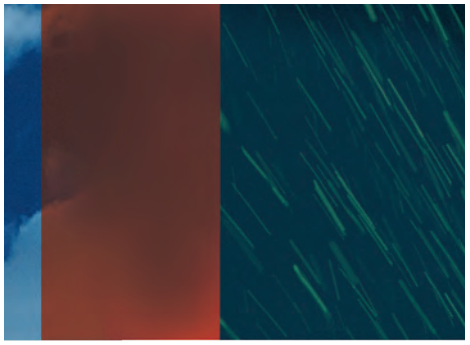

DISEASE MANAGEMENT

Barry Croft, Robert Magarey and Peter Whittle

MANAGEMENT of sugarcane diseases is an important factor in maintaining the competitive advantage of the Australian sugar industry. Diseases have generally been restricted to low levels, helping the industry to maintain high yields of high-quality sugar. Two important diseases, downy mildew and gumming disease, were eradicated in the 1950s, relieving the industry of direct yield losses and the expense of control measures. However, the industry has had to face new challenges from diseases.

The finding of sugarcane smut for the first time in Australia in the Ord River Irrigation Area in 1998 and the discovery of the serious root disease, pachymetra root rot, in 1981 have challenged the sugar industry's ability to respond to new threats.

Other diseases, such as Fiji disease, have caused extensive yield losses and disruption to farming systems in the past and continue to threaten the industry. Managing new threats and the ongoing management of endemic diseases such as ratoon stunting disease (RSD) and chlorotic streak will require commitment and innovation from growers, industry personnel and researchers in the future.

This chapter discusses the primary control measures used to manage diseases and then outlines the major diseases present in Australia and some potentially serious exotic diseases. Quarantine is discussed in a separate chapter.

CONTROL MEASURES

Approved varieties and disease resistance

Regulating which varieties can be grown in a district is a powerful disease management tool. Only varieties with sufficient disease resistance to prevent economic losses or build-up of disease should be grown in a district. By removing all susceptible varieties, some diseases can be brought to a low incidence or even to extinction. In Queensland, lists of varieties approved

for each mill area provide this disease management option. It is not always possible to predict accurately in advance a variety's field reaction to a disease. Some varieties that are approved for a district may succumb to disease, or a new disease may arrive. This will require varieties to be removed from the list of varieties approved for growing.

In the January edition of the Queensland Government Gazette each year, a list of varieties approved by the Chief Executive Officer of BSES for planting in each mill area in Queensland is published. This list is reproduced in the January edition of the *BSES Bulletin*.

A second list is also published with varieties that can be ratooned but not planted. By law (*Sugar Industry Act 1999 Qld.*), only varieties on these lists may be grown for commercial production. Varieties not on these lists may be grown under permit from the Chief Executive Officer of BSES. The location and area of unapproved varieties should be included in an application for a permit.

If an unapproved variety is grown without a permit, it is considered to be a pest and under the Queensland *Plant Protection Act 1989,* the Chief Executive Officer can order the cane to be destroyed. In some cases, certain varieties may be considered to pose a disease risk and a permit would not be issued for their planting.

The Chief Executive Officer takes into consideration a variety's productivity, resistance to diseases and pests, and milling characteristics before approving the variety. Once a variety is approved, it will remain on the list as long as no unforeseen problem, such as a disease risk, develops and the variety remains productive on at least some farms in a district. If a variety is no longer considered to be suitable in a district, it is removed from the list of varieties approved for planting and is placed on the list of varieties approved for ratooning only.

It may remain on this list until the last fields of the variety are ploughed out or until a time specified by the Chief Executive Officer. In the case where a variety is removed from the list of varieties approved for planting because of a disease epidemic, the Chief Executive Officer may advise the industry that the variety will be removed from the ratooning list after a set number of years. All fields of the variety must be removed by this date. This provision has been used to restrict the development of epidemics of Fiji disease, leaf scald, mosaic, red rot and eye spot.

All new varieties are screened for resistance to seven diseases in disease resistance trials before they are released to industry. These diseases are Fiji disease, leaf scald, mosaic, red rot, pachymetra root rot, chlorotic streak and RSD. If a variety is too susceptible to one or more of these diseases, and the disease is important in the district for which the variety is being considered, the variety will be discarded from the plant improvement program.

Other diseases such as yellow spot, orange rust and common rust are noted in plant improvement trials and, if high levels of disease occur, yield is reduced and the variety is discarded naturally.

To reduce the number of varieties that have to be discarded because of disease susceptibility, all parent varieties are also rated for resistance to the major diseases and restrictions are placed on the crosses that are made. For example, two parent varieties that are susceptible to Fiji disease will not be crossed, because there is a high probability that their progeny will also be susceptible and will have to be discarded later in the improvement program.

Sugarcane varieties have recently been genetically modified with genes conferring resistance to sugarcane mosaic, leaf scald and Fiji disease. These genetically modified plants are being tested in field and glasshouse trials; none have been grown commercially. This technology offers an exciting new way of producing disease resistant, high yielding varieties for the sugar industry.

Disease-free planting material

Approved seed

Sugarcane is clonally propagated (grown from stalk cuttings), and a number of serious diseases can be carried within the planting material. It is, therefore, essential to ensure that disease-free cane is used to establish new crops. Because of the bulkiness of sugarcane planting material (5–10 t/ha of cane) and its relatively short viability after cutting (3–4 weeks in tropical areas), most commercial planting material is grown on-farm. Systems have been developed to supply growers with a nucleus of disease-free planting material or approved seed, to be propagated on the farm.

In each mill area or region, the Cane Protection and Productivity Boards (CPPBs) operate approved-seed plots. Approved seed is produced under strict quality assurance guidelines that reduce the risk of diseases being present. In most districts, the highest quality disease-free planting material is planted into a mother plot. This material is hot-water treated each year and the resulting mother-plot cane is used to plant the approved-seed distribution plot. All mother-plot and approved-seed-plot cane is inspected for diseases on a regular basis and sampled for RSD, which has no external symptoms. Approved seed is distributed to growers and, ideally, each grower should obtain enough approved seed each year to plant their commercial crops after 1–2 years of further multiplication. Against RSD and leaf scald, this system is highly effective if appropriate hygiene measures are observed during propagation. However, if Fiji disease or mosaic is present in the commercial planting area, it may be necessary to grow plant sources some distance away to avoid infection by insect vectors. Approved seed must be propagated in a field where the previous crop has been completely destroyed, so that volunteer plants cannot carry over diseases into the approved seed.

Normally, the first and second progeny of approved seed could be planted without further inspection for disease. However, in some areas where insect-vectored diseases such as Fiji disease and mosaic are active, it may be necessary to inspect all plant sources including the first and second progeny of approved seed. CPPBs offer an inspection service in most areas.

Alternative planting material and inspections for diseases

In some cases, farmers may not have sufficient quantities of first or second progeny of approved seed to plant their fields. Alternative plant sources should be used only after inspection by an experienced inspector, who would look for visual symptoms of diseases and take samples for testing for RSD.

Hot-water treatment

Hot-water treatment by certain procedures can free sugarcane of RSD, leaf scald, and chlorotic streak.

Long hot-water treatment (LHWT)

Long hot-water treatment (3 hours at 50ºC) is used to control RSD. All cane planted in mother plots or cane that has not come from a mother plot but is to be planted into an approved seed plot should receive a LHWT. In some districts, growers bring cane from their own farms to the treatment facilities to be treated. Cane to be treated from farms should be the cleanest source on the farm and should test negative for RSD, as well as being of good quality for planting. To recover a variety that is infected with RSD, it should be treated in consecutive years until it tests negative in two consecutive years. Cane to be treated should be stripped of trash and treated at 50°C for 3 hours as bundles of whole stalks or as setts. Temperature control is critical, as temperatures over 50°C adversely affect cane germination, and temperatures below 50°C (<49.8°C) reduce the effectiveness of disease control.

The treatment facilities in most districts have the capacity to treat loads of 1–2 tonnes of cane and are heated by electricity or steam from the sugar mill.

Cold-soak long hot-water treatment (CSLHWT)

The CSLHWT is used to control leaf scald. Varieties susceptible to leaf scald, or which come from areas with endemic leaf scald, should receive CSLHWT before planting in propagation plots. Cane stalks should be trimmed to approximately 1.5 m in length or treated as two-bud or one-bud setts. Cane that is treated as stalks should be cut to 1.5 m in length and should be stacked (loosely) in layers approximately three stalks deep with 50 mm spacers between the layers (e.g. a piece of timber). The cane should be soaked in water at ambient temperature for at least 40 hours with a slow input of fresh water.

Within 6 hours of removing the cane from the cold water, the cane should be treated in hot water at 50°C for 3 hours. Treated cane should be planted in areas considered to be at a low risk of re-infection, i.e. 500 m from known infected blocks or large drains with heavy weed infestation.

Short hot-water treatment (SHWT)

In some districts, short hot-water treatment (SHWT) may be necessary to control chlorotic streak or the spread of insect pests (e.g. weevil borer). SHWT follows the same procedure as LHWT, but the treatment is 50°C for 30 minutes. This treatment generally stimulates germination. SHWT does not control RSD or leaf scald.

Hygiene and destruction of diseased crops

Hygiene

Hygiene is an important disease control strategy, particularly for diseases transmitted on cutting implements and general machinery. These include RSD and leaf scald. RSD is a highly transmissible disease; planting one infected stalk in a trash planter may lead to infection of the next 100 stools of cane, through the transfer of infected juice on the blades of the planter. A cane harvester can similarly spread the disease.

All cutting implements which come into contact with either planting material or standing crops, or from which contaminated juice can be blown or drip onto freshly cut surfaces, (including cane knives, chain saws, plant cutters, planters, bins used to transport billets for planting, harvesters, juice samplers, etc.), should be disinfected.

Several methods are suitable; the best disinfectant for use will vary with the type of equipment to be disinfected. Saturating the surfaces of cutting implements with methylated spirits (diluted to 70% with water) is ideal for use with cane knives and secateurs, etc. Its flammability makes it unsuitable for harvesters.

Commercial, registered sterilising agents based on benzalkonium chloride are also suitable for cane knives, plant cutters, harvesters and other equipment where fire may be a hazard. Disinfection with these products requires all surfaces to be thoroughly cleaned before application and kept wet with the product for at least 5 minutes. It is important that a number of parts of the cane harvester are treated to prevent disease transmission, including the base-cutter blades, chopper box and extractors. Steriliser solution should be discarded when dirty or not fresh, according to label instructions.

In planting machines, it is essential to flush and disinfect recirculating fungicide spray systems, since they can harbour RSD and leaf scald bacteria.

General hygiene practices for preventing disease include minimising the incidence of alternative hosts around the cane farm. Some diseases such as sugarcane mosaic and the exotic disease, downy mildew, may increase rapidly if infected hosts are growing adjacent to sugarcane fields. Australian diseases with alternative hosts include bacterial mottle, chlorotic streak, leaf scald and sclerophthora. Some common hosts include guinea grass (*Panicum maximum*), corn (*Zea mays*) and elephant grass (*Pennisetum purpureum*).

Destruction of diseased crops

Diseased crops may act as reservoirs for spread into neighbouring crops. Fiji disease is one example where this is important. Rapid build up of Fiji disease occurs where infected blocks of cane are ratooned and when high populations of the vector (a planthopper) are present.

In the late 1970s, a severe epidemic developed in the Bundaberg district of southern Queensland. For several reasons, infected crops of the susceptible variety NCo310 were not destroyed at the optimal time. This led to rapid spread of the disease through the district, severe financial hardship on some farms, and the loss of many varieties of intermediate resistance, which could not be grown because of the extremely high disease pressure.

When Fiji disease was found in the Mackay district, the presence of suitable alternative (more resistant) varieties and the rapid elimination of diseased fields minimised the economic effects of Fiji disease. The Bundaberg district suffered for a number of years, while minimal effects were experienced in Mackay.

In a number of cases, the industry has attempted to stem epidemics by roguing diseased crops in preference to crop destruction. Past a certain level of disease incidence, roguing will be ineffective, since many infected plants will be non-symptomatic. Moreover, the labour cost of roguing on a large scale is now prohibitive.

To eradicate disease infections and prevent their further spread, the Queensland *Plant Protection Act 1989* provides for an inspector to direct that diseased crops be destroyed (as well as other necessary quarantine precautions). Such directions can only be given for a declared pest or disease.

It is a wise precaution to destroy volunteer cane growing in abandoned cane fields, since this cane may harbour pests and diseases that may remain undetected due to neglect of the fields. Volunteer cane is a pest under the Queensland *Plant Protection Act 1989* and its removal can be directed.

Cultural control

Farming systems may influence disease levels; disease is a result of the interaction of a range of factors including weather, planting times, drainage, varietal resistance and in some cases nutrition.

Time of planting and harvesting

Late-spring planting usually means that crops are tillering and producing fresh new growth when the wet season begins. Active growth in wet weather favours the spread of some diseases such as red stripe (top rot), a bacterial disease spread from infected shoots, or soil, by water-splash.

The noble cane (*Saccharum officinarum*) Badila suffered significant losses from this disease when late-planted, but minor losses when planted in early spring or autumn. In recent times, varieties such as CP51-21 have also suffered significantly from the disease. Fiji disease also spreads more rapidly in spring-planted crops, since planthopper populations reach a peak when the crops are young and in their most susceptible condition.

Drainage

Wet soil conditions may favour the transmission of diseases. Chlorotic streak, the classic case, can be transmitted in floodwater, and infection is greater when crops are ratooned under wet soil conditions. In areas where flooding and poor drainage are prevalent, such as the Herbert River and Rocky Point, a large proportion of crops may be diseased. Infection can be minimised by improving drainage and preventing the waterlogging of fields. Mitigation of floodwaters may also reduce the levels of the waterborne diseases bacterial mottle and sclerophthora.

Re-cycling irrigation drainage water can spread chlorotic streak from one field to another. Use of fresh irrigation water, or a mixture of fresh and recycled water, minimises disease spread.

Hilling-up

Basal stem, root and sheath rot is a fungal disease affecting plant crops early in their development. The fungus attacks the root buds of germinating setts and young shoots. In severe cases, a poor and inadequate root system causes very unthrifty growth and in some cases stool death. Hilling-up of diseased stools (crops) may provide sufficient healthy soil for root development from buds higher up the stalk, and crop recovery.

Fallowing and crop rotation

Fallowing and crop rotation are important disease control practices in most cropping systems. In the Australian sugarcane industry, fallowing is practised in the 4–12 months break between the ploughing out of a crop and its replanting. This generally occurs once every 4–8 years depending on the number of ratoon crops. During this fallow period, the field is either left uncultivated, allowing weeds to grow, or is planted to a legume such as cowpeas or soybeans. Usually the legume is a green manure crop, but increasingly the soybean crop is harvested as a commercial crop. In Bundaberg, vegetable crops may also be planted during the fallow. These fallow periods provide an opportunity to remove all volunteers from the previous crop, which may be harbouring diseases, and to reduce the populations of some soil pathogens such as nematodes. A short fallow does not significantly reduce the populations of the pachymetra root rot fungus. Longer rotations of 2–3 years or longer are practised on a small scale in some districts with bananas or other crops sometimes being rotated with sugarcane. These rotations are very beneficial for controlling sugarcane root diseases. They are so successful that care has to be taken when selecting varieties to be grown after a long fallow. Less vigorous, high sugar-content varieties may perform better for the first crop after a long break from sugarcane.

In many districts, there has been an increase in the practice known as ploughout-replant. The previous crop is ploughed out and replanted within weeks of the final harvest. This management practice can lead to build-up of some diseases.

Other factors

Plant nutrition is also known to influence disease. One example is brown stripe, a minor disease that usually occurs on soils of low fertility. Potassium and perhaps phosphorus deficiencies have been linked with this disease. A slow-growing pathogen has also been linked to the disease called sunny-side up, which has been associated with silicon deficiency. Better nutrition alone will not prevent the occurrence of all diseases, but poor cultural practices may increase the extent and severity of some. In contrast, highly fertile soils and good irrigation can increase the susceptibility of cane to the virus diseases, Fiji disease and mosaic, by increasing the activity of insect vectors of the viruses.

The risk of epidemics can be reduced by maintaining a mix of varieties on farms and within districts. On several occasions, some districts have been dominated by one variety, with no variety rotation. The variety NCo310 constituted over 80% of the crop in the central and southern districts in the 1970s. NCo310 was susceptible to Fiji disease, which spread rapidly throughout the southern district. A similar situation occurred in the late 1970s with the widespread planting of Q90 in northern Queensland. The introduction of common rust and the build up of pachymetra root rot caused large yield losses. Reliance on one variety increases the risk of economic loss.

Chemical control

Chemicals are only used to control one disease in the Australian industry. The fungus that causes pineapple disease enters the cut ends of setts or billets at planting. Entry may also occur through growth cracks or cracks caused by billet injury. Several fungicides are available to control this disease, and they are

applied during planting by spray nozzles or dips mounted on planting machines. Complete coverage of the whole sett or billet is critical. Vegetable dyes can be used to gauge the effectiveness of spray coverage. Registered fungicides include a mercurial, and others belonging to triazole, and other chemical groups. Application of the fungicides in Queensland costs around $1 million each year. When planting sugarcane, some diseases can be spread in fungicide systems. It is important to empty and sterilise systems between plant sources. Fungicide should be changed at regular intervals (according to the label) to ensure freshness and activity.

MAJOR DISEASES OF SUGARCANE IN AUSTRALIA

Bacterial diseases

Ratoon stunting disease

Ratoon stunting disease (RSD) was probably present in Australia from the inception of the industry, but it was not until 1944 that BSES pathologists discovered it. Since then, it has been recognised worldwide as probably the most economically important disease of sugarcane. RSD is found in all districts in eastern Australia, but it has not been found in Western Australia. The incidence of the disease is associated with how well control measures are followed. Generally RSD is present in fewer than 5% of fields, but in a few districts RSD incidence is at least 40%. RSD causes yield losses from 5–60% depending on the susceptibility of the variety and the weather conditions (Figure 1). Yield losses are higher when the cane is suffering moisture stress. Over a range of conditions, the average yield losses are 15–20%.

RSD produces no external symptoms other than stunting, partially explaining the long period before its first detection. Diseased fields often have an 'up-and-down' appearance due to differing levels of stunting in adjacent stools. The only visual symptoms are red-orange dots or 'commas' in the vascular traces in the nodal tissue, which can be seen when stalks are sliced open with a sharp knife, and a faint pink discolouration of the growing point of young plants. These symptoms are not always present and some varieties can show similar symptoms when not infected.

The disease is caused by the bacterium *Clavibacter xyli* subsp. *xyli* Davis *et al.,* which infects the xylem (water transport) vessels of the sugarcane plant. The bacterium is rod shaped, typically with a slight bend and measures 0.25–0.5 µm by 1–4 µm. It is most readily found in sugarcane sap extracted by blowing compressed air through a stalk piece. The bacteria can be observed by an experienced person using a phase-contrast compound microscope at 1000 times magnification, a method that is used in many areas for rapid diagnosis of RSD. For large numbers of samples, the evaporative-binding enzyme-linked immunoassay (EB-EIA, often referred to as ELISA) is used to diagnose the disease. In this test, antibodies specific to the bacterium are combined with other specific ingredients, to give a colour reaction when bacteria are present. Two laboratories test approximately 50,000 samples each year using EB-EIA, for the industry and the BSES plant improvement program. For some research purposes, an even more sensitive test is available, based on

Figure 1. *Comparison of healthy and RSD infected cane (diseased on left, healthy on right).*

the polymerase chain reaction (PCR), in which millions of copies of bacterial DNA are produced from the few cells that may be present in a sample. The bacterium is extremely fastidious and slow growing on artificial culture media, taking 4–5 weeks to produce visible colonies.

Sampling for RSD diagnosis involves collecting at least 16–20 stalks per variety and block. Selecting stunted stools in a field can increase the chances of detecting the disease if it is present.

Since the RSD bacterium inhabits sugarcane sap, the primary methods of spread of the disease are by planting infected cuttings and by cutting implements. The bacterium is highly contagious and can be spread for many metres down a row after a planter or harvester cuts a diseased stalk or plant. Any implement that cuts the stalk or comes in contact with the freshly cut end of the sett or billet readily spreads RSD. Some of the more common implements that can spread RSD are cane knives, whole-stalk and billet planters, harvesters (base-cutter, butt lifter rollers, topper, chopper box, extractor fans and elevators), cane-stripping machines, haul-out vehicles used to transport billets to planters, and chain saws used to trim bundles of stalks. On planting machines, the recirculating fungicide spray system can carry the bacteria and spread the disease. An implement can be disinfected by first removing all soil and plant material with water and detergent under high pressure, then spraying it with a registered product containing 0.1% benzalkonium chloride. The disinfectant should be left in contact with the implement for 5 minutes before using or rinsing it.

New crops become infected with RSD during the first harvest, if diseased volunteer plants from the previous crop are present. The increasing practice of 'ploughout-replant' (when a new crop is planted within a few weeks of ploughing out the previous crop) has resulted in a sharp increase in the incidence of RSD, because without a fallow period it is almost impossible to prevent the growth of volunteers. Ploughout-replanting should only ever be practised when the previous crop is known to have been free of RSD, and seed should never be propagated on a ploughout-replant block.

RSD is not known to infect naturally any plant species other than sugarcane. The bacteria will survive for up to 1–2 days in soil and for 4–7 days on cutting implements. Some incidences of RSD have not been explained by the known methods of transmission, systemic infection of planting material or poor hygiene of machinery. These cases are not yet understood but appear to be restricted to certain localities. Disease-free (approved) seed and disinfection of cutting implements are the main control measures used to fight RSD. Control has been effective in most districts of the Australian sugar industry. In districts where there is either a high acceptance of approved seed or a high percentage of plant sources are inspected for RSD each year, the disease has been kept at low levels. Some districts with initially high incidence of RSD have steadily reduced the incidence of the disease by promoting approved seed and conducting plant-source inspections. Disease-free seed is produced for distribution to farmers by repeatedly hot-water treating nucleus or mother-plot cane.

Some varieties have partial resistance to RSD (e.g. H56-752) and disease spread is restricted in these varieties. Many highly productive varieties, such as Q158, are highly susceptible and may lose substantial yield. BSES has never actively selected varieties for resistance to RSD because other control strategies have been successful. Varieties are rated for resistance but this rating is only used as a guide for growers and CPPB staff.

Leaf scald

Leaf scald disease is found in most districts of the Queensland sugar industry. The disease has caused significant losses in some districts. Losses have not been quantified, but significant yield losses arise through the

death of a significant proportion of stalks that become unsuitable for milling.

Leaf scald is caused by the bacterium *Xanthomonas albilineans* (Ashby) Dowson, which is a motile, rod-shaped bacterium. It infects the xylem vessels of the sugarcane plant. The bacterium is relatively easy to isolate on simple nutrient agar, producing buff-yellow colonies after 3–5 days.

Leaf scald is characterised by the production of regular, long, narrow, and white-to-cream streaks on the leaves. The streaks follow the main veins on the leaf and death of the leaf tissue commences at the leaf margin and extends down the streak. These leaf streaks are not usually the first symptom observed, but they are the diagnostic symptom. The most striking symptoms are death of stalks with the leaves appearing scalded and turning inwards, chlorosis of patches or whole tops of shoots, and side shooting from the base of stalks (Figure 2). Side shoots and tillers formed at the base of diseased stalks usually show a range of streaking and chlorotic symptoms. Internally, infected stalks show red discolouration of the vascular bundles at the nodes. The reddening extends up to 10 mm either side of the nodes unlike RSD, which only forms a red dot or comma at the nodes. Leaf scald may remain latent for many months and only express symptoms when the plants suffer some stress. Sometimes infected plants will die with no obvious symptoms. However, in these cases, there is usually some evidence of side shooting at the base of stalks and symptoms on young tillers. The disease is diagnosed by examination of the symptoms, isolation of the bacteria on artificial media or serological or PCR-DNA assay.

Figure 2. *Death of susceptible varieties is the acute form of leaf scald.*

Leaf scald disease can be spread by wind-blown rain, particularly associated with extreme weather events such as cyclones. It can also be spread by infected planting material and by contaminated cutting implements such as planters and harvesters. Leaf scald bacteria can infect a range of grasses including *Paspalum* spp., *Brachiaria piligera, Imperata cylindrica* (blady grass), *Pennisetum purpureum, Panicum maximum* (Guinea grass), *Rottboellia cochinchinensis* (itch grass) and *Zea mays* (corn). These alternative hosts act as a reservoir for the disease and, whenever a susceptible sugarcane variety is planted in a district where leaf scald is endemic, it becomes infected. This occurs even if no disease has been found in sugarcane in the district for many years. Leaf-scald symptoms and yield losses are favoured by extremes of moisture and temperature.

Control of leaf scald is primarily by planting resistant varieties. All varieties in the

BSES plant improvement program are screened for resistance to leaf scald, and highly susceptible varieties are discarded. Crosses between susceptible parent varieties are restricted to reduce the proportion of susceptible clones coming through the improvement program. Recently varieties have been genetically modified with genes that confer resistance to leaf scald. The genetically modified plants are undergoing field-testing, but have not been grown commercially. Disease-free (approved) seed and disinfection of cutting implements can also assist in the control of leaf scald. The cold-soak, long-hot-water treatment is used to obtain disease-free planting material.

Red stripe (Top rot)

Red stripe and top rot are two forms of the same disease. The disease is found in all districts of the Australian sugar industry, including the Ord River Irrigation Area where it has caused severe damage. It is particularly severe in fields that are planted to sugarcane for the first time.

Red stripe (top rot) is caused by the bacterium, *Acidovorax avenae* subsp. *avenae* (Manns) Willems *et al.* It is a motile rod and is easy to isolate on simple nutrient agar.

The red stripe phase of this disease is characterised by long, narrow, uniform, red stripes on the leaf blade and leaf sheath. The stripes vary in width from 0.5–4 mm and can coalesce to form large red patches on leaves. The streaks are first seen as watery green stripes and they usually commence toward the base of the leaves. The bacteria ooze from the stomata when humidity is high and white flakes of dried bacterial ooze are often seen on the lower surface of leaves.

Top rot is the more damaging form of the disease and is characterised by death of the younger leaves. The spindle leaf dies and is easily pulled out of the heart of the plant, giving a characteristic rotten odour. In badly affected fields, this odour can be detected from the edge of the field. Internally the stalk tissue in the upper nodes and at the growing point is water-soaked, later turning brown to red, and has sunken cavities. Affected stalks will die or side-shoot from lower down the stalk. In severe cases, top rot can cause significant yield losses.

The bacterium which causes red stripe (top rot) is spread by wind-blown rain. Severe outbreaks of red stripe (top rot) have occurred in fields that have never grown sugarcane before, indicating that the bacterium occurs on other grasses; some other hosts have been reported, such as sorghum, maize and millet. Hot, humid conditions and rapid cane growth favour the disease; thus it is usually seen during the wet season.

The only control measure for red stripe (top rot) is the planting of resistant varieties. There is no active screening of clones for resistance to this disease, but clones that show severe symptoms in plant improvement trials are noted. Susceptible varieties should not be planted in fields that have a history of red stripe (top rot), or should not be planted at a time when young plants will be exposed to conditions conducive to the disease.

Fungal diseases

Smut

Sugarcane smut was discovered for the first time in Australia in July 1998 in the Ord River Irrigation Area in Western Australia. Extensive surveys conducted since then in eastern Australia have failed to locate the disease there. Smut is one of the most important diseases of sugarcane worldwide and its occurrence leads to the discard of many commercial varieties and breeding canes.

The causal agent is the fungus *Ustilago scitaminea* Sydow and P. Sydow, which only infects sugarcane and some closely related grasses. The fungus produces masses of brown-black spores in a whip-like structure produced on infected plants. Smut whips vary in length from a few centimetres to about 1.5 m and arise from the growing point

of a shoot. Infected plants can have a grassy appearance with many thin shoots. In susceptible varieties, the disease can be very severe, with affected stools producing no millable cane, leading to large economic losses.

The whip is made up of a central pithy core surrounded by a layer of the brown-black spores, which in turn are covered by a silvery membrane. As the whip matures, the membrane breaks, releasing the spores, which are blown by the wind to infect healthy cane. Individual whips produce billions of spores each day for up to 3 months. Smut only infects cane through dormant or young shooting buds.

Smut has been controlled well in other countries through the breeding and planting of resistant varieties. The introduction of smut usually leads initially to direct yield losses, which are magnified by the necessity to remove susceptible varieties from cultivation quickly. Ongoing long-term losses arise from the need to discard susceptible parent varieties and progeny from the plant improvement program, thus slowing the rate of genetic improvement from breeding. Trials to screen varieties from Queensland and New South Wales for resistance to smut are being conducted in Indonesia and Western Australia. These trials include commercial varieties, promising clones in the plant improvement program and parent varieties. This will allow pre-emptive breeding to prepare the eastern coast industries for the disease, should an outbreak occur in Queensland or New South Wales.

Cane that is infected with smut can be rendered virtually disease-free by hot-water treatment at 50°C for 30 minutes. Overseas research has shown that the addition of certain fungicides to the hot-water treatment tank will partially protect the cane from reinfection when it is planted in infested soil. Fungicide alone does not provide adequate disease control.

In some countries, roguing of diseased fields is undertaken in an attempt to reduce inoculum levels. This may provide some short-term benefit where the cost of labour is cheap, but smut spreads rapidly and roguing is not a commercial or long-term disease control option in Australia.

Pachymetra root rot

The symptoms of pachymetra root rot were first recognised in 1967 on a cane farm in Babinda. However, it was not until 1981 that the causal agent was cultured and the symptoms reproduced in a glasshouse pot experiment. The disease has now been found in many parts of Queensland, but not in New South Wales, the Ord River Irrigation Area, or anywhere else around the world. Affected areas include many parts of northern Queensland, the Herbert River, Central District (Proserpine, Mackay and Sarina) and some parts of southern Queensland (Bundaberg and Maryborough).

The fungus, *Pachymetra chaunorhiza* Croft and Dick causes pachymetra root rot. *P. chaunorhiza* may be isolated from roots at an early infection stage on specific culture media.

The disease principally affects the larger primary and secondary roots of the cane plant. After gaining entry to the roots, the pathogen gives rise to a watery rot leaving only the root epidermis intact. Affected roots are flaccid and filled with the relatively large (30–65 µm) fungal spores, which have large conical projections. As the roots decay, these spores are released back into the soil and will infect other nearby roots. In susceptible varieties, the extent of rotting leads to a very poor and debilitated root system. Affected crops grow poorly and stools may tip out of the ground due to inadequate anchorage from the poor root system. When harvesting a crop with extensive 'stool tipping', excessive soil and roots ('extraneous matter') are sent to the mill. Ratoon crops fail through loss of stools. Yield reductions may be up to 40% in highly susceptible varieties.

Pachymetra root rot does not move rapidly between fields or districts, and has only been

observed on land growing sugarcane. Build up of the disease has occurred after 'new land' in affected districts is planted with susceptible varieties. This may take 5–10 years or longer, depending on the situation. Spread occurs through soil on vehicles, machinery, or the base of stalks used as planting material. Movement in water is likely to be minimal, except when there is heavy water erosion of soil.

Resistant varieties are the main form of control for pachymetra root rot. Some Australian varieties are quite resistant while resistant parent canes are also available for breeding purposes. All varieties in advanced stages of the plant improvement program are screened for resistance, and highly susceptible clones are discarded in badly affected districts. Also, the resistance rating of parent canes is taken into account when choosing crosses. Crosses must contain at least one resistant parent, which increases the likelihood of resistant progeny. Intensity of pachymetra root rot can be maintained at an acceptable level without excessive yield losses by growing resistant varieties, or by rotating resistant varieties with intermediate to susceptible varieties.

Fallowing fields for 6–12 months is not sufficient to reduce the level of pachymetra root rot significantly, because the spores can survive for up to 5 years. Ploughing fields does not reduce spore viability. Cultivation can dilute spore concentrations in soil, by spreading spores out of the row area where most roots are present and into the inter-row. No economic forms of chemical control are available.

Yellow spot

Yellow spot is a disease of the wet tropics, being favoured by warm wet conditions. As a result, it is regularly severe between Tully and Gordonvale, although it can occur in most districts.

The fungus *Mycovellosiella koepkei* (Kruger) Deighton causes yellow spot. It grows slowly on normal nutrient agar.

As the name suggests, yellow spot causes a yellow lesion on the leaf blade of susceptible varieties. Lesions are irregularly shaped and vary in colour and size depending on the sugarcane variety. As lesions age, their size increases from minute chlorotic spots to lesions with a range of colourings and up to 10 mm or more. Lesion colour may be

Figure 3. *Crops affected by yellow spot take on a yellow-brown appearance and suffer a significant loss in green leaf tissue.*

yellow, brick red, red-brown or even brown-black. The canopy of affected crops may appear red-brown, or 'dirty', in colouration (Figure 3). Symptoms are seen after the onset of the wet season and usually peak from the end of April to May, depending on the amount and distribution of rainfall. The onset of cool and dry conditions in winter usually signals the end of the disease epidemic. Spores are produced on the underside of affected leaves; production is associated with fungal growth on the lower leaf surface giving an appearance with some similarities to the down of downy mildew.

Yellow spot is spread by the dispersal of spores from infected crops by wind and water splash. Moisture and warm temperatures favour infection.

Losses caused by yellow spot have not been adequately quantified in Australia, but overseas research suggests they may be over 20% in susceptible varieties, with CCS losses of over two units. CCS reduction results from the fungus reducing the ability of leaves to photosynthesise at a time when sugar accumulation occurs in the maturing crop.

Control of yellow spot relies on the growing of resistant varieties. Varieties are screened for resistance in specific field trials and in the plant improvement program. Most commercial canes in northern Queensland possess some degree of resistance to the disease, though many still show moderate disease levels in favourable seasons.

Blocks of cane with moderate to high disease levels should not be harvested early in the season because the disease causes sugar content to fall. By delaying harvest to mid-season, there is time for sugar levels to rise to acceptable levels.

Common rust and orange rust

There are two forms of rust in Australia, one known as common rust, and the other as orange rust. Orange rust is normally a minor disease seen on occasions throughout Queensland but more frequently in the north. However, in 1999–2000 the disease caused severe leaf damage to the widely grown variety Q124. Common rust is regularly seen in all districts.

Both of these fungal diseases are caused by species of *Puccinia*: common rust by *P. melanocephala* H. & P. Sydow and orange rust by *P. kuehnii* Butler.

The first symptoms of common rust are small, elongated, chlorotic spots on the younger leaves. These soon develop into red-brown to brown, elongated, narrow lesions (Figure 4). The fungus ruptures the lower leaf-surface to cause pustules from which the spores are released. Rubbing a hand along the leaf surface leaves a brown dusty colouration on the fingers as the spores adhere to the skin. Very little green leaf tissue remains in severely diseased crops. The ruptured leaves allow water to escape from the plant, leading to moisture stress.

The rusty brown appearance of diseased crops is readily identified at a distance. Warm, dry days and cool nights with dew are ideal conditions for common rust. The disease is usually more severe in late spring and early summer (September-December), and tends to disappear with the onset of summer rains, though favourable conditions

Figure 4. *Common rust.*

later may lead to further disease development. Older crops are more resistant to rust than younger crops, and this is another reason for less disease as the season progresses.

Orange rust can be distinguished from common rust by several features. Lesions are an orange colour, they tend to occur in clusters, and are not evenly spaced over the leaf blade (Figure 5). Orange rust is more pronounced in humid summer weather. Microscopic features of the two fungi allow an identification to be confirmed.

Wind-blown spores transmit both rust diseases. Because of their small size, rust spores may travel great distances. When common rust entered Australia in 1978, the disease spread from Cairns to New South Wales in one season. Spores land on the leaves, germinate, and invade through the leaf stomata.

Orange rust is normally a minor disease and most commercial canes are highly resistant but, in early 2000, extensive infection of the very important variety Q124 became evident in the Central and Northern Districts (Figure 6). This epidemic is to be monitored and resistance trials are to be initiated as a contingency.

Common rust has been much more important—it regularly infects a number of commercial canes leading to some losses. Resistant varieties are the cornerstone of disease control. Selection for yield in the plant improvement program usually leads to the discard of highly susceptible clones before they proceed very far in the plant improvement program. No specific resistance screening trials are conducted.

Pineapple disease

Pineapple disease is caused by the soil-borne fungus *Ceratocystis paradoxa* (Dade) Moreau and occurs throughout the Australian industry. It is present in the soil where it can live on decaying plant tissues and infect planting material during crop germination. Infection occurs through the cut ends of setts and through any wounds to the stalk surface.

Figure 5. *Leaf symptoms of orange rust.*

Figure 6. *A crop of Q124 heavily infected with orange rust.*

Symptoms of pineapple disease are poor crop germination and rotting of planting material in the ground. The name comes from the smell similar to rotting pineapples, which is released when affected setts are split open. Infection also leads to a reddening of internal stalk tissues. In the advanced stages of infections, abundant black spores are produced in the rotting setts. The pathogen produces a toxin that may kill the growing shoot, particularly when the shoot is reliant on the sett for nutrients and moisture. Pineapple disease is favoured by low soil temperatures, poor soil moisture conditions (either too dry or too moist), and by poor soil preparation (poor soil-sett contact).

The production of well-grown planting material without growth cracks, internal pipes and other stalk damage is an essential element in control of the disease. Blades on a whole-stalk planter should be sharp and well adjusted, as should the blades on a harvester used to cut billets for planting. Rubber rollers are being developed for these harvesters to reduce bruising of billets. The nodes of the sugarcane sett slow the passage of the disease further into the sett, so the use of two or three-bud, rather than one-bud, setts assists in obtaining better germination.

It is essential that a registered, preventative fungicide be applied to the whole billet surface, particularly the cut ends. Without complete coverage, the pathogen may invade sett tissues leading to germination failure. Running a suitable vegetable dye through the sprays on the planter enables spray coverage to be assessed. Nozzles usually can be redirected or replaced to ensure good coverage. It is important that spray coverage is checked each year before planting begins. A range of fungicides is registered, including mercurial compounds, triazoles and several other types of chemicals. The mercurial stimulates germination, an advantage when conditions favour pineapple disease. Even fungicides do not provide adequate protection to damaged billets.

A bare fallow will reduce the concentration of the fungus in the soil. Replanting shortly after ploughing in the previous crop can lead to high levels of disease, because the fungus can build up on decaying residues of the previous crop.

Red rot

Red rot is a disease of the cane stalk and the stool. It occurs in all areas, with severe disease occurring periodically in areas affected by moisture stress.

Red rot is caused by the fungus *Glomerella tucumanensis* (Spegazzini) Arx and Müller, which is isolated relatively easily from diseased tissue on artificial growth media.

The fungus gains entry to standing stalks through stalk wounds and leaf scars. Moisture stress, such as from drought or flood, predisposes the stalks to infection. In susceptible varieties, it spreads up and down the stalk and disrupts normal stalk function. External symptoms may be reddish to purplish colouration of the rind. When stalks are sliced longitudinally, red internal stalk symptoms will be apparent, with characteristic white transverse patches (at right angles to the long axis of the stalk). These white patches may be used to distinguish the disease from other stalk rots that lead to stalk reddening. The affected tissues have a starchy smell. Rapid death of stalks with total loss of yield sometimes occurs. In resistant varieties, rotting of internal stalk tissues is restricted.

Red rot also commonly infects the mid-rib of leaves leading to a bright red lesion. Infection usually occurs where sugarcane planthoppers (*Perkinsiella saccharicida* Kirkaldy) have laid eggs. These lesions have little effect on cane growth, though they may provide an inoculum source for the infection of stalks. In a few cases, red rot will also infect setts of planting material and ratoon stubble.

Spores produced in leaf lesions, infested stalk material, crop debris and infested soil transmit the disease. Spores are spread by wind, rain, heavy dews, and irrigation water,

and in infected planting material. Rain can wash spores from mid-rib lesions on to leaf scars, where infection may take place under suitable conditions. Pathogen entry points are leaf scars, stalk growth rings, root primordia, buds, growth cracks or injury caused by borers.

Disease control relies on the planting of resistant varieties. Promising clones are screened late in the plant improvement program to ensure that commercial varieties have sufficient resistance to the disease.

Good crop management can also reduce the chances of disease development. Providing optimal soil moisture by irrigation will prevent drought stress and minimise disease severity. In wet areas, adequate drainage also minimises disease occurrence. If a crop is badly diseased, rapid harvest is important, because CCS drops quickly when the disease is severe. The production of dextrans and other contaminants makes sugar production from diseased crops difficult, and the mill may place penalties on severely diseased cane.

Eye spot

Eye spot causes sporadic epidemics with significant yield losses in susceptible varieties in Australia. It is favoured by cool and humid conditions. A recent outbreak occurred in Q159 in Mackay and northern New South Wales. Eye spot has also occurred in northern Queensland and led to the demise of Q101 in the mid-1970s.

Eye spot is caused by the fungus *Bipolaris sacchari* (Butler) Shoemaker, which can be cultured easily on nutrient agar. Spores are produced in large numbers on leaf lesions and dispersed by wind or rain.

The symptoms of eye spot are very characteristic. Initial symptoms are minute water-soaked spots on the young leaves. These develop into small reddish-brown lesions that soon increase in size and elongate. The pathogen produces a toxin causing a halo to develop around the growing lesions. As lesions reach their final size (0.5–4.0 mm long, 0.5–2.0 mm wide), movement of water along the leaf carries the toxin in a stream toward the leaf margin. This gives rise to reddish-brown 'runners', with the affected leaf tissue dying. The presence of many lesions and runners leads to extensive leaf tissue death ('firing') and reduced yield. The runners are not produced with any other sugarcane disease and are very useful for diagnosis. Froghoppers, an insect, inject a toxin into the leaf of sugarcane and cause a similar runner on leaves, but there is no lesion at the base of the runner.

Resistant varieties are the sole control measure for the disease. As the disease occurs sporadically, there is no necessity for routine resistance screening trials. Observations made during the plant improvement program are usually sufficient to identify highly susceptible clones, but occasionally, susceptible varieties have been released.

Pokkah boeng

Pokkah boeng is a peculiar name for a disease, derived from an Indonesian word meaning 'tangle top', a fitting description for one of the symptoms of the disease. Pokkah boeng occurs in all cane growing districts of Australia, but it is usually of minor importance.

The disease is caused by the fungi, *Fusarium monoliforme* Sheldon, (teleomorph *Gibberella fujikuroi* (Sawada) Wollenweber) and *Fusarium subglutinans* (Wollenweber and Reiking) Nelson, Tousson & Marasas (teleomorph *Gibberella subglutinans* (Edwards) Nelson, Tousson & Marasas).

Symptoms of pokkah boeng vary. In minor cases, it causes chlorosis at the base of leaves, and occasionally mid-way along the leaf blade. This chlorosis has a distinctive 'granular' (not uniform) appearance. Increased severity leads to twisting and distortion of leaves. When a number of leaves are affected together, young leaves may not separate fully during development. Sometimes the

twisting is accompanied by 'knife cuts' along the leaf margin. These result when fungal invasion kills cells so that they cannot expand with the remaining healthy cells. The leaf literally pulls itself apart, leading to splits in the leaf running at right angles to the margin. Whole sections of the leaf may fail to develop, giving the young shoot the appearance of having been bitten off by an animal. In very severe cases, the disease may kill the growing point, leading to shoot death. Knife-cut-like symptoms may also be found on stalks. When these occur in series on the rind tissue, ladder-like lesions occur. Increased severity leads to the bending or breakage of affected stalks.

Pokkah boeng is favoured by the presence of lush growth (with associated susceptible tissues). For this reason, this disease is more common in fast growing crops early in the wet season, or at other times when growing conditions are ideal.

There is usually no need for any form of disease control. Except when shoot death occurs, crops grow away from pokkah boeng leaving no significant yield loss. Highly susceptible clones are discarded if identified in the plant improvement program.

Viral diseases

Chlorotic streak

The cause of chlorotic streak is unknown, but it may be a virus. It occurs in every area from Mossman to Harwood, often associated with wet and poorly drained fields. Drier regions generally have lower incidence of the disease.

The main symptoms of chlorotic streak are the yellow to white streaks on the leaf blade and, to a lesser extent, on the midrib and leaf sheath. They follow the general direction of the vascular bundles and vary in length from quite short to the full length of the blade. Streaks vary in width from very narrow to 6 mm. This irregularity and the ill-defined border are diagnostic for the disease. The older streaks are usually yellow and more obvious than younger streaks. They also become partly necrotic, with the necrosis characteristically occurring in the centre of the streak. This is different to leaf scald, where the death of tissue starts at the edge of the leaf and extends down the leaf streak. Symptoms of chlorotic streak are notoriously ephemeral and, although a few varieties may show symptoms throughout the year, in most varieties they come and go for no known reason. Internally, diseased stalks often show one to several reddened vascular bundles through the nodes, which can be distinguished by an experienced person from the symptoms of RSD. Other symptoms are wilting even when soil moisture is adequate, reduced and weakened germination and ratooning, and yield loss.

Yields can be reduced by 70% in susceptible varieties. Losses of 1% for every 5% increase in disease incidence have been measured in the Herbert district.

Chlorotic streak is transmitted in soil by water. It is common in flood-prone areas and areas with poor drainage. A heavy wet season will often lead to chlorotic streak in crops on normally disease-free slopes. Within individual fields, it coincides with the wetter areas. Transmission is favoured by ratooning cane when the soil has high moisture content. In recent years, there has been an increase in the practice of re-cycling irrigation tail waters to irrigate fields, leading to a marked increase in the incidence of chlorotic streak in these blocks.

It is suspected that chlorotic streak infects grasses, as similar symptoms have been noted on a number of grasses.

Chlorotic streak is transmitted in planting material. The planting of diseased setts can be disastrous when conditions are favourable for the disease. Germination is poor and slow, and stooling is reduced. Ratoons are also weakened, so much so that in some localities the crop cycle has to be shortened due to failure of older ratoons.

Provision of disease-free seed is important in chlorotic streak control. This disease is so

widespread on the wetlands of the mill areas north of Townsville that in many years, and nearly always at Macknade, disease-free plants are in very short supply. The alternative is to treat the planting material in water at 50°C for 30 minutes. This is quite effective in freeing the plants from the disease and usually gives a bonus in improved germination. It is important that progeny from this hot-water treatment is planted in a disease-free locality.

Improvement in drainage is essential for reducing losses from chlorotic streak. This also results in a general improvement in the crop and greater ease of cultivation and harvesting. The use of re-cycled tail waters for irrigation should be avoided when irrigating potential plant sources.

There is some range of resistance in present varieties but not sufficient for resistance to be actively bred for control of the disease. Some varieties have been too susceptible for planting in areas prone to chlorotic streak disease and warnings have been issued.

Fiji disease

Fiji disease is caused by *Fiji disease virus* (FDV) (genus *Fijivirus,* family Reoviridae). The FDV particles consist of double-shelled, icosahedral structures about 70 nm in diameter, each with 12 protrusions (spikes) at their vertices.

The definitive symptom of Fiji disease is raised whitish galls on the underside of the leaf blade and midrib. The galls normally occur longitudinally in the large vascular bundles and vary considerably in size—from those only visible with magnification to some up to 0.5 m long. The colour can vary from creamy white to green, while the surface of the gall is usually but not always smooth and unbroken. If the epidermis is ruptured, the appearance is brown and granular. Galls usually remain evident on dead leaves, allowing identification of diseased plants after they have died. Galls are solid when pressed with a fingernail. Galls are due to cell proliferation in the phloem and xylem that arise during tissue differentiation near the meristem. Galls contain large numbers of viral particles.

Often, galls similar to Fiji disease ('pseudo-Fiji') can be found on sugarcane leaves where the disease is not present. They are usually small, the gall surface is not as smooth as for Fiji galls, and they are more triangular in cross section. In addition, a cross section of a Fiji gall will show a white core when examined with a good quality hand lens. This is absent from pseudo-galls. Pseudo-galls collapse when pressed with a fingernail, whereas true Fiji galls are firm.

The first symptom of Fiji disease to appear on a newly infected plant is one or more galls on an otherwise normal shoot. As the disease progresses, stalk development slows down and successive leaves become shorter, harsher and stiffer, the whole top develops a fan-like appearance and, in more severe cases, looks as though an animal has bitten it off (Figure 7). Diseased leaves are usually a somewhat darker green than normal. This,

Figure 7. *The effect of Fiji disease on the growth of susceptible varieties: stunting can lead to major yield losses.*

together with the stunted top, is usually the first sign to attract the attention of the inspector who then looks for the confirmatory galls. Fiji disease virus can be detected with very high sensitivity in non-symptomatic plants with a molecular test (RT-PCR) that is used in quarantine to supplement visual indexing.

When diseased stools are ratooned, or when setts from a diseased stalk are planted, the resultant plants will exhibit varying degrees of stunting from a grass-like stool of stiff dark leaves to one that may produce some millable cane, depending on the variety concerned.

Recovery from Fiji disease has never been observed, although apparent recovery may occur when only part of a stool becomes infected and that part dies out, or fails to ratoon, thus leaving only the healthy portion. All the buds on a newly infected stalk may not contain the virus, and consequently some can give rise to healthy plants.

Fiji disease can be transmitted by planting diseased setts or by infected planthoppers during feeding. In Australia, sugarcane planthopper, *Perkinsiella saccharicida* Kirkaldy, is the vector. When *P. saccharicida* breeds on Fiji-diseased plants about 5–40% of the population acquires the virus and become capable of infecting healthy plants. Adults, both male and female, and nymphs can transmit the disease for their whole life. The insect is a relatively inefficient vector of Fiji disease, but can spread it quickly when present in high populations and when diseased plants are common. The population size of *P. saccharicida* cycles naturally through years from rare to swarming, for reasons that are not well understood but include sugarcane variety, weather conditions, growing conditions and presence of natural enemies. The incubation period, that is the time required for symptoms to show after infection takes place, varies considerably depending on the sugarcane variety, the rate of growth of the crop and the age of the plant. Under ideal conditions in insectary trials, it may be as low as 15 days, but under harsh conditions in the field it can be many months. Cane infected late in the summer may not show symptoms until after ratooning, even though conditions for growth are excellent. Planthoppers have also been observed to have different feeding patterns on some varieties, and the total feeding time spent by the insect feeding from the phloem appears to be correlated with susceptibility of that variety to Fiji disease.

Fiji disease epidemics occurred periodically in southern Queensland and New South Wales in the 1900s. In Bundaberg, the number of infected stools reached very high numbers in the 1970s, leading to serious losses, but no diseased plants have been seen since the 1980s. Fiji disease is still active in some mill areas south of Bundaberg and active control programs are necessary. Fiji disease was recorded for the first time in Plane Creek in 1981 and not long afterwards was found in Mackay. An intensive management program has resulted in no reports of the disease since the 1980s. One diseased plant found in Proserpine was destroyed and no further findings have been reported. Fiji disease is not present in north Queensland, although *P. saccharicida* is. Fiji disease is present in Papua New Guinea and a number of islands in the region of Papua New Guinea and Fiji. It has also been reported from Malagasy, Thailand, Malaysia and the Philippines.

Resistant varieties are critical for control of Fiji disease. Susceptible varieties will not be released in the Central and Southern Districts and, in New South Wales, the use of intermediate varieties is restricted. In plant improvement for these areas, crosses must contain at least one resistant parent and all seedlings in advanced stages must be screened for resistance in field trials. These restrictions do not apply for north Queensland. When an epidemic is developing, strict control measures must be taken before varieties with intermediate resistance become heavily diseased and

economic losses become substantial. At this point if unchecked, an epidemic would progress very rapidly.

Disease-free planting material is critical in the control of Fiji disease, since an epidemic escalates rapidly when diseased cane is planted out, compared to when planthoppers are the sole means of spread. Approved seed programs may be escalated when Fiji disease is present, and it may be necessary to inspect all prospective plant sources.

When just a few diseased plants have been detected, roguing diseased stools can be effective. However, this becomes futile as epidemic progresses, since many plants will be diseased but non-symptomatic. It also becomes uneconomic due to the labour cost. At this point, more stringent measures are required, such as destruction of heavily infected crops and restrictions on planting or ratooning susceptible varieties. Planting in autumn rather than spring can reduce the spread of Fiji disease in moderately susceptible varieties.

The ubiquitous presence of *P. saccharicida* in all canegrowing areas means that quarantine of cane movement (see quarantine chapter) is critical to prevent Fiji disease virus from entering north Queensland and re-entering the areas from which it has possibly been eradicated.

Striate mosaic

Recent research has shown the cause of striate mosaic to be a double-stranded RNA virus called sugarcane striate mosaic-associated virus.

Despite a statewide search for this disease, it has been found only in the Burdekin district. There it occurs on both sides of the Burdekin river. Striate mosaic is usually associated with poorer growth areas resulting from excessive sand, shallow soil, reclaimed watercourses and uneven irrigation, but the relationship of the disease with a specific environmental factor has not been established. It occurs in patches of variable size and shape, except when diseased setts are planted. Patches up to about 1 ha have been found. Striate mosaic has so far been reliably reported only from Queensland.

The specific symptoms are the short, fine striations on the leaves, approximately 0.5 mm wide and 0.5–2.0 mm long, that can be just separated by the naked eye. Striations vary in number from a few to so many that the greater part of the leaf blade is covered. They are a lighter green than the normal blade and show first on the youngest exposed leaf. Their best development occurs as the leaf expands and, since they are difficult or impossible to find as the leaf matures and ages, they are less conspicuous during periods of slow growth than when the cane is growing rapidly. The best time to find the disease is in late winter to early spring. Symptoms are difficult to find when temperatures exceed 30°C. They occur in smaller numbers along the major vascular bundles than in the tissue between, so often give a marked striping effect on the younger leaves, or even a yellowing to the entire top. In varieties with reddish stalks, striations can be clearly seen on the rind of the stalk. In Q96, the stalk develops a marked restriction around the node giving the internodes a bulbous appearance.

The highly susceptible variety Q96 at times does not show clear symptoms, the only signs of disease being severely stunted growth and death of stools with a general yellowing of the canopy.

Striate mosaic is sett-transmitted and recovery from the disease has never been recorded. It can be transmitted mechanically by the needle-prick technique sometimes used for mosaic, but the proportion of plants infected by this method is very low. Growing healthy plants in infested soil has successfully transmitted it, as long as the plants and soil are maintained at 25–30°C. Above these temperatures, plants do not express symptoms.

Striate mosaic causes severe stunting, poor stooling and loss of crop in patches in Q96. Affected patches become overgrown with

weeds and can cause farmers to prematurely plough out blocks.

Currently the only economic control measure is the use of resistant varieties. However, varieties thought to be highly resistant have succumbed to the disease when planted after or near to the highly susceptible variety Q96.

Soil fumigation with methyl bromide has eliminated the disease for at least one crop cycle, but this is not economic and methyl bromide has been withdrawn from use because of environmental concerns.

Sugarcane mosaic

Sugarcane mosaic is caused by the *Sugarcane mosaic virus* (SCMV), a member of the potyvirus group of plant viruses. These are characterised by a long flexuous rod shape when viewed with an electron microscope.

There are several strains of SCMV and closely related virus species overseas, but only strain A of sugarcane mosaic has been recorded in Australia. Strain A is considered a relatively mild form of the SCMV virus.

Mosaic derives its name from the mosaic or mottled pattern of contrasting shades of green, or patches of normal green surrounded by paler green or yellowish chlorotic areas. The symptoms are most evident in the young, rapidly growing leaves and can often be seen in the spindle. Older leaves can appear more normal as the chlorotic areas tend to become more normal green with age. The proportion of the leaf blade that becomes chlorotic varies greatly between cultivars, sometimes resulting in the appearance of scattered elongated yellowish stripes, but usually the chlorotic patches dominate and are relatively uniformly distributed over the leaf blade. Chlorotic areas may also be present on the leaf sheath and stalk (particularly on noble canes), but generally stalk symptoms are not common on the commercial hybrid cultivars.

Aphids and infected seed cane are the most important means of spread of SCMV in the field. The principal vector is the corn aphid, *Rhopalosiphum maidis* (Fitch), although other aphids, including *Aphis gossypii* Glover, can also transmit the virus. The virus is transmitted in a non-persistent manner, that is the virus does not live or multiply in the insect, and can be transmitted only for a few hours after feeding on an infected plant. Mosaic can infect a range of grasses and can be carried from outside cane fields into cane crops by aphids. Harvesters and other machinery do not spread mosaic, although plants can be infected artificially in research by scouring their leaves with abrasive mixed with diseased leaf juice.

Infection of sugarcane by SCMV results in stunting of affected plants and yield losses depend on the extent of stunting and the proportion of plants infected. Heavy yield losses can occur. Germination and ratooning ability are adversely affected.

Sugarcane mosaic is an important disease of sugarcane in Australia and is one of the most widely distributed diseases of sugarcane worldwide. Mosaic has been reported in all mill areas, although it was not recorded at Rocky Point until 1976. Mosaic has not been found north of Bundaberg for many years. It is often difficult to eradicate in localities where cane growth is good and weeds, that are alternative hosts, are prevalent in fields and gullies. It can also spread from certain other crops. The Isis region near Bundaberg suffered a SCMV epidemic in the mid-1980s due to large plantings of the agronomically superior but mosaic-susceptible varieties Q95 and Q137. The effect of the epidemic was accentuated by severe drought, and resulted in losses of up to 40% in yield in some fields. As Q124 became dominant in these areas in the late 1990s, it developed widespread infection, although incidence within a crop remained low. Mosaic has spread into Q124 in areas of Bundaberg adjacent to Isis, and both Bundaberg and Isis have developed control strategies to avert significant yield losses.

The current strategies for control of mosaic are resistant varieties and the provision of disease-free planting material. Agronomically promising varieties are assessed in trials at the BSES Pathology Farm for susceptibility to mosaic.

BSES has developed the use of a 'synthetic' resistance gene for mosaic to be incorporated into the sugarcane plant genome by genetic engineering, but none of this cane has been grown commercially. This technique has the potential to 'cure' SCMV susceptibility in agronomically superior canes.

Nematodes

Nematodes are microscopic, worm-like animals that are an important part of animal diversity in the soil. Among the multitude of species, some break down dead organic matter and contribute to nutrient recycling, some attack fungi, bacteria or other nematodes and may have a role in disease suppression, whilst others are serious plant pathogens. Crop damage occurs when the population density of a pathogenic species is high enough to restrict root development, in growing conditions that are sub-optimal for the cane crop.

Losses from nematodes can be severe on sandy soils in the Bundaberg area and control is required there. The common industry understanding, derived from experiments with commercial doses of non-volatile nematicides throughout Queensland in the late 1970s to early 1980s, has been that losses are minor elsewhere. However, recent research has shown that most cane fields in all districts are infested with at least five species of pathogenic nematodes, with damaging species often at high population densities. Experimentally, yield responses of 5–20% were obtained consistently on clay loam and clay soils by repeated applications of nematicides. It is now understood that the earlier experiments and the commercial treatments achieve only partial and temporary nematode control, so they will be efficacious only on sandy soils or with ideal application conditions. It seems that nematodes have insidious and widespread effects that generally are not recognised.

Lesion nematodes [commonly *Pratylenchus zeae* Graham and *P. brachyurus* (Godfrey)] are present in all fields. These attack the root and move into it and through it, destroying cells while feeding and laying eggs. Fresh lesions are reddish purple, becoming necrotic and purplish-black, presumably through the action of bacteria and fungi that enter the lesions. Roots become girdled by the spreading lesions, so that badly affected root systems are reduced in mass and are dark in colour, typical of most cane root systems. These symptoms are not directly diagnostic for nematodes, rather they are associated with the 'yield decline' that occurs when sugarcane is grown in monoculture. Losses are much more widespread than commonly realised and often would be 10–20% even on loamy soils.

Root knot nematodes [commonly *Meloidogyne javanica* (Treub) and *M. incognita* (Kofoid and White)] are widespread, but are particularly important on sandy soils. They produce the most distinctive of nematode symptoms. They penetrate the root tip and stay in that location to feed and reproduce. The root tip swells and grows no further, with swellings and galls obvious on the tips of young sett and shoot roots. Root elongation ceases and with a high nematode population, root systems are truncated severely. Growers on the sandy soils of southern Queensland have been aware since the 1980s of the yield improvements obtained by addressing root knot nematode with nematicide, but nematode damage can occur on sandy soils in any area.

Several species including stubby root nematodes (*Paratrichodorus minor* Colbran), dagger nematodes (*Xiphinema* spp.) and needle nematodes (*Paralongidorus* spp.) attack and move between root tips, causing swelling, deformity and stunting of the root.

Lateral roots produced behind a damaged root tip may also be attacked and stunted.

Nematode damage to roots causes slow and poor development of shoots and stools, giving a diseased crop an open appearance. Yield losses may be as much as 20–50% under poor growing conditions and severe attack, but very commonly the crop damage is less than this and is difficult to identify from crop appearance. Also, these symptoms can be due to various factors besides nematodes. Diagnosis of a nematode problem will involve excavating and washing roots carefully to allow close examination for the symptoms described above. Often, symptoms of more than one nematode type can be seen. A soil sample should be provided to BSES for identification and counting of nematode species. A nematode count provides only a general guide to what damage may be expected, since other crop and environmental factors are important in determining tolerance of the crop. Accuracy of this count depends on following sampling and storage procedures carefully. Using a shovel or probe, at least 10 samples of soil and roots are taken from against a stool, between 50 and 250 mm depth. The soil is bulked together in a bucket and mixed. A 1 L sub-sample with all of the roots should be placed in a plastic bag or other suitable container, kept cool at all times, packaged securely and despatched promptly.

Populations of nematodes cycle in size with the availability of roots on which to feed and reproduce. Populations decline through a fallow of at least 3 months, but 2–3 years of fallow are required to reduce populations to negligible levels. Most sugarcane nematodes have a wide host range, so the presence of weeds or alternative crops will often lead to carry-over to the new crop. After planting, populations rise to their maximum about 6–10 months later and then decline towards harvest. Populations tend to be lower during ratoon crops, but maintain the same cycle.

Present options for control of nematodes are limited to nematicides. Their use normally is limited to sandy soils, where crops are less able to tolerate heavy attack. Nematicides kill or inhibit only a proportion of the nematodes present and populations will recover. Therefore, timing of the application is important. Attention should be paid to the conditions of application, especially watering in the nematicide—often no response has been due to poor application rather than no nematode damage. A farmer considering the use of nematicides should lay out test strips, to guide decision-making for future crops. Nematicides have high mammalian toxicity and care is required in their use to protect the user and prevent contamination of the environment.

Recent work indicates that nematodes are an important component of sugarcane yield decline, along with other biological, chemical and physical factors. Control of nematodes may be addressed in time by changes to the farming system that alleviate yield decline—these will possibly involve crop rotation, minimum tillage, trash retention and addition of organic matter. There are reports of resistance amongst sugarcane varieties to nematodes and, if this research is pursued, this strategy may become available in the long term.

OTHER SUGARCANE DISEASES IN AUSTRALIA

There are a number of diseases which generally cause no significant yield loss but which can cause concern occasionally.

Bacterial diseases

Bacterial mottle is caused by the bacterium *Pectobacterium chrysanthemi* (Burkholder *et al.*, Brenner *et al.*, emend. Hauben *et al.*), which infects a range of grass species. It is moved into sugarcane fields by floodwater and the disease is only found in flooded fields. It causes a mottling of the leaves and severe stunting. It usually only infects a small number of plants in a field and, therefore, does not cause significant losses.

Fungal diseases

Basal stem, root and sheath rot is caused by a basidiomycete fungus. The fungus attacks the base of young plant or ratoon crops, rotting the base of the leaf sheaths, the surface of the stalk and root primordia. The older leaves of the plant can sometimes develop distinct chlorosis between the veins. The leaf sheaths are easily pulled away from the stalk. The disease usually attacks plants that are suffering from moisture stress. If soil is heaped up around the base of the plant, it will produce new roots and grow away from the disease.

Brown stripe is a fungal disease caused by the fungus *Cochliobolus stenospilus* (Drechsler) Mats. and Yamam. (anamorph *Bipolaris stenospila).* It causes a brown stripe on the leaf surface that is 2–4 mm by 1–50 mm. The lesions have a distinct chlorotic halo. The disease is common on soils with nutritional deficiencies.

Ring spot is a very common fungal disease (*Leptosphaeria sacchari* B. de Haan and other fungi) that usually affects the older leaves. It commences as small dark green to brown spots. The spots expand and develop a straw-coloured centre that is surrounded by a well-defined red-brown margin. Ring spot usually has little effect on yield.

The fungus *Sclerophthora macrospora* (Sacc.) Thirum., Shaw and Narashiman. causes sclerophthora disease. The disease occurs in flooded fields. The fungus produces zoospores that can swim in water to attach to a plant and infect it. The disease causes severe stunting, yellowish white streaks on leaves, and the leaves can have a droopy appearance with wavy edges on the leaves. This gives the leaves an appearance similar to maize leaves. The disease usually only affects a few plants in a field and, therefore, does not cause significant economic losses.

Viral diseases

Dwarf is a disease of unknown cause, suspected to be a virus. It has been found in the Central and Southern Districts, including in isolation plots planted in areas that have never grown sugarcane before. Dwarf causes severe stunting in some varieties and short stiff dark green leaves with fine white stripes. It usually only affects a few stools and, therefore, does not cause significant losses.

SUGARCANE DISEASES NOT PRESENT IN AUSTRALIA

Downy mildew

Downy mildew is an important fungal disease of sugarcane. Because it invades the whole plant, yield losses can be severe in highly susceptible varieties. Poor quarantine procedures in the early days of the Australian sugar industry allowed downy mildew to be introduced to Australia and for many years it was one of our most important diseases. Strict controls led to its eradication from Australia in the late 1950s (in 1972 from experimental disease screening plots). It has not been observed since then.

Downy mildew occurs in the western-Pacific region including Fiji, Papua New Guinea, Irian Jaya, the Philippines, and several other southeast Asian countries. Because many Australian varieties are susceptible to the disease, downy mildew poses a threat to the Australian industry.

The disease can be caused by several oomycete pathogens: *Peronosclerospora sacchari* (Miyake) Shirai and Hara, *P. philippensis* (Weston) Shaw and *P. spontanea* (Weston) Shaw. *Peronosclerospora* spp. are obligate parasites; they cannot be cultured in the laboratory.

Leaves affected by downy mildew show a very characteristic leaf striping, varying in colour as the leaves and symptoms age. Initially streaks are light yellow, changing to greenish-yellow to yellow with time. Older streaks turn a brick-red colour. Streak dimensions vary with variety, but are usually 1–3 mm running parallel to the leaf venation for the whole length of the leaf. They have a well-defined margin.

The name 'downy mildew' arises from the production on warm humid nights of a soft downy growth of hyphae principally on the underside of leaves. Down contains spores that spread the disease. Spores may disperse up to 400 m from infected leaves, but they are very susceptible to drying and survive for only a few hours after daybreak. Maximum spore transmission occurs on warm humid nights and early in the morning following such nights.

In winter, oospore (sexual spore of the fungus) production within leaf tissues leads to leaf splitting. The pathogen also influences stalk physiology, leading to the development of some long, thin, brittle stalks (termed 'jump-ups'), which rise above the normal crop canopy.

Downy mildew is also transmitted by infected planting material, and this would be the most probable means for this disease to enter Australia.

It is best controlled by growing resistant varieties, using disease-free planting material, ploughing out highly diseased crops, and restricting growth of highly susceptible alternative hosts (such as maize) close to cane fields. All Australian commercial varieties are screened for resistance to downy mildew in Papua New Guinea under a cooperative arrangement between BSES and Ramu Sugar Limited. BSES pathologists visit Ramu regularly to gain experience with this and other diseases.

Ramu stunt

Ramu stunt is a disease that apparently is unique to Papua New Guinea. Only recognised in 1986, the disease caused large production losses on the estates of Ramu Sugar Limited; production dropped from an expected 360 000 tonnes of cane to only 120 000 tonnes in 1986. A large proportion of the estate was replanted with a highly resistant variety, which allowed the estate to escape commercial disaster.

The causal agent of the disease is uncertain. It is believed to be either a virus or a phytoplasma, transmitted by the planthopper *Eumetopina flavipes* Muir, which is not present in Australian cane-growing areas, but is on the Torres Strait islands and the mainland at Bamaga.

Symptoms of Ramu stunt are variable and may be difficult to recognise. It leads to strong stunting and often stool death in susceptible varieties. In susceptible varieties such as Ragnar, most plants in a crop will be killed as early as first ratoon. Leaf symptoms include mosaic and mottling type patterns with some chlorosis. Leaf growth tends to be short, stiff and erect.

When the vector is abundant, disease transmission may be very rapid. Diseased planting material may also lead to disease spread.

Ramu stunt has been well controlled in Papua New Guinea through the planting of resistant varieties. Regular screening of Australian commercial canes at Ramu Sugar Limited has shown most Australian varieties to be highly resistant to the disease, with symptoms present in about 25% of varieties grown in disease screening trials.

Grassy shoot and white leaf diseases

These two diseases are caused by similar pathogens and have some similarities in symptoms. They cause significant crop losses in some countries in south and southeast Asia. Grassy shoot has been recorded in India, Bangladesh, Malaysia, Nepal, Pakistan, Sri Lanka and Sudan. White leaf is important in Thailand and Taiwan.

The causal agents of the diseases are phytoplasmas (bacteria which lack a cell wall). A similar phytoplasma has been recorded in northern Australia, but is not reported to cause a disease in sugarcane.

Symptoms of grassy shoot consist of stunting, profuse tillering, side shooting and a softening of the leaf tissue. Chlorosis of the whole shoot may occur, but more common is a cream or white coloured striping of the leaves.

With white leaf disease, there is less variation in symptoms with leaf chlorosis being the diagnostic symptom. Early symptoms consist of a single white or cream line parallel to the mid-rib of one young leaf. Later symptoms include multiple stripes, mottling or whole shoot chlorosis. The development of totally chlorotic leaves in the spindle is characteristic of the disease.

The vector of white leaf is the plant-hopper *Matsumuratettix hiroglyphicus* (Matsumura). Maximum disease transmission occurs in summer-autumn when populations of the vector are highest. Various reports have been made of insect transmission of grassy shoot, but the identification of the vector is uncertain. Transmission through infected planting material is the most important mode of transmission with this disease.

Cane affected by grassy shoot can be partially cured by treatment of planting material with hot water (50°C for 2.0–2.5 hr) or moist-hot-air treatment (54°C for 4 hr). Through careful disease-free seed production, and utilising roguing of diseased plants in propagation plots, the disease has been reduced in incidence.

White leaf disease has not been controlled as successfully through hot-water or hot-air treatments. An integrated management approach has been adopted, consisting of resistant varieties (not totally successful alone), planting the cane at a time less favourable for disease transmission (in winter-spring months), roguing of diseased plants, use of disease-free seed-cane, and the ploughing out of infected fields.

Gumming

Gumming disease was once present in Australia, but resistant varieties and roguing led to its eradication. It has been reported from many countries but in recent years has only been of economic concern in Mauritius and Réunion.

The causal agent is the bacterium *Xanthomonas axonopodis* pv. v*asculorum* (Cobb) Vauterin *et al*. The bacterium produces smooth, glistening, round and yellow colonies on various agar media. Several races of the pathogen have been identified, with some differences in varietal reaction and symptom expression.

Gumming disease symptoms are quite characteristic, although variation in the pathogen does lead to variable symptoms. As with leaf scald, there are several categories of disease severity. The least severe form consists of leaf streaking and reflects the first stages of plant infection. Streaks are about 3–6 mm wide and run parallel to the leaf venation, either resulting from bacterial infection of the leaf margin or of wounds on the leaf blade.

As most infections arise from the leaf margin, streaks spread from here. Initially they are yellow to orange in colour with red flecks, later turning necrotic and ashy grey. Streak development depends on varietal resistance and growing conditions.

Systemic (whole plant) infection leads to chlorosis of the shoot, with either partial or complete chlorosis of the leaf lamina. Chlorosis in gumming is indistinguishable from that in leaf scald. In highly susceptible canes this may precede shoot death, but more often shoots remain alive but in an unthrifty condition.

In warm, humid conditions gum exudes from the cut ends of affected setts. Gum leads to milling problems and difficulty in manufacturing sugar from highly diseased cane.

Affected stalks may also be distorted with one side failing to grow properly because of the asymmetric distribution of gum and bacteria.

Gumming is transmitted locally by wind-blown rain (especially during cyclones or storm events), infected planting material, and mechanical equipment (cane knives, planters, harvesters). It could be moved over longer distances (between regions or countries) as infected planting material.

FURTHER READING

Frison, E.A. and Putter, C.A.J. 1993. Technical Guidelines for the Safe Movement of Sugarcane Germplasm. FAO/IBPGR, Rome.

Hughes, C.G., Abbott, E.V. and Wismer, C.A. 1964. Sugar-Cane Diseases of the World. Vol. 2. Elsevier, Amsterdam.

Martin, J.P., Abbott, E.V. and Hughes, C.G. 1961. Sugar-Cane Diseases of the World. Vol. 1. Elsevier, Amsterdam.

Ricaud, C., Egan, B.T., Gillaspie, A.G. and Hughes, C.G. 1989. Diseases of Sugarcane —Major Diseases. Elsevier, Amsterdam.

Rott, P., Bailey, R.A., Comstock, J.C., Croft, B.J. and Saumtilly, S. 2000. A Guide to Sugarcane Diseases. CIRAD, Montpellier.

Stringer, J.K., Magarey, R.C., Samson, P.R. and Webster, D.E. 1999. Recording Pest and Disease Data in the Australian Sugar Industry. BSES, Brisbane.