Flea and Tick Control: Real Medicine

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The cat flea, *Ctenocephalides felis* can inflict repeated painful bites and cause allergic dermatitis in both animals and humans. The cat flea can also serve as the vector of *Rickettsia typhi*, and *Bartonella hensalae*. It is also the intermediate host for filarid and cestode parasites. Therefore, control of flea infestations is necessarily not only because fleas are nuisance pests but also to reduce disease transmission.

Most insecticides do an excellent job of eliminating existing fleas from the host after the initial application. The problem is that reinfestation is a common occurrence. The first step in flea control is the elimination of existing fleas infesting on pets. This is necessary to eliminate pet discomfort. Several insecticides are available that consistently achieve this goal such as fipronil, imidacloprid, nitenpyram and selamectin. Killing fleas resident on the dog and cats is relatively easy with these insecticides. The difficult part of flea control is providing continued pet relief from the fleas emerging from the infested premises.

Historically stopping flea development in the premises (breaking the flea life cycle) and providing continued pet relief was accomplished by repeated application of insecticides and/or insect growth regulators (IGRs) into the premises and frequent applications of sprays, powders, dips or bathes to the pet. Such an approach while technically effective was often impractical for many pet owners. Modern topical and systemic residual insecticides and IGRs have changed the way most flea infestations are eliminated. The current approach is suppression of flea reproduction on the dog or cat or "breaking the flea life cycle at the host level".

Administration of residual insecticides such as fipronil, imidacloprid, and selamectin kill many newly acquired fleas (within 24 hours) before they can initiate reproduction. Alternatively the administration of topical or oral IGRs such as lufenuron, methoprene or pyriproxyfen kills developing embryos or larvae within the egg, effectively halting flea reproduction.[1] While not an IGR, selamectin has also been found to be highly ovicidal.[2] These control methods essentially turn the treated dog or cat into a "living flea vacuum". As treated pets move through the infested premise they acquire newly emerging fleas. These fleas which are either killed rapidly or had their eggs rendered nonviable are unable to produce future generations of fleas. Once all existing life stages have developed and emerged the flea problem is over.

In theory residual monthly flea control products should be adequate to kill or inhibit the viable reproduction of most newly acquired fleas. However, in some cases flea survival and reproduction can occur if the residual activity of the insecticide is less than 100%, insecticide levels diminish and the speed of flea kill slows a couple weeks after treatment, products are infrequently or mis-applied, pets are bathed or swim thereby reducing insecticide levels and genetic isolates with variable susceptibility are encountered.

A combined adulticide and ovicidal approach may offer an effective long term solution. In a comparison study selamectin and fipronil (s)-methoprene provided >97.9% efficacy against adult fleas through day 24.[2] The high rate of flea kill also meant that egg production was markedly reduced. The pronounced ovicidal action of methoprene and selamectin meant that adult flea

emergence either did not occur or was minimal from day 2 through day 37. The combination of ovicidal and adulticidal activity provides for not only reduced egg production but also reduced viability of the few eggs laid. This makes for pronounced suppression of subsequent flea generations.

Similar population suppression can be achieved using a combination of other IGRs and adulticides such as lufenuron and nitenpyram. Field studies conducted in Tampa, FL and Australia demonstrated that lufenuron plus either a pyrethrum spray or nitenpyram tablets were highly effective in eliminating established flea populations, without the need for treatment of the premises.[3-5]

While great advances have been made in flea control technology, tick control can still be a difficult prospect. Controlling tick infestations is important not only because ticks are nuisance parasites of dogs and cats, but also because they are vectors of a variety of bacterial and protozoal diseases. Control of tick infestations is therefore, a major component of disease prevention. Control and prevention of tick infestations should begin with an understanding of the life history of the tick(s) encountered in your area. It is important to understand that control of ticks and tick-transmitted disease is often difficult due to: 1) multiple tick species, 2) prolonged life cycles, 3) high reproductive capacity, 4) numerous wildlife hosts, and 5) other tick life stages in the environment. Veterinarians, technicians and staff need to be educated on the various aspects of tick biology and control so that they can then educate the pet owner. Reinfestation pressure and failure to control massive tick refugia likely causes most real and perceived product failures.

It must be remembered and related to pet owners that most ticks infesting dogs in North America are considered 3-host ticks, with each successive stage feeding on separate hosts after molting. The adult female feeds until engorged and then drops off and deposits her eggs in the environment. Female ticks then die after depositing an egg mass typically numbering in the thousands. The tick species most commonly encountered infesting dogs in the U.S. are *Amblyomma americanum, Amblyomma maculatum, Dermacentor variabilis, Dermacentor andersoni, Ixodes scapularis,* and *Rhipicephalus sanguineus*. Other tick species may occasionally be recovered from dogs including, the one host tick *Otobius megnini*. While not as commonly infested as dogs, cats can be afflicted with *Amblyomma americanum, Dermacentor sp.* and *Ixodes scapularis.*

Historically the most common tick infesting dogs in North America was *Dermacentor sp.* In the typical life history of *Dermacentor variabilis* larva usually feed on small mammals such as various rats and mice.[6-7] Nymphs can be found feeding on dogs, rabbits, raccoons, opossums, and other medium to small sized mammals. The adult tick will feed dogs, horses, cattle, and other large mammals, including man. The *Dermacentor sp*. ticks are primarily of importance because they vector Rocky Mountain Spotted Fever (*Rickettsia rickettsii*) and Cytauxzoonosis (*Cytauxzoon felis*). The species involved in the transmission of *R. rickettsii* in the United States are the American dog tick, *Dermacentor variabilis* and the Rocky Mountain wood tick *Dermacentor andersoni.*

A tick that appears to be increasing in importance across the southern, midwestern and eastern states is *Amblyomma americanum* the Lone Star Tick. Once considered primarily a southern tick, its geographic range appears to have expanded.[8] A variety of environmental and host factors have contributed to the increased range and frequency of occurrence. *Amblyomma americanum* occurs most commonly in woodland habitats with dense underbrush. Reforestation in urban and rural habitats therefore provides increased areas of suitable habitat for survival of these ticks.

While *A. americanum* has a wide host range parasitizing a number of mammalian and avian hosts, increased populations and expansion of range of two different hosts have likely contributed greatly to *A. americanum* expansion.[8] The well-documented expansion of white-tailed deer populations has had a major impact upon *A. americanum* populations. The white-tailed deer is considered a preeminent host for *A. americanum*, because all three-life stages of the *A. americanum* will feed successfully upon the white-tailed deer and ticks generally fall off into wooded habitats after engorgement.[8] Another host that utilizes similar habitats and is an excellent host for larval and nymphal *A. americanum* is the wild turkey. As wild turkey populations have increased across much of North America, they have reintroduced and increased populations of *A. americanum*.[8] Many animals can serve as host for this aggressive tick. Immature stages can be found on a variety of birds, rodents, rabbits, dogs, cats, fox, coyotes, humans, and deer. Adult ticks can also feed on a variety of hosts including deer, cattle, horses, sheep, dogs, cats, and humans.

While *A. americanum* is considered a major nuisance parasite, it is also a vector of *Ehrlichia chaffensis, E. ewingii* & *Borrelia lonestari*. It has also been implicated in the transmission of *Francella tularensis*. While not as common, the Gulf Coast Tick, *Amblyomma maculatum* will infest dogs and carries *Hepatozoon americanum*, the etiologic agent of American Canine Hepatozoonosis. The transmission of this disease is unique in that dogs need to ingest the ticks to become infected.[9]

Another tick that is of considerable importance because its ability to inhabit dwellings and vector *Ehrlichia canis* is the brown dog tick, *Rhipicephalus sanguineous*. [6-7] It also is a 3-host tick but every stage (larva, nymph and adult) can successfully feed on dogs. Larvae will also feed on rats and nymphs on rabbits, but prefer to feed on dogs. It is the only species of tick that infests human dwellings and kennels in North America. Infestations of homes or kennels are distressing to pet owners and are extremely difficult to eradicate.

The last group of 3-host ticks to be discussed in this paper are in the genus Ixodes. *Ixodes scapularis*, the Deer tick or Lyme disease tick is found throughout the Midwest and Eastern US. It is a small 3-host tick, with larvae being the size of a pinhead and nonengorged adults being less than 2mm. Larvae feed on a variety of small mammals, including mice, squirrels, voles, shrews, and raccoons.[6-7] Nymphs feed on mice, squirrels, chipmunks, raccoons, opossums, shrews, cats, and man. Adults feed primarily on white tail deer and occasionally on dogs, raccoons, and other wildlife. *Ixodes scapularis* is the primary vector of *Borrelia burgdorferi* in the central and eastern United States, and is also the vector of *Anaplasma phagocytophilum* (formerly *Ehrlichia phagocytophila*; HGE). In the western US. *I. pacificus* is the vector of *B. burgdorferi* and in parts of the Rocky Mountains *I. neotomae* can vector B. burgdorferi.[10]

Numerous studies have been conducted to evaluate the efficacy of various acaracides against ticks infesting dogs. Differences in study design make comparison of the results of the various studies difficult. The differences encountered in these studies may be due to various factors including; 1) the time post infestation or treatment when live-dead assessments are conducted, 2) tick species used, 3) laboratory or field study, 4) size or hair coat density of dog, 5) unknown acaracide susceptibility of tick isolate, 6) other unknown factors. Regardless of these difficulties the data suggests that three compounds appear to have the greatest activity against ticks; amitraz (impregnated collar), fipronil (spray and spot-on formulations) and permethrin (spray and spot-on formulations).[11-15] The data also indicates that selamectin has activity ticks[16], but the speed of kill of selamectin is slower than the previously mentioned compounds.

Variable results of the published trials mirrors reports this author has received from practicing veterinarians. In various parts of the U.S. practitioners will report that one acaracide

performs better than another product. In addition, sometimes within the same geographic region practitioners may disagree on which performs best. Whether it is practice art or science, perception of efficacy differences do exist. The causes of which are unknown.

Rapid tick kill or prevention of attachment and feeding is important in the prevention of tick transmitted diseases. A recently article reviewed the available data on acaracides and disease transmission times.[17] Various transmission studies have been conducted, none using dogs, to determine time from attachment until transmission of various pathogens occurred. Results varied depending upon organism, host, tick species, numbers of ticks used and other factors.[17] While it is generally accepted that killing ticks within 24 to 48 will prevent disease transmission, this may not always be correct. However, it is true that following host acquisition a period of time is necessary for tick attachment, feeding, pathogen activation and then excretion. If ticks can be repelled, prevented from attaching or killed outright during this time period, then disease transmission may be prevented. However, more research is needed to determine the exact transmission times of the various pathogens to dogs and cats.

Only a few studies have been conducted to evaluate the performance of various acaracides to prevent tick-transmitted diseases.[18-21] When dogs were infested with adult *I. scapularis* containing *B. burgdorferi*, seven days after placement of amitraz-impregnated collars none of the dogs seroconverted to *B. burgdorferi*.[18] Fipronil spray prevented transmission of *B. burgdorferi* after dogs were exposed to adult *I. scapularis* 7 and 28 days after treatment.[19] A permethrin-imidacloprid combination spot-on prevented seroconversion to *B. burgdorferi* when dogs were exposed to infected adult 1 scapularis ticks 7 days after treatment.[20]

To date only one study has been published investigating the efficacy of an acaracides to prevent the transmission of E. canis to dogs by *R. sanguineus*.[21] A year-long, prospective study in Africa was conducted to evaluate the ability of fipronil spot-on to prevent transmission of *E. canis.* Two groups of French army dogs in Djibouti and Dakar were used in this study. Control dogs were native and police dogs. This area was highly endemic for *R. sanguineus* and *E. canis,* demonstrated by seroprevalence in untreated control groups ranging from 21.7% to 100%. Dogs administered monthly applications of fipronil spot-on had seroprevalence rates of 2.7% in Djibouti and 5.5% in Dakar.[21]

While the above studies demonstrate considerable efficacy against ticks and disease transmission, it must be cautioned that pet owners should not be told that a particular acaracide will completely prevent disease transmission. Too many variables exist once a particular product has been dispensed. While the first dose of a product is likely applied in the veterinary clinic, subsequent doses are administered or collars are attached by owners. It must be remembered that you have little control over what a client may or may not do in the timing or diligence of product application.

Occasionally a label recommendation of topical acaracides does not appear to control the problem. This may be real or perceived based upon pet owner expectations of product performance and reinfestation rates. If additional control measures are needed, products may be combined, frequency of application increased or the problem can be approached by attempting to eliminate ticks in the environment. If additional measures are deemed necessary clients should be informed and notations made in the pets record before extra label uses are conducted.

If possible start by destroying tick habitat. Refuge areas for ticks and wild mammals that can also serve as hosts for ticks, such as grass, weeds, and brush piles, between runs and along buildings, should be removed. Controlled burning of forest canopy or grass lands has produced at least short term reduction in populations *of A. americanum* and *Ixodes scaupularis*.[22,23].

If brown dog ticks are the problem, these ticks can crawl up into the ceilings and cracks and crevices along floors to molt or lay eggs. Acaracides should be sprayed into cracks and crevices, behind and under cages and along the boards in the ceiling. Foggers can also be used to spread acaracides into areas difficult to reach with directed aerosols. Effective compounds for environmental tick include cyfluthrin, permethrin and s-fenvalerate. Always follow state laws and label recommendations as to application of insecticides/acaracides into human dwellings. Following application, make sure the acaracide is dry before you allow animals or humans back into the premises to minimize toxicity problems.

These same compounds can also be used outdoors. Rarely is broadcast application of acaracides necessary in the yard. Spot treatment along fences, kennels, or shaded areas is preferred. Finally, restricting pet access from tick-infested environments may be necessary.

Finally we need to remember that fleas and ticks produce disease by consumption of blood, injection of salivary proteins and transmission of infectious agents. Reactive treatments to eliminate existing infestations are frequently too late to prevent disease and distress in pets and their owners. Many dogs and cats would benefit greatly by being placed on preventative flea and/or tick control products throughout their life. Since substantial geographic differences occur in flea and tick prevalence and seasonality, prevention programs should be tailored accordingly.

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