Influence of Posttreatment Temperature on the Toxicity of Insecticides Against *Diaphorina citri* (Hemiptera: Psyllidae)

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ABSTRACT The psyllid *Diaphorina citri* Kuwayama (Hemiptera: Psyllidae) is one of the most important pests of citrus worldwide because it efficiently vectors three bacteria in the genus *Candidatus* Liberibacter that cause the devastating citrus greening disease (huanglongbing). Current management practices for this insect pest rely on multiple sprays of foliar insecticides and one or two applications of soil systemic insecticides per season. Effective psyllid and disease management in Florida requires insecticide applications throughout the entire season over wide ranging temperature and environmental conditions. Using a petri dish bioassay technique, the effect of posttreatment temperature (range, 17–37°C) on the toxicity of selected organophosphate (chlorpyrifos and dimethoate), carbamate (carbaryl), avermectin (abamectin), pyrethroid (bifenthrin, zeta-cypermethrin, fenpropathrin, and lambda-cyhalothrin), and neonicotinoid (acetamiprid, imidacloprid, and thiamethoxam) insecticides was evaluated against adult *D. citri*. The toxicity of both organophosphates showed a positive temperature correlation within the 17–37°C range. Similarly, carbaryl (carbamate) and abamectin (avermectin) exhibited increased toxicity with increasing temperature from 17 to 37°C, with abamectin showing higher overall temperature-dependent toxicity against *D. citri* adults than carbaryl. With the exception of bifenthrin, which showed a positive temperature-dependent toxicity correlation between 27 and 37°C, all other pyrethroids tested exhibited a negative correlation over the temperature range examined. The toxicity of fenpropathrin and lambda-cyhalothrin dramatically decreased with increasing temperature from 17 to 37°C. The neonicotinoids imidacloprid and thiamethoxam exhibited a mixed response to increasing temperature, whereas acetamiprid showed a positive temperature correlation. However, all three neonicotinoids showed positive temperature-dependent toxicity correlations against *D. citri* adults over the temperature range tested. These data will enable citrus growers to choose the most effective insecticides for *D. citri* control from the various classes currently available depending on the prevailing temperature conditions.

KEY WORDS *Diaphorina citri*, citrus, huanglongbing, citrus greening disease, temperature-dependent toxicity

Diaphorina citri Kuwayama (Hemiptera: Psyllidae) is one of the most important worldwide pests of citrus (Halbert and Manjunath 2004). It causes both direct and indirect damage by feeding on plant sap and by acting as a vector of three phloem-restricted bacteria in the genus *Candidatus* Liberibacter, which cause citrus greening (huanglongbing), the most serious disease of citrus worldwide (Catling 1970, Halbert and Manjunath 2004). Citrus greening was confirmed in Florida in 2005 and has since become established throughout all citrus growing regions in the state (Anonymous 2008a). Infected citrus trees initially exhibit symptoms of leaf mottling and chlorosis (Bové 2006). Subsequently, plants become stunted and begin dying from branch tips, potentially resulting in tree death. Diseased trees produce less fruit that are misshapen, bitter-tasting, and unmarketable (Bové 2006). The Florida citrus industry, with an annual farmgate value of $1.5 billion (Anonymous 2008b), is threatened if the greening disease vector is not controlled. Given its firm establishment throughout commercial citrus in Florida, eradication of *D. citri* is impossible and management efforts are focused on stringent control with insecticides (Rogers et al. 2008a, Srinivasan et al. 2008). It is believed that these intensive control practices may slow the spread of greening disease throughout Florida and to other citrus growing states in the United States where this disease has not yet invaded (Srinivasan et al. 2008).

Under the subtropical and tropical climates of Florida, citrus agroecosystems are exposed to a wide range of

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Table 1. Details of insecticides used to test the influence of temp on toxicity against *D. citri*

Insecticide	Trade name	Class	Mode of action	Manufacturer/supplier
Chlorpyrifos[a]	Lorsban 4E	Organophosphate	Acetylcholinesterase inhibitor	Dow AgroSciences, LLC, Indianapolis, IN
Dimethoate	Dimate 4EC	Organophosphate	Acetylcholinesterase inhibitor	Agriliance, LLC, St. Paul, MN
Carbaryl[a]	Sevin XLR Plus	Carbamate	Acetylcholinesterase inhibitor	Bayer CropScience LP, Research Triangle Park, NC
Abamectin	Agri-Mek 0.15EC	Avermectin	Chloride channel activator	Syngenta Crop Protection, Inc., Greensboro, NC
Bifenthrin	Brigade 2EC	Synthetic pyrethroid	Sodium channel modulator	FMC Corporation, Philadelphia, PA
zeta-Cypermethrin	Mustang	Synthetic pyrethroid	Sodium channel modulator	FMC Corporation
Fenpropathrin[a]	Danitol 2.4EC	Synthetic pyrethroid	Sodium channel modulator	Valent USA Corp., Walnut Creek, CA
lambda-Cyhalothrin	Warrior	Synthetic pyrethroid	Sodium channel modulator	Syngenta Crop Protection, Inc.
Acetamiprid	Assail 70WP	Neonicotinoid	Nicotinic acetylcholine receptor agonist/antagonist	Cerexagri, Inc., King of Prussia, PA
Imidacloprid[a]	Provado 1.6F	Neonicotinoid	Nicotinic acetylcholine receptor agonist/antagonist	Bayer CropScience LP
Thiamethoxam	Actara 25WDG	Neonicotinoid	Nicotinic acetylcholine receptor agonist/antagonist	Syngenta Crop Protection

[a] Currently recommended for *D. citri* control on citrus in Florida (Rogers et al. 2008b).

temperatures (10–35°C) from early spring when the new leaf flush emerges until late fall. Environmental conditions, mainly temperature, significantly affect the toxicity of insecticides and thus their efficacy (Scott 1995). In general, the effect of temperature on insecticide efficacy against insects is similar within a given insecticide class. For example, the toxicity of organophosphate insecticides is positively correlated with temperature (Scott 1995, Satpute et al. 2007), and the toxicity of pyrethroid insecticides is negatively correlated with temperature (Scott 1995, Musser and Shelton 2005, Satpute et al. 2007). However, some studies have shown that temperature-dependent toxicity of insecticides varies within a given class (Scott 1995). Furthermore, factors such as insect species, the temperature range tested, insecticide mode of action, method of application, and amount of insecticide contacted or ingested are known to influence the temperature–toxicity relationship, which could be positive, negative, or mixed (Sparks et al. 1982, Toth and Sparks 1990). For example, the toxicity of two pyrethroids, *cis*-permethrin and lambda-cyhalothrin, decreased with increasing temperature against *Trichoplusia ni* (Hübner), whereas another pyrethroid, esfenvalerate, showed no trend in toxicity with varying temperature (Toth and Sparks 1990). Similarly, two other pyrethroids, deltamethrin and bifenthrin, showed a mixed response with an overall positive temperature-dependent toxicity correlation between 17 and 37°C against *Chilo suppressalis* (Walker) (Li et al. 2006). The above-mentioned studies indicate that temperature has a considerable effect on insecticide efficacy, and this effect varies with chemistry and insect species. As a result, generalizations of temperature–toxicity trends for a given chemical class cannot be made among different insect species or between crops and plant types. Currently, several soil and foliar insecticides belonging to different classes are applied for *D. citri* control (Rogers et al. 2008a); however, no information is available on how temperature affects the toxicity of these insecticides against *D. citri.* Furthermore, there is a paucity of data on how temperature variation affects toxicity of neonicotinoids. This is particularly true of imidacloprid, which is applied both as a soil drench and foliar spray for *D. citri* control. Temperature-dependent toxicity information generated for insecticides that are currently recommended and will be available in the future for *D. citri* control in citrus will enable growers to choose the most suitable insecticide depending on the prevailing temperature conditions for effective management of *D. citri.* The objective of the current study was to determine the temperature–toxicity relationships of selected insecticides from organophosphate, carbamate, avermectin, pyrethroid, and neonicotinoid classes against *D. citri* adults.

Materials and Methods

Insects. Adult *D. citri* were collected using manual aspirators from a commercial Valencia orange grove in Lake Alfred, FL, during March–May 2008 when the temperatures in the field ranged between 18 and 25°C. This grove did not receive insecticide applications at least 1 mo before or during the psyllid collection period. Insects were carefully transported in plastic vials to the laboratory. Immediately after transportation to the laboratory, insects were placed into plastic cages with rough lemon plants, *Citrus jambhiri* (Lush), and maintained at 27 ± 1°C and 50 ± 5% RH. Insects were allowed to feed and acclimate to rearing conditions for 24 h before subjecting them to insecticide testing, so that previous exposure to field temperatures would not impact laboratory results. Insects that were active and feeding on the subsequent day were selected for insecticide bioassays.

Insecticides and Bioassays. In total, 11 insecticides representing different chemistries and modes of action were selected for testing the effect of temperature (17, 27, and 37°C) on toxicity (Table 1). At each temperature, five concentrations of each insecticide were tested that gave mortality between 1 and 99% based on preliminary studies. Test concentrations for each insecticide were prepared in distilled water by

Table 2. Influence of temp on the toxicity of organophosphate insecticides against *D. citri* adults

Insecticide[a]	Temp. (°C)	LC_{50}[b] (mg [AI]/liter)	95% CL	Slope ± SE	χ^2 (df)[c]	*P* value	Temp. coefficient[d]	
							10°C	20°C
Chlorpyrifos	17	4.22	2.02–7.39	2.77 ± 0.58	12.04 (3)[e]	0.007		
	27	1.12	0.93–1.33	2.70 ± 0.29	4.59 (3)	0.203	+3.76	
	37	0.40	0.34–0.46	2.78 ± 0.28	5.86 (3)	0.118	+2.80	+10.50
Dimethoate	17	4.78	3.84–6.02	1.69 ± 0.20	0.60 (3)	0.894		
	27	3.49	2.00–5.09	3.58 ± 0.62	9.34 (3)[e]	0.025	+1.36	
	37	0.78	0.001–1.36	2.33 ± 0.69	9.53 (3)[e]	0.023	+4.47	+6.12

[a] At each insecticide and temperature combination, three replicates of 10 insects were used per experiment.

[b] Concentration required for 50% mortality in *D. citri* adults.

[c] χ^2 goodness-of-fit statistic and degrees of freedom.

[d] Ratio of higher to lower LC_{50} value for 10 and 20°C differences in exposure temperature. A positive coefficient suggests a lower LC_{50} value at the higher temperature.

[e] χ^2 significantly different from expected ($P \leq 0.05$).

using commercially formulated products on the day of testing. Bioassays were conducted using the petri dish technique developed by Prabhaker et al. (2006) for determining the baseline susceptibility of glassy-winged sharpshooter, *Homalodisca vitripennis* (Germar). In 60-mm-diameter plastic disposable petri dishes (Fisherbrand, Thermo Fisher Scientific, Waltham, MA), 1.5% agar solution was poured and allowed to form a bed after solidification. In preliminary tests, it was found that such agar beds maintain leaf discs fresh for at least 1 wk. Citrus leaves were collected from a Valencia orange grove that was not treated with insecticides, and 60-mm-diameter leaf discs were excised. Excised leaf discs were dipped in test solutions for 30 s and were allowed to dry in a fume hood for 1 h. Leaf discs dipped in water alone served as controls. Treated leaf discs were placed on agar beds and 10 adult psyllids were transferred to each dish using a camel's-hair brush after a brief anesthetization with CO_2. We observed no effect of CO_2 anesthetization on psyllid mortality as survival was always >95% in controls. Each concentration of each insecticide was replicated three times ($n = 30$ psyllids per concentration). Petri dishes with insects were transferred to a growth chamber (Percival Scientific, Inc., Perry, IA) set at 17 ± 1, 27 ± 1, or 37 ± 1°C and 80 ± 5% RH with a photoperiod of 14:10 (L:D) h. Mortality counts of insects were taken 48 h after transfer into growth chambers, which eliminated the possibility that insects were incorrectly scored as dead due to a fumigation or knockdown effect. Insects found on their side or back that were unable to move when a petri dish was tapped gently were considered dead and included in mortality counts. Each experiment was replicated twice and the replicate runs overlapped temporally for the majority of the testing period. Mortality data were corrected for control mortality (<5%) by using Abbott's formula (Abbott 1925). Mortality data were pooled from both experiments for each concentration and subjected to probit regression analysis to calculate the concentration required for mortality in 50% of the population (LC_{50}) for each insecticide at each exposure temperature, with corresponding confidence limits and slopes of regression lines (SAS Institute 2005). Temperature coefficients for each insecticide were calculated as the ratio of higher to lower LC_{50} values (Sparks et al. 1982, Musser and Shelton 2005, Li et al. 2006). The coefficient was considered positive or negative when the LC_{50} value was higher at a lower temperature or higher at a higher temperature, respectively (Sparks et al. 1982).

Results

Organophosphate Insecticides. The organophosphate insecticides chlorpyrifos and dimethoate exhibited positively correlated toxicity within the temperature range (17–37°C) tested (Table 2). The toxicity of chlorpyrifos increased by 3.76-fold from 17 to 27°C and by 2.80-fold from 27 to 37°C, with an overall positive temperature coefficient of 10.50. Similarly, the toxicity of dimethoate registered an overall positive temperature coefficient over the temperature range tested (Table 2).

Carbamate and Avermectin Insecticides. Similar to that observed with the organophosphate insecticides, the carbamate and avermectin insecticides showed positive temperature correlations (Table 3). A similar increase in the toxicity of both carbaryl and abamectin was observed from 17 to 27°C (Table 3). However, the increase was small for carbaryl, whereas it was large for abamectin from 27 to 37°C (Table 3), with overall temperature coefficients of 4.18 and 17.91 for carbaryl and abamectin, respectively.

Pyrethroid Insecticides. In contrast to the results obtained with the organophosphate, carbamate, and avermectin insecticides, the pyrethroid insecticides showed negative temperature correlation coefficients, with the exception of bifenthrin (Table 4). The temperature–toxicity correlation for bifenthrin was negative, with an increase in temperature from 17 to 27°C (Table 4). However, when the temperature was increased from 27 to 37°C, the toxicity of bifenthrin increased by 2.83-fold, which also resulted in an overall increase in toxicity by 1.16-fold over the entire temperature range tested. A 1.80-fold decrease in toxicity of zeta-cypermethrin occurred over the 17–27°C range, and an additional 5.33-fold decrease occurred over the 27–37°C range. There was a 30-fold decrease

Table 3. Influence of temperature on the toxicity of carbamate and avermectin insecticides against *D. citri* adults

Insecticide^a	Temp. (°C)	LC$_{50}$^{b} (mg [AI]/liter)	95% CL	Slope ± SE	χ^2 (df)^c	P value	Temp. coefficient^d	
							10°C	20°C
Carbaryl	17	49.48	10.11–178.49	0.82 ± 0.13	7.37 (3)	0.060		
	27	18.84	0.67–113.37	0.74 ± 0.16	11.78 (3)^e	0.008	+2.62	
	37	11.82	7.23–18.79	0.80 ± 0.09	2.75 (3)	0.431	+1.59	+4.18
Abamectin	17	8.60	4.77–15.27	0.66 ± 0.07	4.36 (3)	0.224		
	27	3.67	0.47–17.01	0.79 ± 0.14	9.30 (3)^e	0.025	+2.34	
	37	0.48	0.09–2.05	0.71 ± 0.10	6.58 (3)	0.086	+7.64	+17.91

[a] At each insecticide and temperature combination, three replicates of 10 insects were used per experiment.

[b] Concentration required for 50% mortality in *D. citri* adults.

[c] χ^2 goodness-of-fit statistic and degrees of freedom.

[d] Ratio of higher to lower LC$_{50}$ value for 10 and 20°C differences in exposure temperature. A positive coefficient suggests a lower LC$_{50}$ value at the higher temperature.

[e] χ^2 significantly different from expected ($P \leq 0.05$).

in toxicity of fenpropathrin over the 17–27°C range, which was the largest decrease within this temperature range among the pyrethroids tested (Table 4). An overall negative temperature coefficient of 65.00 was recorded for lambda-cyhalothrin, which was the largest overall decrease among the pyrethroids tested (Table 4).

Neonicotinoid Insecticides. In contrast to the results obtained with pyrethroids, the neonicotinoids exhibited mixed temperature correlations with one exception (Table 5). The toxicity of acetamiprid increased by 2.42- and 2.90-fold within the 17–27°C and 27–37°C temperature ranges, respectively, with an overall increase by 7.03-fold. In contrast, the toxicity of imidacloprid and thiamethoxam decreased by 3.44- and 1.45-fold, respectively, over the 17–27°C temperature range. However, the toxicity of imidacloprid and thiamethoxam increased by 3.87- and 4.80-fold, respectively, over the 27–37°C range. Thus, both imidacloprid and thiamethoxam registered positive temperature coefficients over the entire temperature range tested (Table 5).

Discussion

The purpose of this investigation was to determine the effect of posttreatment temperature on toxicity of several insecticides against *D. citri.* Suppressing populations of the psyllid vector is critical for minimizing greening disease transmission, and broad-spectrum insecticides are currently the main option for *D. citri* control. Insecticides are exposed to a wide range of temperatures (10–35°C) in Florida over the season. In the current investigation, the organophosphate insecticides chlorpyrifos and dimethoate exhibited a consistent positive temperature-dependent toxicity correlation, with chlorpyrifos being more toxic than dimethoate at the highest temperature tested. The positive temperature coefficient observed with carbaryl (carbamate) against *D. citri* adults in the current study is in contrast to the results of previous findings with other insect species, which reported a slight negative or no temperature coefficient (Scott 1995). However, methomyl (carbamate) showed a mixed response against first-instar *Ostrinia nubilalis* (Hüb-

Table 4. Influence of temperature on the toxicity of pyrethroid insecticides against *D. citri* adults

Insecticide^a	Temp. (°C)	LC$_{50}$^{b} (mg [AI]/liter)	95% CL	Slope ± SE	χ^2 (df)^c	P value	Temp. coefficient^d	
							10°C	20°C
Bifenthrin	17	0.07	0.05–0.09	1.58 ± 0.17	5.28 (3)	0.152		
	27	0.17	0.10–0.26	1.00 ± 0.09	5.47 (3)	0.140	−2.42	
	37	0.06	0.01–0.14	1.49 ± 0.30	10.66 (3)^e	0.013	+2.83	+1.16
zeta-Cypermethrin	17	0.05	0.03–0.07	1.08 ± 0.12	5.10 (3)	0.164		
	27	0.09	0.06–0.14	0.82 ± 0.11	3.59 (3)	0.308	−1.80	
	37	0.48	0.04–1.78	1.22 ± 0.27	13.13 (3)^e	0.004	−5.33	−9.60
Fenpropathrin	17	0.008	0.006–0.012	1.18 ± 0.13	3.54 (3)	0.314		
	27	0.24	0.20–0.29	2.05 ± 0.20	4.20 (3)	0.239	−30.00	
	37	0.33	0.07–1.00	1.13 ± 0.21	8.87 (3)^e	0.031	−1.29	−38.70
lambda-Cyhalothrin	17	0.002	0.001–0.004	1.01 ± 0.13	2.08 (3)	0.554		
	27	0.01	0.01–0.02	1.69 ± 0.17	0.74 (3)	0.861	−5.00	
	37	0.13	0.10–0.17	1.74 ± 0.18	4.06 (3)	0.255	−13.00	−65.00

[a] At each insecticide and temperature combination, three replicates of 10 insects were used per experiment.

[b] Concentration required for 50% mortality in *D. citri* adults.

[c] χ^2 goodness-of-fit statistic and degrees of freedom.

[d] Ratio of higher to lower LC$_{50}$ value for 10 and 20°C differences in exposure temperature. A positive coefficient suggests a lower LC$_{50}$ value at the higher temperature.

[e] χ^2 significantly different from expected ($P \leq 0.05$).

Table 5. Influence of temperature on the toxicity of neonicotinoid insecticides against *D. citri* adults

Insecticide[a]	Temp. (°C)	LC_{50}[b] (mg [AI]/liter)	95% CL	Slope ± SE	χ^2 (df)[c]	*P* value	Temp. coefficient[d] 10°C	Temp. coefficient[d] 20°C
Acetamiprid	17	3.80	2.66–5.25	1.21 ± 0.15	4.12 (3)	0.248		
	27	1.57	0.99–2.47	0.91 ± 0.11	2.37 (3)	0.498	+2.42	
	37	0.54	0.74–2.13	1.04 ± 0.20	10.80 (3)[e]	0.012	+2.90	+7.03
Imidacloprid	17	0.09	0.04–0.20	1.35 ± 0.21	6.59 (3)	0.086		
	27	0.31	0.23–0.41	1.55 ± 0.18	5.36 (3)	0.147	−3.44	
	37	0.08	0.06–0.10	1.70 ± 0.17	5.01 (3)	0.170	+3.87	+1.12
Thiamethoxam	17	0.33	0.21–0.50	0.95 ± 0.10	5.19 (3)	0.158		
	27	0.48	0.31–0.69	1.12 ± 0.13	6.19 (3)	0.102	−1.45	
	37	0.10	0.08–0.12	2.27 ± 0.25	2.37 (3)	0.497	+4.80	+3.30

[a] At each insecticide and temperature combination, three replicates of 10 insects were used per experiment.

[b] Concentration required for 50% mortality in *D. citri* adults.

[c] χ^2 goodness-of-fit statistic and degrees of freedom.

[d] Ratio of higher to lower LC_{50} value for 10 and 20°C differences in exposure temperature. A positive coefficient suggests a lower LC_{50} value at the higher temperature.

[e] χ^2 significantly different from expected ($P \leq 0.05$).

ner) between 24 and 35°C, with an overall positive temperature coefficient (Musser and Shelton 2005). A possible hypothesis explaining this increased toxicity could be increased penetration of carbaryl into the body of adult *D. citri* at higher temperatures. The susceptibility of *D. citri* adults to carbaryl was lower compared with the other compounds tested. As was recorded for both the organophosphates and carbamates tested here, avermectin also exhibited a positive temperature coefficient over the range tested. To our knowledge, this is the first report that demonstrates temperature-dependent toxicity of an avermectin insecticide.

With the exception of bifenthrin, the remaining pyrethroids tested exhibited negative correlations of toxicity with temperature. These findings of negative correlations are congruent with other studies examining the influence of posttreatment temperature on pyrethroid toxicity (Sparks et al. 1983, Musser and Shelton 2005). However, some studies have shown a positive or mixed response with certain pyrethroids depending on the bioassay technique and insect species tested (Sparks et al. 1982, Li et al. 2006). The toxicity of pyrethroids, particularly fenpropathrin and lambda-cyhalothrin, decreased substantially with increasing temperature, suggesting that these compounds are less efficacious against *D. citri* at higher temperatures. Although several studies have shown decreased toxicity of pyrethroids with increasing temperature, only a few have attempted to elucidate the underlying mechanisms for such activity (Narahashi 1992, Narahashi et al. 1995, Song and Narahashi 1996).

Sodium channels are the major target sites for pyrethroids (Narahashi 1992, Narahashi et al. 1995). Pyrethroids modify the gating kinetics of sodium channels leading to prolonged influx of sodium ions (Na^+), which causes hyperexcitation in insects (Song and Narahashi 1996). Narahashi et al. (1995) and Song and Narahashi (1996) showed that the pyrethroid tetramethrin caused repetitive nerve firing between 15 and 20°C, whereas this activity decreased between 30 and 35°C. Furthermore, three parameters of sodium channels were affected with increasing temperature when rat Purkinje neurons were treated with tetramethrin at 3 μM, which could explain the negative temperature dependence of pyrethroid toxicity (Narahashi et al. 1995, Song and Narahashi 1996). An increase in experimental temperature conditions from 15 to 30°C decreased 1) the percentage of tetramethrin modified sodium channels, 2) the time constant for sodium channel tail current decay, and 3) the ratio of Na^+ inflow during tail current to that during peak current (Narahashi et al. 1995, Song and Narahashi 1996). All of these changes would lead to a decreased excitation of nerves and decreased toxicity. Although these results were obtained from studies on mammalian neurons, they are applicable to insect systems because insects respond to pyrethroids in a similar manner with greater sensitivity given their lower body temperatures (up to 10°C) compared with mammals (Narahashi 1992, Song and Narahashi 1996).

In general, the neonicotinoids showed a positive temperature-dependent toxicity correlation, except that there was a negative correlation for imidacloprid and thiamethoxam between 17 and 27°C. Congruent with our findings, Arthur et al. (2004) reported that toxicity of thiamethoxam at 0.5, 1.0, 2.0, and 4.0 ppm increased with increasing temperature between 22 and 32°C against five stored-product insect species. To our knowledge, this is the only other published report in addition to our current study on the effect of varying temperature on toxicity of a neonicotinoid insecticide. Using neuronal membranes from *Myzus persicae* (Sulzer), Wellmann et al. (2004) showed a decrease of 73 and 28% in the total binding of radiolabeled thiamethoxam and imidacloprid, respectively, at 30°C compared with 2°C. This finding may, in part, explain the negative temperature–toxicity correlations observed in the current study for thiamethoxam and imidacloprid between 17 and 27°C. However, the individual positive correlations observed between 17 and 27°C for acetamiprid and between 27 and 37°C for all three neonicotinoids tested, and an overall positive correlation observed for all three compounds in our study and for thiamethoxam by Arthur et al. (2004) cannot be explained by the results of Wellmann et al. (2004).

Quantifying the effect of temperature on the toxicity of recommended and new insecticides against a target pest is essential for informed selection of an insecticide based on prevailing environmental conditions. In Florida, *D. citri* populations occur throughout the year, with fluctuating populations depending upon the availability of newly developed flush and weather conditions, mainly temperature (Rogers et al. 2008a). During early and late spring, when abundant new flush is available, *D. citri* populations increase dramatically, requiring intensive chemical control. However, adult psyllids also require chemical control during the dormant winter period to prevent large population outbreaks in the spring (Rogers et al. 2008a). Based on our laboratory bioassays, pyrethroids such as zeta-cypermethrin, fenpropathrin, and lambda-cyhalothrin should be most effective for controlling *D. citri* during the cooler winter months in Florida when temperatures are lower (10–25°C). However, during the summer and early fall when the temperatures are higher (25–38°C), organophosphate (chlorpyrifos and dimethoate), carbamate (carbaryl), avermectin (abamectin), or neonicotinoid (acetamiprid) insecticides should provide the most effective control of *D. citri.*

Availability of several insecticides with different modes of action for a given range of temperatures will facilitate rotation to reduce selection pressure on *D. citri* populations. Because imidacloprid and thiamethoxam were most toxic at both extreme temperatures, these compounds can be rotated with pyrethroid insecticides at low field temperatures and with organophosphate, carbamate, or avermectin insecticides at high field temperatures for optimal management of *D. citri* populations as a function of posttreatment temperature.

With the exception of dimethoate, fenpropathrin, and thiamethoxam, the regression line slope values were similar at all of the temperatures tested for a given compound, indicating a homogeneous response of the psyllid population investigated. Based on χ^2 values, only in nine of 33 instances the concentration (log)–mortality (probit) data did not fit regression equations well given that the observed values were significantly different from estimated values ($P \leq 0.05$), resulting in wider 95% confidence intervals.

In summary, toxicity of organophosphate, carbamate, and avermectin insecticides showed a positive correlation with temperature against *D. citri,* whereas pyrethroid insecticides exhibited a negative toxicity correlation. In contrast, neonicotinoids exhibited a mixed temperature-dependent response with an overall positive correlation. The information provided herein will enable citrus growers to choose the most effective insecticide chemistry for optimal toxicity against *D. citri* as a function of seasonal temperature.

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