

Susceptibility of the Bed Bug *Cimex lectularius* L. (Heteroptera: Cimicidae) Collected in Poultry Production Facilities to Selected Insecticides¹

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ABSTRACT *Cimex lectularius* L. is a widespread hematophagous insect pest around the world and is currently experiencing a reemergence as a public health pest of concern. One possible source of bed bugs to the human environment is the movement of bed bugs from poultry facilities to human structures by poultry workers. No recent studies have been conducted on the susceptibility of this insect to a wide range of insecticides. In addition, populations of bed bugs from poultry facilities have not been screened against insecticides for over 15 yr. Adult bed bugs collected from three poultry facilities in northwest Arkansas were exposed for 24 or 48 h (25°C) to glass vials treated with various dilutions of 12 insecticides dissolved in acetone to determine the concentration–response relationship. The order of toxicity, from most to least based on the LC₅₀s was: λ-cyhalothrin, bifenthrin, carbaryl, imidacloprid, fipronil, permethrin, diazinon, spinosyn, dichlorvos, chlorfena-pyr, and DDT. Significant differences in LC₅₀ and LC₉₀ values for diazinon was observed among the three populations due to the previous history of repeated exposure to a mixture of tetrachlorvinphos and dichlorvos over a 10 yr period when compared to the LC₅₀s of two populations that had been exposed to the tetrachlorvinphos and dichlorvos mixture during 2–3 flock cycles. Bed bugs in each of the three populations exhibited high levels of DDT resistance, LC₅₀ > 100,000 ppm, which confirms that resistance to this insecticide continues in bed bug populations. This study documents baseline toxicological data for 12 insecticides in three populations of bed bugs and provides the first data on bed bug susceptibility to fipronil, spinosyn, and imidacloprid.

KEY WORDS bed bug, *Cimex lectularius*, insecticide resistance

Cimex lectularius L. (Heteroptera: Cimicidae) is a hematophagous insect that can be a major pest in breeder poultry facilities (Axtell 1985) and has regained worldwide attention due to its recent resurgence into dwellings shared by humans. Both sexes feed on blood and require a blood meal for subsequent molts (Usinger 1966). Although active dispersal of bed bugs can be important, passive dispersal is almost exclusively their dispersal *modus operandi*. This species is easily translocated by passive dispersal and adapts to multiple hosts (Usinger 1966, Marshall 1981, Lehane 2005). Consequently, when host animals including

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humans are unavailable, it is extremely difficult to isolate the origins of recent infestations. However, basic molecular biology tools can be used to elucidate the identity of hosts fed upon by bed bugs, hence providing the potential for forensic applications into host identity from undigested blood obtained from bed bugs (Szalanski et al. 2006).

Passive dispersal is the most important way for the wingless cimicids to reach new hosts. Increasing numbers of humans are moving or being moved across international boundaries in many parts of the world. Bed bugs can be transported by humans in clothing and luggage (Axtell 1999, Boase 2001, Krinsky 2002), and have been detected on people traveling by airplanes, trains, ships, and cars. In poultry production, bed bugs are transported from infested facilities by human shoes, clothes, egg boxes and production equipment (Steelman 2000). Population genetic analysis of bed bugs revealed that mitochondrial 16S rRNA haplotypes are shared between poultry facilities and human structures (Szalanski et al. 2008). In addition, wild and domesticated birds and bats are often hosts of *C. lectularius* and are important in their dispersal. Since the late 1970s, bed bugs have undergone a resurgence, became widespread in the late 1990s, and appears to be cosmopolitan across the developed world (Reinhardt & Siva-Jothy 2007). Reasons for this resurgence may include increased long range air travel (Boase 2001), the ability of bed bugs to disperse locally, reduction in the use of residual insecticides around structures, and movement of bed bugs from birds and bats to domesticated birds and then to humans. Bed bugs are also an important pest of poultry (Axtel and Arends 1990, Usinger 1966, Steelman 2000, Mullen & Durden 2002). Each side of a broiler-breeder house typically has a wooden slatted platform over which the feeders and waters are hung. These wooden slats provide an ideal environment for bed bugs (Fletcher & Axtell 1993). One of the most plausible explanations for bed bug resurgence lies in this group's adaptive ability to alternate hosts (Meyers 1928, Kemper 1936, Went 1939, 1941, Hase 1964, Overal & Wingate 1976, Stelmazyk 1986) and phoretically translocate with workers in poultry facilities (Jacobs 2005) where they can amass in large numbers (Lyon 1995, Steelman 2000).

Insecticide resistance in bed bugs was first observed with the organochlorine DDT (Lofgren 1958, Busvine 1958), and later to the organophosphates malathion and diazinon (Feroz 1968). Recently, populations of *C. lectularius* collected from human structures in the United States have been shown to have high levels of pyrethroid resistance (Moore & Miller 2006, Romero et al. 2007) and has been previously attributed as a consequence of DDT cross-resistance (Busvine 1958).

This report compares the relative susceptibility of three populations of bed bugs collected from broiler-breeder egg production facilities to 12 insecticidal compounds.

Materials and Methods

The bed bugs used in this study were field collected from Arkansas broiler-breeder poultry facilities located in Washington (Population 1, northwest Arkansas), Carroll (Population 2, northwest Arkansas) and Lafayette (Population 3, southwest Arkansas) counties during July 2007. Broiler-breeder egg production flock cycles have approximate 45 week duration after which time all chickens are removed and generally a 4 week period elapses before the next flock

is placed in the facility. After each flock cycle at the poultry farm where Population 1 was collected the facility was treated with pyrethroids (permethrin and a permethrin- λ cyhalothrin mix), tetrachlorvinphos and dichlorvos (organophosphates), and spinosyn.

The poultry production facilities where Population 2 was collected had been treated between production flocks as well as during the flock cycle with the organophosphate insecticide combination RaVap[®] (tetrachlorvinphos and dichlorvos) for approximately 10 yr in consistent attempts to manage the bed bug population. The poultry farm where Population 3 was collected had been treated between flock cycles with cyhalothrin and tetrachlorvinphos and dichlorvos combined (RaVap[®]) when chickens were present in the facility for 3 yr.

The bed bug adults used in the insecticide susceptibility tests were collected from beneath the egg pad in the nest boxes, along the area where the slatted flooring joined the walls and from the wooden wall studs. A soft bristle brush was used to sweep the adult bugs into a plastic dust pan. The bugs were placed in 30 × 25 × 8 cm plastic storage containers with lids for transportation to the Veterinary Entomology laboratory, University of Arkansas, Fayetteville, AR. In the lab, adult bed bugs were removed from the storage containers using forceps and placed in 150 × 25 mm plastic Petri dishes lined with filter paper. Bed bugs were maintained in the Petri dishes at 25°C. Samples of the adult bed bugs were morphologically identified using descriptions outlined by Usinger (1966) and molecular diagnostics using mitochondrial 16S rRNA sequencing per Szalanski et al. (2008). Voucher specimens were deposited in the Arthropod Museum, Department of Entomology, University of Arkansas, Fayetteville, AR, USA.

The adult vial test (AVT) used in this study was originally developed by Plapp and Plapp (1987) for adult tobacco budworm, *Heliothis virescens* (F.), and it has since been modified for several other insect species (Cilek et al. 1991, Snodgrass 1996, Amalin et al. 2000). This insecticide-coated glass was used to determine the susceptibility of adult bed bugs to 12 pesticides: permethrin, λ -cyhalothrin, bifenthrin, diazinon, dichlorvos, tetrachlorvinphos (Rabon[®]), carbaryl, DDT, spinosyn (Extinosad[®]), imidacloprid (Provado[®]), chlorfenapyr, and fipronil. The spinosyn (Extinosad[®]) utilized was commercial grade, while the permethrin, λ -cyhalothrin, bifenthrin, diazinon, dichlorvos, tetrachlorvinphos (Rabon[®]), carbaryl, DDT, imidacloprid, chlorfenapyr, and fipronil were purchased as the technical grade from Chem Service (West Chester, PA). The 12 insecticides represents eight insecticide classes including pyrethroids, organophosphates, carbamates, organochlorines, spinosyn, chloronicotiny, pyrrole, and phenylpyrazole (Table 1).

All pesticides were dissolved in acetone and serially diluted. Treated vials were hand rotated until all surfaces within vials had been coated and the acetone had completely evaporated, leaving a uniformly applied insecticidal residue on the inner surface. The number and range of concentrations varied for each insecticide tested. Insecticide, number of concentrations, and range of concentrations in ppm per vial, respectively, were: permethrin 5, 5–500; λ -cyhalothrin 5, 0.05–500; bifenthrin, 4, 0.5–500; diazinon, 4, 5–1000; dichlorvos, 3, 5–500; tetrachlorvinphos, 3, 5–500; carbaryl, 3, 5–500; DDT, 5, 5–100,000; spinosyn, 3, 5–500; imidacloprid, 4, 0.5–500; chlorfenapyr, 4, 5–1000; and fipronil, 4, 0.5–50. Vials treated with only acetone were used as controls. For each treatment 10 adult blood-fed bed bugs were used. Each treatment of 10 bed bugs was replicated

Table 1. Concentration – response of *Cimex lectularius*, exposed for 24 or 48 h (25°C) to insecticide treated vials, LC₅₀ and LC₉₀ in ppm with 95% confidence intervals based on log-probit analysis. Population 1 (Brentwood, AR), Population 2 (Berryville, AR 2*), population 3 (Gin City, AR).

Insecticide	Pop	h	n	LC ₅₀ (95% CL)	LC ₉₀ (95% CL)	Slope ± SE	χ ²
Permethrin ^a	1	24	150	8.2 (15.4–21.1) ^A	27.6 (23.9–34.5)	0.23 ± 0.04	30.73
Permethrin ^a	2	24	150	37.1 (32.3–42.8) ^A	53.7 (47.1–65.0)	0.13 ± 0.02	38.05
Permethrin ^a	3	24	150	22.5 (14.9–32.5) ^A	52.3 (40.2–76.5)	0.07 ± 0.01	27.92
λ-cyhalothrin ^a	1	24	150	1.5 (0.9–2.7) ^A	3.4 (2.3–6.5)	1.15 ± 0.30	14.28
λ-cyhalothrin ^a	2	24	150	5.0 (4.2–6.2) ^A	7.5 (6.3–11.0)	0.88 ± 0.23	14.18
λ-cyhalothrin ^a	3	24	150	0.7 (0.3–1.8) ^A	4.3 (2.9–8.5)	0.62 ± 0.18	11.48
Bifenthrin ^a	1	24	150	21.7 (14.1–30.3) ^A	48.7 (38.5–67.0)	0.08 ± 0.01	33.82
Bifenthrin ^a	2	24	150	2.3 (1.4–3.4) ^A	4.1 (3.0–6.1)	1.24 ± 0.26	22.04
Bifenthrin ^a	3	24	150	6.5 (4.6–22.8) ^A	12.5 (8.3–62.6)	0.37 ± 0.16	5.30
Diazinon ^b	1	24	150	28.3 (24.9–34.1) ^A	39.3 (33.7–53.8)	5.66 ± 1.24	20.86
Diazinon ^b	2	24	150	561.5 (457.3–792.1) ^D	900.5 (708.9–1490.9)	3.64 ± 0.79	13.40
Diazinon ^b	3	24	150	9.8 (0.4–19.5) ^A	36.0 (24.6–71.3)	0.08 ± 0.02	12.13
Dichlorvos ^b	1	24	150	750.1 (635.1–865.4) ^D	913.1 (805.8–1089.1)	0.01 ± 0.08	22.11
Dichlorvos ^b	2	24	150	170.9 (110.1–291.0) ^{BC}	358.6 (253.6–627.7)	2.00 ± 0.39	17.61
Dichlorvos ^b	3	24	150	239.9 (148.8–360.0) ^C	643.9 (483.9–1011.2)	0.01 ± 0.01	21.23
Carbaryl ^c	1	24	145	27.7 (20.0–36.8) ^A	45.9 (36.7–61.0)	0.12 ± 0.02	34.55
Carbaryl ^c	2	24	150	3.4 (2.3–4.2) ^A	5.3 (4.5–6.7)	1.16 ± 0.26	20.75
Carbaryl ^c	3	24	150	5.0 (3.2–14.4) ^A	12.1 (9.9–54.5)	0.31 ± 0.13	5.63
DDT ^d	1	24	210	>100,000			
DDT ^d	2	24	90	>100,000			
DDT ^d	3	24	70	>100,000			
Spinosyn ^e	1	48	150	69.3 (52.8–344.2) ^B	111.3 (77.3–825.7)	3.63 ± 1.13	10.30
Spinosyn ^e	2	48	150	169.3 (111.1–293.4) ^B	357.3 (255.6–629.4)	2.01 ± 0.38	18.63
Spinosyn ^e	3	48	150	650.9 (459.1–1427.8) ^D	1269.6 (859.5–3285.2)	2.31 ± 0.50	8.50
Imidacloprid ^f	1	24	150	6.17 (4.9–13.5) ^A	9.9 (7.3–31.5)	0.59 ± 0.24	6.04

Table 1. Continued.

Insecticide	Pop	h	n	LC ₅₀ (95% CL)	LC ₉₀ (95% CL)	Slope ± SE	χ ²
Imidacloprid ^f	2	24	150	3.8 (2.6–5.2) ^A	7.6 (6.0–12.2)	0.57 ± 0.15	15.00
Imidacloprid ^f	3	24	150	0.15 (–5.5–1.69) ^A	5.8 (3.8–17.0)	0.39 ± 0.14	7.15
Chlorfenapyr ^g	1	48	150	617.4 (489.6–987.5) ^D	996.5 (754.5–1899.4)	0.01 ± .001	10.80
Chlorfenapyr ^g	2	48	150	517.6 (425.2–680.0) ^D	832.3 (672.7–1260.7)	0.01 ± .001	16.00
Chlorfenapyr ^g	3	48	150	104.6 (53.6–218.6) ^B	310.1 (203.8–696.1)	0.01 ± .003	11.12
Fipronil ^h	1	48	150	7.8 (1.2–16.3) ^A	34.7 (23.4–70.5)	0.64 ± 0.29	4.95
Fipronil ^h	2	48	150	34.8 (26.7–42.7) ^A	55.3 (46.9–69.2)	0.11 ± 0.02	32.23
Fipronil ^h	3	48	150	30.1 (20.8–38.9) ^A	61.2 (51.4–79.8)	0.07 ± 0.01	25.67

^apyrethroid.^borganophosphate.^ccarbamate.^dorganochlorine.^espinosyn.^fchloronicotinyl.^gpyrrole.^hphenylpyrazole.LC₅₀ data within rows or columns followed by different capital letters are significantly different at $P < 0.05$.

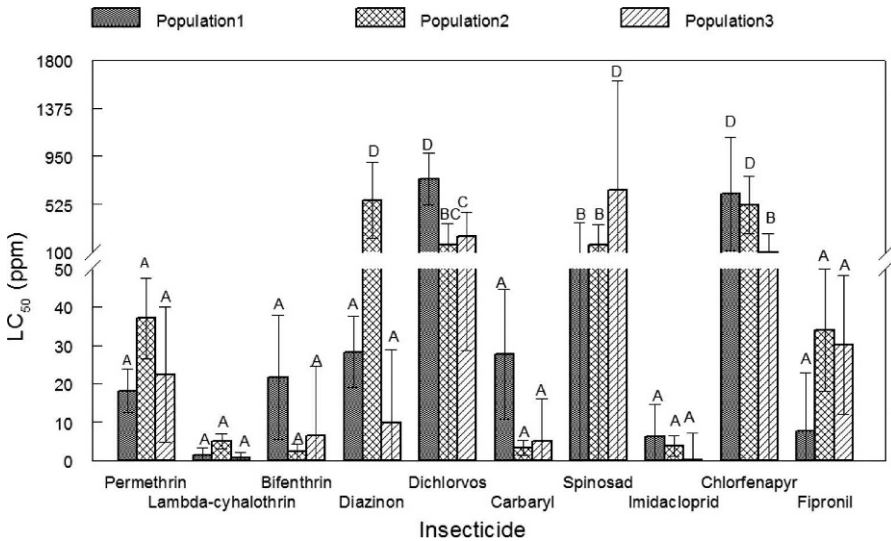


Fig. 1. LC₅₀ (\pm C.L.) response of three bed bug populations to selected insecticides.

3 times for each concentration. Vials were placed upright in a ventilated cabinet within a fume hood and maintained at a constant temperature of 25°C and 80% RH for 24 or 48 h.

Mortality of bed bugs was determined immediately after the 24-h period. A bed bug was considered dead if it was not moving or could not right itself when probed. Percentage mortality was measured as the proportion of 30 bed bugs dead after a 24-h exposure to the pesticides. All data were subjected to probit analysis (PROC PROBIT, SAS Institute 2004).

Results and Discussion

Susceptibility of bed bugs varied among insecticides and populations collected for testing (Table 1). The test for lack-of-fit of the probit model was not significant in all cases (Pearson chi-square test; $P > 0.05$). No control mortality occurred in this study. Because we conducted probit analysis separately on each bed bug population we could not use any kind of design structure. The probit analysis generated one LC₅₀ for each insecticide/population, thus, the lack of LC₅₀ replication prevented the use of an ANOVA for population evaluation. However, the probit analysis generated a Confidence Interval for each LC₅₀ and that allowed us to compare the populations overlaps of the Confidence Intervals. These comparisons were used along with the historical facility treatment information to explain differences in susceptibility response among the three bed bug populations.

Bed bugs from the three poultry farms were all susceptible to the pyrethroids with LC₅₀ values ranging from 0.7 to 37.1 ppm for bifenthrin, λ -cyhalothrin, and permethrin (Table 1 and Fig. 1). In addition, LC₅₀ comparisons indicated that there was no significant difference in the susceptibility responses among the

three bed bug populations to the same synthetic pyrethroid. Historically, Population 1, had been exposed to one spray application of permethrin in 2005, and one spray application of cyhalothrin and permethrin mixed in 2006, with all applications occurring during flock intervals when no chickens were present in the facilities. Population 2 had received no exposure to any pyrethroid while Population 3 had been exposed to facility treatment with cyhalothrin dust between flocks in 2006 and 2007.

Two representative organophosphate insecticides (diazinon and dichlorvos) were used in our laboratory tests of susceptibility. Comparison of confidence limits (CL) indicated that Population 2 was resistant to diazinon and dichlorvos (Fig. 1). No significant difference existed between the LC_{50} s of Population 1 and 3 while both were significantly more susceptible than Population 2. The facility containing Population 2 had been repeatedly treated with RaVap® (a combination of tetrachlorvinphos and dichlorvos) over several years while chickens were in the facility and between flock production cycles. Population 1 was treated three times during 2006 and one time in 2007 with tetrachlorvinphos during the flock production cycles and sprayed with RaVap® two times during each production cycle. All three populations were significantly less susceptible to dichlorvos than to any of the pyrethroid insecticides tested (Fig. 1). Bed bugs from Population 1 was significantly more resistant to dichlorvos than the bed bugs from Population 3 while no significant difference was found between the susceptibility of bed bugs from Population 2 and 3 (Table 1, Fig. 1).

Carbaryl was used in the laboratory tests to represent the carbamate family of insecticides and no significant difference in susceptibility existed among the three populations of bed bugs (Table 1). Historically, no carbaryl or other carbamate had been used in the broiler-breeder facilities. No significant difference existed between the LC_{50} s of the three populations and the LC_{50} s found for the pyrethroids tested on these bed bugs. This is a significant finding since pyrethroids have been implicated as having poor residual control on bed bugs in urban application scenarios (Moore & Miller 2006, Romero et al. 2006).

No previous history of organochlorine use was obtained for any of the facilities containing the three populations of bed bugs utilized in the present studies as all three facilities were constructed after the ban on organochlorine insecticides in the United States over 30 yr ago. However, we included DDT in these studies and found that the LC_{50} s of all three populations were $>100,000$ ppm. It seems probable that all three of these bed bug populations had been exposed to DDT or other organochlorines at some previous time, and certainly before the infestations occurred in the broiler-breeder egg production facilities. High levels of resistance to DDT, effectively acting as 99% exposure, caused only 20% mortality after 96 h of continuous exposure. Bed bugs have had a history of resistance to DDT for over 50 yr. In the 1950s, resistance to DDT was observed in bed bugs from Japan, Korea, Ohio, and U.S. naval vessels (Lofgren et al. 1958). Populations of *C. lectularius* from South Korea had a DDT LC_{50} of 2.8% and a dieldrin LC_{50} of 0.167% (Cha et al. 1970). More recently, bed bugs from Brazil have been found to have a LD_{50} greater than 4% DDT (Nagem & Williams 1992), and high levels of DDT resistance has also been observed in *Cimex hemipterus* (F) from Sri Lanka with 41–88% survival to 2% DDT treated paper (Karunaratne et al. 2006). *C. hemipterus* has also been found to have resistance to the pyrethroids, permethrin and alpha-cypermethrin, from Tanzania (Myamba et al. 2002).

Although cross-resistance from DDT to pyrethroids is believed to exacerbate the ongoing reemergence of this pest, it has not been empirically proven. Resistance has been defined as, “the development of a strain capable of surviving a dose lethal to a majority of individuals in a normal population” (French-Constant & Roush 1990). However, what defines a “normal” population? It is apparent from these field collected populations that the only consistent relationship in the quantal response to insecticides is that a significant population bottleneck constrained most populations from the widespread application of DDT, thus skewing their genetic constitutions. Robertson & Preisler (1992) defines resistance as a significant, genetically based shift in the molecular, biochemical, or behavioral bases of quantal responses in populations of an arthropod species, whereby resistance represents one extreme of response, compared with susceptibility, the other extreme...various degrees of tolerance lie between the two extremes.

New and novel insecticides were also tested to determine the base line susceptibility of bed bugs infesting poultry production facilities. Imidacloprid (a chlorinated analog of nicotine) caused LC₅₀s in all three bed bug populations that were not significantly different and were not significantly different from the pyrethroid LC₅₀s (Table 1, Fig. 1). This product had not been used for bed bug control in any of the broiler-breeder egg production facilities.

The LC₅₀ data for a formulation of spinosyn (a mixture of spinosyn A and D), showed that the bed bugs from the three populations responded much slower to this compound than to the pyrethroid insecticides (Fig. 1). Only the facilities containing Population 1 had previously been treated with a formulation of spinosad; however, no significant difference was found in the LC₅₀ data for Population 1 and Population 2. Both populations were significantly more susceptible than the bed bugs comprising Population 3 (Fig. 1). In addition, the LC₅₀ was reached 48 h after exposure rather than the 24 h obtained for the pyrethroids and organophosphate compounds tested. Chlorfenapyr (halogenated pyrrole) another microbiologically produced compound caused LC₅₀s to the three populations of bed bugs similar to the data obtained for spinosyn. Chlorfenapyr had not been used at any of the three poultry farms. This observation was consistent with results from Miller & Moore (2006), wherein they reported mortality to chlorfenapyr bioassays resulted in LT₅₀ (lethal time) of 48 h after bed bug exposure. Fipronil (a phenylpyrazole) caused significantly lower LC₅₀s to the bed bugs in all three populations than did either spinosyn or chlorfenapyr but were not significantly lower than the LC₅₀s obtained for the pyrethroids. However, as was observed for spinosyn and chlorfenapyr, the LC₅₀s for the three populations were not reached until 48 h after exposure.

As evidenced by the relative susceptibility of bed bugs to pyrethroids in this study, it would be imprudent to suggest that pyrethroids are ineffective in field applications for remedial or preventive control of bed bugs. Rather, each individual control scenario will likely dictate the choice of insecticide, the manner of application, and the level of control afforded by multiple integrated tactics employed by a pest management professional. Careful rotations of insecticides, as has been a relatively common practice for any applicator attempting to sustain the use of an insecticide, and thorough and comprehensive applications will likely control bed bugs in most urban scenarios. However, the lack of registered insecticides that can be applied when poultry are present in infested facilities make bed bug management extremely difficult. The broiler-

breeder egg production cycle is generally 265 d long, thus, bed bug populations reach high numbers before the end of the egg production cycle. Due to the physical environment in the production facilities it is extremely difficult to effectively apply those compounds that are registered for use when the chickens are present. In addition, many avenues of infestations exist ranging from wild birds, poultry production workers moving from house to house as well as movement of egg shipping materials from farm to farm. The use of pyrethroids such as those reported in our present studies and future registration of new and novel insecticides such as imidacloprid, fipronil, spinosyn and bifenthrin, are all viable candidates for effective bed bug management in both urban and poultry production environments.

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