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Effects of Two Carbamates on Infective Juveniles of *Steinernema carpocapsae* All Strain and *Steinernema feltiae* Umeå Strain

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Abstract: Laboratory bioassays were conducted to determine the effects of two carbamates, carbofuran (an acetylcholinesterase inhibitor) and fenoxycarb (a juvenile hormone analog), on survival and infectivity of the infective juveniles (IJ) of *Steinernema feltiae* Umeå strain and *Steinernema carpocapsae* All strain. Both insecticides caused mortality of IJ in a dose-related fashion. The two nematode species were equally sensitive to fenoxycarb (LD_{50} ca. 0.03mg/ml). Whereas IJ of *S. feltiae* were several orders of magnitude more sensitive to carbofuran ($LD_{50} \leq 0.2$ μg/ml) than to fenoxycarb, *S. carpocapsae* IJ displayed approximately the same degree of sensitivity to carbofuran (LD_{50} 0.01–0.03 mg/ml) as they did toward fenoxycarb. Toxicity of the carbamates was the same at all exposure periods from 24 to 168 hours' duration. Determinations of infective doses of nematodes required to cause 50% mortality of *Galleria mellonella* larvae showed that the infectivity of IJ that survived exposure to either of the two carbamates was not compromised by treatment.

Key words: acetylcholinesterase inhibitor, carbamate, carbofuran, entomopathogenic nematode, fenoxycarb, infective junvenile, insecticide, juvenile hormone analog, *Steinernema carpocapsae, Steinernema feltiae.*

In developing integrated pest management (IPM) strategies involving the use of entomopathogenic nematodes (f. Steinernematidae and Heterorhabditidae) and chemical pesticides, it is important to ascertain the degree to which these nematodes may be affected by the chemicals involved. Studies done using chemical insecticides that inhibit acetylcholinesterase activity have yielded inconsistent results, suggesting that it is not possible to generalize with respect to insecticide tolerance. In early laboratory studies, infective juveniles (IJ) of *Steinernema carpocapsae* were unaffected by short-term exposure to a wide variety of insecticides that were toxic to other soil-dwelling nematode species (4). When exposure time to the insecticides was increased beyond 24 hours, however, nematode mortality increased (5). Infective juveniles of the DD136 strain of *S. carpocapsae* were reported to be unaffected by treatment in the laboratory with a variety of insecticides and fungicides (2). However, the bioassay procedure used in the latter study was incomplete and measured only nematode mobility. Ishibashi (11) reported that soil applications of carbamate and organophosphate insecticides with *Steinernema carpocapsae* All strain actually increased the nematode's field efficacy against three species of larval Lepidoptera by stimulating nictating behaviour and infectivity of the IJ.

However, several carbamates and organophosphates adversely affected the in vitro development and reproduction of *S. carpocapsae* All strain, whereas the nematode was unaffected by the chlorinated hydrocarbon methoxychlor or the synthetic pyrethroid fenvalerate (9). Infective juveniles of this nematode displayed partial paralysis and loss of infectivity consequent to being treated with certain of these insecticides (10,14). In a subsequent study, it was determined that IJ of this strain of *S. carpocapsae* were refractory to a variety of carbamates but sensitive to certain organophosphates and the tertiary amine cartap (26). Laboratory bioassays involving exposure of IJ of an Italian strain of *S. carpocapsae* and an unspecified strain of *S. feltiae* to several categories of pesticides showed that while most of the herbicides and fungicides were not toxic, a high proportion

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of insecticides, acaricides, and nematicides induced adverse effects ranging from impaired movement and infectivity to death of the IJ (20). Heterorhabditids were determined to be more susceptible than steinernematids to pesticides (22), with *Heterorhabditis bacteriophora* and *Heterorhabditis heliothidis* displaying approximately the same overall intolerance but differing in their sensitivities to specific pesticides (21).

Due to environmental concerns, current trends are toward decreased reliance on conventional chemical insecticides. Juvenile hormone analogs (JHAs) are insecticidal compounds that offer a biorational approach to pest management since they specifically target the insect's neuroendocrine system and, thus, have minimal deleterious effects on non-target organisms (19). The mermithid nematode *Romanomermis culicivorax* was unaffected by the terpenoid methoprene, one of the first JHAs that was commercially available (6,15). However, the toxicological effects on entomopathogenic nematodes of JHAs, particularly more recently developed nonterpenoid ones, are unknown. The present study was done to determine the effects of two carbamates on the survival and infectivity of IJ of *S. carpocapsae* All strain and *S. feltiae* Umeå strain. Carbofuran is an acetylcholinesterase inhibitor widely used for control of soil-dwelling insects. Fenoxycarb is a recently developed JHA that, according to the supplier, does not inhibit acetylcholinesterase (Technical Data Sheet, 6th ed., 1989; Maag Agrochemicals, Vero Beach, FL).

Materials and Methods

Sources of nematodes: Steinernema carpocapsae All strain was provided by Plant Products Ltd., Brampton, Ontario, Canada. *Steinernema feltiae* Umeå strain was provided by R. West, Natural Resources Canada, Canadian Forest Service (CFS), St. John's, Newfoundland, Canada, from a stock colony that had been initially obtained from Biologic Biocontrol Products, Willow Hill, Pennsylvania, U.S.A. This nematode is marketed as *S. carpocapsae* Umeå strain and was first isolated from soil in Sweden (18). However, we have observed that its morphometric measurements correspond to those of *S. feltiae* (Jagdale and Gordon, unpublished observations). Restriction fragment length polymorphisms of amplified rDNA showed a close association between the Umeå strain and several *S. feltiae* strains (13). Therefore, we believe that the nematode being marketed as *S. carpocapsae* Umeå strain is improperly named and we have designated it as *S. feltiae* Umeå strain. Both nematode isolates were maintained at 25°C by propagation through larval waxmoths, *Galleria mellonella* (25).

Sources of insecticides: Technical-grade carbofuran was provided by FMC, Princeton, New Jersey, U.S.A., and technical-grade fenoxycarb by Elanco, Eli Lilly Canada Inc., Scarborough, Ontario, Canada.

Toxicity of insecticides to IJ: Nematode suspensions were prepared by using a disposable Millipore Filter (0.2 mμ pore diam.) to filter off the dilute formaldehyde solution collected from the White traps (25) in which IJ had emerged. The IJ that were trapped on the filter pad were subsequently washed twice by passing distilled water through the filter, then resuspended in distilled water. Using a stereomicroscope to count the IJ in 1-ml droplets of evenly stirred suspension and adjusting the water volume accordingly, the suspension was adjusted to a concentration of 50 IJ/ml. To allow for better dispersion, insecticide solutions were formulated using the emulsifier 0.01% Triton-X-100 (Sigma Chemical Co., St. Louis, MO). Concentrations of fenoxycarb were formulated such that after dilution with the nematode suspension, final concentrations were 0.001, 0.01, 0.1, 0.5, and 1.0 mg/ml. Final concentrations for carbofuran ranged from 0.00001 mg/ml to 0.01 mg/ml (*S. feltiae*) or from 0.00001 mg/ml to 0.05 mg/ml (*S. carpocapsae*). Equal volumes (10-ml) of nematode suspension and insecticide emulsion

were pipetted into glass petri dishes (60 × 15 mm). Treated and untreated controls were employed using 0.01% Triton-X-100 and distilled water, respectively, in place of the insecticide emulsion.

Nematodes were maintained in an incubator (24°C) in complete darkness. Toxicity was determined after 24-, 72-, 120-, and 168-hour exposure to insecticide by removing 1 ml of suspension containing at least 25 IJ from the appropriate petri dishes and probing each nonmotile nematode in the sample with a lachrymal needle under a stereomicroscope to determine whether it was alive or dead (10). This was necessary because IJ normally display the active or inactive behavioral states. Separate petri dishes containing nematodes and insecticides were set up for each time interval; there were three replicates for each concentration at each time interval.

Effects of insecticides on infectivity of IJ: Infectivity was measured by comparison of LD_{50} values of insecticide treated and untreated (controls) IJ at a fixed insecticide concentration. Since sublethal effects were being monitored, insecticide concentrations were selected that, according to the toxicity studies, resulted in the lowest mortality to IJ. Thus, IJ of each isolate were exposed to 0.001 mg/ml fenoxycarb (about 15% All; <5% *S. feltiae* IJ mortality) or 0.00001 mg/ml carbofuran (about 40% *S. feltiae;* <5% *S. carpocapsae* IJ mortality) for 24 hours and 168 hours, as in the toxicity experiments. A follow-up sequence of experiments was carried out using a higher concentration (0.1 mg/ml) of insecticides and a 168-hour exposure period. Such high-dose studies were not attempted with carbofuran against *S. feltiae* because this species was so sensitive to the compound that the IJ would not have survived treatment. At each exposure time, insecticide was removed from the IJ by Millipore filtration and IJ were then resuspended in distilled water. Nematodes were bioassayed against last instar *G. mellonella.* Using an Eppendorf micropipet, live IJ were removed individually from the nematode suspension under a stereomicroscope and transferred to filter-paper circles lining the bases of disposable petri dishes (60 × 15 mm); it was possible to count the live IJ as they were removed. Doses of 10, 30, 50, and 70 IJ were used and distilled water (≤0.2 ml) added until the filter papers were evenly moist. Ten insects were added to each petri dish, which was closed and incubated in darkness at 24 °C. Thus, doses were 1, 3, 5, and 7 IJ/insect, with five replications of each dose. Insect mortality was determined 5 days after infection; all dead insects were dissected to verify that they harbored steinernematids.

Statistical analysis: In the IJ toxicity studies, the effects of the compounds (% IJ mortality) were determined by allowing for mortality in the controls using Abbott's formula (1). Probit analysis (17) was used to calculate LD_{50} values on $\log_{10}(X + 1)$ transformed data. The same procedure was used to calculate LD_{50} values in the infectivity studies, except that no control correction was necessary as there was no mortality in uninfected insects. In both toxicity and infectivity studies, significant differences between pairs of LD_{50} values were based on the criterion of non-overlap of 95% fiducial limits. To ascertain whether the duration of the incubation time of the IJ affected their infectivity, infectivity LD_{50} values for 24-hour incubated IJ of both nematode isolates, controls and treated, were grouped together and compared to an analogous composite group of 168-hour maintained IJ using a Wilcoxon two-sample nonparametric test (16).

Results

Toxicity of insecticides to IJ: Both insecticides caused death of the IJ. No partial paralysis or other aberrant patterns of locomotory behavior were observed in live treated IJ. The toxicity of the two insecticides was dose related (Figs. 1,2). The two isolates displayed the same level of sensitivity toward fenoxycarb, as evidenced by their comparable LD_{50} values (around 0.03 mg/ml, Table 1). *Steinernema feltiae* was

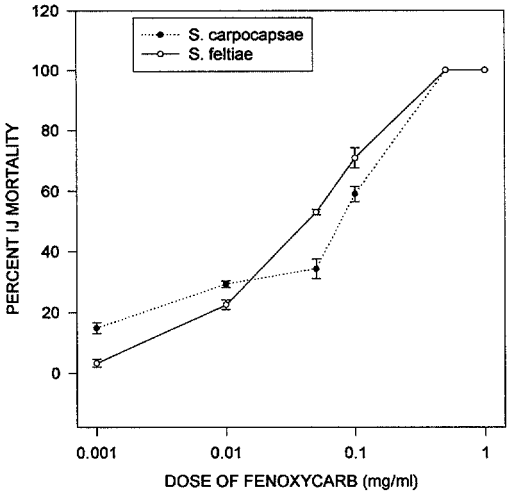

FIG. 1. Dose-response curves, semi-logarithmic plot, for infective juveniles of *Steinernema carpocapsae* and *Steinernema feltiae* treated with fenoxycarb. Since the effect was not time related, mortalities after 24, 72, 120, and 168 hours' treatment were grouped together for each insecticide dose and mean values obtained. Graph shows mean effect of compound, measured as percentage IJ mortality ± SD, as determined by Abbott's formula.

much more sensitive to carbofuran than to fenoxycarb, as its LD_{50} value for fenoxycarb was 2 to 3 orders of magnitude higher than for carbofuran (≤0.2 μg/ml). *Steinernema carpocapsae* IJ showed sensitivity to carbofuran, of the same order of magnitude as to fenoxycarb. Susceptibility to the insecticides was not affected by exposure times in excess of 24 hours, as LD_{50} values for each isolate were the same at all exposure times examined (Table 1).

Effects of insecticides on infectivity of IJ: Neither of the insecticides affected the infectivity of IJ that survived treatment. At the lowest doses used in the toxicity studies (0.01 μg/ml carbofuran; 0.001 mg/ml fenoxycarb), the LD_{50} values for insecticide-treated IJ (24- or 168-hour exposure periods) was the same as in the corresponding controls (Table 2). Using a higher dose of insecticides (0.01 mg/ml) did not alter this pattern. LD_{50} values varied slightly from one cohort of IJ to another. However, in both treated and control IJ, infectivity decreased during incubation, as LD_{50} values of those that had been incubated for 1 week prior to infection were higher than values obtained for 24-hour incubated ones ($P = 0.012$).

Discussion

Carbofuran and fenoxycarb, two carbamate insecticides with different modes of action, were toxic, to varying degrees, to the two species of steinernematids used in this study. No evidence supported the unusual proposition that certain carbamates may be beneficial in IPM by enhancing nictating behavior and, consequently, infectivity of the IJ (11,12). A nictation substrate was provided by the moist filter-paper lining the petri dish bases on which infectivity studies were carried out; however, infectivity of treated IJ was the same as untreated ones. Also, while carbofuran appeared to stimulate locomotory behavior, nematodes that had been treated with either of the two carbamates were either killed or continued to execute normal patterns of movement, unlike those that were stimulated to nictate by insecticides and which displayed a variety of aberrant be-

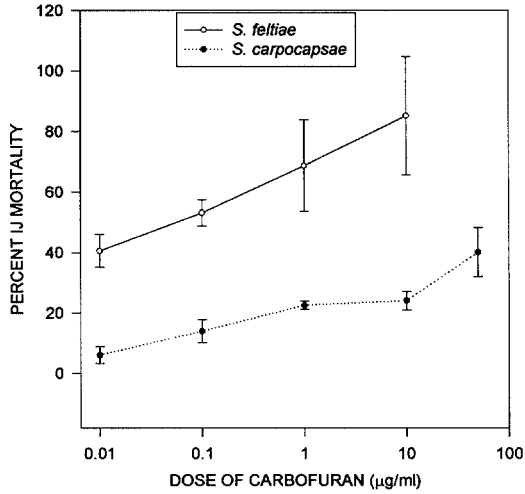

FIG. 2. Dose-response curves, semi-logarithmic plot, for infective juveniles of *Steinernema carpocapsae* and *Steinernema feltiae* treated with carbofuran. Since the effect was not time related, mortalities after 24, 72, 120, and 168 hours' treatment were grouped together for each insecticide dose and mean values obtained. Graph shows mean effect of compound, measured as percentage IJ mortality ± SD, as determined by Abbott's formula.

TABLE 1. Probit analysis of the lethal effects of carbofuran and fenoxycarb on *Steinernema feltiae* and *Steinernema carpocapsae*.

Nematode species	Exposure time (hour)	Slope ± SE	LD_{50} (mg/mL)	Confidence interval
		Fenoxycarb		
S. feltiae	24	1.301 ± 0.093	0.029	0.012–0.060
	72	1.340 ± 0.094	0.029	0.011–0.066
	120	1.325 ± 0.094	0.028	0.012–0.055
	168	1.316 ± 0.094	0.025	0.010–0.052
S. carpocapsae	24	0.953 ± 0.068	0.026	0.001–0.204
	72	0.981 ± 0.069	0.027	0.002–0.199
	120	0.983 ± 0.069	0.032	0.002–0.260
	168	0.927 ± 0.067	0.026	0.001–0.200
		Carbofuran		
S. feltiae	24	0.253 ± 0.061	0.00022	0.0001–0.0008
	72	0.553 ± 0.070	0.00003	0.00000–0.00039
	120	0.405 ± 0.064	0.00003	0.00001–0.00005
	168	0.530 ± 0.071	0.00008	0.00003–0.00016
S. carpocapsae	24	0.514 ± 0.037	0.011	0.001–0.198
	72	0.524 ± 0.037	0.017	0.001–0.543
	120	0.48 ± 0.04	0.029	0.002–18.108
	168	0.62 ± 0.04	0.010	<13.180

havior patterns in purely aqueous films (11,12). It seems unlikely that, at the ranges of concentrations deployed, the carbamates tested in this study affected the nematodes in a positive fashion.

The species, and possibly strain, of nematode appears to be of crucial significance in determining its level of susceptibility to carbamate insecticides that inhibit acetylcholinesterase activity. *Steinernema feltiae* Umeå strain was highly sensitive to carbofuran, whereas *S. carpocapsae* All strain was several orders of magnitude less sensitive. Such relative insensitivity of *S. carpocapsae* All strain IJ to acetylcholinesterase inhibitors accords with observations made by Zhang et al. (26), who reported no toxic effects of several carbamates (not including carbofuran) and minimal effects of a variety of organophosphates. Although detrimental effects on the IJ (10) and on in vitro reproduction (9) have been reported, relatively high (≥0.1 mg/ml) concentrations were required to bring these about. Because the current study showed the infectivity of IJ that survived insecticide exposure was unaffected by the treatment, it seems reasonable to propose that *S. carpocapsae* All strain, but not *S. feltiae* Umeå strain, could be used in IPM involving the use of carbofuran, provided that the operative field doses of the insecticide were sufficiently low to avoid significant nematode mortality.

Carbamates and organophosphates were found to kill a proportion of the IJ of *S. carpocapsae* All and cause partial paralysis and reduced infectivity of the remainder (10). Gaugler and Campbell (7) showed that the carbamate oxamyl stimulated locomotory movement of the IJ at concentrations less than 50 μg/ml, but induced partial paralysis at higher concentrations. Impairment of infectivity of IJ of this nematode was found to occur following exposure to several categories of insecticides, but not after treatment with carbamates (26). The IJ of an Italian strain of *S. carpocapsae,* an unspecified strain of *S. feltiae* (20), and two species of *Heterorhabditis* (21,22) were killed by a variety of insecticides; sublethal effects included loss of mobility and infectivity. The organophosphate pesticide, fenamiphos, killed IJ of the *S. carpocapsae* All strain and compromised the infectivity of those that survived treatment (14). In the current study, however, sublethal effects of treatment were not noticeably manifested with respect to either of the compounds. Nematodes that

TABLE 2. Probit analysis of the effects of fenoxycarb and carbofuran on infectivity of *Steinernema feltiae* and *Steinernema carpocapsae*.

Nematode species	Treatment	Exposure time (hour)	Slope ± SE	LD_{50} (IJ/host)	95% CL
		Fenoxycarb[a]			
S. feltiae	Treated	24	1.40 ± 0.31	0.89	0.34–1.39
	Controls	24	1.00 ± 0.30	0.60	0.06–1.19
	Treated	168	0.97 ± 0.28	2.28	1.07–3.49
	Controls	168	1.43 ± 0.29	2.54	1.73–3.40
	Treated HD	168	1.03 ± 0.29	2.77	1.60–4.25
	Controls	168	1.27 ± 0.29	2.93	1.98–4.13
S. carpocapsae	Treated	24	1.84 ± 0.31	1.50	0.98–1.96
	Controls	24	1.34 ± 0.30	1.27	0.60–1.85
	Treated	168	1.82 ± 0.31	2.32	1.71–2.93
	Controls	168	2.26 ± 0.32	2.24	1.75–2.74
	Treated HD	168	0.69 ± 0.28	4.93	2.69–42.22
	Controls	168	0.79 ± 0.29	5.91	3.52–34.55
		Carbofuran[a]			
S. feltiae	Treated	24	0.98 ± 0.29	1.96	0.82–2.98
	Controls	24	1.40 ± 0.29	1.95	1.20–2.66
	Treated	168	0.99 ± 0.29	2.45	1.26–3.72
	Controls	168	0.86 ± 0.28	2.90	1.43–5.04
S. carpocapsae	Treated	24	1.75 ± 0.30	1.81	1.23–2.35
	Controls	24	1.70 ± 0.30	1.97	1.36–2.55
	Treated	168	1.50 ± 0.30	2.29	1.55–3.04
	Controls	168	1.41 ± 0.29	2.20	1.42–2.96
	Treated HD	168	1.26 ± 0.30	5.61	4.00–10.36
	Controls	168	1.23 ± 0.30	4.97	3.54–8.71

[a] Infective juveniles were exposed (168 hours) to 0.001 mg/ml fenoxycarb or 0.00001 mg/ml carbofuran, except for "Treated HD," which were exposed to 0.1 mg/ml of either insecticide.

survived insecticide treatment executed normal movement and were able to infect hosts as well as those that had not been treated. The range of carbofuran doses used (0.01 to 50 μg/ml) was below that used by other researchers, who reported adverse locomotory effects consequent to treating IJ with the carbamates oxamyl or methomyl (7,10-12). However, the concentrations of fenoxycarb (1 to 10^3 μg/ml) did span the range within which such locomotory effects would have been predicted.

No time-related response was observed for any of the nematode-insecticide combinations (i.e., the effect of the insecticides occurred within the initial 24-hour exposure period and did not increase thereafter). This may indicate a genetic variability within the populations of two nematodes with respect to insecticide tolerance. A proportion of the nematodes were susceptible to the insecticides and were killed within 24 hours' exposure, whereas the remainder of the population were refractory and hence unaffected by prolonged exposure. If validated, such a refractory subpopulation could be subjected to genetic selection, allowing the establishment of a more insecticide-tolerant strain for use in IPM. Clearly, the action of these carbamates on the two nematode isolates is different from that reported for *S. carpocapsae* (unspecified strain), in which toxicity of carbamates increased after 24 hours' exposure (5).

The fact that the two nematodes were equally sensitive to fenoxycarb (albeit three orders of magnitude less than to carbofuran in *S. feltiae* Umeå) is best explained as the consequence of effects of the compound other than the induction of endogenous juvenile hormone sythesis, as occurs in JHA-treated insects. The rapidity of death (<24 hours) is not suggestive of disrupted development resulting from endocrinological dysfunction. Moreover,

although juvenile hormones have been recorded from nematodes, there is no reason to suppose that control of the fundamentally different process of development in nematodes would parallel that of insects (3). Earlier studies on the insect-parasitic nematode *Romanomermis culicivorax* showed that the IJ of this nematode were tolerant to the terpenoid JHA methoprene (6,15). The more recently developed JHAs, many of which are chemically unrelated to natural juvenile hormone, must be viewed in a different light. From the present study, it would appear that conjoint usage of carbamate JHAs with steinernematids in IPM may require applying the chemical and biological control agents in sequence, separated by a time interval that would be sufficient to minimize the toxicity effects on the nematode caused by high levels of the JHA in the soil.

In addition to their use in insect pest management, certain carbamates may be used to control plant-parasitic nematodes (8,23,24). The present findings suggest a cautious approach to such deployment, to minimize possible negative impact upon natural or introduced entomopathogenic nematodes in the soil.

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