An Overview of Recent Research on *Sugarcane yellow leaf virus.*

Susan Schenck
Hawaii Agriculture Research Center, Kunia HI, USA

Abstract

Research on *Sugarcane yellow leaf virus* has been reviewed by Rott *et al*., (2007) and Schenck (2001). The following article covers those reviews with additional updates of recent research.

Yellow leaf caused by S*ugarcane yellow leaf virus* (SCYLV) was the most studied disease of sugarcane during the last decade. The genome of SCYLV has been fully sequenced and characterized, and the virus was recently assigned to the genus *Polerovirus* of the family *Luteoviridae*. Molecular and immunological assays were developed to detect the virus in symptomatic and asymptomatic plants. SCYLV is transmitted by the aphid vectors *Melanaphis sacchari* and *Rhopalosiphum maidis*, and the pathogen was successfully transmitted via these aphids to several species of *Poaceae*. *Saccharum* species are, however, the only known natural hosts of SCYLV. Diversity studies showed that SCYLV is a variable virus, and several genotypes of this pathogen have been described. One genotype was found in all studied geographical locations from Africa, the Americas, and Asia, whereas two other genotypes were found in only 4 of 18 locations. Variation in capacity of infection and in virulence exists between genotypes or isolates of SCYLV and use of sugarcane resistance to infection by the virus appears the most promising means to control yellow leaf in the field.

Introduction

Yellow leaf syndrome is the name given to a disease that appeared in Hamakua, Hawaii on variety H65-7052 in 1989 (Schenck, 1997). Diseased plants exhibited intense yellowing of the leaf midrib and leaf necrosis proceeding from the tip toward the base of the leaf. Subsequently, the same symptoms were reported from several countries (Comstock *et al.*, 1994; Lockhart *et al.*, 1996), especially from Brazil where the disease caused significant yield losses in widely grown cultivar SP71-6163 (Vega *et al*., 1997; Lockhart, Cronjé, 2000). However, genesis of yellowleaf syndrome started most likely earlier because older reports of sugarcane leaf yellowing exist, such as yellow wilt in East Africa in the 1960s (Ricaud, 1968).

Although both sides of the midrib can be yellow, a characteristic symptom of the disease is an intense yellowing of the leaf midrib on the abaxial surface of mature leaves, and this discoloration often occurs while the lamina is still green. In some sugarcane cultivars, leaves show a red coloration of the midrib on the adaxial surface. Yellowing of the midrib and leaf necrosis generally appears on the fourth or fifth and older leaves (Schenck, 2001). Dwarfing of terminal internodes and tissue necrosis can also eventually be observed. Diseased plants show a reduction of sucrose in stalks and an increase of sucrose accumulation in midribs (Fontaniella *et al*., 2003; Gonçalves *et al*., 2005). Stress conditions such as water logging, drought and cool winters favor symptom expression of yellow leaf (Comstock *et al*., 1994; Schenck, 2001.

Izaguirre-Mayoral *et al*., 2002). In 2000, yellow leaf syndrome was reported in more than 35 sugarcane producing countries/locations worldwide (Lockhart, Cronjé, 2000), and this number is still increasing (Avila *et al*., 2001; Garces *et al*., 2005).

First association of RNA with yellow leaf syndrome was reported in Hawaii in 1994 (Borth *et al*., 1994), and a luteovirus was found in diseased sugarcane from Florida and Brazil in 1995 (Lockhart *et al.*, 1996; Vega *et al*., 1997). This virus was successfully transmitted from infected plants to healthy plants with the aphid vectors *Melanaphis sacchari* and *Rhopalosiphum maidis* (Scagliusi, Lockhart, 2000). Additionally, inoculated plants showed symptoms of leaf yellowing, thus providing strong evidence that this virus was a causal agent of yellow leaf syndrome. However, in Cuba, Mauritius and South Africa, a phytoplasma was associated with sugarcane showing yellow leaf syndrome (Cronjé *et al*., 1998; Arocha *et al*., 1999; Aljanabi *et al*., 2001). This phytoplasma was successfully transmitted in Cuba from diseased to healthy sugarcane with the delphacid planthopper, *Saccharosydne saccharivora* (Arocha *et al*., 2005). Leaf yellowing was also observed after inoculation of the sugarcane plants with the phytoplasma, thus providing evidence that another pathogen was involved in yellow leaf syndrome. Recently, in order to differentiate the diseases caused by the virus and the phytoplasma, the International society of Plant Pathologists (ISPP) committee on common Names of Plant diseases suggested to name the virus disease yellow leaf, and the disease caused by the phytoplasma leaf yellows. This suggestion was approved by the ISSCT pathology committee at the 7th ISSCT pathology workshop that was held in Baton Rouge in Louisiana in 2003 (Rott *et al*., 2005).

Since the first description of yellow leaf syndrome in Hawaii in 1989, much research has been devoted to this disease and the associated pathogens. Because the virus appears to be more widespread than the phytoplasma (Smith *et al*., 2001), and because yield losses have been so far demonstrated for the virus only, yellow leaf was the most studied disease of sugarcane during the last decade.

Impact of yellow leaf on sugarcane yields

Characteristics such as stalk height, number of stalk internodes and virus population *in planta* vary according to virus isolates, sugarcane cultivar, and environmental conditions. However, these differences were not reproducible in repeated trials. Additional studies are needed to further investigate importance of SCYLV strains on a world-scale basis and to identify the genetic and environmental effects on virus multiplication and disease expression.

Negative impact of yellow leaf on sugarcane yields has been reported in several countries. The 24% yield losses caused by SCYLV in the late 1980s in sugarcane cultivar SP71-6163 in Brazil are the largest known yield losses reported so far (Vega *et al*., 1997). Because of this disease, cultivar SP71-6163 grown on thousands of hectares was phased out of production in Brazil. Subsequently, potential impact of the disease was also demonstrated with field trials in several countries. The incidence of SCYLV in commercial fields can reach 100% in susceptible cultivars (Comstock *et al*., 1999, 2001; Viswanathan 2002; Rassaby *et al*., 2004), and the disease can cause significant yield losses in susceptible cultivars even if infected plants do not exhibit

overt disease symptoms (Grisham *et al*., 2001 and 2002; Rassaby *et al*., 2003). SCYLV can affect stalk number, total reducing sugars, leaf area, chlorophyll content, and sugar transport (Grisham *et al*., 2001; Izaguirre-Mayoral 2002; Viswanathan, 2002). Infected plants have been shown to have an accumulation of sugars in the leaves. This was thought to be due to disruption of the photosynthetic process (Gonçalves *et al*., 2005). They elucidated alterations in the photosynthetic metabolism, at least partly due to a decrease in the ratio of chlorophyll a to chlorophyll b. Lehrer *et al*. (2007) studied the starch and sucrose concentrations in infected and uninfected plants and measured significant differences that suggested a reduction of carbohydrate export associated with infection.

Diagnosis of SCYLV

Reverse transcription-polymerase chain reaction (RT-PCR) was the first technique developed to diagnose the presence of a virus in symptomatic sugarcane with primers specific to luteoviruses (Lockhart *et al*., 1996). The amplified product from a SCYLV isolate from Florida was sequenced and used to design another set of primers (YLS111 and YLS462) that were specific to SCYLV (Irey *et al*., 1997), and a specific RT-PCR protocol was developed (Comstock *et al*., 1998). Subsequently, this set of primers was widely used to diagnose and detect SCYLV in symptomatic and asymptomatic sugarcane plants (Aljanabi *et al*., 2001; Chatenet *et al*., 2001; Moutia, Saumtally, 2001; Rassaby *et al*., 2004). More recently, other SCYLV specific primers and real-time RT-PCR assays were developed (Gonçalves *et al*., 2002; Korimbocus *et al*., 2002a).

The first antibodies specific to SCYLV were prepared by Ben Lockhart at University of Minnesota with purified virus (Schenck *et al*., 1997; Scagliusi, Lockhart, 2000), and these polyclonal antibodies were used to develop several immunological techniques. Tissue-blot immunoassay (TBIA) is probably the most widely used technique based on its citation in numerous papers (Schenck *et al*., 1997; Comstock *et al*., 1998 and 2001; Rassaby *et al*., 1999; Chatenet *et al*., 2001; Garces *et al*., 2005; Victoria *et al*., 2005). Double antibody sandwich-enzyme linked immunoassay (DAS-ELISA) has also been successfully used to detect the pathogen in infected plant material (Comstock *et al*., 1998; Scaglius, Lockhart, 2000; Viswanathan, 2004). Monoclonal antibodies to SCYLV for use in TBIA were produced using recombinant readthrough protein (Korimbocus *et al*., 2002b). More recently, an antibody to a short peptide sequence was produced for diagnosis of isolates of SCYLV without interfering reactions to sugarcane tissue or other viruses related to luteoviruses or poleroviruses (Wang *et al*., 2005).

For rapid field diagnostic screening, the TBIA is the most efficient, as well as a technique developed by Grisham *et al.* (2010) that detects infection in asymptomatic leaves with hyperspectral remote sensing. The latter technique can be used in the field.

Thanks to these diagnostic techniques, it was possible to demonstrate that most sugarcane varieties infected by SCYLV do not exhibit disease symptoms (Schenck, 2001). The virus was

therefore spread around the world in symptomless but infected material for many years, until efficient diagnostic tools were available, especially in quarantine (Chatenet *et al*., 2001).

Epidemiology of SCYLV

The virus resides in the phloem tissue of plants and is transmitted during vegetative propagation of sugarcane by planting infected cuttings. SCYLV has never been detected in seedlings issued from true seed, suggesting that the SCYLV, like other *Luteoviridae*, is not seed-transmitted. However, it was successfully transmitted by means of several aphid vectors. The sugarcane aphid *Melanaphis sacchari* and the corn leaf aphid, *Rhopalosiphum maidis*, were both able to transmit SCYLV from infected to healthy plants, but transmission efficiency was the highest with *M. sacchari* which is the most common in sugarcane fields (Lockhart *et al*., 1996; Scagliusi, Lockhart, 2000; Schenck, Lehrer, 2000). The virus was detected in inoculated plants which also showed disease symptoms. SCYLV was not transmitted by *Sipha flava*, the yellow sugarcane aphid, but the rice root aphid, *R. rufiabdominalis*, was able to transmit the virus from infected wheat seedlings to wheat and oats (Schenck, Lehrer, 2000). McAllister *et al*. (2005, 2008) showed conclusively that spread of the virus in the field was correlated with infestations of *M. sacchari*. Zhou *et al*. (2006) found another aphid vector, *Ceratovacuna lanigera*, that transmitted SCYLV in China.

Saccharum species (including *S. officinarum*, commercial hybrids, and wild relatives) are the only known natural hosts of SCYLV (Lockhart, Cronjé, 2000; Schenck, Lehrer, 2000; Comstock *et al*., 2001;, Lehrer *et al*., 2001). Numerous grassy weeds were surveyed in Hawaii, but all tested virus-free (Schenck, Lehrer, 2000). However, barley, oats, rice, sorghum, sweet corn and wheat were successfully inoculated with SCYLV using viruliferous aphid vectors.

Incidence of SCYLV varies according to sugarcane varieties, but other factors not elucidated yet must play a significant role. SCYLV occurs in Cuba, Sénégal and South Africa, but its distribution is limited in these countries (Cronjé *et al*., 1998; Arocha *et al*., 1999). Low rates of disease increase and survey results suggested that the industry-wide inoculum levels are low in Louisiana (McAllister *et al*., 2008). In contrast, the incidence of the virus is very high in locations such as Florida (Comstock *et al*., 1999), India (Viswanathan, 2002) and Réunion Island (Rassaby *et al*., 1999). Aphid biotypes with different transmission efficiencies of SCYLV may occur. Although *M. sacchari* is present in Réunion Island, surveys for several crop cycles suggested that SCYLV is mainly transmitted by infected cuttings in this geographical location (Rassaby *et al*., 2004).

Identification and characterization of the causal agent of sugarcane yellow leaf

Isometric virus-like particles of 24 to 29 nm in diameter were found by electron microscopy both in phloem companion cells and in partially purified preparations from symptomatic plants. Size and shape of these virus particles associated with a weak serological reaction using *Barley yellow dwarf virus* (BYDV) serotype PAV antiserum suggested that the causal agent of yellow leaf was a luteovirus (Lockhart *et al*., 1996; Vega *et al*., 1997; Scagliusi,

Lockhart, 2000). Subsequently, the genome of this virus named S*ugarcane yellow leaf virus* (SCYLV) was sequenced and sequence comparisons clearly established that SCYLV was a member of the *Luteoviridae* virus family (Maia *et al*., 2000; Moonan *et al*., 2000; Smith *et al*., 2000). Additionally, phylogenetic studies showed that SCYLV was an emerging virus that has evolved by recombination between luteoviral and poleroviral ancestors (Moonan *et al*., 2000; Smith *et al*., 2000).

The genome of SCYLV is monopartite and consists of a positive-sense single stranded RNA of 5,895-5,898 nucleotides. The viral genome encodes at least six open reading frames (ORFs 0-5) and shows a genome organization typical of poleroviruses. Although SCYLV shares genomic properties with members of the genera *Polerovirus* and *Luteovirus*, it has been assigned to the genus *Polerovirus* of the family *Luteoviridae* by the International Committee on Taxonomy of Viruses on the basis of its striking similarities to the 5' half of the *Polerovirus* genome (D'Arcy, Domier, 2005).

The peptide encoded by ORF0 was recently shown to function as a suppressor of posttranscriptional gene silencing (Albert *et al*., 2005). ORFs 1 and 2 are translated together and code for a multifunctional peptide and an RNA-dependent RNA polymerase (RdRp), respectively. The peptide sequence encoded by ORF1 includes sequence motifs of both a serine proteinase and a putative genome linked viral protein (VPg). ORF3 codes for the coat protein and ORF4 for a movement protein, whereas the peptide encoded by ORF5 is a read-through protein. This latter protein is produced via a translational read-through of the peptide encoded by ORF3 and might be linked to virus transmission by aphids.

Genetic diversity of SCYLV

Following the development of reliable serological and molecular diagnostic techniques, SCYLV was found to be widespread in most sugarcane producing countries (Lockhart, Cronjé, 2000). The worldwide distribution of SCYLV led several research groups to study the genetic diversity of SCYLV. Moonan and Mirkov (2002) identified two groups of the pathogen among virus isolates collected from North, South, and Central America. One group contained only isolates from Colombia (C-population) and the second group (superpopulation) was formed by the isolates from the other countries (Argentina, Brazil, Guatemala, USA/Florida-Louisiana-Texas). Borg *et al*., (2001) showed that fingerprinting the viral sequence from various SCYLV positive cultivars reveals diversity in SCYLV sequences both between, and within, different geographic locations in the world. Based on phylogenetic analyses of sequences of the entire translated genome of SCYLV, Abu Ahmad *et al*., (2006a) described the occurrence of three different genotypes (BRA, ER and REU) within eight virus isolates from worldwide locations. The name given to each of these genotypes was based on the geographical location where it was first detected: Brazil, Peru and Réunion, respectively. Additionally, a virus isolate from Cuba, that was partially sequenced, showed only 77-80% amino acid sequence identity in ORF1 with isolates of genotypes BRA, REU and PER. This result suggested that the Cuban isolate represented another genotype (genotype CUB) or even an isolate of a new virus species (Abu

Ahmad *et al*., 2006a). Recently, another distinct SCYLV isolate was sequenced in China (Wang, Zhou, 2010). It was most closely related to CUB and was named CHN1. Differences in virulence have been shown to exist (Abu Ahmad *et al*., 2007).

Specific RT-PCR primers were successfully developed to analyze the distribution and incidence of SCYLV genotypes BRA, CUB, PER and REU in different geographical locations in the world. However, genotypes BRA and PER, that are phylogenetically relatively close, could not be differentiated so far (Abu Ahmad *et al*., 2006b). These two genotypes were therefore combined and called genotype BRA-PER. In a study that included 18 geographical locations worldwide, only a single SCYLV genotype occurred in most sugarcane producing areas. Genotype BRA-PER was the most widespread and found in all locations, whereas genotypes CUB and REU were each found in four geographical locations only. Several genotypes of SCYLV were found in locations such as Brazil, Colombia, Guadeloupe, Mauritius and Réunion Island, suggesting different virus introductions and/or different evolution histories of the virus after its introduction into a new environment (Abu Ahmad *et al*., 2006b).

Resistance of sugarcane to infection by SCYLV

SCYLV cannot be eliminated from infected stalk cuttings by hot water treatment (Chatenet *et al*., 2001; Moutia, Saumtally, 2001). Virus-free plants can be produced by meristem tip culture (Chatenet *et al*., 2001; Fitch *et al*., 2001; Lehrer *et al*., 2001) and regeneration of plantlets from callus culture (Parmessur *et al*., 2002). These techniques are useful for quarantine and shipping of virus-free plants. However, they are not effective for control of yellow leaf in the field. When the aphid vectors exist in a contaminated sugarcane production area, susceptible sugarcane cultivars will be rapidly reinfected (Schenck, Lehrer, 2000). Sugarcane response to infection by SCYLV and to yellow leaf varies according to the variety, and numerous varieties can be infected by the pathogen without exhibiting disease symptoms (Schenck, 2001). High incidence of infection was reported in CP clones in Florida, suggesting that little resistance exists among these clones (Comstock, Miller, 2003). After inoculation of 29 commercial sugarcane varieties and eight clones of various *Saccharum* and *Erianthus* species with SCYLV in Colombia, incidence of the virus varied between 0 and 100% (Victoria *et al*., 2005). Differences in virus infection rates between different species of *Saccharum* were also reported in Hawaii (Schenck, Lehrer, 2000). In the world collection of sugarcane and related grasses in Florida, incidence of SCYLV ranged from 7% in *S. spontaneum*, the most resistant group, to 76% in *S. officinarum*, the most susceptible group (Comstock *et al*., 2001). Resistance to sugarcane infection by SCYLV and to yellow leaf therefore appears the most promising method to control the disease. In Colombia, a cross between a susceptible female parent and a resistant male parent resulted in mostly resistant progeny (Victoria *et al*., 2005).

In Hawaii, several locally produced varieties always tested virus-free using TBIA both in the field and after controlled inoculation with viruliferous aphids (Schenck, Lehreer, 2000). Other varieties generally gave 30% -35% positive blots in each test, while others gave 85-99% positive blots. Eventually, analyses using quantitative RT-PCR proved that these differences

were due not to infection percentages, but to virus titre (Zhu *et al*., 2010). The TBIA test failed to detect low virus titre that the more sensitive test detected. It is now known that infected Hawaiian varieties vary consistently in virus titre which may constitute a difference in physiological resistance to virus multiplication. No Hawaiian variety has so far been found to be completely resistant to infection.

Resistance to the virus measured as lack of infection in inoculated plants or as lower titer in transgenic lines was obtained through genetic transformation (Rangel, *et al*., 2005; Zhu *et al*., 2011). The nontranslatable SCYLV coat protein construct pFM395 used in these studies was developed by Moonan and Mirkov (1999). Genetic transformation may prove to be effective in producing resistant sugarcane, but raises the question of market acceptance and EPA clearance for commercial use.

Conclusion

Extensive research has been conducted on SCYLV since the discovery of the causal agent of sugarcane yellow leaf in 1996. Diagnostic methods were developed and important knowledge regarding the biology and genetics of SCYLV has been gained. However, more studies are needed to understand the sugarcane/SCYLV pathosystem, especially the interactions between the host, the pathogen and the aphid vector. Future research will most likely be focused on control of the disease, through conventional breeding and biotechnological methods.

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