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Activity of Broad-Spectrum and Reduced-Risk Insecticides on Various Life Stages of Cranberry Fruitworm (Lepidoptera: Pyralidae) in Highbush Blueberry

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ABSTRACT Laboratory and semifield bioassays were conducted to determine the life-stage activity of insecticides for controlling cranberry fruitworm, *Acrobasis vaccinii* Riley (Lepidoptera: Pyralidae), a key lepidopteran pest of highbush blueberry, *Vaccinium corymbosum* L. The organophosphates azinphosmethyl and phosmet, the pyrethroid esfenvalerate, and the carbamate methomyl were lethal to all life stages. The neonicotinoids thiacloprid and acetamiprid demonstrated strong larvicidal and ovicidal activity but were somewhat weaker adulticides than the conventional broad-spectrum compounds. *Bacillus thuringiensis*, indoxacarb, and emamectin benzoate were shown to control *A. vacinii* primarily through their larvicidal activity. Spinosad was toxic to all life stages, including eggs laid on top of residues and those that were treated topically, but larvicidal activity was short lived. The growth regulators pyriproxyfen and novaluron had strong ovicidal activity when eggs were laid on top of residues but had limited larvicidal activity. Tebufenozide was not directly toxic to eggs, but demonstrated larvicidal activity, and ovilarvicidal activity when topically applied to eggs. Azinphosmethyl, phosmet, indoxacarb, thiacloprid, and acetamiprid were all toxic to the egg parasitoid *Trichogramma minutum* Riley. In contrast pyriproxyfen, emamectin benzoate, methomyl, novaluron, and spinosad did not negatively affect the survival of *T. minutum* within *Acrobasis vacinii* eggs. These results help inform the ongoing development of integrated strategies for insect management in blueberry.

KEY WORDS *Acrobasis vacinii*, reduced-risk insecticide, life-stage activity, parasitoid

In eastern North America, highbush blueberries, *Vaccinium corymbosum* L., are at risk of infestation by larvae of cranberry fruitworm, *Acrobasis vaccinii* Riley (Lepidoptera: Pyralidae) (Murray et al. 1986, Dorschner et al. 2009). This insect is the primary lepidopteran pest of blueberries in this region because larvae infest the fruit directly and can web multiple berries together, leaving visible masses of frass in the blueberry cluster at harvest time (Hutchinson 1954). If not controlled, high populations of this insect can result in reduced blueberry yield and detection of cranberry fruitworm larvae in the harvested crop can lead to rejection of entire loads of blueberries by buyers.

A. vaccinii has one generation per year, with moths emerging in May when blueberry bushes are in bloom. Oviposition begins at early fruit set, with female moths laying eggs directly into the calyx cup of berries that are exposed after petals fall from the bushes. The neonate larvae crawl across the fruit surface and enter the berries at the stem end, feeding on a small area of the fruit before penetrating the berry (Hutchinson 1954, Tomlinson 1970). The protected site for egg laying, short distance of travel by neonates before penetrating the fruit, and egg hatch during the late bloom period can make control of *A. vaccinii* challenging.

To minimize the risk of infestation and crop loss, application of bee-safe insecticides may be made during bloom, followed by broad-spectrum insecticides applied after bloom (Wise et al. 2009). A commonly used insecticide for control of *A. vaccinii* in blueberries, azinphosmethyl, is being phased out of blueberry production with registration to end in 2012 (USEPA 2006). Azinphosmethyl was applied to 72.9% of Michigan blueberry acreage during 2007 (R.I., unpublished data). That 81% of those azinphosmethyl applications were made for fruitworm control reflects the current dependency of growers on this compound for fruitworm control. As this product becomes increasingly restricted by Environmental Protection Agency in preparation for complete phaseout in 2012, U.S. blueberry producers will need to use alternatives to maintain fruit protection from this pest.

Alternatives to azinphosmethyl that are registered for use in blueberries include esfenvalerate, phosmet, methomyl, spinosad, pyriproxifen, tebufenozide, acetamiprid, indoxacarb, and *Bacillus thuringiensis* (Dorschner et al. 2009). There are also some promising new insecticides under development for potential

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Table 1. Formulated compounds, field rates and concentrations used for life-stage activity experiments

Formulated name	Chemical class	Active ingredient	Rate/acre	g (AI)/ha	ppm	Company
Asana 0.66EC	Pyrethroid	Esfenvalerate	8 fl oz	46	98	DuPont Crop Protection Wilmington, DE
Assail 70WP	Neonicotinoid	Acetamiprid	2.3 oz	112	241	Cerexagri-Nisso LCC. King of Prussia, PA
Avaunt 30WG	Oxadiazine	Indoxacarb	6 oz	126	269	DuPont Crop Protection
Calypso 4F	Neonicotinoid	Thiacloprid	3 fl oz	105	224	Bayer CropScience Triangle Park, NC
Confirm 2F	Ecdysone agonist Diacylhydrazine	Tebufenozide	16 oz	280	559	Dow AgroSciences LLC, Indianapolis, IN
Diamond 0.83EC	Benzoylphenyl urea	Novaluron	20 fl oz	145	310	Makhteshim Agan of North America, Inc., NY
Dipel 25WG	Biological	*Bacillus thuringiensis* var. kurstaki	1.5 lbs	420	898	Valent U.S.A. Corporation Walnut Creek, CA
Proclaim 5SG	Avermectin	Emamectin benzoate	4.8 oz	17	36	Syngenta Crop Protection, Greensboro, NC
Esteem _ 35WP	Juvenile Hormone Mimic	Pyriproxyfen	5 oz	122	262	Valent U.S.A. Corporation
Guthion_ 50WP	Organophosphate	Azinphosmethyl	1 lb	560	1,198	Bayer CropScience
Imidan 70W	Organophosphate	Phosmet	1.3 lb	1019	2,180	Gowan Company Yuma, AZ
Lannate 90SP	Carbamate	Methomyl	1 lb	1008	2,156	DuPont Crop Protection
SpinTor 2SC	Spinosyn	Spinosad	6 oz	105	224	Dow AgroSciences LLC

All preparations were based on 468 liters/ha (50 gal/acre) spray volume.

future use against this pest, including novaluron, thiacloprid, and emamectin benzoate (Wise et al. 2004, 2006b). Many of these insecticides have selective activity on particular life stages of Lepidoptera (Knight 2000, Charmillot et al. 2001, Brunner et al. 2005, Isaacs et al. 2005), and thus require specific application timing to provide optimal efficacy. Adjusting the growing degree-day–based application timing of selective compounds, such as tebufenozide, has been shown to improve fruit protection against *A. vaccinii* in blueberries, presumably because of enhanced ovicidal or larvicidal activity of the compound (Isaacs et al. 2009). This suggests that understanding the life-stage activity of new insecticides will be important for optimizing the application timing for control of *A. vaccinii* in blueberry integrated pest management (IPM) programs. Life-stage activity and duration of effective residues are key contributors to the overall performance of a compound and the resulting protection of the blueberry crop.

Modification of *A. vaccinii* pest management programs also may have side effects on biological control agents. In Michigan blueberry farms, eggs of this pest are attacked by *Trichogramma minutum* Riley, a generalist lepidopteran endoparasitoid that prevents larval development and eclosion of *A. vaccinii* (Simser 1995, Murray et al. 1986, Pinto 1998). Insecticides vary widely in their toxicity to *Trichogramma* parasitoids of fruit pests (Suh et al. 2000, Brunner et al. 2001), and information on the degree of toxicity to these biological control agents can assist in the selection of effective insecticides that minimize nontarget impacts on beneficial insects.

The objectives of this study were to determine: 1) the toxicity of insecticides to adult *A. vaccinii*, 2) the relative toxicity to eggs of pre- and postoviposition applications of insecticides, 3) the duration of larvicidal activity under field-aged conditions for different insecticides, and 4) the side effects of insecticides on the *T. minutum* egg parasitoid.

Materials and Methods

Materials Tested. Formulated insecticides were prepared at concentrations equivalent to labeled field application rates (468 liters/ha, 50 gal/acre carrier volume) (Table 1). The surfactant Latron B-1956 (Loveland Industries, Inc., Greeley CO) was added to all treatments at 0.125 ml/liter spray volume. Experiments were conducted at the Michigan State University (MSU) Trevor Nichols Research Complex in Fennville, MI, or other nonsprayed blueberry fields in the vicinity. 'Jersey' highbush blueberries containing populations of *A. vaccinii* were used for semifield trials or laboratory bioassays. Because of limitations in the numbers of *A. vacinii* that could be collected and difficulty in holding various life stages in the laboratory, not all insecticides were able to be included in each type of life-stage experiment. Treatment compound selections were prioritized based on known mode of action, representation of chemical classes, and importance of demonstration for fruitworm management in blueberries.

Adult Toxicity. Blueberry clusters infested with *A. vacinii* larvae were collected in mid-June 2004 from nonsprayed blueberry fields returned to the laboratory. Infested fruit clusters were held under ambient conditions in plastic boxes with sand in the bottom until larvae emerged and formed hibernaculae. After they entered diapause, they were held at 4°C and no light for 50 d. Hibernaculae were then removed and held in an environmental chamber at 25°C and a photoperiod of 16:8 (LD) h until adults emerged.

Adult bioassays were performed on 10 and 20 December 2004, with six and five replicates, respectively. Bioassay chambers for insecticide studies consisted of

an 8.9-cm polystyrene petri dish, with a 3.8- by 3.8-cm hole in the top; the top had a piece of fine mesh glued in place to allow gas exchange. The interior of both the top and bottom of each petri dish was treated with 2 ml of insecticide solution at 20 psi by using a Potter Spraytower and allowed to dry before adding moths to the chambers. To maintain consistent coverage of bioassay chambers, each petri dish was individually treated. Two moths were added to each replicate chamber and the mortality and health condition of moths were evaluated after 24-h exposure. Moths observed to have abnormal movement, such as twitching or unable to remain in an upright position, were considered unhealthy. Tebufenozide, pyriproxyfen, emamectin benzoate, indoxacarb, novaluron, and *B. thuringiensis* variety *kurstaki* were not included in the adult toxicity experiments because of the limited availability of *A. vaccinii* adults, and the expectation of no or minimal direct lethal effects on *A. vaccinii* adults.

Egg Toxicity. *Preoviposition Treatment.* On 2 June 2004, blueberry branches with immature fruit were flagged and inspected, with all berries containing insect eggs being removed. These branches were then sprayed with treatment concentrations equal to labeled field rates using a backpack sprayer (Table 1). The following day, the flagged branches were reinspected. All fruit containing *A. vacinii* eggs were removed. These fruit were held peduncle down in moist floral foam (Vans Floral Products, Indianapolis, IN) in an upright position to maintain fruit integrity. Separate containers were used for the separate treatments. Egg eclosion and larval entry into the fruit were measured after 7 d. Due to the random egg laying nature of *A. vacinii* within treatment plots, the number of replications (i.e., egg infested fruit), per treatment ranged from five to 12 eggs. Azinphosmethyl, phosmet, methomyl, esfenvalerate, thiacloprid, acetamiprid, and *B. thuringiensis* variety *kurstaki* were not included in this experiment because the intent was to focus on selective ovicidal insecticides with the limited available *A. vaccinii* eggs.

Postoviposition Treatment. On 11 June 2003 and 11 June 2004, berries containing freshly laid *A. vacinii* eggs were collected from nonsprayed blueberry fields near Covert, MI, and Holland, MI, respectively. Ten of these blueberries were placed in each petri dish that had been lined with floral foam (Vans Floral Products) such that berries could be orientated with the eggs in an upright position. One petri dish was set up for each treatment. These bioassay chambers were then treated with a 2-ml solution of each insecticide at 20 psi atmospheric air delivered by the Potter Spraytower (Table 1). Egg hatch and larval entry into fruit were evaluated after 7 d.

Larval Toxicity. Foliar applications of insecticides were applied to mature blueberry bushes on 18 June 2002 and on 27 June 2003 at a rate of 468 liters/ha (50 gal/acre) with an airblast sprayer (model 1029, FMC Corp., Jonesboro, AR). Treatment plots were arranged in a completely randomized design of three bush plots. The bioassay was conducted in the laboratory by using shoots with fruit clusters clipped from treatment plots at 4 h, 7 d, and 14 d after field application. For uniformity, fruit clusters were thinned to eight treated berries per cluster. Ten replications per treatment of treated clusters were placed in water soaked floral foam to maintain integrity of the fruit. Blueberry clusters with *A. vacinii* eggs were collected from a nonsprayed blueberry field near Holland, MI. Using a razor blade, one *A. vacinii* egg and a sliver of fruit were shaved off and placed in the calyx of one berry in each of the treated clusters. In both 2002 and 2003, each treatment included ten sprayed fruit clusters, and an *A. vacinii* egg placed on one of the eight berries in the cluster. Larval mortality and the number of berries entered were measured after 14 d of exposure. Emamectin benzoate, phosmet, novaluron, acetamiprid, and methomyl were not included in the larval toxicity experiments because of the limited available *A. vaccinii* larvae.

Parasitism Assay. Berries containing *A. vaccinii* eggs were collected from a nonsprayed blueberry field near Holland, MI, on 18 June 2003 and 18 June 2004. Parasitized eggs, those becoming black in color due to the presence of a maturing *T. minutum* parasitoid, were separated for the assay. Twelve blueberries with parasitized eggs were placed in each petri dish that had been lined with floral foam such that the parasitized eggs were in an upright position. One petri dish was set up for each treatment. These petri dish bioassay chambers were then sprayed with the appropriate treatment using a Potter Spraytower as described in the egg toxicity bioassays (Table 1). Treated berries with parasitized eggs were then placed in plastic sealable bags, and the number of parasitoids that emerged was measured after being held for 15 d (3 July 2003 and 4July 2004, respectively). Tebufenozide and *B. thuringiensis* variety *kurstaki* were not included in the parasitism assay because of the limited available parasitized eggs, and because of their anticipated safety to beneficial organisms.

Data Analysis. For each assay described above, data were compared across treatments by using analysis of variance (ANOVA) (PROC MIXED, SAS Institute 2003). Data were analyzed for normality using the Shapiro–Wilk test and for equal variance across treatments by using Levene's test. Adult toxicity, egg toxicity, and larval toxicity data were log $(n + 1)$ transformed before analysis to meet normality assumptions. Parasitism assay data met normality assumptions and no transformation was necessary. Tukey's test was used for multiple comparisons, and the significance level was $\alpha = 0.05$.

Results

Adult Toxicity. Each of the insecticides, except acetamiprid, reduced the survival of *A vaccinii* adults compared with the untreated check ($F = 11.61$; df = 7, 77; $P < 0.0001$) (Fig. 1A). Only azinphosmethyl and methomyl treatments resulted in no live adults. Each of the insecticides reduced the incidence of healthy *A. vacinii* adults compared with the untreated check ($F = 13.26$; df = 7, 77; $P < 0.0001$) (Fig. 1B).

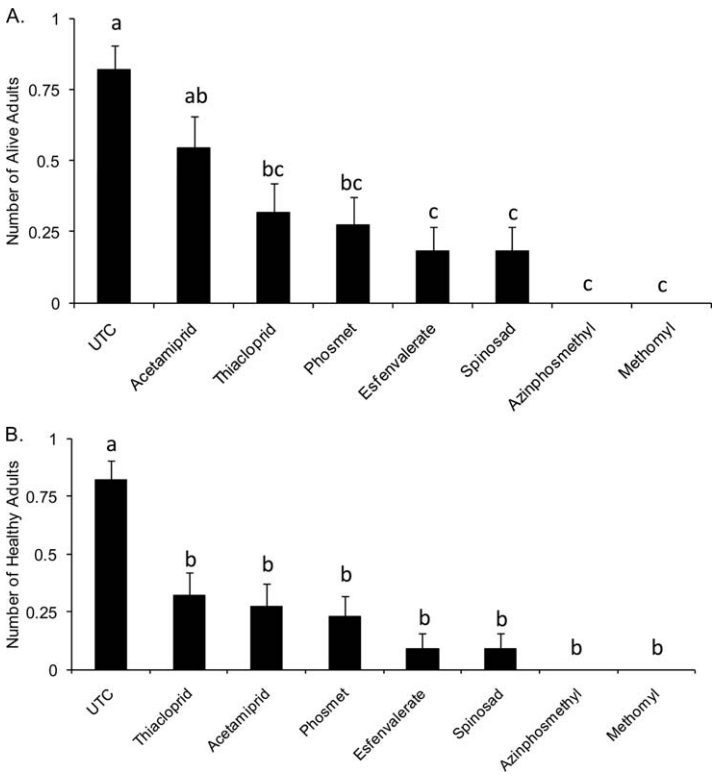

Fig. 1. Mean number ± SE of alive (A) or healthy (B) adult *A. vaccinii* after exposure to residues of different insecticides. Bars with the same letter are not significantly different at $P < 0.05$.

Egg Toxicity. *Preoviposition Treatment.* Spinosad, novaluron, and pyriproxyfen treatments significantly reduced egg survival, whereas eggs laid onto tebufenozide residues were not different from the untreated check ($F = 11.64$; df = 4, 25; $P < 0.0001$) (Fig. 2A). However, there was no significant difference in the number of entry holes in fruit among treatments ($F = 3.36$; df = 3, 8; $P = 0.076$) (Fig. 2B).

Postoviposition Treatment. Only azinphosmethyl, phosmet, methomyl, esfenvalerate, thiacloprid, acetamiprid, and spinosad treatments reduced survival of eggs compared with the untreated check ($F = 7.76$; df = 13, 88; $P < 0.0001$) (Fig. 3A). Among the treatments that did not directly affect egg survival, tebufenozide, emamectin benzoate, and indoxacarb significantly reduced the incidence of larval entries into berries ($F = 4.59$; df = 7, 24; $P = 0.0022$) (Fig. 3B). This indicates that although these compounds do not have true ovicidal activity, ovilarvicidal activity can be expected from these insecticides with spray timings targeting the *A. vacinii* egg-laying period.

Larval Toxicity. Azinphosmethyl, *B. thuringiensis* variety *kurstaki*, pyriproxyfen, esfenvelerate, indoxacarb, thiacloprid, tebufenozide, and spinosad all significantly reduced *A. vaccinii* larval survival, when exposed to field-sprayed fruit clusters 4-h postapplication ($F = 8.42$; df = 8, 72; $P < 0.0001$) (Fig. 4A). In addition, all treatments except for *B. thuringiensis* variety *kurstaki* significantly reduced the number of berries that larvae entered, compared with the untreated check ($F = 7.29$; df = 8, 72; $P < 0.0001$) (Fig. 4B). This suggests that even when the residues are fresh, *B. thuringiensis* may not prevent multiberry infestation before its lethal action is complete.

When larvae were exposed to 7-d-old field-aged residues, only azinphosmethyl, *B. thuringiensis* variety *kurstaki*, esfenvelerate, thiacloprid, and tebufenozide significantly reduced larval survival ($F = 4.50$; df = 8, 72; $P = 0.0002$) (Fig. 4A). Even though these compounds were lethal to larvae, only thiacloprid significantly reduced the number of berries that larvae entered, compared with the untreated check ($F = 7.29$; df = 8, 72; $P < 0.0001$) (Fig. 4B). This indicates that after 7 d of field aging the contact activity of azinphosmethyl and esfenvelerate have diminished, and ingestion is needed to cause mortality. Similarly for *B. thuringiensis* variety *kurstaki* and tebufenozide, additional exposure time is needed to induce larval mortality under the aged residue conditions.

When larvae were exposed to 14-d-old field-aged residues, only spinosad, indoxacarb, esfenvelerate, thiacloprid, and tebufenozide significantly reduced *A. vaccinii* larval survival ($F = 5.00$; df = 8, 72; $P < 0.0001$) (Fig. 4A). None of these treatments, however, reduced the number of berries that were infested, compared with the untreated check ($F = 1.73$; df = 8, 72; $P = 0.11$) (Fig. 4B). This suggests that as the residues

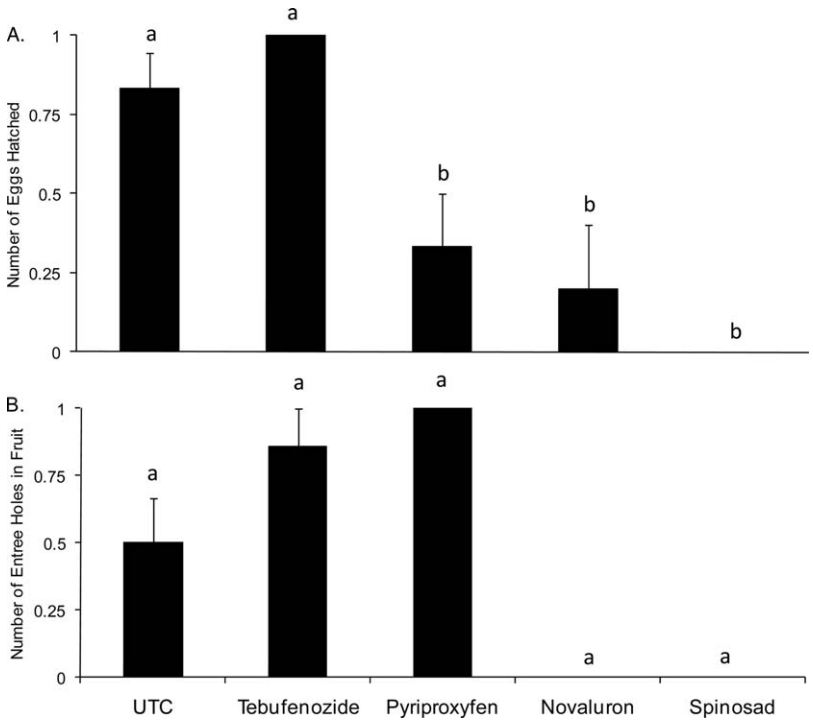

Fig. 2. Mean number ± SE of *A. vaccinii* eggs hatched (A) or entry holes in fruit (B) after exposure to preoviposition treatments of different insecticides. Bars with the same letter are not significantly different at $P < 0.05$.

decline, multi-berry exposure is needed to cause mortality. Thus, after 2 wk of field aging, the remaining lethal activity of these compounds on *A. vaccinii* larvae is not sufficient to protect blueberries from cluster infestation. This is not to say, however, that the combined adulticidal, ovicidal and larvicidal activity under real field conditions will not result in adequate insecticidal performance to justify a 14-d application interval.

Parasitism Assay. When topically applied to parasitized *A. vacinii* eggs, azinphosmethyl, phosmet, indoxacarb, thiacloprid, and acetamiprid significantly reduced the survival of *T. minutum* ($F = 6.37$; df = 10, 106; $P < 0.0001$) (Fig. 5). In contrast, pyriproxyfen, emamectin benzoate, methomyl, novaluron, and spinosad did not negatively affect the survival of *T. minutum.*

Discussion

Protection of blueberries against infestation by *A. vaccinii* is typically achieved using broad-spectrum insecticides, such as azinphosmethyl, that provide high levels of insect control. This study provides evidence for similar performance against specific life stages of *A. vaccinii* by alternative insecticides that possess much lower environmental and worker risk profiles (USEPA 1997). Understanding which insect life stages are susceptible to the available insecticide options will help direct their optimal use in pest management programs. The life-stage activity experiments included here shed new light onto the modes of insecticidal activity responsible for fruitworm control. Even though fruitworm spray recommendations have long targeted *A. vacinii* larvae (Wise et al. 2009), the organophosphate, carbamate, and pyrethroid insecticides have lethal activity on all three *A. vaccinii* life stages. Although the different life-stage experiments were not specifically designed to be comparative in nature, the trends across the different data sets provide some key insights. For example, even though azinphosmethyl has been successfully used on a 2-wk spray interval in blueberries for many years, the insecticide duration experiment shows that azinphosmethyl's larvicidal activity substantially weakens after 7 d of field aging. This suggests that the overall performance of azinphosmethyl is heavily reliant on its ovicidal and adulticidal activity to maintain fruit protection on a traditional 2-wk spray interval. The other organophosphate tested, phosmet, the pyrethroid esfenvalerate, and the carbamate methomyl followed similar life-stage activity patterns, although they tend not to match the overall field performance of azinphosmethyl (R.I. and J.C.W., unpublished data). The neonicotinoid insecticides thiacloprid and acetamiprid were shown to be active on all three *A. vacinii* life stages, with strong larvicidal and ovicidal activity, but were weaker adulticides than the conventional broad spectrum compounds. The reduced health effects associated with acetamiprid may be a reflection of the initial symptoms of toxicity,

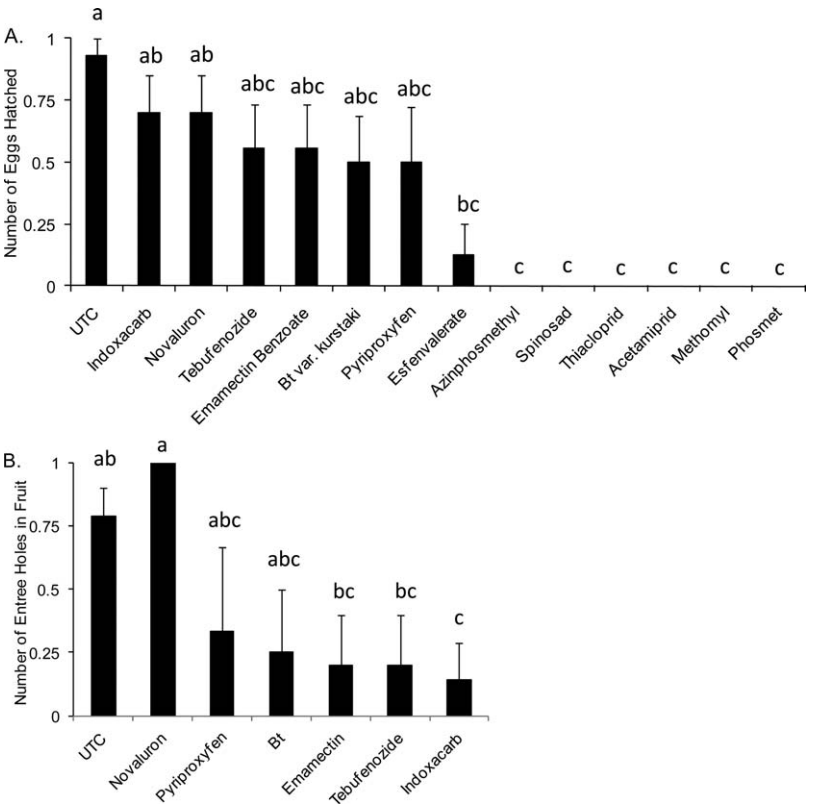

Fig. 3. Mean number ± SE of *A. vaccinii* eggs hatched (A) or entry holes in fruit (B) after exposure to postoviposition treatments of different insecticides. Bars with the same letter are not significantly different at $P < 0.05$.

or they may be indicative of the sublethal neurotoxic effects seen with neonicotinoid exposure with other insect species (Kunkel et al. 2001). Other studies with fruit-feeding beetle species confirm that neonicotinoids have relatively short-lived adult contact toxicity, with the degree of insect mortality being well correlated with surface residue profiles, and adult toxicity declining as residues penetrate plant tissues (Wise et al. 2006a, 2007). Thus, good spray coverage of fruit clusters is essential to maximize the ovicidal and larvicidal activity of neonicotinoids, as demonstrated under field conditions for many of the reduced-risk insecticides being developed and registered for use against *A. vaccinii* in blueberry (Dorschner et al. 2009).

Although this study did not provide complete life-stage data for *A. vaccinii* susceptibility to *B. thuringiensis* variety *kurstaki*, indoxacarb, and emamectin benzoate, these results along with other published research on these materials suggests that they are heavily reliant on larvicidal activity to achieve control of fruitworms (Wise et al. 2000, 2003, 2004, 2005). In addition, it seems that ingestion is an important means of delivery of the toxicant for these materials and that residual activity diminishes after 7 d under field conditions.

The spinosyn compound spinosad showed a surprisingly high level of toxicity to *A. vacinii* adults, and ovicidal activity was demonstrated for eggs that were laid on top of residues as well as for when topically applied. Spinosad's larvicidal activity, however, seems to be relatively short lived under the conditions of these tests. This suggests that spinosad can provide valuable flexibility in fruitworm management programs, and optimal timing may be at or after blueberry petal-fall stage when all fruitworm life stages are present.

The insect growth regulators (IGRs) pyriproxyfen and novaluron had strong ovicidal activity on *A. vacinii* when eggs were laid on top of residues and more limited ingestion-active larvicidal activity. This latter form of activity may result in berry infestation before larval mortality is complete. Therefore, an earlier spray timing, which ensures that residues are present before eggs are laid on fruit, will be necessary for optimal performance of these materials. The diacylhydrazine IGR tebufenozide was not directly toxic to *A. vaccinii* eggs, but it demonstrated ovilarvicidal activity when applied topically after eggs had been laid. Although having excellent residual activity on *A. vaccinii* larvae, tebufenozide's singular life-stage activity demands precise application timing to achieve optimal fruit protection.

Understanding the impact of pesticides on natural enemies is a cornerstone of IPM (Stern et al. 1959, Barbosa 1998), supporting the development of man-

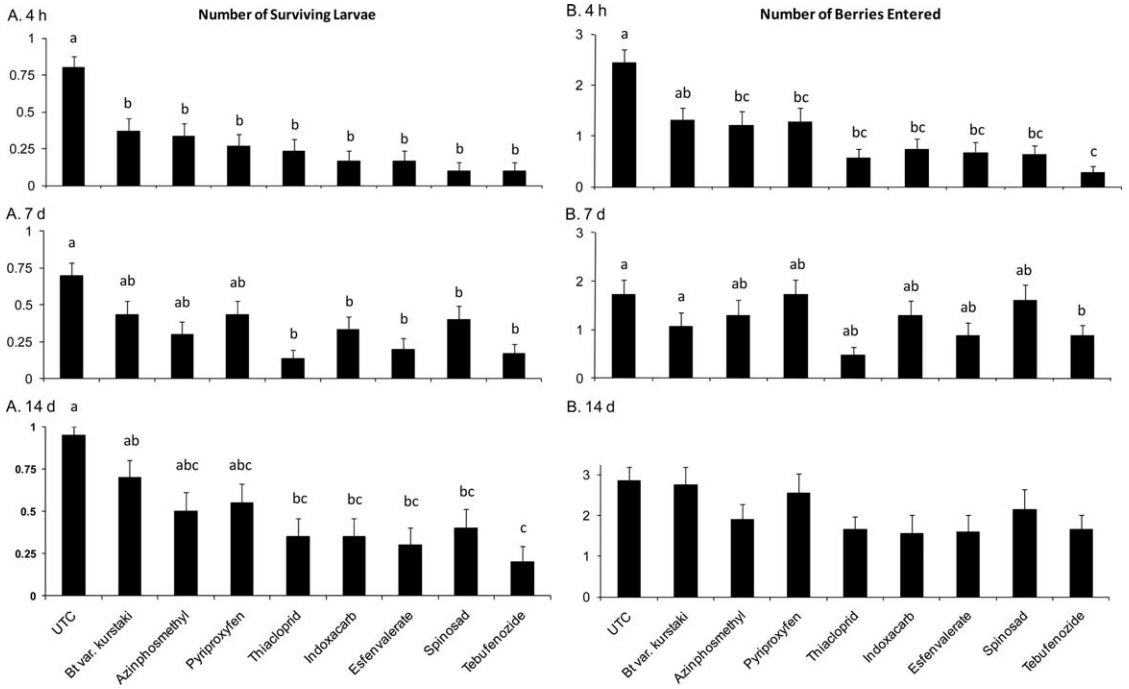

Fig. 4. Mean number ± SE of surviving *A. vaccinii* larvae (A) or berries entered (B) after exposure to 4 h, 7 d, and 14 d field-aged residues of different insecticides. Bars with the same letter are not significantly different at $P < 0.05$.

agement programs that conserve biological control and maintain pests below economic thresholds. Such an integrated approach toward management of *A. vaccinii* is under development and this study provides guidance on the insecticides that will support or undermine the ability of natural enemies to regulate this pest. Azinphosmethyl, phosmet, indoxacarb, thiacloprid, and acetamiprid were all shown to have significant negative impact on *T. minutum*. The toxicity of organophosphates was expected, and because the neonicotinoids are neurotoxic to parasitoid insects (Cordero et al. 2007, Rill et al. 2007) and ovicidal to *A. vaccinii*, their negative effects on *T. minutum* in this study are not surprising. Given that indoxacarb did not show ovicidal activity on *A. vacinii*, the observed reduction in *T. minutum* survival indicates direct toxicity to the parasitoid egg or larval stages. The relatively benign effect of pyriproxyfen, emamectin benzoate, novaluron, and spinosad observed in this study (and the expected lack of effect from tebufenozide and *B. thuringiensis* variety *kurstaki*) on *T. minutum* provide guidance for decision makers when implementing blueberry IPM programs and indicate that these insecticides will be compatible with biological control. These patterns of safety to egg parasitoids also may trans-

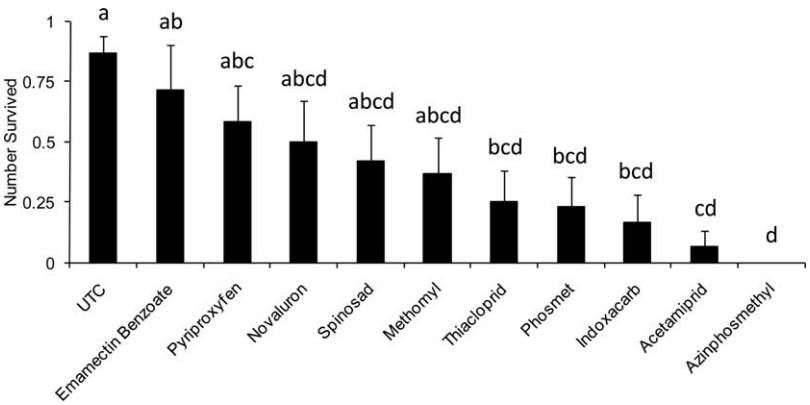

Fig. 5. Mean number ± SE of *T. minutum* surviving after *A. vaccinii* eggs were treated with different insecticides. Bars with the same letter are not significantly different at $P < 0.05$.

late to the larval parasitoids that attack *A. vaccinii* (Murray et al. 1986), although the location of eggs and larvae within the fruit is expected to provide some protection and result in the main effects being on the adult parasitoids.

Our bioassay results indicate that selective insecticides can be deployed for effective control of *A. vaccinii* in blueberry while also conserving biological control. Some of these new options will require that growers make applications timed carefully to ensure optimal exposure of susceptible life stages, and we are currently developing a growing degree-day–based pest development model for this pest to assist with this decision making. Field-scale implementation of IPM programs that integrate these principles and insights will help growers transition from azinphosmethyl to effective alternative management tactics.

Acknowledgments

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