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REVIEW

Biology and management of sugarcane yellow leaf virus: an historical overview

Abdelaleim Ismail ElSayed[1,5] · Ewald Komor[2] · Moncef Boulila[3] · Rasappa Viswanathan[4] · Dennis C. Odero[5]

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Abstract Sugarcane yellow leaf virus (SCYLV) is one of the most widespread viruses causing disease in sugarcane worldwide. The virus has been responsible for drastic economic losses in most sugarcane-growing regions and remains a major concern for sugarcane breeders. Infection with SCYLV results in intense yellowing of the midrib, which extends to the leaf blade, followed by tissue necrosis from the leaf tip towards the leaf base. Such symptomatic leaves are usually characterized by increased respiration, reduced photosynthesis, a change in the ratio of hexose to sucrose, and an increase in starch content. SCYLV infection affects carbon assimilation and metabolism in sugarcane, resulting in stunted plants in severe cases. SCYLV is mainly propagated by planting cuttings from infected stalks. Phylogenetic analysis has confirmed the worldwide distribution of at least eight SCYLV genotypes (BRA, CHN1, CHN3, CUB, HAW, IND, PER, and REU). Evidence of recombination has been found in the SCYLV genome, which contains potential recombination signals in ORF1/2 and ORF5. This shows that recombination plays an important role in the evolution of SCYLV.

✉ Abdelaleim Ismail ElSayed
aelsayed@zu.edu.eg

[1] Biochemistry Department, Faculty of Agriculture, Zagazig University, 44519 Zagazig, Egypt

[2] Plant Physiology, University Bayreuth, 95440 Bayreuth, Germany

[3] Institut de l'Olivier, B.P. 14, 4061 Sousse Ibn-khaldoun, Tunisia

[4] Division of Crop Protection, Sugarcane Breeding Institute, Indian Council of Agricultural Research, Coimbatore 641007, India

[5] Everglades Research and Education Center, Institute of Food and Agricultural Sciences, University of Florida, 3200 East Palm Beach Road, Belle Glade, FL 33430-4702, USA

Introduction

Sugarcane is an important crop that has served as a source of sugar for hundreds of years. The commercial sugarcane cultivars are interspecific hybrids that, under ideal conditions, are capable of storing sucrose in the parenchyma tissues of the stem up to 60 % of the dry weight [67]. Sugarcane has been used as the main source of sugar and, recently, to produce ethanol, an important renewable biofuel energy source. The growing global energy demand and a desire to reduce carbon dioxide emissions from fossil-based energy sources have resulted in increased interest in clean renewable biofuels such as ethanol.

Production of sugarcane can adversely be affected by plant pathogens, including viruses. Several viruses, including sugarcane mosaic virus (SCMV), sugarcane streak mosaic virus (SCSMV), sugarcane streak virus (SSV), sugarcane bacilliform virus (SCBV), sugarcane Fiji disease virus (SFDV), sugarcane mild mosaic virus (SCMMV), sorghum mosaic virus (SrMV), and sugarcane yellow leaf virus (SCYLV) infect sugarcane worldwide. Yellow leaf (YL) caused by SCYLV is one of the most important viral diseases affecting sugarcane. This disease has caused epidemics and loss of a major proportion of the crop in sugarcane production regions, including Brazil, where yield losses of up to 50 % have been reported [90]. Yellow leaf syndrome (YLS) was first observed in 1988 and 1990 in Hawaii [81, 82] and Brazil [90]. Soon thereafter, it was found in many other sugarcane-growing regions of the world [39], and was associated with a mollicute bacterium named sugarcane yellows phytoplasma

(SCYP) [8]. The most characteristic symptom of YL is a distinct yellowing of the lower surface of the leaf midrib, which can extend laterally to the leaf lamina. The yellowing of the midrib may turn pink or have a reddish tinge in some sugarcane varieties due to sucrose accumulation. These symptoms are not specific to YL and can be caused by various biotic and abiotic stresses [59, 82, 90]. There is now considerable evidence from several studies that have conclusively proven an association of SCYLV with YL [80, 90]. SCYLV is a member of the genus *Polerovirus*, family *Luteoviridae* [25]. It is phloem restricted and is transmitted from plant to plant by aphids in a persistent, circulative, and non-replicative manner. Characterization of the genome of SCYLV [66, 87] has revealed a ~6-kb single-stranded RNA genome with six recognized open reading frames (ORFs 0-5) with three untranslated regions (UTRs) that are expressed by a variety of mechanisms [65, 87]. The pathogenic nature of SCYLV was formerly disputed because symptoms were not very specific, and the presence of the virus did not strictly correlate with the symptoms.

The spread and distribution of SCYLV has been attributed to exchange of infected breeding material between global breeding programs. SCYLV exhibits significant genetic diversity, and eight different genetic groups, namely BRA (Brazil), CHN1 and CHN3 (China), CUB (Cuba), HAW (Hawaii), IND (India), PER (Peru), and REU (Réunion Island), have been described based on phylogenetic analysis of partial and/or full-length genome sequences [3, 19, 31, 34, 58, 95] (Fig. 1). Hawaiian isolates of the pathogen differ from the other reported genotypes because they contain a deletion of 48–54 nucleotides (nt) in ORF1 [30, 34]. Variation in pathogenicity among genotypes of SCYLV has also been reported [2]. However, limited information is available on the characteristics of epidemics of YL. In this review, we discuss the epidemiology, biology, and genome characteristics of SCYLV, and the contribution of recombination to its evolution.

Host range of SCYLV

Most luteoviruses have a limited host range, with the exception of beet western yellows virus (BWYV) which has a very wide range of dicotyledonous hosts. Beet mild yellowing virus (BMYV) is the most common beet-infecting luteovirus. While both potato leaf roll virus (PLRV) and BWYV have the same vector, their host ranges are quite different, indicating that the host range of a plant virus is not determined merely by the host range of its vector. In contrast, barley yellow dwarf virus and cereal yellow dwarf virus infect most, if not all, members of the family *Poaceae*.

The host range of the sugarcane aphid *Melanaphis sacchari* (Zehntner) is restricted to members of the genera *Oryza*, *Panicum*, *Pennisetum*, *Saccharum*, and *Sorghum* [27, 86]. Several studies have been conducted to investigate the host specificity of SCYLV and to determine possible alternative sources of viral infection. Schenck and Lehrer [84] placed viruliferous *M. sacchari* on cereal grass seedlings and tested the plants for SCYLV by tissue blot immunoassay (TBIA) after 4 weeks. They observed that more than 90 % of the inoculated wheat (*Triticum aestivum* L.), oats (*Avena sativa* L.) and barley (*Hordeum vulgare* L.), and 10 % of rice (*Oryza sativa* L.) and corn (*Zea mays* L.) contained SCYLV. These results were surprising, because the cereal grasses are less closely related to sugarcane than *Miscanthus* or *Erianthus*, which were SCYLV resistant [46]. In contrast, Komor [46] reported that wheat, rice, and corn in fields next to infected sugarcane fields did not acquire SCYLV during their growth. The same observation was true for wheat, rice, corn, barley, and oats grown in pots outdoors together with pots of infected sugarcane. In contrast, ElSayed [29] reported the first successful transmission of SCYLV by *M. sacchari* to corn plants. Therefore, we speculate that corn can be an alternative host for SCYLV, but because sugarcane may be the preferred host for *M. sacchari*, corn next to sugarcane plants may remain SCYLV free [46]. The morphological similarity between sugarcane and corn leaves might be one of several reasons that aphids (*M. sacchari*) are able to colonize corn. Therefore, future work should be focused on sequencing SCYLV genomes isolated from infected corn to provide more information regarding characterization and identification of possible new virus strains associated with corn. In 2013 and 2014, large populations of *M. sacchari* were found infesting grain sorghum (*Sorghum bicolor*) and johnsongrass (*Sorghum halepense*) in northeast Texas, Oklahoma, eastern Mississippi, northeastern Mexico, Louisiana, and Florida (Fig. 2). These infestations caused up to 50 % grain sorghum yield losses in Texas [91].

Aphid transmission of SCYLV and symptom expression in infected plants

SCYLV can be transmitted from infected to healthy sugarcane by the common aphids sugarcane aphid (*M. sacchari*) and corn leaf aphid (*Rhopalosiphum maidis*), but not by mechanical transmission [80]. So far, a high percentage of transmission of the virus to sugarcane has only been observed with *M. sacchari* [80]. For example, Rassaby et al. [75] observed that of the two aphid species (*M. sacchari* and *R. maidis*) that are known to be able to transmit SCYLV, *M. sacchari* was the more common in Réunion. Some other phloem-feeding aphids commonly

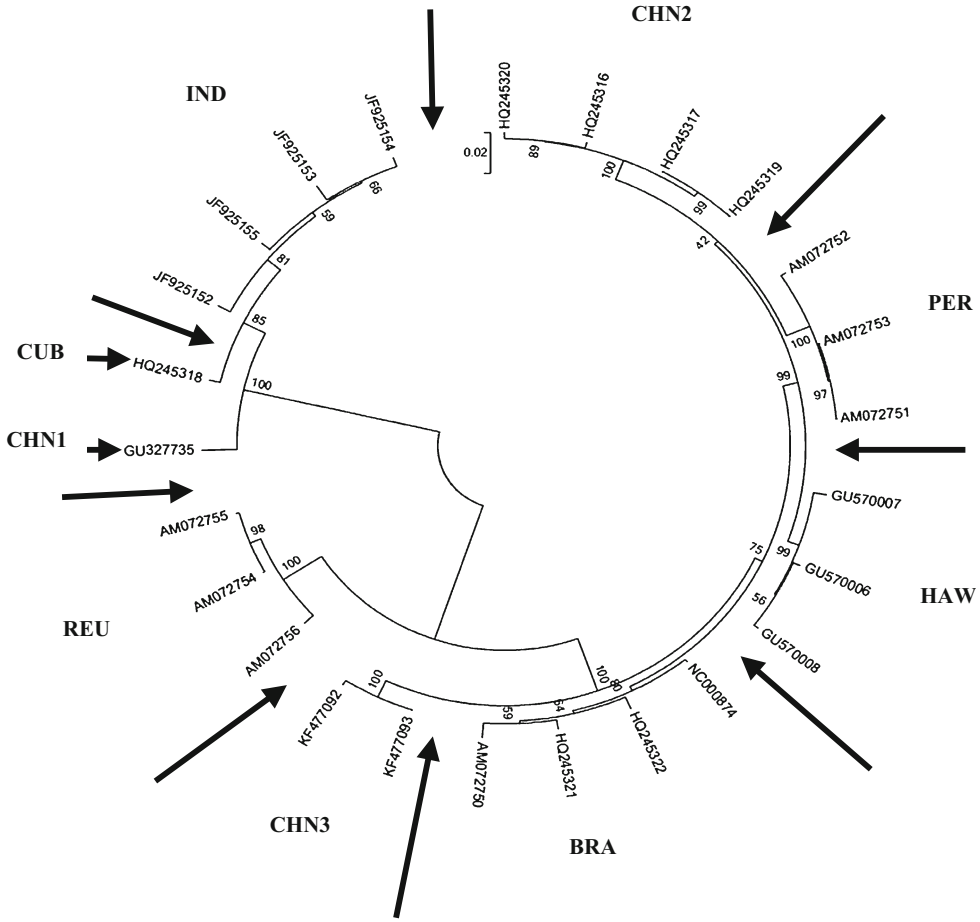

Fig. 1 Phylogenetic tree constructed based on the nucleotide sequence of the RdRp, the most variable segment of the SCYLV genome. The tree was constructed with 25 isolates of SCYLV using the ML algorithm under the assumption of Model K2+G in the MEGA6 software. Bootstrap analysis was performed with 1,000 replicates. The numbers above the branches indicate the bootstrap confidence value. The scale bar shows the number of substitution per nucleotide. The GenBank accession numbers of the sequences used are as follows: NC_000874; SCYLV-A,HQ245316; CHN-GD-JM2, HQ245317; CHN-GD-WY19, HQ254318; CHN-GD-WY20, HQ245319; CHN-GD-ZJ4, HQ245320; CHN-GD-ZJ15, HQ245321; CHN-GD-ZJ17, HQ245322; CHN-YN-KY2, GU570006; HAW87-4094, GU570007; HAW87-4319, GU570008; HAW73-6110, GU327735; SCYLV-CHNl, AM072750; BRA-YL1, AM072751; CHN-YL1, AM072752; PER-YL1a, AM072753; PER-YL1b, AM072754; REU-YL1a, AM072755; REU-YL1b, AM072756; REU-YL2, JF925152; IND1, JF925153; IND2,JF925154; IND3, JF925155; IND4, KF477093; HN-CP502, KF477092; GZ-GZ18

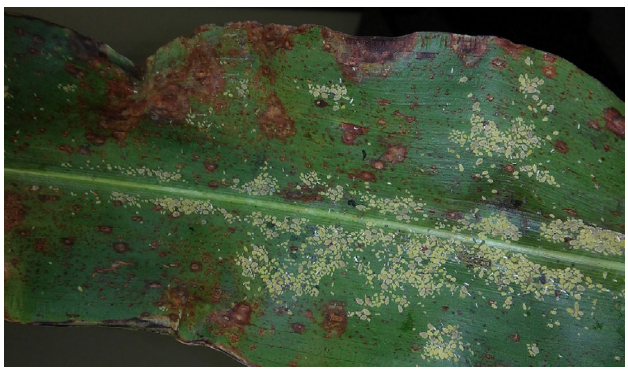

Fig. 2 Sugarcane aphid (*Melanaphis sacchari*) outbreak in sorghum in autumn 2014 in Florida, USA. The leaf showed damage and black sooty mold, which was indicative of a heavy aphid infestation. The picture was taken by Dr. A. ElSayed

found on sugarcane and other plants in Hawaii also transmitted SCYLV, but much less efficiently [54]. In Brazil, the yellow sugarcane aphid (*Sipha flava*) also transmitted SCYLV [60].

Within sugarcane fields, the most important proliferation of SCYLV occurs by planting infected stem cuttings or sections of stalks called "setts". The progression of viral infection via aphids has been estimated to be in the range of 2–5 m per year [54]. The aphids either walk or are carried by ants or strong winds [54]. Also, ambient conditions play a role in the speed of infection progression, as observed by differences in infection rates of plants in the border rows at different locations. Lehrer et al. [54] studied the transmission of SCYLV over middle distances in several plantings of a virus-free cultivar, H87-4094, at

different locations isolated from sugarcane plantations in Hawaii. In that study, at heavily aphid-infested and SCYLV-infected sites at a sugarcane breeding station and a field station, the infection pressure was very high, such that 80 % of virus-free plants became infected within four months. In contrast, virus-free plants at a distance of 1 km from the breeding station remained completely virus free after 4 months. Also, a large plot of virus-free sugarcane 15 km away from infected sugarcane plants remained virus free after 20 years [47]. A 100-m-wide swath of resistant cultivars planted between a plot of infected sugarcane and a plot of susceptible, virus-free sugarcane proved sufficient to completely prevent infection of the virus-free plants for at least 15 months. This shows that propagation of infection by aphids proceeds slowly and sporadically, in the range of a few metres per year. A similar sporadic and patchy spread of infection was observed in test fields on Réunion Island [75]. This may be different in other sugarcane-growing regions, where infectious aphids may be moved over greater distances by wind. For example, SCYLV infection has been reported to spread at a rate of 20–80 % in Florida within 18 months [23]. Generally, the geographical distribution of *M. sacchari* follows the cultivation of sorghum and sugarcane worldwide and covers Angola, Brazil, China, Colombia, Ecuador, Egypt, Ethiopia, Haiti, Hawaii, India, Indonesia, Japan, Jamaica, the Middle East, Nigeria, Pakistan, Peru, the Philippines, Sudan, Thailand, Trinidad, Tobago, Uganda, and Venezuela [86]. The genus *Melanaphis* has 20 species that are associated with members of the family *Poaceae* [12].

SCYLV is often present in infected plants without visible disease symptoms. However, when expressed, the major symptom is leaf yellowing (Fig. 3), appearing first on the abaxial midrib surface and leaf tip of leaves 3–6 from the top expanded spindle leaf. The disease etiology resembles senescence; however, it occurs on younger leaves where senescence would be least expected. The lower surface of the midrib develops a distinct yellow colour, while the upper surface may be unchanged or develop a yellow, pink, or reddish coloration. In addition, symptoms of YL also include shortening of terminal internodes, necrosis of leaves, and sucrose accumulation in the midribs. In many cultivars, the yellowing spreads laterally from the midrib into the lamina, and leaves begin to die from the tip, while in other cultivars, a general yellowing of the leaves occurs. The leaf blade can also become bleached, proceeding from the tip toward the base of the leaf. Lehrer and Komor [50] reported different grades of leaf yellowing during the infection period of sugarcane by SCYLV. The leaf yellowing started from the midrib, proceeded successively to the leaf blade and finally led to completely dry leaf edges. This progression of yellowing was different from yellowing related to plant age or nutrient shortage. Generally, visible symptoms of YL are most often not expressed until late in the growing season as the plant matures [20, 59]. It is notable that some studies have shown that the incidence of yellowing symptoms in sugarcane is not correlated with the presence of SCYLV [5, 87]. Leaf yellowing in sugarcane is not specific to SCYLV, because several biotic and abiotic factors such as nutrient deficiencies or excesses [13, 62], cold or water stresses [22], and phytoplasma infection [87] can cause these symptoms. Whereas Lehrer and Komor [50] found a clear relationship between the presence of SCYLV and YL symptoms [50], these symptoms, as mentioned previously, were in different grades of leaf yellowing. For example, susceptible and moderately susceptible sugarcane cultivars showed severe symptoms, whereas virus-free and resistant sugarcane cultivars rarely showed yellowing leaf symptoms, and if they did, these symptoms were mostly mild [50].

The highly infected cultivar H78-3606 exhibited few symptoms, as did the resistant cultivars, whereas the intermediately infected cultivar H65-7052 showed symptoms as severe as those of the strongly infected susceptible cultivars H73-6110 and H87-4094 [50]. Based on these studies, the correlation between the presence of SCYLV and YL symptom expression is not well understood. Interestingly, some Hawaiian cultivars consistently express a high percentage of leaf symptoms, but the expression of symptoms fluctuates with plant age, as observed with some Hawaiian cultivars and not with other sugarcane cultivars [50]. Consequently, several aspects of these inconsistencies should be investigated – for example, the reliability of the SCYLV detection method and symptom determination and factors other than SCYLV that result in an increase or suppression of symptom expression. Yellow leaf symptoms are normally expressed in 6–8 months in the field. However, Chinnaraja et al. [19] recently observed yellow leaf symptoms at 30 days in sugarcane cv. B38192. Also, ratoon crops exhibited earlier disease expression.

Geographical distribution of SCYLV

SCYLV is widespread in most sugarcane-producing regions of the world. The first report of SCYLV was in Hawaii in the late 1980s [81, 83], and it was later reported in other sugarcane-growing regions of the world [4, 7, 9, 10, 21, 22, 24, 32, 68, 75, 87, 90, 92]. Abu Ahmad et al. [4] reported that SCYLV genotypes BRA-PER, CUB, and REU were found in 137 (56 %), 51 (21 %), and 82 (33 %), respectively, of the 245 sugarcane samples from different geographical regions around the world. These three genotypes of SCYLV are not distributed uniformly across the world. Genotype BRA-PER has been found in 18

Fig. 3 **a** Severe (*Melanaphis sacchari*) colonization in sugarcane. The picture was taken by Dr. A. ElSayed. **b**) Anatomy of the aphid showing how it feeds in the phloem, **c**) The symptoms of SCYLV. Pictures b and c were taken by Dr. E. Komor. **d**) Extensive foliage drying in the maturity phase of the crop due to SCYLV infection. The picture was taken by Dr. R. Viswanathan. This foliage drying causes serious inhibition of crop growth in the field

sugarcane-growing locations (Table 1) in Africa, Asia, and North, Central, and South America, whereas genotypes CUB and REU have been found in only four locations [4]. The worldwide distribution of genotype BRA-PER suggests that YL was originally caused by this genotype, which was spread worldwide by infected plant material when the causal agent of YL was unknown and not intercepted in sugarcane quarantines [16]. Interestingly, the genotype BRA-PER is present worldwide, but its incidence varies according to the sugarcane-growing location [4]. In contrast, the genotype CUB has only been found in South America (Brazil, Colombia) and the Caribbean (Cuba, Guadeloupe). Genotype REU has only been found in locally bred varieties in Guadeloupe, Brazil, Mauritius, and Réunion Island (Table 1), and phylogenetic analysis has shown it to be almost separated in a unique group [3, 19, 30, 34, 38, 96]. The limitation in the geographical distribution of CB and REU genotypes is probably attributed to environmental conditions and/or interactions. Thus, it is reasonable to assume that the aphid vector is important in the evolution of luteoviruses [4]. Furthermore, changing the environment or replicative niche of the virus may have different costs to fitness. The changing environment becomes important in many plant viruses that have a broad host range or that can be transmitted by vectors of several different species [29, 32].

Physiological impact of SCYLV

Infection of sugarcane by SCYLV is characterized by a backup of carbohydrates, mostly starch, in the source leaves and a shift of enzymes that are involved in sucrose and starch metabolism. This is accompanied by ultrastructural changes of bundle sheath and mesophyll chloroplasts and, finally, degradation of chlorophyll, resulting in leaf yellowing [40, 53, 99]. These observations have led to the conclusion that assimilate export is inhibited by SCYLV infection, either by a lack of sucrose transporters or by a physical block of sieve tube mass flow by callose deposition. Stems of susceptible cultivars have less hexoses and starch than resistant cultivars. This has been observed in infected versus virus-free plants of cv. H87-4094 [53]. In general, lower hexose and starch content indicates a more mature state of sugarcane internodes. Therefore, it appears that stem internodes of SCYLV-susceptible cultivars are faster in ripening than resistant cultivars, a feature that is observed in the extreme when sugarcane plants became symptomatic [53]. Because sucrose is delivered to the stem and hexose is produced by invertase, high hexose content will favour starch synthesis. The differences in carbohydrate composition indicate that SCYLV susceptibility has an impact on the carbohydrate physiology of the plants [56]. Lehrer and Komor [51]

Table 1 Dispersal of sugarcane yellow leaf virus genotypes in sugarcane-growing regions of the world

Sugarcane cultivars	Country/ location of origin	SCYLV genotypes	References
B85287, B86776, B9702, BT74209, BT7748, Co6806, DB8811, DB89138	Barbados	BRA-PER	Abu Ahmed et al. [3]
RB80-6043, RB84-5257, RB85-5113, SP71-1406, SP71-6163, SP77-5181, SP81-1763, RB83-5054, SP81-3250, SP83-5073, R570	Brazil	BRA-PER, CUB, REU	Abu Ahmed et al. [3]
CGT73-167, CMT70-611, CP70-1133, CYT93-159, CYT85-1589, CYZ81-173,FR93435, ROC3, FN96-0907, GT93-102, GT96-287, CP93-1309, CGT63-167, CP-4950, Ganzhe 18, Yunrui99-601	China	BRA-PER, CHN1, CHN3	Abu Ahmed et al. [3, 4]; ElSayed et al. [31, 34]; Chinnaraja et al. [19]; Lin et al. [58]
C1616-75, CC92-2227, CC92-2376, CC92-2885, Co421, CP38-34, JA64-20,PR77-3007, SP71-6163, C1051-73, C236-51, C2655-74, CC84-75, CC85-96, CC87-505, CC92-2867, CC92-2882, CC92-2885, CC93-3811, CP38-34, Mex64-1487, POJ2878, CC85-96	Colombia	BRA-PER, CUB	Abu Ahmed et al. [3]
JA64-11, CP43-62, C13-281	Cuba	BRA-PER, CUB	Abu Ahmed et al. [3]; ElSayed et al. [34]
CP85-1491, CP86-1633, CP89-2143, CP87-3388	Florida	BRA-PER	Abu Ahmed et al. [3]
B37-161, CP67-412, FR9453, SP79-1169, B68409, FR9707, FR9976, FR99101, FR0020, FR0048, FR00102, FR00170, SP71-6163, CB56-171, FR90714, FR9707, FR9856, FR9979, FR9983, FR00306, R570, R578,SP71-3146, FR99273, FR9604	Guadeloupe	BRA-PER, CUB, REU	Abu Ahmed et al. [3]
R570, H73-6110, H87-4094, H87-4319, H78-3606, H78-7750	Hawaii	HAW-PER	Abu Ahmed et al. [3]; ElSayed et al. [34]
B 38192, Co 86010, CoC 85061, CoLk 97154, Co 86032, CoV 92101, CoV 92102, Co 93009, Co 62399, Co 7219, Co C671, Co99016, CoJn 862035, CoLk 97154,93 A 53,Black Tanna,D1135, Co 6304, Co 85019, Co 91010, Co 94005, Co 94006, Co 94008, Co 99016, CoC 671, CoV 94101	India	IND, BRA/ HAW-PER	Viswanathan et al. [92]; ElSayed et al. [34]; Chinnaraja et al. [19]
TC2, TC4	Malaysia	BRA-PER	Abu Ahmed et al. [3]
B7623, B7656, FR91180, FR91485, FR91816	Martinique	BRA-PER	Abu Ahmed et al. [3]
M124/59, M253/58, M1186/86, M2024/88, R570, SP71-6163, M1658-78, M2350-7, M99/48	Mauritius	BRA-PER, REU, BRA/ HAW-PER	Abu Ahmed et al. [3, 4]; Viswanathan et al. [92]; ElSayed et al. [34]; Chinnaraja et al. [19]
H32-8560, H50-7209	Peru	HAW	Abu Ahmed et al. [3, 4]; Viswanathan et al. [92]; ElSayed et al. [34]; Chinnaraja et al. [19]
R570, R569, M1371/79, R579, R576, R577, AY7, R490, R81-0834,S17, R84-0408, R85-1102	Réunion Island	REU	Abu Ahmed et al. [3, 4]; ElSayed et al. [34]; Chinnaraja et al. [19]
CP72-2086, CP85-1491, CP86-1664, NA6390	Senegal	BRA-PER	Abu Ahmed et al. [3]
SLC9225, SLC9272, SLC9273, SLC9294	Sri Lanka	BRA-PER	Abu Ahmed et al. [3]
KnB96207, KnB96223	Sudan	BRA-PER	Abu Ahmed et al. [3]
ROC6, ROC10	Taiwan	BRA-PER	Abu Ahmed et al. [3]

observed that the larger decrease in the assimilation rate compared to stomatal conductance led to a higher internal carbon dioxide concentration in the symptomatic leaves compared to the asymptomatic leaves. In this regard, the symptomatic leaves behaved like leaves under salinity stress, which led to a higher internal carbon dioxide concentration [64].

ElSayed et al. [33] compared the expression of the sucrose transporter gene (ShSUT1A) in virus-free sugarcane and in infected sugarcane and observed a slightly higher transcript level in infected plants. Therefore, the previous reports that SCYLV-infected plants seem to suffer under assimilate export inhibition cannot be traced back to a lower expression of sucrose transporter ShSUT1A in source leaves. The SCYLV-infected internodes, which definitely contain the virus in the companion cells of the bundles, also seem to contain a higher transcript level than internodes of virus-free plants [33], which would

appear to conform to the slightly higher sucrose levels in stems of infected plants [53]. However, a high sucrose level may also result from premature maturation because of virus-caused inhibition of apical growth, which occurs when infected plants turn symptomatic [50]. Deficiencies in sucrose transporter expression (assuming that the transcript levels mirror the protein levels) is not the cause for decreased assimilate export in infected plants. The viral movement protein increases the size exclusion limit of plasmodesmata and may thus inhibit phloem loading, as was postulated for transgenic plants expressing the movement protein of potato leafroll luteovirus [42]. This might be due to the movement protein containing domains that interfere with the regulation of photoassimilate translocation and partitioning [71]. However, there may also be indirect effects of viral infection on metabolism or growth, such as inhibition of sugar transport proteins, sugar signaling, or metabolic network regulation [44, 101]. Regarding carbohydrate metabolism and starch content in SCYLV-infected plants, Yan et al. [99] reported that SCYLV-infected leaves had significant amounts of starch grains in Kranz cells and mRNA of ADP-glucose pyrophosphorylase (AGPase) was detected in bundle sheath and Kranz cells, of infected leaves. AGPase is thought to be the enzyme that controls starch biosynthesis [70]. Furthermore, Yan et al. [99] found lower levels of metabolites in the young leaves of infected plants than in virus-free plants. These metabolites act as precursors for sucrose and starch biosynthesis, which may be a consequence of a higher sucrose and/or starch biosynthesis rate. In contrast, in older leaves, where assimilates backed up, these metabolites increased, possibly by reversal of sucrose synthase activity in the direction of UDP-glucose. The increase in adenosine triphosphate (ATP) concentration in infected leaves may reflect photosynthetic and respiratory ATP generation without an equivalent consumption of ATP. Elevated AGPase in bundle sheath cells and Kranz mesophyll indicates that SCYLV infection directly or indirectly stimulates transcription of these genes in these cells and also probably increases enzymatic activities [99]. SCYLV appears confined to the sieve tube-companion cell complex. Neither SCYLV RNA nor SCYLV coat protein has been observed outside the phloem, which shows that the effects on carbohydrate metabolism enzymes in bundle sheath and Kranz mesophyll must be indirect and are probably a result of sugar backup due to export inhibition. Conceivably, the presence of elevated levels of viral movement protein reduces the osmotic pressure in the phloem by counteracting the pressure buildup by active sucrose transport. Unfortunately, pressure determinations in sieve tubes are not possible so far in sugarcane (or any plant) [99]. The change in chlorophyll content in infected sugarcane has been reported by Yan et al. [99]. They observed that the change in the chlorophyll a/b ratio in infected sugarcane leaves was similar to the change in the chlorophyll a/b ratio at the onset of senescence, even though the change in the total chlorophyll content was only marginal. A lower chlorophyll a/b ratio had been observed under low-light conditions, where it is thought to be an adaptation that broadens the light absorption spectrum [6].

Viswanathan et al. [93] studied the physiological changes of SCYLV infection in sugarcane by comparing symptomatic, asymptomatic and virus-free plants derived through meristem culture. Virus infection in sugarcane adversely affected various physiological parameters, such as photosynthetic rate, stomatal conductance, and chlorophyll concentration in symptomatic plants of the susceptible varieties. The chlorophyll concentration in the leaves was measured using a SPAD meter. There was a reduction of 44-57 %, 47-48 %, 36-47 %, and 30-34 % in photosynthesis rate, stomatal conductance, transpiration rate, and chlorophyll concentration, respectively, in symptomatic plants. Also, virus infection resulted in a reduction of 31-33 % in the leaf area index in symptomatic plants.

Sugarcane yield losses from SCYLV infection

SCYLV is considered to be the most important viral disease of sugarcane worldwide that can cause significant yield losses. Losses in yield due to YL have been reported in several sugarcane-growing regions. Yield losses of 50 % have been reported in Brazil [59], up to 14 % in sugar yield loss in Louisiana [41], 11 % loss of both stalk weight and sugar yield [23] and 14 % in sugar yield loss [37] in Florida, and 11 % and 28 %, respectively, for loss of sugar content and stalk weight in Réunion [75]. In Thailand, YLS reduced sugarcane yield up to 30 %, even when the plants were asymptomatic [52]. SCYLV infection reduced plant growth and juice yield by 39-43 % and 30-34 %, respectively, in susceptible varieties at harvest in India [93]. Sugarcane yields are further decreased when plants are infected by SCYLV in combination with phytoplasma [5].

When SCYLV-free and SCYLV-infected plants of the same cultivar (H87-4094) were compared in Hawaii, Lehrer et al. [55] reported visual differences in growth between the virus-free and infected plants during the first five months. In addition, the stalk number was significantly higher (44 %) in the virus-free sugarcane than in the infected cane when sugarcane was harvested after 11 months. This reduction in stalk number resulted in a 40 % higher biomass in the virus-free plants compared to the infected plants at 11 months. There was no significant difference in sugar concentration, but the sugar yield per plot was 35 % higher in the SCYLV-free plot because of

the difference in biomass. This difference in biomass and sugar yield was transient and had disappeared when the plants were harvested after 16 or 24 months. Because 18 % of the originally virus-free plants became infected during growth in the fields, any small yield advantage of the virus-free plots may have been obscured, and more repetitions would have been necessary to determine possible differences [55]. In addition, Rassaby et al. [76] reported 46 % reduction of stalk weight of the ratoon crop (versus 28 % in the plant cane crop), 13 % reduction of stalk diameter (versus 7 % in the plant cane crop), and significant reduction in tonnage (37 %) as a result of SCYLV infection of cultivar R57. Additionally, significant losses in sugar content (12 %) due to reduced amount and quality of extracted cane juice have been reported. Some studies reported observation of leaf yellowing symptoms in both virus-infected and virus-free plants [52, 76]. These observations are probably attributed to the presence of other pathogens that can interfere with sugarcane growth and increase the severity of leaf yellowing. For example, combined infection of lettuce with beet western yellows virus (BWYV) and lettuce mosaic virus (LMV) resulted in a significantly greater yield loss than that caused by BWYV or CMV infection alone [95]. When sugarcane exhibits mosaic and another disease simultaneously, growth and yield are reduced more than when the plants have only one of these diseases [45].

Elimination of SCYLV

Unlike fungal and bacterial pathogens, viruses are difficult to eradicate by the hot-water treatments used in quarantine protocols [18]. Therefore, such treatments cannot be used to eradicate SCYLV from infected stalks [80]. There are three methods used for elimination of viruses from plants, including thermotherapy, tissue culture, and chemotherapy. However, thermotherapy and chemotherapy often fail to eliminate pathogens when used alone, but their combination with the meristem culture technique has given satisfactory results [11, 74, 97]. The meristem culture technique is the most widely used method for virus elimination in meristematic tissues of apical shoots. This technique takes advantage of the fact that many viruses fail to invade and replicate in the meristematic region [35]. Transfer of the meristem dome, together with one or two leaf primordia, to a culture medium and development into a plantlet may lead to the elimination of viruses. Successful elimination of sugarcane mosaic virus and Fiji disease virus in sugarcane using apex or bud culture has been reported [57, 94]. Meristem tip, axillary bud, and callus culture may be used for elimination of SCYLV from commercial and noble sugarcane cultivars with variable rates of success [16, 36, 72, 74]. The success of meristem tip culture depends on the ability to dissect the meristematic dome with one or two leaf primordia from the mother plant and its successful regeneration. Larger meristem tips (>1 mm) are likely to be infected, whereas smaller ones (<0.3 mm) are unlikely to develop into plantlets [16]. This implies that not all meristem tips established would be guaranteed to be virus-free, emphasizing the need for sensitive diagnostic tools for disease indexing. However, Fitch et al. [36] reported that all plants regenerated from callus derived from meristems or buds produced virus-free plants and remained free from SCYLV for at least 4 years, with the exception of two meristem explants that were ≥1 mm. Chatenet et al. [16] reported that apical meristem culture was an efficient method for the elimination of SCYLV, with a 92 % success rate. In contrast, Parmessur et al. [72] reported 64 % disease-free plantlets with the apical meristem culture. These authors also reported that it is possible to eliminate the virus from infected plants by culturing callus derived from leaf rolls. The elimination of SCYLV using meristem culture may be attributed to the uneven distribution of the virus in the different tissues of the leaf.

Breeding for SCYLV resistance

The resistance and susceptibility of sugarcane cultivars to SCYLV has been investigated in many sugarcane-breeding programs, but the complexity of the sugarcane genome has so far prevented information on the genetics of the disease from being obtained [39, 43]. Seventy percent of Hawaiian commercial sugarcane hybrids have been reported to be SCYLV susceptible [47]. Using hybrids obtained by crossing breeding lines of Hawaiian cultivars, it was shown that the progeny of a susceptible female plant yielded 75 % susceptible plants, and that from a resistant female cultivar yielded 90 % resistant plants (male parents are mostly unknown because of polycross breeding). In addition, a cross between an SCYLV-resistant *S. robustum* (cv. 'Mol 5829') and an SCYLV-susceptible *S. officinarum* (cv. 'LA Purple') yielded 85 % resistant progeny clones, which indicated that SCYLV resistance is a dominant trait [47].

Zhu et al. [102, 103] engineered transgenic sugarcane plants containing a untranslated fragment of the coat protein to reduce the incidence of SCYLV. Six out of nine transgenic lines had an at least 103-fold lower SCYLV titre than the susceptible parent line H62-4671, whereas no difference was found between plants containing NPT II and the non-transformed parent [103]. Yield tests of SCYLV-free lines in the field obtained by meristem culture showed that the absence of SCYLV in a commercial cultivar increased yield for at least a one-year crop cycle [55]. A

field study using cv. H65-7052 showed that field plots with plants of higher virus titer developed YLS, resulting in 54–60 % lower cane and sugar tonnage compared to field plots with plants of low virus titer [102]. Therefore, the transgenic approach to producing high-yielding sugarcane cultivars with resistance to SCYLV seems to be a valuable option for regions with high incidence of the virus, such as Hawaii [103].

Classification, genome organization and gene functions

Luteoviruses have been classified into three genera, namely, *Luteovirus*, *Polerovirus*, and *Enamovirus*, based on their genomic organization, replication strategy, and expression mechanism. RNA sequences of SCYLV revealed that the virus belongs to the genus *Polerovirus*, family *Luteoviridae* [25], but it originated by recombination and had ancestors from all three of these genera (Fig. 4) [61, 87].

SCYLV has a positive-sense, single-stranded genomic RNA (+ssgRNA) of ~6 kb with a small protein, VPg, linked to its 5′ end [66]. Its RNA genome contains six major open reading frames (ORFs) that are expressed by a variety of mechanisms [63]. The three 5′-proximal ORFs are translated directly from the genomic RNA and include ORF1, encoding the 72.5-kDa viral protease and ORF2, which is translated via a ribosomal frameshift within ORF1 to yield the 120.6-kDa viral replicase. ORF2 shows the most similarity to the RNA-dependent RNA polymerase (RdRp) genes of the *Polerovirus* [87]. Three other ORFs are expressed via a subgenomic RNA synthesized in infected cells and include ORF3, encoding the 21.8-kDa viral capsid protein, and ORF4, encoding a 17-kDa putative movement protein. ORF4 permits infection of the phloem tissue of the entire plant [17]. The homologue of ORF4 in PLRV has many biochemical properties of a cell-to-cell movement protein, including nonspecific single-stranded nucleic acid binding and the ability to be phosphorylated and localized to the plasmodesmata [85].

ORF5, which is necessary for aphid transmission [17] and also involved in virus movement, is expressed as a readthrough protein with ORF3 (capsid protein) and codes for a putative aphid transmission factor (PATF). It encodes a 52.1-kDa protein that presumably is involved in aphid transmission of the virus [79, 87]. The poleroviruses have an extra ORF (0) at the 5′ end that is absent in barley yellow dwarf virus (BYDV). SCYLV ORF0 begins at the first AUG codon in the sequence and encodes a 30.2-kDa protein that functions as suppressor of RNA silencing [73]. ORF0 from PLRV induces virus symptoms on its own [89].

Recombination and evolution

Changes in environment, expanding host range, new agricultural practices, and the increasing global movement of human populations and plant products all enforce the heterogeneous nature of plant viruses [78]. Adaptation of living organisms to a changing environment through evolution, which has generated the considerable variability that we encounter every day, requires a compromise between genetic variation and phenotypic selection.

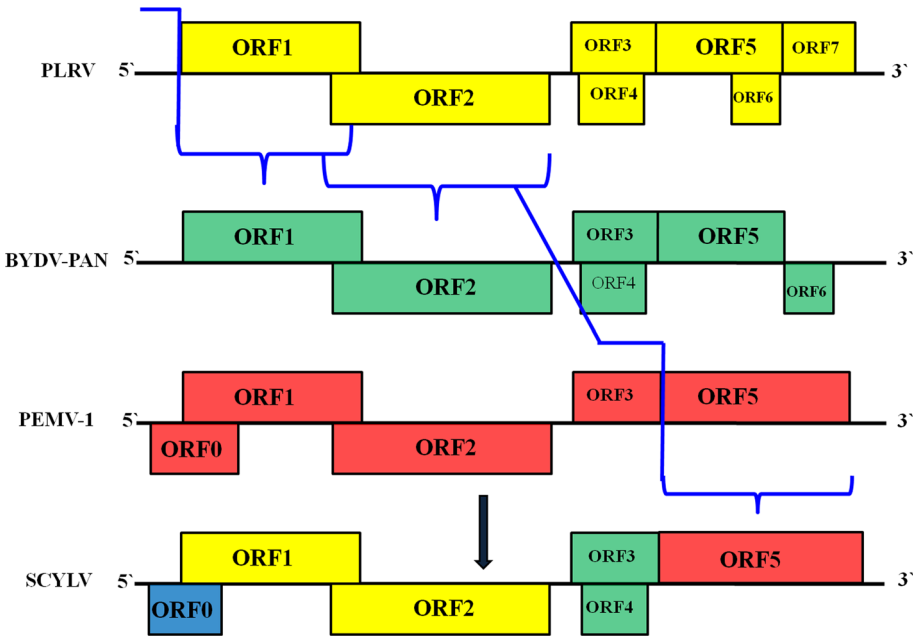

Fig. 4 Evolutionary pathway proposed for the emergence of SCYLV, a member of the family *Luteoviridae*. As shown in the illustration, SCYLV is an emerging virus that resulted from recombination of ancestors belonging to the genera *Luteovirus* (BYDV: barley yellow dwarf virus), *Polerovirus* (PLRV: potato leafroll virus) and *Enamovirus* (PEMV-1: pea enation mosaic virus 1). The graph is based on the results of Maia et al. [61] and Smith et al. [87]

Viruses, particularly RNA viruses, have been shown to have high variability due to evolutionary forces including mutation, reassortment (for viruses with a segmented genome), and recombination [28, 77]. The high mutation rate observed in viral RNA replication is attributed to the lack of proofreading-repair activity of viral RNA-dependent RNA polymerases (RdRp).

RNA recombination is thought to rescue viral genomes by repairing mutation errors in essential viral genes or in structures that could be introduced during RNA replication [14, 49]. Recombination events may play an important role in generating genome diversity. It has been shown that RNA recombination enables exchange of genetic material not only between the same or similar viruses but also between distinctly different viruses [98]. Furthermore, it also results in crossovers between viral and host RNA [1, 69]. Recent reports strongly suggest that RNA recombination is linked to virus replication and that it occurs by a copy-choice mechanism. Inter-species recombination has frequently occurred in the evolution of members of the *Luteoviridae*. RNA recombination events probably created the divergence observed between members of the genera *Luteovirus* and *Polerovirus* [26]. Two major forces, recombination and positive selection, drive the molecular evolution of viruses. An essential step in any phylogeny-based analysis is to screen for and quantify evidence for recombination [48]. Generally, recombination rates vary considerably among plant RNA viruses. This might be due to the different levels of precision of viral replication proteins (i.e., variations in the error-prone nature of the replicase) during RNA replication and the presence or absence of recombinationally active sequences (recombination hotspots). However, environmental and host effects are likely to influence the rate of RNA recombination, in addition to the better-characterized viral factors. Natural selection of the recombinant and parent viruses ensures the survival of only the fittest. Depending on the precision of recombination events, RNA recombination can lead to various genetic changes. These include sequence insertions and duplications if the recombination end breakpoint in one of the recombining RNAs is upstream relative to the endpoint of the other RNA. Reversal of the positions of recombination endpoints on the viral RNAs can lead to deletions. Furthermore, exchanged genetic material may lead to a progeny through different mechanisms, such as intramolecular recombination when polymerases switch templates [98], or homologous or non-homologous recombination. The most variation in the RNA sequence and deduced amino acid sequence of SCYLV was found in the RNA-dependent RNA polymerase gene, as reported previously by Moonan and Mirkov [65]. Recombination events located in the RdRp domain of the Hawaiian SCYLV isolates were detected by ElSayed et al. [30] using two methods (RDP v.4.3 and RECCO), which revealed that the two Hawaiian isolates (Haw73-6110 and Haw87-4094) were recombinants.

Viruses with RNA genomes are known to have mutation rates per site per replication that are three to four orders of magnitude higher than those of viruses with DNA genomes [28]. This difference is attributed to the error-prone nature of the viral RNA-dependent RNA polymerase. Like many other plant RNA viruses, SCYLV appears to undergo recombination events [30]. In order to understand the reasons for variation among SCYLV isolates, ElSayed et al. [31] investigated sequence diversity and occurrence of recombination events in the RdRp and putative aphid transmission factor (PATF) coding genes of 25 SCYLV isolates. This study showed that the RdRp and PATF coding genes are potential locations for recombination using the GARD algorithm. Screening and quantifying evidence for recombination were necessary to avoid errors in phylogenetic analysis and to account for selection pressure that might act on the encoded proteins. Negative and positive selection have been observed for SCYLV, but the frequency of mutants is relatively low [31]. New viruses may have RNAs that are taxonomically distinct but interdependent [100]. Additionally, the clustering patterns of SCYLV isolates were clearly influenced by recombination events that occurred in the RdRp domain. Partial sequences of the SCYLV RdRp gene displayed higher diversity than the PATF gene [31]. Another study that might contribute to our understanding of recombination events in SCYLV genome has been conducted by ElSayed and Boulila (unpublished). They investigated possible recombination events located in ORFs 0, 1, and 3 of SCYLV using three programs, namely, TOPALi v2.5, RECCO, and the RDP package. It is noteworthy that the TOPALi v2.5, and RECCO methods strongly indicated the presence of recombination in aligned sequences of ORFs 0, and 1. In contrast, no recombination signals were detected in ORF3 using those methods. The RDP package did not reveal any recombination signals in ORFs 0 and 3, but in ORF1, numerous accessions were identified as potential recombinants.

It has been proposed that recombination along with mutation can be advantageous for RNA viruses, as it can create high fitness genotypes more rapidly than mutation alone [15]. Changes in the environment or replicative niches of the virus may have required recombination for fitness. This becomes especially important in plant viruses that have a broad host range or can use several vector species for transmission. Viruses that replicate in both plants and the insects that transmit them from plant to plant probably experience dramatically different selection pressures in each host [77].

Conclusion and future prospects

There is increased interest in RNA virus-plant systems for several reasons. First, more than 95 % of plant-infecting viruses are RNA viruses. Second, RNA molecules can affect practically every stage of plant gene expression. Third, plants can utilize RNAi as a specific antiviral mechanism. Finally, RNA-dependent RNA polymerases, the enzymes mediating RNA recombination, are encoded by both viruses and plants [88].

The changing environment becomes important in many plant viruses that have a broad host range or that can be transmitted by several different vector species. Viruses that replicate in both plants and in the insects that transmit them probably face greatly different selection pressures in each host [77]. Therefore, we should place emphasis on studying the impact of environmental conditions on SCYLV replication and evaluation. There is a need to further investigate the biological significance of the genetic diversity found in SCYLV, as well as understanding the genome dynamics of SCYLV. Consequently, it is essential to improve our knowledge of SCYLV, its vector, its hosts other than sugarcane, and its causal agent in order to manage the important diseases of sugarcane, especially with regard to screening and cultivation of resistant cultivars. Two combined strategies are proposed to confine SCYLV infection to a low level. One is to identify and deploy resistant varieties [84], and the other is to employ a cultivation scheme in which virus-free cane plants, generated by meristem tip culture, are grown for seed piece production in fields remote from commercial sugarcane fields.

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