Integrated Flea Control: Flea Control For The 21st Century

Michael W. Dryden DVM, Ph.D.
Professor of Veterinary Parasitology
Department of Diagnostic Medicine/Pathobiology
1800 Denison Ave.
College of Veterinary Medicine,
Kansas State University
Manhattan KS. 66506 USA.

Alberto B. Broce Ph.D.
Professor of Entomology
Department of Entomology
Kansas State University
Manhattan KS. 66506 USA.

Introduction

Flea infestations of pets and the home are a common occurrence and attempts at elimination can be expensive and time consuming. Fleas are hematophagous ectoparasites and are responsible for the production and transmission of several diseases of humans and their pets. The flea primarily responsible for these infestations is the cat flea, *Ctenocephalides felis felis* (Bouché)

In the last 5 to 6 years, important advances have been made in development of new insecticides and formulation technology. These advances have dramatically altered veterinarian and pet owner expectations of flea control. While many of these new products provide excellent flea control, they also pose the potential for selection of resistant flea populations. Proper stewardship of these products may necessitate developing an integrated approach to flea control.

Flea Species Associated With Dogs And Cats:

While several flea species have been found on dogs and cats, only *C. felis felis, C. canis*, and *Pulex* sp. occur with enough regularity to be of importance as nuisance pests in North America (Amin, 1976; Harman et al., 1987; Dryden 1988). Studies in other countries revealed that *C. felis felis* was most common on dogs in Egypt (Amin, 1966), Denmark (Kristensen et al., 1978) and England (Beresford-Jones, 1981), while *C. canis* was most prevalent in Austria (Ressl, 1963), Ireland (Baker and Hatch, 1972) and New Zealand (Guzman, 1984). With the exception of Ireland and New Zealand, *C. felis felis*, the **cat flea**, is considered the most common flea infesting both dogs and cats worldwide.

Medical and Veterinary Importance of Cat flea infestations: or Why do we need to control fleas on dogs and cats?

The cat flea is the cause of severe nuisance and irritation in humans and animals, being responsible for the production of allergic dermatitis. The cat flea can also serve as the vector of typhuslike rickettsia and is the intermediate host for filarid and cestode parasites.

Flea Allergy Dermatitis (FAD) is an immunologic disease in which a hypersensitive state is produced in a host as a result of the injection of antigenic material from the salivary glands of fleas. The condition is found worldwide and is the most common dermatologic disease of dogs and a major cause of feline miliary dermatitis. Fleas, as hematophagous insects, can in heavy infestations produce iron deficiency anemia , particularly in young animals. Fleas in the genus *Ctenocephalides* have been reported to produce anemia, and rarely, death in dogs, cats, goats, cattle, and sheep (Blackmon and Nolan, 1984; Yeruham et al., 1989; Dryden et al., 1993). Murine typhus, caused by *Rickettsia typhi,* is a febrile disease characterized by development of headaches, chills, and skin rashes, with infrequent involvement of the kidneys and central nervous system (Azad, 1990). The disease occurs in humans and many small mammals, including rats and mice. Traditionally, the primary vector of the organism was the Oriental rat flea, *Xenopsylla cheopis*. However, *C. felis* has also been shown to be a vector of this organism. *Dipylidium caninum*, the common intestinal cestode of dogs, cats and rarely children, develops as a cysticercoid in *C. felis*, *C. canis*, and *P. irritans*.

In addition to the above diseases, flea infestations can also be a severe nuisance. In-door flea infestations and subsequent flea bites can have pronounced effect upon humans, causing irritation, allergic reactions and may place strains on the human-animal bond.

Flea - Host interactions that play a role in understanding the importance of topical and systemic flea control:

Adult cat fleas begin feeding almost immediately once they acquire a host and defecate flea feces (partially digested blood) in as little as 8 to 9 minutes of initiation of feeding (Dryden, 1990). Consumption of blood is necessary for ovarian development and reproduction. Mating occurs after fleas have fed and egg production begins within 24 to 48 hours of females taking their first blood meal (Hudson and Prince, 1958; Akin, 1984). Female cat fleas remain on their host, laying 40 to 50 eggs per day within the pelage, with the eggs dropping off to be deposited any place the flea infested host has access (Dryden, 1989). Adult cat fleas are considered permanently host-associated external parasites. Since fleas cause considerable discomfort to their host and it is on the host where reproduction occurs, elimination of fleas and cessation of reproduction can both be accomplished at the host level.

Flea Control Principles:

Most insecticides do an excellent job of eliminating existing fleas from the host during the initial application. The problem is that reinfestation is a common occurrence. Historically, flea control was achieved through repeated application of on-animal products and application of insecticides and insect growth regulators into the premises. These programs were designed to eliminate existing populations on the host and reduce populations of developing and emerging adult fleas in the surrounding environment. The difficulty with this approach was getting pet owners to consistently follow treatment protocols. Because pet owner compliance was problematic, pets repeatedly acquired new fleas from the premises and infestations became a recurring, if not a continuous, problem. The recent development of insecticides and insect growth regulators with convenient dosage formulations (spot-on, collar, pill, oral suspension, and injectables) and prolonged residual activity, have improved dramatically pet owner compliance and have helped to eliminate recurrent infestations.

Several of these new insecticides and insect growth regulators have even been shown to be extremely effective in eliminating flea infestations in even the most difficult climatic conditions. Field studies conducted in Tampa, FL (USA) demonstrated that fipronil, imidacloprid, and lufenuron (+pyrethrin spray or +nitenpyram tablets) were 100% effective in eliminating established flea populations, without the need for treatment of the premises (Dryden et al. 1999, Dryden et al 2000a, Dryden et al 2000b). Flea infestations can be eliminated using topical and systemic approaches because fleas are either killed prior to initiating reproduction or reproduction is directly inhibited.

While providing the pet relief from fleas is one aspect of treatment, the other goal should be eliminating fleas before they can initiate reproduction. If flea products are applied at the appropriate dose and treatment intervals, there should be adequate residual activity between applications to kill most if not all newly acquired fleas before egg production is initiated. However, flea survival and reproduction may occasionally occur for a variety of reasons. Even with the extended intervals required between applications with the new flea control products, infrequent product application commonly occurs because of owner concerns of pet toxicity or simple lack of compliance with label or veterinary instructions. The inabilities of some pet owners to adequately apply the insecticide and therefore under-dose the pet often allow fleas the opportunity to reproduce between product applications. Bathing and swimming can also reduce insecticide levels of most products. In addition, genetic variability of different flea populations means that none of the currently available residual flea products are 100% effective against all cat flea strains between labeled reapplication periods. These same factors that often allow flea infestations to be persistent and could possibly be the scenario that allows for genetic selection of resistant populations of fleas.

Resistance:

While insecticide resistance is certainly an important issue, the effects of resistance on cat flea control have not been fully ascertained. Of the species of fleas tested, the cat flea is resistant to the greatest number of different categories of insecticides (Rust and Dryden 1997). A field-collected population of cat fleas from Puerto Rico was shown to be resistant to DDT, dieldrin, and malathion (Fox et al., 1968), while a field-collected cat flea strain from Kentucky showed evidence of organophosphate resistance (Schwinghammer et al., 1985). El-Gazzar et al. (1986) found that resistance ratios of adult fleas from a field-collected strain in Florida was 10.0, 9.4, 7.2, 28, 26, and 20-fold resistant to chlorpyrifos, diazinon, propetamphos, bendiocarb, malathion, and carbaryl, respectively. Similarly, Lemke et al. (1989) found that the Florida strain was 6.8-, 5.2-, and 4.8-fold more resistant to cyfluthrin, cypermethrin, and fluvalinate than was the susceptible strain, respectively.

Currently the extent or prevalence of insecticide resistance to fipronil, imidacloprid, lufenuron or selamectin in cat flea populations has not been determined. Resistance is often cited as the reason a particular product has failed, however an alternative explanation for lack of control in many homes may be poor application technique, lack of an understanding of flea biology, and too infrequent reapplications. While pet owner compliance is likely the cause of most control failures it is likely that the historic decrease in performance of permethrin, pyrethroid and organo-phosphate flea products can be attributed at least in part to the development of resistant populations of fleas.

Management of flea infestations and resistance:

Veterinarians should be aware that while the currently available residual adulticides such as fipronil, imidacloprid, selamectin, and insect growth regulators (IGRs) are highly effective, their continued use will exert selection pressure on flea populations and will most likely result in the selection for resistant populations. We must understand that as we apply an insecticide to control an insect population, and kill the more susceptible individuals, we are on the path toward making such populations resistant to the used insecticide; what differs among cases is the speed with which resistance develops (a function of the level of mortality achieved). To delay the development of resistance, provide for long-term flea control, and ensure client satisfaction, veterinarians should institute an integrated approach to flea control.

At first glance, it would appear that the strategies employed in the control of fleas on dogs and cats, and in premises could be incorporated into an Integrated Pest Management (IPM) program. Two basic tenets of the IPM concept are 'management' of the pest population instead of 'controlling' it, and, as applied to pests of animals, that the hosts do not require complete freedom from the attack of the pest to produce or do well. However, the nature of the cat flea/host relationship, in terms of the need for almost complete flea control to prevent FAD, makes this insect difficult to be considered as a pest that can be 'managed' under the IPM requirements and guidelines. Nevertheless, there is a series of strategies available to be implemented under the old 'Integrated Pest Control' concept; these include: 1) educating the home owner on the biology and habitat of fleas infesting their home and pets, 2) use of population assessment techniques to determine presence of fleas and action thresholds, 3) implementation of mechanical control systems, 4) use of biological control methodologies, 5) application of insect growth regulators topically and into the premises, and 6) administration of adulticidal insecticides. A true integration of all these strategies would have a profound impact upon flea populations and likely delay development of resistant population of fleas, especially those that rely on non-chemical control. However, the reality is that while veterinarians or trained technicians and staff often give recommendations for flea control, treatment is ultimately conducted not by trained professionals but by pet owners. Thus, the time, effort, knowledge and cost of implementing a truly integrated control program might be impractical. Therefore, we must find practical alternatives that will provide control and hopefully delay resistance.

It should be remembered that the goals of flea control are elimination of existing fleas on the pet(s), provide for continued elimination of fleas acquired from the flea infested premises and prevention of subsequent reinfestations. Therefore, the first step in a program is the use of an effective flea adulticide to eliminate existing flea infestations on the pet(s). Several currently available insecticides provide excellent elimination of established flea infestations on both dogs and cats; these include imidacloprid, fipronil, nitenpyram, and selamectin. For example, fipronil not only provides for initial flea elimination but also provides for continued residual activity for at least 30 days in both dogs and cats.

As previously indicated, none of these residual adulticides are 100% effective throughout their labeled duration of activity; in addition, compliance problems also allow for flea survival. These surviving fleas may be capable of producing viable eggs. Continued reproduction must be halted to prevent persistent flea infestations and selection for resistant fleas. The reproductive process can be prevented by administration of topical or systemic IGRs. Olsen (1985) found that the fur of cats with 2-10 mg/kg of methoprene prevented eggs from developing into normal adult fleas for at least 43 days. Research results suggest that ovicidal activity is also due to exposure of the adults to IGRs (Palma et al., 1993). Female cat fleas treated with either methoprene or pyriproxyfen and then placed on untreated cats failed to produce viable eggs for 76 and 94 hours, respectively. The use of topical IGRs provides for prolonged residual ovicidal activity, interrupting future flea development, even after residual activity of an insecticide is diminished (Donahue and Young, 1992).

Not only have topically applied IGRs been shown to be ovicidal but orally administered lufenuron also produces ovicidal activity. Female fleas feeding on cats treated with lufenuron at 30 mg/kg produce nonviable eggs for approximately 2 weeks (Zackson et al., 1992) then, as the concentration of lufenuron declines in the blood, some of the eggs will be viable; however, the larvae hatching from those eggs subsequently die. Studies have shown that a single dose of lufenuron can inhibit 98.2 to 100% of egg or larval development for 32 to 44 days (Hink et al., 1994; Blagburn et al., 1994).

While the combined approach of an adulticide and IGR may hold considerable promise, it must be remembered that selection for multiple resistance is possible. The use of mechanical control procedures should be an essential part of integrated flea control. The pet owner will conduct the mechanical control, therefore; education of the client on important aspects of flea biology is essential if any degree of compliance is expected. Helpful procedures include having the pet owner wash the pet's blanket, throw rugs, and pet carrier, in addition pet sleeping and resting areas should be vacuumed thoroughly to help remove flea eggs and larvae. Seat cushions and pillows on sofas and chairs should be removed and vacuumed, and special attention should be given to crevices in sofas and chairs and to areas beneath sofas or beds where flea eggs and feces may drop from the pet and accumulate. Mechanical control should be directed under sofas, beds, chairs, or other structures where pets may not have direct access but where larvae may develop. When pets are allowed to seek shade under the house, this can lead to a rapid buildup of a heavy concentration of fleas.

There are several potential pathogens, parasites or predators mentioned in the literature as possible biological control agents for cat fleas, however, only the entomopathogenic nematode *Steinernema carpocapsae* has been used commercially with any degree of success and is not commonly available in many countries. In addition successful use of these nematodes require soil moisture levels of $\geq$20%, which can be difficult to achieve in many outdoor environments during the summer.

As indicated above, several new topical and systemic compounds can eliminate existing flea populations without the need for premise treatments. However, in cases of massive flea infestations or severe pet or human flea allergy, applications of adulticides and IGRs into the premises may be necessary.

Summary:

A practical integrated flea control program that can be put into practice by pet owners should encompass: 1) educating the pet owner on important aspect of the flea life cycle, 2) implementation of mechanical control procedures, 3) the use of an effective adulticide and 4) administration of a residual IGR to suppress reproduction once the activity of the adulticide falls below 100%. We believe that a product or combination of products that provide both adulticidal activity and insect growth regulating activity would greatly benefit flea control efforts and may delay the on-set of resistance. In addition, it is likely that pet owner compliance could be increased substantially if a single residual acting product that contains or has both adulticidal and egg inhibition activity could be used.

References:

Akin DE. 1984. Relationship between feeding and reproduction in the cat flea, *Ctenocephalides felis* (Bouche). M.S. Thesis, University of Florida, Gainesville, FL. pp 125.

Amin OM. 1966. The fleas (Siphonaptera) of Egypt: distribution and seasonal dynamics of fleas infesting dogs in the Nile Valley and Delta. J. Med. Entomol. 3:293-298.

Amin OM. 1976. Host associations and seasonal occurrence of fleas from southeastern Wisconsin mammals, with observations on morphologic variations. J. Med. Entomol. 13:179-192.

Azad AF. 1990. Epidemiology of murine typhus. Annu. Rev. Entomol. 35:535-569.

Baker KP, Hatch C. 1972. The species of fleas found on Dublin dogs. Vet. Rec. 91:151-152.

Beresford-Jones WP. 1981. Prevalence of fleas on dogs and cats in an area of London. Small Anim. Pract. 22:27-29.

Blackmon DM, Nolan MP. 1984. *Ctenocephalides felis* infestation in Holstein calves. Agri. Prac. 5:6-8.
Blagburn BL, Vaughan JL, Lindsay DS, Tebbitt GL. 1994. Efficacy dosage titration of lufenuron against developmental stages of fleas (C*tenocephalides felis felis*) in cats. Am. J. Vet. Res. 55:98-101.
Bowen PM, Caldwell NJ. 1982. Use of cythioate to control external parasites on cats and dogs. Vet. Med. Sm. An. Clin. 77:79-80.
Donahue WA, Young R. 1992. Evaluating a synergized pyrethrin/(S)-methoprene spray against feline flea infestations. Vet. Med., 87:999-1007.
Dryden MW. 1988. Evaluation of certain parameters in the bionomics of *Ctenocephalides felis felis* (Bouche, 1835). M.S. thesis, Purdue Univ., W. Lafayette IN.
Dryden MW. 1989. Host association, on-host longevity and egg production of *Ctenocephalides felis felis*. Vet. Parasitol. 34:117-122.
Dryden MW. 1990. Blood consumption and feeding behavior of the cat flea, *Ctenocephalides felis felis* (Bouché 1835). Ph.D. Dissertation, Purdue University, W. Lafayette, IN. pp 128.
Dryden MW, Broce BA, Moore WE. 1993. Severe flea infestation on dairy calves. J. Am. Vet. Med. Assoc. 203:1448-1452, 1993.
Dryden MW, Perez H, Ulitchny D. 1999. Control of flea populations on naturally infested dogs and cats and in private residences with either topical Imidacloprid spot application or the combination of oral lufenuron and pyrethrin spray. Am. J. Vet. Med. Assoc.1;215(1):36-9.
Dryden MW, Magid-Denenberg T, Bunch S. (In-Press) Control of fleas on naturally infested dogs and cats and in private residences with topical spot applications of fipronil or imidacloprid. Vet. Parasitol.
Dryden MW, Magid-Denenberg, Bunch S, Boyer J. (In-Press) Control of fleas on naturally infested dogs and cats and in private residences with lufenuron and nitenpyram. Comp. Cont. Ed. Pract. Vet.
El-Gazzar LM, Milio J, Koehler PG, Patterson RS. 1986. Insecticide resistance in the cat flea (Siphonaptera: Pulicidae). J. Econ. Entomol. 79:132-134.
Fox I, Rivera GA, Bayona IG. 1968. Toxicity of six insecticides to the cat flea. J. Econ. Entomol. 61:869-870.
Guzman RF. 1984. A survey of cats and dogs for fleas; with particular reference to their role as intermediate hosts of *Dipylidium caninum*. N.Z. Vet. J. 32:71-73.
Harman DW, Halliwell RE, Greiner EC. 1987. Flea species from dogs and cats in north-central Florida. Vet. Parasit. 23:135-140.
Hink WF, Zakson M, Barnett S. 1994. Evaluation of a single oral dose of lufenuron to control flea infestation in dogs. Am. J. Vet. Res. 55:822-824.
Hudson BW, Prince FM. 1958. A method for large scale rearing of the cat flea, *Ctenocephalides felis felis* (Bouché). Bull. W.H.O., 19:1126-1129.
Kristensen S, Haarov N, Mourier H. 1978. A study of skin diseases in dogs and cats. IV. Patterns of flea infestation in dogs and cats in Denmark. Nord. Vet.-Med. 30:401-413.
Lemke LA, Koehler PG, Patterson RS. 1989. Susceptibility of the cat flea (Siphonaptera: Pulicidae) to pyrethroids. J. Econ. Entomol. 82:839-841.
Olsen A. 1985. Ovicidal effect on the cat flea, *Ctenocephalides felis* (Bouché), of treating fur of cats and dogs with methoprene. International Pest Control 27:10-13, 16.
Palma KG, Meola SM, Meola RW. 1993. Mode of Action of pyriproxyfen and methoprene on eggs of *Ctenocephalides felis* (Siphonaptera: Pulicidae). J. Med. Entomol. 30:421-426.

Rust MK, Dryden MW. 1997. The biology, ecology and management of the cat flea. Ann. Rev. Entomol. 42:451-473.
Schwinghammer KA, Ballard EM, Knapp FW. 1985. Comparative toxicity of ten insecticides against the cat flea *Ctenocephalides felis* (Siphonaptera: Pulicidae). J. Med. Entomol. 22:512-514.
Yeruham I, Rosen S, Hadani A. 1989. Mortality in calves, lambs and kids caused by severe infestation with the cat flea, *Ctenocephalides felis felis* (Bouche, 1835) in Israel. Vet. Parasitol. 30:351-356.
Zakson M, Hink FW, Mackichan JJ. 1992. Fate of the benzoylphenyl urea cga-184699 in the cat flea *Ctenocephalides felis*. Pestic. Sci. 35:117-123.