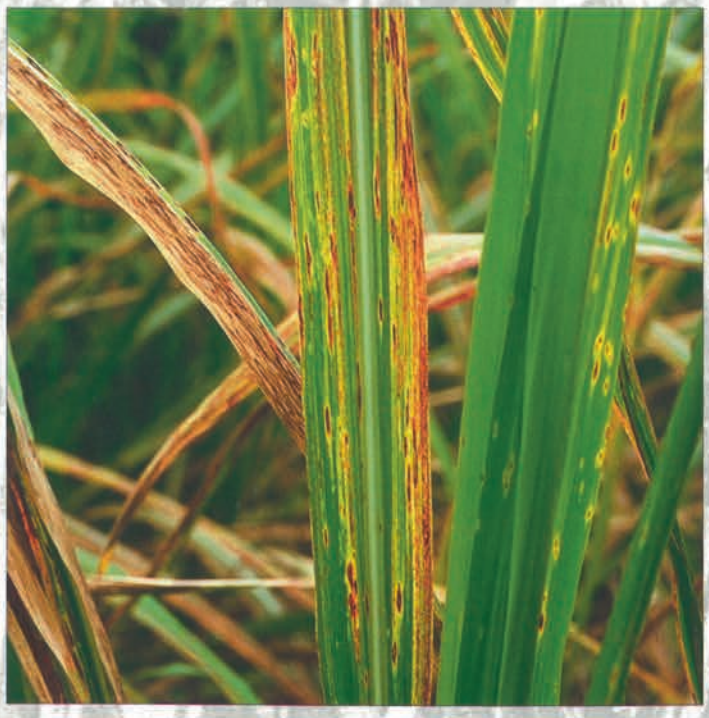
REPÈRES

A guide to sugarcane diseases

Philippe Rott, Roger A. Bailey,
Jack C. Comstock, Barry J. Croft,
A. Salem Saumtally
Editors



CIRAD ISSCT

THE EDITORS

The editors are all sugarcane pathologists and members of the ISSCT (International Society of Sugar Cane Technologists) pathology section committee: P. Rott at CIRAD (Centre de coopération internationale en recherche agronomique pour le développement, Montpellier, France), R.A. Bailey at SASEX (South African Sugar Association Experiment Station, Mount Edgecombe, South Africa), J.C. Comstock at USDA-ARS (United States Department of Agriculture, Agricultural Research Service, Canal Point, USA), B.J. Croft at BSES (Bureau of Sugar Experiment Stations, Woodford, Australia) and A.S. Saumtally at MSIRI (Mauritius Sugar Industry Research Institute, Réduit, Mauritius).

CIRAD

The Centre de coopération internationale en recherche agronomique pour le développement (CIRAD) is a French centre for international co-operation in development-oriented agricultural research, with emphasis on tropical and subtropical zones. It carries out research, agricultural development, and training operations in about 50 countries.

ISSCT

The International Society of Sugar Cane Technologists (ISSCT) is an association of scientists, technologists, institutions and companies/corporations concerned with the technical advancement of the cane sugar industry and its co-products. Over the years, ISSCT has played a prominent role in promoting technical publications for the industry and, since 1961, has published three volumes on sugarcane diseases.

Cover photo
Eye spot disease, caused by Bipolaris sacchari, *Mexico (P. Rott)*

A guide to sugarcane diseases

REPÈRES

A guide to sugarcane diseases

Philippe Rott, Roger A. Bailey,
Jack C. Comstock, Barry J. Croft,
A. Salem Saumtally
Editors

CIRAD ISSCT

ISBN (papier) : 978-2-87614-386-9
ISBN (pdf) : 978-2-87614-806-2
DOI : 10.19182/agritrop/00220

Contents

67 Diseases caused by fungi

Preface

Since its IXth Congress in 1956, the International Society of Sugar Cane Technologists (ISSCT) has encouraged its Pathology Section to publish books on sugarcane diseases by specialists in this field. Two volumes were published through the Elsevier Publishing Company, Amsterdam, The Netherlands in 1961 and 1964, the first volume dealing with the most important and documented diseases and the second with diseases either of lesser importance or less common.

At the XVIth Congress of the ISSCT in 1977, it was felt that new editions of these volumes were needed. However, following investigations, it was decided to publish a new book dealing with major diseases of sugarcane in place of the previous volume 1. The volume which was subsequently published in 1989, included most of the diseases treated in the first volume 1, together with certain diseases which had been published previously in volume 2, but were now included because of their increased importance.

In 1992, ISSCT decided to proceed with a new edition of the previous volume 2, but at the XXIInd Congress in 1995, it was concluded that the publication of a practical field guide for all sugarcane diseases would be more useful.

The Pathology Section Committee entrusted the work to an editorial team comprising P. Rott, Chairman (CIRAD, France), R.A. Bailey (SASEX, South Africa), J.C. Comstock (USDA-ARS, USA), B.J. Croft (BSES, Australia) and A.S. Saumtally (MSIRI, Mauritius), all members of the committee.

CIRAD (the French Centre for International Co-operation in Development-oriented Agricultural Research) offered to publish the book within its series REPERES and to provide the financial investment needed. It was also decided to follow the same format adopted by CIRAD for its D-CAS software used as an interactive PC aid for the diagnosis of sugarcane diseases, and to produce at the same time as the hard copy of the book, a CD-ROM of an up-dated version of the software, both products being under the auspices of ISSCT and CIRAD.

I have no doubt that this volume and the CD-ROM will prove extremely useful to all those who have to deal with the management and control of sugarcane diseases world-wide. A good deal of the information included is derived from the previous three volumes, updated with new information where applicable, and with recent results on improved molecular diagnostic techniques.

On behalf of the ISSCT, I should like to express our thanks to CIRAD for its support and collaboration, to the editors and contributors of the various chapters for their efforts, and to all the organizations with which they are associated, for enabling them to undertake this work.

Claude Ricaud
Permanent Secretary
International Society of Sugar Cane Technologists
September 1999

Résumé

De nombreux changements ont eu lieu en pathologie de la canne à sucre au cours des dix dernières années. De nouvelles connaissances sont apparues sur des maladies connues, de nouvelles maladies ont été décrites et identifiées et, grâce aux progrès de la biologie moléculaire, de nouvelles techniques de diagnostic ont été mises au point. C'est pourquoi la section pathologie de la Société internationale des technologues de la canne à sucre (ISSCT) a décidé de publier un nouveau livre sur les maladies de la canne à sucre. Son objectif est de fournir une information actualisée relative aux connaissances scientifiques et aux aspects pratiques de la lutte, sans pour autant décrire de façon exhaustive les maladies. Chaque maladie fait l'objet d'un chapitre illustré de nombreuses photographies en couleurs, dans lequel sont rassemblées des données sur son agent causal, sa distribution géographique, ses symptômes, sa transmission, son spectre d'hôtes, son épidémiologie et son importance économique. Des informations concernant les souches de l'agent pathogène, le diagnostic et la lutte ainsi qu'une bibliographie complètent chacun de ces chapitres.

Sugarcane morphology, anatomy and physiology

G. Claude Soopramanien

Sugarcane is a member of the family Gramineae and it belongs to the genus *Saccharum* (S.) (Tribe: Andropogoneae). The six known species are perennial grasses which originated in the Old World. *Saccharum spontaneum* and *S. robustum* occur in the wild, while the four other species are considered as cultigens.

Saccharum spontaneum L. (2n = 40–128) has profuse tillering, aggressive rhizomes, stalks 0.3 to 8 m in height, hard and pithy internodes with little juice and sucrose. Breeders use it for vigour, hardiness and resistance to major diseases.

Saccharum robustum Brandes & Jeswiet ex Grassl (2n = 60–194) is indigenous to New Guinea and has erect or recumbent stalks which may reach 10 m in height. It is used to a limited extent in breeding programmes because of its susceptibility to leaf scald and *Sugarcane mosaic virus*.

Saccharum officinarum L. (2n = 80) is known as the 'Noble' cane due to its thick and sweet stems. It probably originated in New Guinea and is grown in the south-western Pacific for chewing. *Saccharum officinarum* reached Hawaii about AD 800 and was first used in India to make sugar. The species is adapted to tropical conditions and the stalks have a comparatively high sucrose and low fibre content. The noble canes are often susceptible to major diseases.

Saccharum barberi Jeswiet (2n = 82–142), *S. sinense* Roxb. (2n = 118) and *S. edule* Hassk. (2n = 74) also have a lower fibre and higher sucrose content than the two wild species. They are also susceptible to diseases.

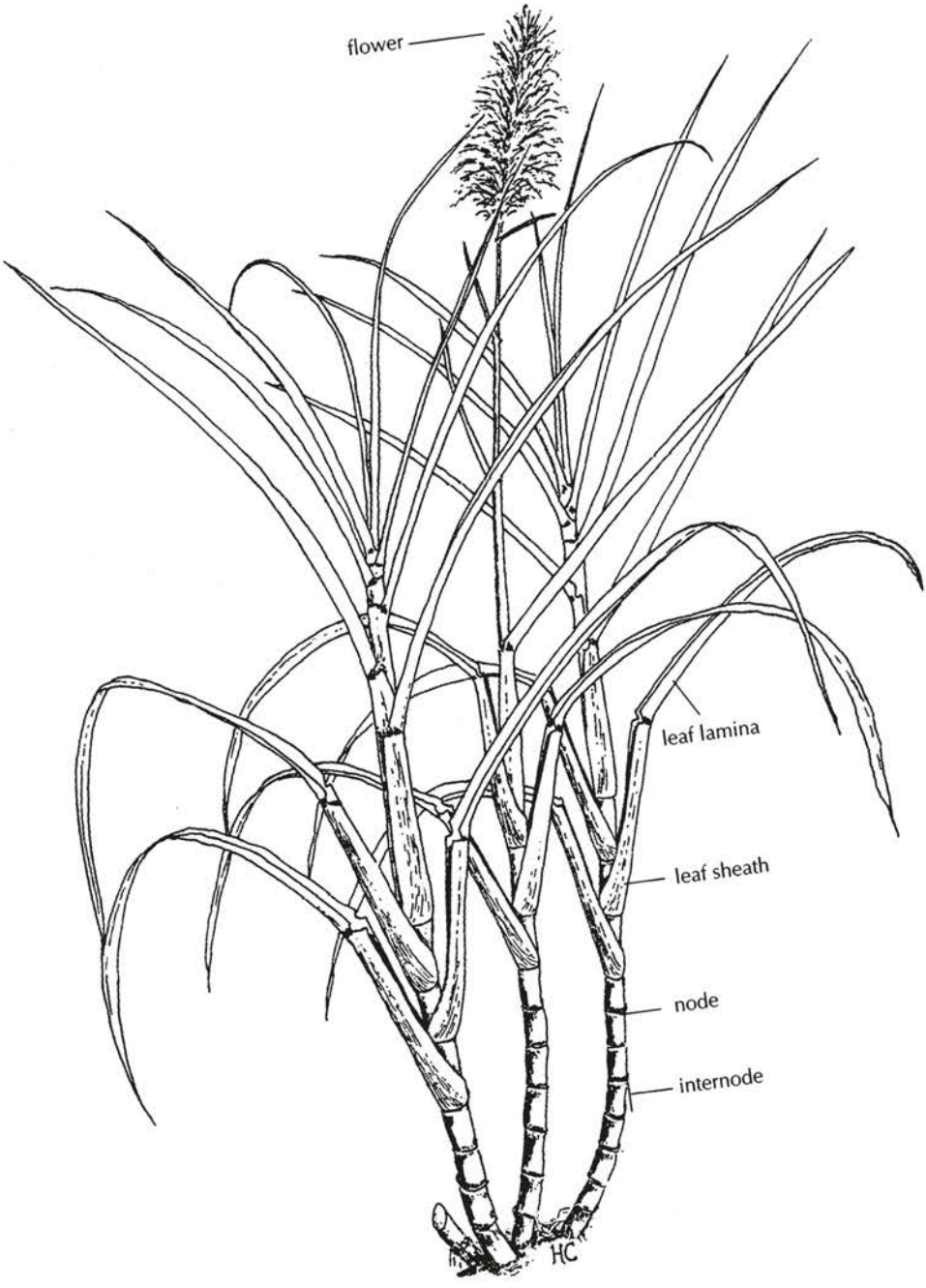


Figure 1. Sugarcane plant (H. Chaillet).

The sugarcane, as all tufted grasses, consists of a clump of stems (Figure 1), also referred to as shoots, stalks or tillers, with a fibrous root system. The stalk is made up of a number of phytomers (a phytomer is made up of one node, an internode and its subtending leaf lamina and leaf sheath) with the oldest ones at the base. A brief description of the morphology and anatomy of the sugarcane is given below together with an outline of the physiology of its growth and development. For more details the reader should refer to: ALEXANDER, 1973; BLACKBURN, 1984; FAUCONNIER and BASSEREAU, 1970; HUMBERT, 1963; JULIEN *et al.*, 1989; LYON, 1920; MARTIN, 1961; and VAN DILLEWIJN, 1952.

Morphology and anatomy

The root system

When a piece of cane stalk, referred to as a cutting or stem cutting or cane sett, is planted under favourable conditions, roots (known as sett roots) develop from primordia in the region of the root band (Figure 2). These roots are thin and branched and their main function is to provide water and nutrients to the young shoot developing from the lateral bud. The life span of these roots is relatively short (2 to 3 months) and their function is subsequently taken over by shoot-roots. The shoot-roots develop from primordia within the root bands on the lower portion of the developing shoots. They are thick and whitish with few branches at first. In an established clump (stool) three types of roots may be

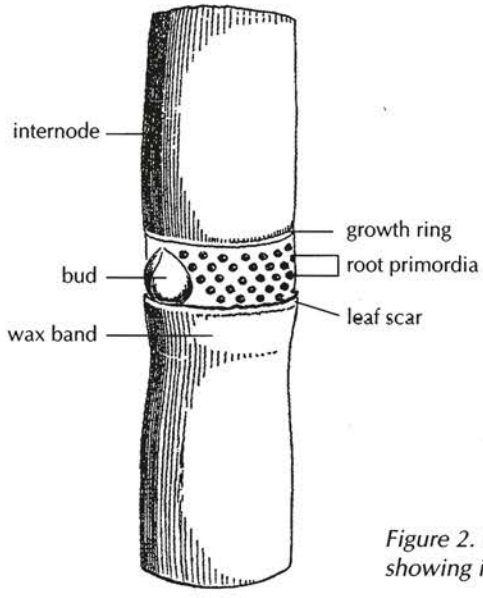


Figure 2. Portion of a sugarcane stalk showing its different parts (J. Guého).

distinguished, their relative lengths or abundance being dependent upon genotype and soil conditions. The three types are known as: superficial roots, buttress roots and the 'rope system'. The superficial roots are thin and branched with numerous root hairs. They occupy the upper layers of the soil and provide water and nutrients for the stalk. The buttress roots are slightly thicker and are deeper in the soil. Their main function is to anchor the plant although they may also absorb water. The 'rope system', which is made up of two or more intertwined roots, grows more or less vertically down the soil profile, sometimes up to 2 or 3 m. These roots may provide the plant with water under dry conditions.

The root of the sugarcane plant is similar to those of other monocotyledons, i.e. with a root cap or calyptra, a growing point and an elongation zone with unicellular root hairs.

In cross-section (Figure 3), the sugarcane root is seen to be made up of three rings of tissue with a central core. The outer ring is made up of an epidermal layer adjacent to the exodermis and the schlerenchyma (thickened cells). The larger ring (the cortex) is made of thin parenchyma cells. The third ring is the endodermis which encloses the vascular tissues, i.e. the xylem and the phloem.

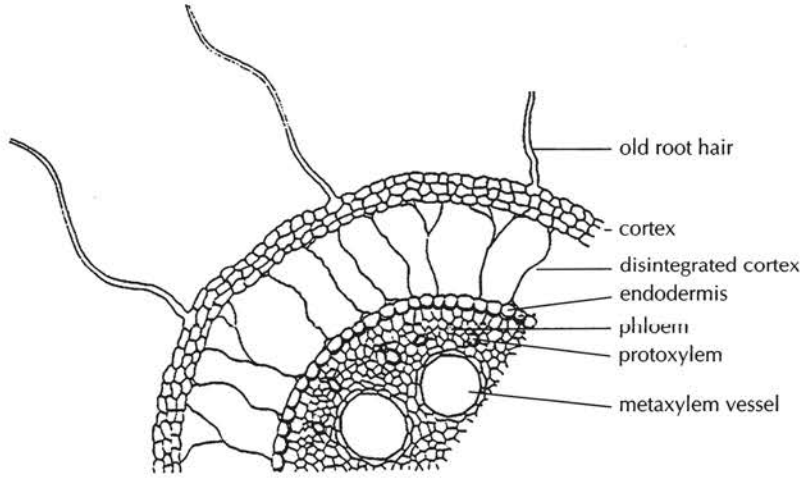


Figure 3. Transverse section through young sett root of sugarcane (J. Guého).

The stem

The stem is made up of a series of nodes and internodes with the youngest ones at the top of the stalk enclosed in the young developing leaves. As in most monocotyledons, the vegetative apical meristem gives rise to phytomers after differentiation.

The mature cane internode is cylindrical in shape, and its length, diameter and colour vary with genotype as well as environmental conditions. The internode may have corky cracks and/or corky patches which differ from the growth cracks that occur when the stalks split lengthwise under certain conditions, e.g. alternating wet and dry conditions.

Between the internode and the node, a light coloured ring called the growth ring may be observed. It resumes its activity, e.g. when the stalk is lodged. At the node there is a root band consisting of root primordia, a lateral bud and the leaf scar. The lateral bud varies in shape according to the genotype. The leaf scar marks the place where the leaf sheath was attached to the node.

The stem is made up of a large mass of storage tissue (mainly for sucrose) consisting of parenchyma cells within which are interspersed the vascular bundles (Figure 4). The outer layer of the stem is made up of an epidermal outermost ring and an adjacent layer called the cortex or rind. These cells may contain some pigment and are lignified.

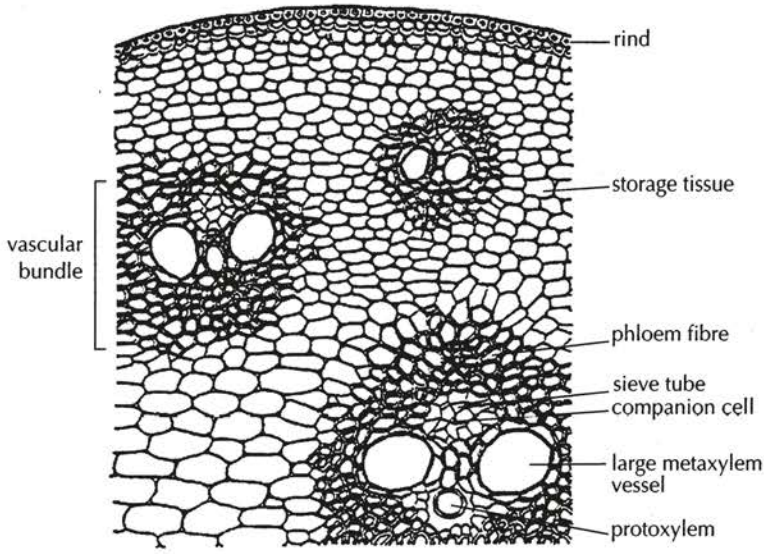


Figure 4. Cross-section of stem of sugarcane showing main features (J. Guého).

The leaf

The leaf of the sugarcane is made up of two parts, the leaf sheath and the leaf lamina. The leaf sheath is the lower portion of the leaf; it is tubular, wraps around the internode and its margins overlap at the base. The outer surface of the leaf sheath may bear variable numbers of hairs.

The leaf lamina is strap-shaped and tapers towards the leaf tip. The margins are sharp and may be coarsely toothed. The midrib is quite conspicuous and is usually white on the upper surface. The blade joint is the point where the leaf lamina meets the upper limit of the leaf sheath. The inner surface is known as the throat, whilst the outer region is known as the collar or leaf triangle (sometimes referred to as the dewlap). The colour of the leaf lamina varies from light to dark green.

The leaf of the sugarcane may be various shades of green but the sheaths, when still enclosed, are light green or whitish. A cross-section of the leaf blade (Figure 5) reveals an upper and a lower epidermis in between which are found small and large vascular bundles, fibre cells, parenchyma or mesophyll (similar to palisade) cells (Kranz type anatomy). The bundle sheath is a ring consisting of a layer of parenchyma cells containing chloroplasts. The mesophyll cells also contain chloroplasts and, together with the vascular bundle sheath cells, participate in photosynthesis.

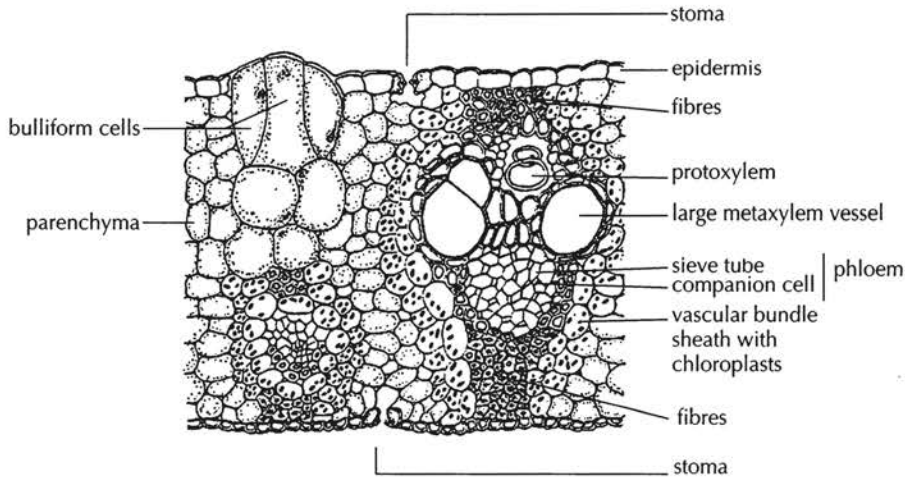


Figure 5. Vertical section through leaf blade of sugarcane showing two vascular bundles and associated tissues (J. Guého).

The flower

The sugarcane flower consists of a main axis which bears primary and secondary branches. On the latter are found pairs of spikelets. Each spikelet has a lemma, a palea, an inner and outer glume as well as a bifid stigma (purplish in colour), as well as three anthers. On the branches are also found numerous hairs which give a silvery appearance to a field with profusely flowering plants.

Physiology of growth and development

The sugarcane plant produces viable seeds but under commercial conditions it is vegetatively propagated by means of stem cuttings (setts) (or whole stalks in a few countries). The use of infected cuttings plays a major role in the transmission of systemic diseases. This explains the setting up of special nurseries for clean planting material in some countries. The lateral bud develops into a shoot within 10 to 15 days under moist and warm conditions (>10°C). Shoot roots are formed from the root primordia on the compressed phytomers below ground level. The lateral buds on this mother or primary shoot give rise to secondary shoots (or tillers) and the latter will give rise to tertiary tillers, etc. (i.e. tillering). Lateral buds on a growing stalk will develop only if the apical meristem is either damaged (by diseases, chemicals such as ripeners, insect pests such as the stalk borer) or after flowering. The lower lateral buds may also develop after a hurricane or cyclone or after prolonged flooding.

Tiller number increases after germination and after a peak density stage, tillers start to die until eventually a more or less stable population of shoots (millable stalks) is achieved up to harvest. The number of stalks at the peak density stage and at harvest depends upon factors such as variety, soil moisture and temperature, available nitrogen, sunlight and the incidence of pests (particularly the stalk borer) and diseases such as smut.

Stalk elongation is the second developmental phase of the sugarcane. Cell multiplication, differentiation and elongation take place during a given period. Under tropical conditions, with day temperatures above 25°C, night temperatures above 15°C, ample soil moisture and sufficient radiation levels, stalk elongation rates of between 1 and 2 cm/day can be recorded.

The C-4 type of photosynthesis takes place in sugarcane, the rate depending upon the variety and environmental conditions. Diseases which reduce the effective leaf area affect the rate of photosynthesis.

Nitrogen, phosphorus and potassium are major nutrients which may influence dry matter accumulation. Assimilated dry matter is used for growth and maintenance (dark respiration) and any excess is stored mainly in the form of sucrose. During elongative growth, as much as 60% of accumulated dry matter can be used for growth and maintenance. Mineral deficiencies or toxicities influence growth and development and a number of these give typical foliar symptoms.

After a certain stage of growth, the sugarcane stalk experiences a more rapid accumulation of sucrose – this phase is known as the ripening phase. The partitioning of dry matter is then in favour of sucrose storage and may reach 65–70% of the total carbon assimilated. Apart from the varietal factor, warm days and cool nights, as well as mild moisture stress, favour ripening. Ripening can be enhanced through irrigation control or the use of chemicals known as cane ripeners.

After its juvenile phase the sugarcane stalk may flower if it experiences a sequence of day-length (11.5–12.5 h). Temperatures below 18°C, water stress or interruption of the night period with light (artificially) are factors which are unfavourable for flowering. Flowering may be deleterious to yield under some specific conditions. After flowering has taken place side shoots will develop on the upper nodes (4th, 5th and 6th). After harvest, buds on the basal part of the stalks left in the ground develop to give rise to another crop referred to as the ratoon. The number of ratoon crops harvested before a field is replanted varies among the various cane producing countries, depending upon the prevailing agronomic and economic factors.

References

ALEXANDER A.G., 1973. Sugar Cane Physiology. Amsterdam, The Netherlands, Elsevier Scientific Publishing Company, 752p.

BLACKBURN F., 1984. Sugar Cane. Harlow, UK, Longman Group, 414p. (Tropical Agriculture Series)

FAUCONNIER R., BASSEREAU D., 1970. La Canne à Sucre. Paris, France, Maisonneuve et Larose, 468p. (Techniques Agricoles et Productions Tropicales 18–19)

HUMBERT R.P., 1963. The Growing of Sugar Cane. Amsterdam, The Netherlands, Elsevier Publishing Company, 710p.

JULIEN M.H.R., IRVINE J.E., BENDA G.T.A., 1989. Sugarcane anatomy, morphology and physiology. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 1–20. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

LYON H.L., 1920. The Cane Plant. Hawaii Plant Record 23: 293–316.

MARTIN J.P., 1961. The anatomy of the sugar cane plant. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 3–52. Amsterdam, The Netherlands, Elsevier Publishing Company.

VAN DILLEWIJN C., 1952. Botany of the Sugar Cane. Waltham, USA, Chronica Botanica, 520p.

Diseases caused by bacteria

Bacterial mottle

Barry J. Croft

Cause

Pectobacterium chrysanthemi (Burkholder *et al.* 1953) Brenner *et al.* 1973 *emend.* Hauben *et al.* 1998, bacterium.

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

Pectobacterium chrysanthemi 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 1 to 2 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 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 (×400–1000 magnification).

Strains of the pathogen

Pectobacterium chrysanthemi causes disease in a wide range of plant species (HAUBEN *et al.*, 1998). The bacterium which infects grasses may be a distinct strain (DOWSON and HAYWARD, 1960) but no definitive studies have been conducted.

Transmission

Bacterial mottle is associated with fields that have been flooded. It is apparent that 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 less important 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 setts 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 (*Panicum maximum*), para grass (*Brachiaria mutica*) and elephant grass (*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.

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

Figure 2. Chlorotic leaf mottling (ISSCT).

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).

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.

References

DOWSON W.J., HAYWARD A.C., 1960. The bacterial mottle pathogen of Queensland sugar cane. International Sugar Journal 62: 275.

HAUBEN L., MOORE E.R.B., VAUTERIN L., STEENACKERS M., MERGAERT J., VERDONCK L., SWINGS J., 1998. Phylogenetic position of phytopathogens within the *Enterobacteriaceae*. Systematic and Applied Microbiology 21: 384-397.

STEINDL D.R.L., 1964. Bacterial mottle. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 2–11. Amsterdam, The Netherlands, Elsevier Publishing Company.

False red stripe

Eder A. Giglioti and Sizuo Matsuoka

Cause

Xanthomonas sp., bacterium.

Significant differences were not found between the false red stripe (FRS) and the sugarcane gumming disease (*Xanthomonas axonopodis* pv. *vasculorum*) pathogens 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 in restriction digest patterns of DNA was found between the pathogens. Additionally, the genetic profiles obtained for isolates of the FRS bacterium did not resemble the profiles of other *Xanthomonas* species attacking sugarcane and other Poaceae (Giglioti *et al.*, 1999). The symptoms described for FRS are also different from those of the other sugarcane diseases caused by xanthomonads and, therefore, there is strong evidence that the FRS bacterium is a unique pathogenic agent. Efforts should be directed towards precisely determining its taxonomic position.

Geographical distribution

Brazil.

Symptoms

FRS is characterized by the presence of narrow stripes (1 mm) parallel to the leaf midrib (Figure 1). Frequently, the stripes have a mixture of reddish and yellowish colours, extending progressively from the tip towards the middle of the leaf (Figure 2). The stripes can extend to the base of the leaf but have not been observed on the leaf sheath. The apical meristem is not affected nor are top rot symptoms observed. When viewed against sunlight, the stripes appear partially translucent and the yellow colour becomes more evident. In highly susceptible genotypes, the stripes frequently coalesce becoming several millimetres wide and have a more intense and predominant reddish colour. In

such situations, shortening of the internodes has been observed. In contrast, fewer stripes, usually only one or two, are observed on the leaves of resistant genotypes. Medium aged and adult plants appear to be more susceptible to the disease and symptoms are more severe in older leaves.

FRS was mistakenly identified as red stripe disease (*Acidovorax avenae* subsp. *avenae*) for many years (Figures 3 and 4). A differential response among genotypes 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; GIGLIOTI *et al.*, 1998; GIGLIOTI *et al.*, 1999). Differences in symptoms between FRS and red stripe disease can be used to differentiate the diseases. The leaf lesions of FRS originate near leaf tips and progress towards the base of the leaf, whereas those of red stripe are usually more basal. FRS affects more mature plants, whereas red stripe affects young leaves on young, less mature plants. FRS does not exhibit a top rot phase that is sometimes prevalent with red stripe. Plants affected by red stripe tend to be distributed in clusters and incidence may vary from 15 to 50% in intermediate and susceptible genotypes, respectively, under conditions conducive for disease occurrence; in contrast, in a field affected by FRS, all plants generally show at least one leaf stripe.

Diagnosis

Presumptive diagnosis can be based on distinctive symptoms. Serological procedures (Dot-Blot and ELISA) have been tested. Serological variability was observed among strains of the pathogen and must be taken into consideration. Isolation is a useful approach because the pathogen grows readily on relatively simple media. The medium frequently used is a modification of DYGS medium (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 modified 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 borders, convex and viscous. Upon ageing, the bacterium causes a slight darkening of the medium. In addition to colony colour and growth rate, serological microprecipitation tests on glass microscope slides can be used to distinguish rapidly the FRS pathogen from other yellow contaminant colonies that grow on isolation media.

Strains of the pathogen

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

Figure 1. Leaf stripes parallel to the midrib (S. Matsuoka).

Figure 2. Foliar symptoms in the field (S. Matsuoka).

Figure 3. Leaves showing false red stripe (left) and red stripe symptoms (right) (S. Matsuoka).

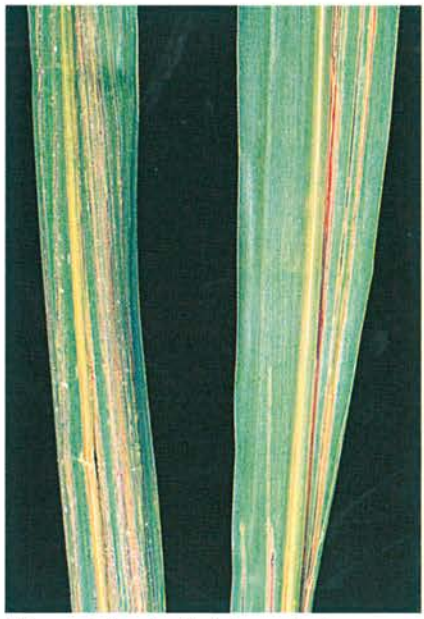


Figure 4. Leaf with false red stripe symptoms (left) and leaf with red stripe symptoms (right) (S. Matsuoka).

Transmission

The natural means of transmission of the FRS bacterium is unknown. However, the generalized distribution of diseased plants and the greater severity of symptoms at the edges of sugarcane fields suggest that the pathogen is mainly spread by aerial means, and that infection occurs mainly through small lesions caused by contact of 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 (Gigliotti *et al.*, 1999). Information regarding spread through infected cuttings is unavailable.

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 symptomology, epidemiological characteristics are important features that can be used to differentiate FRS from red stripe (Gigliotti *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 favoured by high humidity and soil fertility which are common in sugarcane fields in lowland areas and in river valleys. Symptoms become more intense in the spring and summer because of rain and higher temperatures. During these periods, most of the sugarcane plants are 8–12-months-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 genotypes. A high frequency (22–23%) of genotypes that are moderately or highly susceptible to FRS have been noticed in the middle stages of selection in breeding programmes in Brazil.

Control

Use of resistant cultivars is the most effective means of control. The planting of susceptible genotypes should be avoided in locations with high relative humidity, fertility and mild temperatures that favour the disease. Routine

evaluations and/or tests are necessary to screen sugarcane genotypes for FRS resistance and thus increase the frequency of resistant genotypes being produced by variety improvement programmes.

References

ALMEIDA I.M.G., 1994. Bacterial diseases of sugarcane in Brazil. *In*: Current Trends in Sugarcane Pathology. G.P. Rao *et al.* (Eds), p. 73–84. New Delhi, International Books and Periodicals Supply Service.

ALMEIDA I.M.G., CARVALHO M.L.V., RODRIGUES NETO J., 1989. Nova doença bacteriana em cana-de-açúcar ocorrendo nos estados de São Paulo e Paraná. Summa Phytopathologica 15: 19 (abstract).

CARVALHO M.L.V., 1991. Ocorrência e caracterização de *Xanthomonas campestris* em cana-de-açúcar (*Saccharum* spp.) nos estados de São Paulo e Paraná. Piracicaba, ESALQ/USP, 76p. (M.Sc. Thesis).

CCA/UFSCAR, 1997. Relatório anual 1996 do programa de melhoramento genético da cana-de-açúcar, Araras SP, 91p.

GIGLIOTI E.A., BERIAM L.O.S., ALMEIDA I.M.G., MASUDA Y., MATSUOKA S., 1996a. Diferenciação epidemiológica das doenças causadas por *Acidovorax avenae* subsp. *avenae* e *Xanthomonas campestris* em cana-de-açúcar. Fitopatologia Brasileira 21 (Suplemento): 335.

GIGLIOTI E.A., MASUDA Y., MATSUOKA S., MENESES L.L., MARCHETTTI L.B.L., 1996b. Implicações da ocorrência das bactérias *Xanthomonas campestris* e *Acidovorax avenae* subsp. *avenae* em cana-de-açúcar. Anais Do 6° Congresso Nacional Da Stab, Maceió, p. 294–301.

GIGLIOTI E.A., MASUDA Y., MATSUOKA S., 1998. Ocorrência de Estrias Vermelhas em cana-de-açúcar: Perfilhamento, resistência parcial de variedades e exigência em condições ambientais restringem os danos. STAB, Piracicaba 16(3): 10.

GIGLIOTI E.A., ALMEIDA I.M.G., CLERC F., ROTT P., CHATENET M., BERIAM L.O., CARVALHO M.L.V., MASUDA Y., MATSUOKA S., 1999. False Red Stripe: A new disease of sugarcane in Brazil. Proceedings International Society of Sugar Cane Technologists Congress 23: 410–411.

RODRIGUES NETO J., MALAVOLTA JR. V.A., VICTOR O., 1986. Meio simples para o isolamento e cultivo de *Xanthomonas campestris* pv. *citri* tipo B. Summa Phytopathologica 12(1, 2): 16.

Gumming

A. Salem Saumtally and Asha Dookun

Cause

Xanthomonas axonopodis pv. *vasculorum* (Cobb 1894) Vauterin *et al.* 1995, bacterium.

Geographical distribution

Antigua, Argentina, Belize, Brazil, Colombia, Cuba, Dominica, Dominican Republic, Fiji, Ghana, India, Madagascar, Madeira, Malawi, Mauritius, Mozambique, Panama, Puerto Rico, Réunion, St Kitts and Nevis, St Lucia, St Vincent, South Africa, Swaziland, Zimbabwe.

The disease has been eradicated from Australia. It was reported in the first half of the 20th century but has not been seen for many years in Barbados, Guadeloupe and Martinique.

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. There are two distinct phases: the foliar stage and the systemic stage. In the foliar stage, infection usually starts at the edge of the leaf, as distinct yellow stripes, 4–5 mm wide, often associated with reddish dots. As it progresses towards the base of the leaf, the older part of the stripe becomes necrotic (Figure 1). Several stripes may be present on the same leaf, but not all leaves of a stalk show symptoms. A distinctly red foliar stripe as opposed to the yellow one has also been described (RICAUD and AUTREY, 1989) (Figure 2).

In susceptible varieties, stripes progress at an angle to the midrib and penetrate the leaf sheath and stalk to induce the systemic phase. External symptoms are partial or total chlorosis of new leaves in mature cane (Figure 3), similar to the chlorotic symptom produced by leaf scald disease (*Xanthomonas albilineans*). However, this symptom is not necessarily observed in all cases of systemic

infection. Chlorosis can also be present in young ratoons after harvest, as a result of transmission by contaminated knives. Chlorotic leaves may revert back to their normal colour if conditions favourable 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 at the nodal and internodal tissues and growing point. In a highly infected cane, gum exudation can be observed when the stalk is cut transversely (Figure 4). Death of the mature stalk can occur if the growing point is affected.

Diagnosis

Leaf stripes may not be easily distinguishable from those produced by the leaf scald bacterium upon aerial transmission. A piece of the stripe tissue mounted in water on a microscope slide produces a milky bacterial exudate visible to the naked eye after a few minutes. This feature distinguishes gumming disease from the leaf scald stripe where no or little exudation occurs. The bacterium is readily isolated on sucrose peptone medium from an infected stalk or leaf stripe. The latter, after surface sterilization, is streaked directly on to the medium. Isolation from the stalk is carried out by slicing the nodal part thinly, allowing it to diffuse in sterile distilled water and a loopful streaked 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. 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 immunofluorescence.

Strains of the pathogen

Significant heterogeneity exists in the population of the gumming bacterium. Strains that show a differential host reaction to sugarcane varieties have been reported, and three races are known to occur in Mauritius (MAURITIUS SUGAR INDUSTRY RESEARCH INSTITUTE, 1983). Race 1 attacks the noble canes, *Saccharum officinarum*, while races 2 and 3 are observed in hybrid cultivars. The races can be distinguished on Kelman tetrazolium medium (RICAUD and AUTREY, 1989), by ELISA using monoclonal antibodies (DOOKUN *et al.*, 1996), by DNA-based techniques including restriction enzyme analysis, random amplified polymorphic DNA (RAPD) and restriction fragment length polymorphism (RFLP) (SAUMTALLY, 1996). Using the latter technique, QHOBELA and CLAFLIN (1992) distinguished two geographical groups: Southern Africa and Eastern Africa. VAUTERIN *et al.* (1995) recognized two groups of the bacterium based on

DNA-DNA hybridization studies and reclassified the strains as *X. axonopodis* pv. *vasculorum* and *X. vasicola* pv. *vasculorum*. The latter name was, however, not considered valid by the Sub-Committee on Taxonomy of Plant Pathogenic Bacteria (YOUNG *et al.*, 1996). Further variation within the gumming causal agent has been reported by SAUMTALLY (1996), and five groups were distinguished amongst strains obtained from different countries and hosts by RAPD and ERIC-PCR (enterobacterial repetitive intergenic consensus-polymerase chain reaction).

Five groups of the pathogen were also found by fatty acid analysis. Group A corresponds to strains from *Thysanolaena maxima* and is weakly pathogenic on sugarcane. Group B consists of strains belonging to race 1 isolated from sugarcane, palms and bamboo grass and equivalent to *X. axonopodis* pv. *vasculorum* (VAUTERIN *et al.*, 1995). Group C comprises strains isolated in South Africa, Zimbabwe and Madagascar and is related to *X. vasicola* pv. *vasculorum* (VAUTERIN *et al.*, 1995). Group D consists of strains of races 2 and 3 while group E is associated with the red stripe symptoms of gumming disease (DOOKUN *et al.*, 2000).

Transmission

Gumming disease is transmitted by wind-blown rain, infected setts and cutting implements. Violent winds and rains, especially during stormy conditions, facilitate its spread over wide areas. High humidity and warm temperatures are favourable for disease transmission as during 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 infection. Knife transmission may occur either during the preparation of cuttings or at harvest. Mechanical harvesters can efficiently spread the bacterium through the cutting action of the blade and 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 is not very important and the role of these hosts as reservoirs of the disease has not yet been clearly defined. Natural hosts of gumming disease 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*). In the latter, two *Xanthomonas* species producing identical symptoms have been reported, one is weakly pathogenic to sugarcane while a second one has similar properties to the sugarcane strain (RICAUD and AUTREY, 1989).

Figure 1. Yellow leaf stripes turning necrotic (MSIRI, P. Rott).

Figure 2. Red foliar stripes (MSIRI).

Figure 3. Partial chlorosis of leaves (MSIRI).

Figure 4. Gum exudate from cut stalks (MSIRI, P. Rott).

Epidemiology

Epidemics are correlated with intense cyclonic periods that are followed by a dry and cool season. The effect of the disease is more pronounced when the plant is stressed under conditions of low temperatures, drought and is close to maturity. Incidence of the disease gradually declines over the years if turbulent climatic conditions do not persist.

Economic importance

With the cultivation of resistant interspecific hybrid cultivars, gumming disease has become less important. However, it remains potentially a serious problem under cyclonic weather conditions that favour the build-up of the bacterium. Experimentally, the disease has been shown to cause a decrease in stalk length and diameter, as well as a reduction in cane and sugar yield. Systemic infection can reduce cane yield by 45% (Autrey *et al.*, 1986).

The pathogen also interferes in the manufacture of sugar. Xanthan gum, which is produced in systemically 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 effective therapy is available, but it is recommended to treat cuttings in a hot water bath at 50°C for 30 min followed by 50°C for 2–3 h the next day. This treatment reduces infection in contaminated cuttings. The hot water treatment is recommended for the establishment of nurseries in conjunction with roguing of diseased plants. Other measures required to control the disease include the elimination of alternative hosts, and knife disinfection during harvest and in the preparation of cuttings.

Disease-free plantlets can be produced by tissue culture technique and these are recommended for the exchange of germplasm. The occurrence of strains highlights the quarantine importance of this disease.

References

Autrey L.J.C., Dhayan S., Sullivan S., 1986. Effect of race three of gumming disease pathogen on growth and yield in two sugar cane varieties. Proceedings International Society of Sugar Cane Technologists Congress 19: 420–428.

Dookun A., Autrey L.J.C., Jones P., 1996. Monoclonal antibodies specific to race 1 of *Xanthomonas campestris* pv. *vasculorum*, causal agent of gumming disease of sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 22: 380–389.

DOOKUN A., STEAD E.D., AUTREY L.J.C., 2000. Variation among strains of *Xanthomonas campestris* pv. *vasculorum* from Mauritius and other countries based on fatty acid analysis. Systematic and Applied Microbiology 23: 148–155.

MAURITIUS SUGAR INDUSTRY RESEARCH INSTITUTE (MSIRI), 1983. Diseases. *In*: MSIRI Annual Report 1982, p. 34–41. Réduit, Mauritius.

QHOBELA M., CLAFLIN L.E., 1992. Eastern and Southern African strains of *Xanthomonas campestris* pv. *vasculorum* are distinguishable by restriction fragment length polymorphism of DNA and polyacrylamide gel electrophoresis of membrane proteins. Plant Pathology 41: 113–121.

RICAUD C., AUTREY L.J.C., 1989. Gumming disease. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr. and C.G Hughes (Eds), p. 21–38. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SAUMTALLY S., 1996. Genetic variability in *Xanthomonas campestris* pv. *vasculorum* (Cobb) Dye, causal agent of gumming disease of sugarcane. PhD thesis. University of Reading, UK, 191p.

VAUTERIN L., HOSTE B., KERSTERS K., SWINGS J., 1995. Reclassification of *Xanthomonas*. International Journal of Systematic Bacteriology 45: 472–489.

YOUNG J.M., SADDLER G.S., TAKIKAWA Y., DE BOER S.H., VAUTERIN L., GARDAN L., GVOZDYAK R.I., STEAD D.E., 1996. Names of plant pathogenic bacteria 1864–1995. Review of Plant Pathology 75(9): 721–763.

Leaf scald

Philippe Rott and Michael J. Davis

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, Fiji, Ghana, Grenada, Guadeloupe, Guatemala, Guyana, Hawaii, India, Indonesia, Iraq, Jamaica, Japan, Kenya, Madagascar, Malawi, Malaysia, Martinique, Mexico, Morocco, Mauritius, 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, Swaziland, Tahiti, Tanzania, Taiwan, Thailand, Trinidad, 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 parallel to the main veins on leaves. These are generally white to yellow, narrow 'pencil-line' streaks (Figure 1), but they can also be several millimetres wide (Figure 2). Their colour 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. The streaks also tend to widen and become more diffuse on leaves reaching maturity (Figure 2). The fine central line which is characteristic of the disease can, however, always be seen in the centre of the lesion. The widening of lines coincides with the chlorosis or bleaching of the leaf tissue (Figure 3). Chlorosis

or bleaching may affect all the leaves and this discoloration is followed by withering of leaf extremities which curl inwards, giving the shoot or the stalk a tapered aspect (Figure 4). The stalks can be stunted and the leaves wilted, brown and bent at the ends (Figure 5). 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 6). These diseased side shoots may show similar symptoms to those on the main stalk. Longitudinal sections of diseased stalks show reddening of the vessels near the nodes and sometimes in the internodes (Figures 7 and 8). Lysigenous cavities may be observed in severely diseased canes. In susceptible cultivars the whole stalk may die (Figure 9).

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. This acute form often occurs after a period of rain followed by a period of prolonged dry weather, but seems to be 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 (Figure 10). 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, either at harvest, or because the environmental conditions favour 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. Furthermore, the fastidious nature of the pathogen complicates the use of isolation on culture medium as a means of diagnosis. To overcome these problems, serological methods (immunofluorescence, FI ISA, 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; Pan *et al.*, 1999). Because at least three serovars of *X. albilineans* exist, serological variability of the pathogen must be taken into consideration (Rott *et al.*, 1994). Nevertheless, isolation can be a useful

Figure 1. Leaf showing a white 'pencil-line' parallel to the midrib (P. Rott).

Figure 2. Varying intensity of leaf symptoms (P. Rott).

Figure 3. Bleaching of leaves (P. Rott).

Figure 4. Inward curling and drying of leaf tips (P. Rott).

Figure 5. Withering of leaf extremities (P. Rott).

Figure 6. Stalk side shoots showing leaf scald symptoms (P. Rott).

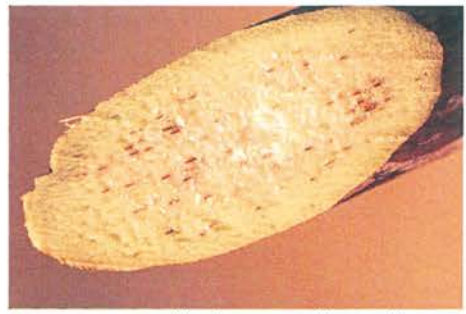


Figure 7. Cut stalk showing red vascular bundles colonized by the pathogen (P. Rott).

Figure 8. Longitudinally cut stalks showing healthy and diseased (red vascular bundles) tissues (P. Rott).

Figure 9. Scalded stools (P. Rott).

Figure 10. Young shoot showing leaf chlorosis and necrosis (P. Rott).

approach. To aid in isolation, selective media were developed (DAVIS *et al.*, 1994). One of the medium 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 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. As a species, *X. albilineans* can be readily distinguished from other xanthomonads (VAN DEN MOOTER and SWINGS, 1990).

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 chronic or acute form 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). The recent outbreak of leaf scald in Florida was closely associated with the appearance of a genetically new strain of the pathogen (DAVIS *et al.*, 1997).

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 recently been reported in Guadeloupe and Mauritius (KLETT and ROTT, 1994).

Host range

Sugarcane is the main host of *X. albilineans* but maize and several grasses (*Brachiaria piligera, Imperata cylindrica, Panicum maximum, Paspalum* sp., *Pennisetum* sp., *Rottboellia cochinchinensis*) have been reported as natural hosts of the pathogen (MARTIN and ROBINSON, 1961; 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 favoured 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).

Economic importance

Leaf scald can cause large yield losses and totally destroys 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 behaviour 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). This technique can easily be applied in quarantine or in other situations with a limited amount 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 volunteer sugarcane shoots and diseased stools, disinfection of cutting tools by brushing 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.

References

AUTREY L.J.C., SAUMTALLY S., DOOKUN A., MÉDAN H., 1995. Studies on variation in the leaf scald pathogen, *Xanthomonas albilineans*. Proceedings International Society of Sugar Cane Technologists Congress 21: 485–497.

COMSTOCK J.C., IREY M., 1992. Detection of the leaf scald pathogen, *Xanthomonas albilineans*, using tissue blot immunoassay, ELISA, and isolation techniques. Plant Disease 76: 1033–1035.

DAVIS M.J., ROTT P., BAUDIN P., DEAN J.L., 1994. Evaluation of selective media and immunoassays for detection of *Xanthomonas albilineans*, causal agent of sugarcane leaf scald disease. Plant Disease 78: 78–82.

DAVIS M.J., ROTT P., WARMUTH C.J., CHATENET M., BAUDIN P., 1997. Intraspecific genomic variation within *Xanthomonas albilineans*, the sugarcane leaf scald pathogen. Phytopathology 87: 316–324.

DAVIS M.J., ROTT P., ASTUA-MONGE G., 1998. Nested, multiplex PCR for detection of both *Clavibacter xyli* subsp. *xyli* and *Xanthomonas albilineans* in sugarcane. *In*: Offered papers abstracts, Vol. 3, 7th International Congress of Plant Pathology, Edinburgh, Scotland, 9–16 August 1998. Abstract 3.3.4. Edinburgh, International Society for Plant Pathology.

KLETT P., ROTT P., 1994. Inoculum sources for the spread of leaf scald disease of sugarcane caused by *Xanthomonas albilineans* in Guadeloupe. Journal of Phytopathology 142: 283–291.

MARTIN J.P., ROBINSON P.E., 1961. Leaf scald. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 79–107. Amsterdam, The Netherlands, Elsevier Publishing Company.

PAN Y.B., GRISHAM M.P., BURNER D.M., LEGENDRE B.L., WEI Q., 1999. Development of polymerase chain reaction primers highly specific for *Xanthomonas albilineans*, the causal bacterium of sugarcane leaf scald. Plant Disease 83: 218–222.

RICAUD C., RYAN C.C., 1989. Leaf scald. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 39–58. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

ROTT P., DAVIS M.J., BAUDIN P., 1994. Serological variability in *Xanthomonas albilineans*, causal agent of leaf scald disease of sugarcane. Plant Pathology 43: 344–349.

STEINDL D.R.L., 1972. The elimination of leaf scald from infected planting material. Proceedings International Society of Sugar Cane Technologists Congress 14: 925–929.

VAN DEN MOOTER M., SWINGS J., 1990. Numerical analysis of 295 phenotypic features of 266 *Xanthomonas* strains and related strains and an improved taxonomy of the genus. International Journal of Systematic Bacteriology 40: 348–369.

Mottled stripe

A. Salem Saumtally

Cause

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

Geographical distribution

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

Symptoms

Mottled stripe is characterized by fine narrow stripes running on both sides of the midrib and more commonly abundant at the base of the leaf. The colour of the stripes is not uniform and consists of parts that are white, cream and red (Figure 1). However the red colour predominates and a heavily infected leaf has a distinct reddish appearance. At an early stage of infection, only one stripe may be present on each leaf. In advanced infection, several stripes are present and they tend to fuse towards the base of the leaf with mottled red and white bands (Figure 2). The stripes, which are also formed on the midrib, do not enter the leaf sheath and are more abundant on older leaves. Some stripes may be short (5–10 cm) whereas some may extend over 1 m. The width of a stripe is about 2 mm and stays narrow over the whole of its length with a sharp margin.

Diagnosis

Mottled stripe produces symptoms similar to red stripe and the two diseases are often confused. In contrast to red stripe (*Acidovorax avenae* subsp.

avenae), mottled stripe is not bright red over the whole length of the stripe, it is not accompanied by a bacterial exudation visible on the surface of the leaf as a pellicle and does not cause a top rot.

The bacterium was transferred from the genus *Pseudomonas* to *Herbaspirillum* on the basis of its habitat, DNA-rRNA and DNA-DNA hybridization studies, as well as substrate utilization (BALDANI *et al.*, 1996). BRADBURY (1967) summarized the biochemical, physiological and morphological properties of the bacterium. It is Gram-negative, rod-shaped (0.8 × 2.5 µm), with 4–6 polar flagella. 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. In contrast to *A. avenae* subsp. *avenae*, malonate is utilized and lipid (Tween 80) is not broken down. Starch hydrolysis is variable. Acid is produced oxidatively from arabinose, fructose, galactose, glucose, glycerol and sorbitol, but not from cellobiose, maltose, meso-inositol, raffinose, salicin or sucrose.

Oligonucleotide probes based on the partial sequences of the 23S rRNA of *H. rubrisubalbicans* have been constructed for its diagnosis (BALDANI *et al.*, 1996). The bacterium can be identified by thin layer chromatography (KHAN and MATSUYAMA, 1998). The *in situ* localization of the bacterium in *Sorghum bicolor* was effected using *H. rubrisubalbicans*-specific antibodies and immunogold labelling (JAMES *et al.*, 1997).

Strains of the pathogen

No race variation has been reported.

Transmission

The major route of transmission of the bacterium from field to field is by wind and rain. Although stripes extend to the base of the leaf blade, the pathogen has not been found to invade the leaf sheath and cause a systemic infection. Consequently, transmission by cuttings is not known. OLIVARES *et al.* (1997) found that on inoculation into the apex of a susceptible variety, the xylem-conducting elements in the diseased regions were filled with the bacterium. The pathogen was abundant in the intercellular spaces of the mesophyll tissue adjacent to the xylem as well as the sub-stomatal cavities. In a resistant variety, xylem vessels contained encapsulated bacteria.

Host range

Sugarcane is the main host of *H. rubrisubalbicans* but it has also been reported on sorghum species as the bacterial leaf stripe of sorghum (HALE and WILKIE, 1972).

Epidemiology

The pathogen develops under mild temperatures and humid conditions. STEINDL and EDGERTON (1964) observed an abundance of foliar stripes during summer months. The bacterium is considered to be an endophytic diazotroph (nitrogen-fixing). It has been found to colonize the roots, stems and predominantly, the leaves of sugarcane (OLIVARES *et al.*, 1996) but there is no report on sett transmission. On soil inoculation, the population of the bacterium declines rapidly. However, in the presence of sorghum, it colonizes the roots and multiplies within the plant tissues (OLIVARES *et al.*, 1996).

Economic importance

Mottled stripe is considered a mild pathogen of sugarcane and no economic loss has been reported. The use of *H. rubrisubalbicans* in biological nitrogen-fixation has been discussed by DOBEREINER (1992) and MUTHUKUMARASAMY *et al.* (1994).

Control

No control measure has been advocated for the disease.

Figure 1. Narrow cream and reddish stripes on both sides of the midrib (MSIRI).

Figure 2. Narrow cream and reddish foliar stripes (MSIRI).

References

BALDANI J.I., POT B., KIRCHHOF G., FALSEN E., BALDANI V.L.D., OLIVARES F.L., HOSTE B., KERSTERS K., HARTMANN A., GILLIS M., DOBEREINER J., 1996. Emended description of *Herbaspirillum*; inclusion of [*Pseudomonas*] *rubrisubalbicans*, a mild plant pathogen, as *Herbaspirillum rubrisubalbicans* comb. nov.; and classification of a group of clinical isolates (EF group 1) as *Herbaspirillum* species 3. International Journal of Systematic Bacteriology 46: 802–810.

BRADBURY J.F., 1967. *Pseudomonas rubrisubalbicans*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 128. Kew, UK, Commonwealth Mycological Institute.

DOBEREINER J., 1992. Recent changes in concepts of plant bacteria interactions: endophytic N_2-fixing bacteria. Ciencia-e-Cultura 44: 310–313.

HALE C.N., WILKIE J.P., 1972. Bacterial leaf stripe of sorghum in New Zealand caused by *Pseudomonas rubrisubalbicans*. New Zealand Journal of Agricultural Research 15: 457–460.

JAMES E.K., OLIVARES F.L., BALDANI J.I., DOBEREINER J., 1997. *Herbaspirillum*, an endophytic diazotroph colonizing vascular tissue in leaves of *Sorghum bicolor* L. Moench. Journal of Experimental Botany 48: 785–797.

KHAN A.A., MATSUYAMA N., 1998. Rapid identification of phytopathogenic bacteria by TLC. Journal of the Faculty of Agriculture, Kyushu University 42: 3–4.

MUTHUKUMARASAMY R., REVATHI G., SOLAYAPPAN A.R., 1994. Biofertilizers – a supplement or substitute for chemical nitrogen for sugarcane crop. Cooperative Sugar 25: 287–290.

OLIVARES F.L., BALDANI V.L.D., REIS V.M., BALDANI J.I., DOBEREINER J., 1996. Occurrence of the endophytic diazotrophs *Herbaspirillum* spp. in roots, stems, and leaves, predominantly of Gramineae. Biology and Fertility of Soils 21: 197–200.

OLIVARES F.L., JAMES E.K., BALDANI J.I., DOBEREINER J., 1997. Infection of mottled stripe disease-susceptible and resistant sugar cane varieties by the endophytic diazotroph *Herbaspirillum*. New Phytopathologist 135: 723–737.

STEINDL D.R.L., EDGERTON C.W., 1964. Mottled stripe. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 12–16. Amsterdam, The Netherlands, Elsevier Publishing Company.

Ratoon stunting

Michael J. Davis and Roger A. Bailey

Cause

Clavibacter xyli subsp. *xyli* Davis *et al.* 1984, bacterium (Davis *et al.*, 1984).

Geographical distribution

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

Symptoms

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 the accompanying production of thinner and shorter stalks and sometimes a reduction in the number of stalks when the disease is severe (Figure 1). In stubble or ratoon crops, diseased plants are slower to initiate growth, and death of individual plants of extremely susceptible varieties may occur. Some highly susceptible varieties may show wilting under moisture stress and develop a necrosis of leaves at the tips and margins. 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, but these symptoms are often ephemeral (Figures 2 and 3). They appear as yellow to 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. The vascular tissue of healthy stalks can sometimes discolour, and healthy controls may be needed for comparison.

Diagnosis

RSD is most often detected using laboratory techniques because of the lack of reliable diagnostic symptoms in sugarcane. The pathogen is a small, xylem-inhabiting, coryneform bacterium that may be detected in xylem sap extracts using phase-contrast or dark-field microscopy (×1000) (Figure 4). The effectiveness of microscopic examination depends largely on pathogen population size. Pathogen populations vary among varieties and are greatest in the basal portion of mature stalks during the later part of the growing season.

The pathogen is extremely fastidious in its nutritional requirements and can only be grown in axenic culture on special media such as the SC medium (Davis *et al.*, 1980). 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.

The bacteria can be detected with various immunochemical tests. 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 colonized vascular bundles in stalk cross-sections. EB-EIA is a modified ELISA procedure for analysis of vascular sap extracts.

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 and may provide greater sensitivity for detection than other molecular means (Davis *et al.*, 1998; Fegan *et al.*, 1998; Pan *et al.*, 1998).

Strains of the pathogen

There is no evidence so far for the existence of strains within *C. xyli* subsp. *xyli*.

Transmission

The pathogen has been found only in sugarcane in nature and has no known insect vectors. Systemic infection of the xylem takes place through wounds. The pathogen can be mechanically transmitted on the blades of equipment used to cultivate and harvest crops and can be spread by propagation with

Figure 1. RSD-infected stools (left) next to healthy plants with normal growth (right) (J. Hoy).

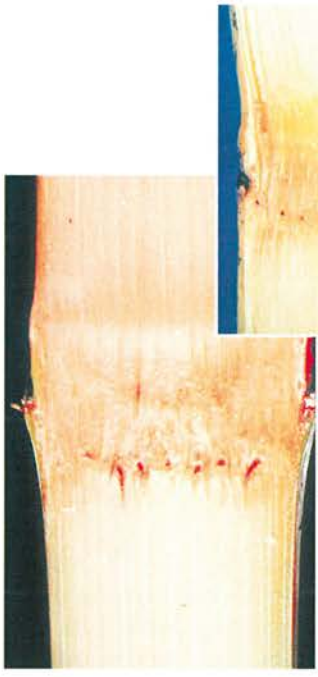


Figure 2. Reddish discoloration of vascular bundles at the nodes (BSES).

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

Figure 4. RSD bacterial cells observed by negative-stain transmission electron microscopy (R.H. Lawson).

infected cuttings. It is not seed transmitted. The pathogen can remain viable and infectious for several months apparently in either moribund plant debris or the soil itself, contributing to the persistence of ratoon stunting disease in areas where the disease is common (BAILEY and TOUGH, 1992). Volunteer regrowth from former infected crops provides a common source of infection of new plantings.

Host range

Sugarcane is the only known natural host. Numerous grasses have been determined to be hosts after experimental inoculation, including *Zea mays, Sorghum* spp., *Brachiaria mutica, Brachiaria miliiformis, Chloris gayana, Cynodon dactylon, Echinochloa colonum, Imperata cylindrica, Panicum maximum, Pennisetum purpureum* and *Rhynchelytrum repens* (GILLASPIE and TEAKLE, 1989).

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. Moisture stress, as a result of either drought or waterlogging, can increase yield reduction due to the disease. A synergy between sugarcane mosaic disease and ratoon stunting disease 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, yet paradoxically few other diseases of sugarcane are less conspicuous. Yield losses have frequently been estimated at 5 to 10% overall. Losses may be negligible in some years but in other years they may be 30% or greater. The importance of RSD is largely dependent on two factors, varietal susceptibility 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).

Control

Planting healthy cane can be used to control the disease. Sanitation is important in keeping healthy cane from becoming infected, since the pathogen is easily transmitted mechanically. Cutting implements can be disinfected by thorough washing and application of a disinfectant such as 5–15% Lysol (a neutralized cresylic acid solution), 1% Dettol (a mixture including pine oil and para-chlorometaxylenol), 50–70% ethanol, or 0.1% Mirrol or Roccal (solutions of quarternary ammonium compounds). Seed cane can be monitored for freedom from the disease using appropriate diagnostic techniques. Continued vigilance in the selection of seed cane over several years has resulted in a reduction in the incidence of ratoon stunting disease in both plantings for seed cane production and commercial crops.

Seed cane can be heat-treated to eliminate the pathogen (GILLASPIE and DAVIS, 1992; GILLASPIE and TEAKLE, 1989; STEINDL, 1961). Hot water, hot air, moist air and aerated steam treatments have been used. Hot water treatment at *c.* 50°C for 2–3 h has been the most commonly used method. The two major problems limiting the effectiveness of heat-treatment, in addition to the expense, are reductions in germination and lack of complete control. Consequently, heat-treatment is often used to establish 'pathogen-free' nurseries that are then used to supply planting material for commercial fields.

Although immunity to infection is not known to occur in sugarcane, substantial resistance to injury has been found in some varieties. The TB-EIA and EB-EIA techniques have been incorporated into sugarcane breeding programmes in Florida (DAVIS *et al.*, 1994) and in Australia (CROFT *et al.*, 1994), respectively, to permit large-scale screening for resistance to the disease based on estimates of disease incidence and the extent of colonization by the pathogen. It has been suggested that control of the disease might best be obtained by disregarding the effects of the disease on 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 such programmes are the exception and control is mainly achieved through traditional practices.

References

BAILEY R.A., MCFARLANE S.A., 1999. The status of ratoon stunting disease of sugarcane in southern and central Africa. Proceedings International Society of Sugar Cane Technologists Congress 23: 338–346.

BAILEY R.A., TOUGH S.A., 1992. Ratoon stunting disease: survival of *Clavibacter xyli* subsp. *xyli*, in field soil and its spread to newly planted sugarcane. Proceedings South African Sugar Technologists' Association Annual Congress 66: 75–77.

CROFT B.J., GREET A.D., LEAMAN T.M., TEAKLE D.S, 1994. RSD diagnosis and varietal resistance screening in sugarcane using the EB-EIA technique. Proceedings Australian Society of Sugar Cane Technologists Conference 14: 143–151.

DAVIS M.J., DEAN J.L., MILLER J.D., SHINE J.M. JR, 1994. A method to screen for resistance to ratoon stunting disease of sugarcane. Sugar Cane 6: 9–16.

DAVIS M.J., GILLASPIE A.G. JR, HARRIS R.W., LAWSON R.H., 1980. Ratoon stunting disease of sugarcane: Isolation of the causal bacterium. Science 210: 1365–1367.

DAVIS M.J., GILLASPIE A.G. JR, VIDAVER A.K., HARRIS R.H., 1984. *Clavibacter*: A new genus containing some phytopathogenic coryneform bacteria, including *Clavibacter xyli* subsp. *xyli* sp. nov., subsp. nov. and *Clavibacter xyli* subsp. *cynodontis* subsp. nov., pathogens that cause ratoon stunting disease of sugarcane and Bermudagrass stunting disease. International Journal of Systematic Bacteriology 34: 107–117.

DAVIS M.J., ROTT P., ASTUA-MONGE G., 1998. Nested, multiplex PCR for detection of both *Clavibacter xyli* subsp. *xyli* and *Xanthomonas albilineans* in sugarcane. *In*: Offered papers abstracts, Vol. 3, 7th International Congress of Plant Pathology, Edinburgh, Scotland 9–16 August 1998. Abstract 3.3.4. Edinburgh, International Society for Plant Pathology.

DEAN J.L, DAVIS M.J., 1990. Losses caused by ratoon stunting disease of sugarcane in Florida. Journal of the American Society of Sugarcane Technologists 10: 66–72.

FEGAN M., CROFT B.J., TEAKLE D.S., HAYWARD A.C., SMITH G.R., 1998. Sensitive and specific detection of *Clavibacter xyli* subsp. *xyli*, causal agent of ratoon stunting disease of sugarcane, with a polymerase chain reaction-based assay. Plant Pathology 47: 495–504.

GILLASPIE A.G. JR., DAVIS M.J., 1992. Ratoon stunting of sugarcane. *In*: Plant Diseases of International Importance. Diseases of Sugar, Forest, and Plantation Crops, Vol. 4. A.N. Mukhopadhyay, J. Kamar, H.S. Chaube and U.S. Singh (Eds), p. 41–61. New Jersey, USA, Prentice Hall.

GILLASPIE A.G. JR., TEAKLE D.S., 1989. Ratoon stunting disease. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 59–80. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

PAN Y.B., GRISHAM M.P., BURNER D.M., DAMANN K.E. JR, WEI Q., 1998. A polymerase chain reaction protocol for the detection of *Clavibacter xylii* subsp. *xyli*, the causal bacterium of sugarcane ratoon stunting disease. Plant Disease 82: 285–290.

STEINDL D.R.L., 1961. Ratoon stunting disease. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 433–459. Amsterdam, The Netherlands, Elsevier Publishing Company.

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, encompassing the entire length of the leaves, appear on some cultivars. The lesions turn red to reddish-brown and may develop whitish to straw-coloured centres (Figure 1). These bleached centres are quite prominent on some cultivars. In severe infections, lesions coalesce, forming large discoloured necrotic blotches resulting in death of the leaf portions distal to the affected areas. Older leaves harbouring 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 cultivars. 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 at 25–28°C. The red streak inciting strains are rather slow to produce levan, compared to the strains causing canker and blight on stone fruits and wheat, respectively (RAHIMIAN, 1995).

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 toxin deficient variant capable of inciting the disease in sugarcane has, thus far, been isolated. Differences in electrophoretic profiles of cell proteins among strains isolated from sugarcane in the same and different areas of Iran have been noted (Rahimian, unpublished). Despite such heterogeneity, no noticeable difference in pathogenicity or virulence appears to exist between strains from sugarcane. Strains producing red streak are as a group distinct from those inciting canker in stone fruit trees or blight in barley and wheat (RAHIMIAN, 1995).

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 on sugarcane strains of *P. syringae* pv. *syringae* have been carried out. The causal bacterium has not been isolated from wheat or barley plants grown adjacent to infected sugarcane nor has it been possible to infect these 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 in cool wet springs and autumns and especially during periods of high or frequent rainfalls. Hot, dry weather effectively checks its development and spread in the field and under glasshouse conditions.

Economic importance

No estimate of yield losses inflicted by the red streak disease is available.

Control

Cultivation of resistant or tolerant cultivars is the most economical and promising means for the control of red streak. Reducing plant density through proper row spacing tends to reduce the incidence and severity of the disease (RAHIMIAN, 1995; Rahimian, unpublished). Application of some bactericides, including streptomycin, has given some levels of control (YANO *et al.*, 1986; Rahimian, unpublished). Use of healthy cuttings is also recommended.

References

FUKUCHI N., ISOGAI A., YAMASHITA S., SUYAMA K., TAKEMOTO J.Y., SUZUKI A., 1990. Structure of phytotoxin syringomycin produced by a sugar cane isolate of *Pseudomonas syringae* pv. *syringae*. Tetrahedron Letters 31: 1589–1592.

RAHIMIAN H., 1995. The occurrence of bacterial red streak of sugarcane caused by *Pseudomonas syringae* pv. *syringae* in Iran. Journal of Phytopathology 143: 321–324.

YANO H., SUYAMA K., FUJII H., 1986. Causal bacteria isolated from red-streak symptom of sugarcane leaves, and its control. Journal of Okinawa Agriculture 21: 24–28.

Figure 1. Reddish-brown leaf streaks (H. Rahimian).

Red stripe (top rot)

Philippe Rott and Michael J. Davis

Cause

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

Geographical distribution

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, Guadeloupe, Guam, Guatemala, Guyana, Hawaii, Honduras, India, Indonesia, Iran, Iraq, 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, Uganda, Uruguay, USA, Venezuela, Vietnam.

Symptoms

The disease is characterized by two forms: leaf stripe and top rot. These can occur individually or simultaneously (MARTIN and WISMER, 1961 and 1989).

Leaf stripes are often more prevalent in young, 4–6-months-old cane in both plant and ratoon crops. They first 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 colour, and finally become maroon or dark red. They are 0.5 to 4 mm wide, and from several centimetres long to the entire length of the leaf blade (Figures 1 and 2). 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.

Top rot can arise from leaf infections or directly from stem and bud infection. The pathogen may attack the unrolled leaves of the leaf spindle causing rotting of the leaf spindle (Figure 3). 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 (Figure 4). Affected internodes may exhibit sunken lesions which are at first water-soaked in appearance and later brown to red in colour. Reddish-brown disoloration may also develop within internodes and as the rotting progresses, large cavities may form. Upper internodes are usually affected. 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 odour 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 usually die (Figure 5).

Unusual symptoms caused by *A. avenae* subsp. *avenae* have been described in specific cultivars. In Central America and Mexico, cultivar B4362 showed red stalk markings in the region of the root primordia, and shrunken watery nodes with several longitudinal cracks along the affected internodes (FORS, 1978 and 1980, cited by MARTIN and WISMER, 1989). In Australia, cultivar Trojan was affected by a rot that originated some distance from the growing point 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).

Diagnosis

The pathogen can be easily isolated from young lesions and the bacteria grown on various culture media, such as modified Wilbrink's medium used to isolate *Xanthomonas albilineans*, causal agent of sugarcane leaf scald. *Acidovorax avenae* subsp. *avenae* is a Gram-negative bacterium, rod shaped (0.7 × 1.6 µm), and motile with a single polar flagellum. Colonies on YDC medium are white-cream with tan to brown centres, convex, smooth, 2–3 mm diameter after 3 days at 30°C. Old colonies 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-ß-hydroxybutyrate (PHB) granules intracellularly when grown on 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). The pathogen can also be identified by PCR using specific primers (SONG *et al.*, 1997).

Strains of the pathogen

No significant variation of pathogenicity between isolates of *A. avenae* subsp. *avenae* has been found so far (RAMUNDO and CLAFLIN, 1990). However, symptom expression of sugarcane cultivars can vary with the isolate of the pathogen (ALMEIDA *et al.*, 1988).

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 favoured 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 cuttings.

Host range

The bacteria can naturally infect different cultivated gramineaceous plants such as maize, sorghum and millet. However, these hosts play an insignificant role in the epidemiology of the disease in sugarcane. The pathogen was also isolated in Mauritius from several grasses such as *Paspalum nutans*. Strains of *A. avenae* subsp. *avenae* appeared to be more virulent to sweet corn than to maize or sugarcane, and only weakly virulent or avirulent to oats (HU *et al.*, 1997).

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 favours disease development in Queensland (MARTIN and WISMER, 1989).

Economic importance

The disease can cause significant economic losses when susceptible cultivars are affected. The top-rot form of the disease is the most economically damaging. However, clones that are highly susceptible to the disease are normally eliminated during the varietal selection process.

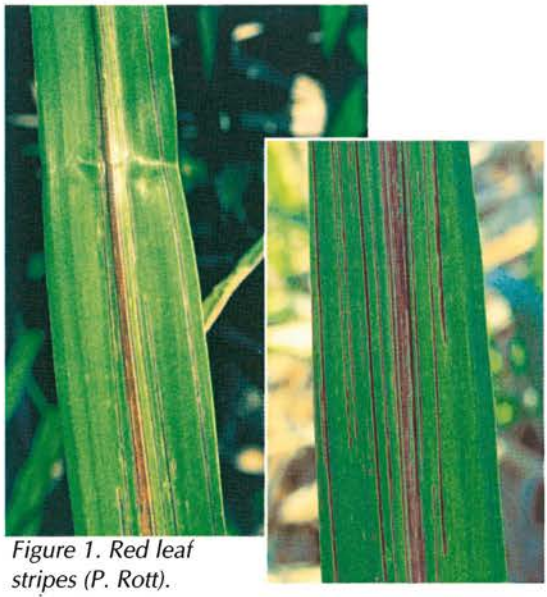


Figure 1. Red leaf stripes (P. Rott).

Figure 2. Maroon leaf stripes (ISSCT).

Figure 3. Top rotting of the leaf spindle (ISSCT).

Figure 4. Stool affected by red stripe (ISSCT).

Figure 5. Sugarcane row affected by the top rot form of the disease (ISSCT).

Control

Use of resistant varieties is the most effective means of control and, therefore, susceptible cultivars should be eliminated in breeding programmes. Top rot can be reduced considerably by altering the planting date of moderately resistant cultivars to avoid periods when infection of young plants is more likely.

References

ALMEIDA I.M., RODRIGUES, NETO J., CARDELLI M. A., 1988. Reaction of sugarcane cultivars to red stripe caused by *Pseudomonas rubrilineans*. Summa Phytopathologica 14: 191–195.

CROFT B.J, RYAN C.C., KINGSTON G., 1979. Stem rot of Trojan caused by *Pseudomonas rubrilineans* (Lee *et al.*) Stapp. Sugarcane Pathologists' Newsletter 23: 17–18.

HU F.P., YOUNG J.M., TRIGGS C.M., WILKIE J.P., 1997. Pathogenic relationships of the subspecies of *Acidovorax avenae*. Australasian Plant Pathology 26: 227–238.

MARTIN J.P., WISMER C.A., 1961. Red stripe. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 109–126. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., WISMER C.A., (revised by C.C. RYAN) 1989. Red stripe. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 81–95. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

RAMUNDO B.A., CLAFLIN L.E., 1990. Demonstration of synonymy between the plant pathogens *Pseudomonas avenae* and *Pseudomonas rubrilineans*. Journal of General Microbiology 136: 2029–2033.

SADDLER G.S., 1994. *Acidovorax avenae* subsp. *avenae*. IMI Descriptions of fungi and bacteria No. 1211. Mycopathologia 128: 41–43.

SONG W.Y., HATZILOUKAS E., KIM H.M., SCHAAD N.W., 1997. Development of PCR primers for detection of *Pseudomonas avenae*. Phytopathology 87: S92–S93.

WILLEMS A., GOOR M., THIELEMANS S., GILLIS M., KERSTERS K., LEY J. DE, 1992. Transfer of several phytopathogenic *Pseudomonas* species to *Acidovorax* as *Acidovorax avenae* subsp. *avenae* subsp. nov., comb. nov., *Acidovorax avenae* subsp. *citrulli*, *Acidovorax avenae* subsp. *cattleyae*, and *Acidovorax konjaci*. International Journal of Systematic Bacteriology 42: 107–119.

Spindle rot

A.S. Patil

Cause

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

Geographical distribution

India.

Symptoms

The disease was first described by PATIL and HAPASE (1994). Symptoms appear initially on the leaf spindle as light to dark-brown elongated areas which can be several centimetres long (Figure 1). The tissue softens in some parts and subsequently rots. At an advanced stage, dark-brown to chocolate-brown soft rotting tissues become prominent (Figure 2). The spindle withers and dries from the tip downwards to the affected portion (Figure 3). The infection is mainly confined to the youngest leaf spindle which cannot be easily pulled out from the growing point (Figure 4). The rotted parts have no unpleasant odour in contrast to the top-rot phase of red stripe. Side shoot formation in the uppermost buds does not occur. Occasionally, other pathogens such as *Gibberella fujikuroi,* causal agent of pokkah boeng, develop in the diseased tissues (Figure 5). Spindle rot is not a truly vascular disease and does not spread to the leaf sheath, apical meristem or stalk. It develops mostly on young canes of about 4–5 months and rarely on mature plants.

Symptoms of spindle rot are not typical of those of red stripe or top rot (RYAN, 1989) caused by *Acidovorax avenae* subsp. *avenae.*

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 ×100 or ×400.

The bacterium can be isolated on King's B medium (2% protease peptone, 1% glycerol, 0.15% $MgSO_4$ $7H_2O$, 0.15% K_2HPO_4, 2% Bacto agar at 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 at pH 6.8–7.0) can be used. Single colonies appear within 72–96 h at 30°C. They are translucent, smooth, raised with entire margins and produce a diffused pigmentation. The morphological, cultural and biochemical characteristics of the pathogen are similar to those described for *Acidovorax avenae* subsp. *avenae*.

The bacterium is aerobic, rod-shaped measuring 1.6 × 0.7 µm, motile with one polar flagellum, and Gram-negative. Gelatin is liquefied, nitrate is reduced to nitrite. Acid, but not gas, is produced from glucose, fructose, glycerol and galactose but not from lactose, sucrose, raffinose, maltose or cellobiose. Citrate test is negative. Hydrolysis of starch is weak. Abundant growth occurs between a pH range of 5.5 and 7.2 on King's B agar medium at 30°C. Growth does not occur at 40°C. Nutrient agar does not support good growth of the pathogen.

A pathogenicity test can be performed by an artificial inoculation method as suggested by CHINEA *et al.* (1978). Forty five days after inoculation, typical symptoms identical to those of spindle rot develop, but only on spindle leaves.

The name *Pseudomonas rubrilineans* pv. *spindulifoliens* was tentatively given to the bacterium by PATIL and HAPASE (1994), but has not been validated by the Sub-Committee on Taxonomy of Plant Pathogenic Bacteria (YOUNG *et al.*, 1996). The taxonomic position of the spindle rot pathogen needs further clarification. A culture has been deposited at the International Collection of Micro-organisms from Plants (ICMP), DSIR, New Zealand under accession No. ICMP 9562.

Strains of the pathogen

Unknown.

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 and no alternative hosts are known. The bacterium has been observed on the major commercial varieties in India such as CoC671, Co740, Co7219, Co8014 and Co86032.

Figure 1. Newly developed spindle leaf showing rotted tissues (A.S. Patil).

Figure 2. Brown elongated lesion on spindle leaf (A.S. Patil).

Figure 3. Withered spindle leaf (A.S. Patil).

Figure 4. Spindle leaf killed by spindle rot (A.S. Patil).

Figure 5. Simultaneous infections of spindle rot and pokkah boeng (A.S. Patil).

Epidemiology

Spindle rot is not prevalent throughout the season. It seems to be more severe in areas with a cool climate and in locations with significant variations in climatic conditions. Infection occurs rarely in summer.

Economic importance

Although no economic loss has been observed, its occurrence in several areas in India indicates that it could become a disease of economic importance.

Control

No information is available on disease management.

References

CHINEA A., MARQUEZ F.R., CANADA A., 1978. Methods for artificial inoculation of the causal organism of red stripe in sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 16: 337–345.

KING E.O., WARD M.K., RANEY D.E., 1954. Two simple media for the demonstration of pyocynin and fluorescin. Journal of Laboratory Clinical Medicine 44: 301.

PATIL A.S., HAPASE D.G., 1994. Spindle rot – A new bacterial disease of sugarcane in India. *In*: Current Trends in Sugarcane Pathology. G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin, V.P. Agnihotri and C.T. Chen (Eds), p. 85–94. Delhi, India, International Books and Periodicals Supply Service.

RYAN C.C., 1989. Red stripe. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G., Gillaspie Jr and C.G. Hughes (Eds), p. 81–95. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

YOUNG J.M., SADDLER G.S., TAKIKAWA Y., DE BOER S.H., VAUTERIN L., GARDAN L., GVOZDYAK R.I., STEAD D.E., 1996. Names of plant pathogenic bacteria 1864–1995. Review of Plant Pathology 75(9): 721–763.

Diseases caused by fungi

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 millimetres 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 odour. Diagnosis is by symptoms and by inoculation of susceptible varieties such as Q63.

Strains of the pathogen

No information is available.

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 favoured by hot dry conditions and generally only affects young plant crops or less frequently young ratoon crops. 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.

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 this disease.

References

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 and C.A. Wismer (Eds), p. 94–98. Amsterdam, The Netherlands, Elsevier Publishing Company.

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

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

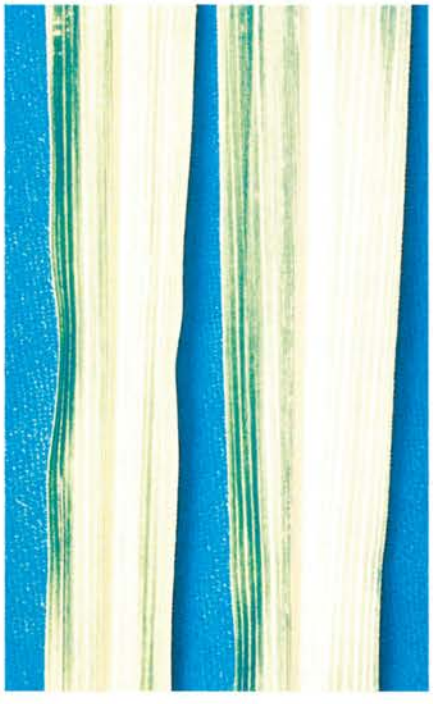


Figure 3. Narrow white leaf stripes along the vascular bundles (B.J. Croft).

Banded sclerotial disease

Claude Ricaud

Cause

Thanatephorus sasakii (Shirai) C.C. Tu & Kimborough, or *T. cucumeris* (A.B. Frank) Donk, basidiomycete fungi (MORDUE, 1974; SIVANESAN and WALLER, 1986).

Geographical distribution

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 (Figures 1 and 2). These bands are initially dirty green, then brownish and finally straw-coloured with a well-defined reddish-brown border. Similar symptoms may occasionally occur on the sheaths (Figures 1 and 3). 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 coloured when immature, but turning dark brown to black later, are frequently observed on the surface of diseased areas (ABBOTT and TAKASHI MATSUMOTO, 1964).

Diagnosis

The fungus can be recognized by the presence of sclerotia on the disease patches and can be easily isolated on potato dextrose agar. Specific characteristics of the fungus have been described by MORDUE (1974).

Strains of the pathogen

Thanatephorus cucumeris is a cosmopolitan and complex species with variable parasitic patterns, morphological and physiological characteristics, host range and specificity. *Thanatephorus sasakii* is considered to be a form of the same fungus; it has been reported as causing a disease in maize with symptoms similar to the sugarcane disease (BARUAH and LAL, 1981).

Figure 1. Irregularly shaped patches across the blades and sheaths of older leaves (ISSCT).

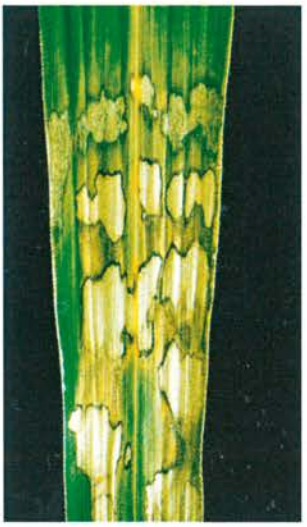


Figure 2. Irregularly shaped patches across the leaf blade (ISSCT).

Figure 3. Irregularly shaped patches across the leaf sheath (Taiwan Sugar).

Transmission

The fungus is a soil inhabitant and infection occurs by leaf contact with the soil or with an infected weed.

Host range

The pathogen infects different grass weeds, especially Bermuda grass (*Cynodon dactylon*) which is often found at the edges of sugarcane fields.

Epidemiology

The fungus survives as sclerotia in soil, on host residues and on grass weeds. It spreads to sugarcane only during periods of high humidity (ABBOTT and TAKASHI MATSUMOTO, 1964).

Economic importance

The disease has usually been considered of little economic importance since infection is confined to older leaves. Differences in varietal susceptibility have recently been reported in India, with some yield reductions in severely affected clones (SINGH *et al.*, 1994).

Control

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

References

ABBOTT E.V., TAKASHI MATSUMOTO, 1964. Banded sclerotial disease. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 19–20. Amsterdam, The Netherlands, Elsevier Publishing Company.

BARUAH P., LAL S., 1981. Host range of *Rhizoctonia solani* f. sp. *sasakii*, the incitant of banded sclerotial disease of maize. Indian Phytopathology 34(4): 494–496.

MORDUE J.E.M., 1974. *Thanatephorus cucumeris*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 406. Kew, UK, Commonwealth Mycological Institute.

SINGH G.P., ATUL SINGH, TIWARI T.N., 1994. Physiological changes associated with banded sclerotial disease. Sugar Cane 6: 17–20.

SIVANESAN A., WALLER J.M., 1986. *Thanatephorus* Donk. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 12–13. Slough, UK, CAB International.

Black leaf spot (tar spot)

Claude Ricaud

Cause

Phyllachora sacchari Hennings, ascomycete fungus (ANAHOSUR and SIVANESAN, 1978).

Geographical distribution

Argentina, Bangladesh, Borneo, India, Italy (Sicily), Indonesia, Malaysia, Myanmar, Nigeria, Pakistan, Papua New Guinea, Philippines, Solomon Islands, Taiwan.

Symptoms

Initial symptoms are small raised black spots (tar spots) on the blades of the lower leaves (Figure 1). Later, necrotic areas develop around the spots. These coalesce and large areas of the lamina may become necrotic and straw-coloured. Under favourable conditions infection may develop on the younger leaves and the older leaves dry up prematurely (KOIKE, 1988).

Diagnosis

Examination of stromata on the spots reveal the presence of characteristic fruiting bodies of the fungus: perithecia, asci and ascospores. Perithecia, up to five per stroma, measure 90–155 × 150–900 µm. Asci are 80–130 × 13–20 µm, and ascospores 16–25 × 8–11 µm. Additional characteristics have been described by ANAHOSUR and SIVANESAN (1978).

Host range and strains of the pathogen

The fungus or its closely related species infects several sorghum species and some grasses. Severe infection has been reported in some sorghum lines, varieties and hybrids (ANAHOSUR and SIVANESAN, 1978).

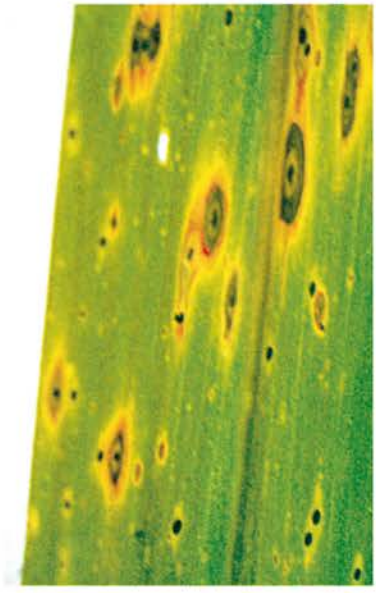


Figure 1. Leaf with tar spots and necrotic areas (H. Koike).

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 favourable to infection.

Economic importance and control

The disease is of minor economic importance in sugarcane and does not require any control measure.

References

ANAHOSUR K.H., SIVANESAN A., 1978. *Phyllachora sacchari*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 588. Kew, UK, Commonwealth Mycological Institute.

KOIKE H., 1988. Sugar-Cane Diseases. A guide for field identification. Rome, Italy, Food and Agriculture Organization of the United Nations, 127p.

Brown spot

A. Salem Saumtally and Sydney Sullivan

Cause

Cercospora longipes E.J. Butler, deuteromycete fungus.

Geographical distribution

Argentina, Bangladesh, Brazil, Burundi, Cameroon, Colombia, Cuba, Democratic Republic of the Congo, Ethiopia, 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, Swaziland, Taiwan, Tanzania, Thailand, Uganda, USA (Florida, Louisiana, Texas), Venezuela, Zimbabwe.

Symptoms

As the name indicates, brown spot causes reddish-brown to dark-brown spots on sugarcane leaves. The lesions vary in size from pinpoint to about 3 × 15 mm. The spots are oval in shape, often surrounded by a yellow halo and are equally visible on both sides of the leaf (Figure 1). Under conditions that are not well defined, the lesion and halo may be as large as 10–15 × 30–40 mm (Figure 2). The long axis of the spot is usually parallel to the midrib. Leaf spots are present on the older leaves and are generally well distributed over the entire surface. The percentage leaf area infected depends on the resistance of the 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. This produces the 'fired' appearance of affected fields, late in the season (ABBOTT, 1964). Lesions can also be observed on the leaf sheath (EDGERTON, 1955; NOLLA, 1965; HSIEH and TSENG, 1978).

Diagnosis

Although the symptoms may be confused with other leaf diseases such as brown stripe, eye spot or ring spot, brown spot can be distinguished by the 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 towards the leaf tip from the lesions. In contrast, ring spot is irregular in shape, 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 (Figure 3). The spores are more abundant on the lower leaf surface.

A description of *Cercospora longipes* is given by MULDER and HOLLIDAY (1974). It belongs to the subdivision Deuteromycotina. 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, 20–200 × 3–8 µm, and are hyaline, broader at the base, straight or curved at the tip and distinctly multi-septate (3–11).

In culture, conidia production is at a maximum at 22°C (HSIEH and TSENG, 1978). 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.

Strains of the pathogen

In Taiwan, a new race of *C. longipes* was identified on the basis of its pathogenicity (HSIEH and TSENG, 1978).

Transmission

Spores of the pathogen are wind-borne. The fungus can also be transported on infected leaves adhering to stalks or cuttings as well as on leaf trash according to ABBOTT (1964). Survival on young sugarcane regrowth has been observed, the disease being less pronounced in seasons following winters severe enough to kill all above-ground parts (EDGERTON, 1955).

Host range

Sugarcane (*Saccharum officinarum* and *Saccharum* hybrids) is the main host but the disease is also present on *S. spontaneum*, which is reported to be susceptible (ABBOTT, 1964; HSIEH and TSENG, 1978).

Epidemiology

In most countries, the disease is generally present throughout the year. However, infection is more readily observed after heavy rains and humid conditions as well as in mild temperatures around 20–22°C.

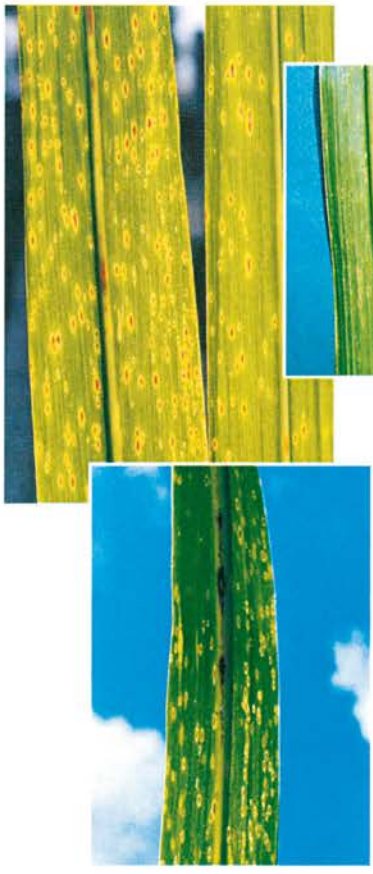


Figure 1. Brown, oval leaf spots surrounded by a yellow halo (ISSCT, MSIRI)

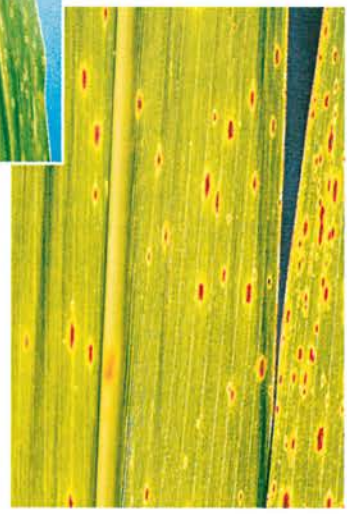


Figure 2. Leaf showing large brown spot lesions (atypical symptoms) (MSIRI).

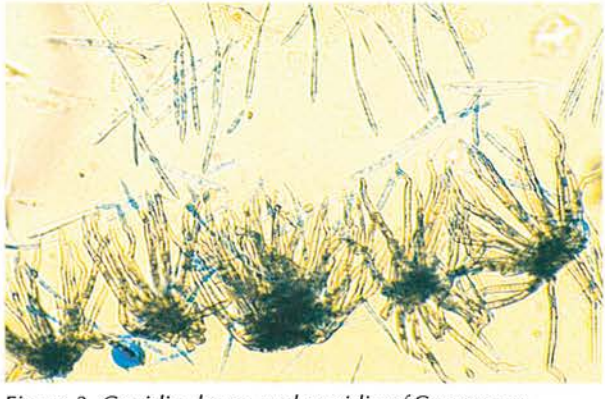


Figure 3. Conidiophores and conidia of Cercospora longipes *(×400) (MSIRI).*

Economic importance

Susceptibility of several varieties to the disease has been well documented, and in one instance a yield reduction of 12% of sugar has been reported (ABBOTT, 1951). Observations from various countries show that variety NCo310 is resistant while NCo376 is susceptible. In general, brown spot is considered to be a minor disease.

Control

The disease is controlled using resistant varieties. Screening methods have been described (WISMER and KOIKE, 1965; WANG and LEE, 1983) although most selection programmes do not conduct formal trials to identify resistant clones. Fungicides such as mancozeb, copper oxychloride, copper oxychloride + zineb are effective in the control of the disease (MUTHUSAMY and SITHANANTHAM, 1974) but in practice no chemical treatment is applied.

References

ABBOTT E.V., 1951. The brown spot leaf disease in Louisiana. Sugar Bulletin 29: 139–141.

ABBOTT E.V., 1964. Brown spot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 24–28. Amsterdam, The Netherlands, Elsevier Publishing Company.

EDGERTON C.W., 1955. Sugarcane and its Diseases. Brown spot. Baton Rouge, USA, Louisiana State University Press, 290p.

HSIEH W.H., TSENG D., 1978. Brown spot of sugarcane in Taiwan. Report Taiwan Sugar Research Institute 79: 29–35.

MULDER J.L., HOLLIDAY P., 1974. *Cercospora longipes*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 418. Kew, UK, Commonwealth Mycological Institute.

MUTHUSAMY S., SITHANANTHAM S., 1974. Varietal susceptibility to brown spot disease (*Cercospora longipes* Butler) of sugarcane. Sugarcane Pathologists' Newsletter 11/12: 15–17.

NOLLA J.A.B., 1965. Brown spot of sugar cane in Puerto Rico. Proceedings International Society of Sugar Cane Technologists Congress 13: 1131–1132.

WANG Z.N., LEE C.S., 1983. Evaluation of sugar cane varieties for resistance to brown spot. Proceedings International Society of Sugar Cane Technologists Congress 18: 771–776.

WISMER C.A., KOIKE H., 1965. Testing sugarcane varieties against eye spot, brown spot, red rot and leaf scald diseases in Hawaii. Proceedings International Society of Sugar Cane Technologists Congress 13: 1144–1153.

Brown stripe

Peter J.L. Whittle

Cause

Cochliobolus stenospilus (Drechsler) T. Matsumoto & W. Yamamoto (note: invalid name as no Latin description was provided), ascomycete fungus; anamorph = *Bipolaris stenospila* (Drechsler) Shoemaker.

Bipolaris stenospila is normally found associated with the disease.

Geographical distribution

Australia, Bangladesh, Barbados, Belize, Brazil, China, Côte d'Ivoire, Cuba, Dominican Republic, Ethiopia, Fiji, 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, 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 75 mm long. Lesions are surrounded by a yellow halo, which is only slightly wider than the lesion itself (Figures 1 and 2). In severe cases, lesions may coalesce and kill large areas of leaf (Figure 3). Varieties may differ slightly in the appearance and severity of symptoms (Figure 4).

Diagnosis

Leaf symptoms are characteristic. Lesions are similar to those of eye spot, but lack the 'runner'. 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 can be identified from its conidia (ELLIS and HOLLIDAY, 1971). 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. A key for distinguishing *Bipolaris* species is given by SIVANESAN (1987).

Strains of the pathogen

Physiologic races of the pathogen were 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 over short and long distances.

Host range

The major host is sugarcane; the pathogen infects a range of grasses artificially, but this is not confirmed in nature.

Epidemiology

Conidia develop on dead leaves in large numbers. After movement by wind, they germinate on leaves in the presence of free moisture from winter to summer. 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 and under dry conditions. The role of the disease in these losses is difficult to quantify.

Control

Development of brown stripe is minimal when conditions are favourable for cane growth. The addition of potassium and/or phosphorus may be beneficial. Substitution of susceptible varieties may be necessary. Resistance screening can be undertaken in the field or on seedlings enclosed in plastic bags during inoculation. Fungicides may be efficacious, but are unlikely to be economical.

Figure 1. Brownish-red leaf lesions surrounded by a yellow halo (BSES).

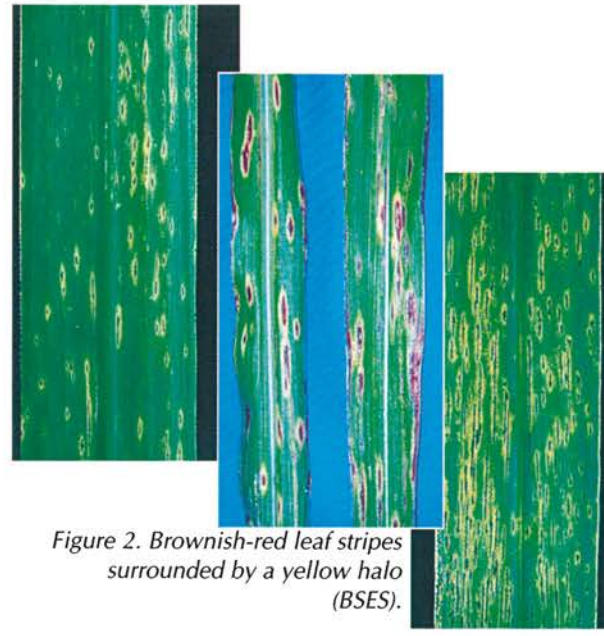


Figure 2. Brownish-red leaf stripes surrounded by a yellow halo (BSES).

Figure 3. Diseased field (BSES).

Figure 4. Leaves showing various degrees of brown stripe lesions (BSES).

References

ELLIS M.B., HOLLIDAY P., 1971. *Drechslera stenospila*. CMI Descriptions of Plant Pathogenic Fungi and Bacteria No. 306. Kew, UK, Commonwealth Mycological Institute.

MARTIN J.P. (revised by B.T. EGAN), 1989. Brown stripe. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 97–106. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SIVANESAN A., 1987. Graminicolous species of *Bipolaris, Curvularia, Drechslera, Exserohilum* and their teleomorphs. Mycological Paper No. 158. Kew, UK, Commonwealth Mycological Institute, 261p.

Common rust

Richard N. Raid and Jack C. Comstock

Cause

Puccinia melanocephala H. & P. Sydow, basidiomycete fungus.

Geographical distribution

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

Symptoms

Common rust symptoms are characterized by pustules (uredinia) that occur mainly on the underside of the leaves (RYAN and EGAN, 1989). The pustules are 2–20 mm long by 1–3 mm wide and lie parallel to the vascular bundles (Figures 1 and 2). At maturity the pustules erupt, exposing orange-brown masses of urediniospores (Figure 3). Severely infected leaves have large numbers of pustules that coalesce, causing large areas of leaves to become necrotic (Figure 4). The pustules of common rust appear quite similar to orange rust (caused by *P. kuehnii*) and the two rusts can be mistaken for one another. Common rust pustules are more reddish-brown to brown and never orange-brown.

The first symptoms of rust infection are small, elongate yellow flecks that are visible on both leaf surfaces. The flecks become reddish-brown on susceptible plants within 3–4 days, they elongate and within 10–14 days become mature sporulating pustules.

Diagnosis

Common rust is distinguished by the reddish-brown to brown sporulating pustules that occur on the undersides of the leaf surface. Positive identification is

by identification of the fungal pathogen. Urediniospores of *P. melanocephala* are smaller (21–40 × 17–27 µm) than those of *P. kuehnii* (25–57 × 17–34 µm) (RYAN and EGAN, 1989). Moreover, the urediniospores are darker with more prominent pores with no apical thickening. The spines on the urediniospores can also be used to distinguish the two organisms. The spines on *P. melanocephala* urediniospores are closer, spaced 1–1.5 µm apart in a regular pattern whereas those of *P. kuehnii* are 3–4 µm apart and are not as regularly spaced.

Strains of the pathogen

Rust races have been reported from India and Florida. Based on the differential reactions of varieties in India, at least six races were characterized (SRINIVASAN and MUTHAIYAN, 1965). In Florida, DEAN and PURDY (1984) indicated two races based on changes in varietal resistance. A race change was also suggested in Florida when the resistant cultivar CP78-1247 developed severe symptoms and had to be withdrawn from commercial production in Florida. In contrast, Australia reported a single rust race from results on a detached leaf test. Most countries probably have not evaluated the presence of races since there are no reports.

Transmission

Sugarcane rust is primarily a wind-disseminated disease. Urediniospores produced within pustules, typically during periods of high humidity and/or leaf wetness, are increasingly subject to passive release as relative humidity decreases during early daylight hours. Dislodged from the pustule by gravity, wind currents or foliar movement, spores become air-borne. Once aloft, rust spores may travel distances ranging from a few centimetres to hundreds of kilometres. There is even evidence that sugarcane rust may have spread from Africa to the Western Hemisphere by trans-oceanic high altitude air currents (PURDY *et al.*, 1985). The wind-disseminated nature of rust spores accounts for the disease's ability to spread great distances over a relatively short period of time.

Rain-splash and water transport are also means of urediniospore dissemination. However, the effective range of dissemination for this type of transport typically ranges from millimetres to several metres. Transport methods other than urediniospore dispersal are unimportant. Since the rust pathogen is not systemic within sugarcane stalks, it is not spread via seed cane.

Host range

Species of *Saccharum* are the main hosts of *P. melanocephala*, although sporulating pustules also have been observed on *Erianthus fulvus* and *Narenga porphyrocoma*. Resistant-type symptoms, in the form of red flecks, have been reported on *Bambusa* sp. and additional *Erianthus* sp.

Figure 1. Scattered rust pustules on the lower side of a leaf (J.C. Comstock, P. Rott).

Figure 2. Lower side of leaf with 'banded' areas of rust pustules (J.C. Comstock).

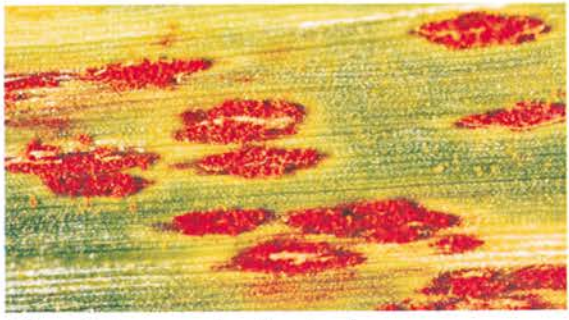


Figure 3. Rust pustules showing the masses of urediniospores emerging (J.C. Comstock).

Figure 4. Leaves with severe rust symptoms (P. Baudin).

Epidemiology

Leaf wetness and temperature are reported to be the environmental factors most influential to rust development (COMSTOCK and RAID, 1994). Common rust is considered to be a cool to moderate temperature disease. Although urediniospores germinate over a fairly broad range of temperatures from 5 to 34°C (PURDY *et al.*, 1983; SOTOMAYOR *et al.*, 1983), appressoria formation, necessary for successful infection, is drastically reduced at temperatures exceeding 30°C. In Florida, maximum daily temperatures above 30°C appear to limit the progression of rust epidemics in the field. Temperature limitations also explain why common rust is frequently more severe at higher elevations in the tropics. Rust spores require adequate periods of leaf wetness or high relative humidity for germination (>8 h).

Rust severity can rapidly increase within a short time period because of the short reproductive cycle. A rust urediniospore can land on a leaf, infect and develop into a sporulating pustule within 14 days. Within 6 weeks, a field planted with a susceptible cultivar may appear reddish-brown when observed from a distance.

Host genotype and plant age are important factors in sugarcane rust development. Wide ranges in cultivar susceptibility have been observed. Depending on host genotype, symptoms may range from an immune response to abundant pustule formation with profuse sporulation. Young plants, 2–6-months-old, have been reported as being more susceptible to rust than more mature plants. The optimum plant age for rust development appears to occur between 4 and 6 months (COMSTOCK and FERREIRA, 1986; VICTORIA *et al.*, 1990). Disease severity gradually decreases with increasing age and rust may almost disappear by 8 months.

Associations between rust severity and soil factors, such as soil pH and nutrient status, have been reported (ANDERSON *et al.*, 1990), however it is difficult to make any generalizations in this regard. There is some agreement that rust severity is promoted by high nitrogen levels.

Economic importance

Common rust was once considered to be economically unimportant. However, with its spread to the Western Hemisphere in 1978, the disease took on new significance (PURDY *et al.*, 1983). With large tracts of land being planted with highly susceptible varieties, most importantly B4362, the disease caused much damage during the years immediately following its spread to the Americas. Yield losses as high as 50% were indicated for certain areas, necessitating the rapid replacement of these susceptible varieties with more resistant varieties.

Yield losses of 10–40% due to rust are not uncommon on susceptible varieties (COMSTOCK *et al.*, 1992). Losses result from the combined effects of reductions in the numbers of millable stalks and stalk biomass.

An indirect loss is the need to develop rust resistant cultivars. This is a costly addition to variety development programmes and many potentially high yielding varieties are lost due to rust susceptibility. Variants within rust pathogen populations demand an ongoing breeding effort in this regard.

Control

Host-plant resistance is the only economic means of control (COMSTOCK and RAID, 1994). Cultivar diversification is also recommended due to the possible presence of rust variants. Although fungicides have proven effective in controlling rust in research studies, their commercial use is not economically feasible at this time.

References

ANDERSON D.L., RAID R.N., IREY M.S., HENDERSON L.J., 1990. Association of sugarcane rust severity with soil factors in Florida. Plant Disease 74: 683–686.

COMSTOCK J.C., FERREIRA S.A., 1986. Sugarcane rust: Factors affecting infection and symptom development. Proceedings International Society of Sugar Cane Technologists Congress 19: 402–410.

COMSTOCK J.C., RAID R.N., 1994. Sugarcane common rust. *In*: Current Trends in Sugarcane Pathology (Prof. K.S. Bhargava Festschrift), G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin Filho, V.P. Agnihotri and C.T. Chen (Eds), p. 1–10. Dehli, India, International Books and Periodicals Supply Service.

COMSTOCK J.C., SHINE J.M. JR, RAID R.N., 1992. Effect of rust on sugarcane growth and biomass. Plant Disease 76: 175–177.

DEAN J.L., PURDY L.H., 1984. Races of sugarcane rust, *Puccinia melanocephala*, found in Florida. Sugar Cane 1: 15–16.

PURDY L.H., LIU L.J., DEAN J.L., 1983. Sugarcane rust, a newly important disease. Plant Disease 67: 1292–1296.

PURDY L.H., KRUPA S.V., DEAN J.L., 1985. Introduction of sugarcane rust in the Americas and its spread into Florida. Plant Disease 69: 689–693.

RYAN C.C., EGAN B.T., 1989. Rust. *In*: Diseases of Sugarcane: Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 189–210. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SOTOMAYOR I.A., PURDY L.H., TRESE A.T., 1983. Infection of sugarcane leaves by *Puccinia melanocephala*. Phytopathology 73: 695–699.

SRINIVASAN K.V., MUTHAIYAN M.C., 1965. A note on physiologic races in *Puccinia erianthi* Padw. and Khan affecting sugar-cane varieties. Proceedings International Society of Sugar Cane Technologists Congress 12: 1126–1128.

VICTORIA J., MORENO C., CASSALETT C., 1990. Genotype-environment interaction and its effect on sugar cane rust incidence. Sugar Cane 4: 13–17.

Downy mildew

Sidney Suma and Robert C. Magarey

Cause

Peronosclerospora sacchari (T. Miyake) Sharai & K. Hara, and *P. philippinensis* (W.H. Weston) C.G. Shaw, oomycete fungi; *Peronosclerospora spontanea* (W.H. Weston) C.G. Shaw can also infect sugarcane.

Geographical distribution

The disease is restricted to the Pacific and South East Asian regions: Indonesia, India, Fiji, Japan, Papua New Guinea, Philippines, Taiwan, Thailand.

Symptoms

The disease is characterized by leaf streaks, varying in colour as the leaves and symptoms age. Streaks initially are pale to light yellow in colour, but with increasing age change from greenish-yellow to a more definite yellow, and finally to a brick-red colour (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; 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 (Figure 3). 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.

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' (Figure 4). 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 5). The jump-up stage is often associated with the production of oospores in diseased leaves. Oospores are produced between the vascular elements and are so large

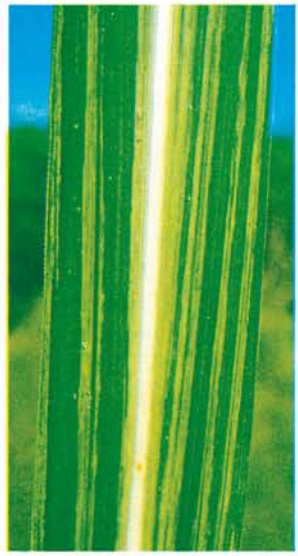


Figure 1. Yellow-green leaf streaks interspersed with normal green leaf tissue: early symptoms (R. Magarey).

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

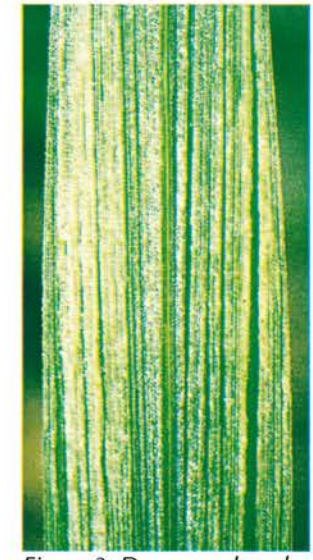


Figure 3. Down produced on the underside of an infected leaf (R. Magarey).

Figure 4. Elongated thin diseased stalks called 'jump-ups' (ISSCT).

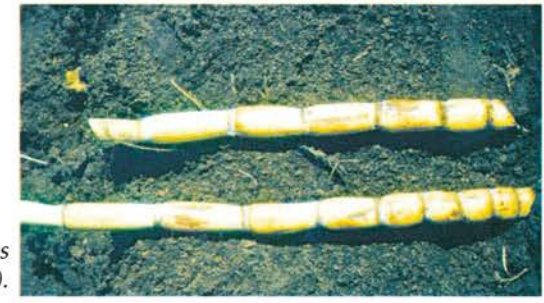


Figure 5. Brown elongated lesions on diseased stalks (ISSCT).

that they push the adjacent elements apart; this leads to leaf shredding (Figure 6), an obvious symptom under certain conditions. Other *Peronosclerospora* spp. may cause leaf shredding.

Diagnosis

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 the various species of *Peronosclerospora* are currently being developed.

Strains of the pathogen

The presence of a second strain of *Peronosclerospora sacchari* has been reported in Papua New Guinea (EGAN, 1991). The second strain of *P. sacchari* was tentatively named strain B, strain A being the original strain (SUMA and PAIS, 1996). Further characterization is required to confirm the existence of the second strain.

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 23.30 h 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.

Oospores have no known role in disease transmission. Spread between regions, or countries, has 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 sacchari infects a number of other hosts including *Zea mays* (maize, which is as susceptible as cane), some sorghum lines, teosinte (*Euch-*

laena mexicana), *Sorghum halepense* (Johnson grass) and *S. sudanense* (Sudan grass). *Saccharum* species infected by the downy mildew pathogens of commercial crops is unclear since there are other *Peronosclerospora* pathogens producing similar symptoms and there are currently no definitive assays.

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 23.30 h onwards and peak around 1.30–2.30 h 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.

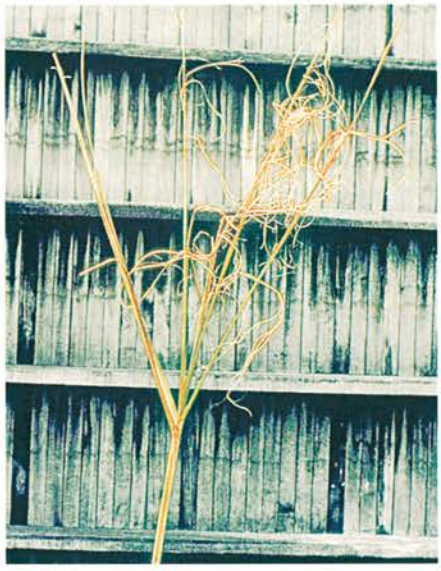


Figure 6. Leaf shredding symptoms (R. Magarey).

Figure 7. Sugarcane field affected by downy mildew (ISSCT).

Economic importance

The disease 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 7), and (2) loss of potential yield from not growing susceptible, but otherwise agronomically superior, varieties (LEU and EGAN, 1989). In Papua New Guinea, SUMA and PAIS (1996) reported direct yield losses of up to 15% of total tonnage harvested, while HUSMILLO (1982) estimated losses in commercial cane in the Philippines (due to *P. philippinensis*) at 38% (tons of cane) and 58% (tons of sugar/ha).

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). Breeding programmes must take resistance to downy mildew into consideration in regions where the disease is prevalent, such as Papua New Guinea.

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 Papua New Guinea (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, and 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 tolerant 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 can be 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).

References

EASTWOOD D., MALEIN P.J., 1998. Further observations on the use of fungicides to control *Peronosclerospora sacchari* (T. Miyake) Sharai and K. Hara in sugarcane in Papua New Guinea. International Journal of Pest Management 44(2): 71–73.

EGAN B.T., 1986. New disease problems appear in Papua New Guinea. Bureau of Sugar Experiment Stations Bulletin 16: 8–10.

EGAN B.T., 1991. Notes on sugarcane diseases in Papua New Guinea. International Society of Sugarcane Technologists Technical Newsletter 3: 3.

HUSMILLO F.R., 1982. Assessment of yield loss due to downy mildew of sugarcane caused by *Peronosclerospora philippinensis* (Weston) C.G. Shaw. Sugarcane Pathologists' Newsletter 28: 17–24.

JAMES G.L., 1983. The effect of pre-plant seed cane treatment with the fungicide acalalanine. Proceedings International Society of Sugar Cane Technologists Congress 18: 855–866.

LEU L.S., EGAN B.T., 1989. Downy mildew. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 107–121. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

MAGAREY R.C., 1996. Downy mildew, Ramu stunt and Ramu orange leaf. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 113–115.

MALEIN P.J., 1993. Fungicidal control of *Peronosclerospora saccahari* (T. Miyake) Sharai and K. Hara in Papua New Guinea. International Journal of Pest Management 9(3): 325–327.

SUMA S., PAIS E., 1996. Major diseases affecting sugarcane production on Ramu sugar estate, Papua New Guinea. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 30–33.

Dry top rot

Jack C. Comstock

Cause

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

Geographical distribution

Present in USA (Florida); reported in Puerto Rico, Barbados, Colombia, Cuba, Guyana, Panama and Venezuela but may not currently be present.

Symptoms

The symptoms of dry top rot affected plants are somewhat similar to drought stress. Symptoms become evident late in the crop on maturing plants (ABBOTT, 1964; MATZ, 1920). Initial symptoms are the withering of spindle leaves, drying of leaf tips and edges of the leaf blades (Figure 1). Stalk growth is eventually retarded. The upper internodes are shortened and become withered. In the most severely affected plants, one or all the stalks die. The dead stalks usually remain standing and become more shrivelled in time (COMSTOCK *et al.*, 1994). Rind disease may also occur on the affected stalks. Leaves remain attached to the dead stalks for a period of time and then they may become detached with only the stalk remaining (Figures 2 and 3). There is no side shooting on the affected stalks.

Diagnosis

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 orange-pink vascular bundles located in the internodes at the base of dry top rot symptomatic plants (COMSTOCK *et al.*, 1994; MATZ, 1922) (Figure 4). The discoloured vascular bundles are pale in colour and not as red as those discoloured by leaf scald and ratoon stunting disease (COMSTOCK *et al.*, 1994). The discoloured vascular bundles can be dissected from the stalk using a sharp scalpel

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

Figure 2. Drying of leaves and loss of some leaves (J.C. Comstock).

Figure 3. Withered and shrunken dead stalks (J.C. Comstock).

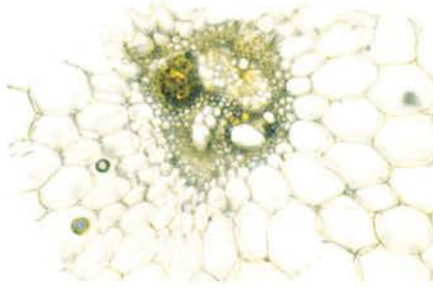


Figure 4. Spores of the pathogen in xylem cells of a vascular bundle (J.C. Comstock).

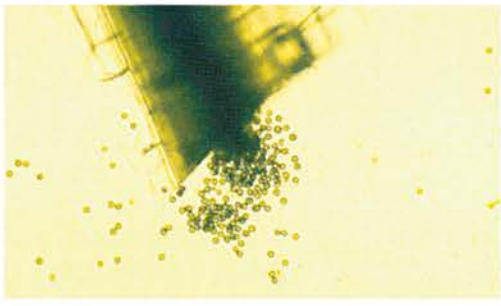


Figure 5. Spores of Ligniera vasculorum *escaping from a cut vascular bundle (J.C. Comstock).*

(Figure 5). Spores are rarely found in non-discoloured vascular bundles located in the same internode. Although the discoloured vascular bundles and spores can be found in several internodes above the soil line, they are most prevalent in the older internodes. Progressing up infected stalks, there is a transition in the xylem vessels from spores to 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 as a result 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 available.

Transmission

The dry top rot pathogen, *L. vasculorum*, can be transmitted by planting seed pieces cut from infected stalks. Germination and survival of new plants from severely affected seed cane are 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 wet poorly drained areas in Puerto Rico. High water tables for at least a portion of the year favour the disease (COOK, 1929).

Economic importance

Because of dry top rot's limited distribution, primarily to a small area (1 ha) on the USDA-ARS Sugarcane Field Station at Canal Point, Florida, it is not causing economic losses (COMSTOCK *et al.*, 1994). 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.

Control

Control is by planting resistant cultivars and preventing the spread of the disease by planting healthy seed cane. Hot water treatments (52°C for 45 min) gave partial control (Comstock *et al.*, 1994).

References

Abbott E.V., 1964. Dry top rot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 110–113. Amsterdam, The Netherlands, Elsevier Publishing Company.

Comstock J.C., Miller J.D., Farr D.F., 1994. First report of dry top rot of sugarcane in Florida: Symptomatology, cultivar reactions, and effect of stalk water flow rate. Plant Disease 78: 428–431.

Cook M.T., 1929. Life history of *Ligniera vasculorum* (Matz) Cook. Journal of the Department of Agriculture Porto Rico 13(1): 19–29.

Cook M.T., 1937. The organism causing the dry top rot of sugar cane. Journal of the Agriculture Department of the University of Puerto Rico 21(1): 85–97.

Matz J., 1920. A new vascular organism in sugar cane. Journal of the Department of Agriculture Porto Rico 4(1): 41–46.

Matz J., 1922. Dry top rot of sugar cane, a vascular disease. Journal of the Department of Agriculture Porto Rico 6(3): 28–47.

Eye spot

Jack C. Comstock

Cause

Bipolaris sacchari (E.J. Butler) Shoemaker, deuteromycete fungus.

Geographical distribution

Andaman Islands, Antigua, 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, Guadeloupe, 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, 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 by 0.5–3.0 mm wide). Reddish-brown runners often extend toward the apex from the lesion and may be 3–6 mm wide and extend up to 90 cm (Figures 1, 2 and 3). The runners are the result of 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 4).

Diagnosis

The typical elongate eye spot lesion with the characteristic reddish-brown runner extending toward the leaf margin is diagnostic. *Bipolaris sacchari* will

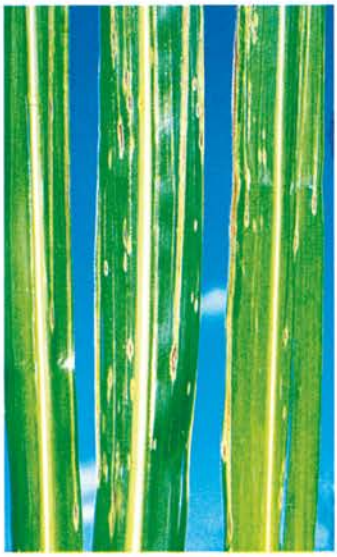


Figure 1. Long reddish-brown runners extending from elliptical eye-shaped lesions toward the leaf margins (G.W. Steindl).

Figure 2. Eye spot leaf symptoms (P. Rott).

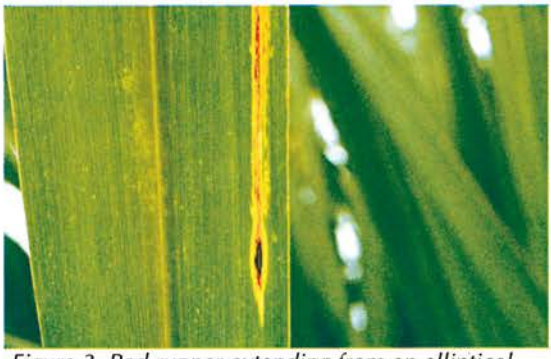


Figure 3. Red runner extending from an elliptical eye-shaped lesion (P. Rott).

Figure 4. Leaves showing severe eye spot symptoms (P. Rott).

sporulate within 3–4 days from young lesions that have been placed in moist chambers after being surface 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, man and setts are not important.

Host range

Bipolaris sacchari is primarily confined to *Saccharum* species. The eye spot pathogen has been isolated from *Pennisetum purpureum* and *Cymbopogon citratus*. However, only the isolates from *C. citratus* were pathogenic to sugarcane.

Epidemiology

Eye spot is favoured by cool wet weather which favours *B. sacchari* infection. Wind and rain help to disperse *B. sacchari* conidia (MARTIN, 1961). Cool weather is also required for susceptible plants to be sensitive to the host-specific toxin of *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 cutivar is released and disease outbreaks occur. In areas that are conducive for the disease, variety development programmes screen clones and eliminate susceptible cultivars in their selection programme. This not only increases the cost of variety development programmes but also eliminates some potentially high yielding cultivars because of their susceptibility (COMSTOCK and STEINER, 1989).

Control

Resistant cultivars are the only practical means of control. Screening programmes 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). Control by fungicides is not economical.

References

BYTHER R.S., STEINER G.W., 1975. Heat-induced resistance of sugarcane to *Helminthosporium sacchari* and helminthosporoside. Plant Physiology 56: 415–419.

BYTHER R.S., STEINER G.W., 1976. Summer-induced resistance to eye spot disease of sugarcane. Sugarcane Pathologists' Newsletter 15/16: 54–56.

COMSTOCK J.C., STEINER G.W., 1989. Eye spot. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 123–133. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

DEAN J.L., MILLER J.D., 1975. Field screening of sugarcane for eye spot resistance. Phytopathology 65: 955–958.

MACKO V., 1983. Structural aspects of toxins. *In*: Toxins and Plant Pathogenesis. J.M. Daly and B.J. Deverall (Eds), p. 41–80. New York, USA, Academic Press.

MARTIN J.P., 1961. Eye Spot. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 167–185. Amsterdam, The Netherlands, Elsevier Publishing Company.

STEINER G.W., STROBEL G.A., 1971. Helminthosporoside, a host-specific toxin from *Helminthosporium sacchari*. Journal of Biological Chemistry 246: 4350–4357.

Floral smuts and ergot

Claude Ricaud

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* (Kühn) K. Vánky (syn. *Ustilago crucnta* Kühn), *Sphacelotheca erianthi* (H. & P. Sydow) Mundkur (syn. *Ustilago erianthi* H. & P. Sydow), and *Sporisorium schweinfurthianum* (Thümen) K. Vánky (syn. *Ustilago schweinfurthiana* Thümen), all basidiomycete fungi.

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

Ergot: *Claviceps purpurea* (E.M. Fries) L.R. Tulasne and *C. pusilla* Cesati, 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.

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*, ascospores 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).

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

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

No specific information is available. Wild sugarcane and certain grasses may be a source of infection.

Economic importance and control

These infections of the inflorescence are not of economic importance in cultivated sugarcane. Exceptionally *Sporisorium cruentum* has been found to affect growth. However, they present a serious drawback in a breeding programme.

No specific control measure is required in cane fields but movement of true seed (fuzz) for a breeding programme must be subject to strict quarantine and fungicide treatment.

References

BACHCHHAV M.B., 1978. First record of false floral smut from Maharashtra, India. Sugarcane Pathologists' Newsletter 20: 32–33.

CHONA B.L., MUNJAL R.L., 1951. A new smut of sugar cane. Current Science (India) 20: 301–302.

HOLLIDAY P., 1980. Fungus Diseases of Tropical Crops. Cambridge, UK, Cambridge University Press, 607p.

MUNDKUR B.B., THIRUMALACHAR M.J., 1952. Ustilaginales of India. Kew, UK, Commonwealth Mycological Institute, 83p.

ROBINSON P.E., 1964. Fungus diseases of the sugar-cane inflorescence. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 141–156. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. Sugarcane Diseases. Phytopathological Paper No. 29. Slough, UK, CAB International, 88p.

Fusarium sett or stem rot

Barry J. Croft

Cause

Gibberella fujikuroi (K. Sawada) H.W. Wollenweber, ascomycete fungus; anamorph = *Fusarium moniliforme* J.L. Sheldon. *Fusarium tricinctum* (Corda) P.A. Saccardo has also been isolated from sugarcane showing stem rot symptoms (KOIKE, 1978). This fungus is generally not a pathogen but it can attack sugarcane that has suffered from frost damage.

Geographical distribution

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

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 1). 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 disease by the lack of the characteristic odour of over-ripe pineapples produced by pineapple disease and the more intense red colouring of the tissues.

STEM ROT

In mature stalks of standing cane infection is often associated with damage from borers or other stalk injury (Figure 2). 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 3). Leaves on infected stalks wilt, turn yellow and die. This form of the disease has been associated with the disease known as wilt.

Fusarium stalk rot has been reported as causing extensive losses in a disease complex with red rot in Thailand (ANONYMOUS, 1991). Symptoms were reported to be a mixture of the two diseases.

Diagnosis

The disease is diagnosed by examination for the symptoms and isolation of *G. fujikuroi*. This fungus can be easily isolated on simple nutrient agars such as potato dextrose agar or corn meal agar. *Gibberella fujikuroi* produces white mycelium which turns greyish-violet to greyish-magenta with age. Pigmentation in the agar varies from no pigmentation to violet or magenta.

Fusarium moniliforme produces macroconidia in pale orange sporodocia, the spores are slender, falcate to almost straight, usually 3–5 (or more) septate and thin walled. They measure 25–60 × 2.5–4.0 µm (BURGESS and LIDDELL, 1983; BOOTH, 1971). The apical cell is slightly curved and tapers to a point. The base of the basal cell is foot shaped or notched. Microconidia are produced abundantly in chains from phialides on branched conidiophores or phialides formed directly on the hyphae. The microconidia are clavate, single celled with a flattened base and measure 5–12 × 1.5–2.5 µm.

Strains of the pathogen

Fusarium moniliforme is variable in its cultural characteristics but there is no clear relationship between these different cultural types and virulence.

Transmission

Fusarium moniliforme can grow saprophytically on decaying plant debris and is spread by wind and rain. The fungus can be carried on the outside of stalk pieces or within the stalk and in contaminated soil.

Host range

Fusarium moniliforme has a wide host range on both monocotyledons and dicotyledons. It causes a range of diseases in these hosts (BOOTH, 1971).

Epidemiology

Fusarium stem rot is associated with standing cane suffering stress, such as waterlogging or drought or damage by boring insects. The sett rot is also

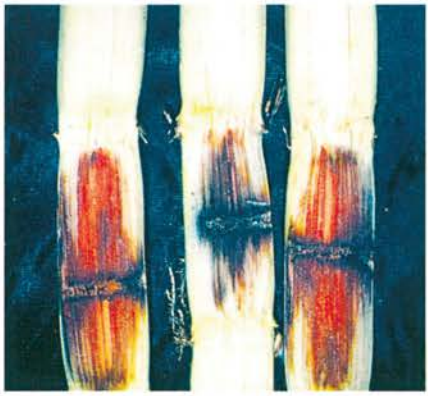


Figure 1. Purplish-red discoloration of internal tissues of sugarcane setts (ISSCT).

Figure 2. External stalk lesions (ISSCT).

Figure 3. Purplish-red discoloration of internal stalk tissues (ISSCT).

associated with insect-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

Fusarium sett or 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 *Glomerella tucumanensis* with a serious outbreak recently reported in Thailand (ANONYMOUS, 1991).

Control

The most effective control of Fusarium stem rot is to avoid stress by improving drainage and irrigation and to reduce damage from stem borers. Where this is not possible, resistant varieties should be grown. Fusarium sett rot can be controlled 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. Mercurial fungicides are reported to control Fusarium sett rot but there has been no study of the effectiveness of more recently developed fungicides.

References

ANONYMOUS, 1991. Control of red rot wilt disease of sugarcane in Sing Buri. Annual Report of the Suphan Buri Field Crops Research Centre, Suphan Buri, Thailand, p. 14–16.

BOURNE B.A., 1961. Fusarium sett or stem rot. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 186–207. Amsterdam, The Netherlands, Elsevier Publishing Company.

BOOTH C., 1971. The Genus *Fusarium*. Kew, UK, Commonwealth Mycological Institute, 237p.

BURGESS L.W., LIDDELL C.M., 1983. Laboratory Manual for *Fusarium* Research. Sydney, Australia, University of Sydney Press, 162p.

EGAN B.T., MAGAREY R.C., CROFT B.J., 1997. Sugarcane. *In*: Soilborne Diseases of Tropical Crops. R.J. Hillocks and J.M. Waller (Eds), p. 277–302. Wallingford, UK, CAB International.

KOIKE H., 1978. Pathogenicity of *Fusarium tricinctum* to sugarcane: a first report. Sugarcane Pathologists' Newsletter 20: 24–26.

Leaf blight

W.H. Hsieh and Jack C. Comstock

Cause

Leptosphaeria taiwanensis W.Y. Yen & C.C. Chi, loculoascomycete fungus; anamorph = *Stagonospora tainanensis* W.H. Hsieh.

Geographical distribution

India, Japan, Philippines, Taiwan.

Symptoms

Mature leaf blight lesions are elongate, 1–50 mm long by 1–3 mm wide (Figures 1 and 2). Lesion size decreases as the number of lesions on the leaf increases. The colour of the lesions varies from bright red, reddish-brown, light yellow, and light yellow with a reddish tint depending on the clone. Often multiple lesions coalesce forming broad bands of infected tissue. Necrotic tissues dry out giving a reddish-brown appearance from a distance (Figure 3). Lesions may also occur on leaf sheaths (YEN, 1964; YEN and CHI, 1953).

Diagnosis

Leaf blight is diagnosed by symptoms and the presence of either perithecia of *Leptosphaeria 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 to asci length. 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).

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 sugarcane hybrids and *Saccharum* species (LEU *et al.*, 1974). *Miscanthus* may be a host since *Miscanthus* blight described in the USA has either the same or a closely related pathogen (O'NEILL and FARR, 1996).

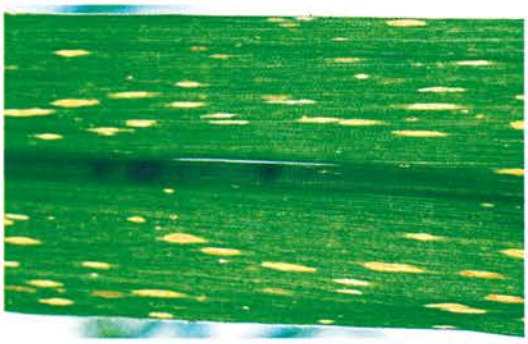


Figure 1. Elongated leaf blight lesions (J.C. Comstock).

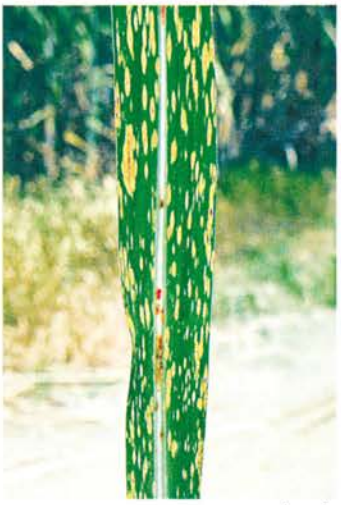


Figure 2. Leaf with numerous elongated lesions (J.C. Comstock).

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

Epidemiology

Leaf blight is favoured 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 on susceptible clones in Taiwan causing a loss of cane tonnage and sucrose.

Control

Resistance is recommended for leaf blight control. Sugarcane can be screened in the field using pycnidiospores for inoculation and in glasshouse tests (LEU *et al.*, 1976; LEU and HSIEH, 1971). Ratings for both tests were similar to results obtained under natural infection.

References

HSIEH W.H., 1979. The causal organism of sugarcane leaf blight. Mycologia 71: 892–898.

LEU L.S., HSIEH W.H., 1970. Leaf blight of sugarcane in Taiwan. 1. Outbreak of leaf blight in 1967–68 and preliminary testing of oil sprays as a method of control. Plant Protection Bulletin 12: 59–67.

LEU L.S., HSIEH W.H., 1971. Leaf blight of sugarcane in Taiwan. 4. Resistance trial. Sugarcane Pathologists' Newsletter 7: 4–7.

LEU L.S., WANG Z.N., HSIEH W.H., 1974. Leaf blight of sugarcane in Taiwan. 5. Host range and hypersensitive reaction. Report of the Taiwan Sugar Research Institute 63: 59–67.

LEU L.S., WANG Z.N., LIN K.H., KWO C.Y., SHANG K.C., 1976. Leaf blight of sugarcane in Taiwan. 6. Preliminary studies on the mode of inheritance of resistance in sugarcane against the disease. Report of the Taiwan Sugar Research Institute 71: 45–54.

O'NEILL N.R., FARR D.R., 1996. Miscanthus blight, a new foliar disease of ornamental grasses and sugarcane incited by *Leptosphaeria* sp. and its anamorphic state *Stagonospora* sp. Plant Disease 80: 980 987.

YEN W.Y., 1964. Leaf blight. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 32–36. Amsterdam, The Netherlands, Elsevier Publishing Company.

YEN W.Y., CHI C.C., 1953. The life history of the causal organism of leaf blight of sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 8: 939–944.

Leaf scorch

Ching-Shiou Lee and Yaw-Guang Liang

Cause

Two different pathogens cause similar symptoms: *Stagonospora sacchari* T.T. Lo & K.C. Ling, deuteromycete fungus, and *Leptosphaeria bicolor* W.J. Kaiser, Ndimande & D.L. Hawksworth, ascomycete fungus; anamorph = *Stagonospora* sp. The imperfect stage of *L. bicolor* has not been identified but it is not *S. sacchari*.

Geographical distribution

The first pathogen, *S. sacchari*, caused epidemics in Taiwan and the Philippines. It was also reported in Argentina, Bangladesh, Cuba, Fiji, India, Indonesia, Japan, Nigeria, Panama, Papua New Guinea, South Africa, Thailand, Venezuela, Vietnam.

Leptosphaeria 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 (Figure 1). 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-coloured 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 *L. 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 2). Conidia 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).

Figure 1. Various degrees of leaf scorch lesions (C-S. Lee).

Figure 2. Conidia of Stagonospora sacchari *(C-S. Lee).*

Strains of the pathogen

The existence of strains is suspected but has not been proven yet.

Transmission

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

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. 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 favour 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 Co290 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).

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. Methods for screening varieties for resistance to *S. sacchari* have been developed and are still being used in Taiwan.

References

EXCONDE O.R., 1963. Leaf scorch of sugar cane in the Philippines. The Philippine Agriculturist 47: 271–297.

KAISER W.J., NDIMANDE B.N., HAWKSWORTH D.L., 1979. Leaf scorch disease of sugarcane in Kenya caused by a new species of *Leptosphaeria*. Mycologia 71: 479–492.

LIN J.T., 1952. The relationship between cane varieties and leaf scorch. Part 1. The degree of susceptibility of various cane varieties. Report Taiwan Sugar Experiment Station 9: 185–199.

LO T.T., 1961. Leaf scorch. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 209–221. Amsterdam, The Netherlands, Elsevier Publishing Company.

LO T.T., LEU L.S., 1989. Leaf scorch. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 135–143. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SAMPANG R.C., REYES T.T., 1980. Virulence pattern, gross morphology of Philippine isolates of *Stagonospora sacchari* Lo and Ling, and assessment of yield loss on sugarcane (*Saccharum officinarum* Linn). Proceedings of the Philippines Sugar Technologist Association Congress 27: 474–481.

SURANTO T.A., HARSANTO U., 1989. Trials on leaf scorch disease in Indonesia. Proceedings International Society of Sugar Cane Technologists Congress 20: 763–767.

Marasmius basal stem, root and sheath rot

Barry J. Croft

Cause

Marasmius sacchari Wakker and *M. stenospilus* Montagne, basidiomycete fungi.

Geographical distribution

Marasmius sacchari has been reported in numerous countries including: Antigua, 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, Uganda, USA, Zimbabwe.

Marasmius stenospilus is found in a more limited area: Indonesia, Mauritius, Peru, Philippines, USA.

Symptoms

Marasmius is usually associated with sugarcane plants growing poorly because of environmental or nutritional stresses, or are affected by other pathogens. 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 leaf 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 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.

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

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

Figure 2. Fruiting bodies of the pathogen at the base of stalks (J. Hoy).

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

No strains have been reported.

Transmission

Transmission is by mycelial strands and soil-borne spores.

Host range

None reported.

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.

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 and C.A. Wismer (Eds), p. 89–93. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. Marasmius Fr. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 11. Slough, UK, CAB International.

Orange rust

Robert C. Magarey

Cause

Puccinia kuehnii E.J. Butler, basidiomycete fungus.

Geographical distribution

The Asian-Oceania region: Australia, China, Fiji, Guam, India, Indonesia, Japan, Malaysia, Myanmar, New Caledonia, Pakistan, Papua New Guinea, Philippines, Samoa, Solomon Islands, Sri Lanka, Taiwan, Thailand, Vietnam.

Symptoms

The two most frequently encountered rust diseases in sugarcane, orange rust and common rust (*Puccinia melanocephala*), may be readily distinguished, though some similarities are also readily apparent. As a rust, orange rust is characterized by leaf lesions which, at maturity, break through the leaf epidermis giving the surface a rough appearance.

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 colour develops depending on the cane variety (Figures 1, 2 and 3). Unlike common 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 and more lesions toward the leaf base.

Symptoms are more prevalent in semi-mature to mature cane, and the disease is favoured by humid summer, and warm to cool autumn (fall) conditions rather than in young cane in spring as is often the case with common rust.

The identity of the disease can be confirmed by the characteristics of the two spore types of the pathogen, which distinguish it from common rust (see the diagnosis section). Urediniospores of orange rust are usually orange in colour (which is the reason for the orange symptoms).

Diagnosis

Orange rust is identified either by symptomatology, or by characteristics of the urediniospores or teliospores (Figures 1, 4, 5 and 6). As mentioned above, orange and common rusts may be distinguished by the aggregation and orange coloration of lesions in orange rust, the orange vs. brown colouring of the urediniospores (orange rust), and the size and shape of urediniospores and teliospores. The urediniospores of *P. kuehnii* have an apical thickening, are paler in colour, 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 with 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.

Strains of the pathogen

There is some evidence for the existence of strains of *P. kuehnii*. Previous to the 1999-2000 season, the variety Q124 was resistant to orange rust in Queensland, though the disease was seen at low intensity in Q78. In the March-May period in 2000, Q124 suffered heavy infection while Q78 maintained only minor disease levels. In addition, Q124 appears to be susceptible to the disease in Indonesia, but not in Papua New Guinea.

Transmission

Rust fungi are usually transmitted by wind and water splash, and orange rust is no exception. SREERAMULU and VITTAL (1970) monitored urediniospores above a 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 (10.00–14.00 h).

Movement of orange rust has not been as well characterized as common rust, where it has been suggested that air mass movements spread the disease around the world in the late 1970s. EGAN (1964) noted that *P. kuehnii* moves more readily during periods of hot humid weather in summer and warm to cool periods in autumn (fall) in Queensland. Under Indian conditions, *P. kuehnii* is more prevalent in the cooler months (SRINIVASAN and CHENULU, 1956).

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). Commercial cane varieties

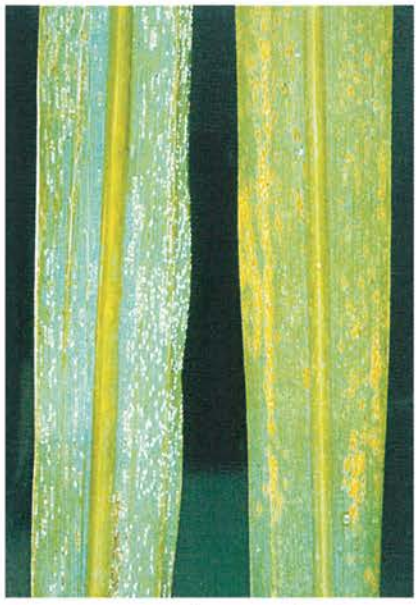


Figure 1. Leaves showing whitish teliospore lesions (left) and orange urediniospore lesions (right) (W.H. Hsieh).

Figure 2. Leaf with orange rust pustules (R. Magarey).

Figure 3. Leaf with numerous orange rust pustules (R. Magarey).

Figure 4. Leaf showing teliosori containing teliospores (W.H. Hsieh).

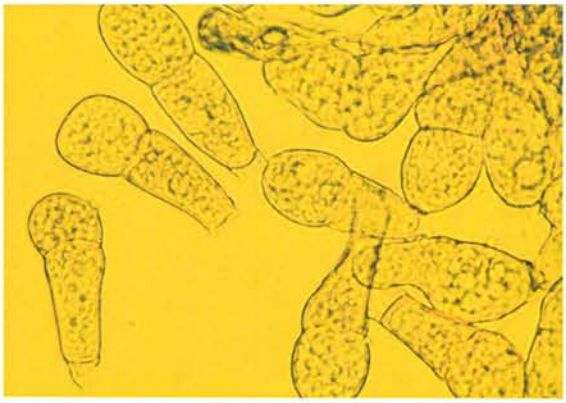


Figure 5. Teliospores of Puccinia kuehnii *(W.H. Hsieh).*

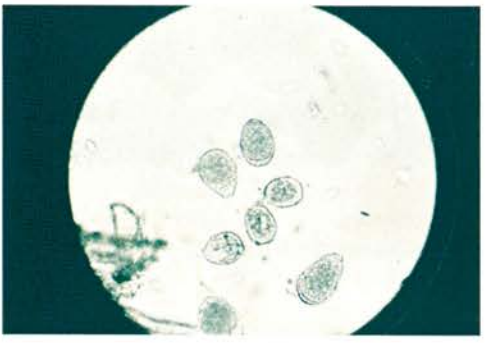


Figure 6. Urediniospores of Puccinia kuehnii *showing thickened apices (R. Magarey).*

Figure 7. Death of foliage (R. Magarey).

rarely show high susceptibility, though Australia's most widely-grown variety, Q124, supported very high disease levels in 2000.

Epidemiology

Wind and large air masses, readily spread the pathogen. Environmental conditions favouring disease development include warm humid summer, and cool autumn (fall), conditions.

Economic importance

The disease is rarely of economic importance but a new strain of *P. kuehnii* has caused extensive yield losses in the Australian sugar industry in the year 2000. A few instances have been noted, as for many minor diseases, where some varieties have shown economically unsustainable yield losses and have been discarded (Figure 7), for instance in Fiji and Australia (NORTH, 1915). In Australia, Q124 and a few other commercial varieties are highly susceptible to what is considered a new strain of the pathogen.

Control

The main form of control for the disease is varietal resistance. Most commercial varieties possess more than adequate resistance to the disease.

References

BUTLER E.J., 1918. Fungi and Diseases in Plants. Calcutta, India, Thacker, Spink and Company.

EGAN B.T., 1964. Rust. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 60–68. Amsterdam, The Netherlands, Elsevier Publishing Company.

NORTH D.S., 1915. Extracts from recent reports by Mr D.S. North on control of diseases and pests at the mills. New South Wales, Colonial Sugar Refining Company Limited.

RYAN C.C., EGAN B.T., 1989. Rust. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 189–210. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SREERAMULU T., VITTAL B.P.R., 1970. Periodicity in the uredospore content of air within and above a sugarcane field. Journal of the Indian Botany Society 50: 39–44.

SRINIVASAN K.V., CHENULU V.V., 1956. A preliminary study of the reaction of *Saccharum spontaneum* variants to red rot, smut, rust and mosaic. Proceedings International Society of Sugar Cane Technologists Congress 9: 1097–1107.

Pachymetra root rot

Barry J. Croft and Robert C. Magarey

Cause

Pachymetra chaunorhiza B.J. Croft & M.W. Dick (Dick *et al.*, 1989), oomycete fungus.

Geographical distribution

Australia (Queensland only).

The disease is unique to Queensland cane fields (Magarey *et al.*, 1987) and has never been observed in any other part of Australia or the world. The disease has also never been observed on land where sugarcane has not been growing. Within Queensland, the disease is widespread in northern, Herbert, central, and some parts of the Bundaberg district.

Symptoms

Pachymetra root rot is characterized by extensive rotting of the larger roots of the sugarcane plant, with consequent debilitated root development and 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 length of the root. Where high levels of the disease occur 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, and also a loss in stool anchorage with consequent stool tipping and failed ratooning.

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

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

Diagnosis

Diagnosis of the disease relies upon the observation of rotted primary roots, and the observation, under a microscope, of the distinctive oospores in flaccid sections of these roots (CROFT and MAGAREY, 1984 and 1989). Oospores are relatively large (25–65 µm in diameter) and have large blunt projections on the oogonial wall (Figure 4), making them larger and distinctive compared to the various *Pythium* species. Staining of spores is unnecessary under normal conditions.

An assay has been developed for assessing *P. chaunorhiza* inoculum densities in soil from the field (MAGAREY, 1989). The assay uses the selective sieving of the spores from the soil (using a wet sieving technique), followed by bleaching of the collected material and the semi-selective staining of spores. Spores are counted under a microscope at ×60 magnification (Figure 5).

Strains of the pathogen

Some evidence has been obtained suggesting that there are differences in the pathogen in different parts of Queensland. Molecular techniques have been used to show 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 has failed to identify large variation in varietal reaction between the two groups of isolates but there are differences with one or two varieties.

Transmission

Unlike *Pythium* species, *Pachymetra chaunorhiza* does not produce zoospores and, therefore, has no motile spore stage (MAGAREY, 1991). Transmission to healthy fields relies on the transfer of soil on implements and machinery. Build-up of the disease in fields occurs through the production and deposition of spores in the soil from diseased roots; these roots are filled with the characteristic oospores. Spore levels may rise quickly, particularly beneath diseased stools, when susceptible varieties are grown (MAGAREY and MEWING, 1994).

Host range

Very few other hosts of *Pachymetra chaunorhiza* have been detected (PERRY, 1985). There are a number of highly susceptible varieties of commercial sugarcane; *Saccharum spontaneum, S. robustum* and *S. edule* can also be infected,

as can *Erianthus* spp. (MAGAREY and CROFT, 1996). A few other grass species have also been infected including sorghum, maize (low levels of disease) and the native grass *Imperata cylindrica* (blady grass).

Epidemiology

Pachymetra root rot is favoured by high rainfall; the disease tends to be more severe in the wet, tropical region of far northern Queensland (MAGAREY and SOPER, 1992). One of the most important factors governing disease levels is the resistance of the varieties grown in previous crops. CROFT (1989) showed that the Australian commercial germplasm contains a wide range of resistance to Pachymetra root rot. MAGAREY and MEWING (1994) showed that varietal resistance greatly influences soil inoculum density, which has been shown to correlate closely with disease levels (MAGAREY, 1989). Susceptible varieties lead to the build-up of high inoculum densities. MAGAREY and MEWING (1994) found it took 5 years for spore numbers to fall to 40% of the original population when sugarcane land was fallowed. Soil temperature is also important; the disease is favoured by temperatures between 25 and 30°C.

Economic importance

Pachymetra root rot can have a large effect on sugarcane yields. Experiments in northern Queensland in a susceptible variety showed a 40% reduction in yield attributable to Pachymetra root rot (MAGAREY, 1994). A trend towards resistance in commercial canes, selected under field conditions on yield characteristics, tends to confirm that the disease affected commercial yield over a wide part of the region. Yield losses in some other districts are likely to be less than 40%.

Control

Control is centred on varietal resistance. All commercial varieties are screened for resistance to the disease in the course of the plant improvement programme. Susceptible clones are discarded in the latter stages of the programme, based on the resistance rating obtained from glasshouse-based screening trials (CROFT, 1989). Parent canes are also screened, and the crossing programme is based on the selection of parents with mid-parent ratings with sufficient resistance to result in a suitable level of resistance in the seedling canes to match the level of disease within a district. An assay based on quantification of the oospores of the fungus in soil (MAGAREY, 1989) has allowed district-wide surveys to be undertaken to assess severity of the disease in each district.

Other controls examined include biocides (no biocides with commercial potential have been located), and minimum till planting of cane in the inter-

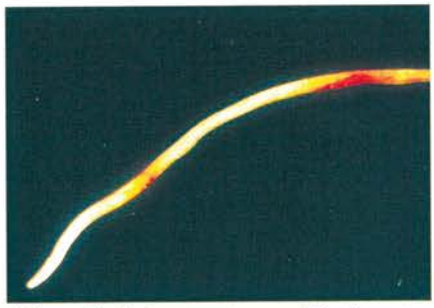


Figure 1. Early symptoms of Pachymetra root rot (R. Magarey).

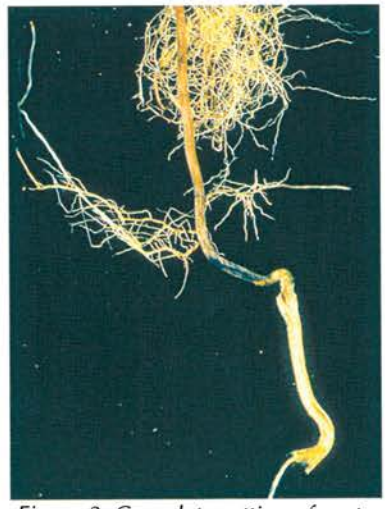


Figure 2. Complete rotting of root tip region (R. Magarey).

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

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

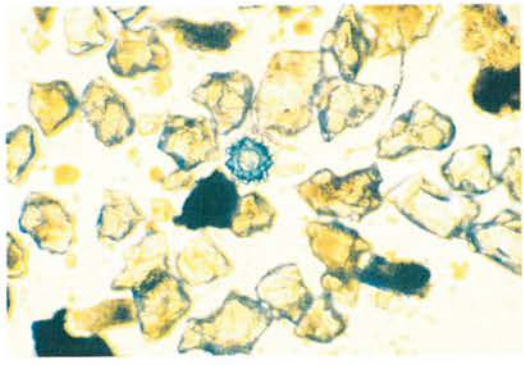


Figure 5. Blue-stained oospores of the fungus in soil extract (R. Magarey).

space to avoid high inoculum pressure resulting from the build-up of disease directly beneath the sugarcane stool. The latter has not been adopted commercially.

References

CROFT B.J., 1989. A technique for screening sugarcane cultivars for resistance to Pachymetra root rot. Plant Disease 73: 651–654.

CROFT B.J., MAGAREY R.C., 1984. Pathogenic fungi associated with Northern Poor Root Syndrome of sugarcane. Proceedings of the Australian Society of Sugar Cane Technologists Conference 6: 55–61.

CROFT B.J., MAGAREY R.C., 1989. A review of research into Pachymetra root rot, an important new fungal disease of sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 20: 686–694.

DICK M.W., CROFT B.J., MAGAREY R.C., CLARK G., 1989. *Pachymetra*, a new genus of the Verrucalvaceae (Oomycetes). Botanical Journal of Linnean Society 99: 97–113.

MAGAREY R.C., 1986. Symptoms and etiology of the root diseases caused by *Pythium graminicola* and an unidentified oomycete, in relation to the Poor Root Syndrome of sugarcane. Proceedings of the Australian Society of Sugar Cane Technologists Conference 8: 161–166.

MAGAREY R.C., 1989. Quantitative assay of *Pachymetra chaunorhiza*, a root pathogen of sugarcane in Australia. Phytopathology 79: 1302–1305.

MAGAREY R.C., 1991. Pachymetra root rot of sugarcane. PhD Thesis. Brisbane, Australia, University of Queensland, 150p.

MAGAREY R.C., 1994. Effect of Pachymetra root rot on sugarcane yield. Plant Disease 78: 475–477.

MAGAREY R.C., MEWING C.M., 1994. Effect of sugarcane cultivars and location on inoculum density of *Pachymetra chaunorhiza* in Queensland. Plant Disease 78: 1193–1196.

MAGAREY R.C., CROFT B.J., 1996. A review of root disease research in Australia. Proceedings International Society of Sugar Cane Technologists Congress 22: 505–513.

MAGAREY R.C., SOPER S.A., 1992. Involvement of Pachymetra root rot in stool tipping in varieties Q117 and Q124 in north Queensland. Proceedings of the Australian Society of Sugarcane Technologists Conference 14: 68–74.

MAGAREY R.C., TAYLOR P.W.J., RYAN C.C., 1987. Distribution of the root rot fungus involved in Poor Root Syndrome in cane fields from Ingham to Rocky Point. Proceedings of the Australian Society of Sugarcane Technologists Conference 9: 105–107.

PERRY J., 1985. Study of the taxonomy and host range of the fungal pathogens associated with poor root syndrome of sugarcane in Queensland. Undergraduate research project, Department of Botany. Brisbane, Australia, University of Queensland, 61p.

Pineapple disease

Jean-Claude Girard and Philippe Rott

Cause

Ceratocystis paradoxa (Dade) C. Moreau, ascomycete fungus; anamorph = *Thielaviopsis paradoxa* (de Seynes) F.X.R. von Höhnel.

Geographical distribution

Antigua, Argentina, Australia, Barbados, Belize, Brazil, Burkina Faso, China, Colombia, Congo, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, Fiji, 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, Swaziland, Tahiti, Taiwan, Thailand, Trinidad and Tobago, Uganda, Uruguay, USA, Venezuela, Vietnam, Zimbabwe; and probably in almost all countries where sugarcane is grown.

Symptoms

Pineapple disease is primarily a disease that causes rotting of sugarcane seed pieces or setts. Poor germination and/or wilting of the young sugarcane shoots at emergence in the field may indicate the presence of the disease. When the setts are cut longitudinally at an early stage of the disease, the internodal tissues are red in colour. 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 disease 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).

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 disease for black rot of sugarcane, caused by *Ceratocystis adiposa*, although the latter is rather uncommon (see the chapter 'Minor fungal diseases'). 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. 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 MORGAN-JONES (1967), SIVANESAN and WALLER (1986), WISMER (1961) and WISMER and BAILEY (1989).

Strains of the pathogen

There is little information available on variation of the pathogen. BYTHER and STEINER (1974, cited by WISMER and BAILEY, 1989) found little difference in virulence among isolates collected from sugarcane in Hawaii. 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). SASTRY *et al.* (1989) showed that three isolates of *C. paradoxa* from arecanut, coconut and sugarcane, respectively, were cross-pathogenic. However, they distinguished two groups on the basis of the proportion of macrospores to microspores produced *in vitro* and the symptoms on the three hosts; one group comprised the arecanut and coconut isolates, and the other group was formed by the sugarcane isolate.

Transmission

Pineapple disease 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 (MIRALLES 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).

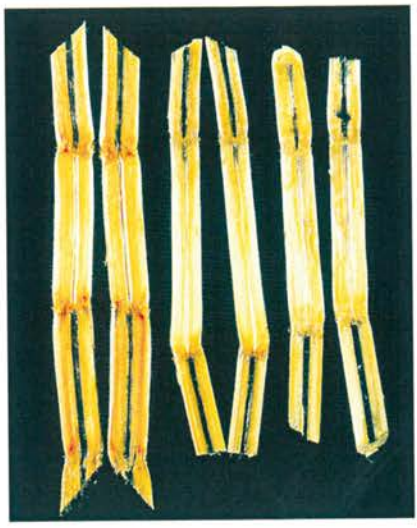


Figure 1. Black-coloured internodal tissues of setts (ISSCT).

Figure 2. Red and black discoloration of internodal tissues of setts (ISSCT).

Host range

Ceratocystis paradoxa can infect a variety of plants including arecanut, banana, cocoa, coconut, maize, oil palm, *Phoenix canariensis* and pineapple (MORGAN-JONES, 1967; WISMER, 1961).

Epidemiology

Factors which slow down germination of cuttings favour 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 disease if they are not associated with a fungicide treatment (WISMER, 1961).

Economic importance

Pineapple disease can cause considerable damage at emergence of sugarcane if the conditions are favourable 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 disease 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 several 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). RAID *et al.* (1991) felt 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 the susceptible cultivar CP74-2005.

A method aimed at the physical protection of setts from the pathogen was recently proposed by CROFT (1998): he demonstrated that polyethylene coating of short, hot water treated setts significantly improved the control of pineapple disease, especially when the setts are also treated with a fungicide.

Some promising experimental results were obtained when different species of *Trichoderma* and two species of *Gliocladium* were used as biological control agents for sugarcane pineapple disease (GUEVARRA, 1990; SAMPANG, 1991). The efficiency of these fungi on a larger scale has, however, yet to be proven.

References

CROFT B.J., 1998. Improving the germination of sugarcane and the control of pineapple disease. Proceedings of the Australian Society of Sugar Cane Technologists Conference 20: 300–306.

GUEVARRA M.M., 1990. Evaluation of *Gliocladium* spp. in the control of pineapple disease of sugarcane caused by *Ceratocystis paradoxa* (Dade) Moreau. Philippine Sugar Quarterly 1(4): 20–28.

MILLANES VIRELLES P., HERRERA ISLA L., 1994. Estudio de la supervivencia de *Thielaviopsis paradoxa* (de Seynes) Hon. sobre restos de caña de azúcar y piña en un suelo con carbonato típico. Centro Azúcar 21: 66–72.

MORGAN-JONES G., 1967. *Ceratocystis paradoxa*. Description of Pathogenic Fungi and Bacteria No. 143. Kew, UK, Commonwealth Mycological Institute, 2p.

RAID R.N., PERDOMO R., POWELL G., 1991. Influence of seedpiece treatment and seeding density on stalk population and yield of a pineapple disease susceptible sugarcane cultivar. Journal of the American Society of Sugar Cane Technologists 11: 13–17.

SAMPANG R.C., 1991. Biological control with *Trichoderma* spp. of pineapple disease of sugarcane caused by *Ceratocystis paradoxa*. Philippine Sugar Quarterly 2(5): 29–36.

SASTRY M.N.L., PATIL-KULKARNI B.G., HEDGE R.K., HIREMATH P.C., 1989. Studies on the disease caused by *Thielaviopsis* state of *Ceratocystis paradoxa* (Dade) Moreau on arecanut, coconut and sugarcane. 1. Morphological, cultural and cross inoculation studies of three isolates of *Ceratocystis paradoxa*. Mysore Journal of Agriculture Sciences 23: 44–48.

SIVANESAN A., WALLER J.M., 1986. *Ceratocystis* Ell. & Halsted. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 14–16. Slough, UK, CAB International.

WISMER C.A., 1961. Pineapple disease. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 223–245. Amsterdam, The Netherlands, Elsevier Publishing Company.

WISMER C.A., BAILEY R.A., 1989. Pineapple disease. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillapsie Jr and C.G. Hughes (Eds), p. 145–155. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

Pokkah boeng

Peter J.L. Whittle and Irawan

Cause

Gibberella fujikuroi (K. Sawada) H.W. Wollenweber, ascomycete fungus; anamorph = *Fusarium moniliforme* J.L. Sheldon.

Gibberella subglutinans (E. Edwards) P.E. Nelson, T.A. Tousson & Marasas, ascomycete fungus; anamorph = *Fusarium subglutinans* (H.W. Wollenweber & O.A. Reinking) P.E. Nelson, T.A. Tousson & Marasas.

Pokkah boeng is sometimes known as Fusarium pokkah boeng.

Geographical distribution

Andaman Islands, Angola, Antigua, 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, Ethiopia, Fiji, 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, Swaziland, Taiwan, Tanzania, Thailand, Togo, Trinidad, Uganda, Uruguay, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

The most obvious symptom is a malformed or twisted top, which gives this disease its name from the 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. Development of further symptoms is dependent on the susceptibility of the

Figure 1. Stalks affected by pokkah boeng (ISSCT).

Figure 2. Twisted top of sugarcane stalk (Irawan).

Figure 3. Twisted leaves with chlorotic areas (ISSCT)

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 (Figure 4). Ladder-like, necrotic lesions may develop on leaf sheaths and midribs. Infection in the spindle may reach the growing point and continue into the stalk. Dark, reddish streaks and ladder-like, necrotic lesions may form either inside the stalk or on the rind (Figure 5), over a length of a few centimetres and a width of about a quarter of the stalk's diameter. In some cases, lesions may penetrate across much of the stalk to give the appearance of a horizontal knife-cut (Figure 6). Most infected shoots grow away from the symptoms and recover, but sometimes, the growing point is killed (top rot) (Figure 7), resulting in shooting of the lateral buds.

Diagnosis

Usually, symptoms are restricted to the top distortion. The distinctive, ladder-like lesions and knife-cuts, or top rot tend to be restricted to severe cases. Superficially, the leaf distortion can resemble Fiji disease, but galls are not present. Widespread occurrence of symptoms during rainy weather suggests pokkah boeng.

Both the causal fungi are isolated readily in their anamorph states on a variety of standard growth media. The teleomorphs are rare. Identification to the

Fusarium genus is usually straightforward, from the presence of typical macroconidia produced in pale orange sporodochia (MARTIN *et al.*, 1989) and this will usually be adequate for diagnosis. However, identification of *Fusarium* species requires specific culturing procedures that give rise to consistent taxonomic characters (WINDELS, 1992).

Strains of the pathogen

The two pathogen species may occur together and act similarly. *Fusarium moniliforme* also causes a sugarcane sett and stem rot, but it is not clear whether this fungus is a different race to that which causes pokkah boeng.

Transmission

Conidia are wind-blown between localities and are deposited on plants, then washed by rain into infection sites. Pokkah boeng may be transmitted in cuttings, but normally this is of little economic importance.

Host range

Both fungi are widespread in the parts of the world where sugarcane is grown. They infect a wide range of species including monocotyledons and dicotyledons, causing various diseases such as seedling blight, stalk and root rot, abnormal stunting or hypertrophy (MARTIN *et al.*, 1989). Some hosts may be significant alternative hosts for the sugarcane pathogen.

Epidemiology

Pokkah boeng is favoured by warm, moist growing conditions, with symptom development typically beginning early in the rainy season during rapid and vigorous growth periods. Conidia enter the spindle along the margin of partly unfolded leaves and are carried by water to the base of the spindle where they germinate (BOLLE, 1936; VAN DILLEWIJN, 1950). The mycelium 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).

The incidence and severity of pokkah boeng can vary greatly between years, escalating when susceptible varieties are common and seasonal conditions are favourable.

Economic importance

While outbreaks of pokkah boeng can appear spectacular, they usually are of little economic importance. Although some stalks may die, the production of

Figure 4. Twisted leaf with reddish tissue (ISSCT).

Figure 5. Necrotic lesions on the rind of a diseased stalk (BSES).

Figure 6. Horizontal knife-cut lesion of diseased stalk (BSES).

Figure 7. Cane top killed by pokkah boeng (BSES).

new and unaffected leaves generally allows stalks to recover. Nevertheless, serious epidemics have taken place in very susceptible varieties. In Java, up to 38% of affected stalks of POJ 2878 died (MARTIN *et al.*, 1989). Sometimes, outbreaks in new seedling varieties have occurred and these varieties have been withdrawn before economic losses could occur.

Control

The use of resistant varieties is the only satisfactory method of control. Mostly, this is accomplished by eliminating susceptible seedlings during field selection. However, at the Indonesian Sugar Research Institute (ISRI), Pasuruan, seedlings are tested 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).

Disease-free seed cane may need to be chosen where infection is high. Chemical control using fungicide can reduce the percentage infection of pokkah boeng, but has never been applied on a commercial scale due to the high cost.

References

BOLLE P.C., 1936. Idem. Bijdrage II. Verh. v.d. Leden Proefst. Java-suikerind. 18: 857–886.

HAN LIOE HONG, 1956. Fusarium-pokkah boeng resistance trial. Proceedings International Society of Sugar Cane Technologists Congress 9: 1023–1029.

LYRENE P.M., DEAN J.L., JAMES N.I., 1977. Inheritance of resistance to pokkah boeng in sugarcane crosses. Phytopathology 67: 689–692.

MARTIN J.P., HAN LIOE HONG, WISMER C.A., 1961. Pokkah boeng. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 247–261. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., HANDOJO H., WISMER C.A. (revised by H. HANDOJO), 1989. Pokkah boeng. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 157–168. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

VAN DILLEWIJN C., 1950. Fusarium-pokkah boeng. Proceedings International Society of Sugar Cane Technologists Congress 7: 473–498.

WINDELS C.E., 1992. *Fusarium. In*: Methods for Research on Soilborne Phytopathogenic Fungi. L.L. Singleton, J.D. Mihail and C.M. Rush (Eds), p. 115–128. St. Paul, USA, American Phytopathological Society.

Pythium root rot

Jeffrey W. Hoy

Cause

Pythium arrhenomanes Drechsler, oomycete fungus; *Pythium* spp.

Pythium arrhenomanes Drechsler 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). Another species, *P. graminicola* Subramanian, closely resembles *P. arrhenomanes* in morphology and ecology, and confusion over identification of isolates exists in the literature. The two have now been demonstrated to be distinct species (CHEN and HOY, 1993), and in pathogenicity tests, *P. graminicola* was only weakly pathogenic to sugarcane.

Geographical distribution

Widely distributed; 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 cultivars. Leaf chlorosis, curling and necrosis, wilting and plant death are rarely observed. 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. *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 1 and 2). 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 colour (HOY and SCHNEIDER, 1988a; MAGAREY, 1984) (Figure 3). Lesions on primary roots where lateral roots once attached are elongate (1–3 × 1–2 mm) and red-black in colour. Primary roots also may show general reddish-black discoloration.

Diagnosis

Certain diagnosis of Pythium root rot is 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. Isolation and positive identification of the pathogen requires agar medium and expertise in the morphology of *Pythium* species reproductive structures. Isolation of *Pythium* is most easily achieved on selective agar medium; however, water agar also may be used. The medium used by the author contains 10 g each of corn meal agar and agar autoclaved in 1 l deionized water amended after cooling to 45°C with 300 mg each of vancomycin and spectinomycin dissolved in 10 ml sterile water, 15 mg pentachloronitrobenzene dissolved in 95% ethanol, and 0.4 ml of pimaricin (10 mg a.i.). Other less expensive selective media have utilized rifampicin (25 ppm) with pimaricin (2.5%) or sodium ampicillin (300 mg/l). 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. To identify *P. arrhenomanes*, characteristic sexual and asexual reproductive structures (VAN DER PLAATS-NITERINK, 1981) must be produced and observed (Figure 4). With experience, it is not difficult to identify the pathogen. However, initial investigations will probably require the co-operation of an expert for positive identification.

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 cultivar 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. Primary inoculum

Figure 1. Sugarcane root system affected by Pythium root rot showing damage to roots resulting from infection and reduction in number of shoots produced (left), and healthy root system (right) (J. Hoy).

Figure 2. 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 3. Rotted root tip showing water-soaked, flaccid tip with red-brown advancing lesion margin (J. Hoy).

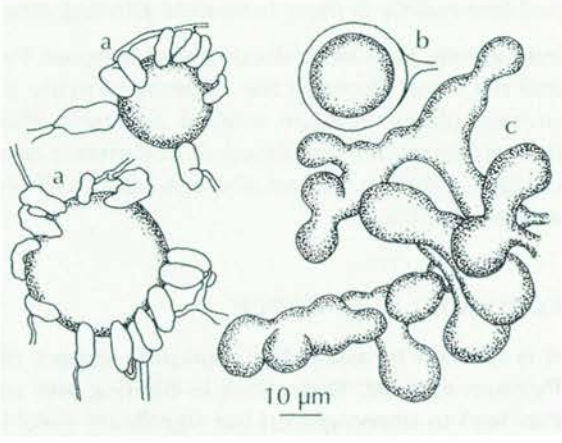


Figure 4. Sexual and asexual reproductive structures of Pythium arrhenomanes*: a. oogonia with multiple antheridia; b. oospore; c. inflated, lobate sporangium (Redrawn with permission from A.J. van der Plaats-Niterink, 1981, by B.R. Corns).*

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.

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 (DISSANAYAKE *et al.*, 1997). Roots of Johnson grass (*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

The amount of damage caused by Pythium root rot depends on cultivar susceptibility, growth conditions, and the inoculum potential of the pathogen. Zoospore production and dispersal are favoured 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 favourable for growth and sporulation of the pathogen but unfavourable 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

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% have been obtained with treatments of the fungicide metalaxyl in Louisiana, but overall, treatment results have been erratic (HOY and SCHNEIDER, 1988b). The total body of evidence suggests that in regions where sugarcane must persist through periods with conditions unfavourable 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

Cultivar 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 cultivars 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 programmes are currently screening for disease resistance. Instead, most industries are relying on the generally improved resistance of the interspecific hybrids. Knowledge about the heritability of resistance and efficiency of selection are unknown.

The prospects for successful chemical 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, and sub-surface drainage during the winter months has increased sugarcane yields. In the absence of better control measures, growing interspecific hybrid cultivars under the best possible drainage conditions will minimize the damage from Pythium root rot.

References

ADAIR C., 1972. Ecological relationships of *Pythium* species in the rhizosphere of sugarcane and possible significance in yield decline. Hawaiian Planters Record 58: 213–240.

CHEN W., HOY J.W., 1993. Molecular and morphological comparisons of *P. arrhenomanes* and *P. graminicola*. Mycological Research 97: 1371–1378.

CROFT B.J., 1988. First report of *Pythium myriotylum* on sugarcane in Australia. Plant Disease 72: 1077.

DISSANAYAKE N., HOY J.W., GRIFFIN J.L., 1997. Weed hosts of the sugarcane root rot pathogen, *Pythium arrhenomanes*. Plant Disease 81: 587–591.

HOY J.W., SCHNEIDER R.W., 1988a. Role of *Pythium* in sugarcane stubble decline: Pathogenicity and virulence of *Pythium* species. Phytopathology 78: 1688–1692.

HOY J.W., SCHNEIDER R.W., 1988b. Role of *Pythium* in sugarcane stubble decline: Effects on plant growth in field soil. Phytopathology 78: 1692–1696.

KOIKE H., 1965. Methods for testing sugarcane for resistance to Pythium root rot. Proceedings International Society of Sugar Cane Technologists Congress 12: 1183–1187.

MAGAREY R.C., 1984. Glasshouse studies on the symptoms and etiology of Northern poor root syndrome of sugarcane. Proceedings of the Australian Society of Sugar Cane Technologists Conference 6: 51–54.

RANDS R.D., 1961. Root rot. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 289–309. Amsterdam, The Netherlands, Elsevier Publishing Company.

RANDS R.D., DOPP E., 1938. Pythium root rot of sugarcane. United States Department of Agriculture, Technical Bulletin No. 666.

SRINIVASAN K.V., 1968. The role of rhizosphere microflora in the resistance of sugarcane to Pythium root rot. Proceedings International Society of Sugar Cane Technologists Congress 13: 1224–1236.

VAN DER PLAATS-NITERINK A.J., 1981. Monograph of the genus *Pythium*. Studies in Mycology 21: 1–242.

Ramu orange leaf

Sidney Suma and Robert C. Magarey

Cause

An unidentified fungus (belonging to the order Exobasidiales).

Geographical distribution

The disease has only been observed in Papua New Guinea.

Symptoms

Disease symptoms are first apparent as the spindle leaf unrolls, 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 look like downy mildew, though the streaks are thinner. The margins of affected leaves remain green.

With time, symptoms change from pale-green to yellow (Figure 2) and finally to yellow-orange (Figure 3). Symptoms are more pronounced on the lower leaf surface, especially once the pathogen has sporulated. The orange-coloured spores are produced on erumpant 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.

With further development, older leaves will 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 4).

Diagnosis

Diagnosis relies on the recognition of leaf symptoms (MAGAREY, 1996).

Figure 1. Very fine leaf stripes running parallel to the leaf venation (R. Magarey).

Figure 2. Yellow leaf stripes (P. Rott).

Figure 3. Yellow-orange leaf stripes (S. Suma).

Figure 4. Diseased stool (S. Suma).

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*), *S. edule* and interspecific 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 thoughout 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.

References

Magarey R.C., Suma S., Egan B.T., 1995. New sugarcane diseases observed in commercial cane at Ramu Sugar Estate, Papua New Guinea. Proceedings International Society of Sugar Cane Technologists Congress 22: 472–476.

Magarey R.C., 1996. Downy mildew, Ramu stunt and Ramu orange leaf. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 113–115.

Suma S., Pais E., 1996. Major diseases affecting sugarcane production on Ramu sugar estate, Papua New Guinea. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 30–33.

Red leaf spot (purple spot)

Barry J. Croft

Cause

Dimeriella sacchari (van Breda de Haan) Hansford, ascomycete fungus.

Geographical distribution

Australia, Bangladesh, Colombia, Cuba, Fiji, Guatemala, Indonesia, Japan, Mexico, Nepal, Panama, Papua New Guinea, Philippines, Taiwan, Tanzania, Thailand, Trinidad, 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 (Figure 1). In some cases, the clusters of red lesions have a distinctly rhizoid appearance. The lesions are more common on older leaves and in severe cases the lesions can coalesce to form red patches (Figure 2). 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 colour 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 3). 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 1. Red leaf spots (B.J. Croft).

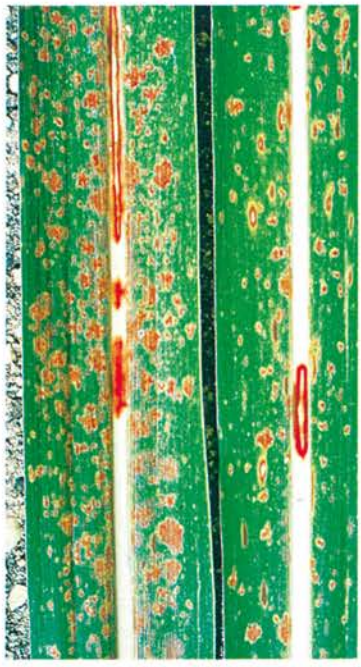


Figure 2. Red leaf spots and patches (B.J. Croft).

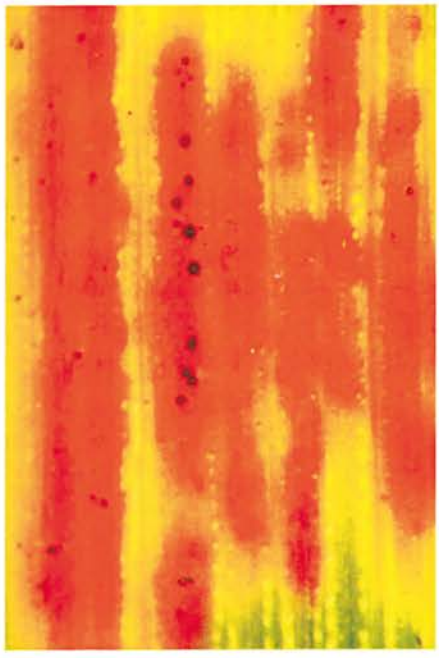


Figure 3. Red leaf lesion showing globose perithecia of the pathogen (B.J. Croft).

Strains of the pathogen

No information is available.

Transmission

Spread of the fungus is presumably by wind or rain-borne spores.

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.

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.

References

ABBOTT E.V., 1964. Red leaf spot (Purple spot). *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 43–44. Amsterdam, The Netherlands, Elsevier Publishing Company.

RYAN C.C., BIRCH R.G., 1980. Red leaf spot of sugarcane caused by *Dimeriella sacchari* in Australia. Plant Disease 64: 501–502.

Red rot

R.P. Singh and Sunita Lal

Cause

Glomerella tucumanensis (Spegazzini) von Arx & E. Muller, ascomycete fungus; anamorph = *Colletotrichum falcatum* F.A. Went.

Geographical distribution

The disease is widespread: Afghanistan, Angola, Antigua, 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, 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, Swaziland, Taiwan, Tanzania, Thailand, Togo, Trinidad, Uganda, Uruguay, USA, Vanuatu, Vietnam, Zimbabwe.

Symptoms

The red rot pathogen can infect seed pieces or cuttings, standing stalks, leaf sheath, lamina and midribs, but rotting of the stalk is of primary importance (Abbott and Hughes, 1961; Singh and Singh, 1989). On infection, whether due to seed-borne inoculum or otherwise, the affected plants show some purplish discoloration on the external surface of the internodes near the point of infection (Figure 1). Later on, yellowing and drying of foliage are observed. However, classic diagnostic symptoms are observed when diseased canes are split longitudinally (Figure 2). In split stalks of susceptible varieties, internodal tissues show a red discoloration interrupted by whitish patches perpendicular to the axis of the stalk (Figure 3). These stalks emit a slightly acidic and starchy odour. The white patches or spots vary in size and appearance. Sometimes the affected tissues appear mottled. The nature of the white spots is also related to

host resistance/susceptibility. In comparatively resistant varieties, the white spots are absent and only the central pith shows red streaks. The nodal tissues of infected stalks may rot and this is related to host reaction.

On the leaves, the pathogen produces elongate red spots on the upper surface of the midribs. They can remain small or elongate along the midrib (Figure 4). Initially, the spots are dark red, then become straw-coloured with purple margins that may be covered with small black flecks which are the fruiting bodies of the fungus (acervuli). 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 red rot causal organism was reclassified by VON ARX AND MULLER (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 isolates. In general, they are bulbous at the base tapering towards the tip, measure 100–200 × 4 µm. Conidia germinate producing appressoria. Sometimes appressoria are produced on hyphal tips. These appressoria are smooth but thick-walled and cinnamon-buff in colour. The perithecia, when produced, 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.

Strains of the pathogen

Genetically different strains of the red rot pathogen occur in nature (SINGH *et al.*, 1992). Pathogenic strains to different varieties may arise independently from genetically different groups. Pathogenic changes in existing isolates take place under host pressure selection during favourable environmental conditions for disease development. Different pathotypes were identified (SATYANARAYANA and SATYANARAYANA, 1976; KHIRBAT *et al.*, 1980). Isolates also show variations in pathogenic behaviour in respect to the stalk and foliage. Stalk isolates may cause infection on the foliage but isolates from the midrib generally do not infect stalks.

Figure 1. Discoloration on the external surface of internodes (R. Viswanathan, BSES).

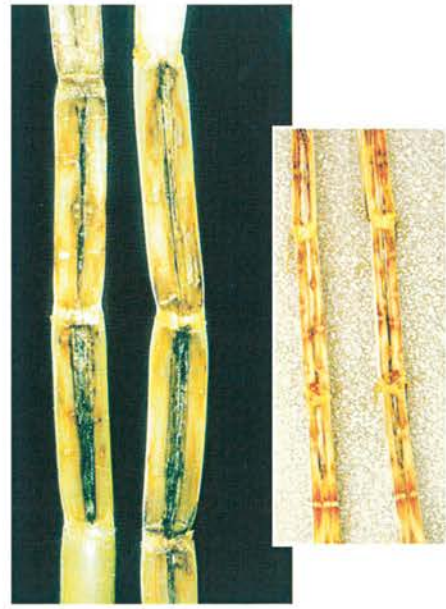


Figure 2. Red discoloration of internal stalk tissue (BSES, G.P. Rao).

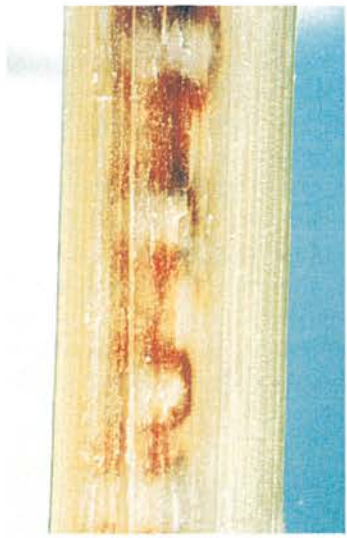


Figure 3. White spots in diseased stalk (J. Hoy).

Transmission

Red rot is spread primarily through planting 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, developing crops. Though the fungus is not soil-borne it remains viable for 1 to 2 months. Spores washed down in the soil may cause infection in the setts planted during that period. Perithecia produced on old dried leaves may provide ascosporic inoculum for new infections but its importance is not known.

In addition to planting infected seed cane, inoculum is disseminated by rain, heavy dews, irrigation water and wind. The conidia produced on cane rind can easily be washed down and 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 conidiospore matrix and/or ascospores.

Host range

Besides sugarcane, the fungus has been isolated from sorghum and Johnson grass leaves. 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 (SINGH *et al.*, 1983 and 1988). Red rot is most destructive during the rainy season from July to September in tropical and subtropical regions. In Australia, red rot is more severe in cane suffering from drought or waterlogging. Under low temperature and less humid conditions, even the susceptible varieties show some degree of resistance. This results in the development of the dormant state of nodal infections. In the case of planted setts, disease is favoured by both high and low soil moisture content where plant germination is affected but not the pathogen. At times, the attack by secondary pathogens like *Fusarium subglutinans* or *Acremonium implicatum* enhances the magnitude of losses.

Economic importance

Red rot has caused much damage in subtropical areas even including complete crop loss. These losses are caused by the degradation of the cuttings and stools, death of the stalks and stools (Figure 5), and reduction in sugar content and cane purity.

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. 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

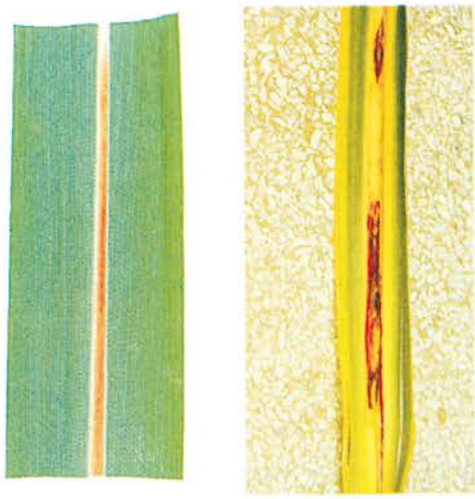


Figure 4. Red leaf midrib (W.H. Hsieh, G.P. Rao).

Figure 5. Stools killed by red rot (R. Viswanathan).

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. Virulent isolates from genetically different groups of the pathogen should be used for screening. Five to six-months-old plants are inoculated at the nodes or in internodal tissues of standing canes.

No fungicidal treatment has been effective in controlling seed-borne infection. However, secondary spread of the disease can be minimized by spraying the crop with fungicides during the monsoon season (July to September) in India. The disease has been effectively managed by using healthy seed cane. Thermotherapy of the cuttings can eliminate the pathogen borne in seed cane. A moist hot air treatment at 54°C for 2 h is used in India (SINGH *et al.*, 1980). The use of healthy seed cane produced from moist hot air treated seed material

coupled with some other prophylactic measures, has proved 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. Appropriate planting times and proper soil conditions at the time of planting help in germination and minimize rotting setts due to seed-borne infection. Timely roguing of diseased plants, burning of trash, avoiding ratoons in the affected fields, and crop rotation are necessary to manage the disease and prevent secondary spread. Avoiding stress such as waterlogging and drought can reduce the risk of serious epidemics of red rot.

References

ABBOTT E.V., HUGHES C.G., 1961. Red rot. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 262–287. Amsterdam, The Netherlands, Elsevier Publishing Company.

AZAB Y.E., CHILTON S.J.P., 1952. Studies on inheritance of resistance to red rot disease of sugarcane. Phytopathology 42: 282.

KHIRBAT S.K., SATYAVIR, BENIWAL M.S., 1980. Physiological and pathological variability in sugarcane red not pathogen *Colletotrichum falcatum* Went in Haryana. Indian Phytopathology 33: 296–299.

SATYANARAYANA Y., SATYANARAYANA B., 1976. Variation in the light isolates of red rot fungus in Andhra Pradesh. Cooperative Sugar 7: 389–392.

SINGH K., SINGH R.P., 1989. Red rot. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 169–188. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SINGH K., MISRA S.R., SHUKLA U.S., SINGH R.P., 1980. Moist hot therapy of sugarcane: control of sett-borne infections of G.S.D., smut and red rot. Sugar Journal 43(5): 26–28.

SINGH R.P., LAL SUNITA, SINGH K., 1983. Development of dormant nodal infections of *Colletotrichum falcatum* Went in sugarcane. Proceedings Annual Convention of Sugar Technologists' Association of India, p.Ag. 79–90.

SINGH R.P., LAL SUNITA, SINGH K., 1988. Effect of ambient temperature on red rot development in sugarcane. Indian Phytopathology 41(1): 86–91.

SINGH R.P., LAL SUNITA, MISRA S.C., 1992. Identification of genetically different isolates of red rot pathogen. *In*: Annual Report of the Indian Institute of Sugarcane Research, Lucknow, p. 50–59.

SPEGAZZINI C., 1896. Hongos de la caña de azúcar. Review Faculty Agronomy y Veterinary 2: 227–258.

VON ARX J.A., MULLER E., 1954. Die Gattungen der Amerosporen Pyrenomyceten. Beitr. Kryptogamenflora Schweiz. 11: 5–434.

WENT F.A.F.C., 1893. Het Rood Snot. Archief voor de Java Sukerindustrie 1: 265–282.

Red rot of the leaf sheath

Claude Ricaud

Cause

Corticium rolfsii 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, Uganda, USA, Vietnam, Zimbabwe.

Symptoms

The disease affects the lower leaf sheaths causing an orange-red discoloration over irregular areas, sharply delineated from the healthy tissue (Figure 1). The fungus develops through the tissue to the inner leaf sheaths, binding them slightly together. Fungal mycelium grows between the leaf sheaths over the infected areas with small, round, 1 mm diameter, brownish sclerotia. Some invasion of the rind beneath the infected areas of the sheath also occurs giving a light brown discoloration with distinct margins as on the sheaths. When the stalks mature, affected cells become necrotic and dry, resulting in shallow cankers. In advanced stages, the leaf sheaths become necrotic and disintegration of tissues between the veins may occur, leaving the vascular bundles exposed. Leaf blades may show a slight discoloration and premature drying (TAKASHI MATSUMOTO and ABBOTT, 1964).

Diagnosis

The fungus can be recognized by the sclerotia and absence of typical fructifications of other sheath-invading fungi such as *Cytospora sacchari* and *Glomerella tucumanensis*. It can be isolated on potato dextrose agar. Cultural and physiological characteristics have been described by MORDUE (1974).

Figure 1. Delineated orange-red discoloration of leaf sheath (Taiwan Sugar Corporation, ISSCT).

Strains of the pathogen and host range

Corticium rolfsii is a widely distributed fungus that infects several plant species. There is little evidence of host specialization.

Transmission and epidemiology

The fungus is a facultative parasite and survives in the soil, on crop residues and weed hosts. The disease is favoured by warm, wet weather.

Economic importance

The disease is not of economic importance.

Control

No control measure is warranted.

References

TAKASHI MATSUMOTO, ABBOTT E.V., 1964. Red rot of the leaf sheath. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 45–48. Amsterdam, The Netherlands, Elsevier Publishing Company.

MORDUE J.E.M., 1974. *Corticium rolfsii*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 410. Kew, UK, Commonwealth Mycological Institute.

Red spot of the leaf sheath

Claude Ricaud

Cause

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, USA, Venezuela, Vietnam, Zimbabwe.

Symptoms

Ellipsoid bright red spots are present on the leaf sheaths, sharply delimited from the normal green colour of the surrounding tissues (Figure 1). The spots enlarge and coalesce to form irregular reddish lesions which penetrate through to the inner sheaths (Figure 2). In later stages a greyish-green mould 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 mould. Conidiophores are olivaceous brown, branched, 1–5 septate, 40–120 × 3–5 µm. Conidia are hyaline to straw-coloured, 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.

Figure 1. Ellipsoid bright red spots on leaf sheaths (ISSCT).

Figure 2. Reddish lesions on inner leaf sheath (ISSCT).

Transmission

The disease spreads by air-borne conidia.

Host range

No other host of the fungus has been reported.

Epidemiology

The disease is favoured 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.

References

ABBOTT E.V., 1964. Red spot of the leaf sheath. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 49–50. Amsterdam, The Netherlands, Elsevier Publishing Company.

KIRK P.M., 1982. *Mycovellosiella vaginae*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 725. Kew, UK, Commonwealth Mycological Institute.

SIVANESAN A., WALLER J.M., 1986. *Mycovellosiella* Rangel. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 47–49. Slough, UK, CAB International.

Rind disease and sour rot

Jack C. Comstock and Roger A. Bailey

Cause

Phaeocytostroma sacchari (J.B. Ellis & Everhart) B. Sutton, coelomycete fungus.

Geographical distribution

Andaman Islands, Angola, Antigua, Australia, Barbados, Belize, Bolivia, Brazil, China, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Egypt, Fiji, 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, Tahiti, Taiwan, Tanzania, Thailand, Trinidad, Uganda, USA, Vietnam, Zimbabwe; probably all sugarcane producing countries.

Symptoms

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 and 2). The pustules can also occur on the leaf sheaths and midribs. Severely affected stalks appear straw-coloured and shrivelled. Internally, an orange-brown rotting occurs, initially at the nodes and later extending through the internode tissues, and may turn dark later (Figure 3). When severe, entire stalks may be rotted (Figure 4). Affected stalks often have a distinctive sour odour (GOODALL *et al.*, 1999). Leaf yellowing and premature desiccation may occur (ABBOTT *et al.*, 1964).

Diagnosis

The black, coiled, thread-like spore masses extruding from the black pustules on infected stalks are diagnostic (Figure 5). Conidia are pale brown, single celled, ellipsoidal to ovoid, and measure 10–15 × 3–4 µm (ABBOTT *et al.*, 1964; SIVANESAN and WALLER, 1986; SUTTON and WATERSTON, 1966).

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

Phaeocytostroma sacchari is very common but is usually regarded as a weak pathogen, damaging 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, 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

Rind disease aggravates crop deterioration by reducing the juice quality of stalks that are weakened for other reasons. The entire stalk may rot in these situations. Since *P. sacchari* is a weak pathogen, it seldom causes direct economic damage by itself. Severe damage can result if mature crops are subjected to prolonged drought. 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).

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.

Figure 1. Sugarcane stalk showing short spore masses on the rind (J.C. Comstock).

Figure 2. Spore masses of the pathogen erupting from the rind of an infected stalk (R.A. Bailey)

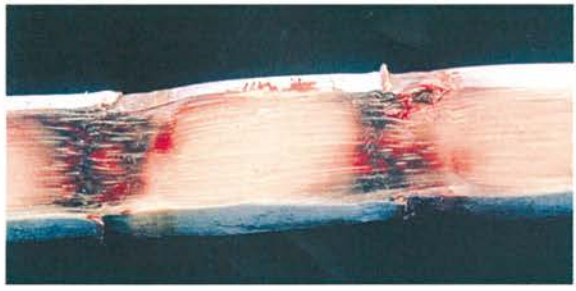


Figure 3. Internal dark discoloration at the sugarcane stalk node (J.C. Comstock).

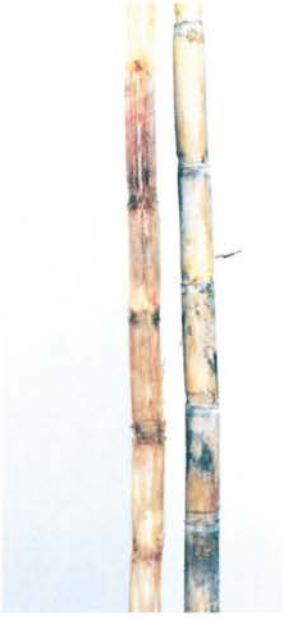


Figure 4. Orange-red rot of a cane affected by the stalk rot phase (R.A. Bailey).

Figure 5. Long thread-like coiled tendrils of the pathogen spore masses extending from a stalk (J.C. Comstock).

References

ABBOTT E.V., WISMER C.A., MARTIN J.P., 1964. Rind disease. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 124–130. Amsterdam, The Netherlands, Elsevier Publishing Company.

GOODALL J.L., MCFARLANE S.A., ROUX C., 1999. An unusual stalk rot caused by *Phaeocytostroma sacchari* in the KwaZulu-Natal Midlands. Proceedings South African Sugar Technologists Association Congress 74 (in press).

SIVANESAN A., WALLER J.M., 1986. *Phaeocytostroma* Petrak. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 54–56. Slough, UK, CAB International.

SUTTON B.C., WATERSTON J.M., 1966. *Phaeocytostroma sacchari*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 87. Kew, UK, Commonwealth Mycological Institute.

Ring spot

Barry J. Croft

Cause

Leptosphaeria sacchari van Breda de Haan, ascomycete fungus.

Geographical distribution

Andaman Islands, Angola, Antigua, Argentina, Australia, Bangladesh, Barbados, Belize, Benin, Bolivia, Brazil, Burkina Faso, Burundi, Cambodia, Cameroon, Central African Republic, China, Colombia, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Egypt, El Salvador, Fiji, 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, 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-coloured with a well-defined red-brown margin (Figure 1). 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, 1–5 × 4–18 mm. Ring spot is more common on older leaves but can also affect young leaves (Figure 2). On older spots the small black fruiting bodies of *L. sacchari* are usually present.

Diagnosis

The characteristic ring-shaped lesions and the presence of the fruiting bodies of *L. sacchari* are usually sufficient for diagnosis of the disease. The perithecia

of *L. sacchari* 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).

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.

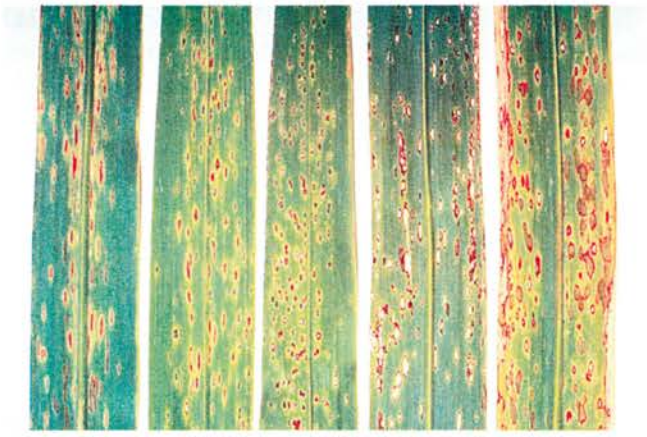


Figure 1. Various leaf lesions of ring spot (B.J. Croft).

Figure 2. Leaves severely affected by ring spot (B.J. Croft).

Host range

Sugarcane is so far the only reported host plant for ring spot.

Epidemiology

Ring spot can be seen throughout the year but is not common on young cane.

Economic importance

Ring spot is not considered to be economically important.

Control

No control is warranted. Varieties differ in susceptibility to the disease.

References

ABBOTT E.V., 1964. Ring spot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 53–58. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Leptosphaeria* Cas. & de Not. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 30–34. Slough, UK, CAB International.

Root and basal stem rot

Jing-Gwo Fang and Ching-Shiou Lee

Cause

The disease is caused by two species of *Xylaria*: *Xylaria* cf. *warburgii* Hennings (in Taiwan) and *X. arbuscula* P.A. Saccardo (in the USA and Puerto Rico) (ANONYMOUS, 1960), ascomycete fungi.

Geographical distribution

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. The symptoms are rarely found in young sugarcane. Root and basal stem tissues of diseased plants are rotted (Figures 1 and 2), and basal stem tissues appear light brown and reddish (HSIEH, 1980). Black lines are usually found in the diseased stem (Figure 3), 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 4). 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 5). The stunted plants contain fewer stalks that are shorter and thinner than normal. Eventually, diseased plants die.

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 1 and 2). Cultural characteristics of the pathogen on common fungal growth media, such as potato dextrose agar and oatmeal agar, can also be used to diagnose the

Figure 1. Initial white rot symptoms on basal stem (J-G. Fang).

Figure 2. White rot symptom on the basal part of harvested cane stem (J-G. Fang).

Figure 3. Black lines formed on basal stem (J-G. Fang).

Figure 4. White-tipped stroma formed on ratoon cane stubble (J-G. Fang).

Figure 5. Poor germination of ratoon cane caused by Xylaria *cf.* warburgii *(J-G. Fang).*

disease. A typical culture of the pathogen would consist of a mosaic-like, black and white mycelial mat with a number of thin rhizomorphs (HSIEH, 1980; FANG and LEE, 1995; MARTIN, 1967; ROGERS, 1979).

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). 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.* cf. *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 the pathogen: barbate windmillgrass (*Chloris barbata*), Bermuda grass (*Cynodon dactylon*), satintial (*Imperata cylindrica*) and nutgrass (*Cyperus rotundus*). Maize (*Zea mays*) and sorghum (*Sorghum bicolor*) also are highly susceptible to the pathogen (FANG and LEE, 1994).

Epidemiology

Root and basal stem rot of sugarcane is a major production constraint of sugarcane in some regions of Taiwan. The disease was first reported by HSIEH (1980) and was limited to the Yuching cane growing area until 1984, however, it has recently become epidemic (FANG *et al.*, 1994). The disease was found in cane fields at 8 of 21 sugarcane factories. The disease is more severe in fields with sandy or sandy loam soil.

Economic importance

Root and basal stem rot is an extremely important disease of sugarcane in certain localities of Taiwan. More than 700 ha of sugarcane are infected by the

pathogen 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).

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. 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). 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. Efforts to find organisms antagonistic to the pathogen, with a view toward biological control of the disease, have not been successful, so far (Fang, unpublished).

References

ANONYMOUS, 1960. Index of Plant Diseases in the United States. US Department of Agriculture Handbook. No. 165. Washington DC, 531p.

FANG J.G., LEE C.S., HSIEH W.H., 1986a. Perfect state of root and basal stem rot of sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 19: 370–374.

FANG J.G., HSIEH W.H., HU C.H., LEE C.S., 1986b. Survival of *Xylaria* sp. in the soil and the effect of green manure application on disease incidence. Report of the Taiwan Sugar Research Institute 112: 21–26.

FANG J.G., LEE C.S., 1994. Host range of *Xylaria* cf. *warburgii*. Sugar Cane 2: 6–11.

FANG J.G., HSIEH W.H., LEE C.S., 1994. Root and basal stem rot – a new disease of sugarcane. *In*: Current Trends in Sugarcane Pathology. G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin, V.P. Agnihotri and C.T. Chen (Eds), p. 59–64. Pitampura, Delhi, India, International Books and Periodicals Supply Service.

FANG J.G., LEE C.S., 1995. Vegetative compatibility and virulence of *Xylaria* cf. *warburgii*, a causal organism of root and basal stem rot of sugarcane. Proceedings International Society of Sugar Cane Technologists Congress 21: 527–534.

HSIEH W.H., 1980. Root and basal stem rot of sugarcane: A new disease caused by *Xylaria* sp. Report of the Taiwan Sugar Research Institute 87: 15–25.

MARTIN P., 1967. Studies in the Xylariaceae. 1. New and old concepts. Journal of South African Botany 33: 205–240.

ROGERS J.D., 1979. The Xylariaceae: Systematic, biological and evolutionary aspects. Mycologia 71: 1–42.

Sclerophthora disease

Claude Ricaud

Cause

Sclerophthora macrospora (P.A. Saccardo) Thirumalachar, C.G. Shaw & M.J. Narasimhan, siphomycete fungus.

Geographical distribution

Australia, India, Mauritius, Peru, South Africa, USA.

Symptoms

Sclerophthora disease 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 sizes and shapes are found on them (Figure 2).

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 (Figure 3) and of chlorotic blotches of varying shapes and sizes. The streaks may turn reddish. When growth conditions are poor, the leaves are stiff and erect, but under favourable 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.

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 47 µm, 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.

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).

Figure 3. Leaves with irregular yellowish-white streaks (BSES).

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 maize and rice, 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 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.

References

HOLLIDAY P., 1980. Fungus Diseases of Tropical Crops. Cambridge, UK, Cambridge University Press, 607p.

ROTH G., 1967. *Sclerophthora macrospora* (Sacc.) Thirum. *et al.* (Syn.: *Sclerospora macrospora* Sacc.) on sugarcane in South Africa. Journal of Plant Diseases and Protection 74: 83–100.

SIVANESAN A., WALLER J.M., 1986. *Sclerophthora* Thirum., C.G. Shaw & Narasimhan. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 61–62. Slough, UK, CAB International.

STEINDL D.R.L., STEIB R.J., 1961. Sclerophthora disease. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 311–325. Amsterdam, The Netherlands, Elsevier Publishing Company.

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, Japan, Malawi, Mauritius, Mexico, Mozambique, Nicaragua, Pakistan, Panama, Peru, Philippines, Puerto Rico, South Africa, Sri Lanka, Taiwan, Thailand, Turkey, USA (Florida, Louisiana, Texas), Zimbabwe.

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 visible on the outside and inside of the sheath are characteristic symptoms of the disease (Figure 1). If infection is severe, practically the whole sheath turns reddish-brown and eventually becomes necrotic (Figure 2). Leaves dry out from the tip, droop downwards but remain attached to the stalk. Following necrosis of the sheath, fruiting bodies (pycnidia) of the fungus emerge from the plant tissue. The pycnidia are visible to the naked eye as black spines over the surface of the sheath (Figure 3). These structures are hard and make the sheath rough to the touch. Under humid conditions, droplets containing masses of spores of the fungus 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 field affected by the disease and severely affected stalks die (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 aboveground parts. Internal tissues turn greyish whereas a dull brown colour is visible on the outside. The disease tends to be more pronounced on older ratoon stubbles than 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).

Diagnosis

The fungus belongs to the subdivision Deuteromycotina and has been described by SIVANESAN (1983). The hyphal structure bearing the conidia are stromatic, separate, sub-epidermal, dark-brown to black and protrude 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. The pathogen can be grown on vegetable juice agar or potato dextrose agar at 28–30°C (CACERES and FORBES, 1965). Growth has been found to be best in the dark but pycnidia are more readily produced under illumination.

As the rind disease fungus (*Phaeocytostroma sacchari*) also attacks the same plant parts as *C. sacchari,* and both produce similar fruiting bodies, the two 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.*

Strains of the pathogen

No strains of the pathogen have been reported.

Transmission

The conidia of the fungus are spread by water and wind. CACERES and FORBES (1965) found that the pathogen can overwinter on dead sheath and stalk tissue in soil, and plants can be inoculated by burying infected sheaths in the soil. Inoculum, in the form of fungal growth on sterile barley seeds added to soil, also caused infection. They were also able to show by isolation that the fungus was present on leaf scars and dormant buds.

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.*

Figure 1. Brick-red discoloration of leaf sheaths (MSIRI).

Figure 2. Necrosis of leaf sheath (MSIRI).

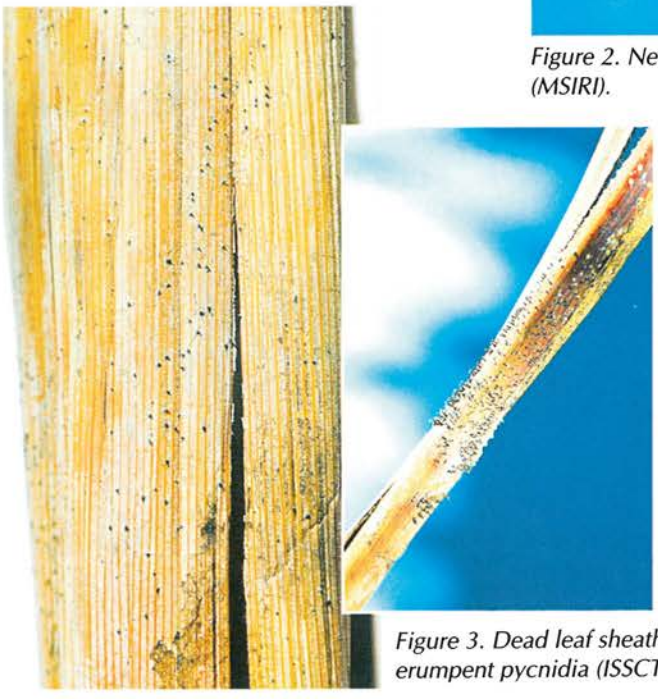


Figure 3. Dead leaf sheath with black erumpent pycnidia (ISSCT, MSIRI).

Epidemiology

The pathogen is considered to be a weak parasite, being able to invade damaged plant tissue or when the tissue is undergoing senescence. Stress (cold, poor drainage, moisture deficit) and the presence of other diseases (red rot, root rot) favour its development (EDGERTON, 1955).

Economic importance

The disease is of common occurrence but is not considered economically important. ABBOTT (1938) reported a reduction in yield of 5% due to mortality of tillers and poor growth of affected stalks.

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.

References

ABBOTT E.V., 1938. Cytospora rot of sugarcane in Louisiana. Proceedings International Society of Sugar Cane Technologists Congress 6: 447–457.

ABBOTT E.V., 1964. Sheath rot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 69–71. Amsterdam, The Netherlands, Elsevier Publishing Company.

AHMED H.U., KHAN M.A., RAHMAN M.H., 1979. Sheath rot disease of sugarcane in Bangladesh. Sugarcane Pathologists' Newsletter 22: 36–37.

CACERES S.F., FORBES I.L., 1965. Studies on Cytospora sheath rot of sugar cane and on the causal organism, *C. sacchari* Butl. Proceedings International Society of Sugar Cane Technologists Congress 12: 1130.

EDGERTON C.W., 1955. Sugarcane and its Diseases. Cytospora rot. p. 79–81. Baton Rouge, USA, Louisiana State University Press, 290p.

SIVANESAN A., 1983. *Cytospora sacchari*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 777. Kew, UK, Commonwealth Mycological Institute.

Smut

Jack C. Comstock

Cause

Ustilago scitaminea H. & P. Sydow, basidiomycete fungus.

Geographical distribution

Afghanistan, Antigua, Argentina, Australia (Western Australia only), 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, Ethiopia, Fiji, Gabon, Guadeloupe, Guatemala, Guyana, Haiti, Hawaii, Honduras, India, Indonesia, Iran, Iraq, Jamaica, Japan, Kenya, Madagascar, Malawi, Malaysia, Mali, Martinique, Mauritius, Mexico, Morocco, Mozambique, Myanmar, Nepal, Nicaragua, Niger, Nigeria, Pakistan, Panama, Paraguay, Peru (?), Philippines, Portugal, Puerto Rico, Réunion, St Kitts and Nevis, Senegal, Somalia, South Africa, Sri Lanka, Sudan, Swaziland, Taiwan, Tanzania, Thailand, Trinidad, Uganda, Uruguay, USA, Venezuela, Vietnam, Zambia, Zimbabwe.

Symptoms

Smut or culmicolous smut of sugarcane produces a whip-like sorus from either the terminal meristem or meristems of the lateral buds of infected stalks (Ferreira and Comstock, 1989) (Figures 1 and 2). The whips vary in size from only a few centimetres to large whips up to 1.5 m long extending high above the foliar canopy. The whips that extend from the lateral side shoots are usually smaller. The emerged whips are composed of a central core of host tissue surrounded by a thin layer of black spores that is covered by a thin silver-white membrane (Figure 3). This membrane quickly disintegrates exposing the spores and central core of host tissue. Older exposed whips that have been weathered usually lack spores since the spores are readily dispersed by wind and rain. Infected stools may appear grassy with an abnormally high number of small diameter stalks with terminal whips.

Besides the characteristic whips other common symptoms are stem galls, bud proliferation and serpentine-shaped whips. There have been reports of modified inflorescences but this is extremely rare (FERREIRA and COMSTOCK, 1989).

Diagnosis

Sugarcane smut is readily diagnosed by the characteristic whip-like sorus that is produced either from the apical meristem or from the lateral buds. Once the whips emerge no other verification is necessary. The dark brown to black teliospores are 5.5–7.5 µm in diameter and are minutely punctate. Detection of infected plants prior to whip emergence is possible using a molecular technique targeting the DNA unique to the smut pathogen, *U. scitaminea*. This technique relies on polymerase chain reaction to amplify a DNA fragment unique to *U. scitaminea*. This technique would be useful in quarantines (ALBERT and SCHENCK, 1996).

Strains of the pathogen

Smut races have been reported based both on observations and inoculation studies. Usually races are either indicated or suggested when a cultivar succumbs to smut after being grown for several years without being infected. Races have been reported in Hawaii, Pakistan, the Philippines and Taiwan (FERREIRA and COMSTOCK, 1989). A glasshouse test using seven sugarcane clones inoculated with six different isolates of *U. scitaminea* collected from different countries indicated different strains (GILLASPIE *et al.*, 1983). The smut reaction of certain cultivars differs between countries. A cultivar may be resistant in one country and susceptible in another indicating pathogenic variation. The presence of pathogenic races complicates the development of resistant cultivars.

Transmission

Sugarcane smut is transmitted aerially by wind-blown spores and by planting infected setts. The large terminal whips produce literally millions of spores daily which can be spread long distances as well as locally infecting sugarcane through the lateral buds (FERREIRA and COMSTOCK, 1989).

Host range

Saccharum hybrids and *Saccharum* spp. are natural hosts. Natural infection of *Imperata arundinacea* and *Erianthus saccharoides* and artificial inoculation of *Sorghum bicolor* and *Rottboellia exaltata* have been reported but are unimportant as far as sugarcane smut epidemiology is concerned.

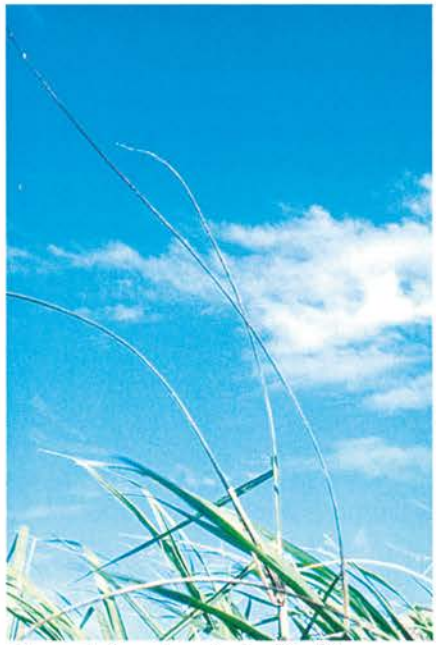


Figure 1. Long terminal whip-like sorus extending high above the sugarcane plant (J.C. Comstock).

Figure 2. Whip-like sorus produced from the terminal meristem of an infected stalk (ISSCT, P. Rott).

Figure 3. Black spores of a whip covered by a thin silver-white membrane (R. Fauconnier).

Epidemiology

Smut is generally favoured by hot dry weather conditions. High rainfall lessens the severity of smut development. Spore survival is decreased rapidly by soil moisture (Hoy *et al.*, 1993). Disease severity usually increases through ratoon crops but this varies with cultivar. The incidence of smut in some cultivars decreases in the ratoon crops. Plant stress increases frequency of whip development; cultivars that normally would not have whips may show symptoms under high stress. In subtropical areas the level of smut may decrease after severe winters; the severe winters probably cause smut infected plants to die (Hoy and Grisham, 1988).

Economic importance

Economically sugarcane smut has caused severe losses. Yield losses range from negligible to above 50%. Both cane tonnage and sugar recoverability are reduced. The disease may not cause any losses for many years and then reappear to cause extensive crop damage (Ferreira and Comstock, 1989).

Control

The best control practice is the use of resistant cultivars. Most variety development programmes screen cultivars for smut resistance where the disease is a threat. The heritability values for smut resistance are sufficiently high to make adequate progress in developing resistance. Most screening programmes rely on immersing seed pieces in an aqueous suspension of teliospores for inoculation. Spore concentrations vary from 2.5×10^6 to 5×10^8 teliospores/ml. Natural infection tests are probably the next often used method. In Taiwan, a bud puncture technique is used to inoculate lateral buds. While most countries use a 1 to 9 scale to rate the smut reaction of clones, different rating scales are used that vary in the percentage of whips for each grade (Ferreira and Comstock, 1989).

Some cultivars are more susceptible in artificial inoculation tests than in natural infection tests. Some of these 'susceptible' clones are sufficiently resistant for practical purposes and can be grown commercially without smut.

Other control practices are used to reduce smut incidence and reduce yield losses. Roguing diseased stalks/stools is used in some locations. Although roguing is not always effective in commercial fields or economically feasible, it can be of use in seed cane fields that have a low incidence of smut as a means of further reducing smut infected seed cane. The hot water treatment of seed cane can reduce and/or eradicate the smut pathogen in seed cane depending on the treatment. A 52°C treatment for 30 min was routinely used in Hawaii in the late 1970s and early 1980s to treat seed cane for commercial plantings in

smut-prone areas that used seed cane with a low smut incidence. This treatment eliminated the pathogen in 95% of the smut infected seed cane. Planting disease-free seed cane permitted harvesting the plant crop and one or more ratoons in smut-prone areas before the disease level caused substantial losses (COMSTOCK *et al.*, 1983).

For quarantine purposes a 52°C treatment for 45 min will essentially eradicate *U. scitaminea* in smut infected seed pieces and allow some buds to germinate.

References

ALBERT H.H., SCHENCK S., 1996. PCR amplification from a homolog of the *b*E mating-type gene as a sensitive assay for the presence of *Ustilago scitaminea* DNA. Plant Disease 80:189–192.

COMSTOCK J.C., FERREIRA S.A., TEW T.L., 1983. Hawaii's approach to control of sugarcane smut. Plant Disease 67: 452–457.

FERREIRA S.A., COMSTOCK J.C., 1989. Smut. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 211–229. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

GILLASPIE A.G. JR, MOCK R.G., DEAN J.L., 1983. Differentiation of *Ustilago scitaminea* isolates in greenhouse tests. Plant Disease 67: 373–375.

HOY J.W., GRISHAM M.P., 1988. Spread and increase of sugarcane smut in Louisiana. Phytopathology 78: 1371–1376.

HOY J.W., ZHENG J., GRELEN L.B., GEAGHAN J.P., 1993. Longevity of teliospores of *Ustilago scitaminea* in soil. Plant Disease 77: 393–397.

Sooty mould

Claude Ricaud

Cause

Capnodium sp. and *Fumago sacchari* Spegazzini, deuteromycete fungi; both are saprophytic fungi growing on honeydew associated with insect feeding: aphids, leaf hoppers, mealybugs and scale insects.

Geographical distribution

Argentina, Australia, Bangladesh, Brazil, Cuba, Guadeloupe, Guyana, Hawaii, India, Jamaica, Japan, Madagascar, Malaysia, Mauritius, Mozambique, Myanmar, Nicaragua, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Solomon Islands, South Africa, Swaziland, Taiwan, Thailand, Trinidad, Uganda, USA, Vietnam, Zimbabwe; probably most sugarcane growing countries.

Symptoms

Leaves and stalks of affected plants are covered with a black film or crust which appears as soot (Figures 1 and 2). Chlorotic patches on leaves may result under the covered areas. There is a sticky secretion from the insects associated with the disease. Following rain, the films are washed from the leaves (ABBOTT, 1964; MARTIN, 1938).

Diagnosis

Microscopic examination reveals the presence of fungal mycelium.

Strains of the fungus, transmission and host range

No information is available.

Epidemiology

Factors favouring 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.

Figure 1. Leaf covered with a black film (J. Daugrois).

Figure 2. Leaves showing sooty mould symptoms (J. Daugrois).

Economic importance and control

Although the fungi are not parasitic on the plant, they are slightly harmful to growth. Attacks are, however, sporadic and are not considered of economic importance. No treatment is usually required, except for material in multiplication plots when it may be necessary to control the insect infestations.

References

ABBOTT E.V., 1964. Sooty mould. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 72–73. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., 1938. Sugar Cane Diseases in Hawaii. Honolulu, Hawaii, Hawaiian Planters' Association Experiment Station, 295p.

Veneer blotch

Sidney Suma

Cause

Deightoniella papuana D.E. Shaw, adelomycete fungus.

Geographical distribution

The disease occurs in Indonesia, Papua New Guinea, Philippines and Solomon Islands.

Symptoms

The first sign of the disease is the development of small oval spots of a light green or straw colour 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-coloured and finally to light brown. 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 microscopic observa-

tion of the conidia shows that the mature conidia are globose to nearly ovoid, aseptate, minutely echinulate, very pale olivaceous brown in colour, 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. pupuana*.

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*).

Figure 1. Group of elongated leaf lesions surrounded by a thin reddish-brown margin (P. Rott, H. Koike).

Host range

The hosts of the disease include *S. officinarum, S. robustum, S. edule, 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.

References

KOIKE H., 1988. Sugarcane Diseases. A guide for field identification. Rome, Italy, Food and Agriculture Organization of the United Nations, 127p.

SHAW D.E., 1964. Veneer blotch. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 79–81. Amsterdam, The Netherlands, Elsevier Publishing Company.

SHAW D.E., 1984. Microorganisms in Papua New Guinea. Research Bulletin. Konedobu, Papua New Guinea, Department of Primary Industry, 314p.

White speck (white rash)

Claude Ricaud

Cause

Elsinoe sacchari T.C. Lo, ascomycete fungus; anamorph = *Sphaceloma sacchari* T.C. Lo.

Geographical distribution

Brazil, Cuba, Guam, Hawaii, Japan, Malaysia, 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 (Figure 2). 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).

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.

Economic importance and control

The disease is of minor economic importance and does not warrant any control measures.

References

KOIKE H., 1988. Sugar-Cane Diseases. A guide for field identification. Rome, Italy, Food and Agriculture Organization of the United Nations, 127p.

LO T.C., 1964. White rash. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 82–85. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Elsinoe* Racib. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 23. Slough, UK, CAB International.

Figure 1. Initial leaf symptoms: minute, round to elliptic spots (S. Matsuoka).

Figure 2. Whitish-grey elliptic spots on leaf blade (Taiwan Sugar).

Wilt

G.P. Rao and V.P. Agnihotri

Cause

Fusarium sacchari (E.J. Butler) W. Gams, *Acremonium implicatum* (J.C. Gilman & E. Abbott) W. Gams, *Acremonium furcatum* (F. & V. Moreau) ex W. Gams, *Fusarium oxysporum* Schlechtendahl; all hyphomycete fungi.

Butler and Khan (1913) described the causal organism of wilt as *Cephalosporium sacchari* E.J. Butler. Subsequently, several workers reported *Fusarium moniliforme* var. *subglutinans* as the pathogen of stalk rot and wilt (see Agnihotri and Singh, 1989). Booth (1971) and Gams (1971) reviewed the taxonomy of *C. sacchari* and considered it *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.

Singh *et al.* (1975) reported that besides *F. sacchari*, two species of *Acremonium* viz. *A. implicatum* (J.C. Gilman & E. Abbott) W. Gams and *A. furcatum* (F. & V. Moreau) ex W. Gams were also involved in the wilt syndrome of sugarcane. *Fusarium oxysporum* has also been associated with wilt. More investigations are required to resolve the controversy of the identity of the actual causal organism of sugarcane wilt.

Geographical distribution

Argentina, Bangladesh, Barbados, Benin, Brazil, Burkina Faso, Central African Republic, Colombia, Cuba, El Salvador, Guadeloupe, Guatemala, India, Iran, Malaysia, Mauritius, Mexico, Mozambique, Myanmar, Nepal, Niger, Pakistan, Panama, Papua New Guinea, Philippines, Réunion, St Kitts and Nevis, Senegal, South Africa, Thailand, Togo, Trinidad, Uganda, USA, Zimbabwe.

Symptoms

Symptoms first appear on 4–5-months-old sugarcane. Leaves of affected stalks gradually turn yellow and become dry (Figure 1). Eventually plants become completely dry and die leaving hollow stalks that are a total milling loss (Figures 2 and 3). Typical internal symptoms can be observed after splitting the

diseased stalks longitudinally (Figure 4). The internal pith tissue, particularly of the lower internodes, is light to dark purplish-brown. The tissue discoloration is more prominent at the nodes. The vascular tissues appear as dark reddish-brown streaks that pass through the internodal tissues from one internode to another. The wilt affected canes do not emit a foul odour. There are no white spots in internodal tissues that are typical of red rot affected canes.

However, shrinkage of internodal tissues takes place due to the loss of moisture resulting in the formation of longitudinal, spindle-shaped cavities in the middle of the internodes. Sometimes, mycelium and spores of the pathogen fill the cavities. Tissue discoloration and cavity formation are relatively more prevalent in the basal portion of the affected canes.

Initially, the roots of affected plants do not display distinct symptoms but subsequently the affected roots die and vascular bundles of the shoot become brownish as the stalks die (SRINIVASAN, 1964).

Diagnosis

Externally, sugarcane wilt can be diagnosed by the drying of the leaves. Affected stalks display typical internal wilt symptoms. However, reddening of the internodal tissue in wilt affected canes creates confusion with red rot and borer. The disease can be confirmed by isolations from roots, nodes and internodes of wilt affected canes, which usually yield *F. sacchari* and *A. implicatum*. The frequency of isolation of the former is higher in roots and internodal tissues, while that of the latter is higher in the nodes. This suggests that *F. sacchari* is a pathogen of parenchymatous tissues, and *A. implicatum* a pathogen of vascular tissues (AGNIHOTRI,1983). The presence of the two fungi in diseased tissues makes the isolation of *A. implicatum* difficult because it is a slow growing fungus, whereas *F. sacchari* is fast growing. It seems that a selective medium for isolating *A. implicatum* is needed. Potato dextrose agar medium supports luxuriant growth of both fungi. Serological diagnosis of the wilt pathogen(s) is not available yet but would be useful, and it also could help to solve the confusion about the true identity of the wilt syndrome causal organism(s).

Strains of the pathogen

Variability in morphological characters and in pathogenicity has been reported for *F. sacchari* and *Acremonium* sp. (SINGH and SINGH, 1983; AGNIHOTRI and SINGH, 1989).

Transmission

The pathogen *(F. sacchari)* is transmitted by infected cuttings. Secondary spread occurs through wind, rain and irrigation water. The fungus can survive in sugarcane debris in the soil. Soil transmission has been reported in soils having a pH of 7.0–8.0 (SRINIVASAN, 1964).

Figure 1. Drying of leaves of infected sugarcane (G.P. Rao).

Figure 2. Sugarcane stool affected by wilt (G.P. Rao).

Figure 3. Stalks killed by wilt in association with red rot (G.P. Rao).

Figure 4. Hollow stalk with dark purplish-brown pith tissue (G.P. Rao).

Host range

Sugarcane is so far the only reported host plant for wilt.

Epidemiology

The pathogen is seed piece transmissible and secondary spread is through wind, rain and irrigation water. The disease is favoured by neutral to alkaline soil (pH 7.0–8.0) and low moisture regime (SARMA, 1976). The wilt fungus can survive in soil for 2.5 to 3 years (GANGULY and CHAND, 1963). A high C/N ratio in the soil also favours disease development.

Many epidemics in India have proved that moisture stress during summer months coupled with high day temperature and low humidity increases wilt incidence (GANGULY, 1964). Wilt is more dangerous and causes enormous damage to the crops in association with red rot (Figure 3), pineapple disease, stalk borer or scale insects. These disease complexes have become a threat to sugarcane growers (AGNIHOTRI, 1983).

Economic importance

Wilt has caused significant losses in India where several epidemics have occurred (AGNIHOTRI, 1983). The disease adversely affects germination. It also causes young tillers to wither and stalks to die. Drying of shoots takes place at 4–5 months of age. During the monsoon period, considerable mortality of affected shoots occurs and occasionally all the shoots in a clump dry out resulting in a gappy crop stand. SARMA (1976) reported that yield losses may reach 65%. Wilt incidence is always higher in ratoon crops compared with the plant crop.

Besides yield reduction, wilt disease also causes 14.6–25.8% reduction in juice extraction and 3–20% in sugar recovery. There is a deterioration in juice quality because of a decrease in sucrose content and an increase in reducing sugars, gums, titrable acidity, flavonoids and soluble salts (SINGH and WARAITCH, 1981); these adversely affect the processing of white sugar in the mill.

Control

No single method is known to control wilt disease syndrome in sugarcane. However, use of resistant varieties is the major method of control. 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. Proper irrigation should be applied as drought conditions favour wilt development. A 3-year crop rotation has also been recommended in case of wilt infested soils.

Wilt incidence under field conditions can be reduced appreciably by applying 40 ppm boron or manganese to wilt infested soils. Similarly sett treatment with

boron (40 ppm) before planting also reduces 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% bavistin considerably reduces wilt incidence as well as the population of wilt pathogen(s) (AGNIHOTRI and SINGH, 1989). New approaches in controlling wilt disease have been recommended using antagonists (BHATTI and CHOHAN, 1970) or natural plant products (RAO and SRIVASATAVA, 1994). Domestic quarantine should be applied before transporting seed cane to a new locality.

References

AGNIHOTRI V.P., 1983. Wilt. *In*: Diseases of Sugarcane and Sugarbeet, p. 87–101. New Delhi, India. Oxford and IBH Publishing Company Pv Ltd.

AGNIHOTRI V.P., SINGH R.P., 1989. Sugarcane wilt: current status. *In*: Prospective in Plant Pathology. V.P. Agnihotri, N. Singh, H.S. Chaube, U.S. Singh and T.S. Dwivedi (Eds), p. 227–237. New Delhi, India, Today and Tomorrow's Printers and Publishers.

BHATTI D.S., CHOHAN J.S., 1970. Antagonism of certain microorganisms to *Cephalosporium sacchari* Butl. Journal of Research (Punjab Agriculture University) 7: 631–635.

BOOTH C., 1971. The Genus *Fusarium*. Kew, UK, Commonwealth Mycological Institute, 237p.

BUTLER E.J., KHAN A.H., 1913. Some new sugarcane diseases. Part 1. Wilt. Agriculture Memoir Botanical Series 6: 180–190.

GAMS W., 1971. *Cephalosporium*-artige Schimmelpilze (Hyphomycetes). Stuttgart, Germany, G. Fischer, 262p.

GANGULY A., 1964. Wilt: *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 131–137. Amsterdam, The Netherlands, Elsevier Publishing Company.

GANGULY A., CHAND J.N., 1963. Longevity of *Cephalosporium sacchari* Butler causing wilt disease of sugarcane. Science and Culture 29: 347–348.

RAO G.P., SRIVASATAVA A.K., 1994. Toxicity of essential oils of higher plants against fungal pathogens of sugarcane. *In*: Current Trends in Sugarcane Pathology. G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin, V.P. Agnihotri and C.T. Chen (Eds), p. 347–365. Delhi, India, International Books and Periodicals Supply Service.

SARMA M.N., 1976. Wilt disease of sugarcane. Sugarcane Pathologists' Newsletter 15/16: 30–33.

SINGH K., SINGH R.P., 1983. Variability in the isolates of *Acremonium terricola*, a causal organism of sugarcane wilt. Indian Sugar 32: 917–920.

SINGH O., WARAITCH K.S., 1981. Effect of wilt and red rot induced disease stress on quality deterioration of sugarcane. Sugarcane Pathologists' Newsletter 27: 25–30.

SINGH K., SINGH R.P., AGNIHOTRI V.P., 1975. Taxonomy and pathogenicity of fungi causing sugarcane wilt syndrome. Indian Phytopathology 28: 86–91.

SRINIVASAN K.V., 1964. Some observations on sugarcane wilt. Journal of Indian Botanical Society 3: 397–408.

Yellow spot

L. Jean Claude Autrey and A. Salem Saumtally

Cause

Mycovellosiella koepkei (W. Krüger) Deighton, deuteromycete fungus.

Geographical distribution

Argentina, Australia, Bangladesh, Barbados, Borneo, Brazil, Burundi, Cambodia, Cameroon, Chad, China, Colombia, Congo, Cuba, Fiji, Gabon, Ghana, Guadeloupe, Guatemala, Guyana, India, Indonesia, Japan, Kenya, Madagascar, Malawi (?), Malaysia, Mauritius, Myanmar, New Caledonia, Panama, Papua New Guinea, Philippines, Réunion, Samoa, Solomon Islands, South Africa, Sri Lanka, Taiwan, Tanzania, Thailand, Trinidad, Uganda, USA, Vietnam.

Symptoms

Symptoms induced by yellow spot disease usually appear on the second to third youngest leaf as minute chlorotic dots (Figure 1). As infection progresses the lesions increase in size, become irregular, coalesce and may attain 1 cm in diameter on mature leaves (Figure 2). Spots are present on both surfaces of the 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. The yellow spots turn brick-red to reddish-brown especially in varieties with red stalks (Figures 3 and 4). In varieties with yellow stalks the spots may give a golden colour to the foliage. Severe infection leads to necrosis from the tip to the base of the leaf and ultimately severe defoliation occurs, particularly in flowering stalks of highly susceptible varieties (Figure 5). In non-flowering stalks, as the conditions favouring infection subside, a flush of green leaves is produced.

Diagnosis

Under cool and moist conditions, the fungus typically produces a dirty grey mass consisting of conidiophores, conidia and hyphae usually on the lower surface of the lamina. This mass is not as profuse as the down associated with

Figure 1. Initial symptoms: minute chlorotic dots on the leaf (MSIRI).

Figure 2. Yellow leaf spots (MSIRI).

Figure 3. Brick-red to reddish-brown leaf spots (MSIRI).

Figure 4. Leaves showing old (left) and young (right) lesions (ISSCT).

Figure 5. Leaves severely affected by yellow spot (ISSCT).

downy mildew disease. The spots are different in size, shape and colour from those induced by other foliar pathogens such as brown stripe, brown spot, eye spot or ring spot but can be confused with them by the inexperienced observer. 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 (DEIGHTON, 1979) (Figure 6). The dimensions of the conidiophores and conidia vary (RICAUD and AUTREY, 1989). The fungus can be isolated on Czapek medium with some difficulty (HUGHES and OCFEMIA, 1961). The colonies are yeast-like in appearance (Figure 7) but give a fluffy, cottony grey mycelium with slow growth and low sporulation on potato dextrose agar. However, growth and sporulation are enhanced in culture on cane leaf agar especially with extract of leaves from susceptible varieties (AUTREY and SAUMTALLY, 1986).

Strains of the pathogen

Although changes in varietal reaction have been observed in Mauritius (RICAUD *et al.*, 1978), and in Australia (EGAN, 1970 and 1972), there is no definite proof of strain variation in the pathogen.

Transmission

Wet, cool weather is conducive to yellow spot infection. Conidia are splashed by rain and are wind-blown to other leaves within the same field or to adjacent ones and establish the disease as minute spots on young leaves. The disease is not transmitted by setts. Seasonal carry-over takes place by inoculum on old leaves of bull-shoots in areas such as Mauritius, where selective harvesting is carried out, from volunteer stools of highly susceptible varieties and plant material other than setts (RICAUD and AUTREY, 1989).

Host range

Wild canes *Saccharum robustum, S. spontaneum* and *S. edule* (DIGNADICE and ORILLO, 1953; ROACH, 1975), *Miscanthus sinensis* and *M. floridulus* (DEIGHTON, 1979) as well as an unidentified grass in Queensland (HUGHES and OCFEMIA, 1961), can harbour the disease. Apart from wild canes, the role of the other hosts in disease carry-over is unclear.

Epidemiology

Rainfall and the resulting higher relative humidity within the canopy, together with cool temperatures, are the most determinant environmental factors in the severity of the disease. Epidemics occur if susceptible varieties are grown in areas prone to infection.

Economic importance

Damaging epidemics of yellow spot have been reported in Australia, India, Indonesia and Mauritius. Losses have been assessed in Mauritius in variety Saipan 17 in which some 8000 tonnes of sugar were lost in 1978 and an estimated 20 000 tonnes from 1976 to 1980 (RICAUD *et al.*, 1980). Losses in both cane and sugar yield have been assessed under epidemic conditions (RICAUD *et al.*, 1980; AUTREY *et al.*, 1983). The disease is particularly damaging in profusely flowering varieties in which reduction of sucrose content can range from 10 to 25%.

Control

Only resistant varieties should be cultivated in high rainfall areas favourable to the disease. A screening method has been fully described by 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). Delaying harvest has also been found to be a good strategy for allowing sugar accumulation in the plant after infection has subsided (RICAUD *et al.*, 1983). Chemical control with benomyl

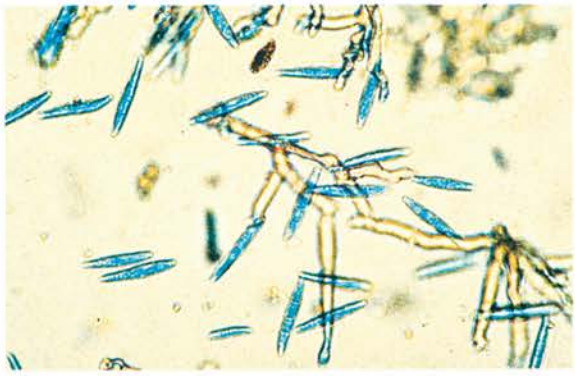


Figure 6. Conidiophores and conidia (blue) of the pathogen (×400) (MSIRI).

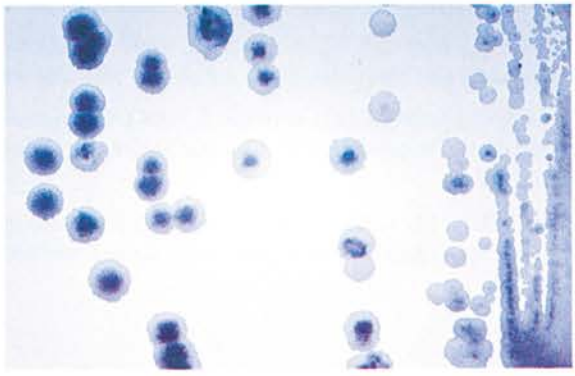


Figure 7. Yeast-like colonies of the pathogen on Czapek medium (MSIRI).

was resorted to in variety Saipan 17 before the removal of the variety from high rainfall areas of Mauritius (AUTREY *et al.*, 1983). However, fungicide application is considered as a palliative since the ultimate objective is the use of resistant varieties.

References

AUTREY L.J.C., SAUMTALLY S., 1986. Cultural characteristics of *Mycovellosiella koepkei* (Krüger) Deighton and their relationship with varietal reaction on cane-leaf agar. Proceedings International Society of Sugar Cane Technologists Congress 19: 336–343.

AUTREY L.J.C., RICAUD C., SULLIVAN S., DHAYAN S., 1983. Control of yellow spot disease of sugar cane by aerial application of fungicide. Sugar y Azúcar 78(7): 23–25.

DEIGHTON F.C., 1979. Studies on *Cercospora* and allied genera. 7. New species and redispositions. Mycological Paper 144: 13–26.

DIGNADICE P.B., ORILLO F.T., 1953. Yellow spot of sugar cane. Philippines Agriculture 37(1–2): 36–46.

EGAN B.T., 1970. Probable existence of strains of the yellow spot pathogen, *Cercospora koepkei*. Sugarcane Pathologists' Newsletter 5: 26–27.

EGAN B.T., 1972. The 1971 yellow spot epidemic in North Queensland. Proceedings Queensland Society of Sugar Cane Technologists Conference 39: 201–207.

HUGHES C.G., OCFEMIA G.O., 1961. Yellow spot disease. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 357–368. Amsterdam, The Netherlands, Elsevier Publishing Company.

RICAUD C., 1974. Factors affecting yellow spot development, its control and effect on cane and sugar yields. Proceedings International Society of Sugar Cane Technologists Congress 15: 354–364.

RICAUD C., AUTREY L.J.C., 1989. Yellow spot. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 231–245. Elsevier Science Publishers B.V.

RICAUD C., AUTREY L.J.C., SULLIVAN S., 1978. Recrudescence of yellow spot disease in Mauritius. Sugarcane Pathologists' Newsletter 20: 36–39.

RICAUD C., AUTREY L.J.C., SULLIVAN S., 1980. Losses from the recurrence of yellow spot epiphytotics in Mauritius. Sugar y Azúcar 75(7): 28–39.

RICAUD C., AUTREY L.J.C., SULLIVAN S., 1983. Integrated control of yellow spot disease of sugar cane. Proceedings International Society of Sugar Cane Technologists Congress 18: 797–805.

ROACH B.T., 1975. Resistance to yellow spot disease in *Saccharum* species and hybrids. Proceedings Queensland Society Sugar Cane Technologists Conference 42: 109–114.

Zonate leaf spot

Jack C. Comstock

Cause

Gloeocercospora sorghi D.C. Bain & Edgerton ex Deighton, deuteromycete fungus.

Geographical distribution

Papua New Guinea, Samoa, Solomon Islands, USA.

Symptoms

Initial symptoms are small necrotic lesions that enlarge to 5–6 cm producing a series of concentric rings of alternating dark and light brown zones of necrotic tissue (Figure 1). Lesions are more common on older leaves (BAIN and EDGERTON, 1943; LUTTRELL, 1950).

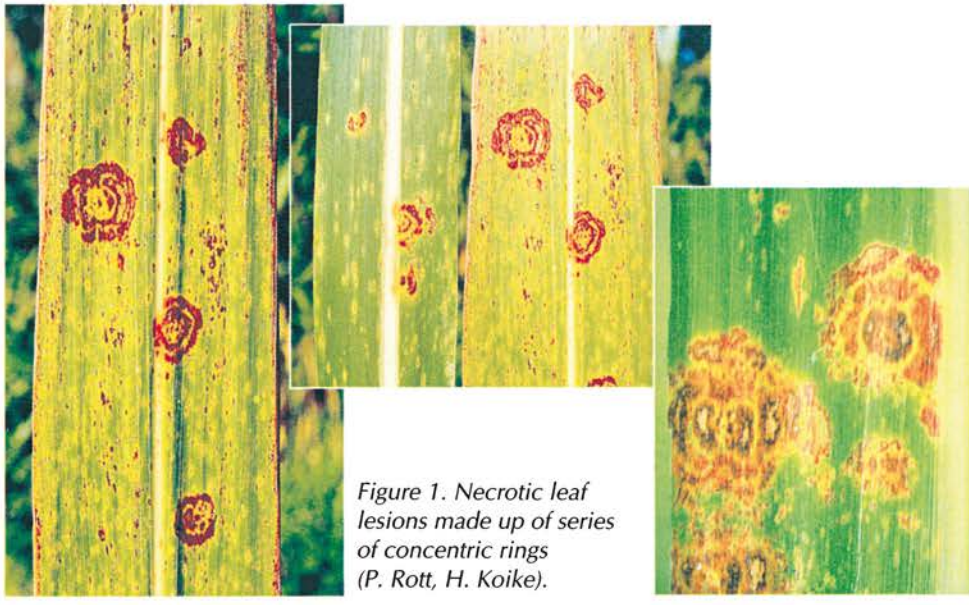


Figure 1. Necrotic leaf lesions made up of series of concentric rings (P. Rott, H. Koike).

Diagnosis

Fruiting structures of *G. sorghi* usually develop on leaf tissue placed in moist chambers within 24 to 48 h. The fungus as described on sorghum has salmon-coloured sporodochia that develop over stomatal openings bearing conidia in a pinkish slimy matrix. The conidia are hyaline, straight to curved, wider in the middle, smooth, with few to many septations varying in length, 20–195 × 2.8 µm (DEIGHTON, 1971). Black hemispherical sclerotia have been reported to be produced by *G. sorghi* beneath the lower epidermis measuring approximately 115–135 × 70–100 µm (LUTTRELL, 1950).

Strains of the pathogen

No information is available.

Transmission

Wind and rain-blown conidia are thought to spread the disease.

Host range

The host range of *G. sorghi* extends to sugarcane, sorghum, Johnson grass and Sudan grass (*Sorghum vulgare* var. *sudanense*) (BAIN and EDGERTON, 1943).

Epidemiology

The disease is favoured by hot humid weather.

Economic importance

Zonate leaf spot 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.

DEIGHTON F.C., 1971. Validation of the generic name *Gloeocercospora* and the specific names *G. sorghi* and *G. inconspicua*. Transactions British Mycological Society 57: 358–360.

LUTTRELL E.S., 1950. Grain sorghum diseases in Georgia – 1949. Plant Disease Reporter 34: 45–52.

Minor fungal diseases

Philippe Rott and Jack C. Comstock

Black rot

Cause: *Ceratocystis adiposa* (E.J. Butler) C. Moreau (anamorph = *Chalara* sp.).

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 disease.

References

ABBOTT E.V., 1964. Black rot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 99–102. Amsterdam, The Netherlands, Elsevier Publishing Company.

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 No. 29, p. 14–16. Slough, UK, CAB International.

Black stripe

Cause: *Pseudocercospora atrofiliformis* (W.Y. Yen, T.C. Lo & C.C. Chi) 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 No. 29, p. 49–52. 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 and C.A. Wismer (Eds), p. 21–23. Amsterdam, The Netherlands, Elsevier Publishing Company.

Collar rot

Cause: *Hendersonia sacchari* E.J. Butler.

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 and C.A. Wismer (Eds), p. 105–107. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Hendersonia* E. Butler. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 53–54. Slough, UK, CAB International.

Dry rot

Cause: *Physalospora rhodina* M.C. Cooke.

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 coloured 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 and C.A. Wismer (Eds), p. 108–109. Amsterdam, The Netherlands, Elsevier Publishing Company.

Iliau

Cause: *Clypeoporthe iliau* (H.L. Lyon) M.E. Barr = *Gnomonia iliau* H.L. Lyon [anamorph = *Phaeocytostroma iliau* (H.L. Lyon) Sivanesan].

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 1).

References

MARTIN J.P., 1964. Iliau. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 114–118. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Clypeoporthe* Höhnel. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 18–19. Slough, UK, CAB International.

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 and C.A. Wismer (Eds), p. 29–31. Amsterdam, The Netherlands, Elsevier Publishing Company.

Leaf splitting

Cause: *Peronosclerospora miscanthi* (T. Miyake) C.G. Shaw = *Sclerospora miscanthi* T. Miyake; *P. northii* (W.H. Weston) C.G. Shaw, *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 2).

References

CHU H.T., 1964. Leaf-splitting disease. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 37–39. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Peronosclerospora* (Ito) Shirai & K. Hara. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 58–61. Slough, UK, CAB International.

Marasmius sheath rot and shoot blight; root rot

Cause: *Marasmiellus stenospilus* (Montagne) R. Singer = *Marasmius stenospilus* Montagne; *Marasmius sacchari* Wakker; *Rhizoctonia* sp.; unidentified oomycete.

Geographical distribution: thought to be widely distributed (Barbados, Indonesia, Mauritius, Peru, Philippines, Puerto Rico, USA).

Symptoms: white mycelium present between leaf sheath and stalk; rotting of leaf sheaths near base; death of stem at or below 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 and C.A. Wismer (Eds), p. 89–93. Amsterdam, The Netherlands, Elsevier Publishing Company.

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 and C.A. Wismer (Eds), p. 94–98. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Marasmius* Fr. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 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 3).

References

ABBOTT E.V., 1964. Myriogenospora leaf binding. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 40–42. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Myriogenospora* Atk. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 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-coloured spots on leaf sheath located close to where leaf attaches; fungus associated with older spots of ring spot.

References

ABBOTT E.V., 1964. Ring spot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 52–58. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., 1938. Phyllosticta spot. *In*: Sugar cane diseases in Hawaii. p. 84–85. Honolulu, Experiment Station of the Hawaiian Sugar Planters' Association.

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 4).

References

STEIB R.J., 1964. Phytophthora seed piece rot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 120–123. Amsterdam, The Netherlands, Elsevier Publishing Company.

SIVANESAN A., WALLER J.M., 1986. *Phytophthora* de Bary. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 62–64. Slough, UK, CAB International.

Seedling foliage blights

Cause: *Alternaria alternata* (E.M. Fries: E.M. Fries) von Keissler; *Cochliobolus hawaiiensis* Alcorn [anamorph = *Bipolaris hawaiiensis* (M.B. Ellis) J.Y. Uchida & Aragaki]; *Cochliobolus lunatus* R.R. Nelson & F.A. Haasis [anamorph = *Curvularia lunata* (Wakker) Boedijn]; *Curvularia senegalensis* (Spegazzini) C.V. Subramanian; *Bipolaris sacchari* (E.J. Butler) Shoemaker; *Setosphaeria rostrata* K.J. Leonard [anamorph = *Exserohilum rostratum* (Drechsler) K.J. Leonard & E.G. Suggs = *Dreschslera halodes* (Drechsler) C.V. Subramanian & P.C. Jain].

Geographical distribution: Cuba, India, Taiwan (*Alternaria*); Argentina, Hawaii, India (*Cochliobolus*); widespread (*Drechslera*); India, Puerto Rico, USA (*Setosphaeria*).

Symptoms: blight of seedlings (*Alternaria* and *Drechslera*). 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 (*Cochliobolus*). Narrow, reddish, elongated broken stripes that become dark brown with age; stunted seedlings (*Setosphaeria*).

References

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.

SIVANESAN A., WALLER J.M., 1986. *Alternaria* Nees. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 39–40. Slough, UK, CAB International.

SIVANESAN A., WALLER J.M., 1986. *Cochliobolus* Drechsler. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 19–22. Slough, UK, CAB International.

SIVANESAN A., WALLER J.M., 1986. *Drechslera* Ito. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 43–47. Slough, UK, CAB International.

SIVANESAN A., WALLER J.M., 1986. *Setosphaeria* Leonard & Suggs. *In*: Sugarcane Diseases. Phytopathological Paper No. 29, p. 37–38. Slough, UK, CAB International.

Target blotch

Cause: *Helminthosporium* sp. Priode.

Geographical distribution: Cuba, India, Japan, South Africa, Thailand, USA, Zimbabwe.

Symptoms: minute, reddish-brown spots; straw-coloured to brownish necrotic areas with irregular, concentric rings roughly resembling a target.

Reference

TODD E.H., 1964. Target blotch. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 74–77. Amsterdam, The Netherlands, Elsevier Publishing Company.

Figure 1. Iliau: binding of leaf sheaths and distortion of emerging stem (S. Matsuoka).

Figure 2. Leaf splitting: split leaves looking like long-tangled coarse hair (J.C. Comstock).

Figure 3. Myriogenospora leaf binding: adhering leaves (BSES).

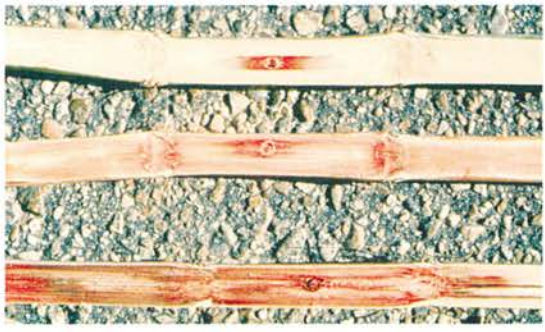


Figure 4. Phytophthora rot of cuttings: orange-red streaking inside cuttings (J. Hoy).

Diseases caused by phytoplasmas

Grassy shoot

R. Viswanathan

Cause

Phytoplasma, mollicute.

Geographical distribution

Bangladesh, India, Iran, Malaysia, Myanmar, Nepal, Pakistan, Sri Lanka, Sudan, Thailand.

Symptoms

Grassy shoot disease is characterized by the production of numerous, thin, slender yellow/chlorotic tillers (Figure 1), chlorosis of the leaves without spotting, mottling of the leaf lamina, premature proliferation of the axillary buds, few or no millable stalks, severe stunting and sterility. The symptoms of the disease can be noticed at all growth stages (EDISON *et al.*, 1977; HUGHES and ABBOTT, 1964; RISHI and CHEN, 1989).

The appearance of a chlorotic leaf among the top leaves in a healthy shoot is the earliest symptom in the plant crop. Then, the number of white leaves appearing in the infected plant increases gradually. If the planted setts are from infected stalks, the emerging shoots will be yellow in colour. Later, many thin, small, white grassy shoots emerge from the base of the affected stalk. These diseased tillers may not survive for more than 1 month. In the field, various degrees of disease severity exist. Occasionally some green leaves may be seen in an otherwise yellow sprout.

If the disease appears in the first half of the crop's stand in the field, the chlorotic sprouts start appearing from the base of the clump (Figure 2) followed shortly by several erect chlorotic grass-like shoots. The older leaves in the infected plant have long narrow, whitish to pale green longitudinal streaks alternating with darker green streaks. The streaks are usually straight, regular and parallel to the vascular bundles. Healthy looking stalks can often be seen in a clump which has many grassy shoots.

In a mature crop, newly emerging leaves turn chlorotic and these leaves do not fully open. In those cases a bunchytop-like symptom with chlorotic leaves can be noticed at the top of the stalks. Later, small chlorotic sprouts are produced from the axillary buds in a basipetal manner. Chlorotic tillers proliferate from the base of infected canes. The tips of the tillers dry out, resulting in the death of the chlorotic tillers.

Round to elongated smaller buds with thin and papery bud scales are common in grassy shoot affected canes. Premature sprouting of lateral buds with pale coloured leaves and leaf sheaths, and the formation of aerial roots is also common. A reduction in size and height of the apical meristem results in the break down of apical dominance which leads to the characteristic little shoots from the axillary buds in diseased cane (Figure 3). In rare cases, proliferated multiple buds produce the sprouted appearance of plants. Grassy shoot affected plants show a significant reduction in leaf size, number, length and breadth of the lamina. This proliferation in a diseased clump is homologous to the bushy, grass-like growth in the vegetative parts of the host plant resulting in the formation of no millable canes or only a few dwarfed ones. The production of inflorescences in diseased plants is almost completely arrested.

In affected stools, immediately after ratooning, thin chlorotic tillers are produced in large numbers. Most of these tillers slowly die. Production of healthy shoots is curtailed in severely affected stools during early crop growth stages in ratoon crops. A field shows crop-free patches in severe cases.

Diagnosis

Foliar symptoms cannot be reliably used for the diagnosis of grassy shoot. Serological techniques can be applied to detect the phytoplasmas associated with the disease. The direct and indirect ELISA techniques were found to be effective for the detection of the pathogen whereas the dot-blot technique was found to be less sensitive. Indirect immunofluorescence technique was also found useful in identifying infected tissues in the host plant (VISWANATHAN, 1994 and 1997). Fluorescence microscopy based on DAPI stain was also successfully used for detecting phytoplasma infection.

Strains of the pathogen

It has been observed in field conditions that certain sugarcane cultivars produce numerous green grassy shoots while most other cultivars produce chlorotic shoots, suggesting the presence of two distinct forms of grassy shoot phytoplasmas in India. Grassy shoot symptoms observed in Thailand primarily included production of green tillers and the disease was, therefore, referred to

Figure 1. Stools affected by grassy shoot (ISSCT).

Figure 2. Chlorotic tiller in a healthy looking stool (R. Viswanathan).

Figure 3. Germination of lateral buds in a grassy shoot affected plant (R. Viswanathan).

as green grassy shoot syndrome (PLIANSINCHAI, 1996). However, the presence of specific strains of the pathogen associated with the syndrome is yet to be proved.

Transmission

The primary method of transmission of grassy shoot is through infected setts. Such transmission is frequently noticed in tropical India with cultivars Co8021, CoC671 and CoC92061 which show high levels of disease incidence under field conditions. No insect vector has been identified.

Host range

Sorghum (*Sorghum bicolor*), elephant grass (*Pennisetum purpureum*), *Brachiaria mutica, Cynodon dactylon* and *Imperata arundinacea* are reported to have symptoms similar to those of sugarcane grassy shoot in India. However, there is no corroborating evidence for the presence of the grassy shoot pathogen. Additionally, recent studies by RFLP and sequencing techniques revealed that phytoplasmas causing grassy shoot in sugarcane and other graminaceous weed hosts in Thailand are different (WONGKAEW *et al.*, 1997).

Epidemiology

Sett transmission plays a major role in the spread of the disease. Crops planted with healthy setts are free from the disease for several months. However, crops planted with setts from infected seed sources show disease incidence within 2 to 3 months. Disease levels in the field generally increase over a period of time. Observations with three cultivars (CoJ64, Co975 and Co1148) showed 6%, 16%, 24%, 28% and 100% of diseased clumps in the third, fourth, fifth, sixth and eighth month, respectively. In some cultivars, however, the incidence never reaches 100% (SANDHU and RAM, 1974). Field observations in peninsular India indicated a higher disease incidence during the summer months than during the monsoon and winter periods. Detailed information regarding seasonal influence on grassy shoot occurrence is not available.

Economic importance

Very high yield losses due to grassy shoot were reported in India for many years. The damage was particularly high when seed material was obtained from infected seed cane. The phenomenon of 'throw off' (recovery) was also observed in severely affected ratoon crops in different states in India. Recent studies conducted at the Sugarcane Breeding Institute showed that

phytoplasma infection can cause 35% reduction in stalk height and 15% reduction in stalk girth. In addition, 50–60% reduction in length of internodes was observed. Above all, disease infection caused a significant reduction in millable cane, especially in ratoon crops. About 50–75% plant crop infection resulted in 100% failure in millable cane production in the ratoon crop of clones such as IS152. In moderately infected cultivars up to 40% reduction in sugar yield was noticed (Viswanathan, unpublished data).

Control

The cuttings used for planting can be disinfected by thermotherapy by soaking in hot water at 50°C for 2.5 h. Aerated steam therapy (AST) was recommended for 1 h at 50°C for inactivation of the phytoplasmas (EDISON and RAMAKRISHNAN, 1972). However, recent findings of the author have shown that AST treatments for 2–3 hours at 50°C or 1 h at 52°C were highly effective in the elimination of phytoplasmas in infected setts. Plants from these treatments were free from the disease for up to 12 months and also later in the 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 grassy shoot phytoplasmas in sugarcane (SHUKLA and SINGH, 1991).

Integrated disease management through a three-tier seed nursery programme is being followed in many sugar factories in India. This programme involves production of disease-free seed material through heat therapy. The programme consists of three stages: (1) foundation seed, (2) certified seed and (3) commercial seed (SINGH, 1977). The seed material employed for raising foundation seed is obtained from a nucleus seed with 100% genetic purity. The seed materials are subjected to AST or MHAT and a foundation nursery is raised. The crop raised from the foundation seed will provide seed for raising certified seed. Good quality certified seed is used for raising commercial seed. No heat treatment is given while raising certified/commercial seed. The nursery fields are inspected at regular intervals and diseased clumps are rogued out to keep the nurseries free from the disease. The permitted limits of grassy shoot incidence is 0% at the foundation nursery and 0.5% in certified and commercial seed. The commercial seed thus raised is distributed to farmers for commercial planting. This system ensures a continuous supply of disease-free seed material to farmers in a sugar factory zone, and this programme is successful in managing the disease in different states.

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

References

BHANSALI R.R., SINGH K., 1991. Grassy shoot mycoplasma free plantlets from apical meristem of lateral buds. Indian Phytopathology 44: 291–294.

EDISON S., RAMAKRISHNAN K., 1972. Aerated steam therapy for the control of grassy shoot disease (GSD) of sugarcane. Mysore Journal of Agricultural Sciences 6: 492–494.

EDISON S., RAMAKRISHNAN K., NARAYANASAMY P., 1977. A study of symptomatology of grassy shoot disease of sugarcane. Cooperative Sugar 8: (8)1–5.

HUGHES C.G., ABBOTT E.V., 1964. Grassy shoot. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 165–170. Amsterdam, The Netherlands, Elsevier Publishing Company.

PLIANSINCHAI U., 1996. Sugarcane green grassy shoot syndrome and its control. *In*: Annual Report 1995–96. Field Crop Research Institute, Suphan Buri, Thailand, p. 10–12.

RISHI N., CHEN C.T., 1989. Grassy shoot and white leaf diseases. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 289–300. Amsterdam, The Netherlands, Elsevier Science Publisher B.V.

SANDHU S.S., RAM R.S., 1974. Grassy shoot disease in Punjab, India. Proceedings International Society of Sugar Cane Technologists Congress 15: 348–353.

SHUKLA U.S., SINGH V., 1991. Inactivation of grassy shoot MLOs in diseased setts by heat treatment. Indian Phytopathology 43: 561–562.

SINGH K., 1977. Sugarcane diseases and three tier seed programme. Sugar News 9: 81–89.

VISWANATHAN R., 1994. Serological techniques for the detection of grassy shoot disease MLOs. Indian Phytopathology 47: 281–282.

VISWANATHAN R., 1997. Detection of phytoplasmas causing grassy shoot disease in sugarcane by ELISA techniques. Journal of Plant Diseases and Protection 104: 9–16.

WONGKAEW P., HANBOONSONG Y., SIRITHORN P., CHOOSAI C., BOONKRONG S., TINNANGWATTANA T., KITCHAREONPANYA R., DAMAK S., 1997. Differentiation of phytoplasmas associated with sugarcane graminaceous weed white leaf disease and sugarcane grassy shoot disease by RFLP and sequencing. Theoretical and Applied Genetics 95: 660–663.

Green grassy shoot

Upsorn Pliansinchai and Siripatr Prammanee

Cause

Phytoplasma, mollicute.

Geographical distribution

Thailand.

Symptoms

Green grassy shoot is characterized by profuse tillering and leaves that are narrow and green without chlorosis (Figures 1, 2 and 3). These latter symptoms are different from those of grassy shoot disease in India and white leaf disease in Thailand, two diseases that are also caused by phytoplasmas.

In the plant cane crop, infected plants produce normal tillers, but small and late profuse tillering can be observed at the base. The number and size of stalks may be reduced which results in yield reduction. Symptoms are more severe in ratoon crops where severe profuse tillering can be observed resulting in decreased yield and ratooning ability. Highly susceptible sugarcane varieties may die (Figure 4).

Symptoms can be recognized 2 months after planting in severely infected cane. In mildly infected cane, symptoms may appear after 5 months or up to the appearance of mature stalks or even up to the ratoon crop. Symptom expression becomes more severe in ratoons resulting in dwarf green grassy symptoms. None or only a few millable stalks are then produced.

Diagnosis

Visible symptoms can be used for diagnosis. Green grassy shoot can be differentiated from white leaf disease (RISHI and CHEN, 1989) by the absence of albinism, from shoot borer infection by the absence of 'dead heart', and from

smut by the absence of a whip-like structure. Examination of ultra-thin sections of infected leaves shows typical phytoplasma profiles in the sieve tube of the phloem cells (PLIANSINCHAI and PRAMMANEE, 1995) (Figure 5).

Serological techniques using white leaf phytoplasma antiserum cannot be used to detect the green grassy shoot pathogen. Antisera against the green grassy shoot phytoplasma are not available yet. Few positive detections of phytoplasmas in plants exhibiting green grassy shoot symptoms using white leaf phytoplasma antiserum were attributed to mixed infections with the causal agent of white leaf (PLIANSINCHAI *et al.*, 1995; SARINDU and PLIANSINCHAI, 1996). DNA probes, PCR, PCR-RFLP and sequence analysis have also been developed for detection and diagnosis of the disease. It was shown that the green grassy shoot (initially called grassy shoot) phytoplasma differed from the white leaf phytoplasma in Thailand by dot-blot hybridization with a specific DNA probe, PCR-RFLP and sequencing (PRAMMANEE *et al.*, 1996; SDOODEE *et al.*, 1999; WONGKAEW *et al.*, 1997).

The green grassy shoot phytoplasma is not serologically related to the white leaf phytoplasma in Thailand. The latter is, however, serologically related to the grassy shoot phytoplasmas from Sri Lanka (SARINDU and CLARK, 1993) and India (VISWANATHAN, 1997). The green grassy shoot (Thailand) and grassy shoot (India or Sri Lanka) pathogens have not yet been directly compared.

Strains of the pathogen

No information is available.

Transmission

The disease can be transmitted through infected cuttings but not by mechanical means (PLIANSINCHAI and SUCHATO, 1995). Sett propagation plays a major role in the spread of the disease, but evidence exists for other means of transmission (PLIANSINCHAI *et al.*, 1997). In a naturally infected field, disease incidence increased from 13.3% in plant cane crop to 56.0% in first ratoon crop. In an experimental plot, healthy planted cane showed disease incidence up to 30% when infected cane was planted alternately between rows (PLIANSINCHAI *et al.*, 1998). Sixteen plant and leafhopper species were identified in a green grassy shoot outbreak area (PLIANSINCHAI *et al.*, 1998). Insect ability to carry the phytoplasma was tested with a specific DNA probe, but the pathogen has not been found in any tested insects.

Host range

Sugarcane is the only reported host plant for green grassy shoot.

Figure 1. Plant affected by green grassy shoot (left) and healthy cane (right) (U. Pliansinchai).

Figure 2. Healthy looking mature cane associated with small and late profuse tillering (U. Pliansinchai).

Figure 3. Typical symptom of profuse tillering (U. Pliansinchai).

Figure 4. Cane killed by green grassy shoot (U. Pliansinchai).

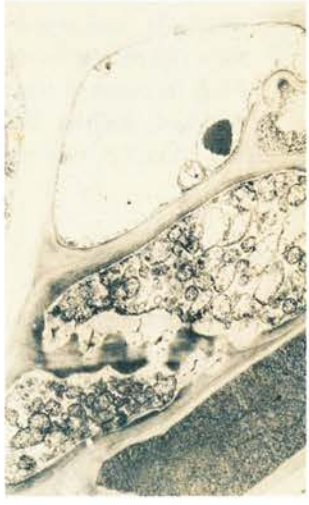


Figure 5. Green grassy shoot phytoplasma observed by electron microscopy in the phloem sieve tubes (U. Pliansinchai).

Epidemiology

Disease incidence is higher when sugarcane is planted during the cool and humid months of the year (August–December).

Economic importance

The disease can cause yield losses up to 40% and a reduction of ccs (commercial cane sugar) around 0.5–0.8 (PLIANSINCHAI *et al.*, 1995). The losses are greater in ratoon crops when diseased plants show profuse green grassy shoots that do not develop into millable stalks. The first disease outbreak occurred in Western Thailand in susceptible variety U-thong 1 which had to be replaced by new varieties more resistant to the disease.

Control

As infected cuttings play a major role in propagation of green grassy shoot, the use of disease-free sugarcane is recommended for planting. Multiplication plots should, therefore, be established with treated cane. A hot water treatment of cuttings or whole stalks at 50°C for 2 h significantly reduced the incidence of the disease. Hot water treatments for 0.5 h or 1 h at 52°C, for 1 h at 50°C, or a hot air treatment at 54°C for 4 h were less efficient (PLIANSINCHAI *et al.*, 1998).

Roguing is also useful in reducing the inoculum in contaminated fields, and at the end of a crop, diseased plants should be eradicated before establishing a new crop.

Use of resistant varieties is the most effective means of control. However, the use of resistant varieties is limited due to the absence of efficient screening methods, and the difficulties encountered in combining resistance and high agricultural performance. However, disease incidence was nil under natural field infestation for several commercial clones from various origins: Alunan, B34664, Badilla, Bo14, C323-68, CB47-15, Co775, CP57-603, F136, My55-14, NCo387, Phil54-52, U-thong3, etc. (PLIANSINCHAI *et al.*, 1998).

References

PLIANSINCHAI U., PRAMMANEE S., 1995. Electron microscopy to detect causal organism of sugarcane green grassy shoot. Proceedings of the 21st Thailand National Science and Technology Conferences, 25–27 October 1995, Bangkok, Thailand, p. 572–573.

PLIANSINCHAI U., SUCHATO W., 1995. Sett transmission of sugarcane green grassy shoot disease. Proceedings of the 2nd National Plant Protection Conference, 9–11 October 1995, Chiang Mai, Thailand, p. 243–248.

PLIANSINCHAI U., SARINDU N., PRAMMANEE S., 1995. Sugarcane green grassy shoot disease and their effects on cane yield and quality. Proceedings of the 2nd National Cane Sugar Conference, 2–3 May 1995, Bangkok, Thailand, p. 253–263.

PLIANSINCHAI U., SARINDU N., PRAMMANEE S., THANAWAN T., YIMSA-ARD M., 1997. Etiology, symptomatology of sugarcane green grassy shoot and heat therapy. Proceedings of the 35th Kasetsart University Annual Conference, 3–5 February 1997, Bangkok, Thailand, p. 399–406.

PLIANSINCHAI U., LAEBWAN U., SUCHATO W., 1998. Sugarcane green grassy shoot disease: epidemiology, transmission and control. Proceedings of the 3rd National Cane and Sugar Conference, 6–8 May 1998, Khon Khen, Thailand, p. 432–453.

PRAMMANEE S., SARINDU N., PLIANSINCHAI U., 1996. Nucleotide sequence analysis of PCR amplification from MLO infected periwinkle is the effectively specific DNA probe to detect MLOs-diseases and genetic related to palm yellow necrotic and sugarcane grassy shoot diseases. *In*: Abstracts of the 8th Annual Meeting of the Thai Society for Biotechnology and the 1996 Annual Meeting of the National Center for Genetic Engineering and Biotechnology. 14–15 November 1996, Hua Hin, Prachuap Khiri Khan, Thailand, p. 31.

RISHI N., CHEN C.T., 1989. Grassy shoot and white leaf diseases. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 289–296. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SARINDU N., CLARK M.F., 1993. Antibody production and identity of MLOs associated with sugarcane white leaf disease and Bermuda-grass white leaf disease from Thailand. Plant Pathology 42: 396–402.

SARINDU N., PLIANSINCHAI U., 1996. Detection of mycoplasma-like organisms in grassy shoot sugarcane by ELISA. Thai Journal of Sugar and Sugarcane 3(1): 33–38.

SDOODEE R., SCHNEIDER B., PADOVAN A.C., GIBB K.S., 1999. Detection and genetic relatedness of phytoplasmas associated with plant diseases in Thailand. Journal of Biochemistry, Molecular Biology and Biophysics 3: 133–139.

VISWANATHAN R., 1997. Detection of phytoplasmas associated with grassy shoot disease of sugarcane by ELISA techniques. Journal of Plant Diseases and Protection 104: 9–16.

WONGKAEW P., HANBOONSONG Y., SIRITHORN P., CHOOSAI C., BOONKRONG S., TINNANGWATTANA J., KITCHAREANPANYA R., DAMAK S., 1997. Differenciation of phytoplasmas associated with sugarcane and gramineous weed white leaf disease and sugarcane grassy shoot disease by RFLP and sequencing. Theoretical and Applied Genetics 95: 660–663.

Ramu stunt

Sidney Suma and Phil Jones

Cause

Early attempts to identify viruses in Ramu stunt affected cane were inconclusive (JONES *et al.*, 1989). A phytoplasma has been associated with Ramu stunt disease, using a nested polymerase chain reaction (PCR) of general phytoplasma primers from the 16S rDNA (CRONJÉ *et al.*, 1999).

Geographical distribution

Ramu stunt has only been reported from Papua New Guinea.

Symptoms

The most striking effect of Ramu stunt is its ability to suddenly and rapidly reduce the growth of affected cane. WALLER *et al.* (1987) give examples of nodes being reduced from 9–13 cm to 1–2 cm in the space of three nodes (1 month's growth). Leaf symptoms of the disease are variable and can depend on the variety affected (Figure 1). The variety Yassawa, for example, shows stunting and chlorosis while in Q124 these symptoms are even more pronounced with tiller proliferation giving a grassy shoot appearance. Typically symptoms start as short irregular streaks or flecks, pale to creamy green in colour and which may resemble a mosaic. As the symptoms develop the streaks become yellowish-green in colour and can vary from several millimetres in length to run the full length of the leaf blade. Streaks, which can be continuous or interrupted, range in width from 2 to 5 mm or more, and are interspersed by apparently healthy (green) tissue. There are no symptoms on the leaf sheath. Leaves of infected plants are much shorter than those of healthy plants.

Overall, the disease gives the cane a trashy appearance (Figure 2): leaves are short, stiff and erect, often partially rolled, and the rolling is pronounced on hot sunny days. As a result the leaves become prematurely senescent. Infected stools are severely stunted (Figures 3 and 4), and there is a progressive death of stalks. The root systems of infected stools are severely stunted, they collapse and become necrotic. Diseased stools invariably fail to ratoon, hence it is not uncommon to find total ratoon failure in a block of a susceptible variety in the field.

Figure 1. Leaf symptoms in cultivar Ragnar (P. Jones, R. Magarey).

Figure 2. Trashy appearance in Ramu stunt affected crop (rear: healthy cane) (G. James).

Figure 3. Diseased sugarcane row (G. James).

Figure 4. Diseased stool of cultivar Ragnar (R. Magarey).

Diagnosis

Diagnosis of the disease is based on visual symptoms which can be backed up by a DNA amplification test for the phytoplasma using a nested PCR (Cronjé *et al.*, 1999) which amplifies a fragment *c.* 1250 bp in size. Primers used are: SN910601 (5'-GTT TGA TCC TGG CTC AGG ATT) (Namba *et al.*, 1993) and P6 (5'-CGG TAG GGA TAC CTT GTT ACG ACT TA) (Deng and Hiruki, 1991) in the first round; followed by R16F2n (5'-GAA ACG ACT GCT AAG ACT GG) and R16R2 (5'-TGA CGG GCG GTG TGT ACA AAC CCC G) (Gundersen and Lee, 1996). A 25 µl reaction mix using 'Ready To Go PCR beads'™ (Pharmacia Biotech), containing 1 µl of each primer and 1 µl template DNA diluted to a final concentration of 50 µg/ml; 1 µl of reaction product was used as a template for the nested PCR.

Reaction conditions are 95°C for 3 min followed by 35 cycles of denaturation (95°C) for 30 s; annealing (first round at 53°C; nested at 56°C) for 1 min; and extension (72°C) for 1 min 30 s; followed by a final extension step (72°C) for 10 min. Reaction mixtures containing only healthy plant DNA or sterile distilled water substituted for template DNA serve as negative controls.

Analysis of PCR amplification products: aliquots (5.0 µl) of each reaction are analysed by electrophoresis through 1% agarose gels (Gibco BRL) containing ethidium bromide in 1x TBE (90 mM Tris-borate; 2 mM EDTA).

Further aliquots (5.0 µl) can be digested by the addition of 0.5 U of restriction enzyme followed by incubation at the appropriate temperature for a minimum of 4 h. The restriction enzymes *Rsa*I, *Alu*I and *Hae*III are recommended, products should be analysed by electrophoresis through 1.5% NuSieve GTG agarose gels (FMC BioProducts, supplied by Flowgen Instruments Ltd, UK) with 1x TBE (90 mM Tris-borate; 2 mM EDTA) as running buffer.

Strains of the pathogen

No information is available about strains of the causal agent. The 16S-23S intergenic spacer sequence of the type strain has been submitted to Genbank (accession no. AF 106061) (Cronjé *et al.*, 1999).

Transmission

The disease is systemic and is primarily spread through the use of infected stem cuttings. It is also transmitted by the leafhopper, *Eumetopina flavipes* Muir (Hemiptera: Delphacidae) (Kuniata *et al.*, 1994) (Figure 5).

Host range

Symptoms of the disease were first observed on interspecific hybrids of sugarcane. They also occur on noble (*Saccharum officinarum*) and wild (*S. robustum*) canes (Figure 6) and intergeneric hybrids of sugarcane. Disease

symptoms similar to those of Ramu stunt have been observed on *Imperata cylindrica* (Suma, unpublished), but these plants have not been subject to molecular analysis by PCR.

Epidemiology

Ramu stunt has been observed from sea level to an altitude of 2000 m. The incidence of the disease is higher during the wet season when vector populations are also high. However, the effects of the disease are exacerbated by moisture stress. Generally infected stools survive for only one season.

In a 1 ha plot planted with a susceptible variety of disease-free planting material, when infection occurs within the first 3 months, by 6 months of age about 50% of the stools will be dead, at harvest about 90% will be dead and the first ratoon will fail. If the infection occurs at 3–6 months, about 50% of the stools will be dead before harvest, less than 10% will germinate as first ratoon and will die before 6 months of age. When the infection occurs 6 months after planting less than 10% will die before harvest, about 50% will ratoon but all of the stools will die before the next harvest.

Economic importance

In Papua New Guinea, Ramu stunt is a major disease of sugarcane. It has caused up to 40% loss in total sugarcane production, and was responsible for the near closure of the Ramu Estate following the crop disaster in 1986 (Eastwood, 1990). In an attempt to minimize losses all imported varieties are screened for their susceptibility to Ramu stunt and only those rated resistant are passed for further evaluation. Between 1979 and 1992, as many as 30% of all introductions were discarded due to susceptibility to Ramu stunt (Suma and Pais, 1996).

Figure 5. Eumetopina *sp. the probable vector of Ramu stunt (R. Magarey).*

Figure 6. Leaf symptoms in wild cane (R. Magarey).

Because of its destructive nature and rapid spread cane breeding programmes must take account of resistance to Ramu stunt and continued screening of major varieties is essential. Ramu stunt disease is a major quarantine threat particularly to the neighbouring sugarcane industries in Australia and Indonesia.

Control

The use of resistant varieties is the only control measure known. The small breeding programme in Ramu, Papua New Guinea, considers Ramu stunt disease resistance as one of its major criteria for crossing and selection. The effect of other methods of disease control, including hot water treatment on Ramu stunt has not been adequately investigated.

References

CRONJÉ C.P.R., BAILEY R.A., JONES P., SUMA S., 1999. The phytoplasma associated with Ramu stunt disease of sugarcane is closely related to the white leaf phytoplasma group. Plant Disease 83(6): 588.

DENG S., HIRUKI C., 1991. Amplification of 16S rRNA genes from culturable and non-culturable mollicutes. Journal of Microbiological Methods 14: 53–61.

EASTWOOD D., 1990. Ramu stunt disease, development and consequence at Ramu Sugar Limited. Sugarcane 2: 15–19.

GUNDERSEN D.E., LEE I.M., 1996. Ultrasensitive detection of phytoplasmas by nested-PCR assays using two universal primer pairs. Phytopathologica Mediterranea 35: 144–151.

JONES P., ANTONIW J.F., EASTWOOD D., 1989. Investigation into the aetiology of Ramu stunt, a sugarcane disease in Papua New Guinea. L'Agronomie Tropicale 44: 179–182.

KUNIATA L.S., YOUNG G.R., PAIS E., JONES P., NAGARAJA H., 1994. Preliminary observation on *Eumetopina* sp. (Hemiptera: Delphacidae), a vector of Ramu stunt disease of sugarcane in Papua New Guinea. Journal of the Australian Entomological Society 33: 185–186.

NAMBA S., KATO S., IWANAMI S., OYAIZU H., SHORAWA H., TSCHZAKI T., 1993. Detection and differentiation of plant pathogenic mycoplasmalike organisms using polymerase chain reaction. Phytopathology 83: 786–791.

SUMA S., PAIS E., 1996. Major diseases affecting sugarcane production on Ramu sugar estate, Papua New Guinea. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 30–33.

WALLER J.M., EGAN B.T., EASTWOOD D., 1987. Ramu stunt, an important new sugarcane disease in Papua New Guinea. Tropical Pest Management 33: 347–349.

White leaf

C.T. Chen and Anusorn Kusalwong

Cause

Phytoplasma, mollicute.

Geographical distribution

Japan (ARAI and UJIHARA, 1989), Pakistan, Sri Lanka, Taiwan and Thailand.

Symptoms

The symptoms of sugarcane white leaf disease are mainly on the leaf blade. The initial symptom of secondary infection is the appearance of a single white or cream streak on the young spindle leaf of the infected stalk. At a later stage, three types of symptoms, i.e. entirely white, stripe and mottled patterns, may develop on the leaves. The stripe, however, always appears; it consists of a single to several stripes following the direction of the veins and usually extending along the entire length of the leaf, but rarely to the upper portion of the leaf sheath. Finally, the spindle leaves become entirely white, which is considered to be the typical symptom of sugarcane white leaf disease (Figures 1, 2 and 3). A few tillers sometimes emerge at the base of an infected plant, and their leaves are usually completely bleached white (Figure 4). The infected plant without any stripe on the stem is stunted. There are no side shoots on the upper part of infected stalks, but the leaves at the top are crowded due to the slow growth of the upper portion of the stem. Besides tillers, buds located a few nodes above the soil surface of infected stalks may also sometimes sprout.

Young cane plants grown from diseased cuttings are smaller when compared with healthy ones. The typical white leaf symptoms always appear on their leaves (Figure 5). However, the symptoms of sugarcane white leaf disease are frequently masked at low temperatures, but reappear as the temperature rises, with chlorosis of the spindle leaves, while the outer leaves remain green and normal. Numbers and size of cane stalks in the diseased plants are reduced resulting in yield reduction (Figure 6). In ratoon crops, plants are severely stunted with all leaves being

white. Death of the diseased stalks regardless of size in the field is notable during the hot and dry season (RISHI and CHEN, 1989) (Figures 7 and 8).

Diagnosis

Visible symptoms are diagnostic for the disease. Electron microscopic examination of ultra-thin sections of infected leaves shows the existence of phytoplasmas in the phloem sieve tubes (Figure 9). Diseased tissues stained with DAPI fluoresce in fluorescence microscopy. Serological methods (ELISA) and molecular biology techniques (DNA probes, DNA amplification by PCR, RFLP and sequencing) have been developed for detection of the causal agent (SARINDU and CLARK, 1993; KLINGKONG and SEEMULLER, 1993; NAKASHIMA *et al.*, 1994 and 1996; NAKASHIMA and HAYASHI, 1997; VISWANATHAN, 1997; WONGKAEW *et al.*, 1997).

Strains of the pathogen

There is no information available.

Transmission

The primary transmission method for the disease is through infected setts. Secondary spread is by the leafhopper *Matsumuratettix hiroglyphicus* Matsumura in a persistent manner.

Host range

Several species of Gramineae including *Saccharum spontaneum, Cynodon dactylon, Brachiaria subquadripara, Dactyloctenium aegyptium, Chloris barbata* and *Sporobolus fertilis* have been found in fields showing white leaf symptoms similar to those on sugarcane. However, only *S. spontaneum* has been confirmed as a natural host of sugarcane white leaf disease (LIN and LEE, 1969; CHEN, 1983). Besides sugarcane, the white leaf agent can infect the *S. officinarum* clone Otaheite, *S. robustum* clone 28NG251 and *S. edule* clone Talur, but *S. officinarum* clone Badila, *S. robustum* clone 28NG219 and *S. sinense* are resistant (CHEN, 1983).

Epidemiology

Planting diseased cane cuttings and leaving infected plants scattered throughout fields are important factors in disease spread, as this is where the insect vector feeds and transmits the disease as a secondary infection. Even high populations of the insect vector in the absence of diseased plants are unable to transmit the pathogen. In Taiwan, the insect vector is abundant from

Figure 1. Leaf symptoms after secondary infection (insect transmission) (C.T. Chen).

Figure 2. Profuse tillering associated with white leaves (C.T. Chen).

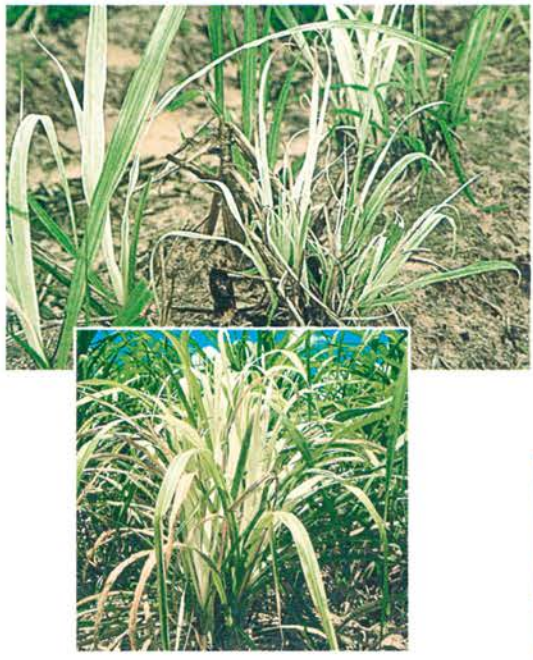


Figure 3. Stools showing white leaf symptoms (A. Kusalwong).

Figure 4. Diseased tillers appearing after secondary infection (insect transmission) (C.T. Chen).

July to October, but the population declines rapidly in December and remains low until April. The disease is more severe for cuttings planted from July to October than for those planted from December to March. The incidence of sugarcane white leaf disease is apparently correlated with the population dynamics of the vector when volunteers are present in the field; hence the greatest incidence is for the July/October planting. The lower incidence of the disease in spring cane is related to lower vector numbers, low temperature effects on the sugarcane white leaf agent in the host plant and vector, and the reduced number of diseased plants due to harvesting operations. The females of the vector 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 soil. The association of dry and warm climate with infertile sandy soil favour high disease incidence.

Economic importance

The sugarcane white leaf disease can cause severe yield losses, particularly when the planting material is obtained from infected sources, or when disease transmission occurs during the early stages of plant crop growth. Cane fields abandoned due to severe infections have been recorded in Taiwan and Thailand. More than 3000 ha of sugarcane plants grown from first ratoon crops in the area of Northeastern Kumpawapee sugar mill in Thailand died during a severe outbreak of the disease. Sugarcane white leaf disease has been almost completely eliminated in Taiwan since the 1980s, and has been under control in Thailand since 1990.

Control

Use of resistant clones to control sugarcane white leaf disease is limited due to the lack of varieties combining high yield with disease resistance. Planting disease-free cuttings, roguing of diseased plants and the prohibition of ratooning in infected fields are, therefore, recommended to control the disease.

Attempts to use thermotherapy to control the disease has not been successful. Hot water treatments for 2–3 h at 50°C or 1 h at 52–53°C are ineffective, and only partial control is obtained with a hot air treatment at 54°C for 8 h. A hot water treatment at 54°C for 50 min kills the causal agent, but germination of the cuttings is also greatly reduced. In Taiwan, when sugarcane white leaf disease was prevalent, adjustment of the planting time (i.e. planting sugarcane in spring to escape secondary infection) was effectively used to control the disease. In Thailand, the disease is now under control in infected areas by the routine use of healthy nurseries, hot water treatment of cuttings for 2 h at 50°C, micropropagation of disease-free plantlets, strict quarantine regulations and various soil amendments (bagasse, filter mud, green manure, waste water from alcohol factories as a nitrogen source...).

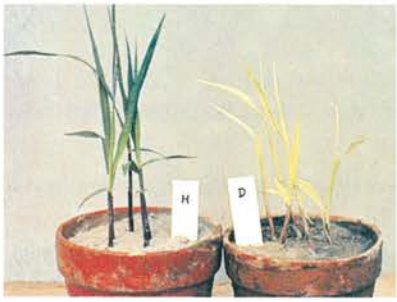


Figure 5. Symptoms of primary infection (right) and healthy cane (left) (C.T. Chen).

Figure 6. Sugarcane row affected by white leaf (A. Kusalwong).

Figure 7. Cane killed by white leaf disease (A. Kusalwong).

Figure 8. Field affected by white leaf disease (A. Kusalwong).

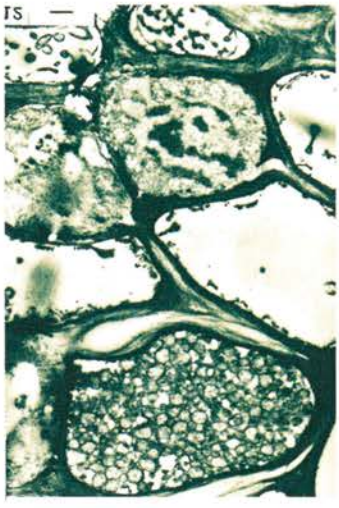


Figure 9. White leaf phytoplasma observed by electron microscopy in the phloem sieve tubes (C.T. Chen).

References

ARAI K., UJIHARA K., 1989. Studies on sugarcane white leaf disease occurred in Tanegashima-island. Technical Bulletin of the Faculty of Agriculture, Kagoshima University 39: 9–16.

CHEN C.T., 1983. Host range of sugarcane white leaf disease. Proceedings International Society of Sugar Cane Technologists Congress 18: 473–485.

KLINKONG S., SEEMULLER E., 1993. Detection and differentiation of the mycoplasma-like organisms association with sugarcane white leaf disease using cloned extrachromosomal DNA probe. Kasetsart Journal, Natural Sciences 27(1): 98–103.

LIN S.C., LEE C.S., 1969. Studies on sugarcane white leaf disease. 2. The relationship between sugarcane white leaf disease and *Saccharum spontaneum* L. Report of Taiwan Sugar Experiment Station 48: 1–9.

NAKASHIMA K., CHALEEPROM W., WONGKAEW P., SIRITHORN P., 1994. Detection of mycoplasma-like organisms associated with white leaf disease of sugarcane in Thailand using DNA probes. Japan International Research Center for Agricultural Sciences Journal 1(1): 57–67.

NAKASHIMA K., HAYASHI T., CHALEEPROM W., WONGKAEW P., SIRITHORN P., 1996. Complex phytoplasma flora in Northeast Thailand as revealed by 16S rDNA analysis. Annals of the Phytopathological Society of Japan 62: 57–60.

NAKASHIMA K., HAYASHI T., 1997. Sequence analysis of extrachromosomal DNA of sugarcane white leaf phytoplasma. Annals of the Phytopathological Society of Japan 63: 21–25.

RISHI N., CHEN C.T., 1989. Grassy shoot and white leaf diseases. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 289–300. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

SARINDU N., CLARK M.F., 1993. Antibody production and identity of MLOs associated with sugarcane white leaf disease and bermuda-grass white leaf from Thailand. Plant Pathology 42(3): 396–402.

VISWANATHAN R., 1997. Detection of phytoplasmas associated with grassy shoot disease of sugarcane by ELISA techniques. Journal of Plant Diseases and Protection 104: 9–16.

WONGKAEW P., HANBOONSONG Y., SIRITHORN P., CHOOSAI C., BOONKRONG S., TINNANGWATTANA T., KICHAREONPANYA R., DAMAK S., 1997. Differentiation of phytoplasmas associated with sugarcane and gramineous weed white leaf disease and sugarcane grassy shoot disease by RFLP and sequencing. Theoretical and Applied Genetics 95: 660–663.

Diseases caused by viruses

Fiji disease

Grant R. Smith

Cause

Fiji disease virus (FDV), type species of the genus *Fijivirus*, family *Reoviridae*.

Geographical distribution

Australia, Fiji, Indonesia, Madagascar (FDV not detected since 1971), Malaysia, New Caledonia, Papua New Guinea, Philippines, Samoa, Solomon Islands, Thailand, Vanuatu.

Symptoms

The definitive diagnostic symptom of Fiji disease is raised whitish-yellow enations (galls) on the abaxial (back) side of the leaf blade and midrib (Figure 1). The galls are the result of hypertrophy (expansion and multiplication) of the phloem and xylem cells in the vascular bundle resulting from viral infection and replication within the cells. Galls generally measure from a few millimetres to 5 cm, although some can be observed extending most of the length of the leaf.

Other symptoms associated with Fiji disease can also be noted but these are not definitive. These symptoms include death of the apical meristem, side shooting, a knife-cut appearance to the edge of newly emerged leaves (Figure 2), stunting and death of the plant (Figure 3). The first symptoms usually observed on a young tiller are a few galls on the leaves. Growth of the stalk slows and the newly formed leaves are shorter and stiffer. The top of the plant becomes fan-like, and in some instances it appears as if the top of the plant has been bitten off (Figure 4). The spindle leaf then dies and is easily detached from the stalk. Plants grown from infected setts are usually stunted, with small, stiff, dark green leaves and often die relatively quickly.

The time between infection and first symptom development can be quite variable depending on the cultivar, age of the plant, level of infection and environmental conditions. Time to symptom development can range from 15 days to several months, although ratooning of a suspect plant will often induce symptoms in the ratoon growth.

Diagnosis

In the field Fiji disease is diagnosed by the presence of the distinctive, definitive galls. Diagnosis of suspect or asymptomatic plants either relies upon the application of molecular assays or waiting for symptoms to develop. Whilst some high specificity, high titre antisera are now available in research laboratories (Soo *et al.*, 1998), earlier sera generally had low titres and limited specificity (Francki *et al.*, 1986), and hence, had limited use for diagnosis. Examination of samples via electron microscopy is not recommended as the virus is in low concentration prior to gall development (Figure 5). The FDV genome consists of ten segments of linear double-stranded RNA. Extraction of the dsRNA and electrophoresis through agarose or acrylamide will reveal the characteristic pattern of the segments. In general, galls are required to extract sufficient dsRNA to conduct this assay, so the characteristic pattern will only confirm, not predict infection.

Both probe and RT-PCR tests have been developed for diagnosis of FDV. The best cDNA probes identified by Smith *et al.* (1994) hybridize within segment 9. The probe, pFDV7, could detect 0.5 pg of purified FDV dsRNA when biotinylated and chemiluminescent substrate was used. In some instances this probe could detect the presence of the virus in the plant prior to symptom development but lacked sufficient sensitivity to routinely detect virus in asymptomatic plants. An RT-PCR system was developed using primers selected from the sequence of pFDV7, namely FDV7F and FDV7R (Smith *et al.*, 1992). This assay was approximately 10^4-fold more sensitive, capable of detecting 100 ag of purified dsRNA. Efforts to improve the sensitivity of this assay, via selection and evaluation of other primer pairs from cDNA clones was not successful (Smith and Van de Velde, 1994). FDV7F and R remain the best RT-PCR primer pair identified to date for molecular diagnosis of *Fiji disease virus.*

Strains of the pathogen

No strains of FDV are officially recognized, although little work has been conducted. There is one report of variation in virulence between FDV isolates from New South Wales, Australia (Hayes, 1974). No research to test these isolates on sets of plant differentials has been reported.

Transmission

FDV is transmitted in infected plant material and by species of the delphacid planthopper genus *Perkinsiella.* Species noted as transmitting FDV are *P. saccharicidia, P. vastatrix* and *P. vitiensis.* There is circumstantial evidence that *P. lalokensis* is also a vector. The virus is transmitted propagatively by adults and second to fifth instar nymphs, after feeding for some hours to acquire the virus. The percentage of adults able to transmit the virus varies (Francki *et al.*, 1986). FDV is not mechanically transmittable.

Figure 1. Galls on the dorsal surface of the leaf blade and midrib (G. Smith, ISSCT).

Figure 2. 'Knife-cut' symptom on newly emerged leaves (G. Smith, ISSCT).

There is some published data on efficiency of viral acquisition by the different nymph instars and adults, but the conclusions from the different studies vary. The most consistent observation is that acquisition of the virus is highest with the youngest instars. CHANG (1977) provided some evidence for transovarial transmission of the virus. The molecular RT-PCR data of PICKERING *et al.* (1998) support the hypothesis that transovarial transmission of FDV occurs.

Host range

Fiji disease virus appears to be limited to members of the *Saccharum* genus, although the disease has been noted in *Erianthus maximus* in Fiji. *Saccharum officinarum* and *S. edule* are relatively susceptible, whilst *S. spontaneum* and *S. robustum* clones are reasonably resistant. Reports of galls in grasses such as Job's tears (*Coix lacryma-jobi*), or wallaby ear of maize should be treated with caution as the structure of the gall is quite different from that found in Fiji disease. FDV has been experimentally transmitted to maize and sorghum by *P. saccharicida* nymphs in glasshouse experiments, but these species have never been naturally observed with Fiji disease (EGAN *et al.*, 1989).

Economic importance

Fiji disease is one of the most important diseases of sugarcane. Despite the use of healthy planting material in Bundaberg, Australia, the susceptible cultivar NCo310 suffered 50% yield loss in the plant crop and 100% loss by the second ratoon. The economic impact of the disease via loss of production, early ploughing up or death of infected stools is further compounded by effects on breeding programmes. During the Bundaberg epidemic approximately 80% of parents were discarded from the breeding populations because of susceptibility to Fiji disease. Considerable genetic potential was lost from the breeding programme, and it is generally considered that it took 10–15 years to recover this lost genetic potential in the southern Queensland breeding programme.

Control

Control of Fiji disease relies upon the use of resistant cultivars, planting of disease-free material, inspection and roguing of diseased stools or the early harvest and ploughing up of diseased crops. Resistance via genetically engineering is being actively pursued in Australia.

The best method to control Fiji disease is via the identification and exploitation of plant resistance in breeding programmes. While the basis of resistance is unknown, highly resistant cultivars have been successfully selected and grown commercially. Screening agronomically elite germplasm for resistance to Fiji disease is resource intensive. If planthopper numbers are low, or only limited

Figure 3. Stunting of sugarcane caused by Fiji disease (ISSCT).

Figure 4. 'Bitten off' aspect of the sugarcane top (ISSCT).

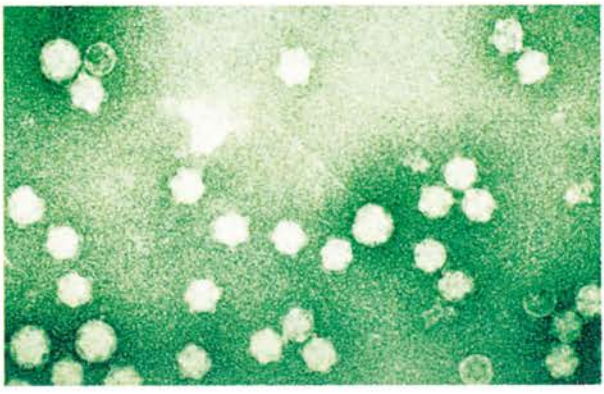


Figure 5. Purified particles of FDV (diameter 50–70 nm) observed by transmission electron microscopy (R. Harding).

infection is noted in the standards and test cultivars, then there is low confidence in the ratings obtained often resulting in a repeat of the trial.

A programme of phytosanitary selection is indispensable in order to obtain healthy material for planting. This system involves a series of regularly inspected nurseries, which may be used to supply planting material if no disease is found. As the incident of a disease increases, or less resistant cultivars are used, then the importance of the remoteness of the nursery from infected fields increases.

Phytosanitary purification via the detection, removal or destruction of infected stools by roguing or ploughing up is an important component of any control programme. The crop can be decontaminated if the level of infection, and the possibility of reinfection, are low. Whilst ploughing up diseased blocks is the ultimate method of roguing, it has serious economic ramifications. Whilst this approach will slow the epidemic, it is potentially more useful after the peak of the infection to remove the remaining sources of infection (EGAN *et al.*, 1989).

References

CHANG V.C.S., 1977. Transovarial transmission of the Fiji disease virus in *Perkinsiella saccharicida* Kirk. Sugarcane Pathologists' Newsletter 18: 22–23.

EGAN B.T., RYAN C.C., FRANCKI R.I.B., 1989. Fiji disease. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 263–287. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

FRANCKI R.I.B., RYAN C.C., HATTA T., ROHOZINSKI J., GRIVELL C.J., 1986. Serological detection of Fiji disease virus antigens in the planthopper *Perkinsiella saccharicida* and its inefficient ability to transmit the virus. Plant Pathology 35: 324–328.

HAYES A.G., 1974. Fiji disease of sugar cane. Evidence for different strains of the causal virus at Condong, N.S.W. Proceedings Conference of the Queensland Society of Sugar Cane Technologists 41: 105–110.

PICKERING L., DIETZGEN R.J., HENDERSON J., SMITH G.R., WHITTLE P.J., MACLEAN D.J., 1998. Development of RT-PCR-ELISA and TaqMan™ assays for Fiji disease virus in sugarcane and its planthopper vector. *In*: Offered papers abstracts, Vol. 2, 7th International Congress of Plant Pathology, Edinburgh, Scotland, 9–16 August 1998. Abstract 1.13.1. Edinburgh, International Society for Plant Pathology.

SMITH G.R., CLARKE M.L., VAN DE VELDE R., DALE J.L., 1994. Chemiluminescent detection of Fiji disease virus in sugarcane with biotinylated DNA probes. Archives of Virology 136: 325–334.

SMITH G.R., VAN DE VELDE R., 1994. Detection of sugarcane mosaic virus and Fiji disease virus in diseased sugarcane using the polymerase chain reaction. Plant Disease 78: 557–561.

SMITH G.R., VAN DE VELDE R., DALE J.L., 1992. PCR amplification of a specific double-stranded RNA region of Fiji disease virus from diseased sugarcane. Journal of Virological Methods 39: 237–246.

SOO H.M., HANDLEY J.A., MAUGERI M.M., BURNS P., SMITH G.R., DALE J.L., HARDING R.M., 1998. Molecular characterisation of Fiji disease fijivirus genome segment 9. Journal of General Virology 79: 3155–3161.

Mild mosaic

Ben E. Lockhart and L. Jean Claude Autrey

Cause

Sugarcane mild mosaic virus (SCMMV), tentative species of the genus *Closterovirus* (Figure 1).

Geographical distribution

Australia, Costa Rica, Malawi, Mauritius, South Africa, Thailand, USA (Florida).

Symptoms

SCMMV has been found naturally in sugarcane only in mixed infections with *Sugarcane bacilliform virus* (SCBV) (Figure 2). In some *Saccharum officinarum* clones the striate mosaic symptoms observed are probably due to SCBV rather than SCMMV infection. In most cultivars, including commercial *Saccharum* hybrids, mixed infection by SCBV and SCMMV is not associated with any distinct foliar symptoms. SCMMV can be separated from SCBV by passage through *S. officinarum* accessions such as IJ76-391 and IK76-69 which support only limited replication of SCBV. When the separated SCMMV is inoculated in the cultivar CL61-620, mild transient mosaic symptoms, similar to those shown in Figure 3, appear in young leaves of vigorously growing plants. In Mauritius, mixed infections of SCBV and SCMMV have been associated with retarded growth, narrow leaves and withering of shoots.

Diagnosis

SCMMV can be detected reliably only by immuno-electron microscopy (IEM) using partially purified leaf extracts (LOCKHART *et al.*, 1992 and 1996). Available antisera to SCMMV were prepared from plants co-infected with SCBV, and these antisera, therefore, do not discriminate between SCMMV and SCBV in ELISA tests.

Strains of the pathogen

No information is available.

Transmission

SCMMV is spread in infected cuttings and can be transmitted by the pink sugarcane mealybug, *Saccharicoccus sacchari*. Transmission tests have not been carried out with other mealybug species such as *Dysmicoccus boninsis*, the grey sugarcane mealybug. Mealybug-transmitted closterolike viruses also occur in pineapple (GUNASINGHE and GERMAN, 1989) and grapevine (ROSCIGLIONE *et al.*, 1983). No clostero- or closterolike viruses are known to be transmitted by seed or pollen (BAR-JOSEPH and MURANT, 1982), and it is also highly unlikely that SCMMV can be transmitted by mechanical contact with cutting tools or machinery.

Host range

SCMMV occurs naturally in *S. officinarum, S. barberi, S. sinense* and *Saccharum* hybrids including M27-16, N14, NCo376, B41227, Pindar and Waya (GANOO *et al.*, 1998). The virus has been transmitted by mechanical inoculation and by *Saccharicoccus sacchari* to rice (*Oryza sativa*) and *Sorghum halepense*, in which mild mosaic symptoms (Figure 3) occur, resembling those induced in sugarcane.

Epidemiology

No information is available.

Economic importance

There is no information on the actual or potential economic importance of SCMMV. The virus was identified in several sugarcane cultivars in Malawi (NCo376, N14, B41227, Waya) and in Mauritius that showed signs of poor growth or decline. These cultivars were all also infected with SCBV and, therefore, the role of SCMMV in the disease syndrome cannot be determined. It is possible that combined infection of SCBV and SCMMV infection may predispose sugarcane to damage by other stress factors or diseases.

Control

SCMMV is apparently not eliminated from sugarcane setts by standard hot water treatments. For germplasm exchange or field propagation, plant material can be indexed by ISEM using partially purified extracts (LOCKHART *et al.*, 1992).

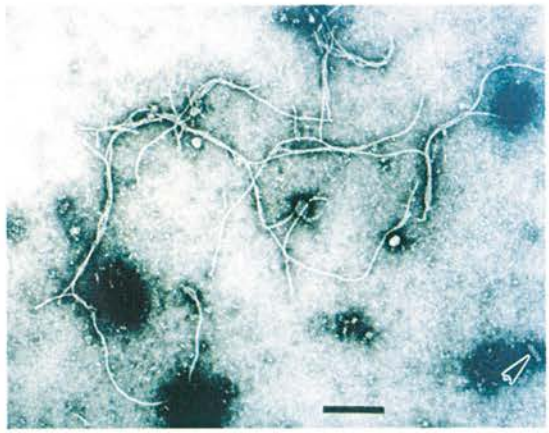


Figure 1. Particles of SCMMV in a partially purified preparation of B41227 from Malawi. This cultivar was also infected with SCBV (indicated by arrowheads); scale bar = 200 nm (B.E. Lockhart).

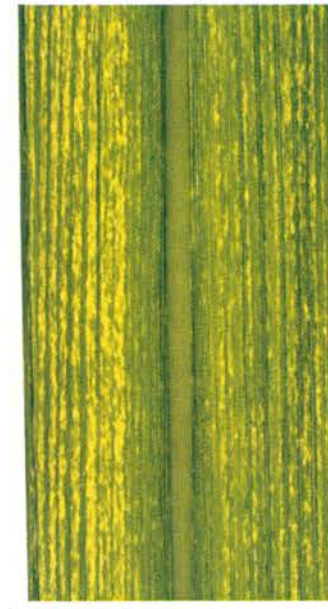


Figure 2. Mild mosaic leaf symptoms in sugarcane (MSIRI).

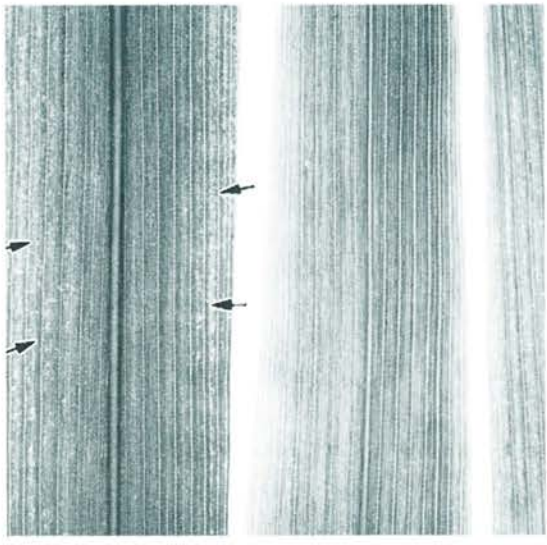


*Figure 3. Mild mosaic symptoms (indicated by arrows) in Johnson grass (*Sorghum halepense*) induced by SCMMV infection following mechanical inoculation. The isolate of SCMMV was obtained from a mixed SCMMV-SCBV infection in the* S. officinarum *accession Selemi Bali and separated from SCBV by passage through IK76-69. Similar symptoms occur transiently in sugarcane infected singly with SCMMV (B.E. Lockhart).*

References

BAR-JOSEPH M., MURANT A.F., 1982. *Closterovirus* group. Descriptions of Plant Viruses No. 260. Kew, UK, Commonwealth Mycological Institute/Association of Applied Biologists, 3p.

GANOO S., AUTREY L.J.C., LOCKHART B.E.L., 1998. Distribution of sugarcane mild mosaic virus in sugarcane germplasm in Mauritius. Sugar Cane 2: 22–24.

GUNASINGHE U.B., GERMAN T.L., 1989. Purification and partial characterization of a virus from pineapple. Phytopathology 79: 1337–1341.

LOCKHART B.E.L., AUTREY L.J.C., COMSTOCK J.C., 1992. Purification and serology of sugarcane mild mosaic virus, a mealybug-transmitted closterolike virus. Phytopathology 82: 691–695.

LOCKHART B.E.L., IREY M.J., COMSTOCK J.C., 1996. Sugarcane bacilliform virus, sugarcane mild mosaic virus and sugarcane yellow leaf syndrome. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 108–112.

ROSCIGLIONE B., CASTELLANO M.A., MARTELLI G.P., SAVINO V., CANNIZZARO G., 1983. Mealybug transmission of grape virus A. Vitis 22: 331–347.

Mosaic

Michael P. Grisham

Cause

Sugarcane mosaic virus (SCMV) and *Sorghum mosaic virus* (SrMV), genus *Potyvirus*, family *Potyviridae*.

Note: mosaic symptoms observed in quarantined sugarcane imported from Pakistan were recently attributed to a new *Rymovirus*, family *Potyviridae: Sugarcane streak mosaic virus* (HALL *et al.*, 1998).

Geographic distribution

Andaman Islands, Angola, Argentina, Australia, Bangladesh, Belize, Bolivia, Brazil, Burundi, Cambodia, Cameroon, Cape Verde, China, Colombia, Congo, Costa Rica, Côte d'Ivoire, Cuba, Democratic Republic of the Congo, Dominican Republic, Ecuador, Egypt, El Salvador, Ethiopia, Fiji, Gabon, Ghana, Guatemala, Haiti, Hawaii, Honduras, India, Indonesia, Iran, Iraq, Italy, Jamaica, Japan, Kenya, Laos, Malawi, Malaysia, Mexico, Morocco, Myanmar, Nepal, Nicaragua, Nigeria, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Puerto Rico, Réunion, St Kitts and Nevis, St Thomas, Sierra Leone, South Africa, Spain, Sri Lanka, Surinam, Swaziland, Tanzania, Taiwan, Thailand, Trinidad, Turkey, Uganda, Uruguay, USA, Venezuela, Vietnam, Zambia, Zimbabwe. The disease was reported but has not been seen for many years in Barbados, Guadeloupe, Madagascar and Martinique.

Symptoms

The general symptom of mosaic is a pattern of contrasting shades of green resulting from varying levels of chlorophyll concentration on the leaf blade (Figure 1). The lighter green or yellowish chlorotic areas usually have diffuse margins, but the response of some cultivars 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 cultivar and virus strain combinations (KOIKE and GILLASPIE, 1989).

Symptoms of mosaic may vary in intensity with cultivar, 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 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 cultivars. With a longitudinal section of the stalk, long necrotic areas are sometimes visible, and the nodal region may have a reddish hue.

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 cultivar 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 (Smith and Van de Velde, 1994; Yang and Mirkov, 1997) protocols are rarely used for routine field diagnosis of mosaic; they have been 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 DNA-based assays are able to confirm that quarantined germplasm or cane to be used for seed cane propagation is mosaic free.

Strains of the pathogen

Within each of the two viruses (SCMV and SrMV) that cause sugarcane mosaic, several strains have been identified on the basis of the symptoms produced on a set of differential host plants. Until recently, all strains were believed to be strains of SCMV. More recent taxonomic studies based on amino acid sequence of the protein coat and nucleic acid analysis resulted in the placement of strains H, I, and M in a new virus taxon, SrMV (McKern *et al.*, 1991; Shukla *et al.*, 1989; Yang and Mirkov, 1997). ELISA can be used to distinguish strains of SCMV from strains of SrMV strains, 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). Both viruses have been reported throughout the world; however, the SCMV strains have been more widely reported among sugarcane production areas. *Johnson grass mosaic virus* (JGMV) does not naturally infect sugarcane. *Maize dwarf mosaic virus* (MDMV) strains B

Figure 1. Mosaic leaf symptoms (M. Grisham).

(SCMV-MDB), D, E, and F do not cause mosaic on sugarcane, but there is a report of an unspecified strain of MDMV being recovered from sugarcane.

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. 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 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, Paspalidium, Paspalum, Pennisetum, Rhynchelytrum, Rottboellia, Setaria, Sorghum, Stenotaphrum, Tripsacum* and *Zea* (KOIKE and GILLASPIE, 1989; RAO *et al.*, 1990). 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. Mosaic in combination with other diseases often reduces growth and yield more than each disease separately. For example, mosaic and ratoon stunting disease (RSD), caused by the bacterium *Clavibacter 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. The diversity of germplasm used in breeding programmes 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) and apical meristem culture. 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 insuring that the early, rapid growth of the sugarcane does not coincide with high levels of vector activity.

References

BENDA G.T.A., 1972. Control of sugarcane mosaic by serial heat treatment. Proceedings International Society of Sugar Cane Technologists Congress 14: 955–960.

GIORDA L.M., TOLER R.W., MILLER F.R., 1986. Identification of sugarcane mosaic virus strain H isolate in commercial grain sorghum. Plant Disease 70: 624–628.

GRISHAM M.P., BURNER D.M., LEGENDRE B.L., 1992. Resistance to the H strain of sugarcane mosaic among wild forms of sugarcane and relatives. Plant Disease 76: 360–362.

HALL J.S., ADAMS B., PARSONS T.J., FRENCH R., LANE L.C., JENSEN S.G., 1998. Molecular cloning, sequencing, and phylogenetic relationships of a new potyvirus: sugarcane streak mosaic virus, and a reevaluation of the classification of the *Potyviridae*. Molecular Phylogenetics and Evolution 10: 323–332.

KOIKE H., 1980. Evidence of resistance in *Saccharum spontaneum* and *Saccharum*-related genera to sugarcane mosaic virus strains H and I. Proceedings International Society of Sugar Cane Technologists Congress 17: 1523–1527.

KOIKE H., GILLASPIE A.G. JR, 1989. Mosaic. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 301–322. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

MCKERN N.M., SHUKLA D.D., TOLER R.D., JENSEN S.G., TOSIC M., FORD R.E., LEON O., WARD C.W., 1991. Confirmation that the sugarcane mosaic virus subgroup consists of four distinct potyviruses by using peptide profiles of coat proteins. Phytopathology 81: 1025–1029.

RAO G.P., SINGH M., SINGH H.N., 1990. Alternative hosts of sugar cane diseases. Sugar Cane, Autumn Supplement: 8–26.

SHUKLA D.D., TOSIC M., JILKA J., FORD R.E., TOLER R.W., LANGHAM M.A.C., 1989. Taxonomy of potyviruses infecting maize, sorghum and sugarcane in Australia and the United States as determined by reactivities of polyclonal antibodies directed towards virus-specific N-termini of coat proteins. Phytopathology 79: 223–229.

SMITH G.R., VAN DE VELDE R., 1994. Detection of sugarcane mosaic virus and Fiji disease virus in diseased sugarcane using the polymerase chain reaction. Plant Disease 78: 557–561.

YANG Z.N., MIRKOV T.E., 1997. Sequence and relationships of sugarcane mosaic and sorghum mosaic virus strains and development of RT-PCR-based RFLPs for strain discrimination. Phytopathology 87: 932–939.

Red leaf mottle

Philippe Rott and Michèle Chatenet

Cause

Peanut clump virus (PCV), genus *Pecluvirus*.

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):
– chlorotic bands, elongated over the length of the leaf, from 1 cm wide to as wide as the blade; they can appear spotted, yellow or rust-coloured (Figure 1); the entire leaf can become dark red with age (cultivar Co1001, B70574, etc.);
– numerous wine-red spots with diffuse edges and no clearly defined shape develop on both sides of the blade in some varieties (cultivar Ragnar, etc.) (Figures 2 and 3);
– white stripes appear on the leaves of some varieties; they resemble the stripes caused by leaf scald but have wavy edges and develop on any part of the blade without being linked to the midrib (Figure 4). A few isolated leaves develop white diamond-shaped or partly diamond-shaped stripes on the surface of the leaves (cultivar 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. Particle lengths range from 200 to 250–300 nm and the diameter is approximately 20 nm (Figure 5). 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).

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 groundnut to groundnut by a soil fungus, *Polymyxa graminis* (THOUVENEL and FAUQUET, 1981). The disease is also transmitted by cuttings taken from symptomatic sugarcane stalks. In groundnut, the virus can also be seed-transmitted (4–14%).

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), and in wheat in India (DELFOSSE *et al.*, 1995). 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 a high pH (7.0 and higher) particularly favour transmission. In some fields, there are areas of infected sugarcane where the disease reappears after replanting, even if a different variety is grown.

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 fields so far.

Control

No efficient method has yet been reported to eliminate the virus from infected cuttings. Soaking cuttings in water for 3 h at 50°C does not eradicate the

Figure 1. Chlorotic elongated bands on the leaf with numerous rust-coloured spots (P. Baudin).

Figure 2. Numerous wine-red leaf spots (P. Baudin).

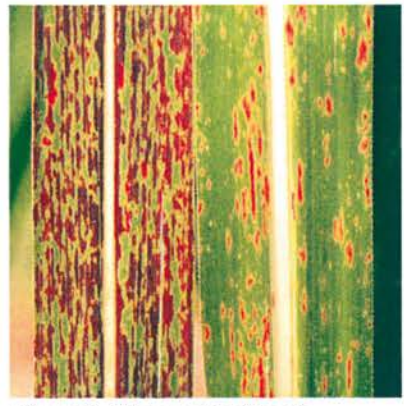


Figure 3. Wine-red leaf spots with diffuse edges on both sides of the blade (P. Rott).

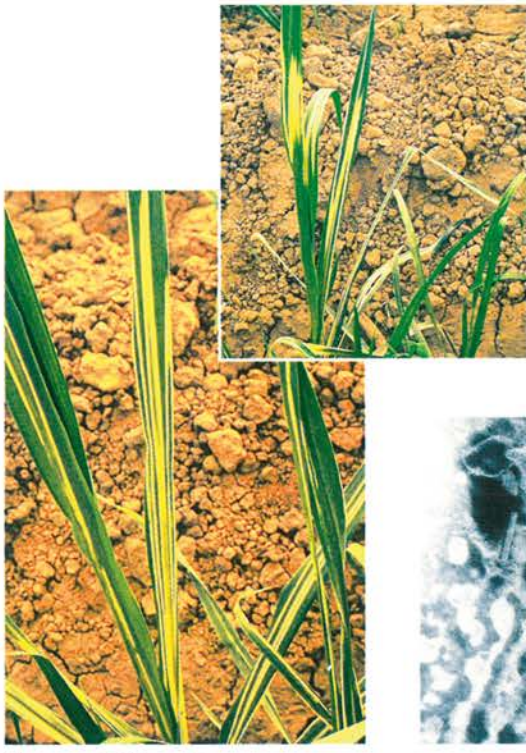


Figure 4. White stripes on the leaves (P. Baudin).

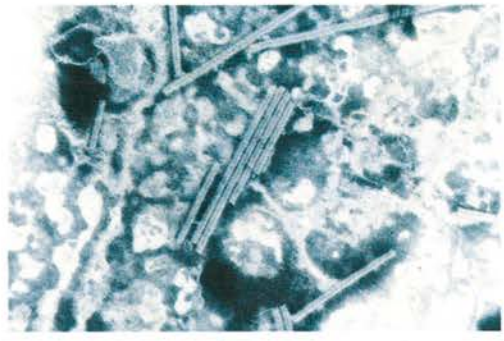


Figure 5. Virus particles (200–300 nm long) observed by transmission electron microscopy (M. Chatenet).

disease. Stools that show symptoms should be eliminated in multiplication plots. 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.

References

BAUDIN P., CHATENET M., 1988. Détection sérologique du PCV, isolat canne à sucre, agent de la marbrure rouge des feuilles. L'Agronomie Tropicale 43: 228–235.

BAUDIN P., SENE A., MARION D., 1989. Effect of peanut clump virus on the yields of two sugarcane varieties. *In*: Current Trends in Sugarcane Pathology (Prof. K.S. Bhargava Festschrift), G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin Filho, V.P. Agnihotri and C.T. Chen (Eds), p. 141–150. Dehli, India, International Books and Periodicals Supply Service.

CHATENET M., SAEED I., 1995. First report of sugarcane red leaf mottle virus in Sudan. Plant Disease 79: 321.

DELFOSSE P., REDDY A.S., DEVI P.S., MURTHY A.K., WESLEY S.V., NAIDU R.A., REDDY D.V.R., 1995. A disease of wheat caused by Indian peanut clump virus (IPCV). Plant Disease 79: 1074.

DOLLET M., FAUQUET C., THOUVENEL J.C., 1976. *Sorghum arundinaceum* a natural host for peanut clump virus in Upper Volta. Plant Disease Reporter 60: 1076–1080.

HUGUENOT C., GIVORD L., SOMMERMEYER G., VAN REGENMORTEL M.H.V., 1989. Differentiation of peanut clump virus serotypes by monoclonal antibodies. Research in Virology 140: 87–102.

RAO G.P., SINGH G.P., 1999. Serological diagnosis of peanut clump virus – Indian sugarcane isolate. *In*: Abstracts of the 51st Annual Meeting and National Symposium on Seed Health Care and Phytosanitation for Sustainable Agriculture of the Indian Phytopathological Society, Lucknow, India 17–19 February 1999. Abstract P.107, p. 55. Lucknow, Indian Institute of Sugarcane Research/Ganna Kisan Sansthan.

REDDY D.V.R., RAJESHWARI R., IIZUKA N., LESEMANN D.E., NOLT B.L., GOTO L., 1983. The occurrence of Indian peanut clump, a soil-borne virus disease of groundnuts (*Arachis hypogea*) in India. Annals of Applied Biology 102: 305–310.

REDDY D.V.R., ROBINSON D.J., ROBERTS I.M., HARRISON B.D., 1985. Genome properties and relationships of Indian peanut clump virus. Journal of General Virology 66: 2011–2016.

ROTT P., 1996. Red leaf mottle. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 125.

THOUVENEL J.C., FAUQUET C., 1981. Further properties of peanut clump virus and studies on its natural transmission. Annals of Applied Biology 97: 99–107.

THOUVENEL J.C., DOLLET M., FAUQUET C., 1976. Some properties of peanut clump, a newly discovered virus. Annals of Applied Biology 84: 311–320.

Streak

Philippe Rott and Michel Peterschmitt

Cause

At least three species of viruses of the genus *Mastrevirus*, family *Geminiviridae*, cause streak symptoms in sugarcane: *Sugarcane streak virus* (SSV), *Sugarcane streak Mauritius virus* (SSMV) and *Sugarcane streak Egypt virus* (SSEV) (Bigarré *et al.*, 1999).

Geographical distribution

Benin, Cape Verde, Côte d'Ivoire, Egypt, India, Kenya, Madeira, Malawi, Mauritius, Mozambique, Pakistan, Réunion, South Africa, Sudan, Uganda, Zimbabwe.

Symptoms

Infected leaves are covered with numerous small spots or streaks which are translucent, narrow and parallel to the veins (Figure 1). 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 from 0.5 to 2 mm in width and vary in length from 0.5 mm to more than 2 cm. 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).

Diagnosis

Symptoms and transmission tests in maize or susceptible sugarcane varieties are useful 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, these techniques cannot be used to distinguish the different virus species causing streak disease.

Serological techniques using monoclonal antibodies prepared against *Maize streak virus* (MSV) isolates can be used to detect the pathogen (DEKKER *et al.*, 1988; PETERSCHMITT *et al.*, 1991). However, none of these antibodies allows the detection of all three species causing streak because the similarity of their coat protein peptide sequences is relatively low, ranging from 73 to 85% (BIGARRÉ *et al.*, 1999). An antiserum against a sugarcane streak isolate is available in South Africa (SASEX) but its efficiency against all three species causing streak in sugarcane has yet to be proven.

PCR tests could be developed for very sensitive and accurate diagnosis since an increasing number of sequences of streak causing mastreviruses are now available (BIGARRÉ *et al.*, 1999).

Nuclear viral aggregates, typically observed for viruses of the family *Geminiviridae*, were detected for sugarcane streak isolates from Egypt and Uganda in sugarcane plants by *in situ* observations using transmission electron microscopy (TEM) (AMMAR, 1994; BOCK and BAILEY, 1989). A simple light microscopy technique developed for the detection of such aggregates in dicotyledonous plants infected by geminiviruses may also be used for sugarcane showing streak symptoms (CHRISTIE *et al.*, 1986). However, in the case of sugarcane streak isolates from Mauritius and South Africa, these *in situ* microscopy techniques may not work because virions were found to be randomly dispersed within the nucleus (PINNER *et al.*, 1993). Alternatively, typical twinned quasi isometric geminivirus particles, approximately 20 × 30 nm in size, can be observed by TEM using purified virus extracts (BOCK and BAILEY, 1989).

Strains of the pathogen

Sugarcane streak disease is caused by at least three virus species, *Sugarcane streak virus* (SSV) isolated in South Africa, *Sugarcane streak Mauritius virus* (SSMV) isolated in Mauritius, Réunion and Nigeria, and *Sugarcane streak Egypt virus* (SSEV) isolated in Egypt (BIGARRÉ *et al.*, 1999). In about 10 years, understanding causal agents of sugarcane streak disease has evolved from several strains of *Maize streak virus* (MSV) to several distinct *Mastrevirus* species. This evolution can easily be explained by the improvement of identification and characterization techniques. Based on biological, physical and immunological techniques, the viruses were considered to be different strains or serotypes of MSV because the viruses causing streak in sugarcane and maize had the same morphology (twinned quasi-isometric particles), were transmitted by the same vectors (species of the genus *Cicadulina*), and were immunologically related (BOCK and BAILEY, 1989; DEKKER *et al.*, 1988; PETERSCHMITT *et al.*, 1991). Using molecular techniques (hybridization, RFLP and sequencing) it was proposed that the streak virus isolates of sugarcane from Mauritius and South Africa be considered as distinct mastreviruses (HUGHES, 1994; HUGHES *et al.*, 1993).

Figure 1. Sugarcane leaves with small spots and narrow streaks (P. Baudin, P. Rott, B. Vercambre).

This new classification was further confirmed by the complete sequences of five more clones showing that sugarcane streak disease is caused by at least three distinct virus species (BIGARRÉ *et al.*, 1999). Nucleotide similarities within the complete sequences of four SSEV clones and between partial sequences of a SSMV clone from Mauritius and one from Réunion are higher than 97% suggesting that intraspecies variability is low (BIGARRÉ *et al.*, 1999). A slightly higher variability was obtained with a SSMV strain isolated from millet in Nigeria since it showed only 92% nucleotide identity in the CP gene when compared to the Réunion and Mauritius SSMV isolates which are 99% similar for this gene. The suggested low intraspecies variability is supported by the fact that SSV isolates generally do not lose their ability to be vector-transmitted, although this function is not required for their survival due to the vegetative propagation of their host. This is, however, consistent with the fact that the coat protein of mastreviruses is not only responsible for insect transmission, but also for movement in the host plant (PALMER and RYBICKI, 1998).

Golden Bantam maize plants infected by SSMV isolates showed milder symptoms than plants infected by SSV and SSEV isolates (BIGARRÉ *et al.*, 1999; PINNER *et al.*, 1988).

Transmission

The sugarcane streak mastreviruses cannot be transmitted mechanically from plant to plant or through seed (BOCK and BAILEY, 1989). They are transmitted through cuttings taken from infected plants and by leafhopper species of the genus *Cicadulina*, particularly by *C. mbila* and *C. bipunctella*.

Host range

The sugarcane streak mastreviruses can be transmitted to maize by *Cicadulina mbila*, and infection results in a pattern of fine streaks resembling those caused by the pathogen in sugarcane (PINNER *et al.*, 1988). The symptoms are, however, milder than those caused by MSV isolates. *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 SSMV 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

Natural infection occurring in *Cenchrus echinatus* is believed to constitute a natural reservoir of streak mastreviruses in Mauritius (BOCK and BAILEY, 1989).

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 cultivars should be eliminated. Resistance of sugarcane to streak can be screened in breeding programmes by planting varieties between rows of diseased plants. When necessary (quarantine etc.), virus-free plants can be obtained from infected sugarcane by tissue culture (PEROS *et al.*, 1990).

References

AMMAR E.D., 1994. Cytopathology and ultrastructure of some African isolates of maize streak and sugarcane streak viruses. Journal of Phytopathology 141: 153–158.

BIGARRÉ L., SALAH M., GRANIER M., FRUTOS R., THOUVENEL J.C., PETERSCHMITT M., 1999. Nucleotide sequence evidence for three distinct sugarcane mastreviruses. Archives of Virology 144: 2331–2344.

BOCK K.R., BAILEY R.A., 1989. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 323–332. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

CHRISTIE R.G., KO N.J., FALK B.W., HIEBERT E., LASTRA R., BIRD J., KIM K.S., 1986. Light microscopy of geminivirus induced nuclear inclusion bodies. Phytopathology 76: 124–126.

DEKKER E.L., PINNER M.S., MARKHAM P.G., VAN REGENMORTEL M.H.V., 1988. Characterization of maize streak virus isolates from different plant species by polyclonal and monoclonal antibodies. Journal of General Virology 69: 983–990.

HUGHES F.L., 1994. Sugarcane streak. *In*: Current Trends in Sugarcane Pathology (Prof. K.S. Bhargava Festschrift). G.P. Rao, A.G. Gillaspie Jr, P.P. Upadhyaya, A. Bergamin Filho, V.P. Agnihotri and C.T. Chen (Eds), p. 131–140. Delhi, India, International Books and Periodicals Supply Service.

HUGHES F.L., RYBICKI E.P., KIRBY R., 1993. Complete nucleotide sequence of sugarcane streak monogeminivirus. Archives of Virology 132: 171–182.

PALMER K.E., RYBICKI E.P., 1998. The molecular biology of mastreviruses. Advances in Virus Research 50: 183–234.

PEROS J.P., BONNEL E., REYNAUD B., 1990. *In vitro* culture of sugarcane infected with maize streak virus (MSV). Plant Cell, Tissue and Organ Culture 23: 145–149.

PETERSCHMITT M., REYNAUD B., SOMMERMEYER G., BAUDIN P., 1991. Characterisation of maize streak virus isolates using monoclonal and polyclonal antibodies and by transmission to a few hosts. Plant Disease 75: 27–32.

PINNER M.S., MARKHAM P.G., MARKHAM R.H., DEKKER E.L., 1988. Characterization of maize streak virus: description of strains; symptoms. Plant Pathology 37: 74–87.

PINNER M.S., MEDINA V., PLASKITT K.A., MARKHAM P.G., 1993. Viral inclusions in monocotyledons infected by maize streak and related geminiviruses. Plant Pathology 42: 75–87.

STOREY H.H., THOMSON G.M., 1961. Streak disease. *In*: Sugar-Cane Diseases of the World, Vol. 1. J.P. Martin, E.V. Abbott and C.G. Hughes (Eds), p. 461–476. Amsterdam, The Netherlands, Elsevier Publishing Company.

Striate mosaic

Barry J. Croft and John W. Randles

Cause

Double-stranded RNA (dsRNA) about 9 kilobase pairs (9 kbp) in size has been isolated from sugarcane striate mosaic disease affected sugarcane leaves (Choi and Randles, 1997). A novel relatively rigid rod-shaped virus approximately 950 nm in length and 15 nm in diameter, whose RNA has nucleotide sequence similar to the dsRNA, has been purified in small amounts from affected sugarcane. It has distant sequence similarity to one *Carlavirus* and two *Trichoviruses* but appears not to fit into either of these two virus genera. This Sugarcane striate mosaic associated virus (ScSMaV) is the possible causal agent of striate mosaic (Choi 1997, Choi *et al.*, 1999).

Geographical distribution

In the Burdekin River district, northern Queensland, Australia. A similar striate mosaic of unknown cause has been reported from India (Nayudu *et al.*, 1971).

Symptoms

Infected plants are severely stunted with a general yellow appearance. They often die or fail to ratoon leaving large bare patches in fields. Symptoms expressed in glasshouse plants grown at 25–30°C are short chlorotic leaf striations on all but the youngest two leaves (Figure 1). Longer and broader chlorotic striations are also visible on some leaf sheaths and on the primary stems of cuttings germinated from affected canes (Figure 2), and on ratoons. Affected canes have shortened internodes and a reduced diameter (Figure 3) (Choi, 1997).

Diagnosis

In the field, the fine chlorotic striations on leaves are the diagnostic symptom but this symptom is not always clear. During hot weather (>30°C) leaf striations are generally not present. dsRNA extraction and assay from glasshouse-grown affected plants gives a characteristic 9 kbp band (Choi, 1997; Choi and Randles, 1997). Detection of this band can be used as a preliminary diagnostic assay in conjunction with symptomatology. A more reliable and sensitive

sequence specific nucleic acid based diagnostic method for ScSMaV is under development (N. Thompson, personal communication).

Strains of the pathogen

Variants of the striate mosaic disease in sugarcane have not been investigated.

Transmission

Striate mosaic can be spread in infected planting material (HUGHES, 1961). Experimentally the disease has been transmitted by the Sein needle-prick technique (HUGHES, 1964) and by planting healthy cuttings in pots of soil from affected fields (C.C. Ryan, personal communication). In the field, there does not appear to be active spread from affected patches. No vector has been identified.

Host range

The disease has been reported only 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 1 ha in area. The disease is associated with sandy or shallow soils in fields, which generally have rich deep alluvial soils. Striate mosaic generally is not detected in plant crops except where diseased planting material has been used. The disease usually develops in ratoon crops.

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. Severe stunting and death of plants in patches of up to 1 ha in area are not uncommon. The 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 up.

Control

Varieties with resistance to striate mosaic have been used to control the disease. Selection of disease-free planting material is essential. 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).

Figure 1. Numerous short chlorotic striations on infected leaf (left) and healthy leaf (right) (N. Thompson).

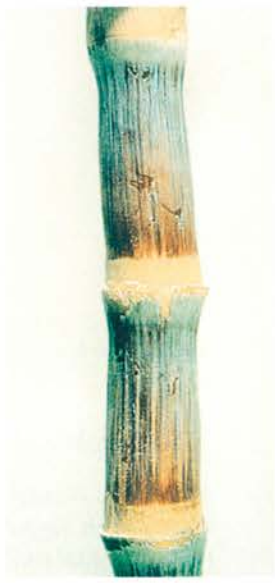


Figure 2. Stalk striations associated with striate mosaic (Y.G. Choi).

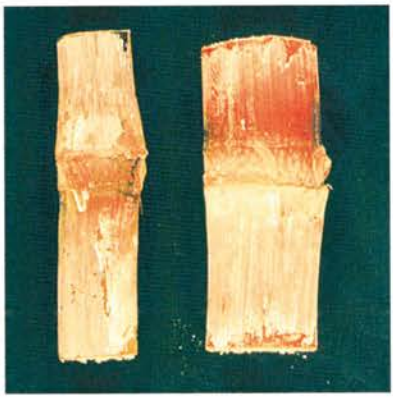


Figure 3. Internodes of diseased (left) compared with normal (right) sugarcane showing reduced cane diameter (Y.G. Choi).

References

Choi Y.G., 1997. The etiology of sugarcane striate mosaic disease. PhD Thesis. Australia, University of Adelaide, 106p.

Choi Y.G., Randles J.W., 1997. Microgranular cellulose improves dsRNA recovery from plant nucleic acid extracts. Biotechniques 23: 610–611.

Choi Y.G., Croft B.J., Randles J.W., 1999. Identification of sugarcane striate mosaic-associated virus by partial characterization of its double-stranded RNA. Phytopathology 89: 877–883.

Hughes C.G., 1961. Striate mosaic: a new disease of sugarcane. International Sugar Journal 63: 298–299.

Hughes C.G. 1964. Striate mosaic. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 192–195. Amsterdam, The Netherlands, Elsevier Publishing Company.

Nayudu M.V., Sastry K.S., Sarma Y.R., 1971. Striate mosaic of sugarcane in India. Sugarcane Pathologists' Newsletter 7: 24.

Sugarcane bacilliform virus

Ben E. Lockhart and L. Jean Claude Autrey

Cause

Sugarcane bacilliform virus (SCBV), genus *Badnavirus* (Figure 1).

Geographical distribution

Argentina, Australia, Barbados, Brazil, Burundi, Cape Verde, Colombia, Cuba, Dominican Republic, Guadeloupe, Hawaii, India, Indonesia, Madagascar, Madeira, Malawi, Mauritius, Mexico, Morocco, Papua New Guinea, Puerto Rico, Réunion, South Africa, Taiwan, Thailand, USA; occurs in all tested clones of *Saccharum officinarum* (COMSTOCK and LOCKHART, 1990), and probably occurs world-wide.

Symptoms

In most sugarcane varieties SCBV infection is not associated with foliar symptoms. Varying degrees of whitish to yellow chlorotic streaking occurs in some *S. officinarum* clones such as Iscambine, NG77-064, M27-16, Selemi Bali (Figures 2 and 3), NG57-239, Jamaica Red. These symptoms are not reproduced when these virus isolates are transmitted to commercial *Saccharum* hybrids. Foliar symptom expression may, therefore, be both virus isolate-dependent as well as host cultivar-dependent. The effect of SCBV infection on other growth parameters of *S. officinarum* has not been determined because no virus-free clones of *S. officinarum* have so far been identified.

Diagnosis

SCBV can be detected by serological methods, including ELISA, which is useful for large-scale indexing. Because of the high degree of serological variability among virus isolates (LOCKHART *et al.*, 1996), DAS-ELISA is unreliable for screening germplasm. A TAS-ELISA protocol (NDOWORA, 1998) gives more dependable results. The virus can be detected with great sensitivity by immuno-electron microscopy (IEM) using partially purified extracts (AHLAWAT

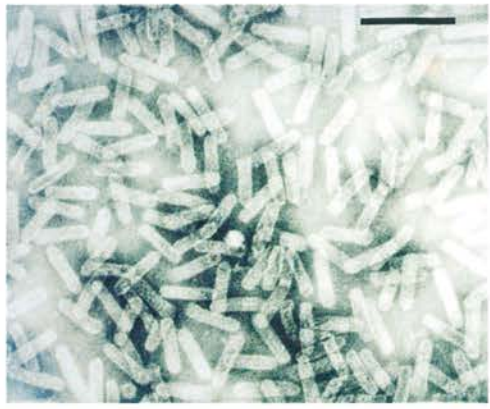


Figure 1. SCBV virions observed by negative-stain transmission electron microscopy (scale bar = 200 nm) (B.E. Lockhart).

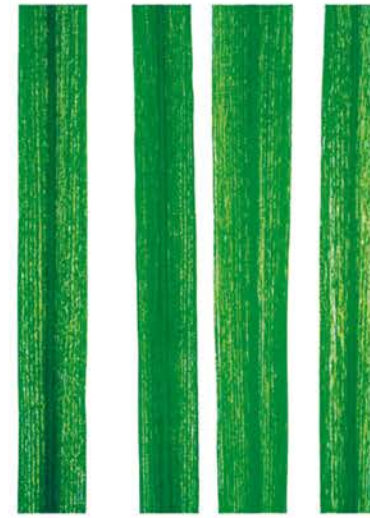


Figure 2. Striate chlorotic mosaic symptoms associated with SCBV infection in four Saccharum officinarum *clones; from left: Selemi Bali, Iscambine, M27-16, NG77-064 (B.E. Lockhart).*

Figure 3. Leaf symptoms of sugarcane infected by SCBV (MSIRI).

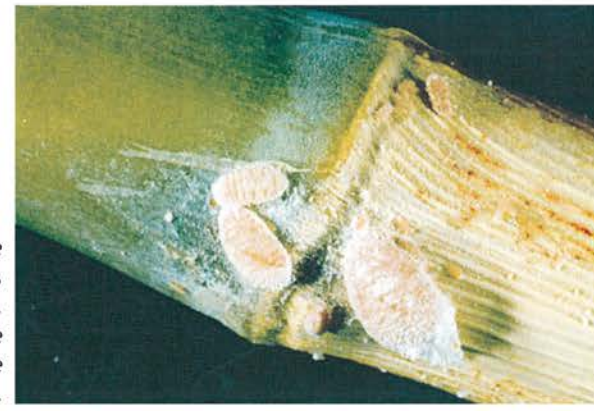


Figure 4. The pink sugarcane mealybug, Saccharicoccus sacchari, *a vector of SCBV, at characteristic feeding site at sugarcane stalk node (B.E. Lockhart).*

et al., 1996; LOCKHART *et al.*, 1996), or by immunocapture PCR (IC-PCR). Detection of SCBV by PCR using total plant DNA (BRAITHWAITE *et al.*, 1995) may be unreliable because of the presence of badnaviral sequences integrated into the host genome (LA FLEUR *et al.*, 1996), similarly to the integration of badnaviral sequences in Musa (LA FLEUR *et al.*, 1996; NDOWORA *et al.*, 1999).

Strains of the pathogen

No information is available.

Transmission

SCBV is transmitted by cuttings and by mealybugs. The pink sugarcane mealybug (*Saccharicoccus sacchari*) (Figure 4) and the grey sugarcane mealybug (*Dysmicoccus boninsis*) are efficient vectors of the virus, which is probably transmitted in a semi-persistent manner (LOCKHART and OLSZEWSKI, 1994). The virus is also transmitted by *Planococcus citri*, the citrus mealybug, which does not normally colonize sugarcane. There is no evidence that SCBV is pollen- or seed-transmitted, even though several other *Badnaviruses*, including the closely related *Banana streak virus* (BSV) are transmitted in this manner (LOCKHART and OLSZEWSKI, 1994; DANIELLS *et al.*, 1995). Like other *Badnaviruses*, SCBV is not easily transmitted by mechanical inoculation, and it is, therefore, unlikely to be spread from infected to healthy canes on cutting tools and machinery during normal cultural operations.

Host range

SCBV is unusual among *Badnaviruses* (LOCKHART and OLSZEWSKI, 1994) in that it infects several different host plant genera and species. The virus occurs naturally in all tested *S. officinarum* clones and in *S. barberi, S. robustum, S. sinense, S. spontaneum, Saccharum* hybrids, *Sorghum halepense, Brachiaria* sp., *Panicum maximum* and *Rottboellia exaltata*, and has been transmitted experimentally to rice (*Oryza sativa*) (Figure 5), banana (*Musa* AAA 'Dwarf Cavendish') (Figure 6) and sorghum (*Sorghum vulgare*). SCBV is closely related to BSV (LOCKHART and AUTREY, 1988), and symptoms induced by SCBV infection in banana (Figure 6) are indistinguishable from those caused by BSV. Although SCBV is readily transmitted by *S. sacchari* from sugarcane to banana, which it colonizes, there is no evidence that this occurs in nature.

Epidemiology

No information is available.

Figure 5. Symptoms induced by SCBV infection in rice: infected leaf showing chlorotic streaks (right) and healthy leaf (left) (B.E. Lockhart).

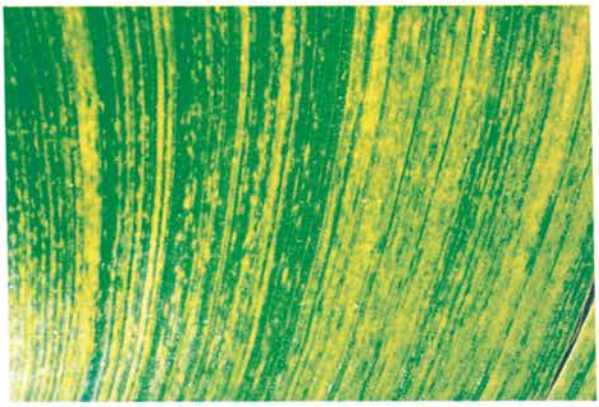


*Figure 6. Striate mosaic symptoms induced by SCBV infection in Dwarf Cavendish banana; the SCBV isolate was transmitted by the citrus mealybug (*Planococcus citri*) from the symptomatic sugarcane accession NG77-064 (B.E. Lockhart).*

Economic importance

There are only limited data on the economic importance of SCBV infection in sugarcane. Variable results were obtained in a 3-year field trial to assess the effect of SCBV infection on biomass production in three commercial sugarcane cultivars. Biomass production was significantly lower in CP63-588, significantly higher in CL61-620 and variably higher or lower in CP65-357 (COMSTOCK and LOCKHART, 1996). Virus titre was highest in CP63-588 and lowest in CL61-620. These results suggest that SCBV infection may lead to significant losses in biomass production in sugarcane cultivars susceptible to high levels of virus multiplication (e.g. noble canes). There appears to be a synergistic interaction between SCBV and *Sugarcane mosaic potyvirus* (SCMV) in sugarcane. This suggests that the effect of mosaic infection may be exacerbated in cultivars infected with SCBV.

Control

SCBV infection in sugarcane is not eliminated by normal thermotherapy, tissue culture or thermotherapy followed by apical meristem culture. Spread of SCBV can be prevented by the use of virus-free planting material. Within-field spread (e.g. in germplasm collections) can be reduced by controlling vectors such as the pink and grey sugarcane mealybugs.

References

AHLAWAT Y.S., PANT R.P., LOCKHART B.E.L., SRIVASTAVA M., CHAKRABORTY N.K., VARMA A., 1996. Association of a badnavirus with citrus mosaic disease in India. Plant Disease 80: 590–592.

BRAITHWAITE K.S., EGESKOV N.M., SMITH G.R., 1995. Detection of sugarcane bacilliform virus using the polymerase chain reaction. Plant Disease 79: 792–796.

COMSTOCK J.C., LOCKHART B.E.L., 1990. Widespread occurrence of sugarcane bacilliform virus in U.S. sugarcane germplasm collections. Plant Disease 74: 530.

COMSTOCK J.C., LOCKHART B.E.L., 1996. Effect of sugarcane bacilliform virus on biomass production of three sugarcane cultivars. Sugar Cane 4: 12–15.

DANIELLS J., THOMAS J.E., SMITH M., 1995. Seed transmission of banana streak virus confirmed. Infomusa 4: 7.

LA FLEUR D.A., LOCKHART B.E.L., OLSZEWSKI N.E., 1996. Portions of the banana streak badnavirus genome are integrated in the genome of its host *Musa*. Phytopathology 86: S100–S101.

LOCKHART B.E.L., AUTREY L.J.C., 1988. Occurrence in sugarcane of a bacilliform virus related serologically to banana streak virus. Plant Disease 72: 230–233.

LOCKHART B.E.L., OLSZEWSKI N.E., 1994. Badnavirus group. *In*: The Encyclopedia of Virology. R.G. Webster and A. Granoff (Eds), Vol. 1, p.139–143. New York, USA, Academic Press.

LOCKHART B.E.L., IREY M.J., COMSTOCK J.C., 1996. Sugarcane bacilliform virus, sugarcane mild mosaic virus and sugarcane yellow leaf syndrome. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 108–112.

NDOWORA T.C.R., 1998. Development of an enzyme immunoassay to detect serologically diverse isolates of banana streak virus and characterization of viral sequences integrated into the *Musa* genome. PhD Thesis. St Paul, USA, University of Minnesota, 83p.

NDOWORA T., GAHAL D., LA FLEUR D., HARPER G., HULL R., OLSZEWSKI N.E., LOCKHART B., 1999. Evidence that badnavirus infection in *Musa* can originate from integrated pararetroviral sequences. Virology 255: 214–220.

Diseases of uncertain etiology

Apex rot

Philippe Rott and Jean 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).

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 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.

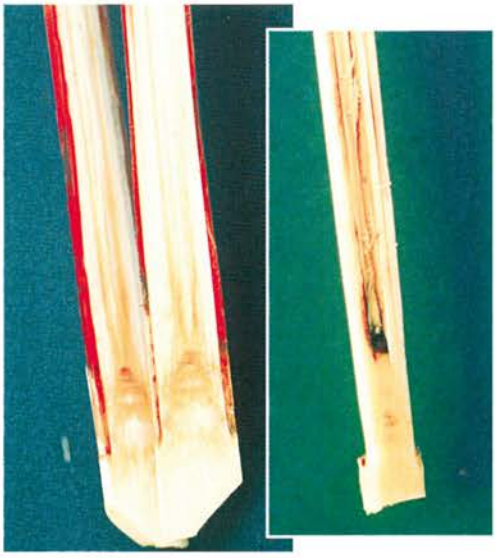


Figure 1. Necrosis of the internal tissues above meristem (P. Feldmann).

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. Daugrois).

Figure 5. Stalks killed by apex rot (P. Feldmann).

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 cultivars 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.

Reference

ROTT P., FELDMANN P., 1991. Les maladies de la canne à sucre en Guadeloupe : situation actuelle et méthodes de lutte mises en place. *In*: Actes de la Première Rencontre Internationale en Langue Française sur la Canne à Sucre, Montpellier, France, 10–15 juin 1991, p. 90–94. Nogent-sur-Marne, France, Association Française de la Canne à Sucre.

Chlorotic streak

Robert C. Magarey and Brian T. Egan

Cause

Causal agent is unknown; some evidence suggests a viral agent.

Geographical distribution

Argentina, Australia, Brazil, Cambodia, China, Colombia, Côte d'Ivoire, Cuba, Dominican Republic, Fiji, Grenada, Guadeloupe, Guyana, Hawaii, Honduras, Indonesia, Jamaica, Madagascar, Mali, Martinique, Mauritius, Mexico, Mozambique, Nicaragua, Pakistan, Panama, Papua New Guinea, Philippines, Puerto Rico, Réunion, St Lucia, Samoa, South Africa, Surinam, Taiwan, Thailand, Trinidad, Turkey, USA, Venezuela.

Symptoms

The disease is characterized by yellow to creamy-white leaf streaks with wavy, irregular margins (Figure 1). Streaks caused by leaf scald have much more defined, straight margins.

Young leaf streaks are irregular, often fragmented, short and faint in colour. As the symptoms progress, streaks generally elongate and become more pronounced. Older streaks take on a yellow colour, and in many instances the central parts of the streak become necrotic, either in sections or along the full length of the streak. These necrotic tissues are ash-grey and may have a red-brown margin where they meet healthy tissue. Leaf tips and margins may also show necrosis, especially where streaks meet the edge of the leaf. Though exhibiting some scalding type symptoms, chlorotic streak symptoms differ from leaf scald in that there is no inward curling of the affected leaves, no pencil-line within leaf stripes, and leaf striping is much more irregular with chlorotic streak.

Leaf streaks vary in width, ranging from very narrow to 10 mm; 3–6 mm is the normal size range. Streaks appear similar on both the upper and lower leaf surfaces, and may be found on the leaf sheath and midrib.

Internal stalk symptoms include discoloured 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. ABBOTT and SASS (1945) investigated the histopathology of chlorotic streak.

Symptom expression is environmentally dependent, mainly on high soil moisture and temperature. This leads to transient symptoms; STURGESS (1962) showed that soil temperature affects the expression of symptoms. Dry conditions do not favour symptom development.

Diagnosis

There is no specific assay for chlorotic streak as the causal agent has not been identified. Identification of the disease relies on the recognition of leaf and internal stalk symptoms. Where symptoms are expressed, this is not usually a problem. Difficulties arise where there is transient symptom expression or where the intensity of symptoms is such that it is difficult to separate chlorotic streak from other scalding type effects (for instance leaf scald or soil-based constraints).

Strains of the pathogen

There is no evidence for the existence of strains of the pathogen, nor have tests been conducted comparing varietal resistance reactions of a set of standard varieties in different countries to see if there is variation in reaction.

Transmission

Much work has been undertaken on the transmission of chlorotic streak (BIRD *et al.*, 1958; EGAN, 1961 and 1963; STURGESS 1961, 1963 and 1965). The disease is not transmitted mechanically by harvesting or planting equipment. Mechanical transmission tests have been unsuccessful except in one case where a few transmissions were obtained by the injection of macerated root material into roots of healthy test plants. The disease is readily spread by infected planting material, in flood waters 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 9 months. Disease transmission is favoured by ratooning under wet conditions. Glasshouse trials showed that inoculation within 3 days of ratooning favoured disease development. Minimum time from inoculation to symptom expression is 12 days.

Host range

Research by EGAN (1965) showed that chlorotic streak infects various species of *Saccharum*. He found that clones of *S. robustum* and *S. spontaneum* were more susceptible than *S. edule; S. sinense* clones were the most resistant. Chlorotic streak is known to occur naturally in several grasses in at least four countries, and these can act as sources of inoculum for commercial crops. These grasses include *Pennisetum purpureum* (elephant grass), *Panicum maximum* (Guinea grass), *Erianthus arundinaceus, Arundo donax, Erianthus maximus* and *Paspalum paniculatum*. Other species into which the disease has been transmitted include *Brachiaria mutica, Erianthus procerus, Miscanthus floridulus, Miscanthus violaceus, Sorghum almum, S. bicolor, S. verticilliflorum*, and *Imperata cylindrica*. Chlorotic streak should be regarded as a disease which can infect a wide range of grasses, including sugarcane, under the right environmental conditions.

Epidemiology

The disease is favoured by soil temperatures between 25 and 30°C and particularly by poorly drained, flood-prone conditions. In such areas, the planting of healthy seed cane does not adequately control the disease because high levels of re-infection 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 disease, even in favoured areas. It may seem for a time that the disease is losing 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 favouring the disease. EGAN (1989) reported that yield losses were significant in various countries, including Australia, Guyana, Hawaii, Mauritius and Puerto Rico. Correlation of yields in healthy and diseased plots in the Herbert River district of Australia, by NIELSEN *et al.* (1986), suggested a 0.24% yield loss for every 1% stalks showing symptoms. 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. EGAN (1962) reported reduced germination of three varieties caused by the disease; reductions were 10, 16 and 22% in the varieties Q66, Q67 and Pindar respectively. 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, WANG and JIANG (1982) showed yield losses from 4 to 14% in six varieties over a plant and first ratoon crop,

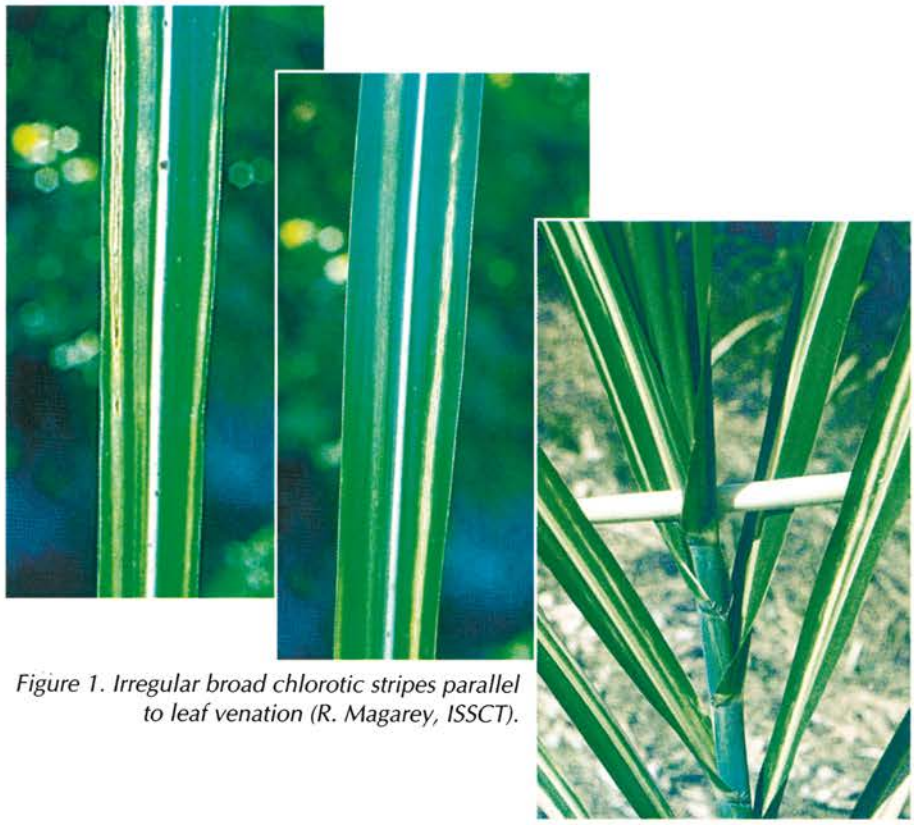


Figure 1. Irregular broad chlorotic stripes parallel to leaf venation (R. Magarey, ISSCT).

Figure 2. Healthy cane (left) and reduced growth of diseased cane (right) (R. Magarey).

with no effect on sugar content. Trials in Mauritius showed significant reductions in stalk numbers, stalk length and diameter, but not in Brix (WIEHE, 1955). Germination and yield loss effects (Figure 2), particularly in weight of harvestable biomass, are generally accepted as characteristics of the disease.

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 growth 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 the propagation of cane hot water treated at 50°C for 30 min or 52°C for 20 min. 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 chlorotic streak and the selection of suitable canes relies on field observations of varietal reaction. Routine trials are being developed in Australia and a diversity of resistance reactions is being noted in commercial canes.

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 absence of an assay for the disease, and the latency of symptoms under certain conditions, makes the selection of disease-free planting material uncertain especially under particular environmental conditions.

References

ABBOTT E.V., SASS J.E., 1945. Pathological histology of sugarcane affected with chlorotic streak. Journal of Agricultural Research 70: 201–207.

BIRD J., CIBES H., TIO M.A., 1958. Transmission of the causal agent of chlorotic streak disease of sugarcane through the roots of plants grown in nutrient solution. Puerto Rico, University of Puerto Rico, Agriculture Experiment Station Technical Paper No. 27, 17p.

EGAN B.T., 1961. Studies with chlorotic streak disease of sugarcane. 2. Transmission in nutrient gravel culture. Bureau of Sugar Experiment Stations Queensland Technical Communication No. 4.

EGAN B.T., 1962. Results from some chlorotic streak yield trials in north Queensland. Proceedings Queensland Society of Sugar Cane Technologists 29: 121–125.

EGAN B.T., 1963. Studies with chlorotic streak disease of sugarcane. 7. Some soil factors affecting streak production and disease transmission. Bureau of Sugar Experiment Stations Queensland Technical Communication No. 1.

EGAN B.T., 1965. Host range and possible sources of resistance to chlorotic streak disease. Proceedings International Society of Sugarcane Technologists Congress 12: 1055–1059.

EGAN B.T., 1989. Chlorotic streak. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 247–262. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

NIELSEN P.J., KAUPILLA N., ROACH B.T., 1986. An evaluation of the effect of chlorotic streak disease on sugarcane crops in the Herbert River district. Proceedings Australian Society of Sugarcane Technologists 8: 135–140.

STURGESS O.W., 1961. Studies with chlorotic streak disease of sugarcane. 3. Transmission by short exposure and incubation period of the disease. Bureau of Sugar Experiment Stations Queensland Technical Communication No. 5.

STURGESS O.W., 1962. Studies with chlorotic streak. 6. The influence of root temperature on production of leaf symptoms. Bureau of Sugar Experiment Stations Queensland Technical Communication No. 2.

STURGESS O.W., 1963. Studies with chlorotic streak disease of sugarcane. 8. Transmission by mechanical methods. Bureau of Sugar Experiment Stations Queensland Technical Communication No. 2.

STURGESS O.W., 1965. An understanding of chlorotic streak disease. Proceedings International Society of Sugarcane Technologists Congress 12: 1044–1054.

WANG C.S., JIANG D.K., 1982. Effect of chlorotic streak disease on the yield of sugarcane. Report of the Taiwan Sugar Research Institute 96: 33–44.

WIEHE P.O., 1955. Mauritius Sugar Industry Research Institute, Annual Report, 1954, p. 47–50.

Dwarf

Robert C. Magarey

Cause

Causal agent is unknown.

Geographical distribution

Australia (Queensland; BELL, 1932).

Symptoms

White stripes on the leaves and 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 centimetres long).

The margins of leaves may be affected by the disease; knife-cut type symptoms and papery and necrotic 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.

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; 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.

Figure 1. Leaves showing leaf striping and 'knife-cuts' along the margins (R. Magarey).

Figure 2. Stunted cane with stiff, fan-like leaf growth (R. Magarey).

Figure 3. Dwarf affected stools (BSES).

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 absence of transmission in 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 disease in susceptible varieties, when no symptoms have been observed in resistant canes, suggests the disease is present either in other hosts that are very persistent or in soil.

Epidemiology

The disease is favoured 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 levels can lead to significant crop loss within diseased crops. However, the limited incidence of the disease, both in number of crops affected and districts in which it occurs, mean that the disease is of only minor economic importance.

Control

The main forms of control have been roguing of diseased stools, the selection of disease-free planting material and the growth of resistant varieties. The disease is not eliminated by hot water, or other curative treatments.

References

BELL A.F., 1932. Dwarf disease of sugarcane. Bureau of Sugar Experiment Stations, Division of Pathology Bulletin No. 3(1), 12p.

STEINDL D.R.L., 1964. Dwarf. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 158–163. Amsterdam, The Netherlands, Elsevier Publishing Company.

Leaf galls (pseudo-Fiji)

Claude Ricaud

Cause

Probably toxicogenic effect of feeding by leafhopper (*Cicadulina mbila* Naude 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 and which bear great resemblance to stem galls (WISMER and HUGHES, 1964); those due to boron deficiency (MARTIN, 1938), and those which resemble Fiji disease galls and which have been observed in the above-mentioned countries. In the case of pseudo-Fiji, the galls are about 1 mm diameter on average but sometimes larger and develop along a vein which 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 millimetres. They are greenish in colour and slightly paler than the 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.

Diagnosis

Histological examination helps to differentiate the leaf galls described above from those due to Fiji disease. In the case of the virus infection the galls result from a proliferation of the phloem. Pseudo-Fiji galls are due to a proliferation of the epidermal cells or vascular bundle sheath cells and some adjoining mesophyll cells (EGAN *et al.*, 1989).

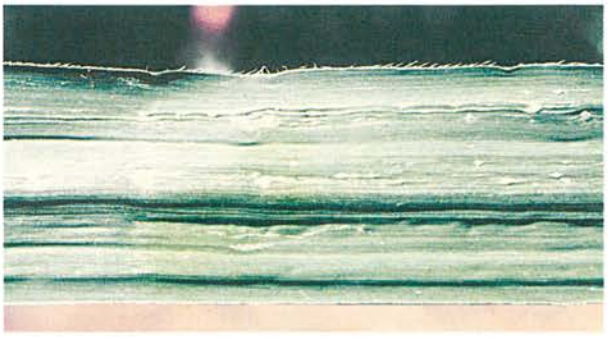


Figure 1. Small leaf galls (ISSCT).

Transmission

Leaf galls are not transmitted by sugarcane cuttings.

Host range

Leaf galls similar to those described above have also been seen on different members of the Poaceae such as *Melinis minutiflora, Phragmites mauritianus* and rice (*Oryza sativa*). Various *Cicadulina* species have been reported to cause somewhat similar leaf galls in maize (SHEFFIELD, 1969).

Epidemiology

Pseudo-Fiji galls tend to disappear on new leaves developing in 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.

References

EGAN B.T., RYAN C.C., FRANCKI R.I.B., 1989. Fiji disease. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 263–287. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., 1938. Sugar Cane Diseases in Hawaii. Honolulu, Hawaii, Hawaiian Planters' Association Experiment Station, 295p.

SHEFFIELD F.M.L., 1969. The cause of leaf galls. Sugarcane Pathologists' Newsletter 3: 25.

WISMER C.A., HUGHES C.G., 1964. Miscellaneous non-transmissible effects. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 297–316. Amsterdam, The Netherlands, Elsevier Publishing Company.

Ramu streak

Robert C. Magarey and Sidney Suma

Cause

Causal agent is unknown.

Geographical distribution

The disease has been observed in Papua New Guinea, and on Saibai Island (Australian territory) in the Torres Strait.

Symptoms

Symptoms consist of yellow-green leaf streaks with some 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.

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*), *S. edule* and interspecific hybrids.

Figure 1. Leaf streaks along the margin of an infected leaf (R. Magarey).

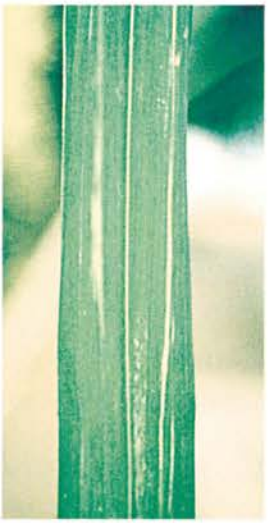


Figure 2. Irregular chlorotic leaf streaks without necrosis (R. Magarey).

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. In 1994 some crops with abundant symptom expression were low yielding, but it is uncertain how much the condition contributed to these low yields.

Control

No specific control measures have been implemented.

References

MAGAREY R.C., SUMA S., EGAN B.T., 1995. New sugarcane diseases observed in commercial cane at Gusap, Papua New Guinea. Proceedings International Society of Sugarcane Technologists Congress 22: 472–476.

SUMA S., PAIS E., 1996. Major diseases affecting sugarcane production on Ramu Sugar Estate, Papua New Guinea. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28–30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 30–33.

Yellow leaf syndrome

Ben E. Lockhart and C. Pieter R. Cronjé

Cause

Yellow leaf syndrome (YLS) of sugarcane has been associated with several biotic and abiotic causes during the past four decades. This condition has probably been known earlier under different names, such as 'yellow wilt' in Africa (RICAUD, 1968; SIDDIQI, 1969; ROGERS, 1970) and 'autumn decline' in Brazil (HUGHES, 1964). No pathogens were associated with these conditions, and no further reports were made on the occurrence of similar symptoms, until an apparent recurrence in the late 1980s (SCHENK and HU, 1991; COMSTOCK *et al.*, 1994). Two separate pathogens have been associated with the recent recurrence of yellow leaf syndrome. One is *Sugarcane yellow leaf luteovirus* (ScYLV) (LOCKHART *et al.*, 1996; VEGA *et al.*, 1997; SCAGLIUSI and LOCKHART, 2000) (Figure 1), and the other is Sugarcane yellows phytoplasma (ScYP) (CRONJÉ *et al.*, 1998) (Figure 2). However, several reports indicate that similar symptoms can also be induced by insect feeding, physiological stress, climate, varietal reaction to stress and other abiotic factors (BAILEY *et al.*, 1996; MATSUOKA and MENEGHIN, 1999).

Geographical distribution

Argentina, Australia (Queensland), Barbados, Brazil, Colombia, Cuba, Dominican Republic, El Salvador, Guadeloupe, Guatemala, Hawaii, India, Iran, Jamaica, Kenya, Malawi, Martinique, Mauritius, Mexico, Morocco, Mozambique, Nicaragua, Papua New Guinea, Peru, Réunion, Senegal, South Africa, Swaziland, Thailand, Uganda, USA (Florida, Louisiana), Venezuela, Zambia, Zimbabwe.

Symptoms

The characteristic diagnostic symptom of YLS is yellowing of the leaf midrib, which occurs in all forms of the disorder (Figure 3). The pattern of midrib yellowing and the nature of associated symptoms are variable, and often depend on plant cultivar and environmental conditions. Symptoms include shortening of terminal internodes, yellowing of terminal leaves, and sucrose accumulation in midribs. In some varieties, the lower surface of the midrib becomes yellow

in older leaves. The upper surface may remain normal (white or greenish white) or it may turn yellow, pink or reddish. This discoloration of the midrib often occurs while the lamina is still green. In some varieties yellowing of leaves can affect all leaves. The yellow discoloration may also spread laterally from the midrib while necrosis begins from the leaf tip and progresses towards the leaf base. It is reported from Africa (Cronjé *et al.*, 1998) that the symptoms occur from leaves three to five, taking the first visible dewlap as leaf one. The symptoms in Africa are usually temporary and fade with the onset of better growing conditions.

Diagnosis

Because the various forms of YLS have similar symptomatology, visual symptoms are not reliable for identifying the cause of the disorder. Environmental factors also appear to have an important effect on symptom expression, further compromising the reliability of disease detection by visual observation. ScYLV can be identified reliably by DAS-ELISA (Scagliusi and Lockhart, 2000) and dot immunoblot assay (DIBA) (Comstock *et al.*, 1998) using antibodies prepared against a mixture of virus isolates from Brazil, Florida and Hawaii (Scagliusi and Lockhart, 2000). Reliable detection of ScYLV by RT-PCR has also been described (Comstock *et al.*, 1998). Detection of ScYP is done by PCR of the 16S rDNA region of the phytoplasma genome, and restriction fragment length polymorphism (RFLP) of the generated products. Further classification and confirmation of identity of the phytoplasmas are achieved by amplifying and sequencing the intergenic spacer region between the 16 and 23S rDNA (Cronjé *et al.*, 1998).

Strains of the pathogens

There is no evidence of differences between isolates of ScYLV from different geographical locations. The virus has most likely been moved about on infected planting material rather than originating locally in alternative host plants. The types of phytoplasma found in YLS-symptomatic canes are largely determined by geographical origin of the samples. Plants are usually infected with the predominant phytoplasma group present in a given location. For instance the Western X type has been found in Hawaii, while the ScWL type has been found in America, and the PPWB type in Jamaica.

Transmission

An essential distinction between the various forms of YLS is that the pathogen-induced syndromes (i.e. those associated with infection by ScYLV and ScYP) are transmissible by vegetative propagation while similar disorders induced by physiological stress or insect feeding are not. Long-distance spread of ScYLV has most probably occurred via infected planting material. ScYLV is also trans-

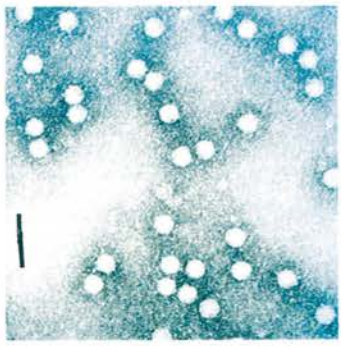


Figure 1. Virions of ScYLV observed by transmission electron microscopy (scale bar = 100 nm) (B.E. Lockhart).

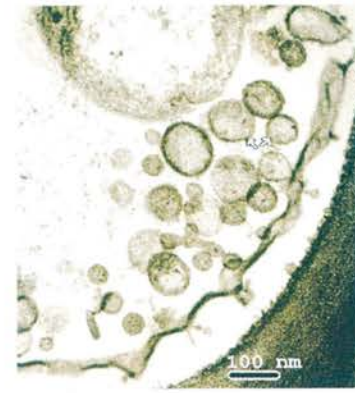


Figure 2. Pleomorphic membrane-bound bodies (possibly phytoplasmas) observed by transmission electron microscopy in the phloem of leaves with YLS symptoms (P. Cronjé).

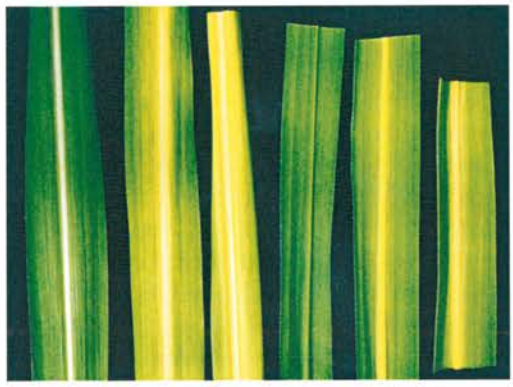


Figure 3. Characteristic midrib yellowing (healthy leaves first and fourth from left) (B.E. Lockhart).

mitted by two aphid species, *Melanaphis sacchari* and *Rhopalosiphum maidis*, which colonize sugarcane. A third aphid species which colonizes sugarcane, *Sipha flava*, is not a vector of ScYLV. Interestingly, *S. flava* is a vector of *Sugarcane mosaic potyvirus* (ScMV) (Blackman and Eastop, 1984), which *M. sacchari* is unable to transmit (Blackman and Eastop, 1984; Noone *et al.*, 1994). Phytoplasma 16S rDNA associated with YLS in cane has been detected in the sugarcane planthopper *Perkinsiella saccharacida*, suggesting that this insect may be a vector of ScYP.

Host range

Sugarcane is the only known host of ScYLV. No wild or cultivated alternative hosts have been identified, and the virus was not transmitted by *R. maidis* to sweet corn, sorghum or Johnson grass. A previous report of ScYLV infection of sweet corn (Lockhart *et al.*, 1996) was based on dsRNA analysis and was not subsequently confirmed by DAS-ELISA and ISEM (Scagliusi and Lockhart, 2000). The phytoplasmas associated with ScYP-YLS have been found in various other host plants, including coconuts and *Cynodon dactylon* (Bermuda grass) growing on the verges of cane fields.

Epidemiology

Failure to identify alternative reservoirs of ScYLV and its aphid vectors suggest that virus spread occurs mainly by vegetative propagation and by aphid transmission from infected to healthy sugarcane. Based on these assumptions, the epidemiology of ScYLV-induced YLS is determined by the frequency of virus in planting material and by the population dynamics of aphid vector species.

Drought, waterlogging and cool winters favour symptom expression of ScYP-YLS. In Africa the symptoms seem to be transient in most varieties. Sudden changes in growing conditions and weather also induce symptom expression.

Economic importance

The most severe yield reductions have been reported from Brazil, where 40–60% losses of recoverable sugar were recorded in the widely grown variety SP71-6163 infected with ScYLV. These initial figures have been revised, and a 20% loss is reported (W. Burnquist, personal communication). Varieties such as SP71-6163, RB72-454, CP65-357, H73-6110 and H65-7052 appear to be highly susceptible to infection by ScYLV. Initial yield loss experiments from elsewhere have shown reductions in the order of 2–20% depending on variety. YLS can also affect processing quality. In South Africa and Hawaii YLS-affected canes have shown a significant increase in the content of complex polysaccharides (gums) which interfere with sugar extraction.

Control

Available evidence suggests that the viral form of YLS spreads within the sugarcane crop by clonal propagation and aphid vectors rather than from external sources. On this basis, effective control of this form of YLS can be achieved by use of virus-free planting material, now that several effective indexing procedures (see above) are available. It also suggests that ScYLV-infected sugarcane varieties which are valued for agronomic and processing traits can be freed of virus by thermotherapy and/or apical meristem culture and re-deployed with a low risk of reinfection provided that infected canes are not grown in close proximity. There is also evidence of important levels of resistance or tolerance to ScYLV in some sugarcane varieties. Production of ScYLV-resistant or ScYLV-tolerant varieties by conventional breeding or transgene introgression, therefore, represents a feasible medium-term control strategy.

A control strategy for ScYP-induced YLS can be developed once the essential features of its epidemiology (vector species and host range) have been clearly established.

References

BAILEY R.A., BECHET G.R., CRONJÉ C.P.R., 1996. Notes on the occurrence of yellow leaf syndrome of sugarcane in southern Africa. Proceedings South African Sugar Technologists Association 70: 3–6.

BLACKMAN R.L., EASTOP V.F., 1984. Aphids on the World's Crops. Chichester, UK, John Wiley and Sons, 466p.

COMSTOCK J.C., IRVINE J.E., MILLER J.D., 1994. Yellow leaf syndrome appears on the United States Mainland. Sugar Journal, March 1994: 33–35.

COMSTOCK J.C., IREY M.S., LOCKHART B.E.L., WANG Z.K., 1998. Incidence of yellow leaf syndrome in CP cultivars based on polymerase chain reaction and serological techniques. Sugar Cane 4: 21–28.

CRONJÉ C.P.R., TYMON A.M., JONES P., BAILEY R.A., 1998. Association of a phytoplasma with a yellow leaf syndrome of sugarcane in Africa. Annals of Applied Biology 133: 177–186.

HUGHES C.G., 1964. Genetical effects. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 267–296. Amsterdam, The Netherlands, Elsevier Publishing Company.

LOCKHART B.E.L., IREY M.J., COMSTOCK J.C., 1996. Sugarcane bacilliform virus, sugarcane mild mosaic virus and sugarcane yellow leaf syndrome. *In*: Sugarcane Germplasm Conservation and Exchange. Report of an international workshop held in Brisbane, Queensland, Australia, 28-30 June 1995. B.J. Croft, C.M. Piggin, E.S. Wallis and D.M. Hogarth (Eds). ACIAR Proceedings No. 67, p. 108–112.

MATSUOKA S., MENEGHIN S.P., 1999. Yellow leaf syndrome and alleged pathogens: A casual not a causal relationship. Proceedings International Society of Sugar Cane Technologists Congress 23: 382–389.

NOONE D.F., SRINSK S., TEAKLE D.S., ALLSOPP P.G., TAYLOR P.W.J., 1994. Ability to transmit sugarcane mosaic virus and seasonal phenology of some aphid species (Hemiptera: Aphididae) in the Isis and Bundaberg districts of Queensland. Journal of the Australian Entomological Society 33: 27–30.

RICAUD C., 1968. Yellow wilt of sugarcane in eastern Africa. Sugarcane Pathologists' Newsletter 1: 45–48.

ROGERS P.F., 1970. Proceedings of the special meeting on Yellow wilt condition of sugarcane. East African Agriculture and Forestry Research Organization, Nairobi, Kenya.

SCAGLIUSI S.M., LOCKHART B.E.L., 2000. Transmission, characterization, and serology of a luteovirus associated with yellow leaf syndrome of sugarcane. Phytopathology 90: 120–124.

SCHENK S., HU J.S., 1991. Update on the cause of sugarcane yellow leaf syndrome. Proceedings Hawaii Sugar Technologists Association Conference 50: 45–46.

SIDDIQI M.A., 1969. Yellow wilt in Malawi. Sugarcane Pathologist's Newsletter 3: 13.

VEGA J., SCAGLIUSI S.M.M., ULIAN E.C., 1997. Sugarcane yellow leaf disease in Brazil: Evidence of association with a luteovirus. Plant Disease 81: 21–26.

Minor diseases of uncertain etiology

Philippe Rott and Jack C. Comstock

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 and C.A. Wismer (Eds), p. 180–181. Amsterdam, The Netherlands, Elsevier Publishing Company.

Sereh

Cause: virus (putative).

Geographical distribution: Indonesia (Java).

Symptoms: small clusters of shoots on affected stalks of variable height, stalks with abundant adventitious roots at nodes, severely affected plants are entirely made up of shoots (Figure 2).

Reference

RANDS R.D., ABBOTT E.V., 1964. Sereh. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 182–189. Amsterdam, The Netherlands, Elsevier Publishing Company.

Spike

Cause: virus (putative).

Geographical distribution: India.

Symptoms: shortened leaves and internodes have a spike-like appearance on normal stalks, leaves dark green with blue tinge.

Reference

EDITORIAL COMMITTEE, 1964. Spike. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 190–191. Amsterdam, The Netherlands, Elsevier Publishing Company.

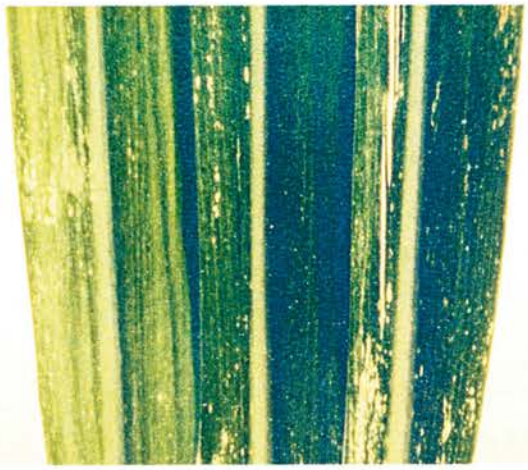


Figure 1. Sembur: leaves with elongate whitish to yellow spots (Irawan).

Figure 2. Sereh: small clusters of shoots on diseased stalk (Irawan).

Nematodes

Graham Stirling and Brenden Blair

More than 275 nematode species in 48 genera have been recorded from the roots and rhizosphere of sugarcane (SPAULL and CADET, 1991). Most cane fields are infested with at least five species. Thus, diseases caused by nematodes always involve a complex of species with different feeding habits and various degrees of pathogenicity. The most important nematodes can be categorized as follows:

– widespread and highly pathogenic: lesion nematodes (various species, but most commonly *Pratylenchus zeae* and *P. brachyurus*) (Figure 1);

– common and highly pathogenic, particularly on sandy soils: root-knot nematodes (commonly *Meloidogyne javanica* and *M. incognita*), stubby root nematodes (commonly *Paratrichodorus minor*), dagger nematodes (*Xiphinema* spp.) and needle nematodes (*Paralongidorus* spp.);

– widespread and moderately or weakly pathogenic: stunt nematodes (*Tylenchorhynchus* spp., with *T. annulatus* the most widely distributed), spiral nematodes (*Helicotylenchus* spp., *Scutellonema* spp. and *Rotylenchus* spp., with *H. dihystera* one of the most common species), lance nematodes (*Hoplolaimus* spp.), ring nematodes (*Criconemella* spp., *Criconema* spp., *Hemicriconemoides* spp. and *Ogma* spp.), sheath nematodes (*Hemicycliophora* spp.) and reniform nematode (*Rotylenchulus parvus*).

Geographical distribution

Plant-parasitic nematodes occur in all sugarcane growing regions, with the genera recorded in 24 countries being listed by SPAULL and CADET (1991). However, much of the published information is based on limited surveys and the taxonomy at species level is often inadequate. BLAIR *et al.* (1999a and 1999b) provide an example of the detailed survey data required to define nematode distribution in a specific region.

Symptoms

Damage by nematodes retards the development of shoots and reduces tillering. Because the canopy is slow to develop, nematode-infested cane tends to have an open appearance. During periods when soil moisture is limiting, leaves may wilt and curl so that the plant has a spikey appearance. Severe infestations may reduce yield by 20–50% due to a reduction in the number and length of stalks. Nematodes also have subtle effects that are usually not recognized because non-infested crops are not available for comparison. Such effects can only be observed by applying a nematicide at a rate sufficient to control nematodes and comparing growth in the nematicide-treated and untreated areas (Figure 2). Commonly, the treated crop will be taller and denser and will produce higher yields.

Root symptoms vary, depending on the nematode species present. Root-knot nematode produces the most distinctive symptoms, with swellings and galls occurring on sett roots and young shoot roots (Figure 3). Because galls often occur at root tips, primary roots cease to elongate and root length can be substantially reduced. Lesion nematode is a migratory endoparasite that causes reddish-purple lesions on newly infected roots. These lesions become necrotic and turn purplish-black, causing the root system to darken in colour. As lesions expand, roots are girdled, so that fine roots are destroyed and root mass is reduced (Figure 4). Ectoparasitic nematodes (e.g. *Paratrichodorus, Xiphinema* and *Paralongidorus*) feed on root tips, causing swelling and malformation of root tips and stunting of roots. Lateral roots produced behind the damaged root tip are also stunted, so that infested root systems may have a 'stubby' appearance.

Diagnosis

Root symptoms in the field are rarely specific enough to definitively diagnose a nematode problem. Galling caused by root-knot nematode can be readily seen on roots of young plant cane, but because galls are small and discrete, they are not easily detected on older plants. Symptoms of other nematodes are relatively non-specific, so that lack of fine roots, swelling of root tips, proliferation of stunted lateral roots, root discoloration and presence of lesions may indicate a nematode problem. However, the poor root growth that is typical of nematode damage can also be due to fungal pathogens, root-feeding arthropods, nutrient deficiencies (e.g. phosphorus) or toxicities (e.g. aluminium), soil compaction and poor aeration.

To diagnose a nematode problem, soil and root samples must be collected and nematodes extracted, identified and quantified. To ensure samples are repre-

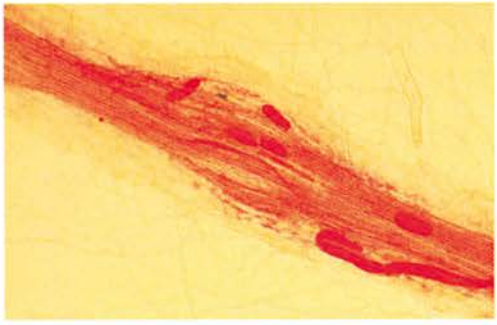


*Figure 1. Lesion nematodes (*Pratylenchus zeae*) and their eggs (stained red with acid fuchsin) in a sugarcane root (B. Blair).*

Figure 2. Response of a plant crop to nematicide treatment (right) in a sandy loam soil (B. Blair).

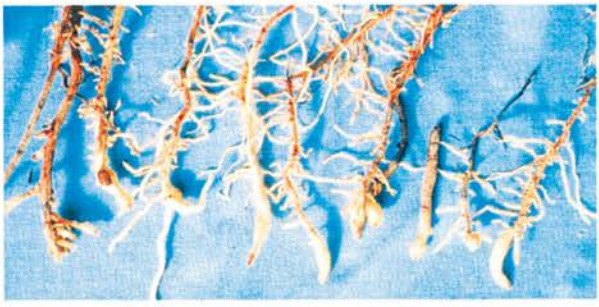


*Figure 3. Terminal galling caused by root-knot nematode (*Meloidogyne *sp.) (B. Blair).*

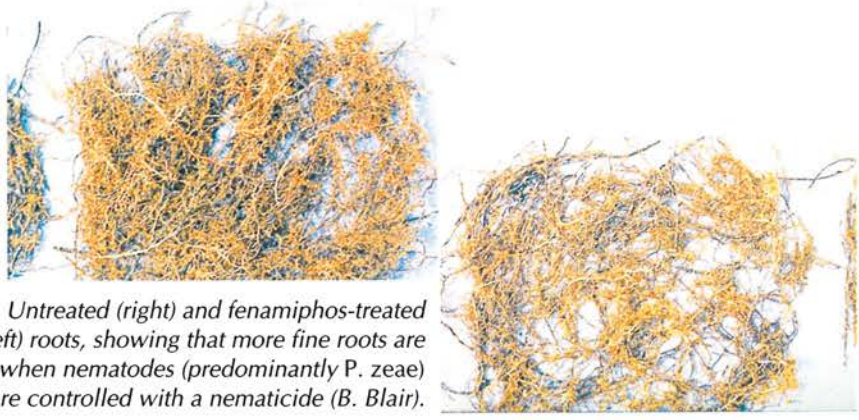


Figure 4. Untreated (right) and fenamiphos-treated (left) roots, showing that more fine roots are present when nematodes (predominantly P. zeae*) are controlled with a nematicide (B. Blair).*

sentative of the sampling area, soil and roots are taken at depths of 5–25 cm from at least ten sampling points within or near rows. Because efficiencies of different extraction techniques vary, correction factors may need to be applied when comparing nematode counts with those provided in published studies. Sampling time must also be taken into account, as nematode population densities are affected by the dynamics of the cane root system. Damage thresholds for nematodes on sugarcane are not well defined, but Table 1 gives some indication of the likely hazard from various densities of root-knot and lesion nematodes.

Table 1. Estimates of the likely hazard to sugarcane of various nematode population densities.

Nematode	Sampling time	Hazard index*			Units**
		Low	Medium	High	
Lesion (*P. zeae*)	Pre-plant	<100	100–300	>300	Nematodes/200 g soil
	Mid-season	<250	250–2000	>2000	Nematodes/200 g soil or /g dry weight of root
Root-knot (*M. javanica*)	Pre-plant	<100	100–200	>200	Nematodes/200 g soil
	Mid-season	<200	200–500	>500	Nematodes/200 g soil or /g dry weight of root

* *Low*: little chance of a reduction in yield due to nematodes. *Medium*: 5–20% losses in yield may occur if environmental conditions are favourable to nematodes or unfavourable to the crop. *High*: yield reductions >20% are possible, but the extent of losses will depend on soil type and the standard of crop management.

** Number of nematodes extracted, assuming an extraction efficiency of 50%.

Nematode identification

Because taxonomic expertise is limited in many sugarcane growing regions, some records of nematodes associated with sugarcane may be incorrect. Unfortunately, most identifications cannot be checked as voucher specimens are not available. Uncertainty about the species of *Meloidogyne* that occur on sugarcane is a typical example of these taxonomic problems. There are numerous records of unidentified *Meloidogyne* and species identification has been based mainly on perineal patterns, which can be unreliable. Recent work in Australia using molecular techniques showed that *M. javanica* was the predominant species (about 75% of samples), with *M. incognita, M. arenaria* and *M. hispanica* also occurring (Blair *et al.*, 1999a and 1999b). Taxonomic information of this nature will be important when attempts are made to identify sugarcane germplasm with nematode resistance. A knowledge of intraspecific variation will also be needed, but to date, no such studies have been attempted.

Host range

Most of the nematodes associated with sugarcane have a wide host range. In some cases (e.g. *Pratylenchus zeae, Rotylenchulus parvus* and *Tylenchorhynchus annulatus*), the host range is largely restricted to grasses. Many other nematodes found on sugarcane (e.g. *Meloidogyne, Paratrichodorus* and *Helicotylenchus* spp.) have a broad host range consisting of hundreds of plant species.

Transmission

Since most of the world's sugarcane is grown in monoculture, newly planted crops will be attacked by nematodes carried over from the previous crop. Nematode populations decline if soil is fallowed for more than about 3 months, but a long period of bare fallow (2–3 years) is required to reduce nematode populations to negligible levels (Pankhurst *et al.*, 1999). Weeds or an alternative crop will often maintain nematode populations, but their effect depends on the species of nematode and the host status of the plant.

Epidemiology

The sugarcane root system is never static. After the crop is planted, sett roots are replaced by shoot roots, which then grow rapidly until the crop approaches maturity. After harvest, a new root system produced by the ratoon crop replaces the roots of the plant crop. This pattern of root development has a significant impact on populations of plant-parasitic nematodes. In general, nematode population densities are lowest at planting, reach a maximum 6–10 months after planting and then decline as harvest approaches (Spaull and Cadet, 1991). A similar cyclic pattern occurs in ratoon crops, but nematode populations are often lower than in plant crops (Blair *et al.*, 1999a).

Economic importance

Nematodes are a major factor limiting cane production in light-textured soils. Results of nematicide trials in soils with a clay content of about 5% or less (summarized by Spaull and Cadet, 1991) showed that nematicide treatment increased yields by 23–81% in the plant crop and by 8–21% in the first ratoon. In sandy loam soils with a clay content of about 10%, yield responses were

11–32% (SPAULL, 1995). However, sandy soils constitute only a small proportion of the area under sugarcane, which means that nematodes are perceived as unimportant in most of the world's sugarcane. Recent observations in Australia challenge that perception. When nematicides are applied to clay loam and clay soils in a manner that suppresses nematode populations for the whole growing season, root health improves (primarily because feeder root density increases), and yield responses of 5–20% are consistently obtained (STIRLING *et al.*, 1999). This suggests that nematodes (particularly *P. zeae*) are having insidious and widespread effects that are generally not recognized within the sugar industry.

Control

Organophosphate and carbamate nematicides can be used to reduce nematode densities early in the season, when crops are most vulnerable to nematode damage. However, the high cost of nematicides usually limits their use to sandy soils, where species of *Pratylenchus, Meloidogyne, Paratrichodorus* and *Xiphinema* often cause heavy losses. Concerns about the high mammalian toxicity of nematicides and their capacity to contaminate groundwater are other limitations to chemical control. Cultivars with resistance to certain species of *Meloidogyne* can be used in situations where these species are the key pest (SPAULL and CADET, 1991). However, resistance cannot be used as a control strategy in most of the sugar industry, as sources of resistance to other important nematodes have not been identified.

The insidious effects of nematodes (particularly *Pratylenchus*) and their role in yield decline is an emerging issue (GARSIDE *et al.*, 1999; PANKHURST *et al.*, 1999) likely to be of universal importance. Since nematodes are only one component of a causal complex that probably involves numerous physical, chemical and biological factors, such problems cannot be solved by concentrating control measures only on nematodes. Sustainable soil management systems that reduce the effects of yield decline are still to be developed, but will possibly involve crop rotation, minimum tillage, trash retention, addition of organic matter and use of varieties with resistance and/or tolerance to nematodes and other soil-borne pathogens.

References

BLAIR B.L., STIRLING G.R., WHITTLE P.J.L., 1999a. Distribution of pest nematodes on sugarcane in south Queensland and relationship to soil texture, cultivar, crop age and region. Australian Journal of Experimental Agriculture 39: 43–49.

Blair B.L., Stirling G.R., Pattemore J.A., Whittle P.J.L., 1999b. Occurrence of pest nematodes in Burdekin and central Queensland sugarcane fields. Proceedings Australian Society of Sugar Cane Technologists Conference 21: 227–233.

Garside A.L., Bell M.J., Cunningham G., Berthelsen J., Halpin N., 1999. Rotation and fumigation effects on the growth and yield of sugarcane. Proceedings Australian Society of Sugar Cane Technologists Conference 21: 69–78.

Pankhurst C.E., Magarey R.C., Stirling G.R., Holt J.A., Brown J.D., 1999. Rotation-induced changes in soil biological properties and their effect on yield decline in sugarcane. Proceedings Australian Society of Sugar Cane Technologists Conference 21: 79–86.

Spaull V.W., 1995. A revised estimate of crop loss in sugarcane caused by nematodes. Proceedings South African Sugar Technologists Association Conference 69: 28–34.

Spaull V.W., Cadet P., 1991. Nematodè parasites of sugarcane. *In*: Plant Parasitic Nematodes in Subtropical and Tropical Agriculture. M. Luc, R.A. Sikora and J. Bridge (Eds), p. 461–491. Wallingford, UK, CAB International.

Stirling G.R., Blair B.L., Whittle P.J.L., Garside A.L., 1999. Lesion nematode (*Pratylenchus zeae*) is a component of the yield decline complex of sugarcane. Proceedings Australasian Soilborne Disease Symposium 1: 15–16.

Nutritional, environmental and chemical disorders

Barry J. Croft

Many disease symptoms can be mimicked by nutritional disorders (Figures 1, 2, 3, 4 and 5), environmental effects (Figures 6, 7 and 8) and phytotoxic chemicals such as herbicides (Figures 9 and 10). For example: the common herbicide paraquat can cause a lesion similar to ring spot disease when fine droplets drift on to sugarcane leaves (Figure 10); magnesium deficiency causes an orange to red-brown spot (orange freckle) on leaves similar to a range of fungal leaf spots; frost can cause a mosaic pattern not unlike *Sugarcane mosaic virus* symptoms, and lightening can cause shredding of the leaves, death of the spindle and purple discoloration of the leaf blade. 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 MARTIN and EVANS (1964), MARTIN *et al.* (1964), ANDERSON and BOWEN (1991) and GRASCHO and TAHA (1972).

Figure 1. Young shoots with iron deficiency (ISSCT).

References

ANDERSON D.L., BOWEN J.E., 1991. Sugarcane Nutrition. Atlanta, USA, Potash and Phosphate Institute, 39p.

GRASCHO G.J., TAHA F.A., 1972. Nutritional Deficiency Symptoms of Sugarcane. Gainesville, USA, University of Florida, 16p.

MARTIN J.P., EVANS H., 1964. Nutritional deficiencies and toxicities. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 197–236. Amsterdam, The Netherlands, Elsevier Publishing Company.

MARTIN J.P., WISMER C.A., HUGHES C.G., 1964. Environmental effects. *In*: Sugar-Cane Diseases of the World, Vol. 2. C.G. Hughes, E.V. Abbott and C.A. Wismer (Eds), p. 237–264. Amsterdam, The Netherlands, Elsevier Publishing Company.

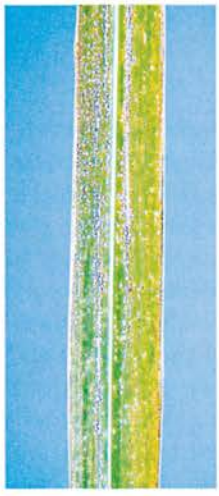


Figure 2. Leaf showing silicon deficiency symptoms: greyish-white freckling present only on the upper leaf surface (MSIRI).

Figure 3. Leaves with molybdenum deficiency symptoms (J.H. Meyer).

Figure 4. Leaves with copper deficiency symptoms (J.H. Meyer).

Figure 5. Leaves with boron deficiency symptoms (R.A. Bailey).

Figure 6. Cane field affected by freeze damage (H. Koike).

Figure 7. Leaves with banded chlorosis (cold injury) (R.A. Bailey).

Figure 8. Cane field affected by lightning damage (ISSCT).

Figure 9. Herbicide (2,4–D) damage (S. Matsuoka).

Figure 10. Leaves with herbicide (paraquat) injury (R.A. Bailey).

Procedures for the safe movement of sugarcane germplasm

Roger A. Bailey, Jack C. Comstock, Barry J. Croft, A. Salem Saumtally and Philippe Rott

Many important diseases of sugarcane can be transmitted in vegetative propagation material. These include all viral and phytoplasmal diseases, most bacterial diseases and several diseases caused by fungal pathogens. Sugarcane germplasm is still mainly exchanged between countries in the form of stalk pieces (setts). Therefore, unless precautions are taken, the exchange of varieties can provide 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 practised for many years. Until recently security was mainly based on the recognition of disease symptoms in glasshouse-grown plants. Before the causal agents of certain diseases had been identified and before modern methods of diagnosis had been developed, the exchange of germplasm was probably instrumental in the world-wide spread of various pathogens, particularly those that cause non-specific symptoms, such as ratoon stunting disease (RSD), or can remain latent, such as leaf scald. There is good evidence that the organisms that are associated with yellow leaf syndrome (YLS) have recently been spread in this manner. The hazards presented by germplasm exchange are now well appreciated and most countries importing germplasm apply strict quarantine procedures (CROFT *et al.*, 1996; GILLASPIE, 1989).

Because of the risk of transmitting diseases by the exchange of germplasm, the avoidance of material from high-risk areas, where the health standard of the material is uncertain, can be an important security measure for the importer.

An important aspect of quarantine security is thermotherapy, applied mainly as the treatment of setts in hot water. Appropriate hot water treatments (HWT) eliminate all seed cane-borne fungal pathogens and certain bacterial pathogens. Additional security is provided by molecular and serological tests for an increasing number of pathogens. Currently these include the causal agents of gumming, leaf scald, ratoon stunting (RSD), Fiji disease, mosaic, streak and yellow leaf syndrome (YLS). The exchange of diagnostic protocols and materials among organizations that exchange germplasm is encouraged. Newer more accurate diagnostic procedures must be continually developed, evaluated and adapted to improve the quarantine process.

Because diagnostic tests are not available for all pathogens, and because not all importers are equipped for modern diagnosis, the basis of quarantine security remains frequent inspections by experienced diagnosticians.

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 specific diseases.

Exchange of setts – actions by the exporter

Although coping with the risks associated with variety exchange is mainly the responsibility of the importer, the exporter should take precautions to ensure, as far as possible, that the material is disease and pest-free.

Setts for export should be taken from propagation plots that were established with seed cane that was subjected to HWT and inspected during growth and found free from symptoms of systemic diseases. Where possible the source plots should be in areas not subjected to hazardous diseases, and the source plants should be indexed for pathogens.

At least two 3-budded setts per variety should be carefully stripped of all trash, washed clean, treated in water at 50°C for 30 min (short HWT), and dipped in a general fungicide and in a general insecticide.

The names of the clones should be 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 packing 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 and given a short HWT.

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 other sugarcane growing area. The French international sugarcane quarantine is located in the South of France where no sugarcane is cultivated (ROTT *et al.*, 1998). 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 SASEX 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 2–3 h at 50°C), 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. The possible duration of the 50°C treatment should, therefore, be tested before application to 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 diseases should be applied.

Provided the plants remain free of disease symptoms and 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.

Pre-export quarantine

A number of organizations have bilateral agreements regarding germplasm exchange. The main feature of these agreements is that the exporter agrees to process material through a quarantine process, which includes diagnostic tests, before dispatch. The main 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, the quarantine unit of CIRAD in Montpellier, France, can be cited. This quarantine unit is located within an international research centre on tropical agronomy and exports varieties to West African countries that do not have the laboratory facilities to test sugarcane for diseases (ROTT *et al.*, 1998).

Tissue cultured plant 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.

Setts are prepared from suitable source plants (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. These are cultured on agar medium under an appropriate light, nutrient and temperature regime.

When the buds have started to produce roots, they are excised and transferred to agar growth medium in transparent containers for further growth.

The young plantlets can be exported, growing on agar medium, after tillers have started to develop and when of a suitable size (Figure 1).

On receipt, the plantlets should be planted into sterilized potting medium in a containment facility and grown for at least one cycle, during which diagnostic tests for pathogens should be applied.

Once the plants are determined to be free from pathogens, setts are prepared for further propagation outside quarantine.

Feed-back of information

To encourage the improvement of standards, importing organizations should routinely inform exporters if any diseases are detected in exchanged material.

References

BAILEY R., BECHET G.R., 1988. Recent improvements in quarantine procedures for sugarcane in South Africa. Proceedings South African Sugar Technologists' Association Annual Congress 62: 190–194.

CROFT B.J., PIGGIN C.M., WALLIS E.S., HOGARTH D.M. (Eds), 1996. Sugarcane germplasm conservation and exchange. ACIAR Proceedings No. 67, 134p.

FRISON E.A., PUTTER C.A.J. (Eds), 1993. FAO/IBPGR Technical guidelines for the safe movement of sugarcane germplasm. Rome, Italy, Food and Agriculture Organization of the United Nations and International Board for Plant Genetic Resources, 44p.

GILLASPIE A.G. JR, 1989. Sugarcane quarantine. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G Hughes (Eds), p. 333–339. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

ROTT P., BOUSQUET J.F., CHATENET M., DARROUSSAT M.J., MULLER M., RIPOLLES M., 1998. A quarantine unit in Montpellier, France, for safe movement of sugarcane germplasm. *In*: Offered papers abstracts, Vol. 3, 7th International Congress of Plant Pathology, Edinburgh, Scotland, 9–16 August 1998. Abstract 4.6.1. Edinburgh, International Society for Plant Pathology.

Figure 1. Tissue cultured sugarcane plantlets (P. Rott).

Common names of sugarcane diseases and their causal agent(s)

Philippe Rott, Roger A. Bailey, Jack C. Comstock, Barry J. Croft and A. Salem Saumtally

Alternaria leaf spot	*Alternaria alternata*
Apex rot	unknown
Arrow rot	*Fusarium* spp.
Australian basal stem, root and sheath rot	unidentified fungus (basidiomycete)
Bacterial mottle	*Pectobacterium chrysanthemi*
Bacterial sun spot	*Pseudomonas* sp.
Baker's leaf spot	*Bakerophoma sacchari*
Banded sclerotial disease	*Thanatephorus sasakii, T. cucumeris*
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	*Ceratocystis adiposa*
Black spot	*Cercospora acerosum*
Black stem rot	*Selenophoma* sp.
Black stripe	*Pseudocercospora atrofiliformis*

Brown rot	*Corticium* sp.
Brown spot	*Cercospora longipes*
Brown stripe	*Bipolaris stenospila*
Chlorotic streak	unknown
Collar rot	*Hendersonia sacchari*
Common rust	*Puccinia melanocephala*
Covered smut	*Sphacelotheca macrospora*
Culm and midrib rot	*Papularia vinosa*
Culmicolous smut (smut)	*Ustilago scitaminea*
Diplodia rot	*Diplodia* sp.
Downy mildew	*Peronosclerospora sacchari, P. philippinensis, P. spontanea*
Dry rot	*Physalospora rhodina*
Dry top rot	*Ligniera vasculorum*
Dwarf	unknown
Ergot	*Claviceps purpurea, C. pusilla*
Eye spot	*Bipolaris sacchari*
False floral smut	*Claviceps* sp.
False red stripe	*Xanthomonas* sp.
Fiji disease	*Fiji disease virus* (FDV)
Floral smut(s)	*Sporisorium cruentum, Sphacelotheca erianthi, Sporisorium schweinfurthianum*
Fusarium sett or stem rot	*Gibberella fujikuroi, Fusarium tricinctum*
Grassy shoot	a phytoplasma
Green grassy shoot	a phytoplasma
Gumming	*Xanthomonas axonopodis* pv. *vasculorum*
Helminthosporium leaf spot(s)	*Helminthosporium purpurascens, H. portoricensis, Setosphaeria rostrata, Drechslera tetramera*
Iliau	*Clypeoporthe iliau*
Inflorescence binding	*Ephelis pallida*
Knife-cut (see pokkah boeng)	*Gibberella fujikuroi, G. subglutinans*
Leaf blast	*Didymosphaeria taiwanensis*

Leaf blight	*Leptosphaeria taiwanensis*
Leaf galls (pseudo-Fiji)	unknown
Leaf scald	*Xanthomonas albilineans*
Leaf scorch	*Stagonospora sacchari, Leptosphaeria bicolor*
Leaf sooty mould (sooty mould)	*Capnodium* sp., *Fumago sacchari*
Leaf splitting	*Peronosclerospora miscanthi, P. northii, Peronosclerospora* sp., *Mycosphaerella striatiformans*
Leaf spots	*Apiospora camptospora, Cochliobolus lunatus, Diplodia* sp., *Glomerella tucumanensis, Magnaporthe grisea, Pyrenochaeta indica*
Marasmius basal stem, root and sheath rot	*Marasmius sacchari, M. stenospilus*
Marasmius sheath rot and shoot blight; root rot	*Marasmius stenospilus, M. sacchari*
Midrib blotch	unknown
Mild mosaic	*Sugarcane mild mosaic virus* (SCMMV)
Mosaic	*Sugarcane mosaic virus* (SCMV), *Sorghum mosaic virus* (SrMV)
Mottled stripe	*Herbaspirillum rubrisubalbicans*
Myriogenospora leaf binding	*Myriogenospora aciculispora*
Orange rust	*Puccinia kuehnii*
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 rot of cuttings (= Phytophthora seed piece rot)	*Phytophthora* spp., *P. megasperma*
Phytophthora seed piece rot (= Phytophthora rot of cuttings)	*Phytophthora* spp., *P. megasperma*
Pineapple disease	*Ceratocystis paradoxa*
Pokkah boeng (including knife-cut symptoms)	*Gibberella fujikuroi, G. subglutinans*
Powdery mildew	*Erysiphe graminis*

Pseudo-Fiji (leaf galls)	unknown
Purple spot (red leaf spot)	*Dimeriella sacchari*
Pythium root rot	*Pythium arrhenomanes, Pythium* spp.
Ramu orange leaf	unidentified fungus (exobasidiale)
Ramu scorch	unknown
Ramu streak	unknown
Ramu stunt	a phytoplasma (tentative)
Ratoon stunting or ratoon stunting disease (RSD)	*Clavibacter xyli* subsp. *xyli*
Red leaf mottle	*Peanut clump virus* (PCV)
Red leaf spot (purple spot)	*Dimeriella sacchari*
Red line	*Fusarium* sp.
Red rot	*Glomerella tucumanensis*
Red rot of the leaf sheath	*Corticium rolfsii*
Red spot of the leaf sheath	*Mycovellosiella vaginae*
Red streak	*Pseudomonas syringae* pv. *syringae*
Red stripe (top rot)	*Acidovorax avenae* subsp. *avenae*
Rhizoctonia sheath and shoot rot	*Rhizoctonia solani*
Rind disease and sour rot	*Phaeocytostroma sacchari*
Ring mosaic	unknown
Ring spot	*Leptosphaeria sacchari*
Root and basal stem rot	*Xylaria* cf. *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
Rust (see common rust and orange rust)	*Puccinia melanocephala, P. kuehnii*
Schizophyllum rot	*Schizophyllum commune*
Sclerophthora disease	*Sclerophthora macrospora*
Sclerotium disease	*Sclerotium* sp.
Seedling foliage blights	*Alternaria alternata, Cochliobolus hawaiiensis, Cochliobolus lunatus, Curvularia senegalensis, Bipolaris sacchari, Setosphaeria rostrata*

Sembur	unknown
Sereh	unknown
Sheath rot	*Cytospora sacchari*
Smut (culmicolous smut)	*Ustilago scitaminea*
Smuts (see covered smut, culmicolous smut, floral smut, false floral smut)	*Sphacelotheca macrospora, 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*
Stellate crystal fungus	*Himanthia stellifera*
Stinking rot	*Pseudomonas desaiana*
Streak	*Sugarcane streak virus* (SSV), *Sugarcane streak Egypt virus* (SSEV), *Sugarcane streak Mauritius virus* (SSMV)
Striate mosaic	Sugarcane striate mosaic associated virus (ScSMaV)
Sugarcane bacilliform virus	*Sugarcane bacilliform virus* (SCBV)
Sugarcane streak mosaic virus	*Sugarcane streak mosaic virus* (SCSMV)
Target blotch	*Helminthosporium* sp.
Tar spot (black leaf spot)	*Phyllachora sacchari*
Top rot (red stripe)	*Acidovorax avenae* subsp. *avenae*
Veneer blotch	*Deightoniella papuana*
White leaf	a phytoplasma
White rash (white speck)	*Elsinoe sacchari*
White speck (white rash)	*Elsinoe sacchari*
Wilt	*Fusarium sacchari, Acremonium implicatum, A. furcatum*
Yellow leaf syndrome	*Sugarcane yellow leaf virus* (ScYLV), Sugarcane yellows phytoplasma (ScYP)
Yellow spot	*Mycovellosiella koepkei*
Zonate foot rot	*Fomes* sp.
Zonate leaf spot	*Gloeocercospora sorghi*

Suggested literature

BAUDIN P., FLORES S., MATSUOKA S., RICAUD C., 1982. Official multilingual list of names of sugarcane diseases. Sugarcane Pathologists' Newsletter 29: 71–88.

ISSCT STANDING COMMITTEE ON SUGARCANE DISEASES, 1989. Sugarcane diseases and their world distribution. *In*: Diseases of Sugarcane. Major Diseases. C. Ricaud, B.T. Egan, A.G. Gillaspie Jr and C.G. Hughes (Eds), p. 341–376. Amsterdam, The Netherlands, Elsevier Science Publishers B.V.

RICAUD C., BAILEY R.A., EGAN B.T., GILLASPIE A.G. JR, MATSUOKA S., 1983. Sugarcane diseases and their world distribution. Proceedings International Society Sugar Cane Technologists Congress 18: 27–68.

SIVANESAN A., WALLER J.M., 1986. Sugarcane Diseases. Phytopathological Paper No. 29. Slough UK, CAB International, 88p.

Sugarcane producing countries/locations and their diseases

compiled by Philippe Rott and Jean-Claude Girard

An updated list of disease distribution is available on the Web site of ISSCT: "http://www.sugarnet.com/issct".

Africa

Angola	common rust, mosaic, pokkah boeng, red rot, rind disease and sour rot, ring spot.
Benin	common 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 or stem rot, Helminthosporium leaf spot (*Setosphaeria rostrata*), leaf scald, pineapple disease, pokkah boeng, ratoon stunting, red leaf mottle, red rot, red rot of the leaf sheath, red spot of the leaf sheath, ring spot, smut, wilt.
Burundi	brown spot, common rust, leaf scald, mosaic, mottled stripe, pokkah boeng, red rot, ring spot, smut, Sugarcane bacilliform virus, yellow spot.
Cameroon	brown spot, common rust, dry rot, eye spot, leaf scald, mosaic, pokkah boeng, ratoon stunting, red stripe, ring spot, smut, yellow spot.
Cape Verde	mosaic, streak, Sugarcane bacilliform virus.
Central African Republic	mottled stripe, pokkah boeng, red rot, red rot of the leaf sheath, ring spot, wilt.

Chad	leaf scald, pokkah boeng, red leaf mottle, red rot, smut, yellow spot.
Congo	common rust, leaf scald, mosaic, pineapple disease, ratoon stunting, red leaf mottle, smut, yellow spot.
Côte d'Ivoire	brown stripe, chlorotic streak, Fusarium sett or stem rot, leaf scald, leaf spots, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, streak, smut.
Democratic Republic of the Congo	brown spot, eye spot, leaf scald, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red stripe, ring spot, smut.
Egypt	eye spot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, rind disease and sour rot, ring spot, smut, streak.
Ethiopia	brown spot, brown stripe, mosaic, pokkah boeng, ratoon stunting, red stripe, smut.
Gabon	brown spot, brown stripe, mosaic, pokkah boeng, red leaf mottle, red rot, smut, yellow spot.
Ghana	gumming, leaf scald, mosaic, pineapple disease, pokkah boeng, red rot, red spot of the leaf sheath, ring spot, yellow spot.
Kenya	brown spot, common rust, eye spot, leaf galls, leaf scald, leaf scorch (*Leptosphaeria bicolor*), mosaic, pokkah boeng, ratoon stunting, red rot, red stripe, ring spot, smut, streak, yellow leaf syndrome, yellow spot.
Madagascar	banded sclerotial disease, basal stem, root and sheath rot (*Dictyophora*), brown spot, chlorotic streak, common rust, eye spot, Fiji disease (not detected since 1971), Fusarium sett or stem rot, gumming, leaf galls, leaf scald, mosaic (not detected for more than 30 years), mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, smut, sooty mould, stellate-crystal fungus, Sugarcane bacilliform virus, yellow spot.
Madeira	eye spot, gumming, pineapple disease, pokkah boeng, red rot, streak, Sugarcane bacilliform virus.
Malawi	brown spot, brown stripe, common rust, eye spot, gumming, leaf galls, leaf scald, mosaic, mottled stripe, Phyllosticta leaf spot, mild mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, rind disease and

	sour rot, ring spot, sheath rot, smut, streak, Sugarcane bacilliform virus, yellow leaf syndrome, yellow spot.
Mali	chlorotic streak, pokkah boeng, ratoon stunting, red spot of the leaf sheath, ring spot, smut.
Mauritius	arrow rot, brown spot, chlorotic streak, collar rot, common rust, eye spot, Fusarium sett or stem rot, gumming, iliau, leaf scald, Marasmius basal stem, root and sheath rot, mottled stripe, Pestalotia leaf spot, mild mosaic, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), Schizophyllum rot, Sclerophthora disease, sheath rot, smut, sooty mould, streak, Sugarcane bacilliform virus, wilt, yellow leaf syndrome (ScYLV and ScYP), yellow spot.
Morocco	brown spot, Fusarium sett or stem rot, leaf scald, mosaic, pokkah boeng, red rot, red stripe, Sugarcane bacilliform virus, smut, yellow leaf syndrome (ScYP).
Mozambique	brown spot, brown stripe, chlorotic streak, common rust, eye spot, gumming, leaf galls, leaf scald, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, sheath rot, smut, sooty mould, streak, wilt, yellow leaf syndrome.
Niger	pokkah boeng, red leaf mottle, red rot, red stripe, ring spot, smut, wilt.
Nigeria	banded sclerotial disease, black leaf spot, eye spot, leaf scald, leaf scorch, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red stripe, smut.
Réunion	arrow rot, basal stem, root and sheath rot (*Armillaria, Dictyophora*), brown spot, chlorotic streak, common rust, eye spot, gumming, leaf galls, leaf scald, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, streak, Sugarcane bacilliform virus, wilt, yellow leaf syndrome (ScYLV and ScYP), yellow spot.
Senegal	brown stripe, eye spot, Fusarium sett or stem rot, pineapple disease, pokkah boeng, red leaf mottle, red spot of the leaf sheath, ring spot, smut, wilt, yellow leaf syndrome.
Sierra Leone	eye spot, mosaic, pokkah boeng, ring spot.
Somalia	smut.

South Africa	arrow rot, basal stem, root and sheath rot (*Mycelia sterilia*), black stem rot, brown spot, brown stripe, chlorotic streak, common rust, Diplodia rot, eye spot, Fusarium sett or stem rot, gumming, inflorescence binding, leaf galls, leaf scald, leaf scorch, Marasmius basal stem, root and sheath rot, mild mosaic, mosaic, mottled stripe, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, reovirus, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), Schizophyllum rot, Sclerophthora disease, sheath rot, smut, sooty mould, stellate-crystal fungus, streak, Sugarcane bacilliform virus, target blotch, wilt, yellow leaf syndrome (ScYLV and ScYP), yellow spot.
Sudan	grassy shoot, pokkah boeng, ratoon stunting, red leaf mottle, ring spot, smut, streak.
Swaziland	basal stem, root and sheath rot (*Mycelia sterilia*), brown spot, common rust, gumming, leaf scald, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, smut, sooty mould, yellow leaf syndrome.
Tanzania	basal stem, root and sheath rot (*Armillaria*), brown spot, common rust, eye spot, leaf scald, mosaic, mottled stripe, pokkah boeng, ratoon stunting, red leaf spot, red rot, red stripe, rind disease and sour rot, ring spot, smut, yellow spot.
Togo	mottled stripe, pokkah boeng, red rot, red spot of the leaf sheath, ring spot, wilt.
Uganda	brown spot, common rust, culm and midrib rot, eye spot, Fusarium sett or stem rot, leaf spots, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, sooty mould, streak, wilt, yellow leaf syndrome, yellow spot.
Zambia	brown stripe, common rust, leaf scald, mosaic, Phyllosticta leaf spot, ring spot, ratoon stunting, smut, yellow leaf syndrome.
Zimbabwe	brown spot, common rust, eye spot, Fusarium sett or stem rot, gumming, leaf galls, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, Phyllosticta leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, rind disease and sour rot, ring spot, sheath rot, smut, sooty mould, streak, target blotch, wilt, yellow leaf syndrome (ScYLV and ScYP).

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, brown spot, brown stripe, collar rot, eye spot, grassy shoot, leaf scorch, mosaic, pokkah boeng, ratoon stunting, red leaf spot, red rot, red stripe, ring spot, sheath rot, smut, sooty mould, wilt, yellow spot.
Borneo	black leaf spot, ring spot, yellow spot.
Cambodia	chlorotic streak, eye spot, mosaic, pokkah boeng, red rot, red stripe, ring spot, smut, yellow spot.
China	Baker's leaf spot, banded sclerotial disease, black rot, brown stripe, chlorotic streak, common rust, eye spot, leaf scald, mosaic, orange rust, Phyllosticta leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, yellow spot.
India	Alternaria leaf spot, arrow rot, banded sclerotial disease, basal stem, root and sheath rot (*Mycelia sterilia*), black leaf spot, black rot, brown spot, brown stripe, collar rot, common rust, downy mildew (*Peronosclerospora sacchari, P. philippinensis*), dry rot, ergot, eye spot, false floral smut, floral smut, Fusarium sett or stem rot, grassy shoot, gumming, Helminthosporium leaf spot, leaf blight, leaf scald, leaf scorch, leaf splitting, leaf spots, Marasmius basal stem, root and sheath rot, midrib blotch, mosaic, orange rust, Periconia leaf spot, Pestalotia leaf spot, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf mottle, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring mosaic, ring spot, root rot (*Rhizoctonia*), Schizophyllum rot, Sclerophthora disease, seedling foliage blight, sheath rot, smut, sooty mould, spike, spindle rot, stinking rot, streak, striate mosaic, Sugarcane bacilliform virus, target blotch, wilt, yellow leaf syndrome, yellow spot.
Indonesia	banded sclerotial disease, black leaf spot, black rot, black spot, brown spot, brown stripe, chlorotic streak, common rust, downy mildew, dry rot, eye spot, Fiji disease, Fusarium sett or stem rot, leaf scald, leaf splitting, leaf scorch, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, orange rust, Pestalotia leaf spot, pineapple disease, pokkah boeng,

	Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), Schizophyllum rot, Sclerotium disease, sembur, sereh, sheath rot, smut, stellate-crystal fungus, Sugarcane bacilliform virus, veneer blotch, yellow leaf syndrome (ScYLV), yellow spot.
Japan	banded sclerotial disease, brown stripe, common rust, downy mildew, eye spot, leaf blight, leaf scald, leaf scorch, Marasmius basal stem, root and sheath rot, mosaic, orange rust, pineapple disease, pokkah boeng, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red streak, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, sheath rot, smut, sooty mould, target blotch, white leaf, white speck, yellow spot.
Laos	mosaic.
Malaysia	banded sclerotial disease, black leaf spot, brown stripe, dry rot, eye spot, Fiji disease, Fusarium sett or stem rot, grassy shoot, leaf scald, leaf spots, mosaic, orange rust, Periconia leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, sooty mould, white speck, wilt, yellow spot.
Myanmar	black leaf spot, dry rot, grassy shoot, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, orange rust, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, sooty mould, smut, wilt, yellow spot.
Nepal	brown spot, common rust, grassy shoot, mosaic, Phyllosticta leaf spot, red leaf spot, red rot, red stripe, ring spot, smut, wilt.
Pakistan	black leaf spot, brown spot, brown stripe, chlorotic streak, collar rot, common rust, eye spot, Fusarium sett or stem rot, grassy shoot, leaf scald, mosaic, orange rust, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red stripe, ring spot, root rot (*Rhizoctonia*), sheath rot, smut, stem canker (*Cytospora sacchari*), streak, Sugarcane streak mosaic virus, white leaf, wilt.
Philippines	Baker's leaf spot, banded sclerotial disease, black leaf spot, black spot, brown spot, brown stripe, chlorotic streak, collar rot, common rust, culm and midrib rot, downy mildew (*Peronosclerospora sacchari, P. philippinensis, P. spontanea*), dry rot, ergot (?), eye spot, Fiji disease, Fusarium sett or stem rot, iliau, leaf blight, leaf scald, leaf scorch, leaf splitting, Maras-

mius basal stem, root and sheath rot, mosaic, orange rust, Pestalotia leaf spot, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, sheath rot, smut, sooty mould, veneer blotch, white speck, wilt, yellow spot.

Sri Lanka — brown spot, collar rot, dry rot, eye spot, grassy shoot, leaf scald, mosaic, mottled stripe, orange rust, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring mosaic, ring spot, Schizophyllum rot, sheath rot, smut, white leaf, yellow spot.

Taiwan — Alternaria leaf spot, banded sclerotial disease, black leaf spot, black rot, black stripe, brown spot, brown stripe, chlorotic streak, common rust, covered smut, downy mildew, eye spot, leaf blast, leaf blight, leaf scald, leaf scorch, leaf splitting, Marasmius basal stem, root and sheath rot, mosaic, orange rust, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red line, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root and basal stem rot, root rot (*Leucoporus*), seedling foliage blight, sheath rot, smut, sooty mould, Sugarcane bacilliform virus, white leaf, white speck, yellow spot.

Thailand — banded sclerotial disease, basal stem, root and sheath rot (*Armillaria*), brown spot, brown stripe, chlorotic streak, common rust, downy mildew (*Peronosclerospora sacchari, P. spontanea*), eye spot, false floral smut, Fiji disease, Fusarium sett or stem rot, grassy shoot (?), green grassy shoot, leaf scald, leaf scorch, Marasmius basal stem, root and sheath rot, mild mosaic, mosaic, mottled stripe, orange rust, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot (purple spot), red rot, red rot of the leaf sheath, red spot of the leaf sheath, rind disease and sour rot, ring spot, Schizophyllum rot, Sclerotium disease, sheath rot, smut, sooty mould, Sugarcane bacilliform virus, target blotch, white leaf, white speck, wilt, yellow leaf syndrome, yellow spot.

Vietnam — banded sclerotial disease, common rust, eye spot, Fusarium sett or stem rot, leaf scald, leaf scorch, mosaic, orange rust, pineapple disease, pokkah boeng, red leaf spot (purple spot), red rot of the leaf sheath, red spot of the leaf sheath, red stripe, red rot, rind disease and sour rot, ring spot, smut, sooty mould, white speck, yellow spot.

Australia and Oceania

Australia	arrow rot, Australian basal stem, root and sheath rot, bacterial mottle, banded sclerotial disease, basal stem, root and sheath rot (*Dictyophora, Mycelia sterilia*), black rot, brown rot, brown stripe, chlorotic streak, common rust, dwarf, ergot, eye spot, false floral smut, Fiji disease, Fusarium sett or stem rot, iliau, leaf scald, Marasmius basal stem, root and sheath rot, mild mosaic, mosaic, mottled stripe, Myriogenospora leaf binding, orange rust, Pachymetra root rot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, Sclerophthora disease, sheath rot, smut (Western Australia only), sooty mould, stellate-crystal fungus, striate mosaic, Sugarcane bacilliform virus, yellow leaf syndrome, yellow spot.
Fiji	banded sclerotial disease, brown stripe, chlorotic streak, common rust, downy mildew, eye spot, Fiji disease, Fusarium sett or stem rot, gumming, leaf scald, leaf scorch, leaf splitting, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, orange rust, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, sheath rot, smut, yellow spot.
Guam	orange rust, pokkah boeng, red rot, red rot of the leaf sheath, red stripe, white speck.
Hawaii	black rot, brown spot, brown stripe, chlorotic streak, common rust, eye spot (not detected for several years), Fusarium sett or stem rot, iliau, leaf scald, leaf splitting, leaf spots, Marasmius basal stem, root and sheath rot, mosaic, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring mosaic, ring spot, Schizophyllum rot, seedling foliage blight, sheath rot, smut, sooty mould, Sugarcane bacilliform virus, white rash, yellow leaf syndrome (ScYLV).
New Caledonia	Fiji disease, orange rust, rind disease and sour rot, yellow spot.
Papua New Guinea	banded sclerotial disease, black leaf spot, brown spot, brown stripe, chlorotic streak, common rust, downy mildew, eye spot, Fiji disease, leaf scald, leaf scorch, leaf splitting (*Mycosphaerella striatiformans* or *Peronosclerospora mis-*

	canthi), leaf spots, mosaic, orange rust, pineapple disease, pokkah boeng, Ramu orange leaf, Ramu scorch, Ramu streak, Ramu stunt, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, Sobemo virus, sooty mould, Sugarcane bacilliform virus, veneer blotch, wilt, yellow leaf syndrome (ScYP), yellow spot, zonate leaf spot.
Samoa	banded sclerotial disease, brown stripe, chlorotic streak, eye spot, Fiji disease, orange rust, pokkah boeng, red rot, ring spot, yellow spot, zonate leaf spot.
Solomon Islands	black leaf spot, eye spot, Fiji disease, orange rust, red rot, ring spot, sooty mould, veneer blotch, yellow spot, zonate leaf spot.
Tahiti	pineapple disease, leaf scald, rind disease and sour rot.
Vanuatu	Fiji disease, leaf spots, red rot, ring spot.

Central America and the Caribbean

Antigua	common rust, eye spot, gumming, Marasmius basal stem, root and sheath rot, pineapple disease, pokkah boeng, ratoon stunting, red rot, rind disease and sour rot, ring spot, smut.
Barbados	apex rot, brown stripe, common rust, dry rot, dry top rot, eye spot, Fusarium sett or stem rot, gumming (not detected for more than 30 years), leaf scald, Marasmius basal stem, root and sheath rot, mosaic (not detected for more than 30 years), mottled stripe, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), smut, Sugarcane bacilliform virus, wilt, yellow leaf syndrome (ScYLV), yellow spot.
Belize	brown stripe, common rust, eye spot, gumming, leaf scald, mosaic, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, rind disease and sour rot, ring spot, sheath rot, smut.
Costa Rica	common rust, eye spot, leaf spots, mild mosaic, mosaic, pineapple disease, ratoon stunting, red stripe, ring spot, smut.
Cuba	Alternaria leaf spot, banded sclerotial disease, brown spot, brown stripe, chlorotic streak, common rust, dry rot, dry top rot, eye spot, floral smut, Fusarium sett or stem rot, gumming,

	iliau, leaf scald, leaf scorch, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, Pestalotia leaf spot, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), Schizophyllum rot, seedling foliage blight, sheath rot, smut, sooty mould, stellate-crystal fungus, Sugarcane bacilliform virus, target blotch, white speck, wilt, yellow leaf syndrome (ScYLV and ScYP), yellow spot, zonate foot rot.
Dominica	leaf scald, gumming, smut.
Dominican Republic	black rot, brown stripe, chlorotic streak, common rust, dry rot, eye spot, Fusarium sett or stem rot, gumming, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, sheath rot, smut, stellate-crystal fungus, Sugarcane bacilliform virus, yellow leaf syndrome (ScYLV).
El Salvador	common rust, eye spot, mosaic, Pythium root rot, ratoon stunting, red rot, red stripe, ring spot, smut, wilt, yellow leaf syndrome (ScYLV).
Grenada	chlorotic streak, eye spot, leaf scald, Marasmius basal stem, root and sheath rot.
Guadeloupe	apex rot, brown stripe, chlorotic streak, common rust, eye spot, Fusarium sett or stem rot, gumming (not detected for more than 30 years), leaf scald, Marasmius basal stem, root and sheath rot, mosaic (not detected for more than 30 years), mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red stripe, rind disease and sour rot, ring spot, smut, sooty mould, Sugarcane bacilliform virus, wilt, yellow leaf syndrome (ScYLV), yellow spot.
Guatemala	common rust, eye spot, leaf scald, mosaic, red leaf spot, red rot, red stripe, smut, wilt, yellow leaf syndrome (ScYLV), yellow spot.
Haiti	common rust, eye spot, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, red rot, red spot of the leaf sheath, rind disease and sour rot, Schizophyllum rot, smut.
Honduras	chlorotic streak, common rust, eye spot, mosaic, pokkah boeng, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut.

Jamaica	apex rot, brown spot, brown stripe, chlorotic streak, common rust, dry rot, eye spot, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, pineapple disease, pokkah boeng, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, sooty mould, stellate-crystal fungus, yellow leaf syndrome.
Martinique	apex rot, chlorotic streak, common rust, eye spot, gumming (not detected for more than 30 years) leaf scald, Marasmius basal stem, root and sheath rot, mosaic (not detected for more than 30 years), mottled stripe, pokkah boeng, ratoon stunting, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, ring spot, Schizophyllum rot, smut, yellow leaf syndrome (ScYLV and ScYP).
Nicaragua	brown spot, chlorotic streak, common rust, Fusarium sett or stem rot, mosaic, mottled stripe, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, sheath rot, smut, sooty mould, yellow leaf syndrome (ScYLV).
Panama	banded sclerotial disease, black rot, brown spot, brown stripe, chlorotic streak, common rust, Diplodia rot, dry top rot, ergot, eye spot, Fusarium sett or stem rot, gumming, leaf scald, leaf scorch, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, Periconia leaf spot, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, seedling foliage blight, sheath rot, smut, sooty mould, wilt, yellow spot.
Puerto Rico	banded sclerotial disease, basal stem, root and sheath rot (*Olpidium*), brown spot, brown stripe, chlorotic streak, common rust, culm and midrib rot, Diplodia rot, dry rot, dry top rot, eye spot, Fusarium sett or stem rot, gumming, Helminthosporium leaf spots, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, Phyllosticta leaf spot, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root and basal stem rot, root rot (*Rhizoctonia*), Schizophyllum rot, seedling foliage blight, sheath rot, smut, sooty mould, stellate-crystal fungus, Sugarcane bacilliform virus, white speck.
St Kitts and Nevis	apex rot, brown stripe, common rust, eye spot, gumming, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, rind disease and sour rot, ring spot, smut, wilt.

St Lucia	chlorotic streak, eye spot, gumming, leaf scald, Marasmius basal stem, root and sheath rot, pineapple disease, pokkah boeng, red rot, rind disease and sour rot.
St Thomas	eye spot, mosaic, pokkah boeng.
St Vincent	gumming, leaf scald, Marasmius basal stem, root and sheath rot.
Trinidad	brown stripe, chlorotic streak, common rust, dry rot, eye spot, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, smut, sooty mould, stellate-crystal fungus, wilt, yellow spot.

Central East

Iraq	leaf scald, mosaic, pokkah boeng, red rot, red stripe, rind disease and sour rot, smut.
Iran	grassy shoot, mosaic, pineapple disease, pokkah boeng, red rot of the leaf sheath, red streak, red stripe, smut, wilt, yellow leaf syndrome.
Turkey	chlorotic streak, mosaic, sheath rot.

Europe

Italy	black leaf spot (Sicily), eye spot, mosaic, pokkah boeng, powdery mildew.
Portugal	rind disease and sour rot, smut.
Spain	mosaic.

North America

Mexico	brown spot, chlorotic streak, common rust, dry rot, eye spot, Fusarium sett or stem rot, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, sheath rot, smut, Sugarcane bacilliform virus, wilt, yellow leaf syndrome.

United States of America (Florida, Louisiana and Texas)	banded sclerotial disease, basal stem, root and sheath rot *(Rhizoctonia)*, black rot, brown spot, brown stripe, chlorotic streak, common rust, dry top rot, eye spot, Fusarium sett or stem rot, Helminthosporium leaf spots, iliau, leaf scald, Marasmius basal stem, root and sheath rot, mild mosaic, mosaic, mottled stripe, Myriogenospora leaf binding, Phyllosticta leaf spot, Phytophthora rot of cuttings, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root and basal stem rot, Schizophyllum rot, Sclerophthora disease, seedling foliage blight, sheath rot, smut, sooty mould, Sugarcane bacilliform virus, target blotch, white speck, wilt (?), yellow leaf syndrome (ScYLV), yellow spot, zonate leaf spot.

South America

Argentina	arrow rot, black leaf spot, brown spot, chlorotic streak, collar rot, common rust, eye spot, Fusarium sett or stem rot, gumming, leaf scald, leaf scorch, leaf spots, mosaic, Myriogenospora leaf binding, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, ring spot, Schizophyllum rot, seedling foliage blight, sheath rot, smut, sooty mould, stellate-crystal fungus, Sugarcane bacilliform virus, wilt, yellow leaf syndrome, yellow spot.
Bolivia	common rust, eye spot, mosaic, ratoon stunting, red rot, red stripe, rind disease and sour rot, ring spot, sheath rot, smut.
Brazil	black rot, brown spot, brown stripe, chlorotic streak, collar rot, common rust, dry rot, eye spot, false red stripe, Fusarium sett or stem rot, gumming, iliau, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, Myriogenospora leaf binding, pineapple disease, pokkah boeng, Pythium root rot, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Schizophyllum rot, Sclerotium disease, seedling foliage blight, sheath rot, smut, sooty mould, stellate-crystal fungus, Sugarcane bacilliform virus, white speck, wilt, yellow leaf syndrome (ScYLV), yellow spot.
Colombia	brown spot, chlorotic streak, common rust, culm and midrib rot, Diplodia rot, dry top rot, ergot, eye spot, false floral smut, gumming, leaf scald, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, Phyllosticta leaf spot, pineapple

	disease, pokkah boeng, purple spot, Pythium root rot, ratoon stunting, red leaf spot, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, root rot (*Rhizoctonia*), smut, stellate-crystal fungus, Sugarcane bacilliform virus, wilt, yellow leaf syndrome (ScYLV), yellow spot.
Ecuador	common rust, leaf scald, mosaic, pokkah boeng, ratoon stunting, red spot of the leaf sheath, smut.
Guyana	brown stripe, chlorotic streak, common rust, dry rot, dry top rot, eye spot, Fusarium sett or stem rot, leaf scald, leaf spots, Marasmius basal stem, root and sheath rot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, sheath rot, smut, sooty mould, stellate-crystal fungus, yellow spot.
Paraguay	Marasmius basal stem, root and sheath rot, mosaic, pineapple disease, pokkah boeng, rind disease and sour rot, ring spot, smut, sooty mould.
Peru	black rot, brown spot, brown stripe, common rust, eye spot, Fusarium sett or stem rot, Marasmius basal stem, root and sheath rot, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of the leaf sheath, red spot of the leaf sheath, red stripe, rind disease and sour rot, ring spot, Sclerophthora disease, sheath rot, smut (?), sooty mould, yellow leaf syndrome (ScYLV).
Surinam	chlorotic streak, eye spot, leaf scald, mosaic, pokkah boeng, red stripe, ring spot.
Uruguay	leaf scald, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red stripe, ring spot, smut.
Venezuela	brown spot, chlorotic streak, common rust, dry top rot, eye spot, Fusarium sett or stem rot, leaf scald, leaf scorch, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red spot of the leaf sheath, red stripe, ring spot, smut, yellow leaf syndrome.

Authors addresses

AGNIHOTRI V.P., Indian Institute of Sugarcane Research, Lucknow 226002, India.

AUTREY L. JEAN CLAUDE, Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

BAILEY ROGER A., South African Sugar Association Experiment Station, Private Bag X02, Mount Edgecombe 4300, South Africa.

BLAIR BRENDEN, Queensland Department of Primary Industries, South Johnston Research Station, South Johnston, Queensland 4859, Australia.

CHATENET MICHÈLE, Centre de coopération internationale en recherche agronomique pour le développement, Département des cultures annuelles, Programme canne à sucre, TA 71/09, Avenue Agropolis, 34398 Montpellier Cedex 5, France.

CHEN C.T., Taiwan Sugar Research Institute, Plant Propagation Center, Tainan, Taiwan, Republic of China.

COMSTOCK JACK C., United States Department of Agriculture, Agricultural Research Service, Sugarcane Field Station, HCR Box 8, Canal Point, Florida 33438, USA.

CROFT BARRY J., Bureau of Sugar Experiment Stations, Lot 207 Old Cove Road, Woodford, Queensland 4514, Australia.

CRONJÉ C. PIETER R., South African Sugar Association Experiment Station, Private Bag X02, Mount Edgecombe 4300, South Africa.

DAUGROIS JEAN, Centre de coopération internationale en recherche agronomique pour le développement, Département des cultures annuelles, Programme canne à sucre, Station de Roujol, 97170 Petit Bourg, Guadeloupe, FWI.

DAVIS MICHAEL J., University of Florida, Tropical Research and Education Center, IFAS, 18905 S.W. 280th Street, Homestead, Florida 33031, USA.

DOOKUN ASHA, Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

EGAN BRIAN T., BTE Consulting, 48 Streeton Parade, Everton Park, Queensland 4053, Australia.

FANG JING-GWO, Taiwan Sugar Research Institute, Department of Horticulture, 54, Sheng-Chan Road, Tainan 701, Taiwan, Republic of China.

GIGLIOTI EDER A., Universidade Federal de São Carlos, Centro de Ciências Agrárias, Via Anhanguera, km 174, Caixa Postal 153, 13600-970 Araras SP, Brazil.

GIRARD JEAN-CLAUDE, Centre de coopération internationale en recherche agronomique pour le développement, Département des cultures annuelles, Programme canne à sucre, TA 71/09, Avenue Agropolis, 34398 Montpellier Cedex 5, France.

GRISHAM MICHAEL P., United States Department of Agriculture, Agricultural Research Service, Southern Regional Research Center, Sugarcane Research Unit, 5883 USDA Road, Houma, Louisiana 70360, USA.

HOY JEFFREY W., Louisiana State University, Department of Plant Pathology and Crop Physiology, Baton Rouge, Louisiana 70803, USA.

HSIEH W.H., National Chung Hsing University, Department of Plant Pathology, Taichung, Taiwan, Republic of China.

IRAWAN, Indonesian Sugar Research Institute, Jalan Pahlawan 25, Pasuruan 67126, Indonesia.

JONES PHIL, Plant Pathology Department, IACR Rothamsted, Harpenden AL5 2JQ, United Kingdom.

KUSALWONG ANUSORN, Plant Pathology and Microbiology Division, Department of Agriculture, Chatuchak, Bangkok 10900, Thailand.

LAL SUNITA, Indian Institute of Sugarcane Research, Lucknow 226002, India.

LEE CHING-SHIOU, Taiwan Sugar Research Institute, Department of Agronomy, 54, Sheng-Chan Road, Tainan 701, Taiwan, Republic of China.

LIANG YAW-GUANG, Taiwan Sugar Research Institute, Department of Agronomy, 54, Sheng-Chan Road, Tainan 701, Taiwan, Republic of China.

LOCKHART BEN E., University of Minnesota, Department of Plant Pathology, 495 Borlaug Hall, 1991 Upper Buford Circle, St Paul, Minnesota 55108, USA.

MAGAREY ROBERT C., Bureau of Sugar Experiment Stations, PO Box 566, Tully, Queensland 4854, Australia.

MATSUOKA SIZUO, Universidade Federal de São Carlos, Centro de Ciências Agrárias, Via Anhanguera, km 174, Caixa Postal 153, 13600-970, Araras SP, Brazil.

PATIL A.S., Vasantdada Sugar Institute, Department of Plant Pathology and Agricultural Microbiology, Manjari (Bk.) 412307, Dist. Pune, Maharashtra, India.

PETERSCHMITT MICHEL, Centre de coopération internationale en recherche agronomique pour le développement, Département d'amélioration des méthodes pour l'innovation scientifique, Programme protection des cultures, TA 40/02, Avenue Agropolis, 34398 Montpellier Cedex 5, France.

PLIANSINCHAI UPSORN, Field Crops Research Institute, Suphan Buri Field Crops Research Center, U-thong, Suphan Buri 72160, Thailand.

PRAMMANEE SIRIPATR, Kasetsart University, Faculty of Liberal Arts and Science, Kamphaengsaen Campus, Nakhon Pathom 73140, Thailand.

RAHIMIAN HESHMATOLAH, College of Agriculture, Department of Plant Protection, Mazandaran University, PO Box 578, Sari, Iran.

RAID RICHARD N., University of Florida, Everglades Research and Education Center, IFAS, PO Box 8003, Belle Glade, Florida 33430-8003, USA.

RANDLES JOHN W., University of Adelaide, Department of Applied and Molecular Ecology, Waite Campus, Glen Osmond, South Australia 5064, Australia.

RAO G.P., Sugarcane Research Station, Kunraghat, Gorakhpur 273008, Uttar Pradesh, India.

RICAUD CLAUDE, International Society of Sugar Cane Technologists, Permanent Secretariat, c/o Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

ROTT PHILIPPE, Centre de coopération internationale en recherche agronomique pour le développement, Département des cultures annuelles, Programme canne à sucre, TA 71/09, Avenue Agropolis, 34398 Montpellier Cedex 5, France.

SAUMTALLY A. SALEM, Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

SINGH R.P., Indian Institute of Sugarcane Research, Lucknow 226002, India.

SMITH GRANT R., Bureau of Sugar Experiment Stations, David North Plant Research Centre, PO Box 86, Indooroopilly, Queensland 4068, Australia.

SOOPRAMANIEN G. CLAUDE, Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

STIRLING GRAHAM, Biological Crop Protection Pty. Ltd, 3601, Moggill Road, Moggill, Queensland 4070, Australia.

SULLIVAN SYDNEY, Mauritius Sugar Industry Research Institute, Réduit, Mauritius.

SUMA SIDNEY, Ramu Sugar Limited, PO Box 2183, Lae, Papua New Guinea.

VISWANATHAN R., Sugarcane Breeding Institute, Plant Pathology Section, Indian Council of Agricultural Research, Coimbatore 641007, India.

WHITTLE PETER J.L., Bureau of Sugar Experiment Stations, David North Plant Research Centre, PO Box 86, Indooroopilly, Queensland 4068, Australia.

Fichier préparé par Nicolas Perrier, société 4P
Imprimé pour vous par Books on Demand (Allemagne)

THE REPÈRES SERIES

This series presents results of research on tropical agriculture, forestry, and livestock production. It is intended for researchers, teachers and students.

ALREADY PUBLISHED

In vitro culture of tropical plants
Claude Teisson, editor

Agricultural research and innovation in tropical Africa
Pierre-Marie Bosc, Ellen Hanak Freud

Integrated pest management of tropical perennial crops
Dominique Mariau, editor

Centre de coopération internationale en recherche agronomique pour le développement

International Society of Sugar Cane Technologists

Many changes have occurred in sugarcane pathology during the last decade. Information on known diseases has been updated, new diseases have been described and identified, and thanks to progress in molecular biology, new diagnostic techniques have been developed. The pathology section of the International Society of Sugar Cane Technologists (ISSCT) decided therefore to publish a new book on sugarcane diseases. The objective of this book is not to exhaustively describe each disease, but to supply sufficient and updated information regarding the scientific knowledge and practical aspects of disease control to those who have to deal with sugarcane. Each disease is described by its causal agent, geographical distribution, symptoms, transmission, host range, epidemiology and economic importance. Information regarding strains of the pathogen, diagnosis and control is also provided. References are cited and each disease is illustrated by several colour pictures.

50,00 €

Diffusion: La Librairie du Cirad
Avenue Agropolis, TA283/04
34398 Montpellier Cedex 5
France