Influence of Post-Exposure Temperature on the Toxicity of Insecticides to the Tobacco-Adapted Form of the Green Peach Aphid (Hemiptera: Aphididae)[1]

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Abstract Abiotic factors, such as temperature, are important in the activity and performance of insecticidal compounds, as they influence biochemical reactions that may either enhance or limit the insecticide effectiveness. The influence of these temperature-mediated factors on the toxicity of insecticides in red and green color morphs of the tobacco-adapted form of the green peach aphid, *Myzus persicae* (Sulzer), was evaluated using leaf-dip bioassays in laboratory incubators. Postexposure temperatures of 15, 20, and 25°C were evaluated for 4 classes of insecticides: organophosphate (acephate), carbamate (methomyl), pyrethroid (lambda-cyhalothrin), and neonicotinoid (imidacloprid). Except for lambda-cyhalothrin, all the insecticides had positive temperature coefficients that indicated increased toxicity to *M. persicae* at both 5 (15 - 20 and 20 - 25°C) and 10°C (15 - 25°C) temperature ranges. Postexposure temperature had similar effects on insecticide toxicity to both color morphs. A temperature increase of 5°C, from 15 - 20°C and 20 - 25°C, caused 1.3- to 3-fold increases in toxicity for methomyl, acephate, and imidacloprid in both color morphs. A change of 10°C (15 - 25°C) increased the toxicity of the three chemicals from 2.9- to 6.0-fold. In contrast, the toxicity of lambda-cyhalothrin decreased as the temperature increased, showing a negative temperature coefficient. Because laboratory bioassays are typically used for monitoring insecticide resistance, this study confirms that using standardized temperatures is necessary for diagnosing problems and making recommendations for resistance management programs in aphids.

Key Words post-exposure temperature, insecticides, toxicity, *Myzus persicae*, tobacco aphid, green peach aphid

Abiotic factors are important in the activity and performance of insecticidal compounds used in insect pest management programs. Temperature influences the rate of biochemical reactions, the resultant arthropod activity, and may either enhance or limit the effectiveness of an insecticide (Horn 1998). It affects both physical and chemical properties of insecticides such as stability, vaporization, penetration, activity, degradation, uptake, and translocation (Johnson 1990). These temperature-mediated factors play important roles, owing to the complexities that each class of insecticides exhibits.

The green peach aphid, *Myzus persicae* (Sulzer), is a highly polyphagous species with several genetically different host-adapted clones (Van Emden et al. 1969, Weber

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1985, 1986, Edwards 2001). It colonizes over 500 species of secondary host plants from at least 40 different families (Blackman and Eastop 2000). The tobacco-adapted form of the green peach aphid, *M. persicae*, is an important pest of tobacco. High populations of the aphid can reduce tobacco yield by 5 - 25% depending on the growing conditions and the level of control (Reed and Semtner 1992). Honeydew produced by the aphid accumulates on the tobacco leaves and a dark, sooty mold often grows on the honeydew (Mistric and Clark 1979). The combination of aphid feeding damage, sooty mold, and honeydew interferes with curing, reduces leaf quality, and often remains on tobacco even after aphids are controlled.

Before 1985, a green morph of the aphid was the only form reported on tobacco in the southeastern U. S. (Blackman 1987). A red morph of the aphid first occurred on tobacco in 1985 and quickly replaced the green morph as the most common form (Blackman 1987, Harlow et al. 1991, Lampert and Dennis 1987). Widespread insecticide resistance to traditional organophosphates (OPs) and carbamates occurred in the late 1980s (Blackman 1987, Harlow and Lampert 1990, McPherson and Bass 1990). The association between the red form and resistance to OP insecticides is well established for the aphid in the U. S. (Harlow and Lampert 1990, Clements et al. 2000). Aphid control depends extensively on insecticide applications and, in Virginia, control expenditures often exceed $500,000 annually (Semtner, pers. observ.). Presently, there are 4 major classes of insecticides (organophosphate, carbamate, pyrethroid, and neonicotinoid) registered for aphid control on tobacco.

Organophosphate and carbamate insecticides generally have stable toxicities at all temperatures, but some studies have found slight positive or negative temperature coefficients to several insect species (McLeod 1991, Musser and Shelton 2005). Pyrethroid insecticides often have reduced efficacy at high temperatures (Horn 1998, Musser and Shelton 2005), although they have a positive temperature coefficient against some species (Valles et al. 1998). Imidacloprid, a neonicotinoid class of insecticide is used extensively to control aphids, but the effect of temperature on the toxicity of this group of compounds is not well studied. To express the difference in the toxicity of insecticides with variation in temperature, temperature coefficients are often used (Gordon 2005). Temperature coefficient is the change in rate or activity of a process for every 1°C change in temperature (IUPS Thermal Commission 2001).

Limited information is available on the influence of temperature on the toxicity of insecticides to the aphid (or *M. persicae* complex) even though insecticides play a major role for its control in many important field and vegetable crops. As temperature-sensitivity varies among insecticide classes and is sometimes pest and product-specific, there is a need for more information to allow those responsible for making pest management decisions to select the best product for the existing environmental conditions. Because insecticide resistance monitoring is primarily evaluated in laboratory bioassays, the effect of temperature on the toxicity of insecticides would play a critical role and should be considered in resistance management programs. As insecticides from the same class often possess similar temperature responses, this study examines the effects of posttreatment temperature on the efficacy of 4 types of insecticides against the aphid.

Materials and Methods

Red and green morphs of green peach aphid were collected by removing infested leaves from untreated tobacco plants grown in the field at the Virginia Tech Southern Piedmont AREC, Blackstone, VA, on 11 July 2007. The infested leaves were kept on

moistened paper towels in ventilated containers for 2 d to allow the colonies to adapt to laboratory conditions. This also insured that the colonies were free of disease or parasitism. Healthy adults and last-instar nymphs were then exposed to pesticides as described below.

Four commercial formulations of insecticides were evaluated: acephate (Orthene 97®, Valent USA), an organophosphate; lambda-cyhalothrin (Warrior®, Syngenta, Greensboro, NC, a pyrethroid; methomyl (Lannate LV®, DuPont, Wilmington, DE), a carbamate; and imidacloprid (Admire 2F®, Bayer CropScience, KS City, MO), a neonicotinoid. Aphids were bioassayed by the leaf-dip method using water dispersions of each formulated insecticide. Leaf disks, 100 mm in diameter, were cut from fresh leaves (midstalk position) from greenhouse-grown tobacco plants (flue-cured cultivar, 'K-326'). Leaf disks were dipped for 5 sec in the designated concentrations, air-dried, and placed on slightly moistened filter papers in labeled Petri dishes (15 X 100 mm). The inside rims of the Petri dishes were coated with Fluon® to keep the aphids on the treated leaves. A camel's hair brush was used to place aphids on each leaf disk. Covers were placed on each Petri dish and secured with Parafilm® (Pichiney Plastic Packaging, Chicago, IL).

Six concentrations along with a deionized water control for each of the 4 insecticide classes were tested to cover the range of expected partial mortality for 2 color morphs of the aphid at 21°C based on preliminary tests. Each concentration was replicated 5 times with 10 aphids per Petri dish for 3 different temperatures (15, 20 and 25°C) tested. A total of 8,400 aphids was used for the study. Mortality of the test populations exposed to imidacloprid was assessed 72 h after treatment to overcome the antifeedant effect (Nauen et al. 1998), and 24 h for the other insecticides. The aphids were touched (or brushed) lightly with the camel's-hair brush. If an aphid did not move or only twitched slightly, it was considered dead. All bioassays were conducted in environmental chambers maintained at 15, 20, and 25°C, 60% RH, and 16:8 (L:D) h photophase. A separate growth chamber was used for each of the 3 temperatures. To insure that temperature effects were independent of the growth chamber used, preliminary assays were conducted in each of the 3 chambers with varying temperatures using aphids on untreated leaf disks.

Abbott's formula (Abbott 1925) was used to correct for control mortality. LC_{50} values were calculated by probit analysis (POLO PC, LeOra Software, Berkeley, CA). Temperature coefficients were calculated, and the likelihood ratios, hypothesis of equality and parallelism for slopes and intercepts of the the dose response curves were tested at $P < 0.05$.

Results

Methomyl, acephate and imidacloprid exhibited positive temperature coefficients for *M. persicae* at both the 5 and 10°C temperature intervals (Table 1). Methomyl, with a temperature coefficient of +5.7 for red morphs and +4.7 for green morphs required 5.7 and 4.7 times less methomyl at 25°C than was needed to achieve equal control at 15°C. The temperature change from 15 - 20°C resulted in almost a 3-fold increase in toxicity for methomyl in both color morphs (Table 1). The difference in the toxicity over the temperature range was tested for the hypothesis of parallelism and equality (slopes and intercepts) was not significant ($P > 0.05$) between the color morphs or for the 3 temperatures. These results show that toxicity of methomyl was impacted more by temperature than by color morph of the aphid, and responses to temperature were consistent for both morphs.

Table 1. Effect of postexposure temperature on the toxicity of methomyl, acephate, imidacloprid, and λ-cyhalothrin insecticide toxicity for red and green morphs of the tobacco-adapted form of the green peach aphid, *Myzus persicae*.

Insecticide	Color Morph	Temperature (°C)	N*	LC_{50} (95% CF) ppm**	Slope ± SE	H†	Temperature Coefficient‡ 5°C	Temperature Coefficient‡ 10°C
Methomyl	Red	15	350	203.9 (155.3 - 269.4) a	1.49 ± 0.14	0.50		
		20	350	68.3 (53.1 - 86.7) b	1.98 ± 0.19	0.64	+ 3.0	+ 5.7
		25	350	35.5 (28.4 - 43.9) c	2.33 ± 0.25	0.92	+ 1.9	
	Green	15	350	163.1 (123.2 - 216.4) a	1.43 ± 0.14	0.83		
		20	350	62.4 (48.7 - 79.3) b	2.16 ± 0.21	1.08	+ 2.6	+ 4.7
		25	350	34.6 (26.6 - 44.1) c	1.89 ± 0.21	0.76	+ 1.8	
Acephate	Red	15	350	364.2 (272.7 - 497.1) a	1.41 ± 0.13	0.53		
		20	350	226.8 (171.2 - 303.8) a	1.61 ± 0.14	1.05	+ 1.6	+ 4.8
		25	350	76.1 (59.8 - 97.5) b	1.82 ± 0.18	0.44	+ 3.0	
	Green	15	350	216.6 (159.2 - 299.1) a	1.25 ± 0.12	0.36		
		20	350	97.3 (74.7 - 127.2) b	1.59 ± 0.15	0.53	+ 2.2	+ 2.9
		25	350	73.8 (56.2 - 96.4) b	1.56 ± 0.15	0.52	+ 1.3	
λ-cyhalothrin	Red	15	350	61.1 (38.8 - 89.9) a	0.95 ± 0.11	0.44		
		20	350	181.1 (127.1 - 261.3) b	1.13 ± 0.11	1.04	- 3.0	- 4.5
		25	350	275.7 (192.5 - 409.5) b	1.02 ± 0.11	0.50	- 1.5	
	Green	15	350	87.2 (49.1 - 143.1) a	0.69 ± 0.10	0.29		

Table 1. Continued

Insecticide	Color Morph	Temperature (°C)	N*	LC_{50} (95% CF) ppm**	Slope ± SE	H†	Temperature Coefficient‡ 5°C	Temperature Coefficient‡ 10°C
		20	350	135.9 (92.8 - 197.8) b	0.98 ± 0.11	0.70	- 1.6	- 3.2
		25	350	276.0 (192.3 - 411.2) b	1.01 ± 0.11	0.64	- 2.0	
Imidacloprid	Red	15	350	6.4 (4.6 - 9.0) a	1.10 ± 0.14	0.18		
		20	350	3.6 (2.3 - 4.9) a	1.54 ± 0.27	0.83	+ 1.8	+ 3.4
		25	350	1.9 (0.4 - 2.7) b	0.64 ± 0.13	0.14	+ 1.9	
	Green	15	350	3.6 (2.4 - 5.2) a	0.96 ± 0.13	0.30		
		20	350	1.2 (0.8 - 1.7) b	1.48 ± 0.18	1.01	+ 3.0	+ 6.0
		25	350	0.6 (0.3 - 0.8) b	1.57 ± 0.21	0.40	+ 2.0	

* Total number of *Myzus persicae* apterae tested for each temperature/color morph/insecticide treatment.

** LC_{50} values followed by the same letter are not significantly different within each insecticide treatment ($P < 0.05$).

† Heterogeneity factor = Observed chi-square value / Degrees of freedom.

‡ Ratio of LC_{50} value at 5 - 10°C differences in temperature. A negative coefficient indicates a higher LC_{50} at the higher temperature. LC_{50} values followed by the same letter are not significantly different within each insecticide treatment ($P < 0.05$).

A similar trend was recorded with acephate, where temperature coefficients of +4.8 for red morphs and +2.9 for green morphs over the 10°C (15 - 25°C) interval were observed (Table 1). A difference of over 10°C (15 - 25°C) were significant to give differential mortality in both color morphs, as indicated by nonoverlapping 95% confidence intervals. The dose response curves subjected to the test for parallelism revealed that the slopes are parallel ($P > 0.05$) with different intercepts ($P < 0.05$).

Imidacloprid also demonstrated a steady increase in toxicity with the increase in post exposure temperature (Table 1). The toxicity is seen as temperature coefficients at the 10°C interval of +3.4 for red morphs and +6.0-fold for green morphs. At 15°C, both red and green morphs had significantly higher LC_{50} values than they did at 20 and 25°C, respectively (likelihood ratio, $P < 0.05$). The 95% confidence interval overlap of the toxicity values at 20°C between the color morphs is seen as the difference in their likelihood ratios (hypothesis of parallelism not rejected at $P > 0.05$). The variations in toxicities were not different at both 20 or 25°C for either color morph.

In contrast to the other insecticides, the toxicity of the pyrethroid, lambda-cyhalothrin, decreased with increased temperature (Table 1). The difference in the toxicity over the 10°C interval was -4.5 for the red morphs and -3.2 for the green morphs. The trend in toxicity remained the same at each temperature for both color morphs and was not statistically significant ($P > 0.05$). Differences between color morphs were significant at 15°C, but not at the two higher temperatures ($P > 0.05$, 95% confidence interval and likelihood ratio test).

Discussion

The biochemical reactions leading to the toxicity of many insecticide molecules usually proceed more rapidly at higher temperatures. Some insecticides, like the pyrethroids, often have negative temperature coefficients and are usually more toxic at lower temperatures (Wadleigh et al. 1991, Satpute et al. 2007).

Results in this study indicate that temperature influences the efficacy of the 4 insecticides tested against the aphid. As insecticide resistance monitoring in aphids is usually conducted at 21 ± 1°C in laboratory incubators, the changes in the temperature over a range of 5 - 10°C could result in drastic changes in the toxicity of an insecticide.

Methomyl had a positive temperature coefficient regardless of the color morph. The temperature change from 15 - 20°C resulted in almost a 3-fold increase in toxicity for methomyl in both color morphs. Similar trends were observed for acephate. Differential toxicity was evident with the nonoverlapping confidence intervals at 15 and 25°C within and between color morphs. Interestingly, for both methomyl and acephate the differences in toxicity between the color morphs disappeared at 25°C. As the efficacy of any insecticide molecule is a function of the interrelationship between vaporization and cuticular penetration, the insecticide formulation plays an important role in determining the actual toxicity of the molecule and not the carrier.

Despite intensive studies between temperature and organophosphorous toxicity to arthropods, little is known about the mechanism responsible for this relationship. Studies have shown that altered distribution within the insect body, target site interactions, penetration, and metabolism of OPs and carbamates differ considerably within insect species (Scott 1995). A positive temperature coefficient, often characteristic of organophosphates and carbamates could be due mainly to higher reactivity (phosphorylation and carbamylation) of the target enzyme (AChE) at higher temperatures

(Aldridge 1971). In the present findings, organophosphate, carbamate, and neonicotinoid insecticides were more effective at higher temperatures, showing positive correlations of temperature with toxicity. Pawinska (1994) reported that organophosphorous compounds had increased activity from 20 - 30°C, which also corroborates the findings in our study. Morytz et al. (1997) also reported increased activity of an organophosphorous compound (chlorpyriphos) between 15 and 35°C against both *Musca domestica* L. and *Leptinotarsa decemlineata* (Say).

Esterase-based resistance is the primary factor conferring resistance to organophosphate and carbamate insecticides in *M. persicae* (Devonshire and Moores 1982, Field et al. 1994). General esterase activity in aphids used in this study was measured according to the method of Van Asperen (1962) as modified by Zhu and Gao (1999) using α-naphthyl acetate as substrate. Both red and green morphs had similar general esterase activity (62 and 54 nmol/min/mg protein, respectively). This further strengthens the fact that the difference in the toxicity seen in this study is mainly due to temperature.

Only a few studies have reported that temperature affects the toxicity of imidacloprid. As the neonicotinoids are among the most heavily used insecticides, any information regarding the activity of these newer compounds may play a critical role in insecticide resistance monitoring. Richman et al. (1999) demonstrated that the toxicity of imidacloprid to cat fleas, *Ctenocephalides felis* (Bouche), was higher at 35°C compared with 20°C. In the current study, imidacloprid toxicity increased steadily with increasing postexposure temperature. At 15°C, both red and green morphs had significantly higher LC_{50} values than the toxicities at 20 and 25°C.

The neurotoxic action of pyrethroids was extensively reviewed by Soderlund and Bloomquist (1989). In general, pyrethroids have negative temperature coefficients due to increased effectiveness at the target site at lower temperatures. Both natural pyrethrins and synthetic pyrethroids have higher toxicity at lower temperatures (Hartzell and Wilcoxon 1932, Harries et al. 1945, Blum and Kearns 1956, Hirano 1979). Electrophysiological studies indicated that pyrethroids have increased activity in the form of repetitive discharges (Gammon 1978, Starkus and Narahashi 1978), increased negative after potential (Narahashi 1962), and conduction block of the central nervous system (Wang et al. 1972). Pyrethroid-induced membrane depolarization (Bloomquist 1993) and sodium currents (Vijverberg et al. 1983) are greatly prolonged with decreasing temperature and depend on several kinetic parameters of the voltage dependent sodium channels that change with temperature. Lambda-cyhalothrin belongs to the type II pyrethroid class that prolongs sodium currents for up to several minutes, which presumably increase as temperature decreases. Recently, the higher toxicity of pyrethroids at lower temperatures on *Chironomus dilutus* Shobanov, Kiknadze & Butler, 1999, was confirmed to be due to a combination of increased accumulation of parent compound and increased nerve sensitivity, exacerbating the toxicity of pyrethroids at 13°C compared with 23°C (Harwood et al. 2008). The increase in toxicity with decrease in temperature in our study is consistent with most studies that have examined the impact of temperature on efficacy of pyrethroids on various species of insects (Scott 1995).

As temperature may positively interact with the expression of insecticide resistance, its effects should be considered in resistance management. In earlier studies, Zareh and Morse (1989) have shown that tolerance of citrus thrips, *Scirtothrips citri* (Moulton), to acephate and fluvalinate decreased as temperatures increased from 16 - 32°C. A field population of *Drosophila melanogaster* Meigen, 1930 selected for cyclodiene resistance displayed a temperature-related paralysis at high temperature (38°C) in

the laboratory, and such an effect could reduce fitness at high temperatures (ffrench-Constant et al. 1993). Brown (1987) noted that a pyrethroid-tolerant strain of *Heliothis virescens* (F.) displayed resistance to fenvalerate, fluocythrinate, and permethrin at 26°C but not at 16°C, indicating that resistance in the field could go unnoticed at low temperatures. Foster et al. (1996) reported apparent loss of insecticide resistance in the green peach aphid, *M. persicae*, after exposure to low temperature and theorized that selection for resistance could be negated by cold winter weather. The biochemical mechanisms responsible for these effects could be due to metabolic resistance, though still need to be thoroughly investigated.

Regardless of other impacts, when insecticides from different classes are available to control a pest, knowledge of a product's temperature coefficient will enable pest managers to select a product that is efficacious under given environmental conditions. Monitoring insecticide resistance with laboratory bioassay methods could be affected by variations in the abiotic factors, especially temperature. This study shows that changes in temperature greatly influence insecticide toxicity to the aphid, but the mechanisms responsible for these effects need to be pursued further.

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