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Prospects for a genetic solution to the management of ratoon stunting disease

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Abstract Ratoon stunting disease (RSD,) caused by a bacterium (*Leifsonia xyli* subsp. *xyli*), is one of the most important diseases of sugarcane in Australia. RSD is an inconspicuous and highly infectious disease and can spread unnoticed causing significant yield loss across entire regions and industries. Developing varieties with resistance to RSD has been proposed at different times as a possible solution. This paper provides a review of the resistance status of the sugarcane germplasm, the effective range in reactions to the disease, and the efficacy of current practices used for RSD management. Examination of the Australian germplasm and historical resistance records show that material with effective RSD resistance has never been identified. Published literature has occasionally suggested that there are resistant varieties/clones, but these putative sources of resistance have failed to demonstrate commercially-effective disease control. Currently, there are no validated sources of resistance available to be used as parents in the breeding program. Evidence from overseas and Australia demonstrates that RSD can be successfully managed through disease-free planting material and farm hygiene. A genetic solution for RSD may only be considered if an effective source of resistance can be identified and validated.

Key words Ratoon stunting disease, RSD, *Leifsonia xyli* subsp. *xyli*, resistance screening , sugarcane

INTRODUCTION

Ratoon stunting disease (RSD), caused by the bacterium *Leifsonia xyli* subsp. *xyli*, is a major disease of sugarcane in sugarcane-growing countries (Davis and Bailey 2000). First discovered in Queensland in 1944-45 in the variety Q28, it causes very substantial stunting, poor vigour and low yields (Gillaspie and Teakle 1989). The bacterial pathogen is xylem-limited and causes vascular plugging that results in general stunting and poor ratooning (Kao and Damann 1980). No reliable external symptoms are evident; stunting and poor ratooning could be related to poor farming practices, low soil moisture, nutrient deficiencies, soilborne fungal diseases or plant-parasitic nematodes (Gillaspie and Teakle 1989; Magarey 1994; Stirling and Blair 2000). Yield losses due to RSD have been estimated at 5% to 10% but may reach 30% under suitable conditions. RSD is mainly spread through infected planting material and mechanically during harvesting and planting operations (Hughes and Steindl 1955; Steib *et al.* 1957). The bacterium can survive in soil on plant debris or in soil itself for up to 3 months (Bailey and Tough 1992). Planting disease-free seedcane, using clean farming equipment, removal of volunteer crops and maintaining farm hygiene are the most important RSD management practices.

Cropping resistant varieties is the most important management strategy for many sugarcane diseases, such as leaf scald, mosaic, pachymetra root rot and sugarcane smut. There is evidence of genetic variation for reaction to RSD in cultivated varieties and parent clones in Australia (Croft and Johnson 2013; Roach 1988, 1992; Roach and Jackson 1992), and development of resistant varieties for controlling RSD has been attempted in Florida, USA (Comstock *et al.* 2001). In Australia, the use of varietal resistance to manage RSD was never pursued (Young 2018), and the disease has been successfully managed by good phytosanitary practice and hot-water treatment (Croft and Johnson 2013). Different research groups have argued that the deployment of RSD-resistant varieties would benefit industry by minimising crop losses due to RSD (Young and Knight 2020; Dean and Davis 1990; Roach 1987).

The objectives of this paper are to review (a) the RSD resistance status of the Australian germplasm, (b) the effectiveness of the highest level of resistance currently available, and (c) the efficacy of current practices used for RSD management.

BREEDING FOR DISEASE RESISTANCE

To prevent or reduce the impact of pests or pathogen attacks, plants have developed a range of defence mechanisms. The two main defence mechanisms that plants use are resistance and tolerance (Pagan and Garcia-Arenal 2018). A resistant variety is able to prevent or minimise the multiplication of the pathogen. Generally, there are two main types of resistance, vertical resistance and horizontal resistance. A variety that exhibits a high degree of resistance to a single race, or strain, of a pathogen is considered to be vertically resistant. Vertical resistance is usually controlled by one or a few major genes (Vanderplank 1963). The term horizontal or partial resistance relates to when the trait is governed by multiple genes, provides partial but durable resistance (Vanderplank 1963; Robinson 1976). Disease tolerance refers to endurance of a biotic or abiotic stress with minimum impact on growth and reproduction. In relation to a plant disease, a tolerant cultivar has a susceptible infection type and supports a similar amount of the pathogen as another 'susceptible' but has significantly better yield and quality (Politowski and Browning 1978). Regardless of the mechanism or genetic complexity, the term 'resistant' is used to describe varieties that exhibit minimal yield or quality losses when exposed to the disease. When growers adopt a variety that is resistant to a particular disease, the expectation is that it provides effective control and no other management measures are required to mitigate infection or losses.

The Australian sugar industry manages several important diseases, such as smut, Fiji leaf gall, leaf scald, pachymetra root rot, orange rust, mosaic and red rot, through resistant varieties. Figure 1 shows the increase in commercial production of smut-resistant varieties since the incursion of the disease. With susceptible varieties now only comprising 20% of crop production, the industry has effectively mitigated the risk of major production losses using a genetic solution. Figure 1 also shows the decline in the proportion of the crop produced from pachymetra-resistant varieties, falling from 60% to 30% following the smut incursion. The recent release of varieties such as SRA11$^{\phi}$, SRA20, SRA22, WSRA24, SRA26 and SRAW30 that combine effective resistance to both diseases with productivity improvements will change the industry exposure to economic losses from pachymetra root rot.

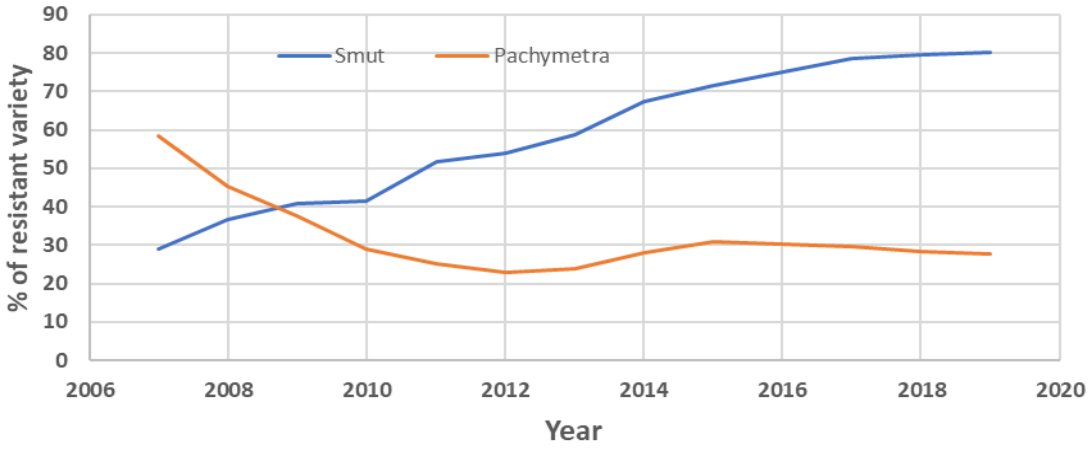

Figure 1. Proportion of crop production with smut and pachymetra root rot resistant (1-3) and intermediate resistant (4) varieties grown from 2007 to 2019.

Although resistant varieties play a major role in disease management, other control methods such as disease-free plant sources, termination of severely affected crops, application of fungicides, quarantine and rouging are important components of an integrated disease management strategy. Sugarcane diseases such as gumming and downy mildew were successfully eradicated from Australia by the introduction of resistant varieties and the use of clean seed (Saumtally and Dookun 2000; https://sugarresearch.com.au/wp-content/uploads/2017/02/Downy_

mildew_IS13089.pdf). In general, if there are cost-effective control strategies that can manage a disease then it is economically better to use management strategies rather than discard a susceptible but otherwise profitable variety (Croft *et al.* 1995). For example, pineapple sett rot disease (*Ceratocystis paradoxa*) of sugarcane has been successfully managed by the application of fungicides at planting for decades (https://sugarresearch.com.au/wp-content/uploads/2017/02/Sett-rot-diseases-IS13100.pdf); hence breeding for resistance is not necessary.

VARIATION IN RSD SUSCEPTIBILITY

Genetic variation in response to RSD has been examined in Australia, USA and South Africa with considerable variations in susceptibility observed in commercial sugarcane and wild *Saccharum* spp. (Davis *et al.* 1988; Roach and Jackson 1992; Comstock *et al.* 1996; Croft 2002). Miller *et al.* (1995) investigated genetic resistance of sugarcane in the USA and found 37% of total variance of clones within families, 4% among families, and that 59% was environmental. This indicates that, while RSD infection is significantly affected by environmental conditions, there is a genetic component to the observed variation. Later Comstock *et al.* (2001) reported a heritability value of $h^2 = 0.53$, obtained by plotting progeny mean cvb (colonised vascular bundles) against parental mid-point for 24 families. In Australia, Roach (1992) screened 309 lines for RSD resistance, including sources of *Saccharum officinarum*, *S. robustum*, *S. barberi*, *S. spontaneum*, *Erianthus arundinaceus* and 16 commercial cultivars. He found a high level of susceptibility among the basic lines; the commercial cultivars H60-6909, Helius and L60-25 showed significantly less susceptibility.

The confirmation of genetic variation for RSD susceptibility encouraged development of improved screening methods such as the enzyme-linked immunosorbent assay (Croft *et al.* 1994), and the development of ratings (Croft 2002). This approach took the range of variation observed in RSD reactions and applied it to the standard scale of 'resistant to susceptible' categories that are universally used in plant pathology. This provided an accurate description of the relative differences in reaction to RSD but was misleading in the sense that the term 'resistant' was used. Growers adopt resistant varieties with the expectation of a genetic solution where no further management intervention is required to avoid production losses (the case for all other sugarcane diseases). With RSD, the term 'resistant' was applied to the 'least susceptible' category of varieties. These 'resistant' varieties can exhibit significant infection and potential production losses due to RSD, and the use of clean seed plus farm hygiene is still needed to effectively manage RSD (Young and Knight 2020; Rotts *et al.* 2018). Perhaps unsurprisingly, some in the Australian industry have interpreted the availability of RSD 'resistant' varieties to mean they do not need to apply cultural practices for disease control, which has significantly increased the risk of production losses. In 2019 SRA amended the description of RSD ratings to remove the resistant category and classify the least susceptible varieties, such as Q200$^{\phi}$, as intermediate-susceptible to better reflect the commercial reality. Infection levels of up to 35% of commercial Q200$^{\phi}$ crops have been reported in the Innisfail-Babinda region (Young and Knight 2020).

Periodically some researchers have advocated breeding for RSD resistance in Australia (Roach 1987; Dean and Davis 1990; Young and Knight 2020). The appeal of a genetic solution is obvious as it generally provides the lowest cost and simplest disease control strategy for growers. To develop a variety with resistance to any disease, the first requirement is for a source of effective resistance to use as a parent. There are various published claims of varieties being resistant to RSD, including the notable H60-6909 (Roach and Jackson 1992). A review of historical screening data shows none of the exotic germplasm or international varieties, including H60-6909, exhibit better resistance to RSD than contemporary Australian varieties, such as Q200$^{\phi}$ and Q208$^{\phi}$, when compared in the same resistance-screening trials.

One potential source of RSD resistance remains to be tested in the Australian context. The old US variety CP72-2086 was reported to be resistant to RSD, exhibiting only 2% incidence (Rott *et al.* 2018). Material has been obtained and a trial established at SRA Woodford to determine if this may be a true source of effective resistance. If the resistance is confirmed, then a breeding strategy could be considered. If CP72-2086 does not exhibit significantly better resistance compared to current benchmarks, then the outlook for a genetic solution is poor.

MECHANISMS UNDERLYING VARIATION IN RSD SUSCEPTIBILITY

The mechanisms underlying differences in reaction of sugarcane to RSD are partly understood. The main cause of stunting is suggested to be physical blockage of the xylem vessels due to the accumulation of *L. xyli* subsp. *xyli* bacteria in vascular cells, lacunae, tracheids, and parenchyma cells in the nodal area of infected stalks (Kao and Damann 1980). A pre-existing physical barrier in xylem vessels of some sugarcane varieties leads to reduced RSD sensitivity. Teakle *et al.* (1978) reported that varieties with complex and profound branching in their xylem vessels slow the flow of bacteria through the nodes and that these were less susceptible. No information is available on molecular or physiological resistance mechanisms to *L. xyli* subsp. *xyli* infection. Regardless of the variation in the physical structures and arrangement of xylem vessels, none of the Australian varieties provide effective RSD resistance. A greater understanding of the plant-pathogen interactions and underlying physiology may provide opportunities to generate effective RSD resistance using genetic modification or gene-editing techniques. However, such information is currently not available.

MANAGEMENT OF RSD

Prevention of disease spread and planting of disease-free seedcane are the two most important factors in managing RSD (Gillaspie and Teakle 1989). Among the sugarcane industries worldwide, the USDA Sugarcane Field Station, Canal Point, Florida, established a resistance selection program for RSD in the early 1990s (Davis et al., 1994). Since then, no RSD-resistant varieties have been developed or released, and clean seed still is considered to be the most effective management strategy in Florida (Rott *et al.* 2018). On the other hand, the Louisiana sugar industry has never selected for RSD resistant varieties. Instead, they focused on commercial clean-seed nursery stocks derived from tissue culture (Hoy 2017). Louisiana is now considered to have one of the lowest levels of RSD of any industries world-wide (Hoy per comm.).

Hot-water treatment of planting material was practised soon after RSD was first recognised as it effectively eliminated the pathogen, especially with successive annual treatments (Gillaspie and Davis 1992; Steindl and Teakle 1974). Hot-water treatment has some potential drawbacks - reduction in germination of seedcane in some varieties and only partial pathogen elimination (if stalks are heavily diseased) (Davies and Bailey 2000). In Australia, hot-water treatment at 50°C for 3 hours is used to produce disease free planting materials (https://sugarresearch.com.au/wp-content/uploads/2017/02/RSD-IS13007.pdf) and is effective in eliminating the pathogen (Roach and Jackson 1992; Fernandes *et al.* 2010), especially with successive annual treatments.

RSD is a highly infectious disease and is easily spread by infected seedcane, and via contaminated machinery, such as harvesters, knives and planters (Comstock 2002). A single pass of a harvester may transmit the disease to healthy stools up to 7.3 m row from the source of infection (Hoy *et al.* 1999). Sanitation to reduce mechanical transmission of the pathogen is important to mitigate the spread of the disease. In Australia, knives and machinery parts that come in contact with the cut surface of sugarcane are treated with sterilizers; registered products include 0.1% benzalkonium chloride (Cane Knife Steriliser) or didecyldimethyl-ammonium chloride (Steri-maX®). Sanitation with such disinfectants successfully restricts RSD spread when applied diligently (Davis and Bailey 2000). Other disinfectants that are effective in killing the bacteria are Lysol®, Dettol®, ethanol and sodium hypochlorite.

Tissue culture is used to establish some nursery or commercial crops in the USA, Australia, Japan and Thailand (Cha-um *et al.* 2006; Homhual *et al.* 1999; Hoy 2017). In Florida and Louisiana, all commercial seedstocks are produced from tissue culture and no hot-water treatment program currently operates (Rott *et al.* 2018). Tissue-culture techniques have also been used in RSD control programs (Hoy and Flynn 2001). Meristem tissue culture can be used to render disease-free RSD planting material. Often disease-free plants, obtained from heat-therapy, are used for micropropagation of disease-free plantlets for expansion as commercial seedcane (Comstock 2002). Surveys in Louisiana in 1984-85, showed a 22% incidence of RSD across all varieties (Damann and Hollier 1991). The incidence of RSD was lower in fields established from commercially-available tissue-culture seedcane compared to those planted with hot-water-treated cane (Hoy 1998). The latest surveys in Louisiana during 1998-2000 determined the mean incidence of infected stalks at 3% or less (Hoy and Flynn 2001).

A review funded by the Australian sugar industry in the late 1990s concluded that varietal resistance to RSD was not an economically viable RSD-management strategy method compared to the existing disease-free seed scheme (Croft *et al.* 1995). It concluded that disease-free planting material is the most effective way to manage RSD, with a high return of up to $251/ha per year (compared to the use of RSD-infected seed cane).

DISCUSSION

Ratoon stunting disease (RSD) is a major disease of sugarcane and poses a very significant yield constraint if not managed adequately. Due to its inconspicuous symptoms and highly contagious nature, RSD is able to spread across industries unnoticed if no preventative measures are applied. Research has shown that the disease can be successfully managed via hot-water treatment (Gillaspie and Davis 1992; Steindl and Teakle 1974), farm hygiene (Comstock 2002), and disease-free seed cane (Hoy 2017; Gillaspie and Teakle 1989). Australian and overseas surveys suggest that where the disease is properly managed, RSD incidence remains consistently low. In Australia, where proper management practices are adopted the RSD incidences are reduced to less than 1% to zero regardless of varietal differences (Mackay, Tablelands and Plane Creek). On the other hand, in the districts where the management practices are inadequately applied the disease incidence can reach as high as >50% (Magarey *et al.* 2021).

RSD spread in northern Queensland has recently been attributed to the use of contaminated fungicide solution in billet-planter dip tanks (https://sugarresearch.com.au/wp-content/uploads/2017/03/RSD-and-billet-planters_Info-Sheet_2020_F.pdf).

A resistance-rating method was developed by Harrison and Davis (1988), based on measuring the number of infected vascular bundles or bacterial cells in inoculated stalks, using enzyme-linked immunosorbent assay (ELISA). Although these tests provided a good indication of bacterial loads in sugarcane stalks, the sensitivity of this assay is inferior to molecular based qPCR tests (Young *et al.* 2016). Since the early 1990s, Australian commercial varieties have been screened to provide industry information through QCANESlelect™ (Croft 2002; Croft *et al.* 2012; Croft and Johnson 2013). The information on RSD susceptibility can be used by growers to understand their level of risk, potentially avoiding highly susceptible varieties if best-practice management is not possible.

The evidence is clear that some commercial varieties such as KQ228$^{\phi}$ and Q253$^{\phi}$ are more sensitive to RSD than varieties such as Q200$^{\phi}$ and Q208$^{\phi}$. There is merit in growers having access to this information to better understand their risk. Trials have been established to generate this information for the key new varieties that are leading grower adoption.

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