Management of *Sweet Potato Leaf Curl Virus* in Sweetpotatoes Using Insecticides[1,2]

D. Michael Jackson,[3,4] Kai-Shu Ling,[3] Alvin M. Simmons,[3] and Howard F. Harrison, Jr.[3]

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ABSTRACT *Sweet potato leaf curl virus* (SPLCV), which is transmitted by the sweetpotato whitefly, *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae), species complex, can severely affect yields of sweetpotatoes, *Ipomoea batatas* (L.) Lam. (Convolvulaceae). This virus is endemic in sweetpotato fields at the U.S. Vegetable Laboratory (USVL), Charleston, S.C. In 2010 and 2011, experiments were conducted to determine if repeated insecticide applications were useful for protecting 'Beauregard' sweetpotato from SPLCV infection. In 2010, plots were untreated or treated twice weekly with imidacloprid. A row of SPLCV-infected sweetpotato genotype 'W-258' was planted between 'Beauregard' plots to serve as a source of whiteflies and SPLCV. A similar test was performed in 2011, except that the plots were sprayed only once a week, and a rotation of four insecticides (in the order of imidacloprid, pyriproxyfen, acetamiprid, and pymetrozine) was used. Yellow sticky traps were placed horizontally in the center of each plot at canopy height to monitor whitefly abundance. Leaf samples were taken every other week to test for SPLCV infection using real-time polymerase chain reaction (PCR) techniques. Over the two-year period, there were significantly fewer whiteflies on sticky cards in the sprayed treatment for only two of the 36 weekly samples, indicating that insecticides were largely ineffective in reducing whitefly populations moving into these plots. By the end of the growing season each year, all of the unsprayed plots were infected with SPLCV as determined by real-time PCR. However only about one-half of the sprayed plots were infected with SPLCV. This indicates that insecticides could be useful in protecting sweetpotatoes from SPLCV. The insecticide sprays would likely be more effective under normal production practices where sources of the virus are not in such close proximity to the uninfected crop.

KEY WORDS *Geminiviridae*, *Begomovirus*, *Ipomoea*, real-time polymerase chain reaction, whiteflies, *Bemisia*

Sweetpotato (*Ipomoea batatas* L.) Lamarck (Convolvulaceae) is one of the most important root and tuber crops, and its world-wide production is surpassed only

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[3]USDA, Agricultural Research Service, U.S. Vegetable Laboratory, Charleston, SC.

[4]Corresponding author; mike.jackson@ars.usda.gov

by potato [*Solanum tuberosum* L. (Solanaceae)] and cassava [*Manihot esculenta* Crantz (Euphorbiaceae)]. Although production is concentrated in China and sub-Saharan Africa, sweetpotatoes are grown widely in many countries with tropical or subtropical climates. In the United States, sweetpotato production is concentrated in several southeastern states and California. One major limiting production factor is the cumulative effects of virus infections on this vegetatively propagated crop. Over 30 plant viruses are known to infect sweetpotato, and many are transmitted by insects (Navas-Castillo et al. 2011, Clark et al. 2012, 2013).

The *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae) species complex (De Barro et al. 2013) is the primary vector of begomoviruses (*Geminiviridae*: *Begomovirus*) affecting sweetpotatoes (Jones 2003, Hogenhout et al. 2008). *Sweet potato leaf curl virus* (SPLCV), which was the target disease in this study, is a whitefly-transmitted *Begomovirus* affecting sweetpotato (Fauquet et al. 2003, Anonymous 2014). Whiteflies also transmit other leaf curl viruses, *Sweet potato chlorotic stunt virus* (*Closteroviridae*: *Crinivirus*) and *Sweet potato mild mottle virus* (*Potyviridae*: *Ipomovirus*), to sweetpotato (Valverde et al. 2004, Navas-Castillo et al. 2011, Clark et al. 2012, 2013). However, little is known about the epidemiology of these viral diseases in sweetpotatoes (Simmons et al. 2009, Ling et al. 2010, 2011, Zhang & Ling 2011).

Begomoviruses have a circular single-stranded DNA, which is encapsulated in twinned quasi-icosahedral virions (Navas-Castillo et al. 2011). The monopartite sweet potato leaf curl viruses, sometimes called 'sweepoviruses' (Fauquet & Stanley 2003, Trenado et al. 2011, Clark et al. 2012) or 'swepoviruses' (Albuquerque et al. 2011, Esterhuizen et al. 2012), form a phylogenetic cluster distinct from and basal to all other begomoviruses (Fauquet et al. 2008, Lozano et al. 2009, Albuquerque et al. 2011). Leaf curl symptoms were first reported from sweetpotato in Taiwan (Liao et al. 1979, Chung et al. 1985). In Japan, Osaki & Inouye (1991) suggested the possible involvement of a geminivirus, and Lotrakul et al. (1998) finally demonstrated SPLCV as the cause of these disease symptoms. Subsequently, SPLCV and related sweepovirus species have been found in the United States and several other countries in Europe, Asia, Africa, and South America (Lotrakul et al. 2003, Zhang & Ling 2011, Clark et al. 2012). Fortunately, none of the sweepoviruses have been reported from the major sweetpotato-producing areas of the United States (Clark et al. 2012, 2013).

SPLCV infection often causes severe yield reductions, frequently without causing visible foliar symptoms (Lotrakul et al. 2003, Clark & Hoy 2006, Ling et al. 2010, Clark et al. 2012). For example, there was a wide variation in visible leaf-curl symptoms and yield reductions among 27 heirloom sweetpotato cultivars grown at the USDA, ARS, U.S. Vegetable Laboratory (USVL), Charleston, SC (Ling et al. 2010). Because sweetpotato is vegetatively propagated, viruses can accumulate in "seed stocks" of stored roots (Bryan et al. 2003a,b, Clark & Hoy 2006). Virus-free sweetpotatoes can become severely infected within one year (Ling et al. 2010), and most sweetpotato genotypes grown from seed as part of the USVL sweetpotato breeding program become infected with SPLCV within the first year.

Geminiviruses are not known to be transmitted through true seeds (Brown & Nelson 1988), and their spread in the field is dependent upon whitefly vectors or infected seed roots (Markham et al. 1994). *Bemisia tabaci* transmits SPLCV in a persistent, circulative manner (Hogenhout et al. 2008, Simmons et al. 2009), and

is restricted to the phloem (Cilia et al. 2012). Disease epidemiology of SPLCV depends on the efficiency of whiteflies to acquire, retain, and transmit this virus (Simmons et al. 2009). When plants were infested with 30 or more viruliferous whiteflies, over one-half of the plants became infected with SPLCV (Lotrakul et al. 1998). Simmons et al. (2009) found that *B. tabaci* needed about a one-day acquisition access period to acquire SPLCV from the Brazilian morning-glory, *Ipomoea setosa* Ker. Gawl. They also reported that these whiteflies required a minimum of 15 min to transmit virus to uninfected plants, and that they remain viruliferous for up to 30 d while feeding on plants that are not virus hosts (Simmons et al. 2009).

Little progress has been made toward developing integrated approaches for managing sweepoviruses in sweetpotato (Clark et al. 2012). Virus diseases have mainly been managed through planting virus-indexed stock derived through meristem tip culture and certified as virus-indexed by grafting techniques or polymerase chain reaction (PCR) testing. In the United States, there are several facilities that produce virus-indexed plant materials that are then propagated and distributed by commercial seedling producers (Bryan et al. 2003a,b). This approach effectively reduces losses to viral diseases but increases the cost of the virus-indexed cuttings.

Control of whitefly vectors with insecticides has met with limited success in preventing the spread of virus diseases (Perring et al. 1999, Gilbertson et al. 2011). The development of resistance by *B. tabaci* to many classes of insecticides (Palumbo et al. 2001, Dennehy et al. 2010) makes vector control even more difficult. Unfortunately, due to extensive exposure, resistance to neonicotinoids is now spread worldwide, and several examples of cross resistance with other insecticide classes have been reported for neonicotinoids (e.g., cross resistance between imidacloprid and pymetrozine) (Nauen & Denholm 2005, Gorman et al. 2010). To help overcome insecticide resistance, the chitin synthesis inhibitor, buprofezin, and the juvenile hormone analog, pyriproxyfen, have been used successfully in rotation with neonicotinoids for management of *B. tabaci* (Stansly & Natwick 2010, Gilbertson et al. 2011).

Nevertheless, the use of insecticides to control whitefly vectors is still a primary strategy for management of *Tomato yellow leaf curl virus* (TYLCV) and other begomoviruses (Polston & Lapidot 2007, Caballero et al. 2013), and newer insecticides with unique modes of action show promise for controlling whiteflies and limiting virus spread (Castle et al. 2009). For example, the systemic anthranilic diamide, cyantraniliprole, has a novel mode of action that leads to a cessation of feeding by *B. tabaci* (Civolani et al. 2014). No cross-resistance of cyantraniliprole with neonicotinoids or pyriproxyfen has been reported; thus, this insecticide could be used in a management rotation with other classes of insecticides (Li et al. 2011). Another relatively new pyridine azomethine insecticide, pymetrozine, is selective against plant-sucking insects. This compound interferes with the feeding behavior of whiteflies, which results in death due to starvation after a few days, thus limiting the spread of TYLCV (Nicholson et al. 1995, Polston & Sherwood 2003). Other new insecticides have antifeedant or arrestant properties in addition to their toxicity, which further disrupts virus transmission (Castle et al. 2009). Therefore, we conducted experiments to determine whether insecticides could be used to protect plots of virus-tested sweetpotatoes from SPLCV infection by controlling whitefly vectors.

Materials and Methods

2010 Experiment. In 2010, a field experiment was performed at the USVL, Charleston, SC. Cuttings of 'Beauregard' were produced from plant beds using indexed (i.e., tested negative for SPLCV) sweetpotato "seed" roots obtained from a commercial grower in North Carolina (Scott Farms, Lucama, NC; http://www.scottfarms.com/) where SPLCV has not been reported. Samples from this plant bed were confirmed to be negative for SPLCV using established real-time PCR procedures (Kokkinos & Clark 2006, Ling et al. 2010, 2011) described below. Stem cuttings were planted on 2 July 2010 [Day of year (DOY) 183] into six replications arranged in a randomized complete block design. Each plot consisted of two adjacent rows of 10 plants each (6.1 m long). The sweetpotato cuttings were planted 30 cm apart within rows, and the centers of the rows were 1.0 m apart.

Insecticide treatments were started on 9 July, and plots were sprayed twice each week (Tuesday and Friday) until 2 November (DOY 306), and weekly (Tuesday) thereafter until roots were harvested on 17 November (DOY 321). The two treatments were an untreated control and an insecticide treatment of imidacloprid (Provado® 1.6 Flowable Insecticide, 51.7 g a.i./ha, Bayer CropScience LP, Research Triangle Park, NC), using a spray volume (270 l/ha.) sufficient to reach "runoff" throughout the season as the plants grew larger.

Individual plots were separated by three meters of bare soil that was kept free of weeds. Within a replication, each double-row treatment plot was separated from the other treatment by a row of the sweetpotato genotype 'W-258' that was confirmed to be infected with SPLCV using real-time PCR. 'W-258' is an advanced breeding line from the USDA-ARS sweetpotato breeding program that is highly symptomatic for SPLCV (Lotrakul et al. 1998, Clark & Hoy 2006, Ling et al. 2010), whereas 'Beauregard' does not show typical leaf curling symptoms. The 'W-258' plots were left unsprayed and they served as a reservoir of whiteflies and SPLCV. Thus, each replication consisted of two rows of unsprayed 'Beauregard' and two rows of insecticide-sprayed 'Beauregard', separated by an unsprayed row of virus-infected 'W-258'.

One 5.1 × 5.1 cm (26 cm^2) yellow sticky card trap (Olson Products, Medina, OH) was placed horizontally in the center of each plot at canopy height to monitor whitefly abundance (Simmons et al. 2008). Sticky traps were collected and replaced weekly for 19 consecutive weeks, 16 July through 15 November (week of the year 28–46). Numbers of adult whiteflies per sticky card for the sprayed and unsprayed plots for each sampling date were subjected to analysis of variance (ANOVA) using the General Linear Model (GLM) procedure (SAS 2009), and means on a particular date were compared using the paired-*t* test (Steel & Torrie 1960).

Leaf samples were taken from each plot approximately every two weeks: 23 July, 6 and 20 August, 3 and 17 September, 1, 15, and 29 October, and 12 November 2010. For each sample, one fully expanded leaf was removed from near the apical meristem of five plants per plot. Leaves were put into plastic bags, refrigerated (4°C), and analyzed within one day for SPLCV using the real-time PCR techniques (Ling et al. 2010, 2011).

Approximately 500 μg of leaf tissue was removed from each of the five leaves per sample and combined. These bulked samples were ground with a mortar and pestle, and DNA was isolated using a DNeasy plant kit (Qiagen Inc., Valencia,

CA) according to the vendor's instructions. Real-time PCR for SPLCV was conducted with a set of improved primers (SPLCV.F2: 5′GAGACAGC-TATCGTGCC and SPLCV.R2: 5′GAAACCGGGACATAGCTTCG), and TaqMan probe (SPLCV.P2: 5′ FAM-TACACTGGGAATGCTGTCCCAATTGCT-TAMRA) described by Ling et al. (2010, 2011). Each reaction mixture consisted of 12.5 µl of IQ Supermix (Bio-Rad Laboratories, Hercules, CA), 0.5 µl of each primer (0.4 µM), 0.5 µl of TaqMan probe (0.2 µM), 0.35 µl diluted reference dye, and 1.0 µl (500 ng/µl) of purified DNA. The thermal profile was set at 95°C for 10 min for denaturation, followed by 40 cycles of 95°C for 30 s, 55°C for 1 min, and 72°C for 30 s. Positive, healthy, and non-template controls were included for each sample run. Based on a cycle threshold (Ct) value of 35 in healthy controls, any Ct value lower than 35 was considered positive and a Ct value above 35 was considered negative for SPLCV (Ling et al. 2010, 2011).

Roots were harvested on 17 November 2010 and cured for one week. Harvested roots were graded, weighed, and then scored individually for insect damage using previously published procedures (Jackson et al. 2012). Among the parameters calculated was the severity index for the WDS complex (Wireworm, *Diabrotica*, *Systena*), which in the Charleston area is comprised of the spotted cucumber beetle (*Diabrotica undecimpunctata howardi* Barber) (Coleoptera: Chrysomelidae), the banded cucumber beetle (*D. balteata* LeConte), the elongate flea beetle (*Systena elongata* F.) (Coleoptera: Chrysomelidae), and wireworm larvae (*Conoderus* spp.) (Coleoptera: Elateridae) (Cuthbert 1967). The WDS index was calculated by averaging the rating given to each root (1 = 1–5 feeding scars, 2 = 6–10 feeding scars, and 4 = 10 or more feeding scars) (Cuthbert & Davis 1971, Schalk et al. 1992). Injury by white grub larvae (*Phyllophaga* spp. and *Plectris aliena* Chapin) (Coleoptera: Scarabaeidae), sweetpotato flea beetles (*Chaetocnema confinis* Crotch) (Coleoptera: Chrysomelidae), and sweetpotato weevils (*Cylas formicarius* F.) (Coleoptera: Brentidae) were calculated as the percentages of total roots that showed any damage by these insects. The percentage of uninjured roots (undamaged by any of the soil insect pests) also was determined. Yields and insect ratings for the sprayed and unsprayed plots were subjected to analysis of variance (ANOVA) (PROC GLM; SAS 2009), and means were compared using a paired-*t* test (Steel & Torrie 1960).

2011 Experiment #1. For the first 2011 field experiment, virus-indexed cuttings of 'Beauregard' were planted on 11 July 2011 (DOY 191) in a randomized complete block design with six replications of double-row plots (two rows of 10 plants each) identical to those of the 2010 experiment. Cuttings were obtained from plant beds of indexed sweetpotato roots obtained from the same commercial grower in North Carolina that provided virus-free roots in 2010. As in 2010, a row of unsprayed 'W-258' that had been confirmed to be infected with SPLCV was planted between the treatment plots. The two treatments were an untreated control and weekly insecticide applications (Thursdays) to runoff (270 l/ha.). Foliar insecticide treatments were started with applications of imidacloprid (Provado®) at 51.7 g a.i./ha. on 21 and 28 July and 4 August. Thereafter, each week one of three insecticides was applied, and they were rotated in subsequent weeks. The insecticides applied were (in order): pyriproxyfen (Knack Insect Growth Regulator®, Valent U.S.A. Corporation, Walnut Creek, CA) at 91.0 g a.i./ha., acetamiprid (Assail® 30SG, Cerexagri-Nisso, LLC, King of Prussia, PA) at 105.3 g a.i./ha., and pymetrozine (Endeavor® 50WG, Syngenta Crop Protection,

Inc., Greensboro, NC) at 20.3 g a.i./ha. The last insecticide spray was made on 16 November, and over that 15-week period, each insecticide was applied five times.

A yellow sticky card trap (26 cm^2) was placed horizontally at canopy height near the center of each plot to monitor whitefly abundance. Sticky traps were collected and replaced weekly for 17 consecutive weeks, 21 July through 10 November (weeks of the year 29–45). Numbers of adult whiteflies per sticky card for the sprayed and unsprayed plots were subjected to analysis of variance (ANOVA) (PROC GLM; SAS 2009), and means on a particular date were compared using a paired-*t* test (Steel & Torrie 1960).

For each leaf sample, one fully expanded leaf was taken from near the apical meristem of five plants per plot. These were put into plastic bags and refrigerated until they could be analyzed by real-time PCR for SPLCV as described above. Leaf samples were taken on seven occasions: 2 and 22 August, 9 and 20 September, 11 and 31 October, and 14 November 2011. Roots were harvested on 18 November, cured, graded, weighed, rated for pest damage, and analyzed as described above for the 2010 experiment.

2011 Experiment #2. For the second 2011 field experiment, virus-indexed cuttings of 'Beauregard' were planted on 12 August 2011 (DOY 224) in six replications of double row plots of 20 plants each. These cuttings were obtained from the North Carolina State University sweetpotato breeding project, and they were tested for SPLCV before planting. For this evaluation, a small piece of leaf was taken from each cutting and bulked by replication. All six replications of these bulked samples were negative for SPLCV using real-time PCR as described above. A drench application of imidacloprid (Admire® 2F, Bayer, Kansas City, MO) was applied to each cutting at transplantation on 12 August. Thereafter, spray applications of the same three insecticides were applied once a week (Thursday) starting on 18 August, and they were rotated in subsequent weeks (in order: acetamiprid, pymetrozine, and pyriproxyfen) until the roots were harvested on 14 November. Over this 13-week period, acetamiprid was sprayed five times, and pymetrozine and pyriproxyfen were sprayed four times each. Yellow sticky card traps and leaf samples were taken on the same dates as the first 2011 field experiment beginning on 22 August. Whitefly data and yields and insect ratings of harvested roots were analyzed as described above for the 2010 experiment. Roots were harvested on 18 November, cured, graded, weighed, rated for pest damage, and analyzed as described above.

Results

Whitefly populations, as determined by yellow sticky cards, increased rapidly in 2010 and peaked in weeks 32–37, then gradually declined (Figure 1). In 2011, whitefly populations remained low until week 35 when the population rapidly increased, then gradually declined for the remainder of the summer (Figure 1). On individual sampling dates, there were significantly fewer whiteflies on sticky cards in the sprayed treatment for only two of 36 sampling periods for both years, week 40 in 2010 ($P = 0.048$) and week 45 in 2011 ($P = 0.007$) (Figure 1). One sample date early in 2011 (week 33) actually had significantly more whiteflies on the sprayed plots ($P = 0.021$) (Figure 1). It should be noted that there was no evidence of resistance by whiteflies to these insecticides throughout the two years of experiments at the USVL.

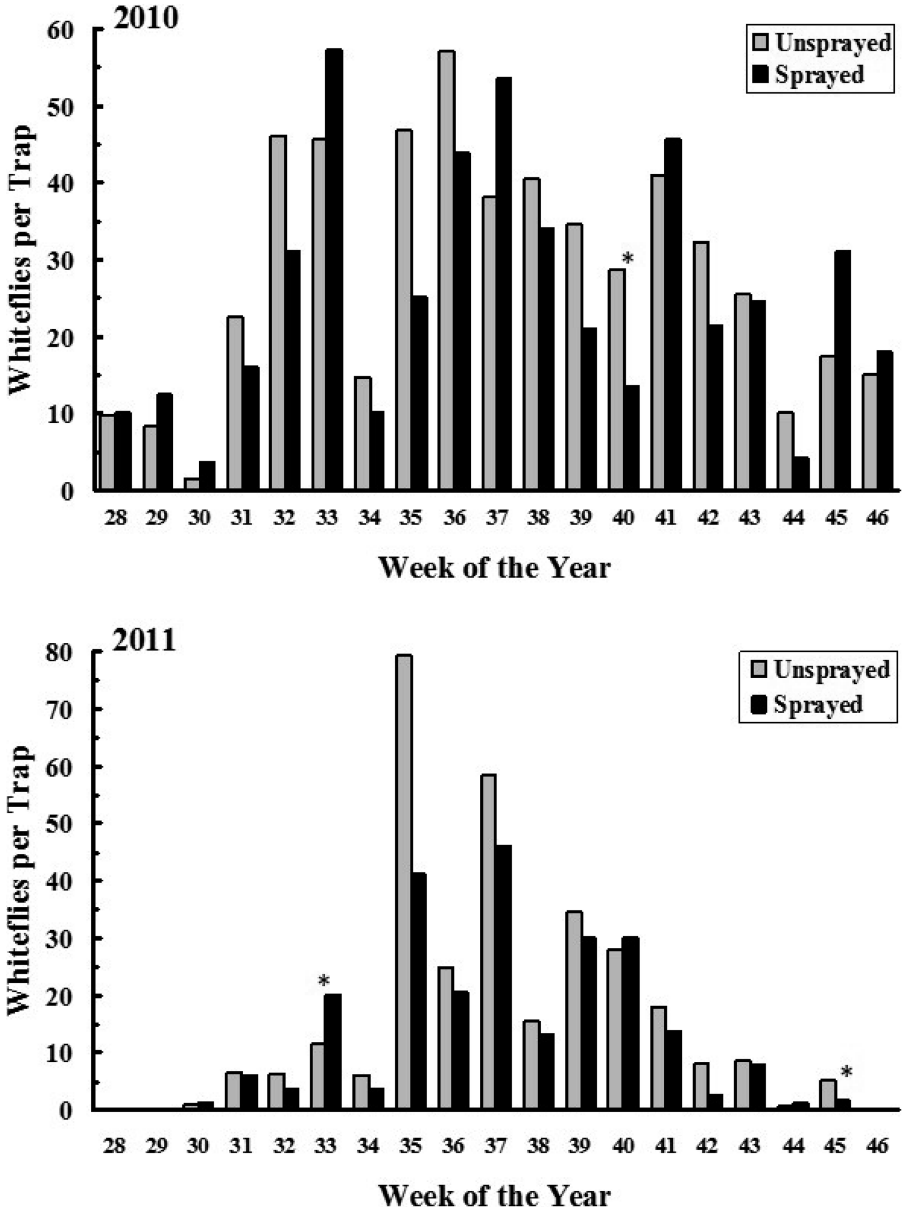

Fig. 1. Mean numbers of sweetpotato whiteflies captured per yellow sticky card trap (n = 6 replications) in sweetpotato fields at the U.S. Vegetable Laboratory (USVL), Charleston, SC in 2010 (top) and 2011 (bottom). Consecutive weekly samples began on 16 July 2010 (week 28) and 21 July 2011 (week 29). Asterisks indicate statistically significant ($P < 0.05$) difference between the sprayed and unsprayed plots according to a paired-*t* test (Steel & Torrie 1960).

For both years, there were no differences in root yields (2010, $P = 0.59$; 2011, $P = 0.62$) between the sprayed and unsprayed plots (data not shown). In addition, there were no significant differences between the sprayed and unsprayed plots in the percentage of roots damaged by flea beetles (2010, $P = 0.33$; 2011, $P = 0.82$), white grubs (2010, $P = 0.14$; 2011, $P = 0.88$), or sweet potato weevils (2010, $P = 0.38$; 2011, $P = 0.11$) (data not shown). In 2011, there also were no significant differences in the overall percentage of uninjured roots ($P = 0.18$) or WDS ratings ($P = 0.44$). However, in 2010, the overall percentage of uninjured roots was significantly higher ($P = 0.026$) and the WDS ratings were significantly lower ($P = 0.003$) for the sprayed plots, indicating some effects of the season-long imadacloprid treatment on soil insect pests.

By the end of 2010 and for the first experiment in 2011, all of the leaf samples from the unsprayed plots were infected with SPLCV; however, only about 50% of the leaf samples in the sprayed plots were infected with SPLCV each season (Figure 2). Interestingly, only two of the six plots in the second 2011 experiment were infected with SPLCV by the end of the season. Because these plots were located close to the first 2011 experiment, they shared the same population of whiteflies. The only differences between these experiments were the date of planting and the use of a drench application of imidacloprid before the cuttings were planted in the second test.

Discussion

As Broadbent (1957) pointed out over 50 y ago, "applying insecticides to crops has, more often than not, failed to decrease the incidence of virus diseases in the sprayed crops and has sometimes actually increased it, even though field inspections of the crops indicated that the insecticide has 'controlled' the specific insect vector." However, much has changed since Broadbent (1957) published this review article, and comprehensive pest management programs, including the use of newer insecticides with unique modes of action, and a better understanding of the epidemiology of begomoviruses has led to renewed optimism that viruses can be managed in vegetable crops (Schuster et al. 2007, Castle et al. 2009, Adkins et al. 2011). However, the insecticides in the experiments reported herein were largely ineffective in reducing whitefly populations, at least as indicated by yellow sticky trap captures. Whiteflies appear to have moved equally into the unsprayed and sprayed 'Beauregard' plots from the nearby unsprayed 'W-258' plants or from other source plants. Although yellow sticky card traps have been used to monitor whitefly populations in several studies (Simmons et al. 2008), they only measure the presence of whiteflies. Sticky card traps do not indicate whether whiteflies actually settled, established, and fed on the sweetpotato plants, which is necessary to transmit SPLCV. Perhaps these data point to the inadequacy of yellow sticky traps to measure the impact that whiteflies have on the plants.

Nevertheless, insecticide applications reduced the incidence of SPLCV in the sprayed plots in this study, perhaps because some of the newer insecticides affect whitefly feeding behavior or otherwise interfere with virus transmission (Castle et al. 2009). Because SPLCV is a persistent, circulative virus, a period of uninterrupted feeding, a latent period, and movement to another plant are all necessary for this virus to be spread. Insecticides expressing modes of action

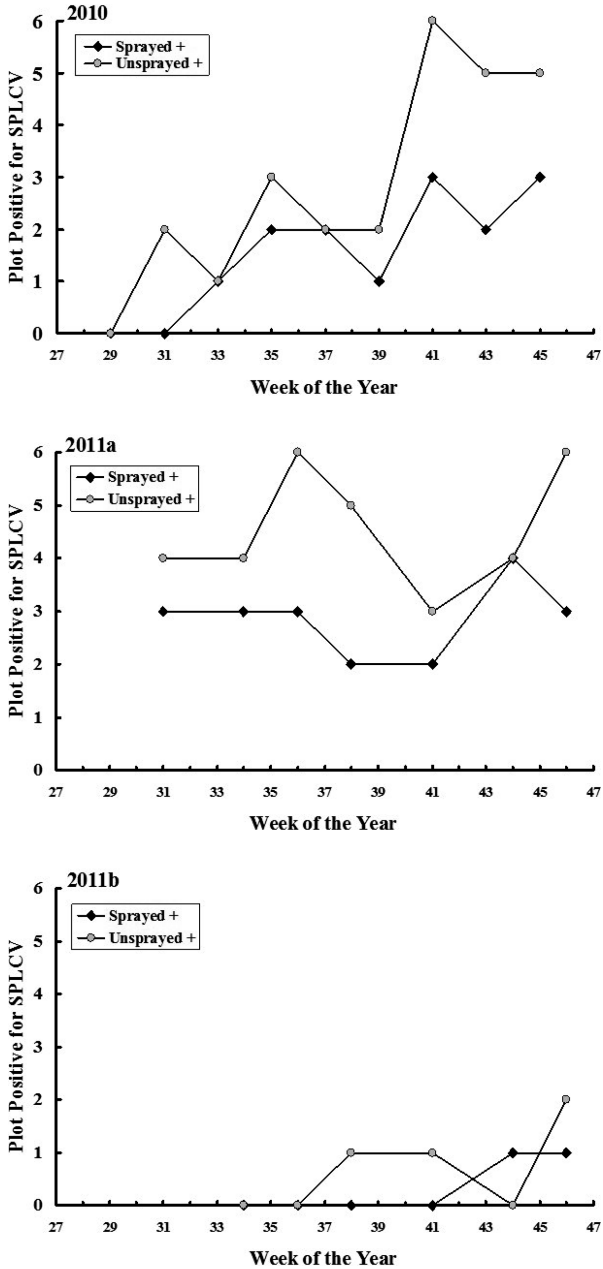

Fig. 2. Number (out of 6 replications) of sprayed or unsprayed sweetpotato plots that had bulked leaf samples (5 leaves per 20-plant plot) that were positive for *Sweet potato leaf curl virus* (SPLCV) using real-time polymerase chain reaction (PCR) techniques at the U.S. Vegetable Laboratory (USVL), Charleston, SC in 2010 (top) and 2011 (middle, bottom).

other than acute toxicity, such as feeding inhibition or increased flight activity, could disrupt the spread of SPLCV within fields. The neonicotinoid insecticides that we used, imidacloprid and acetamiprid, have systemic characteristics so that treated plants retain lethality toward whiteflies for a considerable period of time (Tomizawa & Casida 2005). We also employed the insect growth regulator, pyriproxyfen, that could disrupt the spread of SPLCV by retarding the development of winged forms. Pymetrozine has anti-feedant properties (Castle et al. 2009) that could interfere with the acquisition phase of SPLCV.

Broadbent (1957) proposed that unlike direct insect damage, "the spread of viruses is not proportional to the extent or duration of insect infestation of a crop. Three conditions must be fulfilled for an insect-transmitted virus to spread. First, there must be a source of the virus; secondly, the insect vector must be present, and, thirdly, the insect must move about." For this study, these three conditions existed, and SPLCV spread through these sweetpotato plots in a manner that was not proportional to the infestation level of whiteflies.

Although whiteflies were not adequately controlled by weekly or twice-weekly sprays, these insecticides provided some degree of protection from SPLCV. However, growers are unlikely to spray sweetpotatoes that often (current recommendations and use guidelines for neonicotinoid insecticides preclude repeated use on this crop), and less frequent sprays may be inadequate to prevent SPLCV from infecting sweetpotato plots. No single method of disease management is likely to keep sweetpotatoes entirely free from plant virus infections, and comprehensive management plans for whiteflies and whitefly-transmitted viruses in several vegetables have been developed (Schuster et al. 2007, Adkins et al. 2011, Webb et al. 2011), which can serve as a guide to sweetpotato growers. Clark et al. (2012) suggest that the best approach for managing sweepoviruses in sweetpotato may be use of resistant germplasm, and a wide range in resistance/susceptibility has been documented (Ling et al. 2010). Also, the current system of providing virus-tested planting materials will continue to be the cornerstone for managing this and other virus diseases in sweetpotato. However, careful use of selected insecticides may prove useful in lowering the populations of whiteflies and slowing the spread of SPLCV if this disease became a problem in a major sweetpotato production area. In conclusion, potential management techniques for SPLCV in sweetpotato should include the use of clean planting materials, host plant resistance, eradication of alternate hosts of the virus in production areas, and judicious use of certain insecticides to reduce the populations of whitefly vectors. It must also be noted that these foliar insecticides had no measurable effects on yields or on insect damage of harvested roots, and therefore these products should not be considered for management of soil insect pests of sweetpotato.

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