Occupational health and safety in small animal veterinary practice: Part II — Parasitic zoonotic diseases

J. Scott Weese, Andrew S. Peregrine, Julie Armstrong

The risk of exposure to zoonotic agents is inherent in the practice of veterinary medicine. While comprising only a small percentage of those with reported occupational injuries or diseases (1), veterinary practitioners should remain diligent to protect their own health and that of their staff. A number of parasites are recognized causes of zoonotic disease. This paper provides a brief description of some of the relevant parasitic zoonoses encountered by veterinarians in small animal practice. Relevant nonparasitic zoonoses were covered in a recent companion paper (Can Vet J 2002; 43:631–636). Not all possible zoonotic parasitic diseases or those that are mainly a concern for immunocompromised individuals are discussed; rather, this report concentrates on diseases that are most relevant to veterinary practitioners in Canada.

Toxoplasmosis

Toxoplasma gondii is a coccidian parasite that is typically associated with cats and with potentially severe congenital disease in humans. The vast majority of *T. gondii* infections in cats are subclinical. Clinical disease is more common in stressed, concurrently ill, or immunocompromised cats and can be manifested as fever, dyspnea, coughing, lymphadenopathy, myalgia, vomiting, diarrhea, icterus, splenomegaly, neurologic abnormalities, retinochoroiditis, or granulomatous panuveitis (2).

Shedding of *T. gondii* oocysts appears to be rare in cats. Studies have reported that $< 1\%$ of domestic cats shed *T. gondii* at any point in time (4,5). Further, because oocysts require 1 to 5 d to sporulate and become infective, the fastidious grooming behavior of most cats makes the presence of *T. gondii* oocysts on the hair coat quite unlikely (4). As a result, there is a very low chance that working with cats in veterinary practice would result in acquisition of *T. gondii* infection (4). In fact, it has been reported that cat ownership appears to be a higher risk for veterinarians to seroconvert than is working with cats in practice (6,7).

The main concern regarding *T. gondii* is its potential for zoonotic transmission to pregnant women and the development of congenital disease. Congenital toxoplasmosis can occur when naïve (seronegative) women acquire a primary infection during the 1st or 2nd trimester of pregnancy (3), which can result in spontaneous abortion, premature birth, encephalitis, or other neurological abnormalities. To decrease the likelihood of congenital toxoplasmosis, the American College of Obstetricians and Gynecologists recommends preconceptional serologic testing (8). If antibodies (IgG) against *T. gondii* are present, it is considered that there is no risk for congenital infection of a fetus (8). Retesting of seronegative women at 20 to 22 wk of gestation, and then near, or at, term to detect primary infection has also been recommended (9).

Risk to female veterinary personnel is through oral contact with cat feces. Avoiding contact with cats is neither practical nor necessary for pregnant (or potentially pregnant) veterinary personnel, as direct contact with cats is an infrequent source of infection. Decreased exposure to cat feces, however, is important. Therefore, pregnant women should not handle cat feces and litter. In addition, other clinic personnel should clean fecal staining of fur promptly, and litter boxes should be cleaned daily with scalding water. In summary, avoiding contact with cat feces and employing measures of personal hygiene can effectively prevent transmission (10).

Cryptosporidiosis

Cryptosporidiosis is caused by the protozoal parasite *Cryptosporidium parvum*. While the disease is commonly encountered in neonatal ruminants, there has been some debate as to whether *C. parvum* is a pathogen in immunocompetent dogs and cats, or whether asymptomatic infections are the rule. Thus, although cases of clinical cryptosporidiosis in dogs and cats have been reported, experimental inoculation of kittens and puppies with *C. parvum* has only resulted in asymptomatic infections (11). Nevertheless, infected animals shed oocysts that are immediately infective for other animals and, potentially, for humans. However, in light of the very low prevalence of cryptosporidiosis in dogs and cats, these animals appear to constitute a minor potential zoonotic threat.

Profuse, watery diarrhea is the most common presentation in human cases of cryptosporidiosis, with a duration that is dependent on the immune status of the individual. The disease is generally self-limiting in immunocompetent humans, with a clinical course that lasts 3 to 12 d (12). However, *C. parvum* may cause

Department of Clinical Studies (Weese), Department of Pathobiology (Peregrine), and Veterinary Teaching Hospital (Armstrong), Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1.

Address correspondence to Dr. J. Scott Weese, e-mail: jsweese@uoguelph.ca.

prolonged, potentially fatal, illness in patients with acquired immunodeficiency syndrome (AIDS). Outbreaks in humans are most commonly associated with contaminated water sources; however, direct transmission of *C. parvum* from animals to humans can occur.

Cryptosporidium oocysts are highly resistant to disinfectants. The most effective form of killing is exposure to extreme temperatures (less than -20°C or greater than 60°C) (13). Prevention of cryptosporidiosis among veterinary personnel involves personal hygiene; particularly, close attention to hand washing.

Giardiasis

Giardiasis is a common cause of diarrheic disease in dogs and cats, especially amongst young animals. In a recent survey of fecal samples from 1216 dogs in 15 veterinary practices across Canada, 7.2% of samples were detected as being positive for *Giardia* antigen, and 73% of infections occurred in animals less than 1 y of age (14). This study also showed that the majority of *Giardia* infections in dogs were asymptomatic, as 64 out of 82 (78%) infected dogs did not have diarrhea.

Giardiasis is also the most common intestinal parasitic disease of humans in North America (11). Most human infections are acquired from contaminated water, and the majority of such contaminations appear to be human in origin (15). However, *Giardia* isolates are generally not highly host-specific (11). Thus, contact with dogs, cats, or both has been shown to be a significant risk for giardiasis in humans (16).

Zoonotic transmission of *Giardia* from animals to humans involves ingestion of the cyst stage of the parasite in feces. Infection in humans results in highly variable clinical signs that, typically, include diarrhea. Disease can be mild and transient; however, severe and chronic infections can occur (17).

Fecal samples from any dog and cat with acute or chronic diarrhea should be considered as potential sources of *Giardia* infection. Proper disposal of feces and handwashing should decrease the risk of zoonotic transmission. Monitoring of dogs and cats for *Giardia* infection should be part of a regular health check program.

Toxocara canis and *Toxocara cati*

Toxocara canis and, less commonly, *T. cati* are associated with visceral larva migrans (VLM) and ocular larva migrans (OLM) in humans. Both roundworms are relatively common in dogs and cats, respectively, particularly in young animals (18). However, estimates of the prevalence of infection are variable and depend on the manner in which animals are maintained. For example, in The Netherlands, the prevalence of *T. cati* in stray cats (21%) was significantly higher than in domestic cats (4.7%) (19). In the United States, *T. canis* was detected in the feces of 4% of dogs that visited the Oklahoma State University Clinic in 1990 (20). Likewise, 2.9% of dogs from Dutch households were detected as being infected with *T. canis* (19). Finally, 3% (14/423) of canine fecal samples examined by using the standard flotation method at the Animal Health Laboratory, University of Guelph, between 1999 and 2001, were positive for *T. canis*. In contrast, 9% (14/152) of cat fecal samples were positive for *T. cati* over the same period (B. McEwen, personal communication).

While VLM and OLM most commonly occur in children, adults can be affected. Human infection is acquired though ingestion of larvated eggs of *Toxocara* spp. from the soil or fomites. After hatching in the small intestine, the larvae penetrate the intestinal mucosa and then reach the liver via the portal circulation. Thereafter, they may disseminate to the lungs, brain, or eyes (21). Clinical presentation is variable, depending on the infective dose, the immune status of an individual, and the site(s) of dissemination. Asymptomatic infection is common. In an examination of 8457 sera from humans considered representative of the general population in the United States, 2.8% of individuals were identified as being seropositive. However, seroprevalence varied with age, culture, and socioeconomic level (22). Visceral larva migrans typically occurs in children from 1 to 4 y of age with a history of pica. Weight loss, pyrexia, malaise, and cough are frequent complaints. Most cases are self-limiting; however, serious disease can occur (12). Pulmonary disease can include bronchiolitis, asthma, and nonspecific pneumonitis. Central nervous system (CNS) infection may be manifested as seizures, psychiatric disorders, or, less commonly, encephalitis. Myocardial or CNS involvement is often fatal (21). Ocular larva migrans is usually unilateral and can result in vision loss or strabismus. Ocular disease is generally seen in the absence of other signs of VLM, and a history of pica is less common than with VLM. The mean age when OLM is diagnosed is 7 to 8 y (22). Within Canada, the zoonotic threat of *Toxocara* spp. is unclear. However, it is worthy of note that VLM is currently a very uncommon diagnosis in humans living in Canada (J. Keystone, personal communication).

Hand washing after handling animals and prompt proper disposal of feces should greatly reduce the chance for exposure. Routine deworming and monitoring for *Toxocara* spp. should be part of a preventive program for both dogs and cats. The earliest that eggs of *T. canis* are shed in the feces of puppies is approximately 3 wk of age, while for *T. cati* in kittens, it is approximately 6 wk of age. Thus, in litters of puppies in which *T. canis* is considered a problem, deworming should begin at 2 wk of age and be repeated every 2 wk until approximately 10 wk of age. Puppies that are first seen at 5 to 6 wk of age should routinely be treated at this time and again 2 and 4 wk later. Thereafter, up to 6 mo of age, they should be treated on a monthly basis. Kittens should be dewormed for the first time at 6 wk of age, again at 8 and 10 wk, and then monthly until 6 mo of age. In general, dogs and cats above 6 mo of age should be monitored once or twice a year for intestinal parasites and treated accordingly. Animals in households with children or immunocompromised individuals should be monitored and treated more frequently.

Baylisascaris procyonis

Raccoons across North America are commonly infected with the ascarid nematode *Baylisascaris procyonis*. The eggs of this roundworm are similar in morphology to those of *Toxocara* spp. and, upon ingestion, larvae that

hatch from infective eggs undergo migration in a wide range of intermediate hosts. Thus, *B. procyonis* is the most frequently identified cause of larva migrans in animals, and it has been associated with severe neurological disease in more than 90 species of mammals and birds (23). The parasite is also an important cause of VLM, OLM, and neural larva migrans in humans (24); it has resulted in death or severe CNS disease in at least 10 children (23). As with *Toxocara* spp., children who are 1 to 4 y of age appear to be at greatest risk of becoming infected with *B. procyonis.*

Within the United States, the prevalence of *B. procyonis* in raccoons is highest on the west coast and in the midwestern and northeastern parts of the country where up to 82% of animals have been detected as being infected (23). In Canada, prevalence estimates range from 51% to 61% for raccoon populations in British Columbia, Ontario, and Québec (25–27). However, a lower estimate of 7% was obtained in Nova Scotia (28).

In light of the high prevalence of *B. procyonis* in raccoons, and both the high fecundity and zoonotic potential of the parasite, the keeping or feeding of raccoons should be strongly discouraged. All raccoons examined by veterinarians should be considered infected, and attention should be paid to personal hygiene. Finally, all raccoons in rehabilitation centers, particularly juveniles, should be dewormed regularly for this parasite.

Hookworms

Cutaneous larva migrans (CLM) or "creeping eruptions" are serpiginous, erythematous, pruritic tracks that occur in the skin of humans in association with the transient migration of hookworm larvae. Infections are due to direct contact of skin with larvae in the environment, and they are usually associated with 1 or 2 hookworms of dogs: *Ancylostoma braziliense* or *A. caninum*. Since *A. braziliense* is generally involved in more severe, protracted cases, CLM is typically diagnosed in humans residing where *A. braziliense* is endemic in the dog population (southeastern parts of the USA and in the Caribbean). Within Canada, CLM is diagnosed very rarely in those humans who have not travelled outside the country (J. Keystone, personal communication). Therefore, the risk to veterinary personnel is minimal. Prompt disposal of dog feces and regular deworming programs will control transmission of hookworms to humans.

Sarcoptic mange (canine scabies)

Sarcoptic mange is a parasitic disease of dogs caused by *Sarcoptes scabiei* var. *canis.* There is no age, breed, or sex predilection, and affected animals typically display an acute onset of intense pruritis. Lesions are more common over the pinnae, face, limbs, and ventrolateral part of the trunk (29). Human scabies is usually associated with *S. scabiei* var. hominis; however, zoonotic disease caused by *S. scabiei* var. *canis* is frequently reported and may occur in 30% to 50% of canine cases (29). It has been suggested that human infection with *S. scabiei* var. *canis* causes self-limiting disease (30); however, persistent infection has been reported more recently (29).

Human infection with *Sarcoptes* var. *canis* can be via direct contact with infected animals or via indirect contact with fomites (bedding, grooming utensils) (29). A single washing of fomites in hot water and detergent is effective at eliminating *S. scabiei.* Canine cases of sarcoptic mange can readily be treated with ivermectin or selamectin.

Conclusion

Animals that appear healthy must not be dismissed as possible sources of zoonotic pathogens, as some animals may be asymptomatic carriers. For example, Hill et al (4) reported the identification of *Cryptosporidium parvum, Giardia, Toxocara cati, Salmonella* Typhimurium, or *Campylobacter jejuni* from 9.4% of healthy client-owned cats and 22.6% of healthy shelter cats. Spain et al (5) reported the detection of enteric zoonotic pathogens from 40.7% of cats either from humane societies or presented to primary care veterinary clinics. Accordingly, attention should be paid to personal hygiene after handling any animal.

Potential exposure to zoonotic disease is an inherent risk in veterinary medicine. While it is virtually impossible to completely prevent exposure to zoonotic agents, measures can be taken to protect veterinarians and staff from acquiring infections. If attention is paid to awareness of disease with zoonotic potential, identification of infected animals, proper handling and housing, and personal hygiene, the risks to veterinary personnel can be greatly reduced.

References

1. Jeyaretnam J, Jones H, Phillips M. Disease and injury among veterinarians. Aust Vet J 2000;78:625–629.
2. August JR, Chase TM. Toxoplasmosis. Vet Clin North Am Small Anim Pract 1987;17:55–71.
3. Tenter AM, Heckeroth AR, Weiss LM. Toxoplasma gondii: from animals to humans. Int J Parasitol 2000;30:1217–1258.
4. Hill SL, Cheney JM, Taton-Allen GF, Reif JS, Bruns C, Lappin MR. Prevalence of enteric zoonotic organisms in cats. J Am Vet Med Assoc 2000;216:687–692.
5. Spain CV, Scarlett JM, Wade SE, McDonough P. Prevalence of enteric zoonotic agents in cats less than 1 year old in Central New York State. J Vet Intern Med 2001;15:33–38.
6. Fox JG, Campbell LH. Serological survey of toxoplasmosis in a selected population of veterinarians in California. Calif Vet 1974;28:32–35.
7. Tizard IR, Caoili FA. Toxoplasmosis in veterinarians: an investigation into possible sources of infection. Can Vet J 1976; 17:24–25.
8. Cunningham FG, MacDonald PC, Gant NF, et al. Williams Obstetrics, 20th ed. Stamford, Connecticut: Appleton and Lange, 1997.
9. Bruckner DA. Serologic and intradermal tests for parasitic infections. Pediatr Clin North Am 1974;32:1063–1075.
10. Moore RMJ, Davis YM, Kaczmarek RG. An overview of occupational hazards among veterinarians, with particular reference to pregnant women. Am Indian Hyg J 1993;54:113–120.
11. Bennett M, Baxby D, Blundell N, Gaskell CJ, Hart CA, Kelly DF. Cryptosporidiosis in the domestic cat. Vet Rec 1985;116:73–74.
12. Tan JS. Human zoonotic infections transmitted by dogs and cats. Arch Intern Med 1997;157:1933–1943.
13. Snowden KF. Cryptosporidiosis. In: Farris R, Mahlow J, Newman E, Nix B, eds. Health Hazards in Veterinary Practice. 3rd ed. Austin, Texas: Texas Department of Health, 1995:27–28.
14. Jacobs SR, Forrester CP, Yang J. A survey of the prevalence of *Giardia* in dogs presented to Canadian veterinary practices. Can Vet J 2001;42:45–46.

15. Wallis PM, Erlandsen SL, Isaac-Renton JL, Olson ME, Robertson WJ, van Keulen H. Prevalence of *Giardia* cysts and *Cryptosporidium* oocysts and characterization of *Giardia* spp. isolated from drinking water in Canada. Appl Environ Microbiol 1996;62:2789–2797.
16. Warburton AR, Jones PH, Bruce J. Zoonotic transmission of giardiasis: a case control study. Commun Dis Rep CDR Rev 1994;4:R32–R36.
17. Snowden KF. Giardiasis. In: Farris R, Mahlow J, Newman E, Nix B, eds. Austin, Texas: Texas Department of Health, 1995:35.
18. Anonymous. *Toxocara canis*. J Small Anim Pract 1997;38: 531–534.
19. Overgaauw PA. Prevalence of intestinal nematodes of dogs and cats in the Netherlands. Vet Q 1997;19:14–17.
20. Roth RM, Gleckman RA. Human infections derived from dogs. Postgrad Med 1985;77:169–180.
21. Glickman LT, Schantz PM. Epidemiology and pathogenesis of zoonotic toxocariasis. Epidemiol Rev 1981;3:230–250.
22. Kazacos KR. *Baylisascaris procyonis* and related species. In: Samuel WM, Pybus MJ, Kocan AA, eds. Parasitic Diseases of Wild Animals. Ames, Iowa: Iowa State Univ Pr, 2001:301–341.
23. Kazacos KR. Visceral, ocular and neural larva migrans. In: Connor DH, Chandler FW, Schwartz DA, eds. Pathology of Infectious Diseases. Stamford, Connecticut: Appleton and Lange, 1997:1459–1147.
24. Ching HL, Leighton BJ, Stephen C. Intestinal parasites of raccoons (*Procyon lotor*) from southwest British Columbia. Can J Vet Res 2000;64:107–111.
25. Berry JF. Phylogenetic relationship between *Baylisascaris* spp. Sprent, 1968 (Nematoda: Ascaridae) from skunks, raccoons and groundhogs in southern Ontario. [M.Sc. thesis]. Guelph, Ontario: University of Guelph; 1985.
26. MacKay A, Robitaille J, Messier S, Villeneuve A. *Baylisascaris* chez le raton laveur au Quebec: possibilite de zoonose. Vet Med du Quebec 1995;25:102–105.
27. Smith SL. The presence of the raccoon roundworm *Baylisascaris procyonis* in *Procyon lotor* in Nova Scotia. [Senior Honours Thesis]. Wolfville, Nova Scotia: Acadia Univ; 1992.
28. Scott DW, Horn RTJ. Zoonotic dermatoses of dogs and cats. Vet Clin North Am Small Anim Pract 117–144.
29. Arlian LG, Runyan RA, Estes SA. Cross infectivity of *Sarcoptes scabiei*. J Am Acad Dermatol 1984;10:979–986.