THE VIRAL AND PHYTOPLASMA FORMS OF YELLOW LEAF SYNDROME OF SUGARCANE

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Abstract

Yellow leaf syndrome of sugarcane (YLS) is a recently recognised disease of sugarcane with a distinctive pattern of yellowing of the leaf midrib. Two etiological agents, Sugarcane yellows phytoplasma (SCYP) and Sugarcane yellow leaf virus (SCYLV) have been found to be associated with the syndrome. SCYP predominates in Africa, while SCYLV appears to be the principal causal agent in the rest of the world. There is good evidence for an effect of infection by SCYLV on growth. Both pathogens are often present in the absence of YLS symptoms and mixed infections can occur. The symptoms caused by the two pathogens appear to be identical and this makes differentiation of the two forms difficult. Diagnosis of the causal agents relies on antiserum or molecular based assays. Taxonomically, both SCYP and SCYLV are extremely interesting pathogens. Molecular profiling via restriction enzyme digestions of PCR products suggests that SCYP is a member of the western × group of phytoplasmas, while analysis of the full genomic sequence of SCYLV indicates that it is a recombinant, combining sequences derived from three different viruses. The current status of the biological properties and taxonomy of SCYP and SCYLV is reviewed.

Introduction

A leaf-yellowing symptom of sugarcane (Saccharum L. interspecific hybrids) was first noted in Hawaii in 1988 (Schenck, 1990) and in south-east Brazil in 1990 (Vega, 1997). Two years later the disease, yellow leaf syndrome (YLS), was widespread in Brazil and it has since been reported from numerous countries including the continental USA (Comstock et al., 1994), Australia (Smith et al., 1995), Mauritius (Anon, 1996), South Africa (Bailey et al., 1996; 1997), French West Indies (Daugrois et al., 1999) and Reunion (Rassaby et al., 1999).

The surface of the leaf mid ribs of plants showing YLS becomes yellow, usually most clearly on the abaxial surface, and sometimes reddened (Comstock et al., 1994; Vega et al., 1997). One sugarcane cultivar, SP71-6163, shows a conspicuous reddening of the adaxial surface of the midrib whilst the abaxial surface is strongly yellow. There is also an increase in the brix content of the leaves of plants with YLS symptoms (Comstock et al., 1994). The apparent close similarity between the symptoms of YLS and yellow wilt, a condition of sugarcane that was widespread in eastern and southern Africa in the 1960s and 1970s (Ricard, 1968; Rogers, 1970) has been noted.

Several causal agents of YLS have been proposed to date including fungi or poor cultivation (Matsuoka and Meneghin, 1997), virus (Irey et al., 1997; Vega et al., 1997) and phytoplasma (Cronje et al., 1998). Direct evidence for a phytoplasma-induced and a virus-induced YLS has been provided by Cronje et al. (1998) and Scagliusi and Lockhart (2000) respectively. These forms of YLS are indistinguishable by symptomatology and differentiation relies upon serum or molecular-based diagnostic assays. Here, we briefly review the recent history of YLS and the current status of the etiology, taxonomy and biology of both forms of yellow leaf syndrome.

Identification of the viral and phytoplasma agents of YLS

The yellow-red symptoms of YLS and the increased brix suggested that a phloem-limited pathogen could be the causal agent. In 1995, small icoshedral particles were found in symptomatic plants (Anon, 1996). A weak serological reaction, and the physico-chemical properties of the particles suggested that a virus, by now commonly referred to as Sugarcane yellow leaf virus (SCYLV), was a luteovirid (Scagliusi and Lockhart, 1997; Vega et al., 1997). This was confirmed by Irey et al. (1997) when a short stretch of sequence amplified with the group-specific luteovirus RT-PCR primers of Robertson et al. (1991) was found to be most similar to the corresponding sequence from the PAV isolate of Barley yellow dwarf luteovirus (BYDV-PAV). In 1997, Scagliusi and Lockhart demonstrated that the aphid Melanaphis sacchari transmitted a virus from infected to healthy sugarcane. Plants infected in this way developed typical symptoms and viral particles were re-isolated. In 2000, Moonan et al. and Smith et al. confirmed that SCYLV was a new member of the Luteoviridae family (a ‘luteovirid’) by

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sequencing the entire genome (Genbank accession AF157029, EMBL accession AJ249447) of the viral isolate infecting cultivar CP65-357.

In South Africa and Swaziland, surveys for the association of a virus with YLS were unsuccessful up to 1997, although symptoms were widespread, leading to the hypothesis that a different causal agent was associated with YLS in these countries. Examination of leaf sections showing YLS symptoms by transmission electron microscopy (TEM) demonstrated the presence of phytoplasmas. A polymerase chain reaction (PCR)-based protocol was developed for the detection of potential phytoplasmas associated with YLS (Cronjé et al., 1998). Sequence data from the 16S-23S rDNA intergenic spacer region suggested that SCYP belonged to the western × group of phytoplasmas.

A survey of YLS-symptomatic sugarcane conducted in the South African and Swaziland sugarcane industries in 1997 led to the conclusion that in these countries, the presence of YLS symptoms and phytoplasma sequence were strongly correlated (Cronjé et al., 1998). SCYLV has since been found in a few varieties in restricted localities in South Africa, although it is common in imported varieties in collections. The evidence suggests that SCYLV was introduced recently into southern Africa and is still rare.

The examination in South Africa of more than 300 leaf samples with YLS symptoms from 19 other countries or regions showed that SCYLV was present in material from all countries, in all symptomatic samples and in 57% of all samples. By contrast, SCYLV was present in 58% of symptomatic samples and in 33% of all samples. SCYLV was found to occur in most countries except in Africa and was found to be common in samples from Mauritius and Reunion (Cronjé and Bailey, 1999). This was in agreement with local results obtained subsequently in these countries. Mixed infections of SCYLV and SCYLV were common (Cronjé and Bailey, 1999).

Subsequently, SCYLV was detected in Mauritius (Aljanabi et al., 2000) and Cuba (Peralta et al., 2000). Surveys on Reunion Island indicated that SCYLV predominated as the cause of YLS. Here the association between SCYLV and symptoms was found to be significant, while SCYLV could only be detected occasionally (Rassaby et al., 1999). Tests for SCYLV in Florida were inconclusive (Davis, 2000).

**Biology and effects of SCYLV**

SCYLV infects a range of *Saccharum* germplasm, including *S. officinarum*, *S. robustum*, *S. spontaneum*, *S. sinensis* (Schenck and Lehrer, 2000) as well as commercial interspecific hybrids. There are conflicting reports as to whether SCYLV infects *Erianthus* spp. (Scaglusi and Lockhart, 2000; Schenck and Lehrer, 2000) whilst a *Miscanthus* hybrid was not infected (Schenck and Lehrer, 2000). SCYLV is transmitted by the aphid species *Melanaphis sacchari*, *Rhopalosiphum maidis* and *R. rufiabdominalis* (Schenck and Lehrer, 2000) but is not transmitted by *Sipha flava*. Like all other luteovirids, SCYLV is not mechanically transmissible (Scaglusi and Lockhart, 2000).

Schenck and Lehrer (2000) demonstrated that SCYLV can be transmitted by *M. sacchari* to wheat (Norstar and Bobwhite), oat (Coker 227), barley, rice and maize (Supersweet #10A), but not by *R. maidis* to maize (Early Sunglow), sorghum (*S. bicolor*), Johnson grass (*S. halepense*) or oat (Clintland 64), nor by *M. sacchari* to oat (Clintland 64) Scaglusi and Lockhart (2000).

SCYLV was reported to have caused estimated losses, based on field observations, of up to 50% in the highly susceptible cultivar SP71-6163 in Brazil. Grisham et al. (2000) reported that SCYLV caused a 6–14% yield loss in cultivar LCP82-89 in a field experiment and it is interesting that it was in the absence of symptoms. Plant resistance has been reported in some cultivars, such as H78-3567, H78-4153 and H78-7750 (Schenck and Lehrer, 2000). Genetically engineered resistance using pathogen-derived transgenes is also being actively pursued, but as yet there are no reports of progress.

**Taxonomy of SCYLV**

Sugarcane yellow leaf virus is an unassigned member of the *Luteoviridae*, a recently defined family in the seventh report of the International Committee on the Taxonomy of Viruses (ICTV) (van Regenmortel et al., 2000). In this system, three genera, *Luteovirus*, *Polerovirus* and *Enamovirus*, are defined within the *Luteoviridae* family to accommodate the previous *Luteovirus* genus groups of subgroup I, subgroup II and the previous assigned genus *Enamovirus*. Thus the type species of the new genera in the *Luteoviridae* are *Barley yellow dwarf virus* (BYDV-PAV), *Potato leaf roll virus* (PLRV), and *Pea enation mosaic virus* (PEMV). This system still recognises the genome organisation of the old sub-group classification system but clearly recognises the similarities between the RNA species 1 and 2 of PEMV and the viruses represented by PLRV and BYDV-PAV respectively. SCYLV demonstrates a Polerovirus-like genome organisation (Moohan et al., 2000; Smith et al., 2000), in that there are six open reading frames (ORFS 0 to 5) and the replicase complex at the 3' end of the genome has a distinct sobemo-like homology. However, closer examination of the genome reveals that SCYLV is a recombinant virus in which the lineages from a *Polerovirus*, a *Luteovirus*, and an *Enamovirus* have been fused (Moohan et al., 2000; Smith et al., 2000). Borg et al. (1999) recognised that SCYLV was a unique virus within the *Luteoviridae* and suggested that SCYLV be the type member of a new genus 'Saccharvirus'. This proposal has yet to be accepted. Further, Smith et al. (2000) recognised that the generic term 'luteovirus' was no longer suitable to describe viruses within the new *Luteoviridae* family and proposed 'luteovirid' as the generic description of viruses in this family.

**Taxonomy of SCYPV**

The initial conclusion from analysis of the phytoplasma sequence data was that SCYP was a member of the western × group (Cronjé et al., 1998). The
PCR-based assay developed for the diagnosis of YLS was applied to a range of plants and diseases of suspected phytoplasma aetiology. The phytoplasmas of the Bermuda grass white leaf (BGWL) and Sugarcane white leaf (SCWL) group seem to move readily between grass species and even into non-grass hosts, such as date and coconut palms (Cronjé and Jones, 2000). The significance of geographical distribution, perennial hosts and latent infection of phytoplasmas was also studied. Geographic location, host plants and the phytoplasma group all have a significant role in the epidemiology of these diseases.

Symptomless phytoplasma infection in sugarcane has been demonstrated to occur widely (Cronjé and Jones, 2000). Survey data obtained from sugarcane and other grasses suggest that these grasses become infected with the phytoplasma group dominant in the different geographic regions. The latent type of infection often seen in sugarcane, and the frequent occurrence of symptoms of suspected phytoplasma aetiology in other perennial grasses, suggest that this could be a common occurrence within the grass species.

Whilst phytoplasmas can be detected in numerous plant samples, these pathogens may be a secondary rather than a primary pathogen within the yellow leaf syndrome. Several samples of sugarcane showing obvious YLS symptoms yielded negative results for both pathogens with the current assays. In several sugarcane industries phytoplasmas have not been detected successfully or ambiguous results are generated by the assay (e.g. Davis, 2000). The role of SCYLV and other phytoplasmas in yellow leaf syndrome of sugarcane requires clarification.

**Conclusion**

There is considerable evidence for a primary role of SCYLV in yellow leaf syndrome of sugarcane in many countries, including molecular analysis of the viral genome, yield loss data and aphid transmissibility. However, SCYLV is rare in southern Africa, where symptoms of YLS are common. Although there is good evidence for a strong association of the phytoplasma SCYP with symptoms of YLS in the absence of SCYLV, there is as yet no evidence that SCYP is a significant pathogen of sugarcane. However, YLS examples that cannot be diagnosed with either SCYLV or SCYP continue to occur, suggesting that the term syndrome is indeed appropriate for this new disease of sugarcane and that the symptoms might have a variety of causes.

**REFERENCES**


