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Research Note

Serological Prevalence of *Neospora caninum* and *Toxoplasma gondii* in Dogs from Kansas

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ABSTRACT: Sera from 229 dogs were examined for antibodies to *Neospora caninum* using an indirect immunofluorescence assay and for antibodies to *Toxoplasma gondii* using a direct agglutination test. Five of the dogs (2%) were positive for antibodies to *N. caninum* and 57 (25%) were positive for antibodies to *T. gondii*. Three (1%) of the dogs had antibodies to both protozoans. Results indicate that *N. caninum* is less prevalent in the canine population than *T. gondii*.

KEY WORDS: *Neospora caninum, Toxoplasma gondii,* dog, prevalence.

Neospora caninum Dubey, Carpenter, Speer, Topper, and Uggla, 1988, is a recently described protozoan parasite of dogs (Dubey et al., 1988a, b). It is similar to *Toxoplasma gondii* Nicolle and Manceaux, 1909, with light microscopy but can be differentiated by using transmission electron microscopy (Dubey et al., 1988a; Speer and Dubey, 1989) or serological and immunohistochemical testing (Bjerkås and Presthus, 1988; Lindsay and Dubey, 1989b, c). Clinical neosporosis in dogs manifests itself as polymyositis, encephalitis, polyradiculoneuritis, and ascending paralysis (Cummings et al., 1988; Dubey et al., 1988a, b). The disease can be fatal in young or old dogs but is more serious in transplacentally infected puppies. Clinical toxoplasmosis in dogs is usually seen in young animals and is associated with concurrent distemper virus infection (reviewed by Dubey, 1985).

Nothing is known about the prevalence of *N. caninum* infection in the canine population, whereas *T. gondii* infection is common (Dubey, 1985). In the present study we examined sera from 229 dogs for antibodies to *N. caninum* and *T. gondii*.

All dogs were patients at the Veterinary Med-

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ical Teaching Hospital, Kansas State University, from 1988 to 1989. Samples were shipped frozen to the Zoonotic Diseases Laboratory, Beltsville, Maryland, and were stored at −20°C until examined. Samples were given a coded number and results of *N. caninum* and *T. gondii* testing were kept separate until all samples had been tested. Case histories were obtained for dogs serologically positive for *N. caninum*.

The indirect immunofluorescent assay (IFA) for detection of IgG antibodies to *N. caninum* was done as described (Dubey et al., 1988b) using tachyzoites of *N. caninum* that were grown in cell cultures (Lindsay and Dubey, 1989a) as antigen. Serum was screened at 1:50 dilution in phosphate-buffered saline (PBS), and positive samples were then titrated to an end-point using doubling dilutions of serum in PBS. Serum from 2 dogs with known *N. caninum* infections and serum from 3 dogs negative for *N. caninum* antibodies were used as positive and negative controls at dilutions of 1:50 in PBS. The direct agglutination test for *T. gondii* was performed as described by Dubey and Desmonts (1987). Serum from each dog was tested at dilutions of 1:25, 1:100, and 1:400. We arbitrarily screened canine sera at 1:50 in the IFA and at 1:25 in the agglutination test but have no evidence that titers less than these are not specific.

Antibodies to *N. caninum* tachyzoites were found in 5 of 229 (2%) serum samples. Antibody titers were 1:100 in 1 dog, 1:200 in 2 dogs, and 1:400 in 2 dogs. None of the dogs had clinical signs that could conclusively be associated with active *N. caninum* infection.

Antibodies to *T. gondii* were found in 57 (25%) of the 229 dogs. Forty (17%) had titers of 1:25, 11 (5%) had titers of 1:100, and 6 (3%) had titers of 1:400.

Three of the 5 dogs positive for *N. caninum* were also positive for *T. gondii*. One dog had titers of 1:200 for *N. caninum* and 1:25 for *T. gondii*, 1 had titers of 1:100 for *N. caninum* and 1:25 for *T. gondii*, and 1 had titers of 1:200 for *N. caninum* and 1:100 for *T. gondii*.

Bitches may have antibodies to *N. caninum*, but show no clinical signs of diseases (Dubey et al., 1988b; Hay et al., 1990). These bitches may give birth to transplacentally infected puppies not all of which show signs of disease (Dubey and Lindsay, 1989b; Hay et al., 1990).

Toxoplasma gondii infection was more common in the dogs examined in this study than was *N. caninum*. Oocysts, infected meat, and transplacental modes of transmission are ways in which animals become infected with *T. gondii* (see Dubey and Beattie, 1988). Presently, all that is known about the transmission of *N. caninum* is that parenteral inoculation of cell culture grown tachyzoites produces infections in some animals (Dubey et al., 1988b; Dubey and Lindsay, 1989a; Lindsay and Dubey, 1989c), that transplacental transmission occurs in dogs and cats (Dubey and Lindsay, 1989b, c), and that infected brain containing both tachyzoites and bradyzoites produced infection in a cat (Dubey and Lindsay, 1989c). Immunocompetent animals are more resistant to inoculation with *N. caninum* than are immunocompromised (Lindsay and Dubey, 1989c) or very young animals (Dubey and Lindsay, 1989a). Other modes of transmission, such as oocysts, probably exist but have not been found. It is possible that *N. caninum* may be less prevalent in the canine population because it is not as readily transmitted as is *T. gondii*.

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Research Note

Potentially Pathogenic Species of *Acanthamoeba* and *Hartmannella* (Protozoa: Amoebida) in Sediment of the Potomac River Near Washington, D.C.

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ABSTRACT: Sediments from 6 stations sampled from the shores of the Potomac River were taken from above the Monocacy River in Maryland to below the Blue Plains Sewage Treatment Plant in Washington, D.C. Sediments were cultured on agar plates streaked with *Escherichia coli* at 20, 37, and 40°C. *Hartmannella vermiformis,* not known to cause human disease, was found at all stations. *Acanthamoeba* species occurred downstream from Great Falls. There was an abrupt increase in the number of species of *Acanthamoeba* at about the limnological fall line, near Chain Bridge. As some of the 7 species of *Acanthamoeba* encountered are serious pathogens, the tidal part of the Potomac below Chain Bridge should be regarded as a potential source of *Acanthamoeba* infections.

KEY WORDS: *Acanthamoeba, Hartmannella,* Potomac, fall line, pathogen, Amoebida, ameba.

Acanthamoeba Volkonsky, 1931, and *Hartmannella* Alexeieff, 1912, may inhabit natural areas such as the warm moist banks of any natural body of water where there is an abundance of organic matter and bacteria (Sawyer et al., 1977). They also occur in freshwater and saltwater sediments and soils throughout the world. Studies of ocean sediments show a positive correlation between the presence of sewage-associated bacteria and the presence of both *Acanthamoeba* and *Hartmannella* (Griffin, 1972; Sawyer et al., 1977; Daggett et al., 1982, 1985).

Free-living amebae of genus *Acanthamoeba* are frequent contaminants of animal tissue culture cells and are potentially pathogenic to man and animals (Sawyer et al., 1977; Daggett et al., 1982). Human infections by *Acanthamoeba* have been recorded throughout the world, and probably occur more commonly than presently recognized (Daggett et al., 1982). Some cases of amebic meningoencephalitis and corneal ulcers have been traced to *Acanthamoeba. Acanthamoeba* may contaminate contact lenses or lens-cleaning/soaking fluids (Centers for Disease Control, 1986). Chronic granulomatous infections of the skin have been reported (Brown and Neva, 1983). Patients with Acquired Immune Deficiency Syndrome (AIDS) are susceptible to infections by many uncommon pathogens, including *Acanthamoeba*; this is true of experimental infections in immunocompromised mice (Wiley, 1987).

Human infections typically occur after contact with banks or bodies of water where *Acanthamoeba* occurs (Daggett et al., 1982; Callicott et al., 1988). No human or animal diseases have been demonstrated to have been caused by any species of *Hartmannella* (Wang and Feldman,

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