Toxicity of Avermectins to Diamondback Moth (Lepidoptera: Plutellidae) Populations: Implications for Susceptibility Monitoring

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ABSTRACT The susceptibility of diamondback moth, *Plutella xylostella* (L.), larvae to 2 avermectins, abamectin and MK-243 (4″-epi-methylamino-4″ deoxyavermectin B_1 hydrochloride), was assessed through toxicity evaluations of 17–19 geographically diverse populations. Both avermectins were very potent to 3rd-instar diamondback moth; LC_{50}s for abamectin and MK-243 ranged from 0.4 to 44.0 ng (AI)/ml and 0.2 to 8.0 ng (AI)/ml, respectively. Although most populations showed resistance to methomyl and permethrin, the populations were not resistant to abamectin or MK-243. Pairwise correlations indicated lack of cross or multiple resistance between the avermectins and methomyl and permethrin. This information will serve as an aid for resistance monitoring programs and permit comparative changes in susceptibility during commercial use. Such data are necessary to assist in decisions to recommend product use appropriately in the context of resistance management.

KEY WORDS *Plutella xylostella*, abamectin, resistance monitoring, emamectin benzoate, MK-243, MK-244

THE DIAMONDBACK MOTH, *Plutella xylostella* (L.), is a ubiquitous pest where mustard crops are grown. The annual cost on a worldwide basis for controlling this pest is estimated to be $1 billion (U.S.) (Talekar and Shelton 1992). Although the diamondback moth has a fairly large parasitoid complex, which may aid in population suppression (Putnam 1973, Lasota and Kok 1986), insecticides are required to maintain populations below economically damaging levels.

Because of selection pressure resulting from intensive pesticide use to control *P. xylostella*, insecticide resistance to this insect has been documented for all synthetic chemical classes registered in the United States. These include organophosphates (methamidophos), carbamates (methomyl), and pyrethroids (permethrin) (Shelton et al. 1993b). In southeast Asia (where as many as 15–20 diamondback moth generations occur per year), resistance to these chemical classes was reported over a decade ago (Sun et al. 1978, Liu et al. 1981). In addition, resistance to the benzoylphenylurea compounds has been reported in diamondback moth populations collected in southeast Asia (Pering et al. 1988). Most recently, field failures and resistance to *Bacillus thuringiensis* Berliner bioinsecticides has been documented (Tabashnik et al. 1990, Ferre et al. 1991, Shelton et al. 1993a). Because of this widespread resistance, there is an urgent need for chemicals with different modes of action and that do not select for cross-resistance to conventional insecticides. Integrating new chemistries into insecticide rotation programs should reduce selection pressure from a single product or products with similar chemistry and mode of action, thus prolonging the usefulness of all products.

Abamectin, a member of the avermectin class of compounds is a potent, broad spectrum acaricide and narrow spectrum insecticide (Lasota and Dybas 1991). Although abamectin has shown differential potency to lepidopterans (Lasota and Dybas 1991), it is highly toxic to *P. xylostella* larvae (Abro et al. 1988, 1989). MK-243 (4″-epi-methylamino-4″ deoxyavermectin B_1 hydrochloride), also referred to as emamectin hydrochloride, is a semi-synthetic analog of abamectin, and is highly potent to a wide range of lepidopteran species (Dybas et al. 1989, Mrozik et al. 1989). Emamectin hydrochloride and emamectin benzoate (MK-244) are the hydrochloride and benzoate salt forms of 4″-epi-methylamino-4″ deoxyavermectin B_1 and have shown comparable insecticidal activity in the laboratory and field (R.A.D., unpublished data). In foliar ingestion bioassays with southern armyworm, *Spodoptera eridania* (Cramer), MK-243 was 1,720, 884, and 268 times more toxic than methomyl, thiodicarb, and fenvalerate, respectively (Dybas et al. 1989). Against tobacco budworm, *Heliothis virescens* (F.),

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this compound was reported to be 3,300, 1,666, and 500 times more toxic than these commercial insecticides (Dybas et al. 1989). Emamectin benzoate is highly efficacious in field control of *P. xylostella* (Leibee et al. 1995). Excellent larval control and reduction in marketable yield losses have been seen with rates of 5.6–22.4 g (AI)/ha. Emamectin benzoate is currently under commercial field development for diamondback moth control in the United States and Japan; abamectin is registered in southeast Asia for control of this pest. Before widespread use of avermectins for diamondback control, an assessment of data on susceptibility of geographically diverse *P. xylostella* populations is necessary. Quantification of the susceptibility of this species to avermectins will serve to aid in resistance monitoring programs to detect changes in sensitivity following commercial use. Thus, information gathered in a monitoring program will aid in decisions to appropriately label and recommend product use in the framework of resistance management.

In the experiments described here, the sensitivities of field-collected diamondback moth populations to avermectins, permethrin, and methomyl were compared. Permethrin and methomyl are 2 of the most widely used commercial insecticides for diamondback moth control (Shelton et al. 1993b). We anticipated differential toxicity to the commercial products based on reports of field failures. Thus, our additional objective was to assess cross- or multiple-resistance between or among the avermectins and methomyl and permethrin. At the time of this study, the avermectins (except for abamectin in Thailand) had not yet been registered for diamondback moth control.

Materials and Methods

In 1989 and 1990, 19 diamondback moth populations were collected from various cole crop growing regions, including 17 populations from the United States. These populations were collected from Florida (7), Ohio (2), Texas (1), New York (2), North Carolina (1), Virginia (1), California (1), Maryland (1), and Michigan (1). In several cases, 2 populations were collected from a given location. One population each from Indonesia and Malaysia also were tested. A population collected from Geneva, NY, in 1988 and reared continuously in the laboratory was used as our standard. Each population originated from a field sample of 50–150 diamondback moth larvae and pupae collected from commercial cabbage fields. Collections were made both in areas where growers had experienced difficulty controlling diamondback moth with methomyl or permethrin, or both, and from areas where specific information on product effectiveness was unavailable.

All populations were reared in the laboratory on rape seedlings (Shelton et al. 1991) for ≥1 generations before testing. Leaf dip insecticide assays (similar to the method described by Tabashnik and Cushing [1987]) were used to estimate dose–response relationships for abamectin (0.15 EC [emulsifiable concentrate]), (Merck, Rahway, NJ), MK-243 (0.16 EC) (Merck), permethrin (Ambush 2 E) (ICI, Wilmington, DE) and methomyl (Lannate 1.8 L [liquid]) (DuPont, Wilmington, DE). Six to 7 concentrations were used for each insecticide, with the following ranges: permethrin, 5.62–0.0018 mg (AI)/ml; methomyl, 100–0.0056 mg (AI)/ml; abamectin, 100–0.316 ng (AI)/ml,; and MK-243, 100–0.1 ng (AI)/ml. Five drops of BOND sticker spreader (Loveland Industries, Loveland, CO) per 100 ml H_2O were added to each of the above concentrations.

Cabbage leaves from the outer layers of the head were cut into disks (6 cm diameter) and dipped into an insecticide mixture for 5 s, held vertically to allow excess solution to drip off, and placed on a drying rack. After 2 h drying time, the disks were placed in a plastic petri plate. Five to 10 third instars per replicate were added to each plate. Each concentration was replicated 3 times, as was the untreated control (distilled water and sticker spreader). We used at least 15 larvae per concentration for each of the 6–7 concentrations per insecticide. Larval mortality was assessed at 24 and 48 h for permethrin and methomyl and 72 and 96 h for abamectin and MK-243. The longer evaluation time for the avermectins was based on previously reported information on stabilization of mortality in diamondback moth (Abro et al. 1988) and other lepidopteran species (Dybas et al. 1989) indicating that these compounds are slow acting ingestion toxicants. Although gut paralysis and cessation of feeding occurs within a relatively short period (within 24 h), maximum mortality cannot accurately be quantified until 72–96 h after treatment. This delay contrasts with the more rapid activity of the carbamate and pyrethroid.

For each population for each compound, probit regressions were estimated after data were corrected by Abbott's formula (Abbott 1925). LC_{50}s are presented. Confidence intervals were computed by using Fieller's theorem (Finney 1978). Computations were performed using PROC PROBIT in SAS (SAS Institute 1990).

The LC_{50}s of diamondback moth populations were considered to differ if their 95% CI did not overlap. Because information regarding field performance of the commercial products to various populations was circumstantial, comparisons among all populations were made by use of 2 types of 95% CI. For single regressions, we used the 95% CI for the individual population LC_{50}s. To account for multiple comparisons (hence, cumulative error) among populations, however, a larger interval was used. The wider interval, controlling for all pairwise comparisons among populations within a compound, was computed by using the 1/2 Tukey honestly significant difference (HSD) test (Kirk 1968) with an overall $\alpha = 0.05$. For mul-

Table 1. Foliar ingestion toxicity of abamectin to 3rd-instar *P. xylostella* larvae, 72 h after treatment (n = 90 larvae per population)

Population	Generation	LC_{50} ng (AI)/ml	Individual 95% CI	Slope ± SE
Bushnell, FL	1	0.44	(<0.01–2.10)	0.66 ± 0.24
Fremont, OH	16	0.53	(0.01–2.02)	0.71 ± 0.23
Donna, TX	1	0.56	(0.11–1.25)	1.13 ± 0.29
Florida	1	0.84	(0.22–1.79)	1.07 ± 0.25
North Carolina	2	0.96	(<0.01–23.54)	0.39 ± 0.18
Rehobeth, MD	1	1.35	(0.44–2.74)	1.45 ± 0.34
Bogor, Indonesia	3	2.20	(<0.32–100.0)[a]	—
Ruhlig, MI	2	2.40	(0.87–4.77)	1.36 ± 0.29
Celeryville, OH	32	3.73	(0.59–13.48)	0.61 ± 0.18
Sanford, FL	34	5.49	(2.09–9.37)	2.16 ± 0.62
Geneva, NY (Standard)	88	5.98	(<0.32–10.0)[a,b]	—
Painter, VA	1	6.36	(2.29–12.81)	1.52 ± 0.36
Zellwood, FL	1	6.83	(0.35–100.33)	1.12 ± 0.32
Cameron, Malaysia	8	12.64	(3.07–26.97)	1.33 ± 0.40
Sarasota, FL	1	13.46	(3.61–19.87)	3.82 ± 1.56
Penasquitos, CA	1	14.37	(0.32–>100.0)[a]	—
Geneva, NY	20	25.34	(<0.32–100.0)[a]	—
Zerlina, FL	9	27.16	(10.32–104.20)	0.96 ± 0.22
Stanley, NY	3	44.18	(4.38–4,340.28)	1.45 ± 0.69

[a] Confidence intervals estimated by using the simultaneous binomial hypothesis test method because assuming the probit model, intervals were infinite.

[b] Results of analysis of Geneva, NY, population eliminating the 2nd highest concentration tested caused by highly variable results at that level.

tiple comparisons, we used Bonferroni's test (Peace 1988). Standard Pearson product–moment correlation coefficients (Snedecor and Cochran 1980) were used to correlate pairs of compounds tested against the same populations (Finney 1978).

Results

Abamectin. LC_{50}s (Table 1) ranged from 0.4 (Bushnell, FL) to 44.4 ng/ml (Stanley, NY) at 72 h and from 0.01–30.0 ng/ml at 96 h (not shown). With 1 exception, we found no difference in LC_{50} among pairs of populations. In this exception, the Zerlina, FL, population (LC_{50} = 27.2 ng/ml at 72 h), collected from a greenhouse growing 'Zerlina' cabbage, was more tolerant than 6 other populations at 72 h and 1 other at 96 h. Although the LC_{50} for the Stanley, NY, population was numerically greater than that of Zerlina, differences between this population and others cannot be accurately described based on separation based on simultaneous confidence limits.

MK-243. LC_{50} estimates (Table 2) ranged from 0.2 (Donna, TX) to 8.0 ng/ml (Penasquitos, CA) at 72 h. Only the Geneva, NY, the Zellwood, FL, and the Sarasota, FL, populations had greater LC_{50}s than the population from Donna, TX (for which the upper 95% CI at LC_{50} was 31.6 ng/ml). This

Table 2. Foliar ingestion toxicity of MK-243 to 3rd-instar *P. xylostella* larvae, 72 h after treatment (n = 90 larvae per population)

Population	Generation	LC_{50} ng (AI)/ml	Individual 95% CI	Slope ± SE
Donna, TX	1	0.16	(0.01–0.52)	0.71 ± 0.21
Bushnell, FL	1	0.83	(0.40–1.36)	2.23 ± 0.56
Ruhlig, MI	2	0.91	(<0.01–13.17)	1.57 ± 0.53
Celeryville, OH	32	1.12	(<0.10–3.16)	—
Florida	1	1.30	(0.10–31.60)[a]	—
Painter, VA	1	1.34	(0.50–2.44)	2.01 ± 0.53
Zerlina, FL	9	1.45	(0.41–4.01)	0.98 ± 0.20
Cameron, Malaysia	8	1.61	(<0.10–10.0)[a]	—
Rehobeth, MD	1	1.68	(0.04–5.91)	1.14 ± 0.41
North Carolina	2	2.60	(0.32–10.0)[a]	1.39 ± 0.51
Geneva, NY (Standard)	88	3.08	(1.0–3.16)[a]	2.32 ± 1.12
Zellwood, FL	1	3.65	(1.0–31.6)[a]	1.45 ± 0.53
Fremont, OH	16	3.87	(<0.10–10.0)[a]	—
Sanford, FL	34	4.25	(<0.10–10.0)[a]	—
Bogor, Indonesia	3	6.20	(<0.10–31.60)[a]	—
Sarasota, FL	1	6.98	(3.05–10.44)	3.82 ± 1.35
Penasquitos, CA	1	7.57	(0.32–10.0)[a]	2.63 ± 1.26

[a] Confidence intervals estimated by using the simultaneous binomial hypothesis test method because assuming the probit model, intervals were infinite.

Table 3. Toxicity of methomyl to 3rd instar *P. xylostella* larvae, 48 h after treatment (n = 90 larvae per population)

Population	Generation	LC_{50} mg (AI)/ml	Individual 95% CI	Slope ± SE
Geneva, NY (Standard)	88	0.03	(0.01–0.08)	0.90 ± 0.19
Celeryville, OH	32	0.06	(<0.01–0.26)	0.90 ± 0.25
PC. Penasquitos, CA	1	0.30	(0.16–0.53)[a]	1.68 ± 0.32
Fremont, OH	16	0.39	(0.06–0.64)	—
Penasquitos, CA	1	0.54	(<0.01–3.16)[b]	—
Painter, VA	1	0.65	(<0.01–1.78)	1.32 ± 0.63
North Carolina	2	0.82	(0.21–2.15)	1.12 ± 0.23
Sanford, FL (A)	34	1.56	(0.83–2.88)	1.99 ± 0.45
Sanford, FL (B)	34	1.92	(<0.01–2.99)	—
Donna, TX	1	2.19	(0.99–3.88)	1.57 ± 0.33
Stanley, NY	3	2.67	(<0.02–17.78)	—
Cameron, Malaysia	8	3.07	(1.34–7.08)	1.28 ± 0.24
Zerlina, FL	9	3.53	(0.56–17.78)	—
Rehobeth, MD	1	5.81	(<0.02–17.78)[b]	2.39 ± 0.91
Ruhlig, MI	2	6.92	(2.30–13.84)	1.56 ± 0.47
Florida	1	7.15	(0.81–136.08)	1.02 ± 0.27
Bushnell, FL	1	8.08	(2.18–38.75)	1.13 ± 0.33
Bogor, Indonesia	3	8.60	(1.48–17.13)	1.76 ± 0.64
Sarasota, FL	1	44.45	(27.26–66.70)	2.73 ± 0.69
Zellwood, FL	1	49.52	(27.84–77.76)	3.61 ± 0.97

[a] Results of analysis of Pensaquitos, CA, population eliminating the highest concentration tested (3.16 mg/ml) because of highly variable results.

[b] Confidence intervals estimated by using the simultaneous binomial hypothesis test method because assuming the probit model, intervals were infinite.

low value, the narrow range of LC_{50} values, and lack of differences among LC_{50} values indicate lack of resistance to MK-243. At 96 h, only the Sanford, FL, and Stanley, NY, populations had LC_{50}s outside 0.03–3.0 ng/ml; values were 20.0 and 10.0 ng/ml, respectively. Similar to the result at 72 h, the Zellwood, FL, and Geneva, NY, populations had greater LC_{50}s than the Donna, TX, population. Many populations could not be compared either because the range of doses was not low enough, the sample sizes were too small, or both.

Methomyl. The Geneva, NY (Table 3), population (LC_{50} = 0.14 mg/ml at 24 h [results not shown], and 0.03 mg/ml at 48 h) was more sensitive than nearly every other population, (versus most others at 24 and 48 h). Thus, most populations tested showed resistance to methomyl. LC_{50}s ranged from 2–42 mg/ml at 24 h, and from 0.3–50 mg/ml at 48 h.

Permethrin. At 24 h (results not shown), most populations had LC_{50}s ranging from 0.2–2 mg/ml; only 5 populations had lower LC_{50}s (from 0.007–0.07 mg/ml). At 48 h (Table 4), most populations had LC_{50}s from 0.1–0.7 mg/ml. All populations except the Celeryville, OH, the Fremont, OH, the Geneva, NY, the Penasquitos, CA, and one of the Sanford, FL, populations exhibited resistance to permethrin; LC_{50}s for these populations (0.001–0.06 mg/ml) indicated resistance in most cases. Most of the populations tested showed resistance to methomyl and permethrin, but not to abamectin or MK-243.

Correlation of Resistance. Correlation of $\log_{10}(LC_{50})$ for pairs of compounds tested against the same populations was assessed to determine relationships of resistance development among the compounds (cross or multiple resistance). $\text{Log}_{10}(LC_{50})$ estimates for methomyl and permethrin were highly correlated (r = 0.83, $P < 0.0001$), indicating a similar resistance pattern for these 2 compounds. Abamectin and MK-243 $\log_{10}(LC_{50})$ estimates were not correlated with those of any of the other compounds, indicating lack of cross- or multiple resistance among the avermectins versus methomyl or permethrin. The pairwise correlations are presented in Table 5.

Discussion

The wide range in sensitivity of diamondback moth larvae to the avermectins may reflect the small sample sizes used in this bioassay, although numbers used did not depart substantially from those suggested for use in laboratory bioassays by Robertson et al. (1984). In their article, a minimum of 120 test organisms is recommended for a reliable dose–response study, with increased reliability at larger total sample sizes (for example, 240). Although precision in estimation of lethal concentration values may have been increased by use of 20 versus 15 larvae for each of the 6 doses, our data provide generally useful ranges for baseline susceptibility across diverse populations. A larger sample size may only have served to lessen the variability in sensitivity between populations for individual compounds and would not alter our conclusions of comparisons between the avermectins and methomyl and permethrin. Our conclusions are based on comparison of our results with those obtained by Shelton et al. (1993b). The only

Table 4. Toxicity of permethrin to 3rd-instar *P. xylostella* larvae, 48 h after treatment (n = 90 larvae per population)

Population	Generation	LC_{50} mg (AI)/ml	Individual 95% CI	Slope ± SE
Fremont, OH	26	<0.01	(<0.02–0.18)[a]	—
Geneva, NY (Standard)	88	<0.01	(<0.01–0.01)	1.01 ± 0.31
Penasquitos, CA	1	0.01	(<0.01–0.01)	1.62 ± 0.27
Celeryville, OH	32	0.02	(<0.01–0.10)	1.38 ± 0.47
Sanford, FL (A)	34	0.06	(0.02–0.12)	1.61 ± 0.37
Bogor, Indonesia	3	0.12	(0.03–0.28)	1.30 ± 0.28
Painter, VA	1	0.13	(<0.01–0.27)	1.73 ± 0.79
Cameron, Malaysia	8	0.16	(<0.01–0.49)	1.15 ± 0.34
Ruhlig, MI	2	0.24	(0.14–0.43)	1.86 ± 0.35
Donna, TX	1	0.29	(0.02–1.78)[a]	—
Stanley, NY	3	0.35	(0.10–0.58)	2.96 ± 1.07
North Carolina	2	0.36	(0.09–0.50)	5.51 ± 2.26
Bushnell, FL	1	0.49	(0.01–6.89)	1.34 ± 0.42
Rehobeth, MD	1	0.49	(0.27–0.75)	2.79 ± 0.75
Sanford, FL (B)	34	0.59	(0.18–1.78)	—
Zellwood, FL	1	0.61	(0.14–1.03)	1.88 ± 0.68
Florida	1	0.62	(0.41–0.85)	2.93 ± 0.62
Zerlina, FL	9	0.67	(0.31–1.06)	2.60 ± 0.80
Sarasota, FL	1	0.74	(0.53–1.0)	3.15 ± 0.63

[a] Confidence intervals estimated by using the simultaneous binomial hypothesis test method because assuming the probit model, intervals were infinite.

difference between their bioassay and ours was a larger sample size; mean sample sizes for methomyl and permethrin were 232 and 236, respectively. The range of 48 h LC_{50} values for methomyl (0.03–49.52 mg/ml) and permethrin (0.00146–0.744 mg/ml) obtained in our study nearly completely overlap the ranges of those obtained by Shelton et al. (1993b) (methomyl, 0.106–82.73 mg/ml; permethrin, 0.002–2.669 mg/ml).

The wide range in diamondback moth response to abamectin (LC_{50} = 0.4–44.0 ng/ml; 72 h after treatment) and MK-243 (LC_{50} = 0.2–8.0 ng/ml; 72 h after treatment) should prompt investigation into the mechanisms by which the avermectins act to kill this pest by ingestion. In addition, differences in response of diamondback moth to avermectin (especially drug detoxification by metabolic processes or possible differences in avermectin binding characteristics) require evaluation. Abro et al. (1989) also suggested that differences in toxicity to this species may be indirectly influenced by variability in translaminar uptake of abamectin by different host plants. Such a situation could be critical in identifying reasons for variability in diamondback moth control under field conditions. In our study, variability in translaminar uptake among leaf disks may account for some of the variation in population responses to the avermectins.

Table 5. Correlation coefficients among $\log_{10}$ (LC_{50}) estimates for pairs of compounds tested against the same populations

	Methomyl	MK-243	Permethrin
Abamectin	−0.31 (P = 0.20)	0.44 (P = 0.08)	−0.11 (P = 0.67)
Methomyl		−0.17 (P = 0.52)	0.83 (P < 0.0001)
MK-243			−0.32 (P = 0.21)

Pearson product–moment correlation coefficients (Snedecor and Cochran 1980).

In summary, most of the populations that we tested (with 1 exception) exhibited resistance to methomyl and permethrin, but not to MK-243 and abamectin. For abamectin, only the Zerlina, FL, population collected from greenhouse-grown cabbage differed from the other populations at 72 h after treatment, although it did not differ from the others at the 96 h evaluation time. Populations from this location and other locations of high insecticide pressure should be closely monitored to ensure maintenance of avermectin sensitivity. Results of our study have indicated that the avermectins do not appear to be cross-resistant with the carbamate and pyrethroid insecticides.

This study has also shown that diamondback moth is very sensitive to the avermectins–abamectin and MK-243–considerable variability was observed in sensitivity to both compounds among geographically diverse populations. Information presented here will serve as an aid in resistance monitoring programs, particularly for emamectin benzoate (MK-244) in the United States, to detect changes in susceptibility during commercial use for diamondback moth control. Results of future monitoring can be used in decisions to appropriately limit and recommend product use (rotation with products with different modes of action) in the context of sound resistance management programs, with the ultimate goal being prolongation of product usefulness. Such a proactive approach has not been widely practiced in the agricultural chemical industry.

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