Epidemiologic investigation of seroprevalence of antibodies to Toxoplasma gondii in cats and rodents

Monica L. DeFeo, MS; J. P. Dubey, MVSc, PhD; Thomas N. Mather, PhD; Richard C. Rhodes III, PhD

Objective—To provide an epidemiologic investigation of the seroprevalence of antibodies to Toxoplasma gondii in populations of cats and wild rodents in Rhode Island and to address the possible epidemiologic role of wild rodents in the spread of toxoplasmosis.

Animals—200 cats and 756 small wild rodents.

Procedure—Serum samples were obtained from 84 cats in animal shelters and 116 cats in veterinary hospitals. Serum samples were also obtained from 756 small wild rodents from multiple sites in Rhode Island. Sera from rodents and cats were assayed for antibodies to T gondii by use of the modified agglutination test.

Results—Overall, 42% (84/200) of cats had serum antibodies to T gondii. Seroprevalence was not significantly different between stray (50%; 42/84) versus client-owned (36%; 42/116) cats, between male (43%; 40/94) versus female (42%; 39/93) cats, or between indoor (26%; 7/27) versus outdoor (39%; 35/89) cats. Seroprevalence rate of trapped rodents was 0.8% (6/756). Six rodents captured in Washington County accounted for 4 of the seropositive rodents. Four of 6 of the seropositive rodents were trapped at a single site in Washington County (an abandoned barn). Five stray cats, known to have resided at the same site in Washington County as 4 of the seropositive rodents, were also found to be seropositive for antibodies to T gondii.

Conclusions and Clinical Relevance—Seroprevalence rate in rodents was not correlated with the seroprevalence rate in cats. Stray cats, especially those known to be feral, may be more likely to perpetuate the cat-mouse cycle of T gondii than client-owned cats. (Am J Vet Res 2002;63:1714–1717)

Cats are definitive reservoirs of Toxoplasma gondii. Toxoplasma gondii, an obligate intracellular protozoan parasite, is a common intestinal coccidian of the cat.1 Infected cats shed T gondii oocysts in feces, thus contaminating an environment. Cats and other animals such as wild mice and voles, wild rats, and small- to medium-sized herbivores ingest oocysts from the contaminated environment and become infected.2-4 Rodents that ingest oocysts can become intermediate hosts; T gondii encysts in intermediate hosts. Cysts can be found in the CNS and muscle tissue of intermediate hosts. Uninfected carnivores, like cats, ingest infected intermediate hosts as prey meals and face the potential of infection, a process referred to as the predator-prey cycle of T gondii.5

In humans, infection with T gondii may be acquired or congenital.1-3 Infection is generally acquired from consuming insufficiently cooked meat containing tissue cysts, or by ingesting sporulated oocysts in food, water, or from a contaminated environment.2-4 Toxoplasma gondii infection is 1 of the most widespread parasitic infections among humans throughout the world. Approximately 30% of the adult populations in the United States and United Kingdom have serum antibodies to T gondii. The prevalence rate is higher in other parts of Europe and Central and South America.6 The ability of the parasite to produce clinical disease or death in patients with depressed immunity and in infants born to women acquiring the infection during pregnancy are compelling public health concerns.7

Public health concerns associated with T gondii clearly indicate the need for epidemiologic investigation of toxoplasmosis. This study was conducted to determine whether the seroprevalence of antibodies to T gondii in populations of cats and wild rodents in Rhode Island were associated, and to address the possible epidemiologic role of wild rodents in toxoplasmosis in Rhode Island.

Materials and Methods

Selection of veterinary hospitals and shelters—Veterinary hospitals listed with the Rhode Island Veterinary Medical Association were considered as prospective participants. Alternatively, all animal shelters (public and private) in the state of Rhode Island were deemed potential sample sources. Veterinary hospitals were randomly selected from the Rhode Island Veterinary Medical Association listing, contacted, and asked to participate. A total of 10 veterinary hospitals participated in our study, providing a study population of cats from the entire geographic region of Rhode Island. Six animal shelters (a minimum of 1/county) also consented to participate in our study. Hence, serum samples from client-owned and stray cats were obtained from every county in the state.

Sample collection from cats—A cat from a veterinary hospital was selected if the cat had been admitted to the clin-
parasite biology, epidemiology, and systematics laboratory, formed by use of the modified agglutination test at the trapped rodents were anesthetized with methoxyflurane Rhode Island’s Wild Rodent Quarantine Facility. In the field, in the field and released or transported to the University of cotton and oats and then set at 10-m increments. Traps were obtained by use of a 1-mL syringe and 25-gauge needle. In another study, study personnel transported blood samples obtained from the veterinary hospitals to a University of Rhode Island laboratory where the blood samples were centrifuged and stored as previously described.

Sample collection from small rodents—All rodents were trapped from locations around the state. Trapping sites were chosen on the basis of whether cats had been observed in the immediate vicinity. All rodent collection sites were considered acceptable if a cat or cats had been observed within the trapping area. Although trapping areas varied by the geography of the site (eg, roadside, backyard, field), no trapping area was > 1,600 m². A total of 600 trap nights were conducted at each of the 13 sites throughout the state, thus accounting for 7,800 trap nights. No traps were set in Bristol or Newport counties; permission could not be secured to trap in these counties.

Live traps were used to capture wild rodents from March 1998 to February 2000. Traps were baited with fresh cotton and oats and then set at 10-m increments. Traps were checked at least once a day, and any animal trapped was bled in the field and released or transported to the University of Rhode Island’s Wild Rodent Quarantine Facility. In the field, trapped rodents were anesthetized with methoxyfluran to effect via a nose cone, and a 0.2-ML cardiac blood sample was obtained by use of a 1-ML syringe and 25-gauge needle. In the University of Rhode Island’s Wild Rodent Quarantine Facility, the animal was removed from the trap and anesthetized with methoxyflurane to effect via a nose cone. Animals were then exsanguinated via cardiac puncture by using of a 1-ML syringe and 25-gauge needle.

All blood samples were collected in serum separator tubes and then centrifuged at 3,000 × g for 10 minutes. Serum samples were decanted into storage tubes and stored at −70°C until analysis. For all rodents for which a blood sample was removed, only information on sex, body weight, trap location, and species was collected.

Serologic examination—All sera from rodents and cats were assayed for antibodies to T gondii. Assays were performed by use of the modified agglutination test at the Parasite Biology, Epidemiology, and Systematics Laboratory, USDA, Beltsville, Md.

Statistical analyses—A titer of ≥ 1:25 was considered positive for serum antibodies to T gondii in cats and wild rodents. Seroprevalence was defined as the percentage of samples testing positive for antibodies to T gondii. The χ² test was used to determine whether cat sex (male vs female), owner status (client-owned vs stray cats), risk exposure (indoor vs outdoor), or county of residence (Bristol vs Kent vs Newport vs Providence vs Washington) was associated with seropositivity for T gondii. The role of sex, owner status, and risk exposure was also evaluated within county by the χ² test. The χ² test was also used to determine whether sex (male vs female), species (white-footed mouse vs shrew vs red-backed vole vs meadow vole vs house mouse vs chipmunk), or trap location (Kent vs Providence vs Washington) was associated with seropositivity for T gondii in wild rodents. A value of P ≤ 0.05 was considered significant.

Confidence intervals of 95% were calculated for populations of male and female cats; client-owned and stray cats; indoor and outdoor cats; and cats of Bristol, Kent, Newport, Providence, and Washington Counties.

Results

Serum samples from 200 cats in Rhode Island were collected in veterinary hospitals (n = 116) and animal shelters (84) between November and December 1998 and tested for antibodies to T gondii. The confidence intervals of each population of seropositive cats were determined at a level of 95% (α = 0.05). The total population of cats (± 95% confidence interval) with serum antibodies to T gondii in the Rhode Island cat population was 42 ± 7% (84/200; Table 1). The number of stray cats with serum antibodies to T gondii was 50 ± 11% (42/84), whereas the number of client-owned cats with serum antibodies to T gondii was 36 ± 9% (42/116). Although the seroprevalence rate in stray cats was numerically higher than that of client-owned cats, the difference was not significantly different. Likewise, the number of seropositive cats did not differ between sex, by exposure risk, or by county.

In 4 of 5 counties in the state, there was no significant difference associated with seroprevalence rate and client-owned versus stray cats. However, in Washington County, ownership status of the cats was significantly related to seroprevalence rate. Of the stray cats, 62.8% (22/35) were seropositive, whereas only 35% (14/40) of the client-owned cats were seropositive.

In the 13 sites in which rodent traps were set, trapping success (rodents caught/night traps) was 9.7% (756/7800). There was substantial variation in the number of small rodents captured in a county. Of the 756 rodents caught, only 6 (0.8%) were seropositive for antibodies to T gondii. Interestingly, all seropositive rodents were from Washington County where a total of 391 rodents were captured. Of the 6 rodents that were seropositive, 4 were animals trapped at a single site—an abandoned barn in Kingstown. The seroprevalence rate of antibodies to T gondii in the small rodents trapped at the abandoned barn was 7.5% (4/53), a rate higher that that found anywhere else in the state. White-footed mice (n = 4; Peromyscus leucopus) and meadow voles (2; Microtus pennsylvanicus) accounted for the 6 seropositive rodents.

Discussion

Seroprevalence of antibodies to T gondii in cats
had serum antibodies to *T. gondii*. Sixty-eight percent (267/391) of the cats trapped was higher than that observed in cats from Ohio shelter.12 Numerous investigators have shown that small prevalence of antibodies to *T. gondii* in rodents was 6.3% in rats, 4.9% in white-footed mice (*Mus musculus*).15 In our study, blood was tested from 756 rodents, and only 6 (0.8%) were found to have serum antibodies to *T. gondii*. The low seroprevalence rate observed among rodents in our study might be attributed to the biological properties of *T. gondii* in rodents. In a field study, 5 of 7 mice that were seronegative (titer from modified agglutination test of < 1:25) for antibodies to *T. gondii* had viable parasites in their tissues.16 In an experimental infection study, 5 of 16 congenitally infected rats with no demonstrable serum antibodies to *T. gondii* harbored viable *T. gondii*.17 Apparently, the congenitally infected rodents developed immunologic tolerance to *T. gondii*.

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Sampling bias in trapping in our study, as in other studies,12 might also result in underestimation of infection. It has been suggested that mice are more susceptible than other intermediate hosts to *T. gondii* infection.12 Numerous investigators have shown that small rodents infected with *T. gondii* have altered behavior patterns that may make them more subject to predation by cats.8,16 It is also likely that some mice may die from clinical toxoplasmosis. Hence, sampling bias and diagnostic sensitivity may have resulted in an underestimation of the prevalence of *T. gondii* infection in rodents in Rhode Island.

The concentration of *T. gondii* infection in tissues of prey and epidemiologic data suggest that many cats may become infected by consuming infected tissues of intermediate hosts,17 even though they may also become infected by ingesting sporulated oocysts or transplacentally. The prey of cats, primarily small mammals and birds, are apparently infected with the parasite when they ingest sporulated oocysts from the environment.1 Small terrestrial mammals are natural reservoirs of *T. gondii*, and small mammals from farms may be at an increased risk of transmitting the parasite because they are in close contact with oocysts excreted by cats.18 Indeed, a number of investigators19-21 have shown that swine in close contact with cats and rodents have a higher exposure risk to *T. gondii*. Furthermore, the number of infected cats on a farm has been positively correlated to the number of infections in humans on the same farm.21 Stray cats of known origin (an abandoned barn) had serum antibodies to *T. gondii*. Further, the seroprevalence of antibodies to *T. gondii* in the rodents trapped at the same site was 7.5% (4/53), the highest seroprevalence rate for small rodents observed anywhere in the state. These results are consistent with those of previous studies18,19,20,21 that evaluated rodent and cat exposure as a factor increasing the risk of infection for other species. Alternatively, investigators have shown that the prevalence of *T. gondii* in rodents was drastically reduced when cats on a farm were vaccinated against oocyst shedding.16 These data support the view that small terrestrial rodents are 1 of the reservoirs for the parasite, and that cats can become infected when they prey on small rodents. These data also support the notion that small rodents probably become infected from a contaminated environment where stray or feral cats shed oocysts within a relatively confined space. There appears to be less risk of cat infection from rodents inhabiting wooded or suburban settings unless seronegative mice routinely harbor cysts.18 Our study provides data that may be used to determine the ecological risk factors within a particular region of Rhode Island. Additional studies on rodents performed within metropolitan and suburban areas, as well as on farms and the areas adjacent to farms, would help clarify the role of stray and feral versus owned cats asshed-
ders of oocysts. Results of our study indicate that controlling either small rodent or stray and feral cat populations may aid in reducing the risk of \( T \) gondii infections in humans and other domestic animals.

\*Longworth traps, Longworth Scientific Instruments, Abingdon, UK.
\*Sherman traps, H. B. Sherman Traps Inc, Tallahassee, Fla.
\*Metofane, J. A. Webster, Sterling, Mass.

References