What's Old Is New

Echinococcus and *Trypanosoma* in Companion Animals

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KEYWORDS

• *Trypanosoma cruzi* • *Echinococcus granulosus* • *Echinococcus multilocularis*
• Trypanosomiasis • Echinococcosis • Zoonosis

KEY POINTS

- Both *Trypanosoma cruzi* and *Echinococcus* spp. Have important zoonotic and public-health implications, with dogs acting as key reservoirs and sentinels in North America.
- *T. cruzi* has multiple transmission routes (vector-borne, oral, congenital, and transfusion). Diagnosis is multimodal and stage-dependent, with no single gold-standard test.
- Clinically, canine Chagas disease ranges from asymptomatic to acute myocarditis and chronic chagasic cardiomyopathy; treatment is largely supportive, with limited antiparasitic efficacy; prevention focuses on vector control.
- *Echinococcus* infections involve dogs as definitive hosts with hydatid cysts mainly in liver/lungs of intermediate hosts; diagnosis relies on imaging and PCR/serology to distinguish CE from AE; treatment combines antiparasitic therapy.
- Prevention and One Health emphasis: reduce environmental exposure, controls on predation and coprophagy, regular deworming, hygiene, and surveillance to curb zoonotic transmission.

TRYPANOSOMA CRUZI

Introduction

Trypanosoma cruzi, the protozoan etiologic agent of Chagas disease, was first described in 1909 by Carlos Chagas and remains one of the most important zoonotic parasitic pathogens in the Americas.[1–3] Historically considered a disease of rural Latin America, Chagas disease is now increasingly recognized as endemic in the southern United States, where established vector populations, infected wildlife, infected domestic animals, and

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Abbreviations	
AE	alveolar echinococcosis
CE	cystic echinococcosis
ECG	electrocardiogram
PCR	polymerase chain reaction
WHO	World Health Organization

autochthonous human cases have all been documented.[2,4,5] Dogs are among the most clinically relevant veterinary hosts because they can develop severe disease, act as domestic reservoirs, and serve as sentinels of environmental transmission risk.[6–8]

Etiology

T cruzi is a hemoflagellate protozoan of the family Trypanosomatidae with 3 major life stages: bloodstream trypomastigotes, intracellular amastigotes, and vector-associated epimastigotes.[1] The parasite exhibits considerable genetic diversity and is divided into discrete typing units (DTU) TcI–TcVI and TcBat, with TcI and TcIV predominating in many canine infections in the United States.[9,10] Parasite strain diversity can potentially contribute to differences in virulence, tissue tropism, immune response, and treatment response.[3]

Transmission classically occurs through contamination of bite wounds or mucus membranes with infected feces from triatomine vectors ("kissing bugs"), but oral transmission through ingestion of infected bugs or infected prey has been proposed as the dominant route in dogs.[2] Additional routes include congenital transmission, blood transfusion, and organ transplant.[11] Experimental studies have demonstrated that disease severity may vary depending on inoculum source, parasite dose, and route of inoculation.[12]

Epidemiology

The parasite is endemic across the Americas from South America through the southern United States.[1,5] In the United States, triatomines have been identified in at least 28 to 32 states, with the highest density of vectors and canine cases in southern regions.[2,5] Estimated pooled canine prevalence in the United States have been reported at approximately 12%, although heterogeneity among studies is high.[13]

Canine seroprevalence varies by population. Multidog kennel environments and working dog populations appear at especially high risk, with seroprevalence values reaching 57.6% in Texas hound kennels,[9] 7.4% to 18.9% in government working dogs along the Texas–Mexico border,[10] around 21% in hunting dogs.[14] Shelter dogs in Louisiana had polymerase chain reaction (PCR) positivity of 15.7%,[15] and canine exposure has also been documented in Virginia and West Virginia hunting dogs.[14,16] Additional prevalence studies from Mexico and Brazil emphasize that dogs remain important domestic hosts throughout endemic regions.[8,17]

Risk factors include outdoor housing, kennel density, rural environment, increasing age, hunting activity, vector exposure, and infected housemates or littermates; one study found that dogs with infected housemates/littermates had 13 fold increased odds of infection.[18] Dogs in impoverished border communities ("colonias") also demonstrate high seroprevalence, paralleling human exposure risk.[7]

Pathogenesis and Clinical Presentation

Following infection, metacyclic trypomastigotes invade host cells and transform into amastigotes, which replicate intracellularly before lysing cells and disseminating as bloodstream trypomastigotes.[1] Although many tissues may be affected, the

myocardium is the principal target organ in dogs. Acute myocardial injury results from direct parasite-mediated cytolysis and inflammatory myocarditis, while chronic disease involves persistent inflammation, fibrosis, conduction system damage, microvascular abnormalities, autonomic dysfunction, and myocardial remodeling.[1,18,19] Similar to humans, pathogenesis likely reflects an interplay among parasite persistence, host immune response, and strain-dependent virulence.[3]

Clinical manifestations in dogs range from asymptomatic infection to fulminant myocarditis, chronic cardiomyopathy, and sudden death.[1,2] Acute disease may develop within 2 to 3 weeks of infection. Clinical signs include lethargy, fever, ascites, generalized lymphadenopathy, hepatosplenomegaly, pale mucus membranes, weakness, and sudden death secondary to myocarditis or arrhythmia.[1,2,18]

Many dogs may enter an indeterminate phase with no clinical signs. Chronic disease may manifest months to years later with exercise intolerance, syncope, weakness, cough, ascites, congestive heart failure, respiratory distress, and sudden death.[20] The hallmark of chronic canine Chagas disease is chagasic cardiomyopathy, characterized by ventricular arrhythmias, atrioventricular block, and myocardial dysfunction. Holter monitoring studies show that infected dogs have significantly more electrocardiogram (ECG) abnormalities than controls, with greater than 75% of positive dogs showing at least 1 abnormality.[1,18,19]

Diagnosis

Diagnosis of *T. cruzi* infection in dogs is challenging because no single test is reliable across all stages of infection. Therefore, a multimodal approach, integrating serologic, molecular, and clinical findings, is recommended. This reflects the biphasic nature of infection, with an acute phase characterized by higher parasitemia and a chronic phase in which parasite burden is low and intermittent.[1–3]

Clinical context is critical. Dogs from endemic areas presenting with arrhythmias, myocarditis, elevated cardiac troponin I, or unexplained cardiomyopathy should be considered suspect regardless of initial test results.[10,18,19]

During the acute phase, PCR is the most useful diagnostic tool, as circulating trypomastigotes are rarely detected on blood smears in clinical settings.[1,21] However, acute infections are often missed due to nonspecific clinical signs and limited testing at this stage. In the chronic phase, diagnosis relies primarily on serology (immunofluorescence assay, enzyme-linked immunosorbent assay, or rapid tests). A major limitation is the absence of a gold-standard assay, and discordant results among tests are common.[10,14] Variability in test performance supports the use of multiple independent assays to improve diagnostic confidence.[14] Despite advances, important gaps remain, including the inability to distinguish active from past infection and the lack of tests to confirm parasitologic cure.[3,14]

Treatment

Treatment of canine Chagas disease is challenging. Ideally, clinical management would include treatment of both the cardiac manifestations and the parasitic infection. Since there is no Food and Drug Administration-approved antiparasitic treatment of canine Chagas disease, treatment is largely focused on the cardiac disease.

Antiparasitic therapy has been explored in dogs, essentially extrapolated from human medicine. Benznidazole and nifurtimox are used in human medicine and appear most effective during acute infection but have limited efficacy in chronic disease and may cause adverse effects.[3] Experimental canine studies suggest benznidazole may reduce parasitemia but may not be able to eliminate chronic infection and prevent myocardial lesions.[22]

Therapeutic vaccination, an emerging area of interest, has demonstrated potential use for reducing parasite burden and slowing the progression of ECG abnormalities in naturally infected dogs by modulating the host response.[23,24]

Prevention

No commercial vaccine is currently available for *T cruzi* infection in dogs. Therefore, prevention of infection focuses on vector avoidance and environmental management. Dogs in endemic regions should ideally be housed indoors overnight, and kennels should be modified to reduce vector invasion by removing woodpiles, brush, debris, and wildlife nesting sites.[2] Structural improvements such as screening and sealing cracks may reduce triatomine intrusion. Insecticide use in the environment and treatment of dogs with isoxazolines may help in decreasing the population of triatomines and, therefore the transmission of the parasite.[5,25]

Dogs should be prevented from ingesting insects and wildlife prey when possible because oral transmission is likely common.[2,5] Blood donor screening should be considered in endemic regions because transfusion-associated transmission remains theoretically possible.

One Health Considerations

Dogs are critical of the *T. cruzi* ecology because they serve both as reservoir hosts and as sentinels for human exposure risk.[9,21] Dogs can infect feeding triatomines, thereby amplifying domestic and peridomestic transmission cycles. However, direct contact with infected dogs is not considered a major zoonotic risk; rather, infected dogs indicate shared environmental exposure to infected vectors.[2]

The relationship between canine and human infection is particularly evident in underserved communities, where canine seroprevalence parallels human risk.[7,9] Because of this, canine Chagas disease strongly supports a One Health approach involving veterinarians, physicians, entomologists, epidemiologists, and public health professionals.

Discussion

Despite increasing recognition, canine Chagas disease remains underdiagnosed due to limited clinician awareness, lack of a gold-standard diagnostic test, and overlap with other causes of myocarditis and cardiomyopathy. Serologic discordance among assays is common, particularly in epidemiologic studies and screening programs.[14] PCR can identify circulating parasite DNA but has reduced sensitivity in chronic infection due to intermittent parasitemia.[21,26]

Current evidence suggests clinicians should include *T. cruzi* infection in the differential list for dogs with unexplained arrhythmias, elevated troponin, myocarditis, particularly in endemic areas.

SUMMARY

T. cruzi is an increasingly recognized cause of myocarditis, arrhythmias, and cardiomyopathy in dogs throughout the Americas. Dogs may remain asymptomatic, develop acute myocarditis, or progress to chronic chagasic cardiomyopathy. Diagnosis remains challenging because of imperfect diagnostics and discordant testing platforms. Treatment is essentially supportive, while prevention focuses on vector control and reduction of exposure. Since dogs serve as both sentinels and reservoirs, they play a central role in the One Health epidemiology of Chagas disease.

CLINICS CARE POINTS

- *T. cruzi* infection should be considered in dogs with ventricular arrhythmias, myocarditis, syncope, or elevated cardiac troponin in endemic areas.
- Many infected dogs may remain asymptomatic.
- No single diagnostic assay is definitive; multiple tests improve diagnostic confidence.
- Discordant serology is common and should not automatically exclude infection.
- Prevention depends primarily on vector avoidance and environmental modification.

ECHINOCOCCUS GRANULOSUS AND *ECHINOCOCCUS MULTILOCULARIS*

Introduction

Echinococcus granulosus and *Echinococcus multilocularis* are emerging zoonotic tapeworms and the causative agents of cystic echinococcosis (CE) and alveolar echinococcosis (AE), respectively. Collectively, echinococcosis affects millions of people worldwide and are considered 1 of the 20 neglected tropical diseases by the World Health Organization (WHO) with low mortality and high morbidity rates.[27] AE is considered the third most significant foodborne parasitic disease in the world by the Food and Agriculture Organization of the United Nations and the WHO.[28] Dogs are clinically relevant veterinary hosts because they increase the risk of zoonotic transmission to humans.[29]

Etiology and Life Cycle

Echinococcus spp. are microscopic tapeworms of the family Taeniidae with adult and metacestode stages.[30] *E. granulosus* and *E. multilocularis* are the 2 species of veterinary importance in North America. *E. granulosus* is a genotypic cluster with 2 strains present in North America, a pastoral strain, *E. granulosus* sensu stricto (G1-3), with a domestic dog-domestic sheep life cycle and a sylvatic strain, *E. canadensis* (G6/7, G8-10), with a wild canid-ungulate life cycle.[31] For both species, adults occur in the small intestine of wolves, coyotes, and domestic dogs after predation and ingestion of infected organs from moose and elk and domestic dogs from sheep offal. The metacestode, unilocular hydatid cysts, develops in the liver and lungs of moose, elk, and domestic sheep after fecal–oral transmission of infective eggs through grazing. The sylvatic life cycle is maintained through a predator–prey relationship and the pastoral life cycle in domestic animal production systems where domestic dogs have uncontrolled access to sheep offal[32] (**Figs. 1** and **2**).

E. multilocularis has different haplotypes named after the geographic location in which they were first found. Historically, North America has 2 indigenous haplotypes, N1 in Alaska and N2 in Indiana and South Dakota.[33] Recently, European and Asian haplotypes have been the predominant in North America.[34] *E. multilocularis* occurs mainly in a sylvatic wild canid-rodent life cycle. Adult tapeworms are present in the small intestines of coyote, red fox, and domestic dog (definitive hosts) from predation on infected Arvicoline rodents. The metacestode, alveolar (multilocular) hydatid cysts, develop in the liver of rodents after fecal–oral transmission of infective eggs from the environment. The sylvatic life cycle is maintained through a predator–prey relationship in coyotes and red foxes and spills over into domestic dogs.[32]

Domestic dogs and cats become aberrant intermediate hosts for both *Echinococcus* spp. after accidental ingestion of eggs from the environment, mainly from

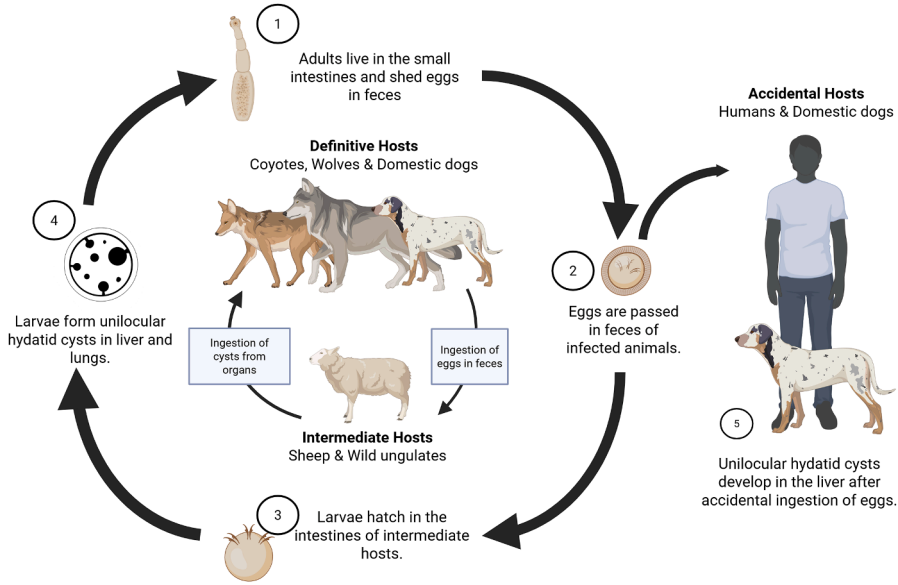


Fig. 1. Life cycle of *E granulosus*. (*Created in* BioRender. Miller, K. (2026) https://BioRender.com/8cmipw1.)

coprophagy. Humans can also be aberrant intermediate hosts after accidental ingestion of infective eggs from the environment through contaminated food, water, and feces from definitive hosts.[35] CE and AE develop slowly, mainly in the liver and lungs, over 1 to 5 years in domestic animals and 5 to 15 years in humans.[36]

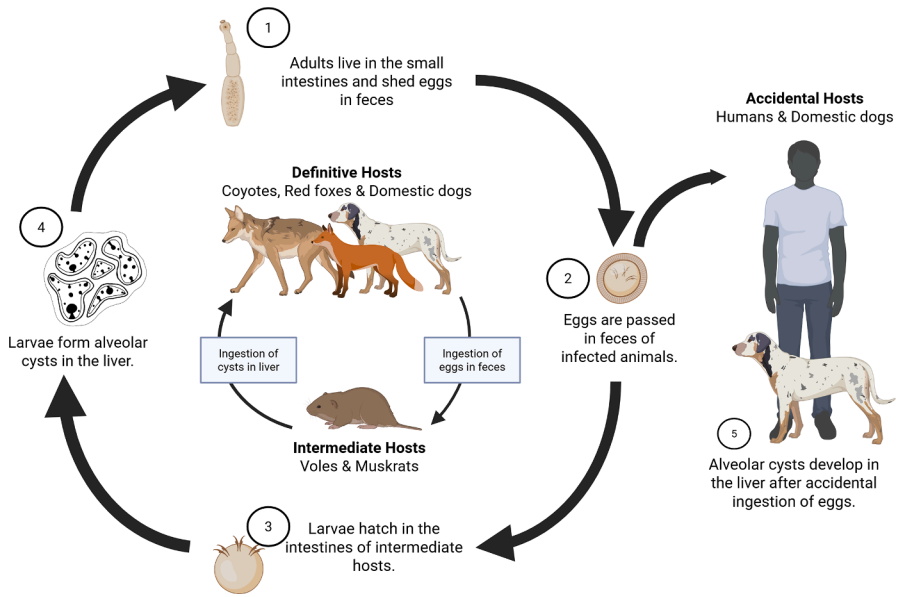


Fig. 2. Life cycle of *E multilocularis*. (*Created in* BioRender. Miller, K. (2026) https://BioRender.com/ziz3lgb.)

Epidemiology

E. granulosus and *E. multilocularis* have been reported in wild canids, domestic dogs and cats, and humans throughout North America. The sylvatic strain of *E. granulosus* has been found in Alaska and Canada with a recent introduction into Tennessee through elk translocated from Canada.[37] The sylvatic strain of *E. granulosus* has a sporadic distribution in the contiguous United States in Arizona, California, New Mexico, Pennsylvania, and Utah.[37]

Historically, the North American haplotype of *E. multilocularis* has been restricted to the Northern Tundra Zone of Alaska and Canada and the North Central Region of North America encompassing the southern half of Canadian provinces and the north central United States.[33,38] The geographic range is expanding in North America with the European haplotype being predominate.[34] In every province in Canada, *E. multilocularis* has been reported in wild canids, domestic dogs, and humans, with Alberta being the province with the highest human and domestic dog AE cases and intestinal echinococcosis in dogs and wild canids.[34] In the United States, there have been new case reports of intestinal echinococcosis in domestic dogs in Colorado, Idaho, Illinois, Kansas, Missouri, Montana, Nevada, and Oregon[29,39] and AE in Missouri, Virginia, and Washington.[40–42] The prevalence of *E. multilocularis* in coyotes was 7% in New York, 37% in Washington, and 2% of red foxes in Pennsylvania.[27,43,44]

Pathogenesis and Clinical Presentation

Intestinal echinococcosis occurs after ingestion of protoscolices within hydatid cysts from infected organs of intermediate hosts. The protoscolices mature in the small intestine and release eggs in the feces of infected definitive hosts. Usually, patent tapeworm infections do not cause clinical disease. However, recent case reports of intestinal echinococcosis in domestic dogs shows a variety of clinical signs ranging from vomiting, diarrhea (with and without frank blood), and abdominal discomfort, to no clinical signs.[29,39]

CE and AE occur after ingestion of eggs from the environment through coprophagy by domestic dogs and cats. Once eggs are ingested, the oncosphere larvae develops into hydatid cysts mainly in the liver and lungs of infected definitive hosts. Unilocular hydatid cysts mimic space occupying lesions when growing causing pressure necrosis on surrounding organs. Clinically, this leads to abdominal distention and pain. Alveolar hydatid cysts mimic infiltrative cancer and grow via budding creating a multivesicular cyst surrounded by connective tissue and a fibrous cyst wall. Hydatid cysts can contain protoscolices and calcareous corpuscles, referred to as hydatid sand, or not. Clinically, infected animals can present with abdominal distention and pain, vomiting, anorexia, lethargy, and muscle wasting.[40,42,45] Complete blood count and chemistry can be normal or could show various abnormalities.[40,42,45]

Diagnosis

Diagnosis of *Echinococcus* spp. is challenging because all adult taeniid tapeworms produce taenia-type eggs. Taenia-type eggs are brown in color with a thick, striated shell (**Fig. 3**). The eggs contain a hexacanth embryo with 3 pairs of hooks and measure 25 to 40 μm in diameter.[30] Teasing eggs out of macroscopic proglottids is a quick way to identify tapeworms in the clinic. Taenia-type eggs found through routine fecal screenings using flotation techniques can be either *Taenia* spp. or *Echinococcus* spp. Therefore, PCR testing through a reference laboratory is recommended.[46] Intestinal echinococcosis should be suspected if there is a history of rodent predation,



Fig. 3. Taenia-type egg. (*Image Courtesy* This image is the courtesy of Parasitology lab, Kansas State University.)

especially if living in an endemic area, or an area with recent case reports and wild canid surveillance.

CE and AE are diagnosed by identifying hydatid cyst in the affected organ(s) of infected animals. Hydatid cysts are found in the internal organs, mainly the liver and lungs, on postmortem examination of naturally infected intermediate hosts. Unilocular hydatid cysts, caused by the pastoral strain of *E. granulosus*, are less pathogenic in domestic dogs and cats, and humans than the sylvatic strain.[37] Hydatid cysts can be identified in the affected organs of domestic dogs and cats using radiographs where there can be loss of serosal detail and mineralization seen around the affected organ(s). Computed tomography can also be used to visualize the extent of cyst development, especially if infection of multiple organs is suspected. Ultrasound can be used to safely remove cyst fluid via fine-needle aspirate and PCR tested if AE or CE is suspected.

Therapy

Treatment of intestinal echinococcosis can be achieved using praziquantel (5 mg/kg, orally or subcutaneously) in domestic dogs and cats. Treatment of CE and AE in domestic dogs and cats can be complicated and is dependent upon the number of cysts and extent of organ involvement. Praziquantel cannot penetrate the hydatid cyst wall and should not be used for CE and AE treatment. The best therapeutic option for hydatid cysts is life-long treatment with albendazole (10 mg/kg, orally, daily).[42,47] It is important to routinely monitor patients on albendazole for bone marrow suppression. However, using albendazole alone is minimally effective at increasing the life span of infected animals. Surgical removal of infected cyst tissue, in combination with albendazole, has been shown to increase the life span of infected animals.[42] If surgery is not an option, a method adopted from CE treatment in humans, called percutaneous aspiration, injection, reaspiration has been used.[42,47] Performed under general anesthesia, cyst fluid is removed using a closed system. Then 1 mL of 95% ethanol/kg is instilled into the cyst and allowed to sit for 10 to 15 minutes and drained again until all cyst fluid is removed. This procedure causes shrinkage of hydatid cysts and can be performed on a bimonthly basis until surgical removal of cysts can be achieved.[42] Because patients diagnosed with CE and AE can also be coinfected with *Echinococcus* spp. adults in the small intestine, praziquantel should also be given.

Prevention

Intestinal echinococcosis can be avoided by preventing predation and scavenging of potentially infected intermediate hosts by domestic dogs and cats. Domestic cats should be kept indoors to prevent predation. Domestic dogs should be prevented from unsupervised outdoor roaming; pets should remain leashed, confined to an enclosed yard, or closely monitored while outside. Monthly preventive deworming should be considered if hunting and roaming behavior cannot be controlled. AE and CE infections can be prevented by discouraging coprophagy.

Public Health Considerations

CE and AE have both been reported in humans in North America. In Canada, case reports of human AE have been found in Alberta and Ontario. In the United States, only 3 autochthonous human cases have been reported in Minnesota in 1977[48] and 2 cases in Vermont.[49,50] In humans, CE and AE can be difficult to diagnose because cysts are often misdiagnosed and fatal if left untreated.[28] Treatment can be prolonged and often results in a decreased quality of life. The zoonotic risk for CE and AE increases when pets are infected with adult *Echinococcus* spp. and contaminate the environment with infective eggs. Individuals with occupations and activities (ie, licensed hunting and trapping) that increase exposure to potentially infected wild canid scat are also at risk. Therefore, it is important to use proper hygiene when in contact with canid feces and carcasses.

SUMMARY

Echinococcus spp. are zoonotic tapeworms that infect domestic dog and cats, and humans. Domestic dogs can be infected with the adult and metacestode stages of *Echinococcus* spp. Although the prevalence is unknown, the geographic range is expanding in North America. The zoonotic risk to humans increases when domestic dogs are infected with adults tapeworms and shed infectious eggs into the environment. Protection of domestic dogs and cats can be achieved by preventing predation on potentially infected rodents and coprophagy.

CLINICS CARE POINTS

- Domestic dogs can be a definitive and intermediate host for *Echinococcus* spp.
- The zoonotic risk to humans increases when domestic dogs are infected with adult *Echinococcus* spp. and shed infectious eggs in the environment.
- Praziquantel is the treatment of adult *Echinococcus* spp. and not the metacestode stages.
- Taenia-type eggs + a macroscopic proglottid = *Taenia* spp., *not Echinococcus* spp.

DISCLOSURE

K.R. Miller reports a relationship with Elanco Animal Health Inc and Boehringer Ingelheim that includes consulting or advising, funding grants, speaking and lecture fees, and travel reimbursement. R.R. Barrios has no conflicts of interest to disclose.

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