Toxicity, Joint Action, and Mean Time of Mortality of Dipel 2X, Avermectin B_1, Neem, and Thuringiensin Against Beet Armyworms (Lepidoptera: Noctuidae)

WILLIAM J. MOAR AND JOHN T. TRUMBLE

Department of Entomology, University of California,
Riverside, California 92521

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ABSTRACT Toxicity of Dipel 2X, avermectin B_1, and neem to beet armyworm, *Spodoptera exigua* (Hübner), was determined using neonate larvae in a diet-incorporation bioassay. LC_{50}'s for Dipel 2X, avermectin B_1, and neem in diet medium were 196 μg/ml, 0.814 μg (AI)/ml, and 0.116 μl/ml, respectively. Combinations of ABG-6162 (an experimental formulation of thuringiensin) and Dipel 2X resulted in potentiation, whereas combinations of neem with avermectin B_1 or Dipel 2X produced an antagonistic effect. There was no significant linear relationship in mean time of mortality across all concentrations within a toxicant except for ABG-6162. Mean time of mortality was significantly different among toxicants. These times for Dipel 2X, avermectin B_1, neem, and ABG-6162 were 3.06, 4.13, 5.42, and 6.23 d, respectively. Mean time of mortality for toxicant combinations was variable and provided insight into modes and sites of action for compounds applied individually or jointly.

KEY WORDS *Spodoptera exigua, Bacillus thuringiensis* var. *kurstaki,* neem, avermectin, thuringiensin

THE BEET ARMYWORM (BAW), *Spodoptera exigua* (Hübner), is a key pest in California on lettuce (Oatman & Platner 1972), celery (Van Steenwyk & Toscano 1981), and tomatoes (Lange & Bronson 1981). Currently, BAW is resistant to some chlorinated hydrocarbons, cyclodienes, organophosphates, and pyrethroids (Georghiou 1981) and has demonstrated potential for resistance to the carbamate methomyl (Alava & Lagunes 1976, Meinke & Ware 1978), which is the predominant chemical used for control (Meinke & Ware 1978, Trumble & Toscano 1983). Therefore, new chemicals or combinations of chemicals that can effectively control BAW must be identified.

Several relatively new chemicals have substantial activity against BAW. Seed extracts from the neem tree, *Azadirachta indica* A. Juss, have insect phagodeterrent and growth-regulating properties (Warthen 1979), which may reduce cosmetic damage in vegetable crops (Arnason et al. 1985). Avermectin B_1 (AVM), a macrocyclic lactone isolated from the mycelia of the soil organism *Streptomyces avermitilis* (Burg et al. 1979), is toxic at low concentrations to many insects, including BAW and *Spodoptera eridania* (Cramer) (Putter et al. 1981; Trumble et al. 1987). The primary mode of action of AVM is to interfere with the action of gamma aminobutyric acid (Fritz et al. 1979, Mellin et al. 1983). AVM has the potential for incorporation into integrated pest management programs in celery and tomatoes for control of the leafminer *Liriomyza trifolii* (Burgess), because this chemical does not damage the parasite-based biological control system (Trumble 1985).

Chemical mixtures may control BAW and other insecticide-resistant insects. Microbial insecticides such as Dipel 2X, which contains the HD-1 isolate of *Bacillus thuringiensis* var. *kurstaki* (Berliner), and Javelin, which contains the NRD-12 isolate, exhibit greater activity against BAW when combined with the β-exotoxin (thuringiensin) produced by some varieties of *B. thuringiensis* (Moar et al. 1986). Tropital increased the growth-disrupting effects of neem 6-fold to *Epilachna varivestis* Mulsant at a ratio of 1:2 (Lange & Schmutterer 1982). Anderson et al. (1986) showed that piperonyl butoxide significantly increased the effectiveness of AVM applied topically to *S. eridania.*

The purpose of this research was to test the toxicity of Dipel 2X, AVM, and neem to BAW in a diet-incorporation bioassay and also to assay combinations of Dipel 2X, thuringiensin, AVM, and neem. Mean time of mortality for each treatment also was evaluated to determine if time of death was affected by toxicant combinations or could be related to the mode of action.

Materials and Methods

Test materials included Dipel 2X wettable powder containing the HD-1 isolate with 32,000 international units (IU)/mg (Abbott Laboratories), AVM

(18 g [AI]/liter emulsifiable concentrate; Merck Sharpe & Dohme), pure neem seed extract (supplied by ARS–USDA, J. D. Warthen Jr., Insect Chemical Ecology Laboratory, Beltsville, Md.), and the liquid ABG-6162, containing 1.5% thuringiensin (Abbott Laboratories). Neonate BAW larvae used in all tests were obtained from a laboratory colony established in 1982 from insects collected in Orange County, Calif., and maintained on an artificial diet (Patana 1969) at 27 ± 1°C and a photoperiod of 16:8 (L:D). Artificial diet medium was used in all bioassays.

Toxicity of Dipel 2X, Neem, and AVM. Bioassays consisted of seven to eight concentrations plus a control for individual toxicants. All toxicants were tested concurrently. Concentrations tested were as follows: Dipel 2X = 25, 50, 100, 200, 300, 400, and 800 μg/ml diet; AVM = 0.1, 0.2, 0.4, 0.6, 0.8, 1.2, 1.8, and 2.4 μg (AI)/ml diet; and pure neem extract = 0.01, 0.02, 0.04, 0.06, 0.08, 0.12, 0.16, and 0.32 μl/ml diet. Suspensions were made by adding materials to an aqueous 0.1% Tween 80 solution to produce 50 ml. Controls consisted of 50 ml of an aqueous 0.1% Tween 80 solution. Suspensions containing Dipel 2X were chilled to 10°C and suspended on a Branson sonic dismembrator (40% power, 30 s) to allow even distribution within the diet medium.

Each concentration was added to 1,300 ml of artificial diet medium after the medium was cooled to 43.5°C in an ice bath. The mixture then was blended for 2 min, and ca. 10 ml was poured into each clear plastic cup (30 ml). The mixture was allowed to dry for 1–8 h before one neonate (0–4 h old) BAW was placed in each cup. Cups were covered with opaque plastic lids and placed in a growth chamber at 27 ± 1°C and a photoperiod of 16:8. Thirty insects were evaluated per concentration, and each concentration was replicated six times. Larval mortality was assessed at day 7 in all tests.

Data initially were analyzed using the Proc Probit procedure (SAS Institute 1985) after correction for control mortality with Abbott's (1925) formula and then judged for suitability as described by Vandekar & Dulmage (1982). Suitable values were pooled. Control mortality was ≤10%.

Joint Action. Bioassays with the LC_{25} of each toxicant (estimated from probit lines) plus controls were prepared as described previously and then concurrently evaluated both individually and in pairs. Toxicant combinations were replicated six times, with 30 larvae per replicate. The LC_{25} for ABG-6162 was determined from previous studies with our laboratory colony (Moar et al. 1986). Toxicant combinations containing Dipel 2X were mixed in the sonic dismembrator before the addition of the other compounds. Mortality was recorded at 24-h intervals for 7 d. Joint effects were analyzed with a χ^2 test as described by Finney (1971) and Salama et al. (1984). If the χ^2 values observed exceeded the calculated value (df = 1; $P < 0.05$), we concluded that effects were not additive. Comparisons between the observed and expected mortality using LC_{25} values from probit lines were based on criteria of Vandekar & Dulmage (1982). Control mortality was ≤10%.

Table 1. Toxicity of Dipel 2X, neem, and AVM to neonate *S. exigua*

Treatment	n^a	Slope ± SEM	LC_{50} (95% FL)[b]
Dipel 2X	1,260	1.93 ± 0.1091	196 (175–217)
Neem	1,440	1.66 ± 0.1068	0.116 (0.104–0.131)
AVM	1,440	3.18 ± 0.2789	0.814 (0.707–0.925)

[a] Number of insects assayed over six replicates. Dipel 2X, seven concentrations; neem and AVM, eight concentrations.

[b] Dipel 2X in micrograms per milliliter of diet, neem in microliters per milliliter of diet, AVM in micrograms (AI) per milliliter of diet.

Mean Time of Mortality. To document the time after which BAW mortality occurred, mean time of mortality (MTM) was calculated. The number of larvae that died on a given day was divided by the total mortality after 7 d. This value then was multiplied by the respective day. Values for days 1–7 were summed to produce a weighted average mortality. MTM was calculated for all concentrations of each pesticide. The relationship between MTM and concentrations within each toxicant were assessed with regression of MTM on concentration. Analysis of variance (ANOVA) followed by mean separation with Duncan's (1955) multiple range test was used to compare toxicant MTM values. Three of the six replicates of Dipel 2X, AVM, neem, and ABG-6162 were evaluated every 24 h. Concentrations of ABG-6162 tested were 2.5, 5.0, 10, 15, 20, 30, 40, and 50 μg (AI)/ml diet and prepared as previously discussed. MTM for all paired combinations of pesticides was calculated from six replicates and differences were evaluated using ANOVA and Duncan's (1955) multiple range test. Differences in MTM values between LC_{25}'s estimated from probit analyses versus all concentrations for a given toxicant were determined by ANOVA and Duncan's (1955) multiple range test.

Results and Discussion

Toxicity of Dipel 2X, AVM, and Neem. The LC_{50}'s for Dipel 2X, neem, and AVM were 196 μg/ml diet, 0.116 μl/ml diet, and 0.814 μg (AI)/ml diet, respectively (Table 1). The LC_{50} for Dipel 2X was less than the 299 μg/ml diet reported in previous studies in which mortality was assayed at 6 d (Moar et al. 1986). Such variation is not uncommon among populations of other lepidopterous insects (Vandekar & Dulmage 1982). The LC_{50} of 0.814 μg (AI)/ml diet for AVM is in agreement with 0.755 μg (AI)/ml diet determined by Trumble et al. (1987). The LC_{50} of 0.116 μl/ml diet (ca. 130 ppm) for neem is similar to the results of Prabhaker et al. (1986), who found that neem at 200 ppm in

Table 2. Potency of four toxicants tested independently and in combination against neonate *S. exigua*

Concn (% mortality)[a]				Expected % larval mortality[b]	Observed % larval mortality	χ^2 value	Effect
Dipel 2X	ABG-6162	AVM	Neem				
87.5 (23)	20 (45)	—	—	57.6	100	56.6	Potentiation[c]
87.5 (23)	—	0.50 (13)	—	32.5	31.8	0.17	Additivity[d]
87.5 (23)	—	—	0.02 (37)	51.1	15.0	44.0	Antagonism[c]
—	20 (45)	0.50 (13)	—	52.4	62.6	3.63	Additivity[d]
—	20 (45)	—	0.02 (37)	66.3	56.7	2.45	Additivity[d]
—	—	0.50 (13)	0.02 (37)	45.0	12.9	40.0	Antagonism[c]

[a] Estimated lethal concentration needed to kill 25% of the test population at day 7 (actual percent mortality observed). Dipel 2X values in micrograms per milliliter of diet; AVM and ABG-6162 in micrograms (AI) per milliliter of diet; neem in microliters per milliliter of diet.

[b] Pooled data from bioassays of independent toxicants, replicated six times with 30 larvae per replicate.

[c] $P = 0.001$.

[d] $P = 0.05$.

artificial diet killed 53% of neonate BAW larvae in 8 d. Thus, the results for each chemical agree with published results of comparable tests.

Joint Action. Combinations of neem with either Dipel 2X or AVM produced an antagonistic effect (Table 2). Because these compounds were mixed before incorporation into the diet medium, they may have interacted either by forming a new, less toxic, compound, or one compound may have interfered with the toxic action of the other. However, because neem has antifeedant as well as growth-regulation properties (Warthen 1979, Kubo & Klocke 1982), less of the Dipel 2X or AVM may have been ingested, resulting in reduced mortality from the combinations. As in previous research (Moar et al. 1986), the combination of Dipel 2X and ABG-6162 resulted in potentiation (Table 2). Because no other insecticide combination produced this effect and the level of potentiation was high ($\chi^2 = 56.6$; $P \leq 0.001$), further research on the physiological and morphological bases for the effects of this combination on BAW is warranted. Independent action was observed for three of the six toxicant combinations (Table 2).

Percentages of observed mortality for independent LC_{25}'s of Dipel 2X, neem, AVM, and ABG-6162 were 22, 36, 12, and 45, respectively (Table 2). With the exception of ABG-6162, these LC_{25}'s were not different (after criteria of Vandekar & Dulmage [1982]) from the expected percentage of mortality using the LC_{25}'s estimated from the probit lines. The relatively high mortality caused by ABG-6162 possibly reflects BAW population variability as discussed previously for Dipel 2X, or resulted from the duration of assay (6 versus 7 d).

MTM. There was no significant linear relationship in MTM across all concentrations within a toxicant for Dipel 2X (slope = 0.060; SEM = 0.407; $R^2 = 0.0011$; $P \geq 0.88$), AVM (slope = −1.085; SEM = 0.628; $R^2 = 0.157$; $P \geq 0.104$), and neem (slope = 0.034; SEM = 0.652; $R^2 = 0.0001$; $P \geq 0.959$). ABG-6162 showed a significant linear relationship in MTM across concentrations (slope = −2.447; SEM = 0.615; $R^2 = 0.418$; $P \leq 0.0006$). Substantial changes in MTM with concentration suggest either a variable mode or site of action, or the presence of an additional toxic component. Just such a change in rate of mortality with concentration was an important factor leading to the documentation of the toxic effects of impurities in malathion (Umetsu et al. 1981). Lack of change in MTM with concentration may provide circumstantial evidence for a consistent mode or site of action.

MTM differed significantly among toxicants after data were pooled across concentrations ($F = 65.38$; df = 8; $P \leq 0.0001$) (Table 3). Results for Dipel 2X are similar to those reported by Moar et al. (1986), who noted that most mortality occurred at ca. 3 d after treatment for Dipel 2X. MTM for neem concentrations was >2 d longer than for Dipel 2X. This increase in time to mortality could be explained partially by either feeding repellency or inhibition of food movement across the midgut epithelium (Cottee & Mordue [Luntz] 1982), which would slow food utilization and thereby extend the developmental period required for molting beyond the normative time of 3.5 d (Fye & McCada 1972). In our experiments, as well as studies by Prabhaker et al. (1986), death occurred predominantly during the molt to the second instar. This molt-related mortality may occur in response to azadirachtin, the most active of the neem limonoids (Arnason et al. 1985), which interferes fatally with ecdysis in *Spodoptera frugiperda* (J. E. Smith) and *Heliothis zea* (Boddie) (Kubo & Klocke 1982).

When compounds were evaluated individually, MTM's were significantly different ($P < 0.05$), with shortest to longest being Dipel 2X, AVM, neem, and ABG-6162, respectively (Table 3). This variation could be due to the differences in modes of action already discussed. Except for AVM, MTM's for a single concentration (LC_{25}) were not significantly different ($P > 0.05$) compared with their values from similar tests that included all concentrations (Table 3). Because low but substantial mortality (45% of the total mortality) occurred on the 1st d (usually in association with control or handling

Table 3. MTM for four toxicants tested individually and in combination against neonate *S. exigua*

Treatment	MTM[a]			
	n^b	Individual analyses[c] (SEM)	n	Joint action[d]
ABG-6162 + neem	—	—	180	6.06a
ABG-6162	720	6.23 (0.126)a	180	5.87a
Neem	720	5.42 (0.156)b	180	5.18a
ABG-6162 + AVM	—	—	180	5.13a
AVM + neem	—	—	180	2.87b
Dipel 2X + AVM	—	—	180	2.83b
Dipel 2X + ABG-6162	—	—	180	2.82b
Dipel 2X + neem	—	—	180	2.54b
AVM	630	4.13 (0.151)c	180	2.41b
Dipel 2X	630	3.06 (0.252)d	180	2.34b

[a] Mean time in days. Means in columns followed by the same letter are not significantly different ($P > 0.05$; Duncan's [1955] multiple range test).

[b] Total number of larvae assayed.

[c] Mean time of mortality based on seven or eight concentrations used in probit analyses.

[d] Mean times based on LC_{25}'s replicated six times.

mortality), the MTM obtained from the AVM (LC_{25}) bioassay could have been artificially lowered (compared with MTM across all concentrations) by the low mortality rate (12.9%).

In trials with pesticide combinations, MTM's generally were distributed into two categories: if Dipel 2X was included, MTM was <3 d; and if ABG-6162 was included (excluding the combination with Dipel 2X), MTM was >5 d (Table 3). Because *B. thuringiensis* is thought to cause a separation of the midgut cells from the basement membrane resulting in leakage of gut contents into the hemocoel (Heimpel & Angus 1959), BAW larvae may have died faster in combination trials because of increased gut permeability, permitting an increase in the flow of the other compound across the gut membrane. Interactions of ABG-6162 with compounds other than Dipel 2X produced an MTM not significantly different ($P \geq 0.05$) from that of ABG-6162. The combination of AVM and neem also produced an intermediate MTM.

There is a need for standardization of procedures among laboratories for those bioassays requiring diet incorporation of toxicants. In comparing our results with those in the literature, it was evident that a short-term bioassay requires higher concentrations for equivalent mortality than bioassays that use longer time periods. Although most bioassays were reported in sufficient detail to allow accurate replication of the study, lack of standardization makes LC values less comparable between studies and thereby reduces the usefulness of the results. Such comparability becomes crucial when joint-action trials are performed; if both compounds are not tested for the same duration of time, comparisons of the MTM for the toxicant combination versus either of the toxicants singly become questionable. We suggest that, for joint-action trials, accurate results require that the minimum acceptable time for bioassay should be longer than the longest MTM for the toxicants selected. Otherwise, results may be based on mortality from an unrepresentative portion of the test population, as in mortality due only to one of the toxicants because of different MTM's.

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