Oral Toxicity of Chlordane, Hydramethylnon, and Imidacloprid to Free-Foraging Workers of *Camponotus pennsylvanicus* (Hymenoptera: Formicidae)

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J. Econ. Entomol. 86(6): 1730–1737 (1993)

ABSTRACT The oral toxicity of the delayed-action insecticide hydramethylnon, in contrast to the acute toxins chlordane and imidacloprid, was evaluated in free-foraging workers of the black carpenter ant, *Camponotus pennsylvanicus* (DeGeer), gathering insecticide-laced, sugar–milk baits. Hydramethylnon was slower acting than either chlordane or imidacloprid. When workers fed upon 500-ppm baits, hydramethylnon killed ants significantly more slowly (LT_{50} [95% CL] = 12.7 [12.5–12.9] d) than did chlordane (1.3 [1.1–1.4] d) or imidacloprid (0.3 [0.1–0.5] d). All toxicants were transferred via trophallaxis (i.e., indirect exposure). In ants exposed to a single forager that fed upon a 500-ppm bait (i.e., the donor ant), hydramethylnon caused death significantly more slowly (LT_{50} [95% CL] = 12.2 [11.9–12.4] d) than did chlordane (4.1 [3.8–4.4] d) or imidacloprid (0.9 [0.8–1.0] d). In both direct and indirect exposures, significant concentration-dependent time delays before mortality were observed with each toxin. Donor ants in indirect exposures survived after gathering and transferring sugar–milk bait laced with chlordane or hydramethylnon but did not survive their exposure to imidacloprid. On the basis of these and other analyses, we identify delayed-action toxins suitable for carpenter ant baits.

KEY WORDS *Camponotus pennsylvanicus*, delayed-action toxicity, toxic baits

THE CARPENTER ANTS, *Camponotus* spp., are increasingly important pest ants throughout the United States and Canada. Once considered a mere nuisance (Fowler 1990), their complicity in damaging wood in service is as significant as their damage to utility poles (Snyder 1910), urban shade trees (Fowler & Roberts 1982), and merchantable timber (Akre & Hansen 1990). Even when they are not causing structural damage, carpenter ants remain unacceptable nuisances in and around urban structures (Dukes 1989). Carpenter ants may account for nearly 40% of all ant control performed by the U.S. structural pest control industry (NuVentures 1992), and Fowler (1986) estimated that $25 million is expended annually in the United States for their control.

Current control measures for carpenter ants rely on chemical and nonchemical techniques, in which ultimate success is largely dependent upon first locating and then destroying the entire colony (Akre & Hansen 1990). This is, at best, difficult to achieve because of their nocturnal nature and their cryptic and polydomous nesting habits. Toxic baits, which are very effective for numerous pest ants (Rust 1986), would obviate the need to locate the nest by exploiting the ants' foraging and recruitment behavior to introduce insecticide into the colony. Baits containing the delayed-action toxicant dechlorane (mirex) were very successful in ant control and, until recently, were still being used in some tropical regions to control leaf-cutting ants. After cancellation of all mirex registrations by the Environmental Protection Agency (Johnson 1976), a number of delayed-action toxicants (e.g., abamectin, hydramethylnon, and sulfluramid) have been developed for bait applications against fire ants (Lofgren & Williams 1982; Williams et al. 1980, 1987). These compounds are now being applied in baiting systems for use against other insects.

Research is underway at many commercial and academic laboratories to develop baits for carpenter ants. To support dosing considerations in developing toxic baits for carpenter ants, we have conducted a series of oral toxicity studies with the black carpenter ant, *C. pennsylvanicus* (DeGeer). We have previously reported results for the delayed-action toxicants dechlorane (mirex) and sulfluramid and the more acutely

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acting abamectin (Reid & Klotz 1992). Here we evaluate the toxicity of hydramethylnon to free-foraging workers of *C. pennsylvanicus* and contrast its activity with an experimental bait toxin, imidacloprid (NTN33893). We also examined chlordane, a historically significant toxicant in ant control (Mallis 1964), because its delayed toxicity is not concentration dependent (Matsumura 1985, Su et al. 1987) and would provide a unique intermediate between delayed-action and rapid acting toxicants.

Materials and Methods

Our studies were conducted during the summer of 1992, and all field work was conducted from 2000 to 2400 hours (EST). Numerous *C. pennsylvanicus* colonies were found nesting in live trees on the grounds of the Indiana Veterans' Home, 1 km N of West Lafayette. Methods used to entice the ants to forage at our feeding stations and the procedures used to expose them to our sugar–milk baits were described in greater detail previously (Reid & Klotz 1992). In brief, the procedures involved training ants from a tree nesting colony to forage at a feeding platform at the terminus of a wooden runway. At this platform we exposed worker ants to a dish of toxic sugar–milk, confining them as they gorged on this bait, and transferred them to a plastic box (7 by 5 by 3.5 cm) after they had fed to repletion (see Fig. 1 in Reid & Klotz 1992). In primary toxicity tests, we gathered ≈30 replete, dosed ants (≈10 ants from each of three colonies). Secondary toxicity tests assessed the effect of social dilution (i.e., trophallaxis) on our liquid bait. Ants were exposed, as described above, until 5 to 10 "donor" ants were dosed; donor ants received a paint mark (yellow gloss enamel, Testor, Rockford, IL) on the abdominal dorsum. Ants from the same colony(s) were collected as they were coming to the feeding station (they had not fed on the bait). These "recipient" ants were placed, in groups of 10, into 3 boxes with 1 dosed, replete donor ant.

All toxic sugar–milk exposures were concluded by mid- to late July to avoid a seasonally variable survivorship of the workers observed in 1991, wherein workers collected in August survived longer than ants that were collected in June (Reid & Klotz 1992). During the 1992 testing program, we collected ants gathering on toxic sugar-milk in mid-June, mid-July, and mid-August to examine formally this seasonal variation in survival. Direct ingestion tests (controls for primary toxicity tests) were conducted at all three time periods, whereas an indirect ingestion test (the control for secondary toxicity tests) was conducted in mid-July.

Technical chlordane (99.0%, Velsicol, Rosemont, IL) and hydramethylnon (98.0%, Clorox, Pleasanton, CA) were diluted in acetone to a concentration of 50 mg/ml, and imidacloprid (95.0%, Miles, Kansas City, MO) was diluted in acetone to 37.5 mg/ml; ≈50 mg of Tween 80 (Sigma, St. Louis, MO) was added for every 25 ml of solution to enhance uniform mixing in our sugar–milk bait. Experimental dilutions were prepared by adding an adequate volume of the acetone/AI/Tween 80 solution to a given weight of the sugar–milk bait (15 g raw sugar dissolved in 50 ml whole milk). Fresh sugar–milk dilutions were prepared daily and transported to the field in ice chests for each evening's test session. Seven concentrations, spanning a 100-fold dilution, were tested for each toxicant in the primary toxicity tests: chlordane and imidacloprid, 1,000, 500, 250, 100, 50, 25, and 10 ppm; and hydramethylnon, 10,000, 5,000, 2,500, 1,000, 500, 250, and 100 ppm. We established these ranges so that the highest concentration tested possessed a slight repellency defined as differential behavior (feeding hesitancy) at toxic baits of differing concentrations. Secondary toxicity tests examined select concentrations from the primary toxicity tests (chlordane and imidacloprid: 500, 250, and 50 ppm; hydramethylnon: 5,000, 2,500, and 500 ppm). These were selected to evaluate three concentrations in a 10-fold dilution, starting with the highest concentration that the ants would readily feed upon (no behavior was observed to suggest feeding deterrence).

In the laboratory (25 ± 5°C, ambient RH) the ants were provided a moistened dental wick and observed for cumulative moribundity and eventual mortality at ≈12-h intervals until all ants died; dental wicks were moistened daily. Median lethal times (LT_{50}s) for each toxicant concentration were estimated by regression analysis of the cumulative probit mortality versus the $\log_{10}$ of time (d); nonoverlapping 95% CL defined significant differences. Probit analysis of primary toxicity data was used to generate graphic interpretation of each toxicant's delayed action based on the effective lethal time to 90% of the population (ELT_{90}), after the manner suggested by Su et al. (1987). In secondary toxicity tests, only recipient ants were considered in probit analysis, whereas survival of donor ants was analyzed in two ways. The mean day of death in each toxicant concentration ($n = 3$) was compared, by a two-tailed t test ($P = 0.05$) with a control group ($n = 3$) of marked, donor ants collected at untreated sugar–milk. Mean death times of donor ants at each of the concentrations were also analyzed in a completely randomized, one-way analysis of variance (ANOVA) for each toxicant, followed by least significant differences (LSD) ($P = 0.05$) to test for concentration dependence in survival of the donor ants. SAS procedures (SAS Institute 1990) were used for all analyses.

Table 1. Seasonal variation in survival (i.e., lethal time to 50%) of *C. pennsylvanicus* workers following direct or indirect ingestion of nontoxic sugar–milk baits

Date	Direct ingestion				Indirect ingestion			
	n^a	Slope ± SE	$LT_{50}{}^b$ (95% CL)	χ^{2c}	n^a	Slope ± SE	$LT_{50}{}^b$ (95% CL)	χ^{2c}
16 June 1992	600	10.33 ± 0.91	17.89 (17.39–18.34)	9.0	—	—	—	—
17 July 1992	510	10.35 ± 0.98	17.89 (17.14–18.62)	23.3*	420	7.75 ± 0.71	7.09 (6.77–7.41)	17.0
14 Aug. 1992	660	6.69 ± 0.55	20.66 (19.82–21.45)	6.1	—	—	—	—

See text for elucidation of methods implied by the terms direct and indirect ingestion.
[a] Number of observations included in this regression analysis.
[b] Number of days before death following ingestion of nontoxic sugar–milk bait.
[c] Pearson's χ^2. All P values >0.10 except: *P = 0.08 and, because of heterogeneity, a t value of 2.13 instead of 1.96 was used to calculate 95% CL.

Results and Discussion

After ingesting nontoxic bait, ant survival times were equal in June and July because the median time of death (LT_{50}) was approximately 18 d at either date (Table 1). This compares favorably with our 1991 estimated median survival time in control ants (LT_{50} [95% CL] = 16.8 [16.6–17.1]; Reid & Klotz 1992). However, a different survival curve was described from ants collected in August 1992, when the median time of death (LT_{50}, ≈21 d) was significantly greater than in June or July (Table 1). The practical significance of this finding is that this, or similar, field bioassays should be conducted in as narrow a time frame as possible so that seasonally variable survival does not confound toxicant-mediated survival. The more heterogeneous distribution of survival times in August indicates the existence of a sub population in each colony which survives longer than do ants collected in either June or July. These results support our speculation (Reid & Klotz 1992) that the worker caste is dominated by overwintered workers in the spring (Pricer 1908, Sanders 1964, Cannon 1990) whereas, later in the season, newly emerged workers begin to forage.

In contrasting direct and indirect ingestion of nontoxic bait, the consequence of receiving reduced amounts of liquid bait by trophallaxis is dramatic. When all ants were allowed to feed to repletion (i.e., direct ingestion), they survived a median of 17.9 d, yet when 10 unfed ants were exposed to a single replete worker (i.e., donor ant), ants receiving sugar–milk bait indirectly survived for only 7.1 d (Table 1). The mean survival time for the donor ants, 7.0 ± 1.3 (mean ± SE) d, was equivalent to the survival time (LT_{50}) of the recipient ants. Traniello (1977) found with *C. pennsylvanicus* that a donor ant would transfer >95% of a single crop load of liquid bait to nestbound cohorts. In the laboratory, through mutual exchange of liquid foods within the group (i.e., the group effect [Gosswald & Kloft 1963]), the sugar–milk bait was likely to be evenly distributed among the donor and recipient ants, thus explaining the equal survival times. From a practical perspective, differing survival times in the two bioassays stress the need to compare toxicity results from either the primary or secondary toxicity bioassay with the appropriate control (i.e., direct or indirect exposure, respectively). This was overlooked in our earlier work (Reid & Klotz 1992), where we failed to conduct controls for the secondary toxicity tests; the ramifications of this are discussed later in this article.

Toxicants examined in this study differ in mode of action: chlordane promotes transmitter release at synapses of the central nervous system (Matsumura 1985), hydramethylnon inhibits mitochondrial electron transport (Hollingshaus 1987), and imidacloprid is an agonist at nicotinic acetylcholine receptors in the postsynaptic regions of neuromuscular junctions (Bai et al. 1991). Accordingly, moribund behavior (e.g., ataxia, excitation, lethargy) varied greatly among these compounds. Given the qualitative, subjective nature of our operational definition for moribund behavior, these data were judged to be too imprecise for analysis; thus, mortality data were analyzed for comparative toxicity. However, the behavioral impairment of moribund individuals would render them functionless in the social context of the colony. Thus, LT_{50}s estimated on mortality in the primary toxicity study overstate the functional survival of poisoned ants by a few hours or days, depending on the toxicant's mode of action. With the respiratory poison hydramethylnon, there was generally a 3–4-d delay between the onset of moribundity and eventual mortality, whereas with the nerve poisons chlordane and imidacloprid, this delay was <1 d (Fig. 1).

In the primary toxicity test, with each dilution of toxicant concentration in the sugar–milk baits, there was a corresponding increase in the LT_{50} values (Table 2); lone exceptions were from 250 to 100 ppm hydramethylnon and from 50 to 25 ppm imidacloprid. All concentrations of hydramethylnon in the primary toxicity tests caused significant reductions in survival time (compare treatment LT_{50}s [Table 2] to control

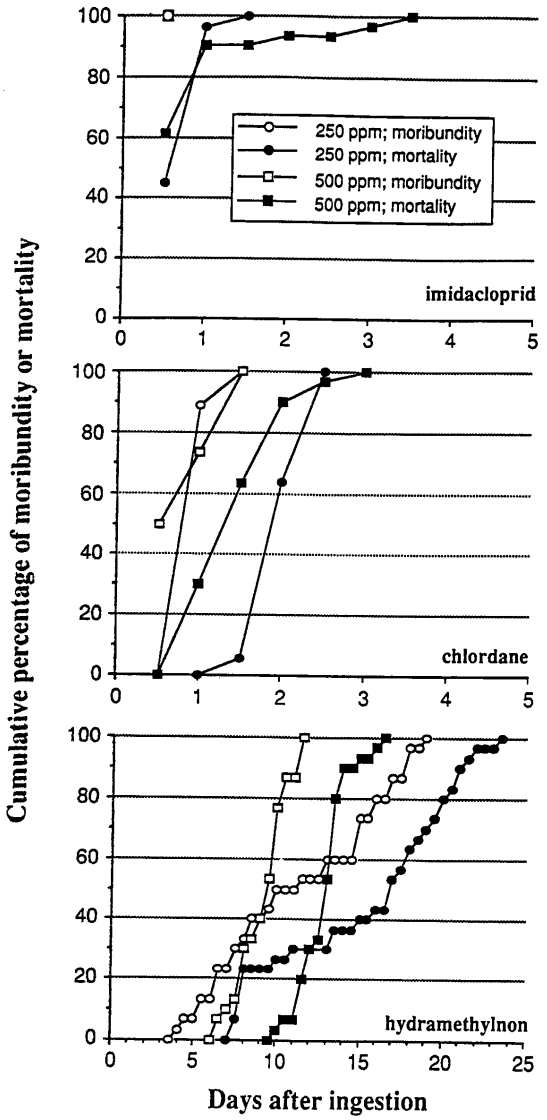

Fig. 1. Relation of moribundity to mortality, illustrating toxicant-dependent delays between the time ants were first affected by a toxicant and their eventual death. Note different scales for X axis between chlordane and imidacloprid (5 d) and hydramethylnon (25 d).

LT_{50}s in June or July [Table 1]). Although significantly reducing the survival time of the ants at higher concentrations, low concentrations of chlordane (25 and 10 ppm) and imidacloprid (10 ppm) were not lethal to the ants in these tests. Ants ingesting hydramethylnon, at any concentration, did not die until (i.e., LT_{50}s) 5–14 d after exposure, demonstrating its delayed action toxicity (Hollingshaus 1987). Ants ingesting chlordane died from (i.e., LT_{50}s) 1–3 d after exposure (1,000 to 50 ppm), whereas ants ingesting imidacloprid were very rapidly intoxicated (Fig. 1) and died (i.e., LT_{50}s) within 1 d, except at 25 and 50 ppm imidacloprid when ants survived for 2 or 3 d, respectively. Based on these results, we conclude that only hydramethylnon possesses the concentration-dependent, delayed-action toxicity considered a prerequisite to successful baiting of social insects (Banks 1990, Stringer et al. 1964). Chlordane and imidacloprid might intoxicate or kill workers too fast to promote recruitment to baits, which would limit the continuous infusion of toxic bait into the colony.

The LT_{50}s estimated from the secondary toxicity tests (Table 2) show that the quantity of hydramethylnon in 5,000, 2,500, and 500 ppm sugar–milk bait received by the recipient workers, after being diluted by trophallaxis with the donor ant, was not lethal (compare treatment LT_{50}s to control LT_{50} in Table 1). Neither the 250- nor 50-ppm chlordane baits were lethal after dilution through trophallaxis, but at 500 ppm chlordane and at all three concentrations of imidacloprid, the survival of recipient ants was significantly reduced (compare treatment LT_{50}s to control LT_{50} in Table 1). The fate of donor ants in the secondary toxicity tests reveal a pattern similar to that seen with recipient ants. At all concentrations of hydramethylnon and chlordane, and at 50 ppm imidacloprid, the mean survival time of the donor ants was not reduced significantly from that observed for donors in control tests (Table 3). Transfer of toxic sugar–milk bait from donor ants to recipient ants rendered benign what should have been (given the primary toxicity data in Table 2) fatal toxicant exposures in donor ants gathering either chlordane or hydramethylnon. However, there was no such effect for imidacloprid because donor survival at 500 and 250 ppm was significantly reduced (Table 3). Dose-dependence in survival of donor ants in the secondary toxicity tests was significant only for hydramethylnon (ANOVA, $F = 105.00$; df = 2, 6; $P < 0.01$); mean separations of survival times may be referenced in Table 3. Survival of donor ants did not vary with concentrations of chlordane ($F = 0.77$; df = 2, 6; $P = 0.51$) and imidacloprid ($F = 2.48$; df = 2, 6; $P = 0.16$) in the sugar–milk baits.

Data from the secondary toxicity trial (Tables 2 and 3) indicated that trophallaxis rendered hydramethylnon nontoxic to both donor and recipient ants involved in the mutual exchange of toxic sugar–milk. If we reexamine the secondary toxicity data in our earlier article (Reid & Klotz 1992) where we failed to conduct a proper control it can be shown that, after trophallaxis, exposures to either dechlorane or sulfluramid were likely not fatal in the donors and recipients. These results suggest the generalization that, if a compound possesses concentration-dependent delayed-action toxicity, trophallaxis acts to attenuate toxic bait exposures in foragers, thus promoting long-term bait collection which results in the accumulation of toxicant in the colony, where

Table 2. Estimates of lethal time to 50% of *C. pennsylvanicus* workers following direct (primary toxicity) or indirect (secondary toxicity) ingestion of toxic sugar-milk baits

Conc, ppm	Primary toxicity				Secondary toxicity			
	n^a	Slope ± SE	$LT_{50}{}^b$ (95% CL)	χ^{2c}	n^a	Slope ± SE	$LT_{50}{}^b$ (95% CL)	χ^{2c}
Chlordane								
1,000	160	7.29 ± 1.09	1.19 (1.06– 1.30)	2.3	—	—	—	—
500	180	6.37 ± 0.87	1.27 (1.12– 1.40)	1.0	360	5.06 ± 0.49	4.08 (3.80– 4.38)	3.4
250	144	17.57 ± 2.74	1.88 (1.79– 1.97)	1.0	510	8.16 ± 0.64	9.14 (8.82– 9.48)	10.8
100	429	3.63 ± 0.46	2.52 (2.11– 2.92)	22.8*	—	—	—	—
50	297	8.55 ± 0.82	2.91 (2.74– 3.07)	6.6	690	3.82 ± 0.32	7.17 (6.68– 7.63)	12.3
25	672	6.87 ± 0.58	17.61 (17.00–18.23)	3.7	—	—	—	—
10	496	9.54 ± 1.17	24.07 (23.39–24.92)	10.4	—	—	—	—
Hydramethylnon								
10,000	493	6.54 ± 0.51	5.38 (5.08– 5.65)	11.0	—	—	—	—
5,000	462	10.03 ± 0.78	6.38 (6.15– 6.60)	5.5	570	8.20 ± 0.58	8.23 (7.93– 8.53)	5.0
2,500	570	6.76 ± 0.55	6.94 (6.56– 7.28)	18.0	450	11.54 ± 0.90	7.95 (7.70– 8.19)	18.4
1,000	690	6.09 ± 0.44	10.66 (10.23–11.09)	18.9	—	—	—	—
500	420	20.72 ± 1.63	12.68 (12.45–12.91)	7.4	595	13.61 ± 0.95	12.17 (11.90–12.44)	3.0
250	990	5.18 ± 0.46	14.45 (13.66–15.27)	58.8*	—	—	—	—
100	390	18.45 ± 1.59	13.40 (13.14–13.66)	7.4	—	—	—	—
Imidacloprid								
1,000	30	—	<0.5[d]	—	—	—	—	—
500	217	2.03 ± 0.43	0.33 (0.12– 0.51)	2.5	150	4.74 ± 0.64	0.88 (0.75– 1.01)	2.5
250	87	6.61 ± 1.56	0.52 (0.43– 0.60)	0.1	300	2.06 ± 0.27	1.87 (1.52– 2.23)	2.0
100	330	2.11 ± 0.27	0.89 (0.64– 1.13)	2.1	—	—	—	—
50	728	1.38 ± 0.15	3.53 (2.86– 4.19)	15.7	1,020	2.25 ± 0.19	5.02 (4.33– 5.71)	55.6*
25	279	2.78 ± 0.37	2.43 (2.11– 2.79)	4.3	—	—	—	—
10	1056	3.87 ± 0.36	20.23 (19.30–21.31)	6.3	—	—	—	—

See text for explanation of exposure methods implied by the terms primary and secondary toxicity.

[a] Number of observations included in this regression analysis.

[b] Number of days before death following ingestion of toxicant diluted in sugar–milk bait.

[c] Pearson's χ^2. All P values >0.10 except: *$0.002 \leq P \leq 0.019$ and, because of heterogeneity, t values of 2.04–2.20 instead of 1.96 were used to calculate 95% CL.

[d] All ants died before the first observation at 12 h, thus data were not analyzed by probit regression.

it later exerts its toxic effects. In contrast, data for chlordane and imidacloprid (Tables 2 and 3), and by extrapolation from Reid & Klotz (1992), the data for abamectin indicate this attenuating effect of trophallaxis is compromised by rapidly acting toxicants. At high concentrations, chlordane (500 ppm), abamectin (500 ppm), and imidacloprid (500 and 250 ppm) reduced the survival of recipient ants as well as the survival of the donor ants. At lower concentrations (chlordane and abamectin at ≤250 ppm or imidacloprid at 50 ppm) there was only a slight attenuation of lethal toxicant exposures by trophallaxis. This attenuating effect would be active only in a narrow range of concentrations because each of these acute toxins were readily diluted to ineffective concentrations by trophallaxis (Table 2).

Table 3. Survival times for donor ants that had collected concentrations of toxic sugar–milk bait after trophallaxis with 10 unfed ants in secondary toxicity tests

Treatment	Concn, ppm	n	Survival time, d[a]	Control comparison, $P > t^b$
Control	—	3	7.00 ± 1.32	—
Chlordane	500	3	4.17 ± 1.20*z*	0.189
	250	3	6.67 ± 2.24*z*	0.904
	50	3	6.67 ± 1.30*z*	0.866
Hydramethylnon	5000	3	6.33 ± 0.17*a*	0.666
	2500	3	8.00 ± 0.58*b*	0.527
	500	3	14.67 ± 0.44*c*	0.005
Imidacloprid	500	3	1.17 ± 0.17*z*	0.046
	250	3	1.67 ± 0.44*z*	0.019
	50	3	5.33 ± 2.46*z*	0.582

[a] Mean ± SEM days before death after the donor ant ingested the toxicant diluted in sugar–milk bait. For each compound, means followed by the same letter were not significantly different ($P > 0.05$; LSD [SAS Institute 1990]).

[b] Donor survival was significantly different from control whenever $P < 0.05$ (t test, df = 4).

By comparing the toxicants tested in this current investigation with those tested in our previous article (Reid & Klotz 1992), we hope to draw broader inferences on the suitability of the toxicants for carpenter ant baits and establish standards with which to judge other toxicants in the future. When all six compounds are compared at 500 ppm, imidacloprid was the fastest acting in both the primary (Fig. 2a) and secondary (Fig. 2b) toxicity trials. Abamectin and chlordane killed ants at similar rates; abamectin killed the ants slightly faster in both bioassays (Fig. 2a and b). In both primary and secondary toxicity bioassays, dechlorane and sulfluramid had virtually identical speeds of action; they are, in fact, nearly superimposed in Fig. 2a and b. Hydra-

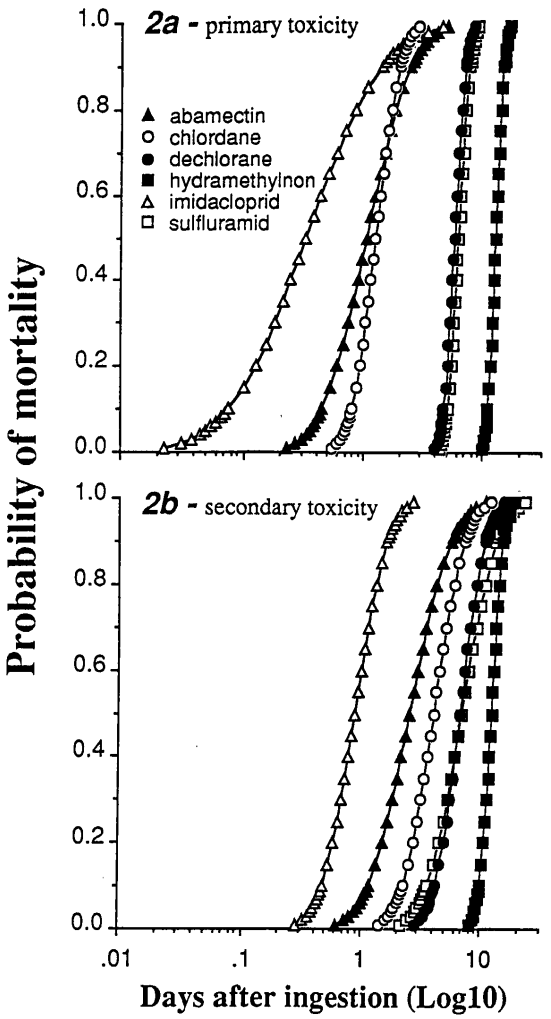

Fig. 2. Comparative activity among three delayed action toxicants (dechlorane, hydramethylnon, and sulfluramid) and three acute toxicants (abamectin, chlordane, and imidacloprid) against free-foraging workers of *C. pennsylvanicus* following direct ingestion of a 500 ppm (2a) bait, and after trophallaxis with a single worker that ingested a 500 ppm (2b) bait. Plotted points and fitted line present the probit regression equation fitted to actual mortality data; data for abamectin, dechlorane, and sulfluramid are taken from Reid & Klotz (1992).

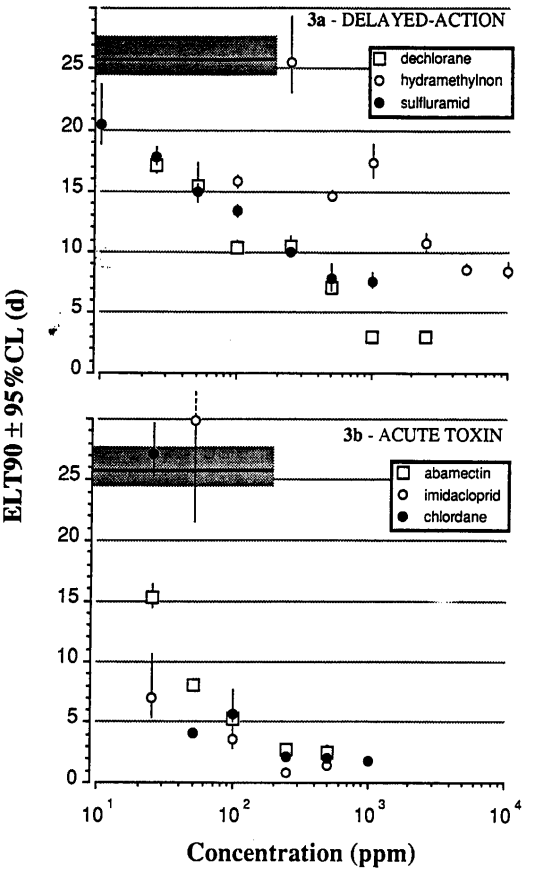

Fig. 3. Time (days) elapsing before 90% mortality (ELT_{90} ± 95% CL) in free-foraging *C. pennsylvanicus* workers ingesting various concentrations of delayed-action toxicants (3a: dechlorane, hydramethylnon, and sulfluramid) or acute toxicants (3b: abamectin, chlordane, and imidacloprid). Some concentrations (10 ppm imidacloprid, 10 ppm chlordane, and 5 and 10 ppm abamectin), which were not lethal (see Table 2 and Reid & Klotz [1992]), have not been plotted to narrow the scale of the X axis. Shaded bar extending from Y axis is the ELT_{90} ± 95% CL for controls conducted in June 1992 (see Table 1).

methylnon was the slowest acting of all the toxicants we have tested.

When the ELT_{90}s were plotted against the $\log_{10}$ of concentration (Fig. 3), further discrimination among, and characterization of, these compounds is possible. For delayed action toxicants (i.e., dechlorane, hydramethylnon, sulfluramid), there was a near linear decrease in ELT_{90}s as the concentration increased (Fig. 3a). This linear response is symptomatic of a compound's concentration-dependent, delayed-action toxicity and can be predictive of its suitability as a toxicant in social insect baiting systems (Stringer et al. 1964, Su et al. 1987). Further, all three compounds possessed delayed action toxicity over a 100 times range of dilutions, thus satisfying the prerequisite for toxicants in ant baits (Stringer et al. 1964, Banks 1990). Again, dechlorane and sulfluramid were nearly superimposable but for hydramethylnon, the linearly decreasing trend in ELT_{90}s was shifted to a higher concentration scale as a result of its lower toxicity (Fig. 3a). Finally, at the higher concentrations of hydramethylnon (10,000 ppm) and sulfluramid (1,000 ppm), the lengthy survival of workers (ELT_{90} values of 8.4 and 7.5 d, respectively) would promote long-term bait collection in the field.

For acute toxicants (i.e., abamectin, chlordane, imidacloprid) the concentration-dependence in

ELT_{90} values was nonlinear and was manifested over a narrower range of concentrations (Fig. 3b) than was the linear concentration-dependence of delayed-action toxicants (Fig. 3a). For example, although ELT_{90}s decreased as abamectin concentration increased, the relationship was curvilinear only over a 10 times range of dilutions (250–25 ppm) before a nonlethal threshold was reached at ≤100 ppm (Fig. 3b). In fact, all acute toxicants were diluted to nonlethal concentrations within the 100 times range studied, whereas only once was a delayed-action toxicant (hydramethylnon at 250 ppm) nonlethal at the ELT_{90} value. Finally, high concentrations (500 ppm) of abamectin and imidacloprid reduced the longevity of workers (ELT_{90} values of 2.5 and 1.4 d, respectively) to levels that could hamper long-term recruitment to and collection of toxic baits in the field.

By virtue of their concentration-dependent, delayed-action toxicity reminiscent of dechlorane, the more promising compounds for use in a carpenter ant bait should be hydramethylnon and sulfluramid. However, abamectin may have potential if the ovicidal effects documented in fire ant queens (Lofgren & Williams 1982, Williams 1985) are manifested against queens of the carpenter ants. Low doses of abamectin that reach the queen after dilution by trophallaxis, if causing sterilization, might compensate for low mortality among workers. Imidacloprid, on the other hand, appears to be too fast-acting to warrant further consideration. We would be remiss were we not to acknowledge the potential for IGRs, either juvenoids or molting inhibitors, to be effective in baiting against carpenter ants. Because the IGRs would have no toxic effect on workers (Banks 1990), they obviate concerns for worker mortality affecting recruitment and bait collection. Fowler (1984) has investigated juvenoid IGRs against carpenter ants, but no one has yet examined a molting inhibitor (e.g., benzoylphenyl ureas).

Acknowledgments

We thank G. Bennett (Purdue University) for support of J.H.K., and K. Kelley-Tunis and R. Yarde (Purdue) for their assistance. The comments of J. Moss (MAVERL, USDA–ARS, Gainesville, FL) improved early drafts of the manuscript. Chlorox and Miles Agricultural Division provided technical hydramethylnon and imidacloprid, respectively, as well as partial funding. This article is Journal Paper 13,780, Purdue University Agricultural Experiment Station, West Lafayette, Indiana.

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Received for publication 15 April 1993; accepted 28 June 1993.