupe formed subgroup VII (Lin *et al*., 2021). This suggested a relationship between SStrV genetic variation and geographical origin of infected plants. Isolates of strains A and B were not only found in sugarcane germplasm in China but also in sugarcane germplasm in the United States, thus most likely sharing a common geographical origin.

Transmission

SStrV is transmitted through infected seed cane (stem cuttings) and most likely by an insect vector that remains to be identified. Sugarcane geminiviruses are mainly transmitted by phloem-feeding insects such as leafhoppers. Mealybugs feeding on diseased sugarcane plants were found infected by SStrV but their role in transmission of this virus has not been verified yet. Mastreviruses are normally not transmissible by mechanical means.

Host range

Although SStrV includes at least four genetic strains, strain A has a much broader host range and is currently more widespread than strains B, C, and D. Isolates of SStrV strain A were detected in eight different host plants, including four *Saccharum* species (*S. officinarum*, *S. robustum*, *S. sinense*, *S. spontaneum*), *Saccharum* spp. local varieties in China, *Saccharum* spp. hybrids, *E. arundinaceus,* and *E. rufipilus*. Strain B was only found in *S. officinarum* and *Saccharum* spp., whereas strains C and D infected *S. barberi* and *S. spontaneum*, respectively. No obvious relationship was identified between the genetic variants of SStrV in China and their respective hosts.

Epidemiology

SStrV was detected in 439 of 1,750 sugarcane leaf samples collected from commercial/farmer fields of the provinces of Guangxi, Guangdong, Yunnan, Hainan, and Fujian in China, with an average incidence of 25.1%. The highest incidence (90.3%) was observed in Guangdong (93/103 positive samples), whereas the lowest incidence (9.9%) was found in Hainan (10/101 positive samples). This high prevalence of the virus in China suggests that sugarcane growing conditions in this country are highly favorable for virus infection and spread. Optimal plant stage and weather conditions for disease development remain to be determined. Absence of the virus in commercial fields in the Western Hemisphere is evidence for absence of the insect vector in this part of the world.

Economic importance

Stalk length, diameter, weight, and fiber content were significantly higher for virus-free versus SStrV-infected plants of variety Badila. Additionally, SStrV infection had a negative effect on germination of seed cane and growth of young sugarcane shoots.

Control

Application of strict quarantine measures is recommended for movement of germplasm in order to avoid further spread of the virus. Healthy seed cane produced in a quality seed farm should be used to establish new sugarcane crops and reduce virus inoculum and spread. Meristem tissue culture in combination with antiviral chemical agents could be employed to develop virus-free planting material (Balamuralikrishnan *et al*., 2002; Cha-um *et al*., 2006; Cheong *et al*., 2012; Ramgareeb *et al*., 2010).

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Yellow leaf

Philippe Rott, Jean Heinrich Daugrois

This chapter has been updated and rewritten based on the original chapter entitled "Yellow leaf syndrome" and authored by Ben E. Lockhart and C. Pieter R. Cronjé in 2000.

Cause

Sugarcane yellow leaf virus or SCYLV (species name = *Polerovirus SCYLV*, family *Solemoviridae*) (Figure 1).

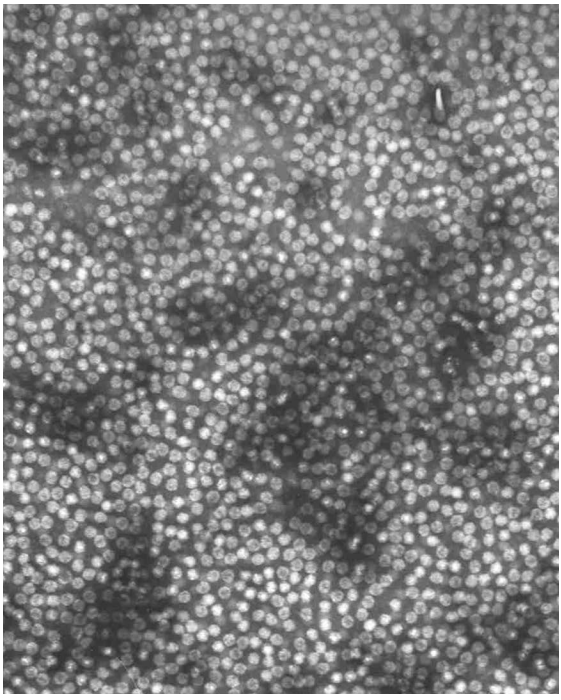

Figure 1. High concentration of particles (24–29 nm) of sugarcane yellow leaf virus observed by electron microscopy after purification from a sugarcane stalk (M. Chatenet).

Two pathogens were associated in the 1990s with symptoms of leaf yellowing known as yellow leaf syndrome or YLS (Lockhart and Cronjé, 2000): sugarcane yellow leaf virus (SCYLV) and sugarcane yellows phytoplasma (SCYP). Later on, the name yellow leaf was attributed to the disease caused by SCYLV and the name leaf yellows was given to the disease caused by SCYP (see book section on diseases caused by bacteria, p. 80). Unusual yellowing of sugarcane leaves called "yellow wilt" in Eastern Africa in the 1960s is considered to be the first report of the disease caused by SCYLV (Ricaud, 1968).

Geographical distribution

Argentina, Australia, Barbados, Brazil, China, Colombia, Cuba, Dominican Republic, Ecuador, El Salvador, Eswatini, Guadeloupe, Guatemala, Hawaii, India, Indonesia, Iran, Jamaica, Japan, Kenya, Malawi, Malaysia, Martinique, Mauritius, Mexico, Morocco, Mozambique, Nicaragua, Papua New Guinea, Peru, Philippines, Réunion, Senegal, South Africa, South Korea, Sri Lanka, Taiwan, Thailand, Tunisia, Uganda, USA (Florida, Louisiana), Venezuela, Zambia, Zimbabwe.

Symptoms

Symptoms usually appear in >7-month-old plants as an intense yellowing of the lower side of the leaf midrib while the color of the upper side remains white or greenish white (Figures 2 and 3). In some varieties, the upper side may also turn yellow or reddish-pink (Figure 4). When the disease progresses, V-shaped necrosis will start from the leaf tip and progress towards the leaf base while the yellow discoloration spreads laterally from the midrib into the lamina (Figure 5). Only lower leaves (starting from the third to fourth fully developed leaves from the top) are affected in some varieties while all the leaves are symptomatic in others. Additional symptoms include accumulation of sucrose in the midribs and shortening of the stalks' internodes. Sugarcane varieties can also be infected with the virus without exhibiting symptoms whose expression may vary according to environmental conditions (Rott *et al.*, 2008).

Diagnosis

Because leaf yellowing and necrosis can be caused by various biotic and abiotic stresses, symptoms are not reliable for identification of SCYLV (Lockhart and Cronjé, 2000). Furthermore, as symptom expression appears to depend on environmental factors and to vary according to sugarcane varieties, specific diagnostic assays need to be applied to detect SCYLV. Antibodies against SCYLV have been prepared and the virus can be identified by Double Antibody Sandwich-Enzyme Linked Immuno Sorbent Assay (DAS-ELISA) (Scagliusi and Lockhart, 2000) and tissue blot immuno-assay (TBIA) (Comstock *et al.*, 1998; Schenck *et al.*, 1997). SCYLV can also be detected by Reverse Transcription-Polymerase Chain Reaction (RT-PCR) assays and at least 13 diagnostic primer pairs were developed in various laboratories. However, some of these primer pairs may not detect all genetic variants of the virus (Rott *et al.*, 2023). Primer pair ScYLVf1/ScYLVr1 proved to match best with 109 complete genome-coding sequences of SCYLV, and these primers efficiently detected isolates representing the currently known genetic diversity of the virus. Reverse-transcription loop-mediated isothermal amplification (RT-LAMP) assays have also been developed for rapid identification of SCYLV in the laboratory and in the field (Holkar *et al.*, 2020). Recently, a RT-recombinase polymerase amplification (RT-RPA) assay (Srivastava *et al.*, 2025), a magnetic nanozyme-enhanced rapid immunoflow-through assay (Thangavelu *et al.*, 2024), and a novel system based on CRISPR-Cas12a (Wang *et al.*, 2024), became also available for detection of SCYLV.

Strains of the pathogen

Based on partial or full genome analyses, at least 10 different genotypes of SCYLV have been reported since 2006 (Abu Ahmad *et al.*, 2006; Lu *et al.*, 2021). In a phylogenetic tree constructed with entire genome-coding sequences, 108 isolates of the virus were recently distributed in three phylogenetic lineages called BRA, CUB, and REU



Figure 2. Mature sugarcane stalk with yellow leaf symptoms (P. Rott).



Figure 3. Lower leaf midrib showing intense yellow discoloration (P. Rott).



Figure 4. Asymptomatic sugarcane leaf (on the left) and two leaves (on the right) with reddish-pink discoloration of the upper side of the midrib (P. Rott).



Figure 5. Asymptomatic sugarcane stalk (on the left) and yellow leaf affected stalk (on the right) with V-shaped necrosis starting from the leaf tip and yellow discoloration of the lamina (P. Rott).

(Rott *et al.*, 2023). Based on its geographical distribution and host range, lineage BRA was the most widespread lineage and probably the most ancient one. Isolates belonging to this lineage have been found in *Miscanthus* sp., *Saccharum* hybrids, *Saccharum edule*, *Saccharum officinarum*, and *Sorghum bicolor*. Although less distributed, lineage CUB also occurs in several countries (China, Colombia, Cuba, Guatemala, India, USA, etc.) and lineage REU is predominant in the Mascarenes (Reunion Island and Mauritius). The significance of this genetic diversity remains to be investigated but variation in infection capacity and in virulence between genotypes of SCYLV has already been reported (Abu Ahmad *et al.*, 2007).

Transmission

In contrast to leaf yellowing caused by abiotic stresses or feeding insects, yellow leaf caused by SCYLV is transmissible by vegetative propagation of sugarcane (stalk cuttings). Long-distance spread of the virus occurs via infected planting material. Within and between fields, SCYLV is transmitted in a circulative, persistent, and non-propagative manner by at least five aphid species. These insects include *Melanaphis sacchari* (Figure 6), *M. sorghi* (Figure 7), *Rhopalosiphum maidis*, *R. rufiabdominalis*, and *Ceratovacuna lanigera* but not the yellow sugarcane aphid (*Sipha flava*) that is unable to transmit SCYLV (Holkar *et al.*, 2020; Pimenta *et al.*, 2025). Poleroviruses such as SCYLV are phloem-limited viruses that require extensive insect feeding on infected plants for efficient virus transmission.

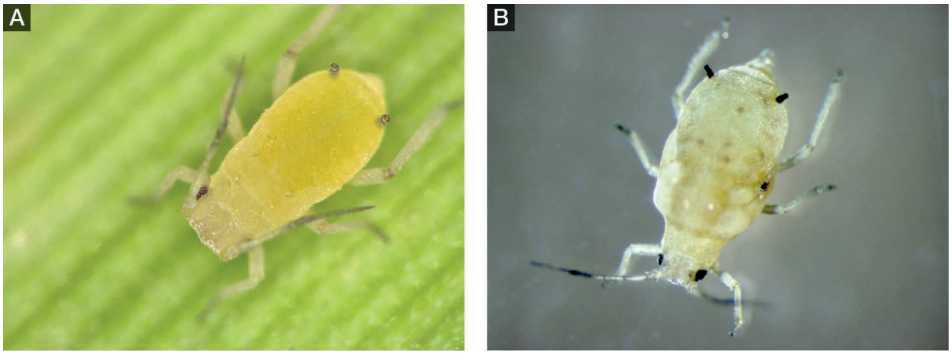

Figure 6. The aphid *Melanaphis sacchari* (nymph on the left and adult on the right) observed under a microscope (A: S. Sood; B: J.H. Daugrois).



Figure 7. Colony of *Melanaphis sorghi* on the underside of a leaf of *Sorghum almum* (P. Rott).

Host range

Modern hybrids of sugarcane are the main hosts of SCYLV but natural infections by this virus were reported in barley (*Hordeum vulgare*), Columbus grass (*Sorghum almum*), grain sorghum (*Sorghum bicolor*), and sugarcane related species such as *Erianthus* sp., *Miscanthus* sp., *Saccharum barberi*, *Saccharum edule*, *Saccharum robustum*, *Saccharum sinense* and *Saccharum spontaneum* (Boukari *et al.*, 2021; Comstock *et al.*, 2001). Using the aphid *Melanaphis sacchari*, the virus was successfully transmitted under controlled conditions to maize (*Zea mays*), oat (*Avena sativa*), rice (*Oryza sativa*), and wheat (Holkar *et al.*, 2020).

Epidemiology

Occurrence of alternative plant reservoirs and aphid vectors suggests that the virus can spread to healthy sugarcane from infected sugarcane or other plants by aphid transmission. Consequently, epidemiology and progress of yellow leaf is determined by the incidence of SCYLV in the sugarcane planting material, by the frequency of the virus in non-sugarcane hosts, and by the population dynamics of aphid vector species. In Guadeloupe, all plants of a field set up with healthy plants became infected by SCYLV within 20 weeks after planting whereas only 7–33% plants became infected in a field trial in Florida surveyed for three years (plant cane and two ratoon crops). Incidence of yellow leaf in India was positively correlated with minimum temperature and relative humidity but it was negatively correlated with rainfall (Saritha *et al.*, 2023). Disease severity was also negatively correlated with temperature, rainfall, and sunshine hours. Rainfall prevents effective plant colonization by winged aphids and subsequent sugarcane infection by the virus (Daugrois *et al.*, 2011).

Economic importance

Stalk diameter and length, sugar content, and the number of millable stalks can be affected in susceptible varieties. Although sugarcane is rarely killed by SCYLV, this virus can cause cane yield losses up to 50% in ratoon crops (Holkar *et al.*, 2020). In yield loss experiments conducted in Florida, yield reductions in ratoon crops varied from non-significant to 27% depending on sugarcane cultivar and soil type (Boukari *et al.*, 2019). Reduction in stalk sugar content of 11–14% were reported in Louisiana, Florida, and Reunion. The disease can also affect processing quality. In South Africa and Hawaii, the content of complex polysaccharides (gums) was increased in SCYLV-infected canes, thus interfering with sugar extraction (Lockhart and Cronje 2000). Degeneration in sugarcane varieties in India has been attributed to SCYLV and other sugarcane infecting viruses (Viswanathan, 2024).

Control

Planting resistant varieties and virus-free seed cane are the most effective means to control yellow leaf. Susceptible varieties exhibiting severe symptoms of the disease should be eliminated (Figures 8 and 9). Virus-free material can be obtained from virus-infected plants by meristem culture as well as osmo- and cryo-therapy (Cele *et al.*, 2024). Healthy planting material should be propagated in controlled nurseries before being used for the planting of commercial fields. Nurseries need to be checked for the absence or low frequency of SCYLV using DAS-ELISA or TBIA that allow the indexing of numerous samples in a reasonable amount of time. Infected alternative reservoir

plants may need to be removed during the peak season of aphid populations. Biological control of the aphids can contribute to limit the spread of the virus (Holkar *et al*., 2020). Molecular markers for resistance to SCYLV have been identified and can potentially be used to screen for resistance to yellow leaf in breeding programs. Genetic transformation or sugarcane genome editing might be used in the future to obtain resistant germplasm from susceptible but high performing varieties. The titer of SCYLV is lower in transgenic lines expressing the coat protein of SCYLV in comparison to non-transformed plants (Zhu *et al*., 2011).



Figure 8. Sugarcane variety severely affected by yellow leaf (P. Rott).



Figure 9. Sugarcane field severely affected by yellow leaf (P. Rott).

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Yellow mosaic

San-Ji Gao

Cause

Maize yellow mosaic virus or MaYMV (Species name = *Polerovirus MAYMV,* family *Solemoviridae*).

Geographical distribution

China, India, Nigeria.

MaYMV has been reported in the following countries: Brazil, Burkina Faso, China, Ecuador, Ethiopia, India, Kenya, Nigeria, Rwanda, South Korea, Tanzania, Uganda. Up to now, sugarcane infections by this virus have been identified in only three locations (China, India, and Nigeria).

Symptoms

Symptoms of yellow mosaic caused by MaYMV in maize include leaf mosaic and yellowing symptoms (Figure 1) (Chen *et al*., 2016). Some maize plants may show yellow stripes associated with leaf yellowing and chlorosis, which tends to begin from the tip and edges of the leaves before extending to the entire leaf blade (Lim *et al*., 2018b). In transmission experiments with aphids, leaf tip reddening and dwarfing has also been observed (Ohlson *et al*., 2024). Such symptoms were also observed in naturally infected maize leaves in China and East Africa (Massawe *et al*., 2018; *Shi et al*., 2022), as well as in weed hosts in China (Zhang *et al*., 2023).

Symptoms of yellow mosaic caused by MaYMV in sugarcane and itch grass (*Rottboellia cochinchinensis*) are mosaic and yellow stripes on the leaf blade (He *et al*., 2022; Nithya *et al*., 2021; Sun *et al*., 2019; Yahaya *et al*., 2017). In contrast to maize, mosaic symptoms in sugarcane look like light green "eye spot" lesions with a dark green center (Figures 2 and 3). These lesions vary from a few mm to 2–3 cm.

Diagnosis

MaYMV was first identified as a novel *Polerovirus* infecting maize in China, and it was discovered by deep sequencing of small RNAs (Chen *et al*., 2016). Subsequently, this virus was found in other plants and other countries using various RNA-sequencing platforms (Adams *et al*., 2017; Guo *et al*., 2022; Lim *et al*., 2018a; Welgemoed *et al*., 2020; Yahaya *et al*., 2017; Zhang *et al*., 2023). Relying solely on visual symptoms to distinguish MaYMV from other plant viruses is challenging due to the prevalence of mixed virus infections in either maize or sugarcane (Asiimwe *et al*., 2020; He *et al*., 2022).



Figure 1. Leaf yellowing and yellow stripes on maize leaves infected by maize yellow mosaic virus (A. Yahaya).



Figure 2. Symptoms of yellow mosaic on a sugarcane leaf (S.-J. Gao).



Figure 3. Eye spot shaped mosaic lesions on a sugarcane leaf affected by yellow mosaic (S.-J. Gao).

RT-PCR assays are commonly used for the detection of MaYMV using specific primers such as MaYMV-F and MaYMV-R[22] (Chen *et al.*, 2016). This primer set amplifies a 753-bp fragment spanning across ORF2 and ORF3 (partial ORF2-3) of the MaYMV genome sequence (Sun *et al.*, 2021). Another primer pair, MaYF and MaYR[23] target the coat protein (CP) of MaYMV and was used for simultaneous RT-PCR detection of four viruses associated with maize lethal necrosis (Li *et al.*, 2021). A one-step reverse-transcription loop-mediated isothermal amplification (RT-LAMP) assay was also developed for the detection of MaYMV[24] in maize (Li *et al.*, 2022).

22. MaYMV-F: 5′-TGCTCACATGTCTTCGAGAAGGAA-3′; MaYMV-R: 5′-TCGGAGATGAACTCCAGTATGATCT-3′.
23. MaYF: 5′-GACGCGCTCGCAATAATAAC-3′; MaYR: 5′-CGCAAAGACCCTCCTACCAC-3′.
24. The four primers of this RT-LAMP assay are: F3 (5′-CAAATCCGCGTCGAGGAC-3′) and B3 (5′-CCACTGAATGCTGCACACT-3′), FIP (F1c+F2) (5′-GCGAGCCTCGTCTGACTCCAttttTCGAAGACGAAGACGACCAA-3′) and BIP (B1c+B2) (5′-TTCAAAGGACTCTCTCACGGGCttttCTGATAAAGACGGCCCGAAG-3′).

Strains of the pathogen

Genome nucleotide identity among isolates of MaYMV varies from 91.1–99.9% (Sun *et al*., 2021). Based on partial or complete genome sequences, MaYMV isolates from different host plants and countries were separated into three phylogenic groups. These groups were closely associated with the geographical origin of the virus isolates i.e., Africa, China, and South America. Environmental adaptation is likely an important evolutionary force for shaping three geographical populations of MaYMV.

Transmission

MaYMV is most frequently transmitted through infected cuttings of sugarcane and/or different insect vectors (aphids). The virus has been experimentally transmitted to maize by three aphids i.e., the corn leaf aphid *Rhopalosiphum maidis*, the bird cherry-oat aphid *R. padi*, and the greenbug aphid *Schizaphis graminum* (Gonçalves *et al*., 2020; Ohlson *et al*., 2024; Stewart *et al*., 2020). Notably, the transmission efficiency of MaYLV by *S. graminum* was similar to *R. padi*, but was lower than *R. maidis* (Ohlson *et al*., 2024). Like other poleroviruses, MaYMV cannot be transmitted mechanically (Gonçalves *et al*., 2020). Aphid vectors are expected to spread MaYMV within sugarcane fields and between close fields whereas long-distance transmission occurs via cuttings taken from infected plants.

Host range

In addition to its first reported host (maize), MaYMV also naturally infects sugarcane (*Saccharum* spp.) (Figure 4), itch grass (*Rottboellia cochinchinensis*), millet (*Panicum miliaceum*) and sorghum (*Sorghum bicolor*) (Lim *et al*., 2018a; Nithya *et al*., 2021; Sun *et al*., 2019; Yahaya *et al*., 2017). Recently, the virus was also found in cultivated wheat (*Triticum aestivum*) and three different common weed species i.e., barnyard grass (*Echinochloa crusgalli*), green foxtail (*Setaria viridis*), and goosegrass (*Eleusine indica*) (Guo *et al*., 2022; Zhang *et al*., 2023). Experimental hosts include cereal crops such as oats (*Avena sativa*), foxtail millet (*Setaria italica*), barley (*Hordeum vulgare*),



Figure 4. Sugarcane plant naturally infected by sugarcane mosaic virus and maize yellow mosaic virus (A. Yahaya).

cultivated rye (*Secale cereale*) and some grass species such as annual ryegrass (*Lolium multiflorum*), switchgrass (*Panicum virgatum*), green foxtail (*Setaria viridis*), and sand love grass (*Eragrostis trichodes*) (Ohlson *et al*., 2024). Plants that were not infected by MaYMV in inoculation experiments include rice (*Oryza sativa*), Indian barnyard millet (*Echinochloa frumentacea*), smooth bromegrass (*Bromus inermis*), orchard grass (*Dactylis glomerata*), meadow fescue (*Festuca pratensis*), foxtail barley (*Hordeum jubatum*), Kentucky bluegrass (*Poa pratensis*) and narrow-leaf meadow grass (*Poa pratensis*) (Ohlson *et al*., 2024).

Epidemiology

Some agricultural production systems, such as mono-cropping with low genetic diversity and high crop density, appear to contribute to epidemics of yellow mosaic. Under field conditions, MaYMV is transmitted by several aphid species and annual or long-term climate change may modify the distribution areas of these insect vectors, and consequently the geographical distribution of an emerging virus like MaYMV. Susceptibility of host plants to viral infection can be increased by heat stress and rising temperatures (Regassa, 2021). Crops grown over large areas (such as sugarcane) and wild grasses are reservoirs of MaYMV that contribute to the spread of the virus and the development of yellow mosaic epidemics.

Economic importance

In maize, MaYMV leads to prominent leaf yellowing, tip reddening, and stunting, which can reduce plant growth and yields from 10–30% (Ohlson *et al*., 2024). However, no detailed cane yield and sugar losses caused by MaYMV have been reported in sugarcane so far.

Control

No large-scale means to control yellow mosaic have been implemented yet to control MaYMV in sugarcane. Nevertheless, important strategies to control and manage other sugarcane diseases are also valid for yellow mosaic, such as:

1. the use of certified disease-free healthy cuttings and resistant varieties;
2. the application of strict phytosanitary regulations and practices to prevent the entry/export of plant material infected by MaYMV or of the vector carrying the virus;
3. breaking the disease cycle and vectors build-up by crop rotation and diversification practices;
4. the use of chemical pesticides to control insect vectors indirectly through cutting treatment and foliar spray.

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VI

Sugarcane-associated viruses with unknown effect

Stephen Bolus, Jean Heinrich Daugrois

Causes

The following four viruses have been recently detected in sugarcane but their role in a disease is currently unknown:
1. Sugarcane umbra-like virus (SULV), family *Tombusviridae*.
2. Sugarcane white streak virus (SWSV; species name = *Mastrevirus saccharumalbusvenae*, family *Geminiviridae*).
3. Sugarcane totivirus 1 (STV 1), family *Orthototiviridae*.
4. Unidentified chrysovirus, family *Chrysoviridae*.

Geographical distribution

The original isolate of SULV was detected in a sugarcane accession from South Africa (Tahir *et al.*, 2021). Closely related isolates have been detected in plants from Ethiopia (Adams *et al.*, 2017) and Sudan (Daugrois *et al.*, 2021). Umbra-like virus sequences were also reported in sugarcane from Florida (Filloux *et al.*, 2018 ; Sood *et al.*, 2026).

SWSV was first identified and characterized in two quarantined sugarcane plants from Egypt using sequence-independent metagenomics (Candresse *et al.*, 2014), and in additional varieties from Chad and Sudan at Cirad's sugarcane quarantine facility in Montpellier, France. SWSV was also recently diagnosed in Côte d'Ivoire and Réunion (Ben Chéhida *et al.*, 2024; Kouman *et al.*, 2025).

A new totivirus (STV 1) and an unidentified chrysovirus have also been detected in sugarcane in India and Florida, respectively (Srivastava *et al.*, 2025; Filloux *et al.*, 2018).

Symptoms

It is unknown whether SULV alone, SWSV, STV 1, or the unidentified chrysovirus can cause symptoms in sugarcane. The original isolate of SULV was detected along with sugarcane yellow leaf virus and sugarcane mosaic virus in a sugarcane accession that

was displaying mosaic symptoms (Tahir *et al.*, 2021). Related virus isolates were also found associated with plants that were co-infected with other viruses and that exhibited disease symptoms (Adams *et al.*, 2017; Daugrois *et al.*, 2021).

The original isolate of SWSV was detected in plants infected by sugarcane streak Egypt virus and these plants exhibited white streak symptoms (Candresse *et al.*, 2014). However, these symptoms were later on not seen in sugarcane varieties infected with SWSV alone.

No leaf symptoms were associated either with sugarcane infected by STV1 or the unidentified chrysovirus. Members of the genera *Totivirus* and *Chrysovirus* are associated with plants, fungi, and insects, and it cannot be excluded that these two viruses were obtained from fungi colonizing sugarcane.

Diagnosis

No diagnostic molecular tests have been described for SULV and the unidentified chrysovirus. Currently, high-throughput sequencing or similar approaches have been successful at detecting these and related viruses (Adams *et al.*, 2017; Daugrois *et al.*, 2021; Filloux *et al.*, 2018; Tahir *et al.*, 2021). A PCR detection assay was developed for SWSV at Cirad's sugarcane quarantine facility in Montpellier, France, based on unpublished primers SWSV_561F and SWSV_1834R[25]. A one-step RT-PCR assay is also available for detection of STV 1 (Srivastava *et al.*, 2025).

Strains of the pathogen

No strains are officially recognized in the recently discovered groups of SULV and SWSV. Full genome analysis of SWSV isolates from several sugarcane growing regions revealed low genetic diversity within this virus (Daugrois *et al.*, unpublished data).

Transmission

No experimental data are available regarding the transmission of these viruses. Mastreviruses from the African continent are spread by *Cicadulina* species, and this could be also the case for SWSV.

Host range

Isolates closely related to SULV infect both sugarcane and maize (Adams *et al.*, 2017; Daugrois *et al.*, 2021). SULV-like sequences were also found in south of France in the wild grass *Trachynia distachya* (Daugrois *et al.*, 2021). Recently, SWSV was detected in *Megathyrsus maximus* in Reunion Island (Ben Chéhida *et al.*, 2024).

Epidemiology and economic importance

No information currently available.

Control

These viruses are not subjected to any field control method yet. In quarantine, SWSV-free plants can be produced by apical meristem tissue culture.

25. SWSV_561F: 5′-TTGTACCGTGGCCTGCTGA-3′; SWSV_1834R: 5′-GAACTGGTAAATCTACGTGGG-3′.

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VII

Diseases of uncertain etiology

Apex rot

Philippe Rott, Jean Heinrich Daugrois

Cause

Causal agent is unknown.

Geographical distribution

Barbados, Guadeloupe, Jamaica, Martinique, St Kitts and Nevis.

Symptoms

The initial symptom of apex rot is the drying of the spindle leaves (Rott and Feldmann, 1991). It is linked to a necrosis of the internal tissues above the meristem which can be seen in a longitudinal section of the upper part of the stalk (Figures 1 and 2).

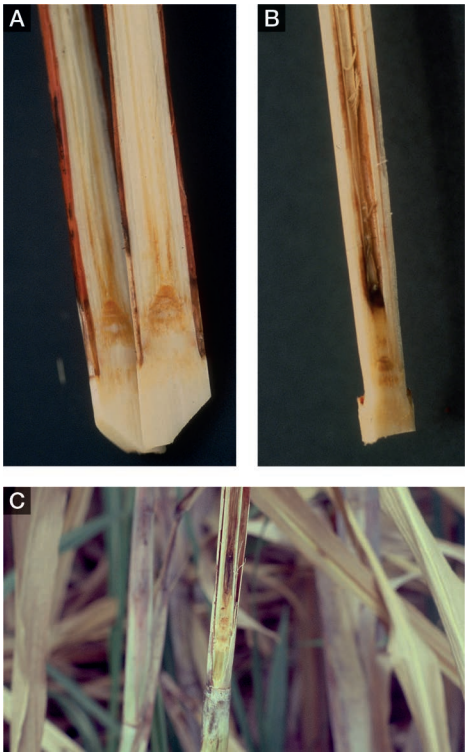

Figure 1. Necrosis of the internal tissues above meristem (A and B: P. Feldmann; C: J.H. Daugrois).



Figure 2. Necrosis of leaf tissue observed after removal of external leaves (P. Feldmann).



Figure 3. Mummified aspect of a stalk affected by apex rot (P. Rott).



Figure 4. Stalk affected by apex rot (J.H. Daugrois).





Figure 5. Stalks killed by apex rot (P. Feldmann).

Rotting progresses towards the base of the stalk, which dies and takes on a mummified aspect (Figure 3). The entire foliage can dry out and fall. No side shoots develop after the death of the apical meristem. Diseased stalks are randomly distributed within sugarcane stools and mixed with healthy stalks (Figures 4 and 5). Up to 15% mummified stalks of cultivar B69566 were observed in sugarcane fields in Guadeloupe.

Diagnosis

Attempts to isolate pathogenic bacteria or fungi from apex rot showing stalks have so far been unsuccessful.

Strains of the pathogen

No information is available.

Transmission

No information is available.

Host range

Sugarcane is the only reported host for apex rot.

Epidemiology

No information is available.

Economic importance

Apex rot might cause yield reduction in susceptible varieties such as B69566 in Guadeloupe, but experimental data are not available.

Control

Use of resistant varieties is the most effective means of control and, therefore, susceptible cultivars should be eliminated.

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Dwarf

Robert C. Magarey

Cause

Causal agent is unknown.

Geographical distribution

Australia.

The disease has only been observed in the Mackay district, Queensland (Bell, 1932).

Symptoms

White stripes on the leaves and the production of short, stiff, fan-like leaf growth are characteristic symptoms of dwarf (Figures 1 and 2). Leaves, particularly young ones, may exhibit numerous fine longitudinal white stripes on the leaf veins. Stripe dimensions vary considerably: width is often 1 mm or less but length varies from quite short to the whole length of the leaf blade. Broad diffuse chlorotic areas may develop toward the margins of the leaves (up to 5 mm wide and several cm long).



Figure 1. Leaves showing striping and "knife-cuts" along the margin (R. Magarey).



Figure 2. Stunted cane with stiff, fan-like leaf growth (R. Magarey).

The margins of leaves may be affected by the disease. Knife-cut type symptoms as well as papery and necrotic leaf margins may be common (Figure 1). Growth, as the name suggests, is considerably reduced with no commercial cane produced in diseased stools of susceptible varieties (Figure 3). Dwarfing varies with varietal susceptibility. In canes of intermediate resistance, leaf striping and knife-cut of leaf margins may be the dominant symptoms.

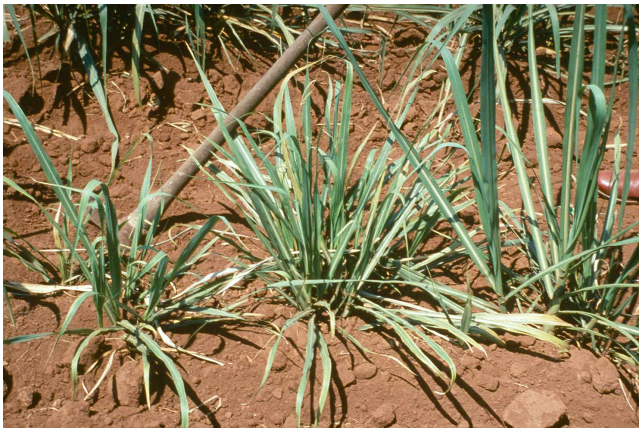

Figure 3. Dwarf affected stools (BSES).

Histological examination of internal leaf structures reveals the following: in white stripes, the vascular bundles may be considerably enlarged, very irregular in shape and attached to other bundles; the chlorophyll-bearing sheath may be entirely absent, and within the bundle there is distortion and changes in position of the bundle elements (Steindl, 1964).

Diagnosis

As the causal agent is unknown, there is no assay for the disease. Diagnosis relies on the recognition of symptoms.

Strains of the pathogen

No strains of the pathogen are known. There is no evidence to suggest variation in the causal agent.

Transmission

Transmission of the disease occurs readily in the field under conditions suited to the disease (wet soil conditions in summer). All attempts to transmit the disease artificially have failed including mechanical transmission and insect vectors.

Host range

The failure of transmission studies and the lack of an assay for diagnosis have meant that no information on host range is available. Symptoms have not been identified in any other hosts. However, the rapid appearance of the disease in susceptible varieties, when no symptoms have been observed in resistant canes, suggests the disease is present either in other hosts that are often present or in the soil.

Epidemiology

The disease is favored by wet soil conditions and warm soil temperatures. Little other epidemiological information is available.

Economic importance

Because the disease can lead to stunting of diseased stools, high disease incidence coupled with severe stunting can result in significant crop losses. However, the limited incidence of the disease, in terms of regions affected and crops diseased, means that the disease is rarely seen and of minor importance.

Control

The main forms of control have been roguing of diseased stools, the selection of disease-free planting material and the cultivation of resistant varieties. The disease is not eliminated by hot water or other curative treatments.

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Leaf galls (Pseudo-Fiji leaf galls)

Claude Ricaud, revised by Philippe Rott

Cause

Probably a toxicogenic effect of feeding by leafhopper (*Cicadulina mbila* Naudé suspected) (Sheffield, 1969).

Geographical distribution

Kenya, Madagascar, Malawi, Mozambique, Réunion, South Africa, Zimbabwe.

Symptoms

Three types of leaf galls have been reported on sugarcane in the literature: those found on leaf sheaths that bear great resemblance to stem galls (Wismer and Hughes, 1964); those due to boron deficiency (Martin, 1938), and those that resemble Fiji leaf galls and that have been observed in the above-mentioned countries. In the case of Pseudo-Fiji leaf galls, the galls are about 1 mm diameter on average but sometimes larger and develop along a vein that may be slightly swollen (Figure 1), on the underside of the leaf blades and the midrib, but rarely on the sheaths. The galls are usually round but may be oblong, extending over a few millimeters. They are greenish in color and slightly paler than normal leaf tissue. The galls may be adjacent to one another along a vein, like a chain of beads, or they may be separated at intervals of 1 to 2 cm.

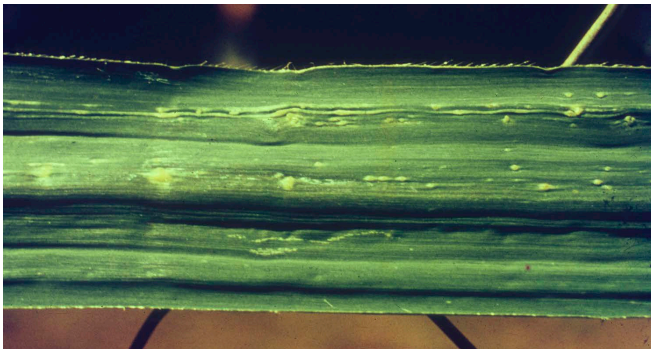

Figure 1. Small leaf galls (ISSCT).

Diagnosis

Histological examination helps to differentiate the leaf galls described above from those due to Fiji leaf gall disease. In the case of the virus infection, the galls result from a proliferation of the phloem. Pseudo-Fiji leaf galls are due to a proliferation of the epidermal cells or vascular bundle sheath cells and some adjoining mesophyll cells (Egan *et al.*, 1989).

Transmission

Leaf galls are not transmitted by sugarcane cuttings.

Host range

Leaf galls similar to those described above have also been seen on different species of the *Poaceae* family such as *Melinis minutiflora*, *Phragmites mauritianus* and rice (O*ryza sativa*). Various *Cicadulina* species have been reported to cause somewhat similar leaf galls in maize (Sheffield, 1969).

Epidemiology

Pseudo-Fiji leaf galls tend to disappear on new leaves developing during the wet season and are not seen on leaves produced as the stalks approach maturity.

Economic importance and control

Leaf galls are not of economic importance and do not warrant any control measure.

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Leaf yellowing and internode stunt

Uriel Green, Philippe Rott

Cause

The causal agent is unknown. Leaf yellowing and internode stunt is a condition that may represent a multifactor syndrome rather than a single-agent disease.

Geographical distribution

Jamaica.

Symptoms

The initial symptom of leaf yellowing and internode stunt is a progressive unilateral yellowing of the leaf blade that appears in 3–6-month-old sugarcane (Figure 1). Later on, this unilateral yellowing spreads to the entire leaf blade. The leaves that show the brightest yellow to orange discoloration are the bottom leaves (around F+6 leaf). In the younger leaves, a close-up observation reveals a very slight yellow-green discoloration in a small area of the leaf.

Mature stalks with normal stalk diameter have lower internodes with normal size but the upper stalk is formed by up to 20 stunted internodes (Figure 2). Additionally, the first 2–4 short internodes from the bottom exhibit side shoots (Figure 3). The upper leaves

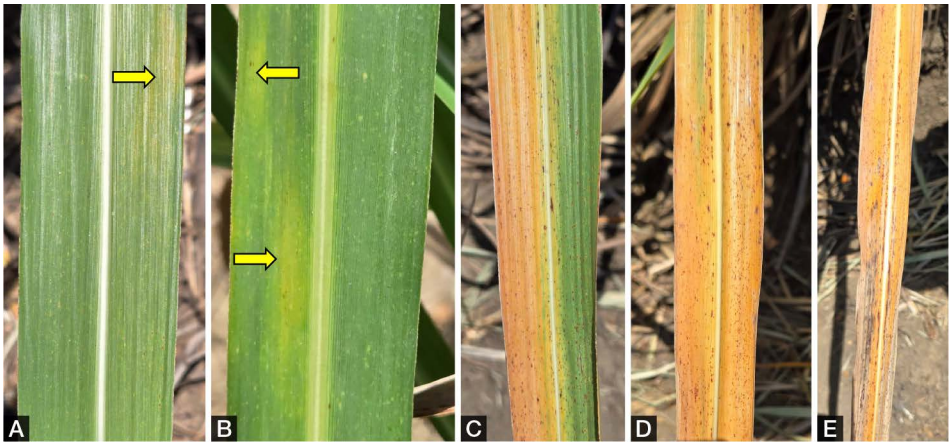

Figure 1. Progressive leaf discoloration of sugarcane affected by leaf yellowing and internode stunt. A. Small and faint discoloration on one side of the leaf blade (yellow arrow). B. Yellow-green discoloration on one side of the leaf blade (yellow arrows). C. Yellow-orange discoloration of one side of the leaf blade. D. Yellow-orange discoloration of both sides of the leaf blade. E. Yellow-orange discoloration and partial necrosis of the sugarcane leaf blade (P. Rott).

remain green or die. Stalks may also have a reduced diameter (up to 50%) or can have stunted internodes from the bottom to the top of the cane (Figure 4). Thin stalks are often flexible ("rubbery cane") and can be bent without breaking (Figure 5). In highly susceptible varieties, the stalk ultimately dies and mummifies (Figure 6). No internal symptoms are visible in stalks with leaf yellowing and stunted internodes, and stalk rotting is only seen in dead sugarcane. The root system of affected stools appears healthy.



Figure 2. Mature sugarcane stalk affected by leaf yellowing and internode stunt (P. Rott).



Figure 3. Side shoots on stunted internodes of a stalk affected by leaf yellowing and internode stunt (P. Rott).



Figure 4. Stalk with stunted internodes (P. Rott).



Figure 5. Thin and flexible sugarcane stalk affected by leaf yellowing and internode stunt (P. Rott).



Figure 6. Mummified aspect of a stalk affected by leaf yellowing and internode stunt (P. Rott).

Leaf-yellowing symptoms can overlap with nutrient disorders, water stress, herbicide injury, root impairment, leaf scald, yellow leaf, ratoon stunt, and other biotic or abiotic conditions.

Diagnosis

Attempts to isolate pathogenic bacteria or fungi from diseased stalks have so far been unsuccessful.

Strains of the pathogen

No information is available.

Transmission

The disease appears to be transmitted by infected cuttings. Transmission by other means (cutting implements, soil, water, etc.) is not excluded.

Host range

Sugarcane is the only reported host for leaf yellowing and internode stunt.

Epidemiology

Symptoms of the disease are often first seen at the edge of sugarcane fields but spread from areas inside fields has also been reported. No vector has been identified yet.

Economic importance

Leaf yellowing and internode stunt can cause significant yield reductions in cane tonnage, sugar and juice quality. Entire fields have been severely affected and destroyed by the disease, especially when grown with highly susceptible variety CR892023. Other symptomatic varieties were BJ78100, BJ7938, BJ9250, BJ9702, and BT80311. Several hundreds of ha were affected by the disease in Jamaica in 2024-2025. Indirect losses are caused by early replanting and the need to screen for resistance.

Control

The use of resistant or tolerant varieties, supported by clean planting material, is the most effective means of control and, therefore, susceptible varieties should be eliminated. Jamaica has implemented a policy to restrict movement of suspect seed cane, as it is the most practical control pathway currently available. In addition, small plots of tolerant varieties have been established under a Certified Seed cane nursery program, with plans for timely expansion, and distribution to growers under a structured variety replacement program.

Ramu streak

Robert C. Magarey, Sidney Suma, revised by Robert C. Magarey

Cause

Causal agent is unknown. Disease status is yet to be proven.

Geographical distribution

Australia, Papua New Guinea.

The disease has been observed in Papua New Guinea and on Saibai Island (Australian territory) in the Torres Strait.

Symptoms

Symptoms consist of thin yellow-green leaf streaks with a few similarities to chlorotic streak, but with a number of distinct differences (Magarey *et al*., 1995; Suma and Pais, 1996) (Figure 1). Streaks are 5–25 cm long, parallel to the midrib and up to 5 cm wide. Streaks may on occasions be short and relatively wide. Unlike chlorotic streak, Ramu streak rarely causes leaf necrosis, the streaks are more yellow-green and are prone to be wider (Figure 2). Reddening of the vascular bundles does not occur with Ramu streak.



Figure 1. Leaf streaks along the margin of an infected leaf (R. Magarey).



Figure 2. Irregular chlorotic leaf streaks without necrosis (R. Magarey).

Diagnosis

As the causal agent is unknown, diagnosis relies on the recognition of leaf symptoms.

Strains of the pathogen

There is no information on the existence of strains.

Transmission

Transmission of the disease has not been investigated. There is a considerable amount of basic research still to be undertaken. Indeed, there is a need to confirm a disease etiology.

Host range

The disease has been observed on noble canes (*Saccharum officinarum*), wild canes (*S. robustum, S. spontaneum), Saccharum edule* and interspecific sugarcane hybrids.

Epidemiology

Little information is available on spread of the disease.

Economic importance

Despite the common occurrence of this disease on the estates of Ramu Sugar at Gusap, Papua New Guinea, its economic impact has not been determined. It is considered a minor disease only. Some crops with abundant symptom expression were low yielding in 1994, but it is uncertain how much the condition contributed to these low yields.

Control

No specific control measures have been implemented.

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VIII

Minor diseases of uncertain etiology

Philippe Rott, Jack Comstock, revised by Philippe Rott

Note: Sembur and Sereh are currently considered as minor diseases but they were major diseases in Indonesia several decades to over a century ago.

Sembur

Cause: virus (putative).

Geographical distribution: Indonesia (Java).

Symptoms: elongate whitish to light yellow spots, 2–10 mm long by 1–3 mm wide, between vascular bundles (Figure 1).

Reference:

Editorial committee, 1964. Sembur. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 180–181.

Hes J.W., 1953. Sembur, leaf spot disease of sugar-cane. *Proceedings International Society of Sugar Cane Technologists Congress*, 8: 891–894.



Sereh

Cause: virus (putative).

Geographical distribution: Indonesia (Java).

Symptoms: short internodes, small cluster of shoots on affected stalks of variable height, stalks with abundant adventitious roots at nodes, and with severely affected plants made up entirely of shoots (Figures 2 and 3).

Figure 1. Leaf symptoms of sembur [Reproduced from Handojo H., 1982 (Penyakit Tebu di Indonesia/Sugarcane diseases in Indonesia), with the authorization of the Indonesian Sugar Research Institute].

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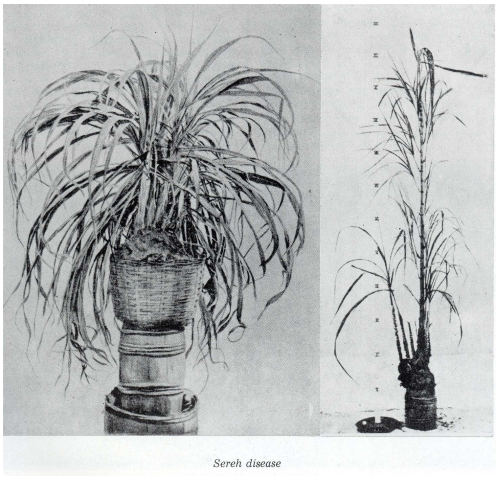

Figure 2. Sugarcane plants affected by sereh (Reproduced from the book "ISRI: An Historical Outline 1887-1987, 100 years", with the authorization of the Indonesian Sugar Research Institute).

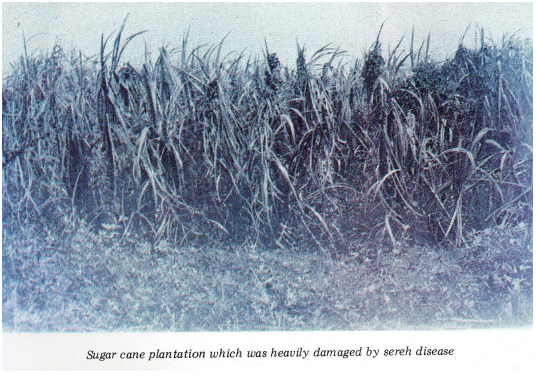

Figure 3. Sugarcane field affected by sereh (Reproduced from the book "ISRI: An Historical Outline 1887-1987, 100 years", with the authorization of the Indonesian Sugar Research Institute).

Spike

Cause: virus (putative).

Geographical distribution: India.

Symptoms: shortened leaves and internodes have a spike-like appearance on normal stalks, the leaves are dark green with blue tinge.

References:

Editorial committee, 1964. Spike. *In*: *Sugar-Cane Diseases of the World, Vol. 2*. C.G. Hughes, E.V. Abbott, C.A. Wismer (Eds). Amsterdam, The Netherlands, Elsevier Publishing Company, 190–191.

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IX

Plant-parasitic nematodes

Shamsul A. Bhuiyan

This chapter has been updated and rewritten based on the original chapter entitled "Nematodes" and authored by G. Stirling and B. Blair in 2000.

Plant-parasitic nematodes are small, multicellular animals (roundworms) found mainly in soil. More than 300 species live in the roots and rhizosphere of sugarcane. Often, more than five parasitic species can be present in the same sugarcane field. Nematodes are divided into two groups based on how they feed: ectoparasites that feed on the outside of root tissue and endoparasites that eat the inside of the roots (Bhuiyan *et al*., 2024). The main types of nematodes can be grouped as follows: (i) most prevalent and damaging species, such as *Pratylenchus zeae* and *P. brachyurus*; (ii) prevalent and highly pathogenic nematodes such as root-knot nematodes (*Meloidogyne javanica* and *M. incognita*), stubby root nematodes (*Paratrichodorus minor*), dagger nematodes (*Xiphinema* spp.), and needle nematodes (*Paralongidorus* spp.), all of which can cause severe crop damage; (iii) widespread but less pathogenic nematodes, including stunt nematodes (*Tylenchorhynchus* spp.), spiral nematodes (*Helicotylenchus*, *Scutellonema*, *Rotylenchus* spp.), lance nematodes (*Haplolaimus* spp.), ring nematodes (*Criconemella*, *Criconema*, *Hemicriconemoides*, *Ogma* spp.), sheath nematodes (*Hemicycliophora* spp.), and reniform nematodes (*Rotylenchulus parvus*).

Host range and geographical distribution

Parasitic nematodes associated with sugarcane have a wide range of hosts. Nematodes such as *Pratylenchus zeae*, *Rotylenchulus parvus*, and *Tylenchorhynchus annulatus* primarily infect grasses. In contrast, *Meloidogyne, Paratrichodorus*, and *Helicotylenchus* species possess a broader host range, affecting hundreds of different plant species.

Plant-parasitic nematodes are found in all regions where sugarcane is grown, with documentation at least from 36 countries (Spaull and Cadet, 1991; Jayakumar and Ganapathy, 2020). Although these nematodes are significant pathogens of sugarcane, their harmful effects often go unnoticed. This is largely due to the absence of visible symptoms aboveground and the microscopic size of the nematodes themselves. Furthermore, limited or insufficient survey data on the impact of these parasitic nematodes often leads to an underestimation of the damage they can cause. Many farmers have a limited understanding of nematode symptoms and may mistakenly attribute the damage to other issues such as nutrient deficiencies or soil toxicity (Bhuiyan *et al*., 2024).

Symptoms and diagnosis

Nematode infestation can show subtle and often unreliable symptoms. Common signs include stunted shoots, reduced tillering, slow growth, and uneven establishment (Figures 1 and 2). In very dry conditions, infested plants may have wilting and curled leaves, giving them a spiky look. Other factors, such as infections by pathogens and nutrient deficiencies, can produce similar symptoms (Bhuiyan *et al.*, 2024; see also "Leaf scald", p. 72).



Figure 1. Root-knot nematodes (*Meloidogyne javanica*) infested a plant crop in sandy soil near Bundaberg, Queensland, Australia (S.A. Bhuiyan).



Figure 2: *Pratylenchus zeae*, lesion nematode infestation in the plant crop has resulted in shorter plants on both sides of a tolerant variety in the middle row, in Harwood Mill area, NSW, Australia (S.A. Bhuiyan).

The symptoms caused by nematodes can vary depending on the specific species involved. The most prominent symptom of root-knot nematodes is the swelling and formation of galls on the tips of sett roots or young shoot roots (Figures 3 and 4). In older or mature plants, galls tend to be less prominent or smaller in size (Figure 5). In contrast, lesion nematodes create red, reddish-purple, or brown lesions on the roots (Figure 6).

Over time, these lesions progress and become necrotic, eventually turning a purple-black color that darkens further (Figure 7).

Symptoms caused by other types of nematodes tend to be more nonspecific and may include a lack of fine roots, discoloration, root lesions, swelling at the root tips, and stunted lateral roots. It is important to note that a variety of biotic factors (such as pathogenic fungi or insects) and abiotic stresses (such as soil toxicity or soil compaction) can produce similar damage, making accurate identification challenging.



Figure 3. Galling at the root tip caused by root-knot nematodes (*Meloidogyne javanica*) (S.A. Bhuiyan).

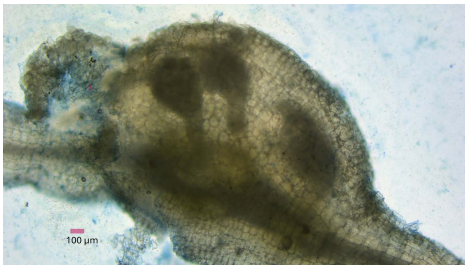

Figure 4. Root swelling caused by root-knot nematodes (*Meloidogyne* sp.), adult females inside (S.A. Bhuiyan).



Figure 5. *Meloidogyne* sp. infected roots of mature plants; note the smaller and inconspicuous galls (S.A. Bhuiyan).



Figure 6. Typical symptoms of root-lesion nematode (*Pratylenchus zeae*) infection in young roots (S.A. Bhuiyan).

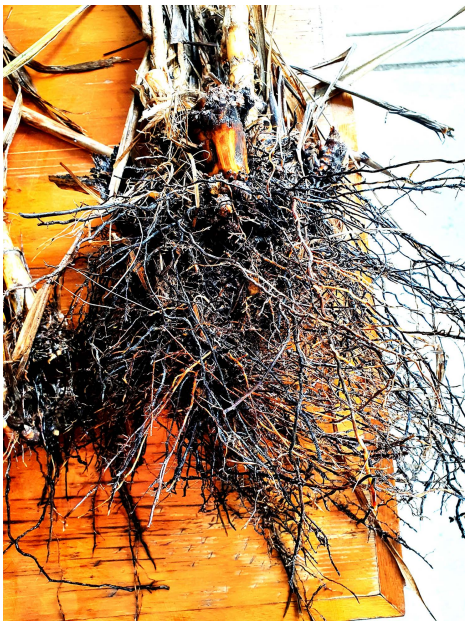

Figure 7. The advanced stage of root-lesion nematode (*Pratylenchus zeae*) infection, characterized by a dark to black-brown color (S.A. Bhuiyan).

Diagnosing nematode species is crucial for developing effective management strategies. Until recently, most nematode diagnoses relied on morphological identification, which involves categorizing them into species or genera (Martinha *et al.*, 2022). Light microscopy has been the primary method for this type of identification. Common morphological features used in the diagnosis of nematodes are size and shape of head, stylet, tail, and positions of the oesophageal gland and vulva (Figures 8 and 9). However, many plant-parasitic nematode genera show only slight morphological variations, and the features that differentiate species within a genus can be so subtle that even the most experienced nematologists may misidentify them (Coomans, 2002). In the past two decades, many nematologists have increasingly employed molecular techniques for identifying plant-parasitic nematodes (Berry *et al.*, 2008; Martinha *et al.*, 2022; Singh *et al.*, 2020).

Transmission

Nematodes can be transmitted through contaminated plant materials. The movement of agricultural machinery, such as tractors, planters, and harvesters, may spread nematodes and their eggs between fields. Additionally, nematodes can be transmitted through flash flooding or irrigation water. Because nematodes can survive in the soil for long periods,

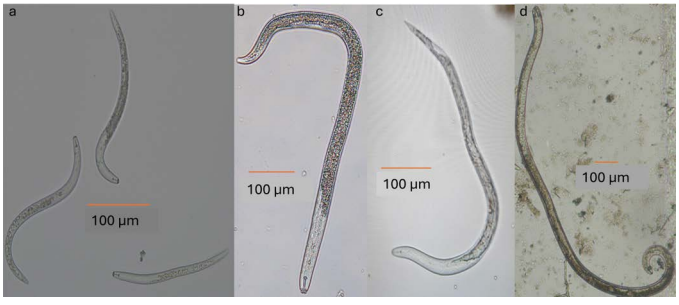

Figure 8. Root-lesion (*Pratylenchus zeae*) and root-knot (*Meloidogyne javanica*) nematodes. Root-lesion nematode: (a) juveniles, and (b) an adult female; root-knot nematode: (c) a juvenile, and (d) an adult male (S.A. Bhuiyan).

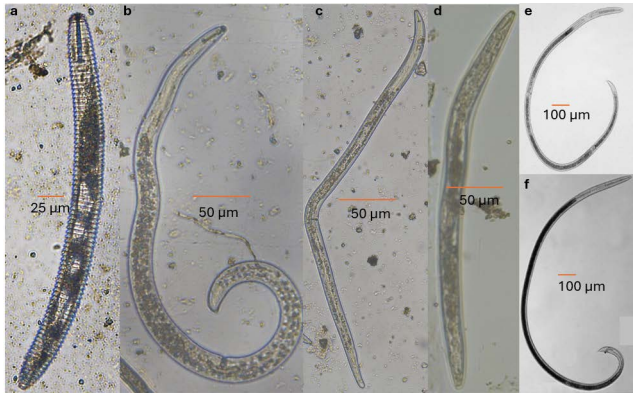

Figure 9. Minor nematodes: (a) ring (*Criconemella* sp.), (b) spiral (*Helicotylenchus* sp.), (c) stunt (*Tylenchorhynchus* sp.), (d) stubby (*Paratrichodorus* sp.), (e) dagger (*Xiphinema* sp., female), and (f) dagger (male) nematodes (S.A. Bhuiyan).

they are carried over from one crop to another. A fallow period of 2–3 years is needed to reduce the population of nematodes to very low levels (Pankhurst *et al*., 1999).

Epidemiology

The sugarcane root system is dynamic and constantly changing. After planting sugarcane stalks or billets, roots known as sett roots develop from the primordia at the nodes. These sett roots are then replaced by shoot roots, which develop from the root primordia of the young shoots. After ratooning, a majority of the root system dies off and is replaced by new roots that emerge from the new shoots (see "Sugarcane morphology, anatomy and physiology", p. 15; Smith *et al*., 2005). The development pattern of sugarcane roots significantly affects the population of plant-parasitic nematodes. Generally, nematode populations are low immediately after planting but gradually increase, reaching their highest levels between six to ten months post-planting. A similar trend has been observed in ratoon crops (Ramouthar and Bhuiyan, 2018). However, other factors, such as soil type, drought, and waterlogging, also play important roles in the distribution of nematode infestations (Maranhão *et al*., 2018). For example, *Pratylenchus zeae* nematodes are found in various soil types, but their populations are higher in clay soils compared to sandy soils (Ramouthar and Bhuiyan, 2018). In contrast, *Meloidogyne* species are more prevalent in sandy soils, with their numbers declining as the proportion of silt and clay in the soil increases (Blair *et al*., 1999). Additionally, nematode populations and species diversity were observed to increase over time, a trend likely driven by the monoculture nature of the sugarcane production system (Dengia *et al*., 2024).

Economic importance

Parasitic nematodes pose significant economic challenges in sugarcane production. Due to a lack of public awareness and often inconspicuous symptoms, the damage caused by these nematodes is frequently underestimated. However, extensive research conducted in Australia, South Africa, and other countries has shown that parasitic nematodes can lead to substantial yield losses in the sugar industry. In some countries, productivity losses attributed to plant-parasitic nematodes can reach up to 30% (Ramouthar and Bhuiyan, 2018). Generally, yield loss is more pronounced in plant crops compared to ratoon crops. In Australia, yield loss in plant crops was estimated at up to 23%, while the first ratoon experienced a loss of up to 12%. In Brazil, the losses were recorded at 29% for plant crops and 16% for the first ratoon. Burkina Faso reported a significant

67% loss in plant crops and only 9% in the first ratoon. In South Africa, both plant and ratoon crops faced a yield loss of 14% (Ramouthar and Bhuiyan, 2018).

Control

Controlling nematodes is essential when a single species dominates a farm and reaches or exceeds economic damage thresholds. Root-knot (*Meloidogyne javanica)* and root-lesion (*Pratylenchus zeae*) nematodes are the most destructive, with damage thresholds of 200–300 and 300–400 nematodes per 200 g dry soil, respectively. Management typically focuses on the predominant pathogenic nematode species in the sugarcane rhizosphere but may overlook the cumulative impact of other nematodes below threshold levels. Bhuiyan *et al.* (2020) developed a hazard index (HI) to assess the combined impact of nematodes based on their relative pathogenicity compared to *Meloidogyne javanica*. For instance, a root-lesion nematode accounts for 80% [hazard factor (HF) of 0.8] and a stunt nematode for 25% (HF of 0.25) of the damage caused by a root-knot nematode. The HF ranges from 0.05 for spiral, reniform and pin nematodes to 1 for the root-knot nematode (Bhuiyan *et al*., 2020 and 2024). The HI is estimated based on the sum of the nematode population size (number of nematodes in 200 g of soil) of all nematode types, each weighted by its respective HF value, using the following formula:

$$HI = \sum [\text{Number of nematodes per type} \times \text{type HF}]$$

This HI may help estimate potential yield losses from nematode infestations with an HI below 300 corresponding to a low risk with less than 5% yield loss, whereas an HI above 800 corresponds to a high risk with above 20% yield loss (Bhuiyan *et al*., 2020 and 2024).

In the USA and South Africa, several nematicides, such as fluopyram, ethoprop, carbofuran, oxamyl, and furfural aldehyde, are registered for nematode control in sugarcane (Crow, 2012; Odero *et al*., 2022). In Australia, only a single nematicide, Nimitz®, is registered for this purpose (Bhuiyan *et al*., 2024).

Other effective methods for reducing nematode populations include crop rotation with non-host crops, long-term bare fallow, minimum tillage, and the amendment of sugarcane soil with organic matter (Stirling *et al*., 2001 and 2005). While it is difficult to find nematode-resistant varieties, recent research in Australia has identified several commercial varieties and advanced clones that are resistant to root-knot nematodes (*Meloidogyne javanica*) (Croft *et al*., 2015). However, no commercial sugarcane variety has shown resistance to lesion nematodes. Tolerant varieties can greatly reduce nematode damage, resulting in higher yields than susceptible varieties. In Brazil and South Africa, yields from tolerant varieties exceeded those of susceptible ones by 40% to 124% (Matsuoka 1980; Spaull and Cadet, 2003). In Australia, some varieties are found to withstand high nematode infestations compared to susceptible ones (Figure 2).

Given the complex nature of parasitic nematode populations in soil, an Integrated Nematode Management (INM) strategy is more effective than relying on a single technique (Sikora *et al*., 2022). For managing nematodes in sugarcane, the judicious integration of two or more techniques is recommended. These techniques include improving soil organic matter, maintaining soil cover with a trash blanket, practicing minimum tillage, planting in low-nematode-infested soil, selecting resistant or tolerant varieties (including resistant rotation crops), maintaining long-term bare fallow, limiting the use of chemical nematicides, and monitoring nematode populations between crop cycles (Robinson, 2004; Stirling, 1991).

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X

Nutritional, environmental and chemical disorders

Barry J. Croft

Many disease symptoms can be mimicked by nutritional disorders, environmental effects, and phytotoxic chemicals such as herbicides (Figures 1 to 23). For example: the common herbicide paraquat can cause a lesion similar to ring spot disease when fine droplets drift on to sugarcane leaves (Figure 14); magnesium deficiency causes an orange to red-brown spot (orange freckle) on leaves similar to a range of fungal leaf spots (Figure 21); frost can cause a mosaic pattern not unlike sugarcane mosaic virus symptoms (Figures 9 and 10), and lightening can cause shredding of the leaves, death of the spindle and purple discoloration of the leaf blade (Figure 19). When confronted with symptoms of a possible disease, it is important to eliminate other possible causes. Nutrient analyses of the soil in the field and records of environmental conditions and chemical treatments applied to the field should be examined to try and identify other factors which may have caused or contributed to the symptoms which have been observed. These effects are outside the scope of this book, but more information can be obtained in Anderson and Bowen (1991), Calcino *et al*. (2022), Grascho and Taha (1972), Martin and Evans (1964), Martin *et al*. (1964), McCray *et al*. (2022).

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Figure 1. Leaf necrosis caused by fire ashes (P. Rott).



Figure 2. Sugarcane field affected by leaf necrosis caused by fire ashes (P. Rott).



Figure 3. Sugarcane field affected by banded chlorosis (cold injury) (P. Rott).



Figure 5. Leaf with banded chlorosis (cold injury) symptom (P. Rott).



Figure 4. Leaf with banded chlorosis and necrotic lesions caused by cold (P. Rott).



Figure 6. Leaves with boron deficiency symptoms (R.A. Bailey).



Figure 7. Leaves with copper deficiency symptoms (J.H. Meyer).



Figure 8. Sugarcane field affected by frost damage (P. Rott).

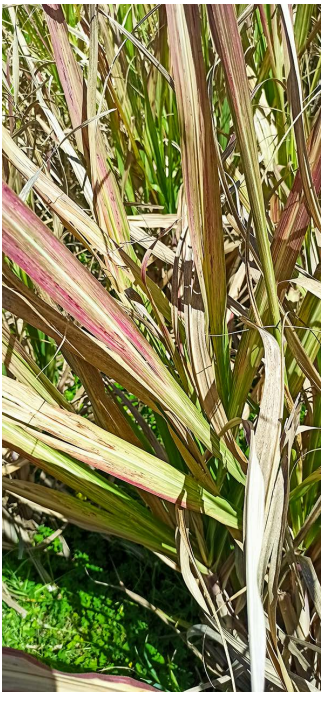

Figure 9. Leaf reddening of sugarcane affected by frost damage (J.H. Daugrois).



Figure 10. Leaf chlorosis and stripping of sugarcane affected by frost damage (J.H. Daugrois).



Figure 11. Herbicide (2,4-D) damage on sugarcane stalks (S. Matsuoka).



Figure 12. Field with herbicide (Glyphosate) damage (R. Viswanathan).



Figure 13. Sugarcane leaves showing herbicide (Glyphosate) damage (R. Viswanathan).



Figure 14. Leaves with herbicide (paraquat) injury (R.A. Bailey).



Figure 15. Elongated red lesions caused by an herbicide (Sulfentrazone) (J.-C. Girard).



Figure 16. Mosaic-like symptoms caused by whiteflies (P. Rott).



Figure 17. Elongated necrotic leaf lesions induced by an insect (P. Rott).



Figure 18. Leaves with iron deficiency symptoms (A: P. Rott; B: S. McFarlane).



Figure 19. Cane field affected by lightning damage (M. Mellin).



Figure 20. Stalk damage caused by lightning (M. Mellin).

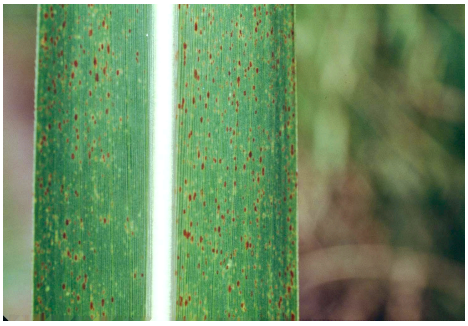

Figure 21. Leaf with magnesium deficiency symptoms (P. Rott).



Figure 22. Leaves with molybdenum deficiency symptoms (J.H. Meyer).



Figure 23. Regular white strips (albinism) on the leaf blades attributed to variegation (A. Mahoungou).

XI

Procedures for the safe movement of sugarcane germplasm

Roger A. Bailey, Jack C. Comstock, Barry J. Croft, A. Salem Saumtally, Philippe Rott, revised by Jean Heinrich Daugrois, Sharon A. McFarlane, Shamsul A. Bhuiyan, Romina Priscila Bertani and Nawshad Joomun

Many important pathogens of sugarcane can be transmitted through vegetative propagation materials. These include all viruses and phytoplasmas, most bacteria, a cercozoan and several fungal pathogens. Despite tissue cultured plantlets being a safer method of sugarcane germplasm exchange between countries or sugarcane-producing locations, stalk pieces (setts or cuttings) are still more commonly used for this purpose. Therefore, unless precautions are taken, sugarcane germplasm exchange provides a means of spreading diseases between countries (Croft *et al*., 1996).

Risks from germplasm exchange and basic control measures

The quarantining of imported germplasm has been widely practiced for many years. Prior to the development of immuno-based and molecular diagnostic assays, the main method for detecting diseases was based on symptom development in glasshouse-grown plants and microscopic detection. Before the causal agents of certain diseases were identified and prior to the development of appropriate diagnostic methods, the exchange of germplasm likely contributed to the worldwide spread of various pathogens. Those that cause non-specific symptoms, such as *Leifsonia xyli* subsp. *xyli* (ratoon stunt), or those that may remain latent in commercial sugarcane (*Saccharum* spp. hybrids) and wild germplasm, such as *Xanthomonas albilineans* (leaf scald), sugarcane yellow leaf virus (SCYLV), sugarcane bacilliform virus (SCBV) and sugarcane mild mosaic virus (SCMMV), pose a particular threat. The risks presented by germplasm exchange are now well appreciated and most countries importing germplasm apply strict quarantine procedures (Croft *et al*., 1996; Gillaspie, 1989; Guinet-Brial *et al*., 2013; Thompson and Wilson, 2020; van Antwerpen *et al*., 2006).

To mitigate the risk of transmitting pathogens through the exchange of germplasm, avoiding material from high-risk areas, where the health standard of the plant material is uncertain, becomes a crucial biosecurity measure for the importers.

Appropriate hot water treatment (HWT) procedures applied to sugarcane setts can eliminate all known sett-borne fungi, certain bacterial pathogens, parasitic nematodes, and a cercozoan (Table 1). While these treatments are still used routinely, efficacy in eliminating pathogens is improved when used in combination with meristem tissue culture procedure, and more recently developed protocols such as cryo/osmotherapy to eliminate viruses. These procedures reduce the risk of inadvertently spreading pathogens to importing regions and countries (Table 1).

Diagnosis

Enhanced biosecurity is provided through molecular and serological testing. Testing procedures are currently in place for the causal agents of gumming, leaf scald, ratoon stunt, leaf yellows, Fiji leaf gall, mosaic, streak mosaic, mild mosaic, sugarcane and maize streak, leaf fleck, yellow leaf and many other damaging viruses. Sharing of diagnostic protocols and materials among organizations that exchange germplasm is encouraged. Frequent reassessment of implemented molecular diagnostic tests is imperative to confirm their ongoing efficacy in detecting the target pathogens. It is essential to continually develop, evaluate, and adapt more accurate and high-throughput diagnostic procedures to enhance the quarantine process. In this respect, high throughput sequencing (HTS) holds much promise for pathogen detection and discovery. It has been successfully developed for detection of known and unknown pathogenic viruses in imported sugarcane (Fernandez *et al.*, 2020; Malapi-Whight *et al.*, 2021; Thompson and Wilson, 2020).

Since diagnostic tests are not available for all pathogens, and to reduce the risk of introducing rare or currently unknown pathogens, or emerging strains of known pathogens, the foundation of quarantine biosecurity relies on frequent inspections by experienced diagnosticians. This is also important because not all importers have access to modern diagnostic tools. Information regarding diagnostic methods is given in each disease chapter of this book.

Recommendations for the safe movement of germplasm

> The following is a summary of the recommendations published in detail by Frison and Putter (1993). This publication should be consulted for further information on quarantine security and for information on precautions against some specific diseases.

Exchange of setts – actions by the exporter

Although the importer primarily bears the responsibility for managing the risk associated with germplasm exchange, the exporter should take measures to ensure, as far as possible, that the material is disease and pest-free.

Setts for export should be sourced from plant stalks grown in a pest and disease-free environment, ensuring they are not exposed to hazardous diseases. These plants should be established using seed cane or tissue cultured plants and must undergo carefully controlled thermotherapy. The plants should be inspected regularly during growth to ensure they are free from symptoms of systemic diseases and the source plants should be indexed for pathogens.

Table 1. Therapy procedures described for important sugarcane disease-causing pathogens.

Type of pathogen	Name of pathogen (disease)	Procedure	Reference
Bacterium	*Leifsonia xyli* subsp. *xyli* (ratoon stunt or ratoon stunting disease)	Hot water treatment or HWT (50ºC/2–3h)	Davis and Bailey (2000), Andreato *et al*. (2022)
Bacterium	*Xanthomonas albilineans* (leaf scald)	Fresh running water soak (2 days) + HWT (50ºC/3 h)	Rott and Davis (2000)
Cercozoa	*Phytocercomonas venanatans* (chlorotic streak)	HWT (50ºC/30 min)	Egan (1989)
Fungus	*Sporisorium scitamineum* (smut)	HWT (50ºC/45 min) and fungicide treatments	Bailey (1979), Bhuiyan *et al*. (2015a and 2015b), James (1971)
Fungus	*Peronosclerospora sacchari* (downy mildew)	HWT (50ºC/2 h)	Hughes and Robinson (1961), Leu and Egan (1989)
Parenchyma-infecting virus	Sugarcane mosaic virus and sorghum mosaic virus (mosaic)	1/ Fresh water soak (24 h) + HWT (50ºC/2 h) + small meristem tip culture from younger germinated plants 2/ (1) + cryotherapy/osmotherapy (Potyvirus) 3/HWT (59°C/10 min) (Potyvirus)	1/ Ramgareeb *et al*. (2010) 2/ Cele *et al*. (2024), González-Arnao *et al*. (2020a and 2020b) 3/ Benda and Ricaud (1978)
Phloem-infecting virus	Sugarcane yellow leaf virus or SCYLV (yellow leaf), African streak virus group (streak)	1/ HWT (50ºC/3 h) + small meristem tip culture 2/ HWT (59°C/10 min) (streak) 3 /Meristem tissue culture followed by osmotherapy/cryotherapy (SCYLV) 4/ Leaf roll callus tissue culture (SCYLV)	1/ Chatenet *et al*. (2001), Ramgareeb *et al*. (2010) 2/ Benda and Ricaud (1978) 3/ Cele *et al*. (2024) 4/ Parmessur *et al*. (2002).
Phytoplasma	*Candidatus* Phytoplasma sacchari (grassy shoot/white leaf)	1/ Hot air treatment at 99% humidity (54°C/2.5 h); HWT (54°C/2 h) 2/ HWT (50°C/2 h) followed by soaking in tetracycline HCl antibiotic for 2 h 3/ Disease-free seed cane from tissue culture	1/ Frison and Putter (1993) 2/ Kaewmanee and Hanboonsong (2011) 3/ Roddee *et al*. (2017)
Phytoplasma	Sugarcane leaf yellows phytoplasma (leaf yellows)	Leaf roll callus tissue culture	Parmessur *et al*. (2002)
Parasitic nematodes	All known species	HWT (50ºC/3 h); avoid roots and soil	HWT criteria cited in the literature for nematodes are lower than the treatment provided here

Setts with up to three buds should be carefully stripped of all cane residue, thoroughly washed, hot water treated at 50°C for 30 to 60 min (short HWT), and treated with a registered fungicide and insecticide following appropriate protocols.

The name of the germplasm line should be clearly written directly on the rind of the setts, the extremities of which are then dipped in low melting point paraffin wax.

The setts should be wrapped in dry paper and protective packaging and dispatched by air freight or courier service.

Exchange of setts – actions by the importer

On receipt, parcels should be unpacked in a secure facility, the setts inspected for rotting and insect damage. The setts should be planted in sterilized potting medium in an insect-proof growth facility (glasshouse or screenhouse).

Ideally, imported sugarcane should be grown in an environment isolated from any sugarcane growing area (Figure 1). The French international sugarcane quarantine (Visacane) is located in the South of France where no sugarcane is cultivated (Guinet-Brial *et al.*, 2013).

When the quarantine is located close to sugarcane fields, the facilities should be built with the highest security standards such as those of the quarantine station of SASRI in South Africa (Bailey and Bechet, 1988).

Two growth cycles are recommended, setts being cut from the first planting, subjected to a long, cold soak HWT (24–48 h in cold running water followed by 3 h at 50°C;



Figure 1. Sugarcane grown in pots in a quarantine facility (Visacane) far away from sugarcane production fields in the south of France (J.H. Daugrois).

Figures 2 and 3), and re-planted in fresh sterilized medium. Note: the application of the 50°C treatment for 3 h may reduce germination of some wild germplasm (*Erianthus,* etc.) or sugarcane cultivars in certain locations. Therefore, the duration of the 50°C treatment should be tested before its application for new material or in a new location.

Once the second planting is established and provided the ratoon regrowth remains free of disease symptoms, the first planting should be destroyed. During both growth cycles, the plants should be inspected frequently by a skilled sugarcane disease diagnostician and any diagnostic tests available for virus and prokaryote pathogens should be applied. Provided the plants remain free of disease symptoms and the diagnostic tests are negative, at the end of the second cycle, setts can be cut, subjected to a long, cold soak HWT, and planted in post-quarantine isolation for further propagation.



Figure 2. Hot water tank with heat treated sugarcane cuttings at Visacane, Cirad's sugarcane quarantine in Montpellier, France (P. Rott).



Figure 3. Hot water treated one-bud cuttings of sugarcane (P. Rott).

Pre-export quarantine

A number of organizations have bilateral agreements for germplasm exchange. The main feature is that the exporter agrees to process material according to an accepted quarantine procedure, which includes diagnostic tests, before dispatch. The primary benefit is to improve quarantine security and to reduce the onus on the importer for a strict, lengthy quarantine process. The latter is of particular benefit when the exporter is better equipped technologically than the importing organization. As an example, Visacane, the quarantine unit of CIRAD in Montpellier, France, can be cited. This quarantine is located within the Plant Health Institute Montpellier research unit. Varieties being imported are tested and cleaned before being exported to tropical and subtropical countries or locations that do not have the laboratory facilities to test sugarcane for diseases or clean up imported material (Guinet-Brial *et al.*, 2013).

Tissue-cultured plants exchange

Increasing use is being made of tissue-cultured plants (*in vitro* cultures) as a safe method of exchanging germplasm, but it requires the exporting organization to have appropriate technological expertise and facility.

Setts are prepared from suitable source plants that are free of known diseases (selected to the same standards as for the exchange of setts). These are planted in sterilized medium in a containment facility. After several months' growth, shoot tip cultures are prepared from the apical bud and upper lateral buds, excised from young stalks under aseptic conditions (Figures 4 and 5). These are cultured on specific media under an appropriate light, nutrient, and temperature regime. When the plantlets originating from buds have started to produce roots, they are transferred to a semi-solid growth medium in transparent containers for further growth (Getnet 2017; Singh, 2003) (Figures 6 and 7).

The young plantlets can be exported, on semi-solid medium or on a decontaminated soilless substrate such as peat moss, coconut coir, perlite or vermiculite, after tillers have started to develop and when of a suitable size.

On receipt, the plantlets should be planted into sterilized potting medium in a containment facility and grown for at least one cycle, during which time appropriate diagnostic tests for pathogens should be applied. Once the plants are determined to be free from known pathogens, setts are prepared for further propagation outside quarantine.

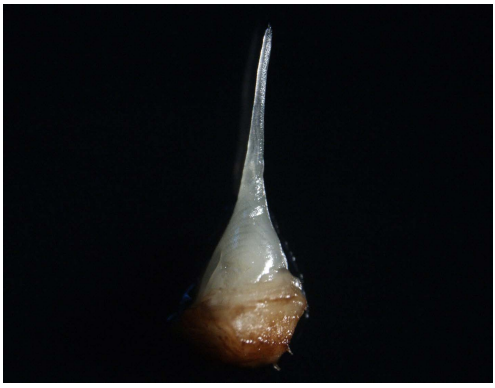

Figure 4. Excised meristematic dome (3 mm) of sugarcane with 1st, 2nd and 3rd leaf primordia (S. Ramgareeb).

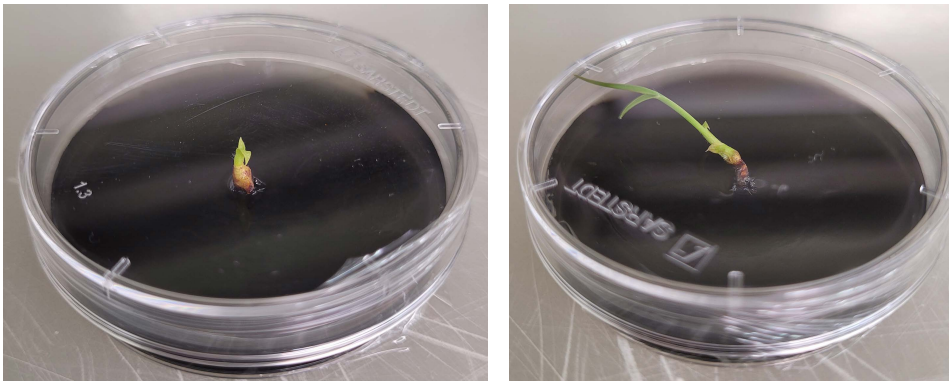

Figure 5. Young sugarcane plantlets obtained from meristem cultures on charcoal-containing medium (P. Rott).



Figure 6. Tissue-cultured sugarcane plantlets grown in transparent containers (P. Rott).



Figure 7. Tissue-cultured sugarcane plantlets grown in glass tubes (P. Rott).

Feedback of information

To encourage the improvement of standards, importing organizations should routinely inform exporters if any diseases are detected in exchanged material.

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XII

Common names of sugarcane diseases and their causal agent(s)

Updated by Philippe Rott

This is a revised version of the lists published by Philippe Rott, Roger A. Bailey, Jack C. Comstock, Barry J. Croft and A. Salem Saumtally in 2000 (A guide to sugarcane diseases, p. 317–322) and by the International Society for Plant Pathology at https://www.isppweb.org/names_sugarcane_common.asp.

Disease common name	**Pathogen name (Syn. = Synonymy*)**
Alternaria leaf blight	*Alternaria tenuissima*
Alternaria leaf spot	*Alternaria alternata*
Apex rot	Unknown
Apiospora mold	*Apiospora arundinis*
Arrow rot	*Fusarium* spp.
Australian basal stem, root and sheath rot	Unidentified fungus (basidiomycete)
Bacterial chlorotic streak	*Xanthomonas sacchari*
Bacterial leaf streak	*Burkholderia gladioli*
Bacterial leaf wilt	*Pantoea stewartii*
Bacterial mottle	*Dickeya poaceiphila* (Syn. *Pectobacterium chrysanthemi*)
Bacterial sun spot	*Pseudomonas* sp.
Baker's leaf spot	*Bakerophoma sacchari*
Banded sclerotial disease	*Rhizoctonia solani* (Syn. *Thanatephorus sasakii*, *T. cucumeris*)
Basal stem rot	*Agroathelia rolfsii*
Basal stem, root and sheath rot (see also Australian basal stem, root and sheath rot; Marasmius basal stem, root and sheath rot; Root and basal stem rot)	*Armilaria* sp., *Dictyophora* sp., *Mycelia sterilia*, *Olpidium* sp., *Rhizoctonia* sp.
Black leaf spot (Tar spot)	*Phyllachora sacchari*
Black rot	*Catunica adiposa* (Syn. *Ceratocystis adiposa*)
Black spot	*Cercospora acerosum***
Black stem rot	*Selenophoma sp.*

Disease common name	Pathogen name (Syn. = Synonymy*)
Black stripe	*Pseudocercospora atrofiliformis*
Brown rot	*Corticium* sp.
Brown rust (formerly Common rust)	*Puccinia melanocephala*
Brown spot	*Cercospora longipes*
Brown stripe	*Bipolaris stenospila*, *B. secalis*, *B. setariae*
Charcoal rot	*Macrophomina phaseolina*
Chlorotic streak	*Phytocercomonas venanatans*
Collar rot	*Hendersonia sacchari*
Covered smut	*Sphacelotheca macrospora*
Culm and midrib rot	*Apiospora camptospora* (Syn. *Papularia vinosa*)
Culmicolous smut (Smut)	*Sporisorium scitamineum* (Syn. *Ustilago scitaminea*)
Diplodia rot	*Diplodia* sp.
Downy mildew	*Peronosclerospora sacchari*, *P. philippinensis*, *P. spontanea*, undescribed *Peronosclerospora* species
Dry rot	*Lasiodiplodia theobromae* (Syn. *Physalospora rhodina*)
Dry top rot	*Ligniera vasculorum*
Dwarf	Unknown
Epicoccum leaf spot	*Epicoccum latusicollum*
Ergot	*Claviceps purpurea*, *C. pusilla*
Exserohilum leaf spot	*Exserohilum rostratum*
Eye spot	*Bipolaris sacchari*, *Curvularia spicifera*
False floral smut	*Claviceps* sp.
False red stripe	*Xanthomonas* sp.
Fiji leaf gall (formerly Fiji disease)	*Fijivirus fijiense* (Fiji disease virus or FDV)
Floral smut(s)	*Sporisorium cruentum*, *Sphacelotheca erianthi*, *Sporisorium schweinfurthianum*
Fusarium diseases: Pokkah boeng and Fusarium sett and stem rot	*Fusarium fujikuroi* (Syn. *Gibberella fujikuroi*, *G. subglutinans*) species complex, *Fusarium tricinctum*
Fusarium leaf spot	*Fusarium sacchari*
Fusarium root rot	*Fusarium* spp.
Fusarium sett and stem rot; see also Fusarium diseases	*Fusarium fujikuroi* (Syn. *Gibberella fujikuroi*, *G. subglutinans*) species complex, *Fusarium tricinctum*
Grassy shoot/White leaf	*Candidatus* Phytoplasma sacchari
Green grassy shoot	Phytoplasma
Gumming	*Xanthomonas axonopodis* pv. *vasculorum* and *X. vasicola* pv. *vasculorum*
Helminthosporium leaf spot(s)	*Helminthosporium purpurascens*, *H. portoricense*, *Exserohilum rostratum* (Syn. *Setosphaeria rostrata*), *Curvularia spicifera* (Syn. *Drechslera tetramera*)

Disease common name	Pathogen name (Syn. = Synonymy*)
Iliau (= Leaf sheath binding)	*Clypeoporthe iliau*
Inflorescence binding	*Balansia oryzae-sativae* (Syn. *Ephelis pallida*)
Knife-cut (see Pokkah boeng or Pokkah boeng twisted top)	*Fusarium fujikuroi* (Syn. *Gibberella fujikuroi*, *G. subglutinans*)
Leaf binding (= Myriogenospora leaf binding)	*Myriogenospora aciculispora*, *M. atramentosa*
Leaf blast	*Didymosphaeria taiwanensis*
Leaf blight	*Saccharicola taiwanensis* (Syn. *Leptosphaeria taiwanensis*)
Leaf fleck	*Badnavirus alphasacchari* (sugarcane bacilliform Guadeloupe A virus or SCBGAV), *Badnavirus betasacchari* (sugarcane bacilliform Guadeloupe D virus or SCBGDV), *Badnavirus gammasacchari* (sugarcane bacilliform IM virus or SCBIMV), *Badnavirus deltasacchari* (sugarcane bacilliform MO virus or SCBMOV)
Leaf galls (Pseudo-Fiji leaf galls)	Unknown (possible toxigenic effect of feeding by leafhopper)
Leaf scald	*Xanthomonas albilineans*
Leaf scorch	*Stagonospora sacchari*, *S. bicolor* (Syn. *Leptosphaeria bicolor*)
Leaf sheath binding (Iliau)	*Clypeoporthe iliau*
Leaf sooty mould (Sooty mould)	*Capnodium sp.*, *Fumago sacchari*
Leaf splitting	*Peronosclerospora miscanthi*, *P. northii*, *Peronosclerospora sp.*, *Mycosphaerella striatiformans*
Leaf spots	*Apiospora camptospora*, *Curvularia lunata* (Syn. *Cochliobolus lunatus*), *Diplodia* sp., *Colletotrichum falcatum* (Syn. *Glomerella tucumanensis*), *Epicoccum latusicollum*, *Fusarium sacchari*, *Pyricularia grisea* (Syn. *Magnaporthe grisea*), *Pyrenochaetopsis indica* (Syn. *Pyrenochaeta indica*)
Leaf yellowing and internode stunt	Unknown
Leaf yellows (formerly Yellow leaf syndrome)	Sugarcane leaf yellows phytoplasma (SCLYP)
Marasmius basal stem, root and sheath rot	*Marasmius sacchari*, *Collybiopsis stenophylla*
Marasmius sheath rot and shoot blight; root rot	*Collybiopsis stenophylla*, *Marasmius sacchari*, *Rhizoctonia* sp., unidentified oomycete
Midrib blotch	Unknown
Mild chlorotic streak	*Mastrevirus striatis* (maize striate mosaic virus or MSMV)
Mild mosaic	*Ampelovirus sacchari* (sugarcane mild mosaic virus or SCMMV)
Mosaic	*Potyvirus sacchari* (sugarcane mosaic virus or SCMV) and *Potyvirus sorghitessellati* (sorghum mosaic virus or SrMV)
Mottled stripe	*Herbaspirillum rubrisubalbicans*
Myriogenospora leaf binding (= Leaf binding)	*Myriogenospora aciculispora*, *M. atramentosa*
Orange rust	*Puccinia kuehnii*

Disease common name	Pathogen name (Syn. = Synonymy*)
Pachymetra root rot	*Pachymetra chaunorhiza*
Periconia leaf spot	*Periconia sacchari*
Pestalotia leaf spot	*Pestalotia fuscescens* var. *sacchari***
Phyllosticta leaf spot	*Phyllosticta hawaiiensis*, *Phyllosticta sp.*
Phytophthora sett rot (formerly Phytophthora rot of cuttings or Phytophthora seed piece rot)	*Phytophthora* spp., *P. megasperma*
Pineapple sett rot (formerly Pineapple disease)	*Ceratocystis paradoxa*, *Thielaviopsis ethacetica*
Pokkah boeng (including knife-cut symptoms) or Pokkah boeng twisted top; see also Fusarium diseases	*Fusarium fujikuroi* (Syn. *Gibberella fujikuroi*, *G. subglutinans*) species complex
Powdery mildew	*Blumeria graminis* (Syn. *Erysiphe graminis*)
Pseudo-Fiji (Leaf galls)	Unknown (possible toxigenic effect of feeding by leafhopper)
Purple spot (Red leaf spot)	*Dimeriella sacchari*
Pythium root rot	*Pythium arrhenomanes, Pythium* spp.
Ramu leaf scorch	Unknown
Ramu orange leaf	Unidentified fungus (exobasidiale)
Ramu streak	Unknown
Ramu stunt	*Mechlorovirus ramuense* (Ramu stunt virus or RmSV)
Ratoon stunt or Ratoon stunting	*Leifsonia xyli subsp. xyli* (Syn. *Clavibacter xyli* subsp. *xyli*)
Red leaf midrib	*Lasiodiplodia theobromae*
Red leaf mottle	*Pecluvirus arachidis* (peanut clump virus or PCV), *Pecluvirus indicum* (Indian peanut clump virus or IPCV)
Red leaf spot (Purple spot)	*Dimeriella sacchari*
Red line	*Fusarium* sp.
Red rot	*Colletotrichum falcatum* (Syn. *Glomerella tucumanensis*)
Red rot of the leaf sheath (now Sclerotium sheath rot)	*Agroathelia rolfsii* (Syn. *Corticium rolfsii*)
Red spot of the leaf sheath	*Passalora vaginae* (Syn. *Mycovellosiella vaginae*)
Red streak	*Pseudomonas syringae* pv. *syringae*
Red stripe (Top rot)	*Acidovorax avenae* subsp. *avenae*, *Acidovorax sacchari*
Rhizoctonia sheath and shoot rot	*Rhizoctonia solani*
Rind disease and sour rot	*Phaeocytostroma sacchari*
Ring mosaic	Unknown
Ring spot	*Epicoccum sorghinum* (Syn. *Leptosphaeria sacchari* and *Phoma sorghina*), *Curvularia ischaemi*, *C. guangxiensis*
Root and basal stem rot	*Xylaria warburgii*, *X. arbuscula*
Root rots (see also Pachymetra root rot; Pythium root rot; Marasmius sheath rot and shoot blight, root rot)	*Leucoporus sacchari, Marasmius sacchari*, *Rhizoctonia* spp., unidentified oomycete

Disease common name	Pathogen name (Syn. = Synonymy*)
Rust (see Brown rust, Orange rust, and Tawny rust)	*Puccinia melanocephala*, *P. kuehnii*, *Macruropyxis fulva*
Schizophyllum rot	*Schizophyllum commune*
Sclerophthora stunt (formerly Sclerophthora disease)	*Sclerophthora macrospora*
Sclerotium sheath rot (formerly Red rot of the leaf sheath)	*Agroathelia rolfsii* (Syn. *Corticium rolfsii* and *Sclerotium rolfsii*)
Seedling foliage blights	*Alternaria alternata, Curvularia hawaiiensis* (Syn. *Cochliobolus hawaiiensis*), *Curvularia lunata* (Syn. *Cochliobolus lunatus*), *Curvularia senegalensis*, *Bipolaris sacchari*, *Exserohilum rostratum* (Syn. *Setosphaeria rostrata*)
Sembur	Unknown
Sereh	Unknown
Sett and seedling rots	*Agroathelia rolfsii* (Syn. *Corticium rolfsii* and *Sclerotium rolfsii*)
Sheath rot	*Cytospora sacchari*
Smut (Culmicolous smut)	*Sporisorium scitamineum* (Syn. *Ustilago scitaminea*)
Smuts (see Covered smut, Culmicolous smut, Floral smut, False floral smut)	*Sphacelotheca macrospora*, *Sporisorium scitamineum* (Syn. *Ustilago scitaminea*), *Sporisorium cruentum*, *Sphacelotheca erianthi*, *Sporisorium schweinfurthianum*, *Claviceps* sp.
Sooty mould (Leaf sooty mould)	*Capnodium sp.*, *Fumago sacchari*
Spike	Unknown
Spindle rot	Related to *Acidovorax avenae* subsp. *avenae*
Stalk bacterial soft rot	*Dickeya zeae*
Stellate crystal fungus	*Himanthia stellifera***
Stinking rot	*Pseudomonas desaiana***
Streak	*Mastrevirus saccharofficinari* (sugarcane streak virus or SSV), *Mastrevirus saccharumegyptense* (sugarcane streak Egypt virus or SSEV), *Mastrevirus saccharumbourbonense* (sugarcane streak Reunion virus or SSRV), *Mastrevirus storeyi* (maize streak virus or MSV), *Mastrevirus sacchari* (saccharum streak virus or SacSV), *Mastrevirus saccharumpallidi* (sugarcane chlorotic streak virus or SCSV).
Streak mosaic (formerly Mosaic)	*Poacevirus sacchari* (sugarcane streak mosaic virus or SCSMV)
Striate mosaic	*Sustrivirus sacchari* (sugarcane striate mosaic-associated virus or SCSMaV)
Striation	*Mastrevirus saccharumstriati* (sugarcane striate virus or SStrV)
Tar spot (Black leaf spot)	*Phyllachora sacchari*
Target blotch	*Helminthosporium* sp., *Curvularia spicifera* (Syn. *Helminthosporium spiciferum*)
Tawny rust	*Macruropyxis fulva*
Top rot (Red stripe)	*Acidovorax avenae* subsp. *avenae*

Disease common name	Pathogen name (Syn. = Synonymy*)
Twisted leaf	*Epicoccum sorghinum* (Syn. *Leptosphaeria sacchari* and *Phoma sorghina*), *Phoma* sp.
Veneer blotch	*Deightoniella papuana*
White leaf/Grassy shoot	*Candidatus* Phytoplasma sacchari
White rash (White speck)	*Elsinoe sacchari*
White speck (White rash)	*Elsinoe sacchari*
White streak	*Mastrevirus saccharumalbusvenae* (sugarcane white streak virus or SCWSV)
Wilt	*Fusarium sacchari*
Yellow leaf (formerly Yellow leaf syndrome)	*Polerovirus SCYLV* (sugarcane yellow leaf virus or SCYLV)
Yellow mosaic	*Polerovirus MAYMV* (maize yellow mosaic virus or MAYMV)
Yellow spot	*Passalora koepkei* (Syn. *Mycovellosiella koepkei*)
White leaf	*Ca*. Phytoplasma sacchari
Zonate foot rot	*Fomes* sp.
Zonate leaf spot	*Gloeocercospora sorghi*

*Synonymy are pathogen names cited in Rott *et al*. (2000).
**Currently invalid or not recognized name.

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For any pathogen (and pest): EPPO Global Database (https://gd.eppo.int/).

For bacterial species names: List of Prokaryotic names with Standing in Nomenclature or LPSN (https://lpsn.dsmz.de/).

For fungal species names: Index Fungorum (https://www.indexfungorum.org/names/names.asp).

For virus species names: International Committee on Taxonomy of Viruses (ICTV) Taxonomy browser (https://ictv.global/taxonomy) and EPPO Global Database (https://gd.eppo.int/taxon/).

XIII

Sugarcane producing countries-locations and their diseases

Compiled by Philippe Rott and Frédéric Mahé

Africa

Algeria

Banded sclerotial disease.

Angola

Brown rust, Mosaic, Mottled stripe, Pokkah boeng, Red rot, Rind disease and sour rot, Ring spot.

Benin

Brown rust, Leaf scald, Mottled stripe, Pokkah boeng, Red rot, Red spot of the leaf sheath, Ring spot, Streak, Wilt.

Burkina Faso

Banded sclerotial disease, Fusarium sett and stem rot, Helminthosporium leaf spot, Leaf scald, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red leaf mottle, Red rot, Red spot of the leaf sheath, Ring spot, Sclerotium sheath rot, Smut, Wilt.

Burundi

Brown rust, Brown spot, Leaf fleck, Leaf scald, Mosaic, Mottled stripe, Pokkah boeng, Red rot, Ring spot, Smut, Yellow spot.

Cameroon

Brown rust, Brown spot, Dry rot, Eye spot, Leaf scald, Mosaic, Orange rust, Pokkah boeng, Ratoon stunt, Red stripe (Top rot), Ring spot, Smut, Streak, Yellow spot.

Cape Verde

Leaf fleck, Mosaic, Streak.

Central African Republic

Mottled stripe, Pokkah boeng, Red rot, Ring spot, Sclerotium sheath rot, Wilt.

Chad

Leaf scald, Mild mosaic, Pokkah boeng, Red leaf mottle, Red rot, Ring spot, Smut, Yellow spot.

Comoros

Ratoon stunt.

Congo

Brown rust, Leaf scald, Mosaic, Pineapple sett rot, Ratoon stunt, Red leaf mottle, Smut, Yellow spot.

Côte d'Ivoire

Brown spot, Brown stripe, Chlorotic streak, Fusarium sett and stem rot, Leaf fleck, Leaf scald, Mild mosaic, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Streak, Streak mosaic, Yellow spot.

Democratic Republic of the Congo

Brown spot, Eye spot, Leaf scald, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Ring spot, Smut.

Djibouti

Ratoon stunt.

Egypt

Chlorotic streak, Eye spot, Leaf yellows, Mild mosaic, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Rind disease and sour rot, Ring spot, Smut, Streak.

Eswatini (formerly Swaziland)

Brown rust, Brown spot, Gumming, Leaf scald, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Smut, Sooty mould, Tawny rust, Yellow leaf.

Ethiopia

Brown rust, Brown spot, Brown stripe, Leaf fleck, Mosaic, Pokkah boeng, Ratoon stunt, Red stripe (Top rot), Smut.

Gabon

Brown spot, Brown stripe, Leaf scald, Mosaic, Orange rust, Pokkah boeng, Red leaf mottle, Red rot, Red stripe (Top rot), Smut, Yellow spot.

Ghana

Gumming, Leaf scald, Mosaic, Pineapple sett rot, Pokkah boeng, Red rot, Red spot of the leaf sheath, Ring spot, Smut, Yellow spot.

Kenya

Brown rust, Brown spot, Chlorotic streak, Eye spot, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf scorch, Leaf yellows, Mosaic, Orange rust, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Ring spot, Smut, Streak, Yellow leaf, Yellow spot.

Madagascar

Banded sclerotial disease, Brown rust, Brown spot, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Gumming, Leaf fleck, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Yellow spot.

Madeira

Eye spot, Gumming, Leaf fleck, Pineapple sett rot, Pokkah boeng, Red rot, Streak.

Malawi

Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Gumming, Leaf fleck, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf yellows, Mild mosaic, Mosaic, Mottled stripe, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Rind disease and sour rot, Ring spot, Sheath rot, Smut, Streak, Yellow leaf.

Mali

Chlorotic streak, Pokkah boeng, Ratoon stunt, Red spot of the leaf sheath, Ring spot, Smut.

Mauritius

Brown rust, Brown spot, Chlorotic streak, Collar rot, Eye spot, Fusarium sett and stem rot, Gumming, Iliau, Leaf fleck, Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mild mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerophthora stunt, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Streak, Wilt, Yellow leaf, Yellow spot.

Morocco

Brown spot, Fusarium sett and stem rot, Leaf fleck, Leaf scald, Mosaic, Pokkah boeng, Red rot, Red stripe (Top rot), Smut, Yellow leaf.

Mozambique

Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Gumming, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf yellows, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sheath rot, Smut, Sooty mould, Streak, Tawny rust, Wilt, Yellow leaf.

Niger

Pokkah boeng, Red leaf mottle, Red rot, Red stripe (Top rot), Ring spot, Smut, Wilt.

Nigeria

Banded sclerotial disease, Black leaf spot (Tar spot), Eye spot, Leaf scald, Leaf scorch, Mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Sclerotium sheath rot, Smut, Streak, Yellow mosaic.

Réunion

Brown rust, Brown spot, Chlorotic streak, Eye spot, Gumming, Leaf fleck, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf yellows, Mild mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Streak, Wilt, Yellow leaf, Yellow spot.

Senegal

Brown stripe, Eye spot, Fusarium sett and stem rot, Pineapple sett rot, Pokkah boeng, Red leaf mottle, Red spot of the leaf sheath, Ring spot, Smut, Wilt, Yellow leaf.

Seychelles

Ratoon stunt.

Sierra Leone

Black leaf spot (Tar spot), Eye spot, Mosaic, Pokkah boeng, Ring spot.

Somalia

Ratoon stunt, Smut.

South Africa

Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Gumming, Inflorescence binding, Leaf fleck, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf scorch, Leaf yellows, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Mottled stripe, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerophthora stunt, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Streak, Target blotch, Tawny rust, Wilt, Yellow leaf, Yellow spot.

Sudan

Grassy shoot/White leaf, Mild mosaic, Pokkah boeng, Ratoon stunt, Red leaf mottle, Red rot, Ring spot, Smut, Streak, Wilt.

Tanzania

Brown rust, Brown spot, Eye spot, Leaf scald, Mosaic, Mottled stripe, Pokkah boeng, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sheath rot, Smut, Yellow spot.

Togo

Mottled stripe, Pokkah boeng, Red rot, Red spot of the leaf sheath, Ring spot, Wilt.

Tunisia

Yellow leaf.

Uganda

Brown rust, Brown spot, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Leaf yellows, Marasmius basal stem, root and sheath rot, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Streak, Wilt, Yellow leaf, Yellow spot.

Zambia

Brown rust, Brown stripe, Leaf scald, Leaf yellows, Mosaic, Phyllosticta leaf spot, Ratoon stunt, Ring spot, Smut, Streak mosaic, Yellow leaf.

Zimbabwe

Brown rust, Brown spot, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Gumming, Leaf galls (Pseudo-Fiji leaf galls), Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Mosaic, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Streak, Target blotch, Tawny rust, Wilt, Yellow leaf.

Asia

Afghanistan

Red rot, Red spot of the leaf sheath, Smut.

Andaman Islands

Eye spot, Mosaic, Pokkah boeng, Rind disease and sour rot, Ring spot.

Bangladesh

Banded sclerotial disease, Black leaf spot (Tar spot), Brown spot, Brown stripe, Collar rot, Eye spot, Grassy shoot/White leaf, Leaf scorch, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Ring spot, Sheath rot, Smut, Sooty mould, Streak mosaic, Wilt, Yellow spot.

Borneo

Black leaf spot (Tar spot), Sheath rot, Yellow spot.

Brunei

Black leaf spot (Tar spot), Yellow spot.

Cambodia

Black leaf spot (Tar spot), Chlorotic streak, Eye spot, Mosaic, Pokkah boeng, Red rot, Red stripe (Top rot), Ring spot, Smut, Yellow spot.

China

Alternaria leaf blight, Apiospora mold, Bacterial chlorotic streak, Bacterial leaf streak, Bacterial leaf wilt, Banded sclerotial disease, Basal stem rot, Black leaf spot (Tar spot), Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Grassy shoot/White leaf, Leaf blight, Leaf scald, Mild mosaic, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red leaf midrib, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Stalk bacterial soft rot, Streak mosaic, Striation, Target blotch, Twisted leaf, Wilt, Yellow leaf, Yellow mosaic, Yellow spot.

Hong Kong

Black leaf spot (Tar spot), Smut.

India

Banded sclerotial disease, Black leaf spot (Tar spot), Black rot, Brown rust, Brown spot, Brown stripe, Collar rot, Downy mildew, Dry rot, Ergot, Exserohilum leaf spot, Eye spot, False floral smut, Floral smuts, Fusarium sett and stem rot, Grassy shoot/White leaf, Gumming, Leaf blight, Leaf fleck, Leaf scald, Leaf scorch, Leaf splitting, Leaf yellows, Marasmius basal stem, root and sheath rot, Mosaic, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf mottle, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerophthora stunt, Sclerotium sheath rot, Seedling foliage blights, Sett and seedling rots, Sheath rot, Smut, Sooty mould, Spike, Spindle rot, Streak, Streak mosaic, Striation, Target blotch, Wilt, Yellow leaf, Yellow mosaic, Yellow spot.

Indonesia

Banded sclerotial disease, Black leaf spot (Tar spot), Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Downy mildew, Dry rot, Eye spot, Fiji leaf gall, Fusarium sett and stem rot, Leaf fleck, Leaf scald, Leaf scorch, Leaf splitting, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Root and basal stem rot, Sclerotium sheath rot, Sembur, Sereh, Sheath rot, Smut, Sooty mold, Streak mosaic, Striation, Target blotch, Veneer blotch, Yellow leaf, Yellow spot.

Japan

Banded sclerotial disease, Black leaf spot (Tar spot), Brown rust, Brown stripe, Downy mildew, Eye spot, Grassy shoot/White leaf, Leaf blight, Leaf scald, Leaf scorch, Marasmius basal stem, root and sheath rot, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red streak, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Streak mosaic, Target blotch, White speck (White rash), Yellow leaf, Yellow spot.

Kazakhstan

Smut.

Laos

Grassy shoot/White leaf, Mosaic.

Malaysia

Banded sclerotial disease, Black leaf spot (Tar spot), Brown stripe, Dry rot, Eye spot, Fiji leaf gall, Fusarium sett and stem rot, Grassy shoot/White leaf, Leaf scald, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, White speck (White rash), Wilt, Yellow leaf, Yellow spot.

Myanmar

Black leaf spot (Tar spot), Dry Rot, Grassy shoot/White leaf, Leaf scald, Marasmius basal stem, root and sheath rot, Mosaic, Orange rust, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Streak mosaic, Wilt, Yellow spot.

Nepal

Brown rust, Brown spot, Grassy shoot/White leaf, Mosaic, Phyllosticta leaf spot, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Ring spot, Smut, Wilt.

Pakistan

Black leaf spot (Tar spot), Brown rust, Brown spot, Brown stripe, Chlorotic streak, Collar rot, Eye spot, Fusarium sett and stem rot, Grassy shoot/White leaf, Leaf scald, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red stripe (Top rot), Ring spot, Sheath rot, Smut, Streak, Streak mosaic, Wilt, Yellow spot.

Philippines

Bacterial chlorotic streak, Banded sclerotial disease, Black leaf spot (Tar spot), Brown rust, Brown spot, Brown stripe, Chlorotic streak, Collar rot, Downy mildew, Dry Rot, Ergot, Eye spot, Fiji leaf gall, Fusarium sett and stem rot, Iliau, Leaf blight, Leaf fleck, Leaf scald, Leaf scorch, Leaf splitting, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mild mosaic, Mosaic, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Striation, Twisted leaf, Veneer blotch, White speck (White rash), Wilt, Yellow leaf, Yellow spot.

Singapore

Black leaf spot (Tar spot), Yellow spot.

South Korea

Yellow leaf.

Sri Lanka

Brown spot, Collar rot, Dry Rot, Eye spot, Grassy shoot/White leaf, Leaf scald, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Streak mosaic, Yellow leaf, Yellow spot.

Taiwan

Banded sclerotial disease, Black leaf spot (Tar spot), Black rot, Black stripe, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Covered smut, Downy mildew, Eye spot, Grassy shoot/White leaf, Leaf blast, Leaf blight, Leaf fleck, Leaf scald, Leaf scorch, Leaf splitting, Marasmius basal stem, root and sheath rot, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Root and basal stem rot, Sclerotium sheath rot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, White speck (White rash), Yellow leaf, Yellow spot.

Thailand

Banded sclerotial disease, Black leaf spot (Tar spot), Brown rust, Brown spot, Brown stripe, Chlorotic streak, Downy mildew, Eye spot, False floral smut, Fiji leaf gall, Fusarium sett and stem rot, Grassy shoot/White leaf, Green grassy shoot, Leaf fleck, Leaf scald, Leaf scorch, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Streak mosaic, Target blotch, White speck (White rash), Wilt, Yellow leaf, Yellow spot.

Turkmenistan

Smut.

Vietnam

Banded sclerotial disease, Brown rust, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Grassy shoot/White leaf, Green grassy shoot, Leaf scald, Leaf scorch, Leaf yellows, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Streak mosaic, White speck (White rash), Wilt, Yellow spot.

Australia and Oceania

American Samoa

Chlorotic streak, Orange rust.

Australia

Australian basal stem, root and sheath rot, Bacterial mottle, Banded sclerotial disease, Black leaf spot (Tar spot), Black rot, Brown rust, Brown stripe, Chlorotic streak, Dwarf, Ergot, Eye spot, False floral smut, Fiji leaf gall, Fusarium sett and stem rot, Iliau, Leaf fleck, Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Mottled stripe, Myriogenospora leaf binding, Orange rust, Pachymetra root rot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ramu streak, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerophthora stunt, Sclerotium sheath rot, Sett and seedling rots, Sheath rot, Smut, Sooty mould, Striate mosaic, Wilt, Yellow leaf, Yellow spot.

Federated States of Micronesia

White speck (White rash).

Fiji

Banded sclerotial disease, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Downy mildew, Eye spot, Fiji leaf gall, Fusarium sett and stem rot, Leaf scald, Leaf scorch, Leaf splitting, Marasmius basal stem, root and sheath rot, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Yellow spot.

French Polynesia

Leaf scald, Orange rust, Pineapple sett rot, Rind disease and sour rot, White speck (White rash).

Guam

Orange rust, Pokkah boeng, Red rot, Red stripe (Top rot), Sclerotium sheath rot, White speck (White rash).

Hawaii

Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Iliau, Leaf fleck, Leaf scald, Leaf splitting, Leaf yellows, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, White speck (White rash), Yellow leaf.

Marshall Islands

White speck (White rash).

New Caledonia

Fiji leaf gall, Orange rust, Rind disease and sour rot, Yellow spot.

New Guinea

Black leaf spot (Tar spot).

New Zealand

Mottled stripe.

Papua New Guinea

Banded sclerotial disease, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Downy mildew, Eye spot, Fiji leaf gall, Leaf fleck, Leaf scald, Leaf splitting, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Ramu orange leaf, Ramu streak, Ramu stunt, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Striation, Target blotch, Veneer blotch, Yellow leaf, Yellow spot, Zonate leaf spot.

Samoa

Banded sclerotial disease, Brown stripe, Chlorotic streak, Eye spot, Fiji leaf gall, Orange rust, Pokkah boeng, Red rot, Ring spot, Yellow spot, Zonate leaf spot.

Solomon Islands

Black leaf spot (Tar spot), Eye spot, Fiji leaf gall, Orange rust, Red rot, Ring spot, Sooty mould, Veneer blotch, Yellow spot, Zonate leaf spot.

Vanuatu

Fiji leaf gall, Red rot, Ring spot.

Central America and the Caribbean

Antigua and Barbuda

Brown rust, Eye spot, Gumming, Marasmius basal stem, root and sheath rot, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Rind disease and sour rot, Ring spot, Smut.

Barbados

Apex rot, Brown rust, Brown stripe, Dry rot, Dry top Rot, Eye spot, Fusarium sett and stem rot, Leaf fleck, Leaf scald, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mild mosaic, Mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Wilt, Yellow leaf, Yellow spot.

Belize

Brown rust, Brown spot, Brown stripe, Eye spot, Gumming, Leaf scald, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Wilt.

Costa Rica

Brown rust, Eye spot, Mild mosaic, Mosaic, Orange rust, Pineapple sett rot, Ratoon stunt, Red stripe (Top rot), Ring spot, Smut, Yellow spot.

Cuba

Banded sclerotial disease, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Dry rot, Dry top Rot, Eye spot, Floral smuts, Fusarium sett and stem rot, Gumming, Iliau, Leaf fleck, Leaf scald, Leaf scorch, Leaf yellows, Marasmius basal stem, root and sheath rot, Mosaic, Mottled stripe, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, Target blotch, White speck (White rash), Wilt, Yellow leaf, Yellow spot.

Dominica

Gumming, Leaf scald, Smut.

Dominican Republic

Black rot, Brown rust, Brown stripe, Chlorotic streak, Dry rot, Eye spot, Fusarium sett and stem rot, Gumming, Leaf fleck, Leaf scald, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root

rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Yellow leaf.

El Salvador

Brown rust, Eye spot, Mosaic, Orange rust, Pythium root rot, Ratoon stunt, Red rot, Red stripe (Top rot), Ring spot, Smut, Wilt, Yellow leaf, Yellow spot.

Grenada

Chlorotic streak, Eye spot, Leaf scald, Marasmius basal stem, root and sheath rot.

Guadeloupe

Apex rot, Bacterial chlorotic streak, Brown rust, Brown stripe, Chlorotic streak, Fusarium sett and stem rot, Leaf fleck, Leaf scald, Marasmius basal stem, root and sheath rot, Mild mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Sooty mould, Striation, Wilt, Yellow leaf, Yellow spot.

Guatemala

Brown rust, Eye spot, Leaf scald, Leaf scorch, Mosaic, Orange rust, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Smut, Wilt, Yellow leaf, Yellow spot.

Haiti

Brown rust, Chlorotic streak, Eye spot, Marasmius basal stem, root and sheath rot, Mosaic, Pineapple sett rot, Pokkah boeng, Red rot, Red spot of the leaf sheath, Rind disease and sour rot, Smut.

Honduras

Brown rust, Chlorotic streak, Eye spot, Mosaic, Pokkah boeng, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Yellow spot.

Jamaica

Apex rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Dry rot, Eye spot, Gumming, Leaf scald, Leaf yellowing and internode stunt, Marasmius basal stem, root and sheath rot, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Smut, Sooty mould, Yellow leaf.

Martinique

Apex rot, Brown rust, Chlorotic streak, Eye spot, Leaf scald, Marasmius basal stem, root and sheath rot, Mottled stripe, Pokkah boeng, Ratoon stunt, Red spot of the leaf sheath, Red stripe (Top rot), Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Yellow leaf, Yellow spot.

Nicaragua

Brown rust, Brown spot, Chlorotic streak, Fusarium sett and stem rot, Mosaic, Mottled stripe, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sheath rot, Smut, Sooty mould, Wilt, Yellow leaf.

Panama

Banded sclerotial disease, Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Dry top Rot, Ergot, Eye spot, Fusarium sett and stem rot, Gumming, Leaf scald, Leaf scorch, Marasmius basal stem, root and sheath rot, Mosaic, Mottled stripe, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, Wilt, Yellow spot.

Puerto Rico

Banded sclerotial disease, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Dry Rot, Dry top Rot, Eye spot, Fusarium sett and stem rot, Gumming, Helminthosporium leaf spot, Leaf fleck, Leaf scald, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mosaic, Mottled stripe, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Root and basal stem rot, Sclerotium sheath rot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, White speck (White rash), Yellow spot.

St Kitts and Nevis

Apex rot, Brown rust, Brown stripe, Eye spot, Gumming, Leaf scald, Marasmius basal stem, root and sheath rot, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Rind disease and sour rot, Ring spot, Smut, Wilt.

St Lucia

Chlorotic streak, Eye spot, Leaf scald, Marasmius basal stem, root and sheath rot, Pineapple sett rot, Pokkah boeng, Red rot, Rind disease and sour rot.

St Thomas

Eye spot, Mosaic, Pokkah boeng.

St Vincent

Leaf scald, Marasmius basal stem, root and sheath rot.

Trinidad and Tobago

Brown rust, Brown stripe, Chlorotic streak, Dry Rot, Eye spot, Gumming, Leaf scald, Marasmius basal stem, root and sheath rot, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Sooty mould, Wilt, Yellow spot.

Central and Middle East

Iran

Exserohilum leaf spot, Grassy shoot/White leaf, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red streak, Red stripe (Top rot), Sclerotium sheath rot, Sheath rot, Smut, Streak mosaic, Striation, Wilt, Yellow leaf.

Iraq

Leaf scald, Mosaic, Pokkah boeng, Red rot, Red stripe (Top rot), Rind disease and sour rot, Smut.

Oman

Smut.

Türkiye

Chlorotic streak, Mosaic, Sheath rot.

Europe

Italy

Black leaf spot (Tar spot), Eye spot, Mosaic, Pokkah boeng, Red stripe (Top rot).

Portugal

Rind disease and sour rot, Smut.

Spain

Mosaic, Ratoon stunt.

North America

Mexico

Brown rust, Brown spot, Charcoal rot, Chlorotic streak, Dry Rot, Eye spot, Fusarium sett and stem rot, Gumming, Leaf fleck, Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Mosaic, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Wilt, Yellow leaf, Yellow spot.

USA

Banded sclerotial disease, Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Dry top Rot, Eye spot, Fusarium sett and stem rot, Gumming, Helminthosporium leaf spot, Iliau, Leaf fleck, Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mild mosaic, Mosaic, Mottled stripe, Myriogenospora leaf binding, Orange rust, Phyllosticta leaf spot, Phytophthora sett rot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Root and basal stem rot, Sclerophthora stunt, Sclerotium sheath rot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, Striation, Target blotch, White speck (White rash), Wilt, Yellow leaf, Yellow spot, Zonate leaf spot.

South America

Argentina

Bacterial chlorotic streak, Black leaf spot (Tar spot), Brown rust, Brown spot, Chlorotic streak, Collar rot, Eye spot, Fusarium sett and stem rot, Gumming, Leaf fleck, Leaf scald, Leaf scorch, Mild mosaic, Mosaic, Myriogenospora leaf binding, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Ring spot, Seedling foliage blights, Sheath rot, Smut, Sooty mould, Wilt, Yellow leaf, Yellow spot.

Bolivia

Brown rust, Eye spot, Mosaic, Orange rust, Ratoon stunt, Red rot, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sheath rot, Smut.

Brazil

Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Collar rot, Dry rot, Eye spot, False red stripe, Fusarium sett and stem rot, Iliau, Leaf fleck, Leaf scald, Leaf yellows, Marasmius basal stem, root and sheath rot, Mild chlorotic streak, Mild mosaic, Mosaic, Mottled stripe, Myriogenospora leaf binding, Orange rust, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, White speck (White rash), Wilt, Yellow leaf, Yellow spot.

Colombia

Brown rust, Brown spot, Chlorotic streak, Dry top Rot, Ergot, Eye spot, False floral smut, Gumming, Leaf fleck, Leaf scald, Marasmius basal stem, root and sheath rot, Mild mosaic, Mosaic, Mottled stripe, Orange rust, Phyllosticta leaf spot, Pineapple sett rot, Pokkah boeng, Pythium root rot, Ratoon stunt, Red leaf spot (Purple spot), Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerotium sheath rot, Smut, Wilt, Yellow leaf, Yellow spot.

Ecuador

Brown rust, Leaf scald, Mosaic, Orange rust, Pokkah boeng, Ratoon stunt, Red spot of the leaf sheath, Smut, Sooty mould, Yellow leaf.

French Guiana

Brown stripe, Chlorotic streak, Gumming, Leaf scald, Pokkah boeng, Ratoon stunt, Ring spot, White speck (White rash).

Guyana

Brown rust, Brown stripe, Chlorotic streak, Dry rot, Dry top Rot, Eye spot, Fusarium sett and stem rot, Leaf scald, Marasmius basal stem, root and sheath rot, Orange rust, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sheath rot, Smut, Sooty mould, Yellow spot.

Paraguay

Marasmius basal stem, root and sheath rot, Mosaic, Pineapple sett rot, Pokkah boeng, Rind disease and sour rot, Ring spot, Smut, Sooty mould.

Peru

Black rot, Brown rust, Brown spot, Brown stripe, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Marasmius basal stem, root and sheath rot, Marasmius sheath rot and shoot blight, root rot, Mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red spot of the leaf sheath, Red stripe (Top rot), Rind disease and sour rot, Ring spot, Sclerophthora stunt, Sclerotium sheath rot, Sheath rot, Smut, Sooty mould, Yellow leaf.

Surinam

Chlorotic streak, Eye spot, Leaf scald, Mosaic, Pokkah boeng, Red stripe (Top rot), Ring spot.

Uruguay

Leaf scald, Mosaic, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red rot, Red stripe (Top rot), Ring spot, Smut.

Venezuela

Brown rust, Brown spot, Chlorotic streak, Eye spot, Fusarium sett and stem rot, Leaf scald, Leaf scorch, Mosaic, Mottled stripe, Pineapple sett rot, Pokkah boeng, Ratoon stunt, Red spot of the leaf sheath, Red stripe (Top rot), Ring spot, Sheath rot, Smut, Yellow leaf, Yellow spot.

List of authors

Virendra P. Agnihotri: formerly at Indian Institute of Sugarcane Research, Lucknow 226002, India.

Jean Claude Autrey: formerly at Mauritius Sugarcane Industry Research Institute, Réduit, Mauritius.

Roger A. Bailey: formerly at South African Sugarcane Research Institute, Mount Edgecombe, 4300, KwaZulu-Natal, South Africa.

Yixue Bao: Guangxi University, Nanning, Guangxi 530005, China.

Romina Priscila Bertani: Qumir nano, Batalla de Ayacucho 449, San Miguel de Tucumán, Argentina. Formerly at Instituto de Tecnología Agroindustrial del Noroeste Argentino (ITANOA), Estación Experimental Agroindustrial Obispo Colombres (EEAOC) - Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), CCT NOA Sur. Av. William Cross 3150. Las Talitas, Tucumán, Argentina.

Shamsul A. Bhuiyan: Sugar Research Australia, 90 Old Cove Road, Woodford, QLD 4514, Australia; Queensland Quantum and Advanced Technologies Research Institute, School of Environment and Science, Griffith University, Australia.

Stephen Bolus: USDA ARS, National Germplasm Resources Laboratory, Beltsville, MD, USA.

Michèle Chatenet: formerly at CIRAD-CA, Programme Canne à Sucre, BP 5035, 34032 Montpellier Cedex 1, France.

Baoshan Chen: Guangxi University, Guangxi Key Laboratory for Sugarcane Biology and College of Agriculture, Nanning 530004, China.

Jack C. Comstock: formerly at USDA-ARS, Sugarcane Field Station, 12990 US Hwy. 441N, Canal Point, Florida 33438, USA.

Barry J. Croft: formerly at Sugar Research Australia (SRA), 90 Old Cove Rd, Woodford, Queensland 4514, Australia.

Jean Heinrich Daugrois: CIRAD, UMR PHIM, 34398 Montpellier, France; PHIM, Plant Health Institute, Univ. Montpellier, CIRAD, INRAE, Institut Agro, IRD, Montpellier France.

Michael J. Davis: formerly at University of Florida, Tropical Research and Education Center, IFAS, 18905 S.W. 280 Street, Homestead 33031, USA.

Asha Dookun-Saumtally: formerly at Mauritius Sugarcane Industry Research Institute, Réduit, Mauritius.

Brian T. Egan (deceased): formerly at BTE Consulting, 48 Streeton Parade, Everton Park, Queensland 4053, Australia.

Jing-Gwo Fang: formerly at Taiwan Sugar Research Institute, Department of Horticulture, 54, Sheng Chan Road, Tainan 701, Taiwan (R.O.C.).

Andre Bueno Gama: Louisiana State University Agricultural Center, Baton Rouge, LA, USA.

San-Ji Gao: National Engineering Research Center for Sugarcane, Fujian Agriculture and Forestry University, Fuzhou 350002, China.

Eder A. Giglioti: Smartbreeder S.A., Av. Cezira Giavanoni, 600. 13414-157. Piracicaba, SP. Brazil. Formerly at Federal University of São Carlos, Via Anhanguera, km 174, 13600-970, Araras SP, Brazil.

Jean-Claude Girard: formerly at CIRAD, UMR PHIM, 34398 Montpellier, France; PHIM, Plant Health Institute, Univ. Montpellier, CIRAD, INRAE, Institut Agro, IRD, Montpellier France.

Uriel Green: Sugar Industry Authority, Kendal Road, Mandeville, Jamaica.

Michael P. Grisham: formerly at USDA-ARS, Sugarcane Research Unit, Houma, Louisiana 70360, USA.

Jean-Yves Hoarau: eRcane, 29 rue d'Emmerez de Charmoy, F-97490 Sainte-Clotilde, Reunion Island; CIRAD, UMR AGAP Institut, F-34398 Montpellier, France; UMR AGAP Institut, Univ Montpellier, CIRAD, INRAE, Institut Agro, Montpellier, France.

Jeffrey W. Hoy: Department of Plant Pathology and Crop Physiology, Louisiana State University Agricultural Center, Baton Rouge, Louisiana 70803, USA.

W.H. Hsieh: formerly at Department of Plant Pathology, National Chung Hsing University, Taichung, Taiwan (R.O.C.).

Alison S. Jensen: Sugar Research Australia (SRA), Bundaberg, Queensland 4670, Australia.

Nawshad Joomun: Mauritius Sugarcane Industry Research Institute, Mauritius Cane Industry Authority, 1 Moka Road, Réduit, Mauritius.

Sunita Lal: formerly at Indian Institute of Sugarcane Research, Lucknow 226002, India.

Ching-Shiou Lee: formerly at Taiwan Sugar Research Institute, Department of Agronomy, 54, Sheng-Chan Road, Tainan, 701, Taiwan (R.O.C.).

Meilin Li: Guangxi University, Nanning, Guangxi, 530005, China.

Yaw-Guang Liang: formerly at Taiwan Sugar Research Institute, Department of Agronomy, 54, Sheng-Chan Road, Tainan, 701, Taiwan (R.O.C.).

Ben E. Lockhart (deceased): formerly at University of Minnesota, Department of Plant Pathology, 495 Borlaug Hall, 1991 Upper Buford Circle, St Paul, Minnesota 55108, USA.

Robert C. Magarey: Sugarcane Disease Consultancy (SDC), (formerly Sugar Research Australia), PO Box 559, Tully, Queensland, Australia.

Frédéric Mahé: CIRAD, UMR PHIM, 34398 Montpellier, France; PHIM, Plant Health Institute, Univ. Montpellier, CIRAD, INRAE, Institut Agro, IRD, Montpellier France.

Sizuo Matsuoka: formerly at Federal University of São Carlos, Via Anhanguera, km 174, 13600-970, Araras SP, Brazil.

Sharon McFarlane: South African Sugarcane Research Institute, Mount Edgecombe, KwaZulu-Natal, South Africa.

Dimitre Mollov: United States Department of Agriculture, Animal and Plant Inspection Service, Plant Protection and Quarantine, 5601 Sunnyside Ave., Beltsville, MD 20705, USA.

Chuong N. Ngo: Sugar Research Australia Limited, GPO Box 133, Brisbane QLD 4001, Australia.

A.S. Patil: formerly at Department of Plant Pathology and Agricultural Microbiology, Vasantdada Sugar Institute, Manjari (Bk.) 412307, Dist. Pune, Maharashtra, India.

Maria Francisca Perera: Instituto de Tecnología Agroindustrial del Noroeste Argentino (ITANOA), Estación Experimental Agroindustrial Obispo Colombres (EEAOC) - Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), CCT NOA Sur. Av. William Cross 3150. Las Talitas, Tucumán, Argentina.

Michel Peterschmitt: CIRAD, UMR PHIM, 34398 Montpellier, France; PHIM, Plant Health Institute, Univ. Montpellier, CIRAD, INRAE, Institut Agro, IRD, Montpellier France.

Lilik K. Putra: Bojonegoro University, Jalan Lettu Suyitno No. 02, Bojonegoro, East Java, Indonesia. Formerly at Indonesian Sugar Research Institute, Jalan Pahlawan No. 25, Pasuruan, East Java, Indonesia.

Heshmatolah Rahimian: Department of Plant Protection, College of Agriculture, Mazandaran University, P.O. Box 578, Sari, Iran.

Chandramani Raj: ICAR-Indian Institute of Sugarcane Research, Lucknow 226002, Uttar Pradesh, India.

John W. Randles: Emeritus Professor, Adelaide University, School of Agriculture, Food and Wile, Adelaide, Australia.

Govind Pratap Rao: Indian Agricultural Research Institute, New Delhi 110012, India.

Claude Ricaud (deceased): formerly at Permanent Secretariat, International Society of Sugar Cane Technologists, c/o MSIRI, Réduit, Mauritius.

Philippe Rott: CIRAD, UMR PHIM, 34398 Montpellier, France; PHIM, Plant Health Institute, Univ. Montpellier, CIRAD, INRAE, Institut Agro, IRD, Montpellier France.

Suchirat Sakuanrungsirikul: Advisory office, Field and Energy Renewable Crops Research Institute, Department of Agriculture, Thailand.

A. Salem Saumtally: formerly at Mauritius Sugarcane Industry Research Institute, Réduit, Mauritius.

Rajan Selvakumar: ICAR-Sugarcane Breeding Institute, Indian Council of Agricultural Research, Coimbatore 641 007, Tamil Nadu, India.

R.P. Singh (deceased): formerly at Indian Institute of Sugarcane Research, Lucknow 226002, India.

Shweta Singh: ICAR-Indian Institute of Sugarcane Research, Raebareli Road, Lucknow 226002, India.

Sushma Sood: Sugarcane Field Station, United States Department of Agriculture, Agricultural Research Service (USDA-ARS), Canal Point, FL 33438, USA.

Sydney Sullivan: formerly at Mauritius Sugarcane Industry Research Institute, Réduit, Mauritius.

Sidney Suma (deceased): formerly at Ramu Sugar Limited, PO Box 2183, Lae, Papua New Guinea.

Nicole Thompson: Program Leader, Plant health and diagnostics, SARDI-PIRSA (formerly Sugar Research Australia), GPO Box 397, Adelaide, South Australia, Australia.

Rasappa Viswanathan: ICAR-Indian Institute of Sugarcane Research, Lucknow 226002, Uttar Pradesh, India.

Ronghui Wen: Guangxi University, Guangxi Key Laboratory for Sugarcane Biology and College of Agriculture, Nanning 530004, China.

Peter J.L. Whittle: formerly at Sugar Research Australia, 90 Old Cove Rd, Woodford, Queensland 4514, Australia.

Muqing Zhang: Guangxi University, Nanning, Guangxi, 530005, China.

Chengwu Zou: Guangxi University, Guangxi Key Laboratory for Sugarcane Biology and College of Agriculture, Nanning 530004, China.

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This new edition of the book *A guide to sugarcane diseases* is a revised and updated version of the first edition published in 2000 by the International Society of Sugar Cane Technologists (ISSCT) and CIRAD, with the same objective of gathering practical and updated information in sugarcane pathology. During the last two decades, new diseases have developed and distribution of known diseases has expanded. Furthermore, significant progress was made in disease diagnosis with the emergence of powerful new molecular methods.

The book starts with an introductory chapter on sugarcane morphology, anatomy and physiology. This latter is followed by chapters on diseases caused by various pathogens i.e., bacteria, fungi and fungus-like organisms, viruses, and even a newly reported protozoan that causes chlorotic streak. The book also contains a chapter on diseases of uncertain etiology and a chapter on plant-parasitic nematodes. The procedures for the safe movement of sugarcane germplasm and the common names of sugarcane diseases and their causal agent(s) have been updated, as well as the listing of the sugarcane producing countries and their diseases.

Forty-one experts contributed to this new edition that is expected to become a useful new resource for plant pathologists, the sugarcane community at large, and all people interested in this subject.

Philippe Rott is the Scientific Director of CIRAD's sugarcane quarantine in Montpellier, France. He has over 40 years of worldwide experience in sugarcane pathology, and he produced over 200 scientific papers, reports and book chapters on sugarcane diseases.

Jean-Claude Girard is retired from CIRAD. He was a plant pathologist in several countries for over four decades. He has worked on bacterial, fungal and virus diseases of sugarcane and other plants. He has an extensive experience in field and laboratory diagnosis of plant diseases.

Jean Heinrich Daugrois is the Manager of CIRAD's sugarcane quarantine in Montpellier, France. He has performed numerous consultations in sugarcane pathology, especially in Africa and the West Indies. He has authored papers on several sugarcane diseases.

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