Banfield PET HOSPITAL

BARK

Putting Knowledge into Practice

Banfield Applied Research & Knowledge Team

April 2010

Flea Literature Review

By PATRICK SHEARER, BVMS, PhD | Contributing Author

CLINICAL BOTTOM LINE

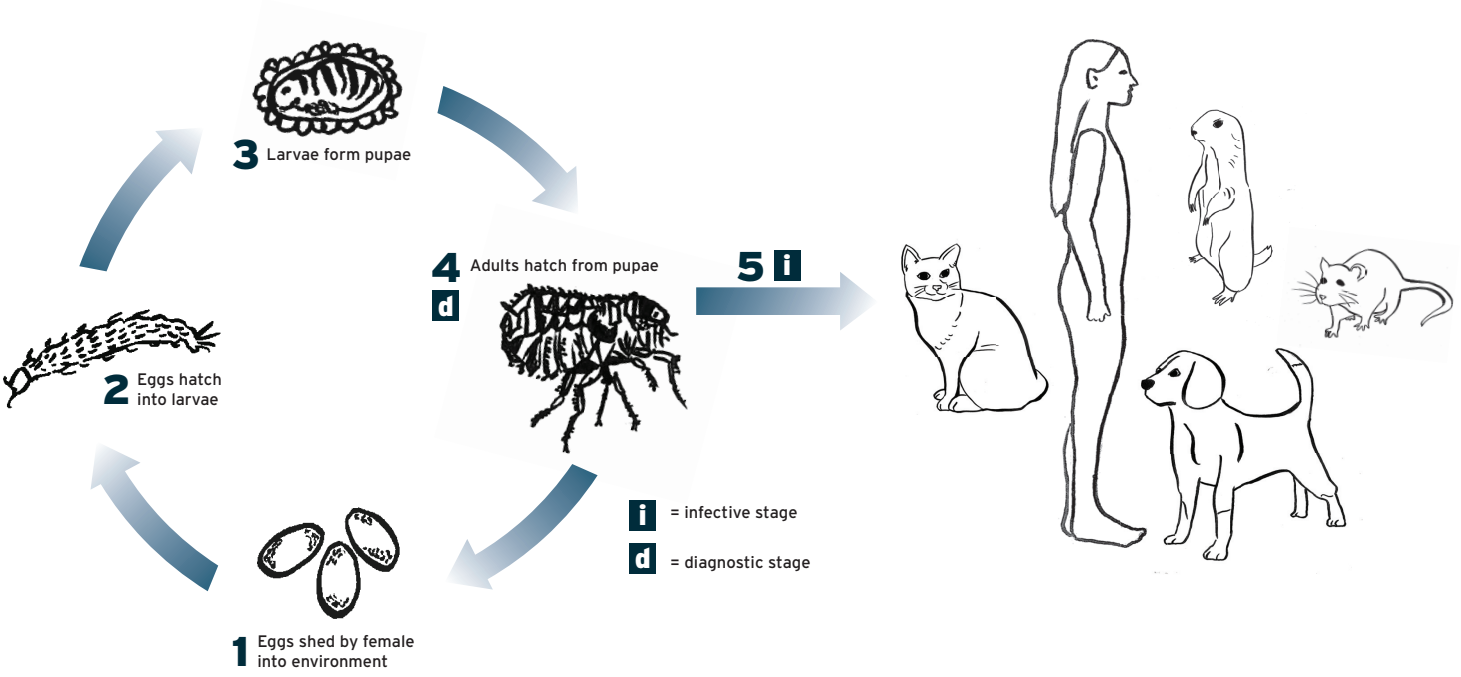
- Fleas are common and important ectoparasites of companion animals. *Ctenocephalides felis* accounts for the majority of fleas on dogs and cats.
- Under the right conditions, fleas are prolific breeders; female fleas may lay up to 2,000 eggs in their lifetime. Fleas can also survive long periods of starvation, and pupated fleas can survive up to 12 months in the pupa in the absence of a host.
- Flea infestation can cause flea allergy dermatitis, which leads to intense pruritis, hair loss and secondary skin infections in affected animals.
- Fleas also transmit a number of diseases, some of which are zoonotic.
- Many suitable products are approved for the treatment and prevention of fleas. These are available in the form of shampoos, rinses, sprays, mists/fogs and spot-ons.
- Effective control and prevention depends on a good knowledge of the life cycle, identification and cleaning of source points and application of appropriate chemicals on a regular basis.

INTRODUCTION AND LIFE CYCLE

Worldwide, about 2,500 species of fleas have been described and of these, *Ctenocephalides felis* is one of the most common.[1,2] *C felis* accounts for 92 to 99 percent of fleas on dogs and cats, respectively, and can also infest humans and other domestic and wild animals. It undergoes four major life stages, from egg to larva, then pupa and adult, with a complete metamorphosis between each stage (*Figure 1,* page 2).[2] The entire life cycle takes between 12 and 174 days to complete, depending on ambient temperature, humidity and availability of hosts.[1,2] At any point in time, approximately 57 percent of fleas exist as eggs, 34 percent as larvae, 8 percent as pupae and 1 percent as adults.[2]

Flea eggs are about 0.5mm long, oval, white and non-sticky, and develop within one to 10 days of being deposited on the host and falling off into the environment.[2,3] Larvae are approximately 1 to 2mm long, thin and covered in short hairs.[2] They live in areas frequented by hosts and seek out dark, warm, humid places—usually carpets, bedding, under furniture or garden debris.[1,2] Larvae feed on organic material and the feces of adult fleas, which are rich in partially digested blood.[1,2] Larvae are very sensitive to heat and desiccation, as they can only survive if humidity is over 50 percent, soil moisture is 1 to 20 percent and temperature is between 3° and 35°C for more than 40 hours per month.[2] Larvae molt twice within two to three weeks, then the mature larvae produce a sticky cocoon from silk made by their salivary glands.[1,2] Environmental debris sticks to the cocoon, aiding camouflage. Pupation takes five to 14 days, depending on environmental conditions. Areas which are suitable for a high rate of survival from pupa to adult are known as "hot spots" or "source points" and are an important aspect of flea control.[2] The pre-emerged adult can survive in the cocoon for six to 12 months if protected from desiccation. Changes in pressure, light, temperature and carbon dioxide levels are thought to be stimuli for emergence.[1,2] Newly emerged fleas survive

Figure 1: Flea Life Cycle



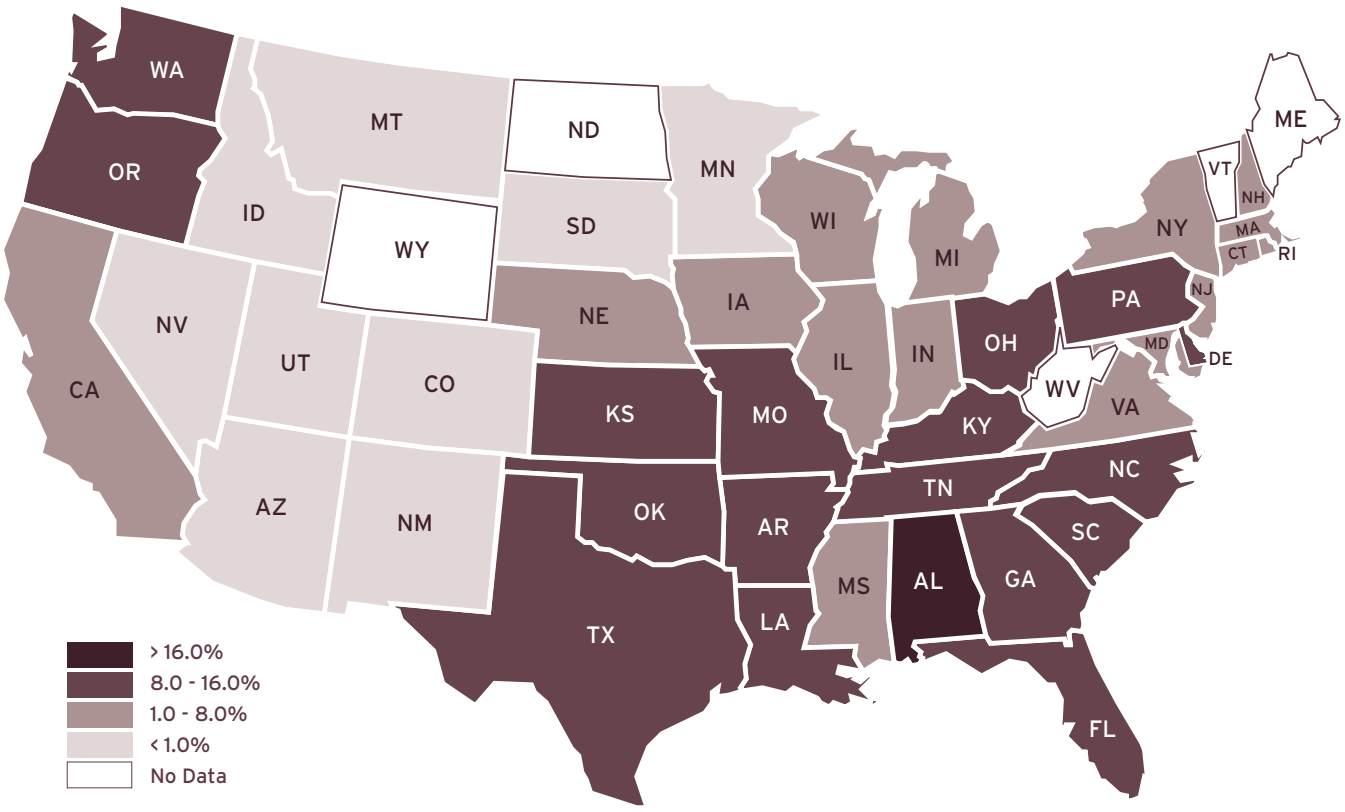
between 10 and 62 days and the total adult lifespan can be up to a year, with long periods of starvation in between.[1-3]

Once an adult flea has found a host, feeding begins almost immediately. Adult fleas feed on capillary blood and pump saliva into the bite wound to act as an anticoagulant.[2] Female fleas consume about 15 percent of their body weight in blood per day and about 72 fleas will remove 1 mL of blood from the host.[2] Male fleas consume less than females but feed more often. While feeding, adult fleas excrete large quantities of partially digested blood. Once fleas feed and begin reproduction, they are dependent on a constant source of blood, or death occurs within days.[2] Adults begin to reproduce very soon after emerging from the cocoon (within eight to 24 hours) and mate multiple times during their lifespan.[1,2] The adult female lays several hundred eggs a day or two later and may deposit up to 700 to 900 within three to four weeks and up to 2,000 eggs in her lifetime.[1,2]

PREVALENCE AND RISK FACTORS

Although the environmental characteristics favorable for flea development have been described, there is little published data on prevalence or risk factors for infestation. The risk for environmental infestation has been modeled in Europe, based on weather patterns and activity matrices (with optimal activity between 20°C to 30°C and >70 percent humidity).[4] The models are available at www.FleaTickRisk.com. It should be noted, however, that this estimate does not

Figure 2: Flea Infestation Prevalence by State—Based on Banfield Data



take the indoor environment into account, so risk for infestation indoors is independent of these models and may be higher depending on household heating and flea prevention habits.[4] In North America, prevalence varies according to geographic region, as shown in *Figure 2*.

CLINICAL CONSEQUENCES OF INFESTATION

Fleas can cause allergic skin disease in dogs and cats and are able to transmit a range of infectious diseases to dogs, cats and humans. *C felis* readily feeds on humans, so flea infestation itself can be a major cause for concern.[5] Flea allergy dermatitis (FAD) is one of the most common veterinary dermatological conditions in the world.[2] The majority of dogs (61 percent) develop clinical signs between 1 and 3 years of age and signs are uncommon in dogs less than 6 months old.[2] Hypersensitivity develops to certain proteins in the flea's saliva, and the degree of hypersensitivity may decrease with age and continued exposure.[2] Clinical signs include salivary stains, papules, crusts, excoriations and erythema in a wedge-shaped pattern over the lumbosacral region, caudal thighs,

proximal tail, ventral abdomen and around the umbilicus. Dogs and cats with chronic disease develop alopecia and skin becomes lichenified and hyperpigmented.[2] Secondary infections develop, leading to a characteristic odor related to proliferation of *Staphylococcus intermedius* and *Malassezia pachydermatis*. Many dogs and cats with FAD may have recurrent tapeworm (*Dipylidium caninum*) infestations resulting from flea ingestion. Diagnosis of FAD is based on the clinical signs, age of onset, distribution of skin lesions and demonstration of fleas and/or flea feces.[2] Flea feces can be detected by brushing the dog or cat's coat and collecting the dirt on a piece of paper, then lightly moistening the paper. The hemoglobin in the flea feces will diffuse out and form a red ring around the specks of debris.[2,3] Demonstration of fleas on dogs or cats with FAD may be difficult, as they typically remove most or all of their fleas as a result of excessive grooming.

Fleas can transmit a variety of infectious diseases to dogs, cats and humans. These diseases and their veterinary or zoonotic significance are listed in *Table 1,* page 5.

TREATMENT AND PREVENTION

Products suitable for the treatment and control of fleas are available in many different formulations, such as shampoos, rinses, sprays, mists/fogs and spot-ons. These formulations often also offer various different combinations of chemicals, depending on their intended use (*e.g.*, indoors vs. outdoors or dogs vs. cats). When applying chemicals to the environment, either indoors or outdoors, hand spraying by professionals or owners is recommended; this allows the product(s) to be applied directly to source points. Large pieces of furniture or debris must be moved to ensure the spray covers areas of larval migration. Outdoor sprays should be concentrated on areas with shade, mild temperatures and organic matter such as shrubs, dog houses, garages or other areas where dogs or cats may rest.

Treatment of FAD is a three-step process, which involves the elimination and prevention of fleas with on-animal and environmental control, providing relief from pruritis with a brief course of short-acting corticosteroids, and treatment of secondary infections with oral and/or topical medications.[2] The aim of this process is to eliminate adult fleas on all animals in the house as well as the immature fleas in the environment. This involves physical/mechanical and chemical methods. Source points, such as bedding and resting areas, should be identified and treated aggressively. This includes washing bedding, blankets and rugs; vacuuming carpets, cushions and pillows, and clearing away dead vegetation from resting areas outside. Chemicals suitable for flea control are described in *Table 2,* page 6.

CONCLUSIONS

Fleas are an extremely common parasite of dogs and cats. Because of their ability to reproduce in great numbers, their ability to feed on a wide variety of hosts and the unpleasant effects of their bite, they are an extremely important ectoparasite in the domestic environment. As well as the direct effect of their parasitism, they are responsible for potentially severe allergic reactions in cats and dogs and can transmit a range of diseases to both humans and animals. Because of their high reproductive rate and the preference of their larvae for hard-to-reach places, flea

Table 1: Infectious Diseases Transmitted by Fleas

Pathogen (Common Name)	Life Cycle/Transmission	Clinical Signs	Diagnosis
Dipylidium caninum (tapeworm)[6-8]	Eggs are produced by adult tapeworms and excreted by the host. Eggs are ingested by flea larvae and cysticercoids develop in adults. Fleas ingested by hosts and tapeworms develop in the gastrointestinal tract.	Usually none. Proglottids are sometimes visible in the feces.	Fecal analysis demonstrating eggs
Dipetalonema reconditum[3]	Fleas ingest microfilariae, which develop to third-stage infective larvae and are re-transmitted to a canine host.	None	Appears similar to *Dirofilaria immitis* on blood smears or microfilarial concentration tests
Feline parvovirus (feline panleukopenia)[3]	Can be transmitted from infected cats to susceptible cats	Vary from subclinical to peracute death	Serology or polymerase chain reaction (PCR)
Rickettsia typhi (Murine typhus)[8-10]	Rats are the typical host. *C felis* can transmit *R typhi* from infected opossums to humans.	Disease has not been reported in animals. Clinical signs in humans include flu-like symptoms and a macular rash.	Serology
Yersinia pestis (plague)[8,11]	Usually transmitted by fleas between wild rodents, but fleas can also transmit *Y pestis* to cats or humans.	**Bubonic:** fever and lymphadenopathy. **Pneumonic:** respiratory disease with hemorrhagic sputum. **Septicemic:** systemic disease without a rash. Cats most commonly develop the bubonic form.	Fluorescent antibody assays on lymph node aspirates
Bartonella henselae (cat scratch disease)[12,13]	Cats are the reservoir host, the agent is transmitted by fighting and by fleas.	Cats are usually asymptomatic. Humans usually develop a benign lymphadenopathy, but can also cause encephalitis, endocarditis, hemolytic anemia, hepatosplenomegaly, glomerulonephritis, pneumonia, relapsing bacteremia and osteomyelitis.	Serology or PCR
Mycoplasma haemofelis and *M haemominutum* (feline infectious anemia)[14,15]	Cats are the reservoir host. The exact method of transmission is unknown, however fleas are suspected to be involved and the disease may be transmitted by blood transfusions.	Mild to severe anemia, with lethargy, weakness and cyclic fevers	Blood smear or PCR
Rickettsia felis[9,10,14,16,17]	*R felis* has been detected in fleas from cats, dogs and opossums and in opossum tissues. The role of mammals and specifically the role of companion animals is unknown.	No clinical signs have been reported in animals. Causes a typhus-like illness in humans.	PCR

Table 2: Products Suitable for the Treatment and/or Prevention of Fleas

Product	Mode of Action	Description	Contraindications/ Comments
Pyrethrins [2,18-20]	Affects sodium channels in nerves, leading to repeated nerve depolarization, paralysis and death	Derived from certain species of chrysanthemum. Most are combined with synergists to enhance stability and effectiveness.	Suitability for use in different species and ages varies for each product
Pyrethroids [2,18,21,22]	Affects sodium channels in nerves, leading to repeated nerve depolarization, paralysis and death	Synthetic analogues of pyrethrins. More stable but slightly more toxic.	Can be toxic to cats, may cause contact dermatitis
Rotenone[2]	Interferes with the electron transport chain in mitochondria	Extracted from the root of the derris plant	Extremely toxic to fish. Safe for use in mammals but not commonly used
D-limonene and linalool [2,18]	Has a solvent action on cuticular lipids, leading to desiccation and death	Derived from citrus pulp	May cause severe toxic reactions in cats
Carbamates (carbaryl, propoxur, bendiocarb)[2,18,19,21]	Inhibition of acetylcholinesterase at nerve junctions	Related to organophosphates. Many different formulations.	Potentially very toxic, especially to cats and young animals
Nitenpyram [2,20,22]	Inhibits the nicotinic acetylcholine receptor of insects	Flea adulticide. Rapid kill (within 30 minutes) but short duration of action (24-48 hours).	Very low mammalian toxicity, but should not be used in animals under 2 lbs of body weight or 4 weeks of age
Imidacloprid[2,18,29,22]	Inhibits the nicotinic acetylcholine receptor of insects	Flea adulticide and larvicide	Do not use on puppies under 7 weeks or kittens under 8 weeks old. The formulation containing permethrin (for use as a flea and tick preventive in dogs) should not be used on cats.
Fipronil[2,20,22]	Phenylpyrazole antiparasitic. Interferes with the passage of chloride ions in gamma-aminobutyric acid (GABA) channels, disrupting central nervous system (CNS) activity.	Adulticide. Often combined with S-methoprene, which mimics juvenile flea growth hormone to prevent production of eggs and larvae.	Do not use on puppies or kittens less than 8 weeks of age. Contraindicated in rabbits. Do not bathe animals within 48 hours of application.
Selamectin[2,22]	Avermectin antiparasitic. Enhances chloride permeability or release of GABA at presynaptic neurons causing paralysis of the parasite.	Has ovicidal and larvicidal activity against fleas	Use with caution in sick, underweight or debilitated dogs and cats or avermectin-sensitive Collies. Not recommended for use in dogs and cats under 6 weeks of age.
Sodium polyborate [2,23]	Poisonous effects of boron	Stomach poison and insect cuticle wax absorber. Larvae ingest the powder and are killed before they pupate.	High margin of safety around animals, but should not be used on animals that groom a lot (*e.g.*, cats and rabbits).
Insect growth regulators (methoprene, fenoxycarb, pyriproxyfen)[2,20,22]	Mimics insect juvenile growth hormone	Halts metamorphosis and larval development. Ovicidal and larvicidal, often combined with an adulticide.	Low toxicity in mammals. Formulations containing pyrethroids should not be used on cats.
Insect development inhibitors (lufenuron) [2,20,22]	Inhibits chitin synthesis	Prevents flea larvae developing into adults. Does not kill adult fleas.	The cat labeled product should not be used in dogs.

control can be frustrating. This, coupled with the wide range of innate defense mechanisms (such as the exoskeleton of adults or the protective cocoon of pupae) has led to the development of a wide range of chemicals for use in treatment and control. Effective control and prevention depends on a good knowledge of the life cycle, identification and cleaning of source points, and application of appropriate chemicals on a regular basis.

ABOUT THE AUTHOR

Patrick Shearer, BVMS, PhD, graduated from Murdoch University School of Veterinary and Biomedical Sciences in Perth, Western Australia. Dr. Shearer joined the Banfield Applied Research and Knowledge (BARK) team as an associate medical advisor in 2009. For more information, or to contact the BARK team, e-mail: BARK@banfield.net.

Banfield® PET HOSPITAL

REFERENCES

1. Mehlhorn H. Armstrong PM. *Encyclopedic reference of parasitology online*. http://parasitology.informatik.uni-wuerzburg.de. Accessed March 11, 2010.
2. Sousa CA. Fleas, flea allergy and flea control. In: *Textbook of Veterinary Internal Medicine*. 6th ed. Ettinger SJ, Feldman EC (eds.). St. Louis, Mo. Elsevier Saunders. 2005;63-65.
3. Bowman DD, et al. Arthropods. In: *Georgi's Parasitology for Veterinarians*. St. Louis, Mo. Saunders. 2003;39-44; 49-59.
4. Beugnet F, Chalvet-Monfray K, Loukos H. Flea Tick Risk: a meteorological model developed to monitor and predict the activity and density of three tick species and the cat flea in Europe. *Geospatial Health*. 2009;4(1):97-113.
5. Corpus LD, Corpus KM. Mass flea outbreak at a child care facility: case report. *Am J Public Health*. 1991; 81(4):497-498.
6. Bowman DD, et. al. Helminths. In: *Georgi's Parasitology for Veterinarians*. St. Louis, Mo. Saunders. 2003;148.
7. Hall EJ, German AJ. Diseases of the small intestine. In: *Textbook of Veterinary Internal Medicine*. 6th ed. Ettinger SJ, Feldman EC (eds.). St. Louis, Mo. Elsevier Saunders. 2005;1332-1378.
8. Hugh-Jones M, Hubbert W, Hagstad H. *Zoonoses: recognition, control, and prevention*. Ames, Iowa. Iowa State University Press. 2000;238, 279,294.
9. Azad AF, et al. Flea-borne rickettsioses: ecologic considerations. *Emerg Infect Dis*. 1997;3(3): 319-327.
10. Parola P, Davoust B, Raoult D. Tick- and flea-borne rickettsial emerging zoonoses. *Vet Res*. 2005; 36(3):469-492.
11. Nelson OL, Sellon RK. Pulmonary parenchymal disease. In: *Textbook of Veterinary Internal Medicine*. 6th ed. Ettinger SJ, Feldman EC (eds.). St. Louis, Mo. Elsevier Saunders. 2005;1239-1266.
12. Chomel BB, et al. Bartonella spp. in pets and effect on human health. *Emerg Infect Dis*. 2006;12(3):389-394.
13. Marcus LC. Public health aspects of small animal general practice. In: *Textbook of Veterinary Internal Medicine*. 6th ed. Ettinger SJ, Feldman EC (eds.). St. Louis, Mo. Elsevier Saunders. 2005;702-705.
14. Kamrani A, et al. The prevalence of bartonella, hemoplasma, and rickettsia felis infections in domestic cats and in cat fleas in Ontario. *Can J Vet Res*. 2008;72(5):411-419.
15. Giger U. Regenerative anemias caused by blood loss or hemolysis. In: *Textbook of Veterinary Internal Medicine*. 6th ed. Ettinger SJ, Feldman EC (eds.). St. Louis, Mo. Elsevier Saunders. 2005;1886-1907.
16. Capelli G, et al. Occurrence of rickettsia felis in dog and cat fleas (Ctenocephalides felis) from Italy. *Parasites & Vectors*. 2009;2 Suppl 1:S8.
17. Pérez-Osorio C, et al. Rickettsia felis as emergent global threat for humans. *Emerg Infect Dis*. 2008;14(7):1019-1023.
18. Kahn CM, Line S, Aiello BS. *The Merck Veterinary Manual online*. Merck & Co. www.merckmanual.com/mvm/index.jsp?cfile=htm/bc/211064.htm+word=pyrethrins. Accessed March 19, 2010.
19. Peterson ME, Talcott PA. *Small Animal Toxicology*. St. Louis, Mo. Saunders. 2001;640-652;687-693.
20. Plumb DC. *Plumb's Veterinary Drug Handbook*. 6th ed. Ames, Iowa. Blackwell Publishing. 2008;1346,1341.
21. Urquhart GM, et al. Ectoparasiticides (insecticides/acaricides). In: *Veterinary Parasitology*. Oxford, England. Blackwell Scientific. 1996;272-275.
22. Bren, L. Taking the bite out of fleas and ticks. *FDA Consumer*. 2001;35(4):6-9.
23. Ware, GW, Whitacre DM. *An introduction to insecticides*. 4th ed. Willoughby, Ohio. MeisterPro Information Resources. 2004. http://ipmworld.umn.edu/chapters/ware.htm. Accessed March 19, 2010.

For more information, or to contact the Banfield Applied Research & Knowledge Team, e-mail: BARK@banfield.net