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MONITORING INSECTICIDE RESISTANCE IN BIOTYPE B OF *BEMISIA TABACI* (HEMIPTERA: ALEYRODIDAE) IN FLORIDA

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ABSTRACT

Biotype B of the sweetpotato whitefly (SPWF), *Bemisia tabaci* (Gennadius) (also known as the silverleaf whitefly, *B. argentifolii* Bellows & Perring), is the key pest of tomatoes in south Florida, primarily as a vector of the begomovirus *Tomato yellow leaf curl virus* (TYLCV). Insecticides are most often used to manage the SPWF and TYLCV. A resistance monitoring program that was initiated in Florida in 2000 was continued from 2008 to 2010 and included 4 neonicotinoid insecticides (imidacloprid, thiamethoxam, dinotefuran, and acetamiprid), the insect growth regulator buprofezin, the pyrethroid bifenthrin, and the organochlorine endosulfan. Ten field populations in 2008 and 9 each in 2009 and 2010 were established with nymphal infested foliage and were tested for susceptibility using a systemic uptake, cut-leaf petiole bioassay with adults for the neonicotinoids; a leaf-dip bioassay with 2nd instars for buprofezin; and a vial bioassay with adults for bifenthrin and endosulfan. Each field population was exposed to the LC₅₀ and LC₉₅ of a known susceptible laboratory colony for each respective insecticide and mortality was compared with that at the same doses predicted from probit analyses of field populations tested in 2007. T-tests were used to determine the significance of differences between the mean mortality at the LC values of each field collected colony compared to the respective LC values of the laboratory susceptible strain. T-tests were also used to determine the significance of differences between the mean of the predicted mortality at the LC values of field collected populations in 2007 and the means of the LC values of field collected populations in 2008-2010 for each insecticide. Results indicated that, based on mortality averaged over all populations evaluated, all of the neonicotinoids indicated decreases in average susceptibility in 2008 and 2009 compared with the 2007 values, although the differences were less for dinotefuran and acetamiprid. The lowest average of mortality at both the LC₅₀ and LC₉₅ in 2008 and 2009 occurred for imidacloprid and thiamethoxam. These neonicotinoids had been in use longer than either of the other two. Mortality values for bifenthrin suggested an overall increase in field susceptibility in 2008 and 2009 while values for endosulfan suggested no change. There were no data predicted for buprofezin in 2007, but the 2008 average mortalities at the LC₅₀ and LC₉₅ were 0.438 and 0.802, respectively, indicating that field susceptibility was at an acceptable level. In 2010 the average susceptibility to the neonicotinoids appeared to have increased compared with previous years; however, the field populations tended to be evaluated after they had been reared in the laboratory without exposure to insecticides for more generations than in previous years. Despite this, average susceptibility to endosulfan appeared to decrease. The results showed the utility of using predicted LC₅₀ and LC₉₅ values, over the use of full dose range, for monitoring changes in susceptibility in field populations thru time. The data presented here provide important information aid to growers and producers in making decisions on insecticide usage.

Key Words: neonicotinoids, endosulfan, bifenthrin; buprofezin, sweetpotato whitefly, pest management, insecticide resistance monitoring

RESUMEN

Biotipo B de la mosca blanca de la batata (MBB), *Bemisia tabaci* (Gennadius) (también conocida como la mosca blanca de la hoja plateada, *B. argentifolii* Bellows & Perring), es la plaga clave de tomates en el sur de Florida, principalmente como vector del begomovirus *Tomato yellow leaf curl virus* (TYLCV). Insecticidas son comúnmente usados para manejar la MBB y TYLCV. Un programa de monitoreo de resistencia que fue iniciado en Florida en 2000 se continuó desde 2008 a 2010 e incluyó cuatro insecticidas neonicotinoides (imidacloprid, thiamethoxam, dinotefuran, y acetamiprid), el regulador de crecimiento de insectos buprofezin, el piretroide bifenthrin, y el organoclorado endosulfan. Diez poblaciones de campo en 2008 y 9 en cada año 2009 y 2010 fueron establecidas con follaje infestado con ninfas que fueron examinadas para susceptibilidad usando un bioensayo de absorción sistémica

de pecíolos de las hojas con adultos para los neonicotinoides; un bioensayo de sumersión de las hojas para 2do estadio para buprofezin; y un bioensayo de viales con adultos para bifenthrin y endosulfan. Cada población de campo fue expuesta a las LC_{50} y LC_{95} conocidas de una colonia susceptible de laboratorio para cada respectivo insecticida y la mortalidad fue comparada para las mismas dosis predictas por análisis probit de poblaciones de campo examinadas en 2007. Pruebas T fueron usadas para determinar las diferencias significativas entre la mortalidad media a los valores de LC de cada población de campo recolectada y comparada a los respectivos valores de LC de la colonia susceptible de laboratorio. Pruebas T también fueron usadas para determinar las diferencias significativas entre la media de la mortalidad predicta a los valores de CL de poblaciones de campo recolectadas en el 2007 y la medias de los valores de CL de poblaciones de campo recolectadas en 2008-2010 de cada insecticida. Resultados indicaron que, basado en el promedio de mortalidad sobre todas las poblaciones evaluadas, todos los neonicotinoides indicaron disminución en el promedio de susceptibilidad en 2008 y 2009 comparadas con los valores del 2007, aunque las diferencias fueron menos para dinotefuran. El promedio más bajo de mortalidad en ambas LC_{50} y LC_{95} en 2008 y 2009 ocurrieron para imidacloprid y thiamethoxam. Estos neonicotinoides han sido usados por más tiempo que cualquiera de los otros dos. Los valores de mortalidad para bifenthrin sugirieron un aumento en general en la susceptibilidad de campo en 2008 y 2009 mientras los valores para endosulfan no sugirieron cambio. No hubieron datos en 2007 para buprofezin pero el promedio de mortalidad en 2008 a las LC_{50} y LC_{95} fueron 0.438 y 0.802, respectivamente, indicando que la susceptibilidad de campo estaba en un nivel aceptable. El promedio de susceptibilidad en 2010 de los neonicotinoides parecieron incrementar comparado a años previos; sin embargo, las poblaciones de campo tendieron ser evaluadas, en general, después de haber sido criadas en el laboratorio sin exposición a insecticidas por más generaciones que en años previos. A pesar de esto el promedio de susceptibilidad para endosulfan pareció disminuir. Los resultados mostraron la utilidad de usar valores predictos de LC_{50} y LC_{95} sobre el uso de un rango completo de dosis, para monitorear cambios de susceptibilidad en poblaciones de campo a través del tiempo. Los datos presentados aquí proveen información importante para ayudar a los agricultores y productores en la toma de decisiones sobre el uso de insecticidas.

Palabras Clave: Neonicotinoides, endosulfan, bifenthrin; buprofezin, mosca blanca de la tatata, manejo de plagas, monitoreo de resistencia a plaguicidas

Biotype B of the sweetpotato whitefly (SPWF), *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae) (also known as the silverleaf whitefly, *B. argentifolii* Bellows & Perring) represents one of the 24 species encompassed in the recent defined species complex of *B. tabaci* (DeBarro et al. 2011). This specific biotype/species and the Tomato yellow leaf curl virus (TYLCV) that it vectors remain key pests of tomato (*Solanum lycopersicum* L.; Solanales: Solanaceae) in southern Florida. Insecticides, particularly the neonicotinoids imidacloprid, acetamiprid, thiamethoxam and dinotefuran; the insect growth regulators buprofezin and pyriproxyfen; the organochlorine endosulfan (various products); and the pyrethroids (various products) remain integral tools for the management of whiteflies and, thus, the spread of TYLCV.

The SPWF has a host range that is far from limited to tomato. Florida's climate allows for overlapping growing seasons and, as a result, almost year-round pesticide use on various hosts of the SPWF and other field crop pests. Availability of hosts and multiple applications of insecticides create the potential for selection pressure to develop resistance in field populations of the SPWF. Due to this potential for resistance development, a program to monitor the susceptibility of field populations of SPWF to imidacloprid and

thiamethoxam was conducted from 2000 to 2007 (Schuster et al. 2010). Based upon LC_{50} values, resistance in *B. tabaci* to imidacloprid and thiamethoxam increased about 7 and 12 fold, respectively, through 2006. The information was used to develop and implement insecticide application strategies as part of a whitefly, TYLCV and resistance management program (Schuster 2007). Because of the value of the information, the resistance monitoring program was continued from 2007 to 2010. The neonicotinoids acetamiprid and dinotefuran and, the pyrethroid bifenthrin and endosulfan, were added to the program in 2007. Buprofezin was included in 2008. Reported herein are the results of the monitoring for 2008-2010.

MATERIALS AND METHODS

Field Populations

The bioassays were conducted using adults reared from foliage infested with nymphs that had been collected from crop fields in southern Florida during the spring 2008 and 2009 crop seasons and the spring and fall crop seasons of 2010. Collections were made at various locations around the state including Collier, Dade, Hendry, Marion and Manatee counties. Subsamples were sent to the

USDA-ARS, U.S. Horticultural Research Laboratory, in Florida, and all of them were determined as B biotype while Q biotype was only found on ornamental plants in protected culture (McKenzie et al. 2004, 2009, 2012). The leaves were placed in cages with non-infested cotton plants and placed in a room at about 28 °C and 12:12 h L:D. The sample leaves were left for several days to allow as many adults as possible to emerge. When the leaves collected from the field were dry, they were removed from the cages and the populations were maintained on the cotton plants for the duration of the resistance testing. When insufficient adults in the F₁ generation were available to conduct bioassays, the field populations were reared to the F₂-F₄ generations until sufficient adults were available. Ten populations were collected in 2008 and 9 each in 2009 and 2010.

Insecticides and Doses

Insecticide formulations evaluated included the neonicotinoids imidacloprid [Admire® 2F (2007) and Admire Pro® (2008-10), Bayer Crop Science, Research Triangle Park, North Carolina], thiamethoxam (Platinum® 2F, Syngenta Crop Protection, Inc., Greensboro, North Carolina), dinotefuran (Venom® 20SG, Valent U.S.A. Corp., Walnut Creek, California), and acetamiprid (Assail® 30SG, Cerexagri Inc., King of Prussia, Pennsylvania). The insect growth regulator buprofezin (Courier® 40SC, Nichino America, Inc., Wilmington, Delaware). Technical materials were used to evaluate the pyrethroid bifenthrin (bifenthrin technical, FMC Corp., Princeton, New Jersey) and the organochlorine endosulfan (endosulfan technical, Makhteshim Agan of North America, Inc., New York, New York). Resistance was estimated in the laboratory by observing adult or nymph mortality at the LC₅₀ and LC₉₅ of a susceptible strain for the respective insecticides. The doses were estimated using standard probit analyses (SAS Institute Inc. 1989) of the mortality of whiteflies exposed to serial dilutions of the insecticides. The susceptible strain used had been in continuous culture in the laboratory since the late 1980's without the introduction of any whiteflies collected from the field, and, therefore, would be expected to be highly susceptible to the insecticides. The respective LC₅₀ and LC₉₅ in ppm a.i. and Fiducial Limits used from 2008-2009 and 2010 are shown in Table 1.

Cut Leaf Petiole Bioassay for Neonicotinoids

For the neonicotinoids, the cut leaf petiole bioassay method was used (Schuster & Thompson 2001, 2004; Schuster et al. 2002, 2003, 2006, 2010; Schuster 2007). Petioles were cut from cotton seedlings and were suspended in aqueous

TABLE 1. LC₅₀ AND LC₉₅ BASELINE ESTIMATED VALUES FOR A SUSCEPTIBLE LABORATORY STRAIN FOR SELECTED INSECTICIDES TO COMPARE WITH ADULTS/NYMPHS OF *B. TABACI* FROM POPULATIONS COLLECTED IN THE FIELD FROM SOUTHERN FLORIDA.

Insecticide	2008-2009		2010	
	LC ₅₀ mg ai L ⁻¹ (95%FL)	LC ₉₅ mg ai L ⁻¹ (95%FL)	LC ₅₀ mg ai L ⁻¹ (95%FL)	LC ₉₅ mg ai L ⁻¹ (95%FL)
Imidacloprid	0.500 (0.118 – 1.749)	5.000 (1.424 – 13.407)	0.347 (0.136 – 0.609)	6.931 (3.305 – 31.584)
Thiamethoxam	—	—	1.804 (1.560 – 2.094)	17.953 (13.240 – 26.324)
Dinotefuran	1.250 (0.473 – 3.415)	10.000 (4.696 – 13.510)	1.004 (0.794 – 1.238)	4.894 (3.547 – 7.904)
Acetamiprid	0.313 (0.157 – 0.586)	2.500 (1.595 – 5.010)	0.615 (0.427 – 0.840)	5.207 (3.227 – 11.273)
Bifenthrin	0.625 (0.349 – 978)	5.000 (4.316 – 15.775)	1.418 (1.110 – 1.789)	10.501 (7.080 – 18.714)
Endosulfan	0.068 (0.025 – 0.253)	23.310 (16.563 – 28.906)	n/a ¹	n/a
Buprofezin	6.790 (4.352 – 9.867)	60.500 (35.700 – 144.500)	16.603 (11.574 – 22.868)	70.139 (45.319 – 159.928)
	0.286 (0.248 – 0.330)	1.604 (1.283 – 2.143)	n/a	n/a

¹Population not available (n/a).

solutions at the LC_{50} and LC_{95} of the susceptible laboratory strain for each of the respective insecticides. Double de-ionized water was included as a control. After 24 h, 10 whitefly adults were confined on each leaf in clip cages and after an additional 24 h, mortality was observed. Whiteflies were considered dead/moribund if they were unable to flip themselves to an upright position after a period of approximately one minute after having been pushed to their backs.

Vial Bioassay for Bifenthrin and Endosulfan

For bifenthrin and endosulfan, a variation of the vial bioassay method described by Staetz et al. (1992) was used. The inner surfaces of 20-mL glass scintillation vials were coated with 0.25 mL of acetone solutions of technical grade bifenthrin and endosulfan at the LC_{50} and LC_{95} of the susceptible laboratory strain. Control vials were treated with 0.25 mL of acetone, but no chemical. Ten whitefly adults were introduced into each vial and caps with organically-covered holes were used to close the tops. The vials were placed caps up in a room at about 28 °C and mortality was observed 6 h later. Whiteflies were pushed to their backs with a small brush and were considered dead/moribund if they were unable to flip themselves to an upright position after a period of approximately 1 min.

Leaf Dip Bioassay for Buprofezin

For buprofezin, a modification of the leaf-dip bioassay method described by Cahill et al. (1996) was used. Ten to 15 adult female whiteflies were confined in clip cages on each leaf of cotton plants with 2 true leaves present. After 24 h the adults were removed and the plants were then held in a clean cage for 10 days in a room at about 28 °C and 12:12 h L:D. After 10 days, live 2nd instars were counted and all other stages and any dead nymphs were removed with a dissecting needle. The leaves were then dipped for 10 s each in aqueous solutions of buprofezin at the LC_{50} and LC_{95} of the susceptible laboratory strain. Control leaves were dipped in double de-ionized water. The plants were placed back in the cage for at least another 10 days, after which time nymph mortality was then observed. Susceptibility to buprofezin was estimated only for populations collected in 2008.

Statistical Analyses

Mortality in 2007 was estimated by exposing whiteflies to serial dilutions of the insecticides and analyzing the data with standard probit analyses (SAS Institute Inc. 1989; Schuster 2007; Schuster et al. 2010). Therefore, in order

to compare changes in susceptibility from 2007 to 2008-2010, the predicted mortality of each of the 2007 populations at the LC_{50} and LC_{95} values that were used in 2007 was statistically extrapolated from the probit analyses of the 2007 populations. T-tests (Proc t-Test) were used to determine the significance of differences between the mean mortality at the LC values of each field collected colony compared to the respective LC values of the laboratory susceptible strain (SAS Institute Inc. 1989). The mortality at the LC_{50} and LC_{95} values are the mean of 4 replicates in each insecticide and each year, with a sample size (n) of 36-44 whiteflies (Tables 2 and 3). T-tests were also used to determine the significance of differences between the mean of the predicted mortality at the LC values of field collected populations in 2007 and the means of the LC values of field collected populations in 2008-2010 for each insecticide (Table 4). Proc T-Test Pooled was used for populations with equal variances and Proc T-Test Cochran for populations with unequal variances (SAS Institute Inc. 1989).

RESULTS

Field populations for the resistance monitoring program were collected largely from tomato in counties in southern Florida, including Collier, Dade, Hendry, and Manatee; however, 2 populations in 2010 were collected in Marion County in north central Florida (Table 2 and 3).

Results of monitoring for 2008 to 2010 are shown in Tables 2 and 3. Considering imidacloprid for 2008 the highest mortality values of approximately 0.40 and 0.90 at the LC_{50} and LC_{95} values, respectively, observed for one population from Collier County (GCC-AG) were not significantly different from the respective mortality values of the susceptible strain ($t = -0.98$; $df = 6$; $P = 0.3634$ and $t = -0.93$; $df = 6$; $P = 0.3867$) (Table 2). The remaining 9 field populations had LC_{50} and LC_{95} mortality values that were significantly lower than the LC mortality values of the susceptible strain: GCC_IT ($t = -4.25$; $df = 6$; $P = 0.0054$ and $t = -8.36$; $df = 6$; $P = 0.0002$), GCC_WE ($t = -9.72$; $df = 3$; $P = 0.0023$ and $t = -6.76$; $df = 6$; $P = 0.0005$), HOMESTEAD_1 ($t = -6.28$; $df = 6$; $P = 0.0008$ and $t = -13.00$; $df = 6$; $P < 0.0001$), HOMESTEAD_4 ($t = -8.36$; $df = 6$; $P = 0.0002$ and $t = -9.02$; $df = 6$; $P = 0.0001$), GCC_SE ($t = -6.08$; $df = 6$; $P = 0.0009$ and $t = -17.31$; $df = 6$; $P < 0.0001$), GCC_CB ($t = -5.98$; $df = 6$; $P = 0.0010$ and $t = -19.29$; $df = 6$; $P < 0.0001$), PARISH_5 ($t = -3.92$; $df = 6$; $P = 0.0078$ and $t = -8.36$; $df = 6$; $P = 0.0002$), MYAKKA CITY ($t = -8.14$; $df = 6$; $P = 0.0078$ and $t = -12.48$; $df = 6$; $P < 0.0001$), and ORGANIC ($t = -6.88$; $df = 6$; $P = 0.0005$ and $t = -3.29$; $df = 3$; $P = 0.0462$) (Table 2). In 2009, all 9 field collected populations had significantly lower LC mortality values compared with the LC values of the susceptible

TABLE 2. MEAN MORTALITY AND STANDARD ERROR (SE) OF FLORIDA FIELD POPULATIONS OF *BEMISIA TABACI* EXPOSED IN THE LABORATORY TO THE LC₅₀ AND LC₉₅ DOSES ESTIMATED FOR A SUSCEPTIBLE LABORATORY STRAIN. THE GENERATION BIOASSAYED IS INDICATED IN PARENTHESES, WITH GENERATION F₁ BEING THE ADULTS THAT EMERGED FROM FIELD-COLLECTED FOLIAGE.

Population	County	Crop	Imidacloprid			Thiamethoxam			Dinotefuran			Acetamiprid		
			LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)
			2008											
Susceptible Strain GCC-IT GCC-WE GCC-AG Homestead-1 Homestead-4 GCC-SE GCC-CB Parrish-5	Manatee	Cotton	0.514 (F ₁) ¹	(0.053)	0.950 (F ₁)	(0.029)	0.650 (F ₁)	(0.132)	0.850 (F ₁)	(0.119)	0.575 (F ₁)	(0.123)	0.925 (F ₁)	(0.048)
	Collier	Tomato	0.111 (F₁)²	(0.079)	0.408 (F₁)	(0.058)	0.025 (F ₁)	(0.025)	0.509 (F ₁) ³	(0.170)	0.100 (F ₁)	(0.041)	0.736 (F ₁)	(0.105)
	Hendry	Tomato	0.000 (F ₁)	(0.000)	0.206 (F ₁)	(0.106)	0.033 (F₁)	(0.033)	0.706 (F₁)³	(0.072)	0.088 (F ₁)	(0.059)	0.719 (F ₁)	(0.065)
	Collier	Tomato	0.400 (F ₁) ³	(0.103)	0.875 (F ₁) ³	(0.075)	0.075 (F ₁)	(0.048)	0.657 (F ₁) ³	(0.128)	0.250 (F₁)³	(0.029)	0.950 (F₁)³	(0.029)
	Dade	Tomato	0.105 (F ₂)	(0.038)	0.231 (F ₂)	(0.047)	0.025 (F ₂)	(0.025)	0.433 (F ₂)	(0.091)	0.025 (F ₂)	(0.025)	0.499 (F ₂)	(0.125)
	Dade	Tomato	0.025 (F ₂)	(0.025)	0.183 (F ₂)	(0.080)	0.050 (F ₂)	(0.050)	0.301 (F ₂)	(0.066)	0.075 (F ₂)	(0.048)	0.663 (F ₂)	(0.055)
	Hendry	Tomato	0.078 (F₂)	(0.048)	0.161 (F₂)	(0.035)	0.025 (F ₂)	(0.025)	0.311 (F ₂)	(0.126)	0.050 (F ₂)	(0.050)	0.680 (F ₂)	(0.022)
	Hendry	Tomato	0.056 (F ₁)	(0.056)	0.362 (F ₁)	(0.010)	0.000 (F₁)	(0.000)	0.259 (F₁)	(0.128)	n/a ⁴	n/a	n/a	n/a
	Manatee	Grape	0.186 (F₁)	(0.065)	0.285 (F₁)	(0.074)	0.025 (F ₁)	(0.025)	0.508 (F ₁) ³	(0.136)	0.131 (F ₁)	(0.051)	0.552 (F ₁)	(0.060)
Myakka City Organic	Manatee	Tomato	0.028 (F ₁)	(0.028)	0.281 (F ₁)	(0.045)	0.025 (F ₁)	(0.025)	0.417 (F₁)	(0.122)	0.063 (F ₁)	(0.063)	0.676 (F ₁)	(0.040)
	Manatee	Tomato	0.132 (F ₁)	(0.017)	0.561 (F ₁)	(0.115)	0.086 (F ₁)	(0.030)	0.467 (F ₁) ³	(0.161)	0.317 (F ₁) ³	(0.010)	0.756 (F ₁)	(0.147)
2009														
Susceptible Strain GCC-1 GCC-2 GCC-3	Manatee	Cotton	0.359 (F ₂)	(0.052)	0.850 (F ₂)	(0.089)	0.451 (F ₂)	(0.072)	0.953 (F ₂)	(0.019)	0.488 (F ₂)	(0.078)	0.863 (F ₂)	(0.057)
	Collier	Tomato	0.046 (F ₁)	(0.046)	0.050 (F ₁)	(0.050)	0.025 (F ₁)	(0.025)	0.075 (F ₁)	(0.048)	0.108 (F ₁)	(0.045)	0.557 (F ₁)	(0.070)
	Collier	Tomato	0.031 (F ₁)	(0.031)	0.250 (F ₁)	(0.250)	0.025 (F ₁)	(0.025)	0.081 (F ₁)	(0.028)	0.023 (F ₁)	(0.023)	0.575 (F ₁) ³	(0.103)
	Collier	Grape	0.023 (F ₁)	(0.023)	0.048 (F ₁)	(0.028)	0.050 (F ₁)	(0.050)	0.073 (F ₁)	(0.046)	0.071 (F ₁)	(0.024)	0.394 (F ₁)	(0.071)
GCC-4 Devil's Garden Homestead - 1 GCC-5 GCC-EV GCC-BW	Collier	tomato	0.025 (F ₁)	(0.025)	0.015 (F ₁)	(0.050)	0.023 (F ₁)	(0.023)	0.203 (F ₁)	(0.107)	0.158 (F ₁) ³	(0.071)	0.856 (F ₁) ³	(0.076)
	Collier	Tomato	0.050 (F ₁)	(0.029)	0.075 (F ₁)	(0.048)	0.023 (F ₁)	(0.023)	0.075 (F ₁)	(0.025)	0.234 (F ₁) ³	(0.077)	0.725 (F ₁) ³	(0.080)
	Dade	Tomato	0.023 (F ₁)	(0.023)	0.093 (F ₁)	(0.064)	0.023 (F ₁)	(0.023)	0.175 (F ₁)	(0.103)	0.223 (F ₁)	(0.050)	0.434 (F ₁)	(0.109)
	Collier	Tomato	0.068 (F ₂)	(0.044)	0.150 (F ₂)	(0.029)	0.025 (F ₂)	(0.025)	0.400 (F ₂)	(0.041)	0.073 (F ₂)	(0.024)	0.448 (F ₂) ³	(0.189)
	Hendry	Tomato	0.000 (F ₂)	(0.000)	0.050 (F ₂)	(0.050)	0.025 (F ₂)	(0.025)	0.461 (F ₂)	(0.068)	0.153 (F ₂)	(0.027)	0.444 (F ₂)	(0.057)
	Collier	Cucumber/ melon	0.050 (F ₂)	(0.029)	0.100 (F ₂)	(0.041)	0.051 (F ₂)	(0.029)	0.350 (F ₂)	(0.087)	0.226 (F ₂) ³	(0.226)	0.675 (F ₂) ³	(0.125)

¹Number in parentheses for the susceptible strain indicate the times that the susceptible colony was tested at the time that field colonies were bioassayed.

²Bold percentage mortalities indicate that a given field population was known to be treated with that specific insecticide.

³LC mean values of field colonies followed by asterisks are not significantly different compared with the LC values of the susceptible strain.

⁴Population not available (n/a).

⁵SE not available, LC values are the average of 4 replicates.

TABLE 2. (CONTINUED) MEAN MORTALITY AND STANDARD ERROR (SE) OF FLORIDA FIELD POPULATIONS OF *BEMISIA TABACI* EXPOSED IN THE LABORATORY TO THE LC₅₀ AND LC₉₅ DOSES ESTIMATED FOR A SUSCEPTIBLE LABORATORY STRAIN. THE GENERATION BIOASSAYED IS INDICATED IN PARENTHESES, WITH GENERATION F₁ BEING THE ADULTS THAT EMERGED FROM FIELD-COLLECTED FOLIAGE.

Population	County	Crop	Imidacloprid			Thiamethoxam			Dinotefuran			Acetamiprid						
			LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)				
			2010															
Susceptible Strain	Manatee	Cotton	0.623 (4) ¹	(0.089)	0.954 (4)	(0.014)	0.606 (5)	(0.076)	0.933 (5)	(0.032)	0.564 (5)	(0.086)	0.923 (5)	(0.052)	0.552 (4)	(0.090)	0.882 (4)	(0.043)
GCC-ST	Collier	Cantaloupe	0.159 (F ₂) [*]	(0.023)	0.500 (F ₂)	(0.108)	0.077 (F ₁)	— ⁵	0.211 (F ₁)	—	0.356 (F ₃) [*]	(0.125)	0.687 (F ₂)	(0.075)	0.350 (F ₂) [*]	—	0.917 (F ₂) [*]	—
GCC-AM-O	Collier	Tomato	0.098 (F ₂) [*]	(0.053)	0.640 (F ₂) [*]	(0.257)	0.105 (F ₁)	—	0.150 (F ₁)	—	0.195 (F₂)	(0.120)	0.595 (F₂)	(0.095)	0.256 (F ₂) [*]	—	0.921 (F ₂) [*]	—
GCC-AM-P	Collier	Tomato	0.250 (F ₂) [*]	(0.025)	0.101 (F ₂)	(0.004)	0.057 (F ₁)	—	0.250 (F ₁)	—	0.075 (F₂)	(0.048)	0.501 (F₂)	(0.084)	0.474 (F ₂) [*]	—	0.974 (F ₂) [*]	—
Cortez-1	Manatee	Tomato	0.028 (F ₂) [*]	(0.028)	0.465 (F ₂) [*]	(0.154)	0.023 (F ₂)	(0.023)	0.271 (F ₁)	(0.060)	0.335 (F ₂)	(0.048)	0.653 (F ₂)	(0.048)	0.265 (F ₂)	(0.022)	0.430 (F ₂) [*]	(0.030)
GCC-JP	Hendry	Tomato	0.236 (F ₂)	(0.087)	0.636 (F ₂)	(0.037)	0.638 (F ₂) [*]	(0.153)	0.932 (F ₃) [*]	(0.044)	0.200 (F ₂) [*]	(0.091)	0.644 (F ₂)	(0.038)	0.141 (F ₂)	(0.024)	0.396 (F ₂)	(0.109)
Citra (Imidacloprid)	Marion	Tomato	0.159 (F ₃)	(0.023)	0.592 (F ₃)	(0.073)	0.297 (F ₂) [*]	(0.137)	0.743 (F ₂)	(0.059)	0.368 (F ₃) [*]	(0.098)	0.845 (F ₃) [*]	(0.077)	0.607 (F ₃) [*]	(0.083)	0.925 (F ₃) [*]	(0.025)
Citra (Dinotefuran)	Marion	Tomato	0.297 (F ₃) [*]	(0.137)	0.743 (F ₃)	(0.059)	0.135 (F ₂)	(0.023)	0.280 (F ₂)	(0.038)	0.136 (F ₂)	(0.079)	0.618 (F ₂) [*]	(0.131)	0.541 (F ₂) [*]	(0.043)	0.682 (F ₂) [*]	(0.079)
Wimauma-1	Manatee	Tomato	0.048 (F ₂)	(0.028)	0.486 (F ₂)	(0.064)	0.023 (F ₂)	(0.023)	0.640 (F ₂) [*]	(0.163)	0.139 (F ₂)	(0.030)	0.691 (F ₂)	(0.045)	0.406 (F ₂)	(0.0524)	0.842 (F ₂)	(0.020)
Wimauma-2	Manatee	Pepper	0.048 (F ₂)	(0.028)	0.666 (F ₂)	(0.029)	0.050 (F ₂)	(0.050)	0.643 (F ₂) [*]	(0.135)	0.159 (F ₂)	(0.044)	0.627 (F ₂) [*]	(0.133)	0.573 (F ₂) [*]	(0.057)	0.783 (F ₂) [*]	(0.152)

¹Number in parentheses for the susceptible strain indicate the times that the susceptible colony was tested at the time that field colonies were bioassayed.

²Bold percentage mortalities indicate that a given field population was known to be treated with that specific insecticide.

³LC mean values of field colonies followed by asterisks are not significantly different compared with the LC values of the susceptible strain.

⁴Population not available (n/a).

⁵SE not available, LC values are the average of 4 replicates.

TABLE 3. MEAN MORTALITY AND STANDARD ERROR OF *BEMISIA TABACI* POPULATIONS EXPOSED IN THE LABORATORY TO THE LC₅₀ AND LC₉₀ VALUES ESTIMATED FOR A SUSCEPTIBLE LABORATORY STRAIN. THE GENERATION BIOASSAYED IS INDICATED IN PARENTHESES, WITH THE F₁ GENERATION BEING THE ADULTS THAT EMERGED FROM FIELD-COLLECTED FOLIAGE.

Population	County	Crop	Bifenthrin			Endosulfan			Buprofezin						
			LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)			
Susceptible Strain GCC-IT GCC-WE GCC-AG Homestead-1 Homestead-4 GCC-SE GCC-CB Parrish-5 Myakka City Organic	Manatee	Cotton	0.513 (1) ¹	(0.101)	1.000 (1)	(0.000)	0.750 (1)	(0.050)	0.889 (1)	(0.111)	0.540 (5)	(0.052)	0.949 (5)	(0.033)	
	Collier	Tomato	0.156 (F₁)²	(0.052)	0.408 (F₂)	(0.106)	0.403 (F₁)	(0.042)	0.700 (F₁)^{2,3}	(0.041)	0.377 (F₁)²	(0.060)	0.756 (F₁)	(0.071)	
	Henry	Tomato	n/a ⁴	n/a	n/a	n/a	0.225 (F₂)	(0.025)	0.850 (F₂)²	(0.029)	0.206 (F₂)	(0.045)	0.725 (F₂)	(0.068)	
	Collier	Tomato	0.442 (F ₁) ²	(0.080)	0.800 (F ₁) ²	(0.108)	n/a	n/a	n/a	n/a	0.385 (F₁)²	(0.091)	0.667 (F₂)	(0.101)	
	Dade	Tomato	0.125 (F ₂)	(0.048)	0.686 (F ₂)	(0.078)	0.293 (F ₁)	(0.082)	0.864 (F ₁) ²	(0.059)	0.630 (F ₁) ²	(0.051)	0.878 (F ₁) ²	(0.063)	
	Dade	Tomato	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	0.588 (F ₁) ²	(0.136)	0.945 (F ₁) ²	(0.034)	
	Henry	Tomato	0.175 (F₃)²	(0.111)	0.625 (F₃)	(0.075)	n/a	n/a	n/a	n/a	0.343 (F₃)²	(0.157)	0.796 (F₂)	(0.055)	
	Henry	Tomato	0.150 (F₂)	(0.029)	0.652 (F₂)²	(0.182)	0.578 (F ₁)	(0.036)	0.952 (F ₁) ²	(0.028)	0.465 (F₂)²	(0.010)	0.833 (F₂)²	— ⁵	
	Grape tomato		0.203 (F ₂)	(0.069)	0.800 (F ₂)	(0.041)	0.431 (F ₁)	(0.100)	0.832 (F ₁) ²	(0.071)	n/a	n/a	n/a	n/a	
	Tomato		0.025 (F ₁)	(0.025)	0.338 (F ₂)	(0.055)	0.418 (F ₁)	(0.098)	0.809 (F ₁) ²	(0.074)	n/a	n/a	n/a	n/a	
	Tomato		0.153 (F ₁)	(0.027)	0.692 (F ₂)	(0.071)	0.455 (F ₁)	(0.064)	0.880 (F ₁) ²	(0.046)	n/a	n/a	n/a	n/a	
	Susceptible Strain GCC-1 GCC-2 GCC-3 GCC-EV GCC-BW Homestead - 1 GCC-4 GCC-5 Devil's Garden	Manatee	Cotton	0.475 (1)	(0.048)	(0.000)		0.438 (2)	(0.112)	0.862 (2)	(0.069)	n/a	n/a	n/a	n/a
Collier		Tomato	(0.086)	0.725 (F₃)²	(0.185)		0.350 (F ₃) ²	(0.065)	0.855 (F ₃) ²	(0.049)	n/a	n/a	n/a	n/a	
Collier		Tomato	0.350 (F ₃) ²	(0.050)	0.807 (F ₃)	(0.036)	0.425 (F ₃) ²	(0.025)	0.875 (F ₃) ²	(0.075)	n/a	n/a	n/a	n/a	
Collier		Grape tomato	0.100 (F ₁)	(0.041)	0.656 (F ₁)	(0.048)	0.459 (F ₃) ²	(0.072)	0.925 (F ₃) ²	(0.025)	n/a	n/a	n/a	n/a	
Henry		Tomato	0.150 (F ₁)	(0.065)	0.750 (F ₁) ²	(0.107)	0.264 (F ₂) ²	(0.076)	0.657 (F ₂) ²	(0.067)	n/a	n/a	n/a	n/a	
Collier		Cucumber/melon	n/a	n/a	n/a	n/a	0.250 (F ₂) ²	(0.087)	0.755 (F ₂) ²	(0.052)	n/a	n/a	n/a	n/a	
Dade		Tomato	0.146 (F ₂)	(0.027)	0.589 (F ₃) ²	(0.135)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	
Collier		Tomato		(0.041)	0.814 (F ₃) ²	(0.074)	0.575 (F ₃) ²	(0.075)	0.782 (F ₂) ²	(0.045)	n/a	n/a	n/a	n/a	
Collier		Tomato	n/a	n/a	n/a	n/a	0.364 (F ₂) ²	(0.038)	0.611 (F ₂)	(0.032)	n/a	n/a	n/a	n/a	
Henry		Tomato	n/a	n/a	n/a	n/a	0.550 (F ₂) ²	(0.029)	0.850 (F ₂) ²	(0.029)	n/a	n/a	n/a	n/a	
Susceptible Strain GCC-ST GCC-JP GCC-AM-O GCC-AM-P Cortez-1 Citra (Imidacloprid) Citra (Dinotefuran) Wimauma-1 Wimauma-2		Manatee	Cotton	n/a	n/a	n/a	n/a	0.462 (1)	(0.137)	1.000 (1)	(0.000)	n/a	n/a	n/a	n/a
		Collier	Cantaloupe	n/a	n/a	n/a	n/a	0.139 (F ₁) ²	(0.108)	0.268 (F ₁)	(0.085)	n/a	n/a	n/a	n/a
	Henry	Tomato	n/a	n/a	n/a	n/a	0.439 (F ₁) ²	(0.085)	0.718 (F₁)	(0.045)	n/a	n/a	n/a	n/a	
	Collier	Tomato	n/a	n/a	n/a	n/a	0.247 (F ₁) ²	(0.035)	0.559 (F ₁)	(0.066)	n/a	n/a	n/a	n/a	
	Collier	Tomato	n/a	n/a	n/a	n/a	0.364 (F ₁) ²	(0.083)	0.684 (F ₁)	(0.044)	n/a	n/a	n/a	n/a	
	Manatee	Tomato	n/a	n/a	n/a	n/a	0.121 (F ₁) ²	(0.074)	0.605 (F ₂)	(0.057)	n/a	n/a	n/a	n/a	
	Marion	Tomato	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	
	Marion	Tomato	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	
	Manatee	Tomato	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	
	Manatee	Tomato	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	
	Manatee	Pepper	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	

¹Number in parentheses for the susceptible strain indicate the times that the susceptible colony was tested at the time that field colonies were bioassayed.

²Bold percentage mortalities indicate that a given field population was known to be treated with that specific insecticide.

³LC mean values of field colonies followed by asterisks are not significantly different compared to the LC values of the susceptible strain.

⁴Population not available.

⁵SE not available, LC values are the average of 4 replicates.

TABLE 4. MEAN MORTALITY AND STANDARD ERROR OF FLORIDA FIELD POPULATIONS OF *BEMISIA TABACI* EXPOSED IN THE LABORATORY TO THE LC₅₀ AND LC₉₅ DOSES ESTIMATED FOR A SUSCEPTIBLE LABORATORY STRAIN FOR SELECTED INSECTICIDES FOR 2007 (PREDICTED) AND FOR 2008 TO 2010 (ACTUAL) FOR ADULTS/NYMPHS OF *B. TABACI* IN SOUTHERN FLORIDA. THE NUMBER IN EACH PARENTHESIS IS THE NUMBER OF POPULATIONS EVALUATED. MORTALITY AT THE LC VALUES FOLLOWED BY ASTERISKS ARE NOT SIGNIFICANTLY DIFFERENT COMPARED TO THE MORTALITY OF THE RESPECTIVE LC VALUE FOR THE SUSCEPTIBLE STRAIN.

Insecticide	2007 (Predicted)			2008 (Actual)			2009 (Actual)			2010 (Actual)		
	LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)	LC ₅₀	(SE)	LC ₉₅	(SE)
Imidacloprid	0.153 (16)	(0.020)	0.534 (16)	(0.048)	0.112 (10)*	(0.023)	0.355 (10)	(0.039)	0.035 (9)	(0.009)	0.082 (9)	(0.015)
Susceptible	—	—	—	—	0.514 (1)	(0.053)	0.950 (1)	(0.029)	0.359 (2)	(0.052)	0.850 (2)	(0.089)
Thiamethoxam	0.167 (18)	(0.016)	0.480 (18)	(0.050)	0.037 (10)	(0.010)	0.457 (10)*	(0.041)	0.030 (9)	(0.009)	0.210 (9)	(0.032)
Susceptible	—	—	—	—	0.650 (1)	(0.132)	0.850 (1)	(0.119)	0.451 (2)	(0.072)	0.953 (2)	(0.019)
Dinotefuran	0.191 (10)	(0.010)	0.660 (10)	(0.029)	0.122 (9)	(0.020)	0.685 (9)*	(0.025)	0.141 (9)	(0.019)	0.568 (9)*	(0.040)
Susceptible	—	—	—	—	0.575 (1)	(0.123)	0.975 (1)	(0.025)	0.488 (2)	(0.078)	0.863 (2)	(0.057)
Acetamiprid	0.398 (6)	(0.031)	0.784 (6)	(0.040)	0.138 (9)	(0.020)	0.707 (9)*	(0.033)	0.242 (9)	(0.037)	0.518 (9)	(0.042)
Susceptible	—	—	—	—	0.495 (1)	(0.058)	0.925 (1)	(0.048)	0.500 (2)	(0.060)	0.842 (2)	(0.071)
Bifenthrin	0.115 (7)	(0.022)	0.478 (7)	(0.049)	0.179 (8)*	(0.028)	0.625 (8)*	(0.042)	0.180 (6)*	(0.027)	0.723 (6)	(0.043)
Susceptible	—	—	—	—	0.513 (1)	(0.101)	1.000 (1)	(0.000)	0.475 (1)	(0.048)	1.000 (1)	(0.000)
Endosulfan	0.451 (8)	(0.021)	0.836 (8)	(0.024)	0.400 (7)*	(0.031)	0.841 (7)*	(0.023)	0.405 (8)*	(0.028)	0.789 (8)*	(0.024)
Susceptible	—	—	—	—	0.750 (1)	(0.050)	0.889 (1)	(0.111)	0.438 (2)	(0.112)	0.862 (2)	(0.069)
Buprofezin	n/a	—	n/a	—	0.438 (7)	(0.079)	0.802 (7)	(0.065)	n/a	n/a	n/a	n/a
Susceptible	—	—	—	—	0.540 (5)	(0.052)	0.949 (5)	(0.033)	n/a	n/a	n/a	n/a

*Population not available (n/a).

strain: GCC_1 ($t = -5.67$; $df = 6$; $P = 0.0013$ and $t = -6.30$; $df = 6$; $P = 0.0007$), GCC_2 ($t = -7.00$; $df = 6$; $P = 0.0004$ and $t = -6.99$; $df = 3$; $P = 0.0060$), GCC_3 ($t = -7.87$; $df = 6$; $P = 0.0002$ and $t = -6.75$; $df = 6$; $P = 0.0005$), GCC_4 ($t = -4.43$; $df = 6$; $P = 0.0044$ and $t = -8.66$; $df = 6$; $P = 0.0001$), DEVIL'S GARDEN ($t = -3.97$; $df = 6$; $P = 0.0073$ and $t = -9.66$; $df = 6$; $P < 0.0001$), HOMESTEAD_1 ($t = -4.52$; $df = 6$; $P = 0.0040$ and $t = -8.44$; $df = 6$; $P = 0.0002$), GCC_5 ($t = -5.36$; $df = 6$; $P = 0.0017$ and $t = -14.64$; $df = 6$; $P < 0.0001$), GCC_EV ($t = -5.67$; $df = 6$; $P = 0.0013$ and $t = -6.30$; $df = 6$; $P = 0.0007$), and GCC_BW ($t = -7.12$; $df = 6$; $P = 0.0004$ and $t = -13.43$; $df = 6$; $P < 0.0001$) (Table 2). For 2010, the mortality of 2 field populations at both the LC_{50} and LC_{95} values were not significantly different from those of the laboratory strain: GCC_AM (O) and CORTEZ_1, range ($t = -1.49$ to -0.96 ; $df = 3$ to 6 ; $P = 0.1865$ to 0.3828 and $t = -2.5$ to -0.874 ; $df = 3$ to 6 ; $P = 0.0848$ to 0.4777). Three more field populations did differ at the LC_{95} value: GCC-ST ($t = -3.25$; $df = 6$; $P = 0.0175$), GCC_AM (P) ($t = -26.22$; $df = 3$; $P = 0.0001$), and CITRA_D ($t = -4.39$; $df = 6$; $P = 0.0219$), but did not differ significantly at the LC_{50} value, ranging ($t = -2.41$ to -0.84 ; $df = 3$ to 6 ; $P = 0.0525$ to 0.4650). Four populations were significantly different at both the LC_{50} and LC_{95} values: GCC_JP ($t = -7.88$; $df = 6$; $P = 0.0002$ and $t = -9.80$; $df = 3$; $P = 0.0023$), CITRA_I ($t = -6.51$; $df = 6$; $P = 0.0006$ and $t = -5.59$; $df = 3$; $P = 0.0113$), WIMAUMA_1 ($t = -7.67$; $df = 6$; $P = 0.0003$ and $t = -6.72$; $df = 6$; $P = 0.0005$), and WIMAUMA_2 ($t = -7.67$; $df = 6$; $P = 0.0003$ and $t = -7.14$; $df = 6$; $P = 0.0004$). Three populations in 2008 and 4 in 2010 were known to have been treated with imidacloprid and all of these treated populations had lower mortality at both LC values compared with mortality at the LC values of the susceptible strain (Table 2). When mortality at the LC_{50} and LC_{95} were averaged over all sites for the actual data in 2008-2010 and compared with the estimated average mortality in 2007, susceptibility of *B. tabaci* appeared to decline from 2007 to 2009 and then rebound in 2010 (Table 4). However, the LC_{50} mean mortality value of the field populations in 2008 was not significant lower than the predicted LC_{50} mean value but it was significantly lower than the LC_{95} value ($t = -1.34$; $df = 48$; $P = 0.1853$ and $t = -2.61$; $df = 54$; $P = 0.0118$). In contrast, in 2009 the mortality at both LC mean values of the field populations were significant lower than the predicted LC mean values ($t = -6.05$; $df = 50$; $P < 0.0001$ and $t = -8.95$; $df = 18$; $P < 0.0001$). The LC mean mortality values of the field populations in 2010 were not significantly different than the predicted LC mean values in 2007 ($t = -0.98$; $df = 46$; $P = 0.3229$ and $t = -0.00$; $df = 49$; $P = 0.9995$) (Table 4). This apparent rebound increase in susceptibility may be due at least in part because the populations in 2010 were evaluated in the F_2 to F_3 generations rather than in the F_1 and F_2 genera-

tions as in 2008 and 2009. Previous research indicated that the longer *B. tabaci* populations are reared in the absence of exposure to imidacloprid the more susceptible they became to the insecticide (Schuster et al. 2010).

Considering thiamethoxam in 2008, all of the 10 field populations had LC_{50} mortality values that were lower and significantly different than that of the susceptible strain, but 5 of the populations had LC_{95} values that did not differ significantly from the susceptible colony: GCC_IT ($t = -4.64$; $df = 3$; $P = 0.0188$), GCC_WE ($t = -3.88$; $df = 5$; $P = 0.0117$), GCC_AG ($t = -4.09$; $df = 6$; $P = 0.0064$), PARISH_5 ($t = -4.64$; $df = 3$; $P = 0.0188$), and ORGANIC ($t = -4.16$; $df = 3$; $P = 0.0253$), and LC_{95} value, ranging ($t = -1.91$ to -1.04 ; $df = 6$; $P = 0.1041$ to 0.3388). The remaining 5 were susceptible and significantly different: HOMESTEAD_1 ($t = -4.64$; $df = 3$; $P = 0.0188$ and $t = -2.78$; $df = 6$; $P = 0.0321$), HOMESTEAD_4 ($t = -4.24$; $df = 6$; $P = 0.0054$ and $t = -4.03$; $df = 6$; $P = 0.0069$), GCC_SE ($t = -4.64$; $df = 3$; $P = 0.0188$ and $t = -3.11$; $df = 6$; $P = 0.0209$), GCC_CB ($t = -4.91$; $df = 3$; $P = 0.0161$ and $t = -3.38$; $df = 6$; $P = 0.0148$), and MYAKKA CITY ($t = -4.64$; $df = 3$; $P = 0.0188$ and $t = -2.55$; $df = 6$; $P = 0.0437$) (Table 2). In 2009, all 9 field populations had mortality values that were significantly lower than those of the susceptible strain at both LC dose levels: GCC_1 ($t = -7.24$; $df = 6$; $P = 0.0004$ and $t = -13.72$; $df = 6$; $P < 0.0001$), GCC_2 ($t = -7.24$; $df = 6$; $P = 0.0004$ and $t = -17.80$; $df = 3$; $P < 0.0001$), GCC_3 ($t = -5.54$; $df = 6$; $P = 0.0015$ and $t = -13.82$; $df = 6$; $P < 0.0001$), GCC_4 ($t = -4.56$; $df = 6$; $P = 0.0038$ and $t = -7.44$; $df = 3$; $P = 0.0050$), DEVIL'S GARDEN ($t = -4.56$; $df = 6$; $P = 0.0038$ and $t = -37.00$; $df = 3$; $P < 0.0001$), HOMESTEAD_1 ($t = -4.56$; $df = 6$; $P = 0.0038$ and $t = -8.00$; $df = 3$; $P = 0.0041$), GCC_5 ($t = -7.06$; $df = 6$; $P = 0.0004$ and $t = -12.32$; $df = 6$; $P < 0.0001$), GCC_EV ($t = -7.06$; $df = 6$; $P = 0.0004$ and $t = -6.94$; $df = 6$; $P = 0.0004$), and GCC_BW ($t = -6.43$; $df = 6$; $P = 0.0007$ and $t = -6.81$; $df = 3$; $P = 0.0065$) (Table 2). In 2010, one population (GCC-JP) had mortality levels approaching 0.50 and 0.95 ($t = -0.80$; $df = 10$; $P = 0.4445$ and $t = -0.70$; $df = 6$; $P = 0.5098$); however, this population was evaluated in the F_3 generation. One population was only significantly different at the LC_{95} value, CITRA_I ($t = -1.76$; $df = 6$; $P = 0.1291$ and $t = -4.39$; $df = 3$; $P = 0.0219$). Two more were significantly different at LC_{50} value only compared to LC values the susceptible strain, WIMAUMA_1 ($t = -18.28$; $df = 6$; $P < 0.0001$) and WIMAUMA_2 ($t = -12.91$; $df = 6$; $P < 0.0001$), and the LC_{95} value range ($t = -2.65$ to -2.20 ; $df = 3$; $P = 0.0768$ to 0.1147). The remaining 5 populations had significantly lower and significantly different mortality at both LC values compared to the susceptible strain: GCC_ST ($t = -2.57$; $df = 7$; $P = 0.0369$ and $t = -3.36$; $df = 3$; $P = 0.0439$), GCC_AM (O) ($t = -2.41$; $df = 7$; $P = 0.0466$ and $t = -3.67$; $df = 3$; $P = 0.0351$), GCC_AM (P) ($t = -2.58$; $df = 7$; P

= 0.0313 and $t = -3.15$; $df = 3$; $P = 0.0511$), CORTEZ_1 ($t = -3.13$; $df = 6$; $P = 0.0203$ and $t = -9.51$; $df = 6$; $P < 0.0001$), CITRA_D ($t = -10.38$; $df = 6$; $P < 0.0001$ and $t = -10.86$; $df = 6$; $P < 0.0001$). Only 3 populations were known to be treated in 2008, 3 and 2 were significantly different at the LC_{50} and LC_{95} , respectively, comparing with the LC values of the susceptible strain (Table 2). The average mortality of the field populations in 2008 did differ significantly from the predicted mortality of the 2007 populations at LC_{50} value but did not differ at the LC_{95} value ($t = 7.31$; $df = 55$; $P < 0.0001$ and $t = 0.34$; $df = 56$; $P = 0.7382$). In 2009 mortality differed significantly from 2007 at both LC values ($t = 8.30$; $df = 52$; $P < 0.0001$ and $t = 4.73$; $df = 52$; $P < 0.0001$) but in 2010 did not differ ($t = -0.29$; $df = 31$; $P = 0.7363$ and $t = -0.75$; $df = 43$; $P = 0.4583$) (Table 4).

Two populations evaluated with dinotefuran in 2008 had mortality values that were not significantly different compared with those of the susceptible strain, one only at the LC_{50} level, ORAGANIC ($t = -2.09$; $df = 3$; $P = 0.1275$ and $t = -3.30$; $df = 6$; $P = 0.0164$), and another at both levels, GCC_AG ($t = -2.57$; $df = 3$; $P = 0.0825$ and $t = -1.52$; $df = 6$; $P = 0.1799$) (Table 2). The remaining 7 populations were significantly different at both LC levels: GCC_IT ($t = -3.66$; $df = 6$; $P = 0.0105$ and $t = -5.93$; $df = 6$; $P = 0.0010$), GCC_WE ($t = -3.57$; $df = 6$; $P = 0.0118$ and $t = -3.08$; $df = 6$; $P = 0.0216$), HOMESTEAD_1 ($t = -4.38$; $df = 3$; $P = 0.0220$ and $t = -4.91$; $df = 6$; $P = 0.0027$), HOMESTEAD_4 ($t = -3.79$; $df = 6$; $P = 0.0091$ and $t = -5.43$; $df = 6$; $P = 0.0016$), GCC_SE ($t = -3.95$; $df = 6$; $P = 0.0075$ and $t = -5.48$; $df = 6$; $P = 0.0016$), PARISH_5 ($t = -3.34$; $df = 6$; $P = 0.0157$ and $t = -10.76$; $df = 6$; $P < 0.0001$), and MYAKKA CITY ($t = -3.71$; $df = 6$; $P = 0.0099$ and $t = -7.49$; $df = 6$; $P = 0.0003$). In 2009, 3 field populations were not significantly different at both LC values compared with values of the susceptible strain: GCC_4, DEVIL'S GARDEN, and GCC_BW, ranging ($t = -1.99$ -(-0.16); $df = 6$; $P = 0.0932$ -0.2913 and $t = -1.48$ -(-0.19); $df = 6$; $P = 0.1905$ -0.8576). Two more populations, GCC_2 ($t = -6.82$; $df = 6$; $P = 0.0005$) and GCC_5 ($t = -8.44$; $df = 6$; $P = 0.0002$), were not different only at the LC_{95} level, range ($t = -2.40$ -(-0.2.11); $df = 6$; $P = 0.0533$ -0.0794). The other populations were significantly different at both LC values: GCC_1 ($t = -5.54$; $df = 6$; $P = 0.0118$ and $t = -3.41$; $df = 6$; $P = 0.0143$), GCC_3 ($t = -6.26$; $df = 6$; $P = 0.0008$ and $t = -5.25$; $df = 6$; $P = 0.0019$), HOMESTEAD_1 ($t = -4.03$; $df = 6$; $P = 0.0069$ and $t = -3.52$; $df = 6$; $P = 0.0126$), and GCC_EV ($t = -6.60$; $df = 6$; $P = 0.0006$ and $t = -5.27$; $df = 6$; $P = 0.0019$). In 2010, one population was not significantly different from the susceptible strain at both LC levels: CITRA_1 ($t = -1.88$; $df = 6$; $P = 0.1096$ and $t = -1.27$; $df = 6$; $P = 0.2524$). Two populations differed significantly only at the LC_{95} level, GCC_ST ($t = -3.19$; $df = 6$; $P = 0.0188$) and

GCC_JP ($t = -3.86$; $df = 6$; $P = 0.0083$), LC_{50} range ($t = -2.22$ -(-2.15); $df = 6$; $P = 0.0678$ -0.0755), while two differed only at the LC_{50} level: CITRA_D ($t = -3.36$; $df = 6$; $P = 0.0152$) and WIMAUMA_2 ($t = -2.85$; $df = 6$; $P = 0.0293$), LC_{95} range ($t = -2.24$ -(-1.60); $df = 3$ -6; $P = 0.1608$ -0.1114). The remaining 4 populations were significantly different at both LC values: GCC_AM (O) ($t = -3.45$; $df = 6$; $P = 0.0137$ and $t = -3.50$; $df = 6$; $P = 0.0128$), GCC_AM (P) ($t = -8.00$; $df = 6$; $P = 0.0002$ and $t = -4.86$; $df = 6$; $P = 0.0028$), CORTEZ_1 ($t = -4.36$; $df = 6$; $P = 0.048$ and $t = -2.63$; $df = 6$; $P = 0.0389$) and WIMAUMA_1 ($t = -3.08$; $df = 6$; $P = 0.0217$ and $t = -4.68$; $df = 6$; $P = 0.0034$). Only one population was known to be treated in 2008 and 3 in 2010, the first was not significantly different but all 3 and 2 in 2010 were significantly different at the LC_{50} and LC_{95} values, respectively, comparing with the LC values of the susceptible strain (Table 2). The average mortality of the field populations in 2008 and 2009 differed significantly from that of the average mortality predicted in 2007 at the LC_{50} level but not at the LC_{95} level ($t = -3.04$; $df = 44$; $P = 0.0040$ and $t = 0.52$; $df = 44$; $P = 0.6069$) and ($t = -2.29$; $df = 44$; $P = 0.0268$ and $t = -1.88$; $df = 39$; $P = 0.0673$), respectively. In 2010, there were no differences at either LC level ($t = 0.87$; $df = 41$; $P = 0.3893$ and $t = -0.18$; $df = 31$; $P = 0.8601$).

Considering acetamiprid, no field population in 2008 had mortality higher than 0.23 at the LC_{50} value and only 3, GCC_IT ($t = -3.95$; $df = 6$; $P = 0.007$), GCC_AG ($t = -3.16$; $df = 6$; $P = 0.0196$), and ORGANIC ($t = -2.87$; $df = 6$; $P = 0.0285$) had mortality not significantly different from the susceptible strain at the LC_{95} value, ranging $t = -0.53$ -0.45; $df = 6$; $P = 0.3139$ -0.6704) (Table 2). The remaining 6 populations were significantly different at both LC values compared with the susceptible strain: GCC_WE ($t = -7.49$; $df = 6$; $P = 0.0003$ and $t = -2.54$; $df = 6$; $P = 0.0438$), HOMESTEAD_1 ($t = -5.55$; $df = 6$; $P = 0.0014$ and $t = -3.19$; $df = 6$; $P = 0.0188$), HOMESTEAD_4 ($t = -6.91$; $df = 6$; $P = 0.0005$ and $t = -3.58$; $df = 6$; $P = 0.0116$), GCC_SE ($t = -4.27$; $df = 6$; $P = 0.0053$ and $t = -4.65$; $df = 6$; $P = 0.035$), PARRISH_5 ($t = -3.97$; $df = 6$; $P = 0.0074$ and $t = -4.86$; $df = 6$; $P = 0.0028$), and MYAKKA CITY ($t = -4.12$; $df = 6$; $P = 0.0062$ and $t = -3.99$; $df = 6$; $P = 0.0072$). In 2009, 3 populations did not differ significantly from the susceptible strain at either LC value: GCC_4, DEVIL GARDEN, and GCC_BW, ranging ($t = -1.17$ -(-0.43); $df = 6$; $P = 0.2857$ -0.6790 and $t = -2.26$ -(-0.12); $df = 6$; $P = 0.0643$ -0.9111). One population differed at the LC_{95} value but not at the LC_{50} value, HOMESTEAD_1 ($t = -1.08$; $P = 0.3213$ and $t = -2.76$; $P = 0.0329$) while 5 differed at both LC values: GCC_1 ($t = -7.76$; $df = 6$; $P = 0.0002$ and $t = -4.41$; $df = 6$; $P = 0.0045$), GCC_2 ($t = -3.62$; $df = 6$; $P = 0.0111$ and $t = -5.03$; $df = 6$; $P = 0.0024$), GCC_3 ($t = -7.21$; $df = 6$; $P = 0.0004$ and $t = -6.33$; $df = 6$; $P = 0.0007$), GCC_5 ($t = -5.40$; $df = 6$; $P = 0.0007$), and

= 6; $P = 0.0017$ and $t = -4.64$; $df = 6$; $P = 0.0035$), and GCC_EV ($t = -4.28$; $df = 6$; $P = 0.0052$ and $t = -2.85$; $df = 6$; $P = 0.0294$). In 2010, 6 populations were not significantly different from the susceptible strain at both LC values: GCC_ST, GCC_AM (O), GCC_AM (P), CITRA_I, CITRA_D, and WIMAUMA_2, ranging ($t = -1.59$ - 1.21 ; $df = 6$ - 7 ; $P = 0.1562$ - 0.9946 and $t = -1.60$ - 1.43 ; $df = 6$ - 7 ; $P = 0.1615$ - 0.6083). The remaining 3 populations were significantly different at both LC values: CORTEZ_1 ($t = -3.09$; $df = 10$; $P = 0.0115$ and $t = -5.85$; $df = 10$; $P = 0.0002$), GCC_JP ($t = -4.90$; $df = 10$; $P = 0.0006$ and $t = -4.47$; $df = 10$; $P = 0.0012$), WIMAUMA_1 ($t = 2.73$; $df = 6$; $P = 0.0342$ and $t = 8.07$; $df = 3$; $P = 0.0040$). There was not information on populations treated (Table 2). The average mortality of the field populations in 2008 differed significantly from that of the average mortality predicted in 2007 at the LC_{50} level but not at the LC_{95} level ($t = -5.15$; $df = 40$; $P = <0.0001$ and $t = -0.93$; $df = 40$; $P = 0.3567$). Average mortality in 2009 was significantly different at both LC values ($t = -2.53$; $df = 82$; $P = 0.0133$ and $t = -3.20$; $df = 23$; $P = 0.0040$) while in 2010 differences were not significant at either LC value ($t = 0.22$; $df = 31$; $P = 0.8298$ and $t = -1.25$; $df = 22$; $P = 0.2240$) (Table 4).

One population evaluated with the pyrethroid bifenthrin in 2008 had mortality approximating both 0.50 and 0.95 which were not significantly different at the respective LC_{50} and LC_{95} values compared with the susceptible strain: GCC_AG ($t = -0.55$; $df = 6$; $P = 0.6017$ and $t = -1.85$; $df = 3$; $P = 0.1612$) (Table 3). Another population differed significantly only at the LC_{50} value: GCC_CB ($t = -3.46$; $df = 6$; $P = 0.0135$ and $t = -1.91$; $df = 3$; $P = 0.1516$) and one only at LC_{95} value: GCC_SE ($t = -2.25$; $df = 6$; $P = 0.0652$ and $t = -5.00$; $df = 3$; $P = 0.0154$). The remaining 5 were significantly different at both LC values: GCC_IT ($t = -3.15$; $df = 6$; $P = 0.0199$ and $t = -5.60$; $df = 3$; $P = 0.0014$), HOMESTEAD_1 ($t = -3.47$; $df = 6$; $P = 0.0133$ and $t = -4.02$; $df = 3$; $P = 0.0277$), PARRISH_5 ($t = -2.53$; $df = 6$; $P = 0.0446$ and $t = -4.90$; $df = 3$; $P = 0.0163$), MYAKKA CITY ($t = -4.70$; $df = 3$; $P = 0.0183$ and $t = -11.95$; $df = 3$; $P = 0.0013$), and ORGANIC ($t = -3.44$; $df = 6$; $P = 0.0137$ and $t = -4.33$; $df = 3$; $P = 0.0277$). During 2009, mortality of one population was significantly different only at the LC_{95} value, GCC_2 ($t = -1.81$; $df = 6$; $P = 0.1210$ and $t = -5.45$; $df = 3$; $P = 0.0122$) while 4 were significantly different only at the LC_{50} value: GCC_1 ($t = -2.46$; $df = 6$; $P = 0.0489$), GCC_EV ($t = -4.04$; $df = 6$; $P = 0.0068$), HOMESTEAD_1 ($t = -6.02$; $df = 6$; $P = 0.0009$) and GCC_4 ($t = -5.96$; $df = 6$; $P = 0.0010$) but not at the LC_{95} , ranging ($t = -3.05$ - (-1.49) ; $df = 3$; $P = 0.0555$ - 0.22335).

Only one population differed significantly from the susceptible strain at both LC values: GCC_3 ($t = -5.96$; $df = 6$; $P = 0.0010$ and $t = -7.12$; $df = 3$; $P = 0.0057$). Unfortunately, there were not enough whiteflies to bioassay for bifenthrin re-

sistance monitoring in 2010. Three populations were known to be treated in 2008, 2 had lower and significantly different mortality at both LC levels comparing with the LC levels of the susceptible strain (Table 3). The average mortality of the field populations in 2008 was not significantly different from the estimated values in 2007 at either LC level ($t = 1.79$; $df = 27$; $P = 0.0856$ and $t = 1.58$; $df = 37$; $P = 0.1227$). In 2009, average mortality did not differ at the LC_{50} value but did at the LC_{95} value ($t = 1.24$; $df = 29$; $P = 0.2241$ and $t = 2.89$; $df = 29$; $P = 0.0073$) (Table 4).

Considering the organochlorine endosulfan, mortality of all 7 field populations tested in 2008 were significantly different at the LC_{50} value but not at the LC_{95} value compared to the mortality of the susceptible strain at the respective LC values: GCC_IT ($t = -5.33$; $df = 6$; $P = 0.0018$), GCC_WE ($t = -9.39$; $df = 6$; $P = <0.0001$), HOMESTEAD_1 ($t = -4.76$; $df = 6$; $P = 0.0031$), GCC_CB ($t = -2.79$; $df = 6$; $P = 0.0314$), PARRISH_5 ($t = -2.84$; $df = 6$; $P = 0.0294$), MYAKKA CITY ($t = -3.02$; $df = 6$; $P = 0.0234$), and ORGANIC ($t = -3.63$; $df = 6$; $P = 0.0110$), LC_{95} value, ranging ($t = -1.60$ - 0.55 ; $df = 6$; $P = 0.1617$ - 0.9405) (Table 3). In 2009, mortality of one of the field populations differed significantly from that of the susceptible strain only at the LC_{95} value, GCC_5 ($t = -0.37$; $df = 3$; $P = 0.7341$ and $t = -3.30$; $df = 6$; $P = 0.0164$). The other 7 populations did not differ significantly at either LC value: GCC_1, GCC_2, GCC_3, GCC_EV, GCC_BW, GCC_4, and DEVTL GARDEN, range ($t = -1.85$ - 0.85 ; $df = 3$ - 6 ; $P = 0.1135$ - 0.9081 and $t = -2.26$ - 0.91 ; $df = 6$; $P = 0.0645$ - 0.9121). The mortality of the 5 field populations evaluated in 2010 was significantly different from that of the susceptible strain at the LC_{95} value: GCC_ST ($t = -8.61$; $df = 3$; $P = 0.0033$), GCC_JP ($t = -6.28$; $df = 3$; $P = 0.0081$), GCC_AM (O) ($t = -6.71$; $df = 3$; $P = 0.0068$), GCC_AM (P) ($t = -7.21$; $df = 3$; $P = 0.0055$) and CORTEZ_1 ($t = -6.97$; $df = 3$; $P = 0.0061$), but not at the LC_{50} range ($t = -2.20$ - (-0.15) ; $df = 3$ - 6 ; $P = 0.0704$ - 0.8890). Two populations known to be treated in 2008 and one in 2010, both differed significantly only at the LC_{50} value in 2008 and one only at LC_{95} value in 2010, comparing with LC values of the susceptible strain (Table 3). The average mortality of the field populations in 2008 and 2009 did not differ significantly from the average predicted mortality of the 2007 populations at either LC value ($t = -1.34$; $df = 32$; $P = 0.1891$ and $t = 0.11$; $df = 34$; $P = 0.9164$) and ($t = -1.31$; $df = 32$; $P = 0.1987$ and $t = -0.95$; $df = 38$; $P = 0.3458$), respectively; however, average mortality in 2010 differed significantly at both LC values ($t = -3.96$; $df = 25$; $P = 0.0005$ and $t = -0.536$; $df = 26$; $P = <0.0001$).

Susceptibility of *B. tabaci* to buprofezin was evaluated only in 2008 when 3 populations approximated 0.50 and 0.95 at the LC_{50} and LC_{95} levels, respectively, and did not differ significantly

from the respective LC values of the susceptible strain, HOMESTEAD_1, HOMESTEAD_4, and GCC_CB, ranging ($t = 0.36$ - 1.43 ; $df = 4$ - 7 ; $P = 0.2291$ - 0.7323 and $t = -0.142$ - (-0.09) ; $df = 4$ - 7 ; $P = 0.2283$ - 0.9321). The mortality of 3 of the populations was significantly different from the susceptible strain only at the LC_{95} value: GCC_IT ($t = -2.64$; $df = 7$; $P = 0.0335$), GCC_AG ($t = -3.30$; $df = 6$; $P = 0.0165$), and GCC_SE ($t = -2.57$; $df = 6$; $P = 0.0424$), LC_{50} value range ($t = -2.07$ - (-1.57) ; $df = 5$ - 7 ; $P = 0.0775$ - 0.1777) (Table 3). Only the GCC_WE population had mortality that was significantly different from the susceptible strain at both LC values ($t = -4.70$; $df = 7$; $P = 0.0022$ and $t = -3.17$; $df = 7$; $P = 0.0156$) (Table 3). From 5 populations known to be treated, one was significant different at the LC_{50} value and 4 at LC_{95} value when comparing with the LC values of the susceptible strain. There were no field populations available to predict the field LC values in 2007 and there was collected field data only in 2008. Therefore, there was no comparison between field predicted LC values and field collected populations; however, the mean mortalities at LC_{50} and LC_{95} values of 0.438 and 0.802, respectively, indicate satisfactory susceptibility at field level (Table 4).

DISCUSSION

The results of the survey in southern Florida demonstrated that, when the mortality was averaged over all populations within a year at both the LC_{50} and LC_{95} values, all of the neonicotinoids indicated decreases in average susceptibility in 2008 and 2009 compared with 2007 (Table 4). The trend was particularly true for imidacloprid and thiamethoxam, especially in 2009. Both of these neonicotinoids were registered for use before either dinotefuran or acetamiprid. Data from the individual populations within years also support these observations. All populations known to be treated with these two insecticides also had lower and significantly different mortality values at both LC values compared with those of the susceptible strain. From 10 populations collected from the field in 2008, mortality of only one exposed to imidacloprid in the laboratory did not differ statistically from that of the susceptible laboratory strain at both LC doses (Table 2). All 10 populations exposed to the LC_{50} value for thiamethoxam differed from the susceptible strain, although 5 populations exposed at the LC_{95} did not. Similarly, from 9 field populations exposed to dinotefuran in the laboratory, only one did not differ significantly from the laboratory strain at both LC values. Another population exposed to dinotefuran at the LC_{50} dose and 3 populations exposed to the LC_{95} value for acetamiprid failed to differ. In 2009, all 9 populations exposed to either imidacloprid or thiamethoxam in the laboratory had mortality that was significantly lower than

that of the laboratory strain at both LC doses. In contrast, 3 populations exposed to either dinotefuran or acetamiprid failed to differ from the laboratory strain at both LC doses. An additional two populations exposed to dinotefuran at the LC_{95} dose and one exposed to acetamiprid at the LC_{50} dose also failed to differ. Of the 9 field populations collected in 2010, the mortality of 5 exposed to imidacloprid, 4 exposed to thiamethoxam, 5 of 6 exposed to dinotefuran and all 6 exposed to acetamiprid failed to differ from the mortality of the susceptible strain at least at one of the LC values. The apparent decline in susceptibility from 2007 to 2009 continues the trend in declining susceptibility that was documented previously from 2000 to 2007 for imidacloprid (the neonicotinoid first registered for use) and from 2003 to 2007 for thiamethoxam (the second neonicotinoid registered). The results further indicate that average susceptibility to the neonicotinoids increased in 2010 to levels observed in 2007. However, the populations of *B. tabaci* in the field were low in the spring of 2010, which necessitated that the populations be reared for one to 3 additional generations in the laboratory before sufficient specimens could be obtained for the bioassays. Susceptibility of populations with apparent tolerance to either imidacloprid or thiamethoxam was shown to increase the more generations the populations were reared in the laboratory without exposure to the insecticides (Schuster et al. 2010). Thus, the apparent increase in susceptibility in 2010 may have resulted at least in part from rearing *B. tabaci* in the absence of exposure to the insecticides rather than from a real increase in susceptibility in the field.

Averaged mortality values for bifenthrin suggested an overall increase in field susceptibility from 2007 to 2009 (Table 4). Mortality of 3 of the 8 field populations exposed to bifenthrin in 2008 and 5 of the 6 in 2009 did not differ from the laboratory populations at least at one of the LC values (Table 3). Average mortality values for endosulfan suggested no change from 2007 to 2009 (Table 4). Mortality of all 7 of the 2008 field populations exposed to the LC_{95} value did not differ from that of the susceptible strain and all but one of the 8 field populations in 2009 differed at both of the LC values. *B. tabaci* appeared to be more susceptible to endosulfan from 2007 to 2009 than to the neonicotinoids or to bifenthrin. However, endosulfan was evaluated on later generations than were the neonicotinoids. Nevertheless, the average susceptibility to endosulfan appeared to decrease in 2010, with mortality of all 5 field populations exposed to the LC_{50} value of endosulfan not differing significantly from that of the laboratory population. In addition, populations known to be treated with bifenthrin or endosulfan support the increase or stabilization of susceptibility as field-treated populations had higher mortalities and

fewer significant differences compared with the LC values of the susceptible strain. There were no data in 2007 predicted for buprofezin, but the 2008 mean field mortality at the LC₅₀ and LC₉₅ values were 0.438 and 0.802, respectively, suggesting that field susceptibility was at an acceptable level. Populations known to be treated with this insecticide also reveals susceptibility at LC₅₀ value.

Because of the demonstrated decrease in susceptibility of *B. tabaci* to the neonicotinoids in this and previous studies (Schuster et al. 2010) and because of the propensity of the whitefly to developed resistance to all of the major classes of insecticides (Palumbo et al. 2001; Horowitz et al. 2007), recommendations for managing not only *B. tabaci* and the TYLCV it transmits but also for managing insecticide resistance on vegetables in Florida were developed by a consortium of University of Florida, chemical industry, commodity organizations, and crop consultant representatives (Schuster 2007). The recommendations included pre- and post-planting cultural practices for delaying the onset of whitefly infestations and for reducing the level of infestations during the crop, thus reducing insecticide use and selection pressure for the development of insecticide resistance. The recommendations also included specific recommendations for the selection, timing and application of insecticides.

The results demonstrate changes in insecticide susceptibility through time of field populations in Florida based upon the mortality of field populations exposed in the laboratory to the estimated field LC₅₀ and LC₉₅ values of a susceptible laboratory strain. The results of the mean of populations per year (2008-2009), compared with the predicted field value of 2007 substantiate the trend of decreasing of susceptibility for the neonicotinoids through time, particularly to imidacloprid and thiamethoxam and to a lesser extent to dinotefuran and acetamiprid. Mortality values for bifenthrin suggested an overall increase in field susceptibility in 2008 and 2009 while values for endosulfan suggested no change. There were no 2007 data for buprofezin, but the 2008 average mortality at the LC₅₀ and LC₉₅ were 0.438 and 0.802, respectively, indicating that field susceptibility was at an acceptable level. In 2010 average susceptibility to the neonicotinoids appeared to increase compared with previous years; however, the field populations tended to be evaluated, in general, after they had been reared in the laboratory without exposure to insecticides for more generations than in previous years. Despite this, average susceptibility to endosulfan appeared to decrease.

Exposing field populations to two doses rather than a range of doses requires fewer insects and less time. Assessing mortality at these two data points provides an indicator of the nature of resis-

tance development within a population. Changes in mortality at the LC₅₀ (slope independent mortality) may differ from changes in mortality at the LC₉₅ (slope dependent mortality). A shallow slope indicates a slow change in decrease of susceptibility, whereas a steep slope indicates a rapid development of tolerance. The data presented here can provide important information that would be helpful to growers and producers for making decisions on a more rational use or abandonment of use of a specific insecticide.

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