Review of Insecticide Resistance in Cat Fleas (Siphonaptera: Pulicidae)

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ABSTRACT   Insecticide resistance often is blamed for failures of insecticides to control cat fleas, *Ctenocephalides felis* (Bouché). Yet the genetics and adaptive advantage of resistance traits remain unexamined. Lethal doses of insecticides that kill 50% of the population fluctuate 7-fold within a cat flea strain. Many reports of flea resistance may be attributable to variable mortality from effects of solvents, substrates, humidities, temperatures, colonization, and ages of fleas. Resistance ratios (ratios of lethal doses of a resistant to a susceptible strain) are <690-fold in fleas; lower than many other arthropods. This, plus strain variability, hinders resistance detection. Relationships between resistance levels, control failures, and health threats are unclear. Insensitive acetylcholinesterase, knockdown recovery, glutathione transferase conjugation, and mixed function oxidase/cytochrome P450 are demonstrated resistance mechanisms in cat fleas. Ecological genetics of resistance in cat fleas probably involves flea transfer among hosts, host movements, refugia, founder effects, and mortality from abiotic factors. Understanding cat flea resistance requires population monitoring before, during, and after insecticide treatments using conventional and rapid molecular bioassays. Sustained insecticide release devices such as flea collars and long-lived insecticide residues for premises possibly contribute to the development of resistance. New systemic and topical insecticides, especially when given prophylactically, may act similarly. Eliminating insecticides prevents insecticide resistance but necessitates application of biorational tactics incorporating mechanical, environmental, and cultural controls. Using high temperatures, low humidities, host grooming and such tactics as decreasing doses, increasing action thresholds, rotating insecticides, and leaving spatial and temporal refugia may suppress cat flea resistance.

KEY WORDS   *Ctenocephalides felis*, insecticide susceptibility, bioassays, systemics, sustained release, flea collars

RESISTANCE WAS REPORTED in 8 species of fleas by 1980 (Georghiou and Mellon 1983), including 3 species of notable public health importance—the cat flea, *Ctenocephalides felis* (Bouché); human flea, *Pulex irritans* (L.); and oriental rat flea, *Xenopsylla cheopis* (Rothschild). *C. felis*, the most common flea in Europe (Vater and Vater 1985) and the United States (Warner 1984, Rust and Dryden 1997), is resistant to more chemical categories than any other flea (WHO 1992, Rust 1993).

Resistance to insecticides is defined by the World Health Organization (WHO) as "development of an ability in a strain of some organism to tolerate doses of a toxicant that would prove lethal to a majority of individuals in a normal population of the same species." (WHO, cited in Ferrari 1996). Ferrari (1996) stated that "resistance has a genetic basis and is the result of a change in the genetic composition of a population as a direct result of the selective effects of a toxicant." However, a better definition of resistance may be a response of an organism or a population to a toxicant that enables the organism or population to withstand future toxicant exposures better, because

gene amplification which may confer resistance does not require selection (Devonshire and Field 1991), and other individual responses to sublethal exposures are included. There have been no studies on the genetic basis of putative resistance in cat fleas, nor on selective advantage of resistance in the field. Much insecticide resistance ascribed to cat fleas simply may be variation in flea susceptibility.

This review examines the current knowledge regarding the measurement, prevalence, and mechanisms of resistance in cat fleas. Emphasis is on the abiotic and biotic factors that might influence the likelihood of resistance development in cat fleas. Finally, consideration is given to potential strategies to avoid resistance development.

Measurement of Cat Flea Resistance

Susceptibilities are measured as percentage mortalities (Bossard 1997) or are evaluated with probit analysis to give lethal doses and resistance ratios (the ratio of lethal doses of a field to a susceptible reference strain). One problem in comparing strain susceptibilities is variation in the susceptibility of a strain over time. Moyses (1995) found 7-fold differences in lethal doses within a susceptible laboratory strain to diazinon over several years. Bossard (1997) assayed cat

fleas from a California colony 4 times over several months using 10 mg/m^2 chlorpyrifos deposits and found mortalities after 24 h ranging from 3.7 to 100%.

Factors contributing to variability include abiotics such as procedure, temperature, humidity, substrate, and biotics such as colonization. Fleas often are shipped as pupae or adults and then immobilized with CO_2 or cold before testing. The susceptibility of cat flea pupae to insecticides was unaffected by handling during transport, but adult susceptibility increased with longer CO_2 or cold exposures (El-Gazzar et al. 1988a). Adult cat fleas exposed to CO_2 with sorting by sex also have increased susceptibility to insecticides (Bossard 1997).

Factors such as the type of solvent (water or acetone, Rust 1993) or the volume of solvent used (Moyses 1997) affect insecticide efficacy in bioassays. Humidity, temperature, and lighting alter insecticide chemistry, modify behavior and physiology of test insects (Hinkle et al. 1989, Rust 1995), and also are probably important in establishing a bioassay baseline. For example, atmospheric humidity affects the residual efficacy of chlorpyrifos, permethrin, and propetamphos against cat fleas (Rust 1993).

Insecticides such as chlordane, propetamphos, and propoxur are more active at higher temperatures, whereas DDT and many pyrethroids are less active (Hinkle et al. 1989, Rust 1995). Fleas move more and may acquire more toxicant as temperatures increase from 20 to 30°C and their susceptibility to dieldrin increases, unlike their DDT susceptibility (Busvine and Lien 1961).

Busvine and Lien (1961) found flea movements decreased in darkness, but they did not examine lighting effects on susceptibility. Activity of cat fleas peaks at dusk (Koehler et al. 1989, Bossard 1997), which coincides with cat activity (Kern et al. 1992). Bossard (1997) detected no changes in chlorpyrifos toxicity toward cat fleas in test tubes under reduced light. Bioassays started at 0700, 1230, 1830 and 0030 hours revealed no mortality differences (Bossard 1997). Humidity, temperature, and lighting often are not standardized among flea bioassays.

Bioassays for cat flea insecticide resistance have used various configurations and substrates. Vertical strips of filter paper (WHO bioassay for fleas) required higher doses of chlorpyrifos and permethrin to kill fleas than did horizontal disks, probably because fleas moved onto untreated areas or were unable to return to treated surfaces rapidly (Bossard 1997).

The substrate of the WHO flea bioassay is a cellulose filter paper strip (Whatman #1) treated with insecticide (Brown 1971, Gratz 1980, WHO 1981, Moyses 1995). Other cat flea bioassays have used glass (Schwinghammer et al. 1985, Bossard 1997), nylon carpet (Koehler et al. 1986), cotton fabric (Rust and Reierson 1988), soil (Palma and Meola 1990), fiberglass (Olsen 1993), wool fabric (Olsen 1993), turfgrass (Metzger et al. 1996), and nylon 6,6 fabric (Bossard 1997, Moyses 1997) for substrates.

The type of substrate on which insecticide is deposited affects its efficacy against fleas. A chemical may be more effective than another chemical on a given substrate, but may be less effective on a different substrate (El-Gazzar et al. 1986, Olsen 1993, Moyses 1995, Bossard 1997).

Bossard (1997) assayed fleas on nylon 6,6, which is used to manufacture most U.S. carpets, in an attempt to duplicate substrate–chemical interactions. Bioassays using glass (Bossard 1997) or topical application (Moyses 1995) may be more precise and generate higher resistance ratios than paper and nylon fabric bioassays, but glass probably does not mimic most field surfaces and therefore may be inaccurate (Bossard 1997). Topical application may have the same disadvantages (Hinkle et al. 1985). Further research is needed to determine which assay best estimates resistance levels in relation to control efficacy.

Colonization changes susceptibilities of reference and field-collected strains. Susceptible normal populations used for bioassay reference strains usually have been maintained in the laboratory for years and appear adapted to those conditions (El-Gazzar et al. 1988b, Moyses 1995, Bossard 1997). After a year in the laboratory, resistance in a cat flea strain increased toward carbamates (bendiocarb, carbaryl, propoxur) probably because of carbamate exposure during rearing (El-Gazzar et al. 1988b). In the same strain, resistance decreased toward chlorpyrifos and malathion, whereas it remained the same with chlorfenvinphos, diazinon, isofenphos, and propetamphos (El-Gazzar et al. 1988b). Bossard (1997) found that laboratory colony strains died more quickly than recently collected field strains during insecticide exposure, but lived longer in control tubes. Whether this was caused by field resistance or laboratory adaptation was undetermined.

Another biotic factor that may affect susceptibility is sex of the test insect. Male insects often are more susceptible to contact insecticides than females (Shepard 1960), probably because usually the males are smaller. With systemic insecticides, male cat fleas are less susceptible than females, probably because of the males' lower feeding rates (Dryden 1992). Because females emerge from cocoons before males (Hudson and Prince 1958) and fleas are usually assayed as groups with varying sex ratio, variability caused by sex differences may be compounded. Nonetheless, there were no consistent differences in susceptibility between sexes in bioassays of fed rat fleas (Busvine and Lien 1961) or of unfed cat fleas (Bossard 1997) using contact insecticides.

Density of insects in experimental units may affect susceptibility (Shepard 1960) because of insect interactions. However, densities at 5, 10, and 30 fleas per tube did not affect percentage mortalities on deposits of chlorpyrifos (Bossard 1997).

The age and developmental stage of fleas affect susceptibility to insecticides. Adult cat fleas older than 48 h were more susceptible to insecticides than younger adults (El-Gazzar et al. 1988a). Larval cat fleas are twice as tolerant of insecticides as adults (Rust 1993).

History of Cat Flea Resistance

Resistant cat fleas were 1st reported in 1952 from dogs in the southeastern United States when 5% DDT dusts failed to control the dogs' fleas, and thereafter *C. felis* from many areas showed resistance to chlordane, dieldrin, and HCH (Brown and Pal 1971) and other insecticides (WHO 1992) (HCH is hexachlorocyclohexane; the gamma isomer is lindane). Fox et al. (1968) reported that adults and larvae in Puerto Rico were tolerant, possibly resistant, to DDT, dieldrin, and malathion. Fox and de Leon (1984) found possible resistance in cat fleas to carbaryl and methoxychlor powders.

Organophosphate resistance was suggested as contributing to control failures of previously effective flea collars and sprays (Schwinghammer et al. 1985). However, they did not assay both resistant and susceptible strains to ascertain the level of resistance. A field-collected cat flea strain from Florida required LD_{50}s of 7.2-, 9.4-, 10-, 20-, 26-, 28-fold more propetamphos, diazinon, chlorpyrifos, carbaryl, malathion, and bendiocarb, respectively (El-Gazzar et al. 1986) and 4.2-, 5.2-, and 6.8-fold more fluvalinate, cypermethrin, and cyfluthrin, respectively (Lemke et al. 1989), than did a susceptible California cat flea colony.

Lemke et al. (1989) concluded that pyrethroids were ineffective against this Florida strain. Possibly, DDT had selected fleas for knockdown resistance (kdr). However, Lemke et al.'s (1989) resistance ratios are less than the expected variability within cat flea strains (Moyses 1995), indicating that adult fleas may not be resistant but inherently tolerant of certain pyrethroid formulations.

In several reports of resistance, determining if resistance ratios are caused by strain differences or bioassay conditions is difficult because field and reference strains were not assayed simultaneously. In Tanzania, cat fleas were reported to be resistant to malathion because a 3.6 mg/cm² dose killed only 92% of cat fleas in a WHO bioassay after 24 h (Kilonzo and Gisakanyi 1988). Kobayashi et al. (1994) found their cat flea strain was 190-fold less resistant to certain insecticides than the Florida strain of El-Gazzar et al. (1988b). The other highest resistance ratio for field-collected cat fleas is 190-fold from an Australian strain (Moyses 1995) (Table 1).

The highest resistance ratio for fleas is 690-fold from a *C. felis* strain selected with malathion in the laboratory (Moyses and Bunchy 1996). For other flea species, the record is from a DDT-selected *X. cheopis* laboratory strain of 222-fold against the DDT-analog prolan (Kalra and Joshi 1974).

Whether laboratory-selected or field-collected, the resistance ratios of fleas are low compared with other insecticide-resistant arthropods such as horn flies against fenvalerate (92,000-fold) (Sheppard and Joyce 1992), diamondback moths resisting *Bacillus thuringiensis* (6,800-fold) (Tabashnik et al. 1993), and the tick *Boophilus decoloratus* to fenvalerate (4,744-fold) (Coetzee et al. 1987). Low resistance ratios, coupled with high strain variability, increase the difficulty of

Table 1. Resistance ratios from literature of *C. felis*

Insecticide	Resistance ratio	Reference
Malathion	690[a]	Moyses and Bunchy 1996
Malathion	190	Moyses 1995
Malathion	108[a]	Moyses 1995
Malathion	77	Kobayashi et al. 1994
Fenitrothion	41	Moyses 1995
Bendiocarb	28	El-Gazzar et al. 1986
Malathion	25	El-Gazzar et al. 1986
Carbaryl	20	El-Gazzar et al. 1986
Diazinon	15	Moyses 1995
Carbaryl	12	El-Gazzar et al. 1988b
Malathion	12[a]	Collart and Hink 1986
Malathion + DEF	12[a]	Moyses 1995
Permethrin	12	Moyses 1995
Diazinon	11	Moyses 1995
Chlorpyrifos	10	El-Gazzar et al. 1986
Fenthion	10	Moyses 1995
Diazinon	9.4	El-Gazzar et al. 1986
Bendiocarb	7.7	El-Gazzar et al. 1988b
Malathion	7.6	Moyses 1995
Propetamphos	7.2	El-Gazzar et al. 1986
Cyfluthrin	6.8	Lemke et al. 1989
Fenitrothion	5.8	Moyses 1995
Chlorfenvinphos	5.6	El-Gazzar et al. 1986
Cypermethrin	5.2	Lemke et al. 1989
Chlorpyrifos	4.7	Moyses 1995
Propoxur	4.4	El-Gazzar et al. 1986
Fluvalinate	4.2	Lemke et al. 1989
Chlorpyrifos	3.6	Moyses 1995
Isofenphos	3.5	El-Gazzar et al. 1986
Chlorpyrifos	3.0	Moyses 1995
Fenvalerate	2.8	Lemke et al. 1989
Propoxur	2.7	El-Gazzar et al. 1988b
Resmethrin	2.2	Lemke et al. 1989
Fenthion	2	Moyses 1995
Malathion	1.7	Moyses 1995
Chlorpyrifos	1.6	El-Gazzar et al. 1988b
Malathion	1.6	El-Gazzar et al. 1988b
Permethrin	1.6	Lemke et al. 1989
Tralomethrin	1.5	Lemke et al. 1989
Profenofos	1.4	Moyses 1995
Chlorfenvinphos	1.3	El-Gazzar et al. 1988b
Diazinon	1.3	El-Gazzar et al. 1988b
D-phenothrin	1.2	Lemke et al. 1989
Isofenphos	1.1	El-Gazzar et al. 1988b
Propetamphos	1.1	El-Gazzar et al. 1988b

[a] Laboratory-selected.

detecting resistance (Moyses 1995). Whether these characteristics of cat flea resistance are caused by biological, operational, or assay characteristics is unknown.

Bossard (1997) produced a range of mortalities in 9 cat flea strains collected from throughout the United States with given insecticide doses and exposures. For example, a 24-h exposure to 25 mg/m² deposits of pyrethrum with PBO killed 97% of cat fleas from a California colony but <1% of a recently field-collected Florida strain. The Florida strain was from a pet repeatedly treated for fleas and tolerated carbaryl and pyrethrum. A similarly treated Texas strain tolerated carbaryl, chlorpyrifos, and malathion.

In contrast, the laboratory strain from California was susceptible to malathion and pyrethrum, but tolerated permethrin. A North Carolina laboratory strain was susceptible to carbaryl, chlorpyrifos, malathion, and permethrin, but not to pyrethrum (Bossard 1997).

Cat fleas show multiple resistance (El-Gazzar et al. 1986, 1988b; Moyses 1995) and possible cross-resistance between carbaryl and the organophosphates chlorpyrifos and malathion (Bossard 1997).

Genetic and Molecular Mechanisms of Cat Flea Resistance

Laboratory experiments suggest a genetic basis for some susceptibility differences (Rust 1993). Cat fleas selected with malathion in the laboratory developed resistance 12-fold higher after 8 generations (Collart and Hink 1986), 108-fold higher after 2 generations (Moyses 1995), and 690-fold higher after 19 generations (Moyses and Bunchy 1996) than did unselected controls. Such experiments may not duplicate resistance development in the field. New techniques such as artificial hosts (Wade and Georgi 1988) and on-host flea chambers (Thomas et al. 1996) may facilitate crossing of susceptible and field strains necessary to investigate the genetics of resistance.

Target site insensitivity of acetylcholinesterase and organophosphate detoxification by glutathione transferase conjugation occur in resistant fleas (Hinkle et al. 1995b). The former mechanism may produce knockdown recovery after 24 h (Bossard 1997).

Piperonyl butoxide synergized D-limonene activity, which indicated detoxification by fleas with mixed function oxidase (Collart and Hink 1986). Negative cross-correlations of cat flea mortality occurring between carbaryl, chlorpyrifos, and malathion to permethrin and pyrethrum also suggested cytochrome P450 (mixed function oxidase) detoxification (Bossard 1997).

Cao and He (1991) quantified specific esterase bands in the fleas *C. felis, X. cheopis,* and *Nosopsyllus laeviceps* (Yagubyants). Roslavtseva et al. (1991) examined inhibition of *X. cheopis* esterases by the insecticide Sh-205 that was synergistic with permethrin.

Esterase activity fluctuates in immature and adult cat fleas (Silver et al. 1997). Because some cat flea esterases degrade the artificial substrate alpha-naphthyl acetate (Silver et al. 1997) and also may hydrolyze toxicants, these developmental changes may explain varying insecticide susceptibilities as fleas become older.

Bossard (1997) used a rapid assay for flea esterase (Kambhampati et al. 1997) and found no correlations between esterase levels and mortalities in 9 cat flea strains when exposed to carbaryl, chlorpyrifos, malathion, permethrin, or pyrethrum. Whether this was caused by the assay or a lack of the esterase resistance mechanism in the assayed cat fleas is unknown.

Resistance caused by modifications in behavior, morphology, or the excretion or sequestration of insecticides have not been reported in *C. felis.* Ingestion of adult flea feces by larval fleas (Hinkle et al. 1991) may produce resistance if feces were contaminated with insecticides.

Certain chemicals repel fleas, including DEET, permethrin (Mehr et al. 1984), and pyrethrum (Linduska et al. 1946). Insecticide avoidance may occur when fleas move away from pyrethroid-treated areas such as flea collars. Fleas avoiding treated surfaces may in part account for the WHO bioassay requiring higher doses than assays without untreated surface areas (Bossard 1997). Exophilous resistance could result when cat fleas move to difficult-to-treat areas in response to darkness or humidity.

Factors Affecting Development of Resistance in Cat Fleas

Cat flea resistance development has not been studied in the field. However, biological factors contributing to insecticide resistance in other insects include isolation, mobility, fortuitous survival, polyphagy, and refugia (Georghiou and Taylor 1986).

Complicated mating behavior of fleas could isolate populations, prevent hybridization between susceptible and resistant strains, restrict gene flow, and affect rate of resistance evolution. This may be countered by flea movement. Blagburn and Hendrix (1989) felt infrequent, short-term contact between infested and uninfested hosts was insignificant for the movement of adult *C. felis.* Movements between hosts of 3–15% of the cat fleas infesting a host are possible (Rust 1994). Dryden and Broce (1993) found cat fleas off the host moved 8 m overnight in response to a light trap.

Mortality caused by abiotic factors is important for fleas, especially immatures. Extreme temperature and low humidity affect *C. felis* reproductive potential by slowing development and reducing survival (Silverman et al. 1981, Silverman and Rust 1983). More annual generations of fleas in warmer latitudes and consequently an increased insecticide application may increase the rate at which resistance develops. Resistant fleas were 1st collected in the tropics (Brown and Pal 1971).

Host resistance, such as grooming, can cause 50% mortality of adult fleas after 1 wk (Wade and Georgi 1988). Host density positively correlates with populations of *X. vexabilis* (Wagner) (Haas 1969) and probably of cat fleas also.

Mortality from noninsecticidal sources should slow resistance development by removing resistance genes (Rosenheim et al. 1996). There also may be fitness costs for resistance genes. These factors, as well as small, isolated populations that are not exposed to intense selection for long periods, may explain the low resistance ratios of cat flea populations.

Features of cat flea population dynamics such as host colonization, host movements, and founder effects may be critical to understanding resistance development. Gene flow of fleas is undoubtedly influenced by movements and home ranges of hosts. Humans and their pets travel and may transport fleas. Cats can have home ranges in urban areas of <1 ha or up to 270 ha rurally, depending on cat density and the availability and distribution of food (Liberg and Sandell 1988). Urban dogs may have home ranges of 1.5–2.6 ha (Beck 1973). Wild animals are mobile, increasingly abundant in urban areas and often serve as alternative hosts for *C. felis.* Host movement and in-

teraction create opportunity for flea exchange (Marshall 1981).

The immense variety of hosts upon which adult cat fleas feed (Hopkins and Rothschild 1953, Lewis 1972) creates populations in refugia which may increase or decrease resistance (Georghiou and Taylor 1986). Probably, refugia suppress resistance by providing susceptible fleas.

For immature fleas and off-host adults, habitat heterogeneity occurs where certain areas of the house and yard are more infested (Osbrink et al. 1986). Byron (1987) collected cat flea eggs mainly where cats slept. Immature fleas develop in these protected microhabitats where dried fecal blood accumulates and the temperature and humidity favor development. Failure to treat these areas creates refugia.

Resistance development also is dependent on operational factors, including insecticidal mode of action, cross-resistance, selection intensity, and life stage selected, particularly if products with similar modes of action are applied to different stages. Inadequate insecticide applications, insecticides with prolonged residual activity, and sustained release devices such as flea collars may kill <100% of the pest insects and contribute to the rapid evolution of resistance (Sheppard et al. 1989, Rust 1995). Flea collars may be analogous to insecticide-treated cattle eartags applied to control horn flies, which resulted in high and extensive resistance and control failures (Sheppard et al. 1989).

With the recent introduction of systemic and topical residual formulations of fipronil, imidacloprid, and insect growth regulators and developmental inhibitors such as lufenuron (Hinkle et al. 1995a, 1997), flea populations may be exposed chronically to sublethal doses, increasing resistance, especially when prophylactically administered. Lufenuron resistance has recently developed in Drosophila melanogaster (Meigen) (Wilson and Cain 1997).

For cat fleas, lufenuron efficacy was 0–98% during 119 d under conditions simulating natural reinfestations (Blagburn et al. 1995). Surviving fleas provide a nucleus for continued exposure and the potential founding of resistant populations. Applicator noncompliance further increases the risk of sublethal exposures.

Rust (1993) noted that for cat fleas, the effectiveness and practicality of resistance management tactics available such as decreased dose, increased action threshold, rotation of insecticides with different modes of action, and leaving untreated refugia have not been investigated. For example, a 1-time treatment might be sufficient to eliminate acute flea problems temporarily, even if not 100% effective. If this treatment is not followed by another insecticide treatment or sustained residual activity until the fleas again become a pest problem, then genes from fleas in untreated refugia may restore susceptibility to the treated population. These acute, temporary insecticide exposures should prevent resistance development better than treatments where fleas continually receive sublethal doses. Such temporal refugia for

fleas are similar to the spatial refugia proposed for managing resistance in crop pests.

Unfortunately, susceptible genes come packaged as biting cat fleas! Therefore, treatments must be harsh enough to solve pest problems caused by fleas but mild enough to avoid creating resistance.

Importance of Insecticide Resistance in Flea Control

Insecticide tolerance and resistance often are blamed for failures to control cat fleas (Fox et al. 1968, Kerr 1977, Davidson 1992). Even low resistance ratios may be enough to affect control (Rust 1993).

However, other factors may contribute to failures to control cat fleas, such as reinfestations from refugia on domestic or wild animals or in off-host environments (Bennett and Lund 1977, Byron 1987). Abundant hosts, larval food, and other favorable environmental conditions may contribute to large flea populations (Lifton 1985).

Control failures also may occur because insecticide applications are not adjusted for variation in humidity and temperature or cultural conditions such as substrates and carpet types (Koehler et al. 1986, Dryden and Reid 1996). Failures also occur when cocooned adult fleas survive insecticidal treatments in refugia behind furniture or in the protective carpet fibers (pupal windows) and emerge weeks after the insecticide treatments (Dryden 1991). The importance of insecticide resistance versus these other factors in causing failures to control cat fleas is not known (Rust and Dryden 1997).

Because flea control has relied heavily on insecticides (Rust and Dryden 1997), chemical failure potentially has a great impact on the development of resistance and flea control technology. As soon as products appear to be ineffective, they are reapplied or replaced. Perhaps no example of control of a household insect of medical–veterinary importance illustrates a pesticide treadmill better than the history of cat flea control.

Noninsecticidal methods without toxicity and pollution that conserve the resource of susceptible pests (Sheppard et al. 1989) and nontarget organisms (Wilson and Cain 1997) are available to control fleas. These include vacuuming and shampooing carpets and animal bedding, setting flea traps, and combing fleas off pets (Hinkle et al. 1997, Rust and Dryden 1997). Too often, these effective methods are not used.

Future Directions in Research

In conclusion, to determine if cat fleas truly are resistant to insecticides requires baseline studies of population susceptibility before and after toxicant exposure. Unexposed populations should be monitored simultaneously. Determination of genetic and other factors causing changes in susceptibility requires conventional and molecular field assays (Brown and Brogdon 1987).

Introductions of new insecticides to control cat fleas such as insect growth regulators represent ideal situations to follow potential resistance development. Veterinarians, pest control operators, and pet owners need to use operational procedures that diminish resistance in cat fleas. Cooperation may be helpful. Integrated pest management and the concepts of injury threshold and treatment levels rarely have been applied to urban entomology (Robinson 1996), but they espouse insecticide reduction, especially with insect pests of companion animals.

Insecticide resistance may be viewed, not as a problem to be overcome, but as a warning of injudious insecticide use. Eliminating insecticide use would prevent insecticide resistance. However, if we continue to rely upon chemicals for flea control, pest management strategies that reduce the likelihood of resistance need to be developed. In either case, the application of biorational approaches that minimize environmental harm (Hinkle et al. 1997) through mechanical, environmental, and cultural controls is needed.

Acknowledgments

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