

Seroprevalences of antibodies against *Bartonella henselae* and *Toxoplasma gondii* and fecal shedding of *Cryptosporidium* spp, *Giardia* spp, and *Toxocara cati* in feral and pet domestic cats

Felicia B. Nutter, DVM; J. P. Dubey, MVSc, PhD; Jay F. Levine, DVM, MPH; Edward B. Breitschwerdt, DVM, PhD, DACVIM; Richard B. Ford, DVM, DACVIM; Michael K. Stoskopf, DVM, PhD, DACZM

Objective—To compare seroprevalences of antibodies against *Bartonella henselae* and *Toxoplasma gondii* and fecal shedding of *Cryptosporidium* spp, *Giardia* spp, and *Toxocara cati* in feral and pet domestic cats.

Design—Prospective cross-sectional serologic and coprologic survey.

Animals—100 feral cats and 76 pet domestic cats from Randolph County, NC.

Procedure—Blood and fecal samples were collected and tested.

Results—Percentages of feral cats seropositive for antibodies against *B henselae* and *T gondii* (93% and 63%, respectively) were significantly higher than percentages of pet cats (75% and 34%). Percentages of feral and pet cats with *Cryptosporidium* spp (7% of feral cats; 6% of pet cats), *Giardia* spp (6% of feral cats; 5% of pet cats), and *T cati* ova (21% of feral cats; 18% of pet cats) in their feces were not significantly different between populations. Results of CBCs and serum biochemical analyses were not significantly different between feral and pet cats, except that feral cats had a significantly lower median PCV and significantly higher median neutrophil count.

Conclusions and Clinical Relevance—Results suggested that feral and pet cats had similar baseline health status, as reflected by results of hematologic and serum biochemical testing and similar prevalences of infection with *Cryptosporidium* spp, *Giardia* spp, and *T cati*. Feral cats did have higher seroprevalences of antibodies against *B henselae* and *T gondii* than did pet cats, but this likely was related to greater exposure to vectors of these organisms. (*J Am Vet Med Assoc* 2004;225:1394–1398)

The number of feral cats in the United States is difficult to estimate accurately, but the overall population is widely considered to be growing. A major concern expressed about feral cats, especially those resident in groups near human habitation, is that they might serve as a reservoir for infectious agents that can be transmitted

to humans.^{1,5} Indeed, several zoonotic agents, including *Bartonella henselae*, *Toxoplasma gondii*, *Cryptosporidium* spp, *Giardia* spp, and *Toxocara cati*, have been associated with feral cat populations.^{4,6} However, it is difficult to put the potential health risk that feral cats pose into perspective without comparing the health status of feral cats with that of pet domestic cats. The purpose of the study reported here, therefore, was to compare seroprevalences of antibodies against *B henselae* and *T gondii* and fecal shedding of *Cryptosporidium* spp, *Giardia* spp, and *T cati* in feral and pet domestic cats.

Materials and Methods

Cats—One hundred feral cats (47 females and 53 males) and 76 healthy pet cats (39 females and 37 males) were included in the study. The study was conducted in conjunction with a project examining the population dynamics of managed feral cat colonies in Randolph County, NC. Feral cats were humanely live trapped in box-type traps and anesthetized with an IM injection of ketamine, tiletamine, zolazepam, and xylazine. Once cats were anesthetized, a complete physical examination was performed and blood and fecal samples were collected. Cats were then vaccinated against rhinotracheitis, panleukopenia, calicivirus infection, FeLV infection, and rabies and treated with ivermectin. Only those cats considered to be at least 6 months old on the basis of eruption of the full permanent dentition were included in the study. Feral cats were included in the study on the basis of age and capture with no consideration for state of health at the time of trapping. Traps were thoroughly cleaned between captures by scrubbing with a detergent solution to remove all organic debris and then spraying with 10% bleach solution. Traps were allowed to stand for at least 15 minutes after being sprayed with the bleach solution and were then rinsed with a high-pressure hose.

Pet cats were enrolled in the study during the same period that trapping of feral cats occurred. Owners bringing domestic cats to the Asheboro Animal Hospital in Asheboro, NC, for routine preventative medical examinations or elective surgical procedures were solicited to participate in the study. Cats were manually restrained or anesthetized for collection of blood and fecal samples, depending on the reason for clin-

From the Environmental Medicine Consortium (Nutter, Levine, Stoskopf) and the Departments of Clinical Sciences (Nutter, Breitschwerdt, Ford, Stoskopf) and Population Health and Pathobiology (Levine), College of Veterinary Medicine, North Carolina State University, Raleigh, NC 27606; and the Animal Parasitic Disease Laboratory, Building 1001, Animal and Natural Resources Institute, Agricultural Research Service, USDA, Beltsville, MD 20705 (Dubey).

Supported in part by the Morris Animal Foundation, the William and Charlotte Parks Foundation, the College of Veterinary Medicine at North Carolina State University, the Randolph County Humane Society, and the North Carolina Zoological Society.

The authors thank Dorsey Kordick, Barbara Hegarty, and Chris Whittier for assistance with molecular diagnostic testing and John Canipe, Leslie Yow, Barbara Wolfe, Michael Loomis, and Roger Powell for assistance with study design.

Address correspondence to Dr. Stoskopf.

ical evaluation. Demographic information on the cats was collected through a questionnaire completed by the owners.

Sample collection—Blood samples were collected from all 100 feral cats and all 76 pet cats by means of jugular or saphenous venipuncture. Samples were divided between plain glass and EDTA-containing collection tubes. Samples in plain glass tubes were allowed to clot, and serum was obtained. Serum samples were frozen at -70°C until analyzed. Samples anticoagulated with EDTA were used for determination of CBCs; samples that were not processed within 1 hour after collection were refrigerated until they could be processed. For feral cats, all CBCs were completed within 6 hours after sample collection. For pet cats, all CBCs were completed within 24 hours after sample collection.

Fecal samples were collected from 87 feral cats (39 females and 48 males) and 66 pet cats (31 females and 35 males). For the feral cats, fecal samples were obtained directly from the trap or by means of digital rectal examination. For the pet cats, fecal samples were provided by the owner or obtained by means of digital rectal examination. Fecal samples were placed in neutral-buffered 10% formalin within 24 hours after collection and held at room temperature until analyzed.

Testing procedures—Serologic testing for antibodies against *B henselae* was performed as described⁷; cats with an antibody titer $\geq 1:64$ were considered seropositive. Frozen serum samples were sent to the USDA Animal Parasitic Disease Laboratory for testing for antibodies against *T gondii*. A modified agglutination test for IgG (sensitivity, 83%; specificity, 90%) was used⁸; cats with an antibody titer $\geq 1:25$ were considered seropositive. Blood or serum was tested for FeLV p27 core antigen and FIV antibody with a commercially available diagnostic test kit⁹; all samples were tested according to the manufacturer's directions.

Fecal samples were concentrated by means of formalin-ethyl acetate sedimentation. Concentrated samples were tested for *Cryptosporidium* spp and *Giardia* spp with a commercially available indirect fluorescent antibody test^b according to the manufacturer's instructions. Concentrated fecal samples were also examined microscopically for *T cati* ova.

Complete blood counts were performed with an automated hematology machine^c; differential cell counts were performed manually on stained blood smears.^d Serum biochemical panels, including determination of alanine transferase, alkaline phosphatase, and amylase activities and albumin, calcium, cholesterol, creatinine, globulin, glucose, potassium, total bilirubin, total protein, and urea nitrogen concentrations, were performed with an automated chemistry analyzer.^e

Statistical analyses—Results of CBCs and serum biochemical analyses; seroprevalences of antibodies against *B henselae* and *T gondii*; and fecal prevalences of *Cryptosporidium* spp, *Giardia* spp, and *T cati* were compared between feral and pet cats. The χ^2 or Fisher exact test was used for dichotomous data, and the Mann-Whitney *U* or Kruskal-Wallis test was used for continuous data. For all analyses, standard statistical software^f was used; values of $P \leq 0.05$ were considered significant.

Results

According to their owners, most pet cats had originally been obtained as strays (42/76 [55%]) or from shelters (8 [11%]), with smaller proportions having originally been obtained from a friend, neighbor, veterinarian, or classified advertisement (10 [13%]); as a

gift (5 [7%]); or from a breeder or pet shop (1 [1%]). Owners of 10 (13%) pet cats did not specify the original source of the cat. Owners of 36 of 76 (47%) pet cats indicated that their cats spent at least part of their time outdoors. Median age of the pet cats was 4 years (range, 3 months to 19 years), with 23 (30%) of the pet cats being ≤ 2 years old and 39 (51%) being < 6 years old. Owners of 32 (42%) pet cats reported giving their cats an enteric parasiticide at least once a year.

For both the feral and pet cats, results of hematologic and serum biochemical analyses were generally within ranges expected for healthy domestic cats.⁹ Values for feral cats were not significantly different from values for pet cats, except that median PCV was significantly lower for feral cats (31%) than for pet cats (38%) and median neutrophil count was significantly higher for feral cats (11,500 cells/ μL) than for pet cats (7,800 cells/ μL).

Percentages of feral and pet cats with positive FeLV and FIV assay results were low (Table 1), and there were not enough cats with positive results to permit evaluation of whether either organism was associated with coinfection with other organisms. Percentages of feral cats seropositive for antibodies against *B henselae* and *T gondii* were significantly higher than percentages of pet cats, with median titers for feral cats (median titer of antibodies against *B henselae*, 1:128; median titer of antibodies against *T gondii*, 1:50; Figure 1) being significantly higher than median titers for pet cats (median titer of antibodies against *B henselae*, 1:64; median titer of antibodies against *T gondii*, 0). Percentages of feral cats with *Cryptosporidium* spp, *Giardia* spp, or *T cati* ova in their feces were not significantly different from percentages of pet cats found to have these organisms in their feces.

For the pet cats, serum titers of antibodies against *B henselae* were significantly ($P < 0.001$) higher in younger than in older cats; however, titers were not significantly ($P = 0.958$) associated with whether cats had outdoor access. Seroprevalence of antibodies against *T gondii* was significantly ($P = 0.009$) higher in older than in younger pet cats; however, the actual specific value of the titer did not vary significantly ($P = 0.08$) with age. Seroprevalence of antibodies against *T gondii* was significantly ($P = 0.04$) higher in pet cats with outdoor access than in pet cats without outdoor access. Among pet cats, prevalence of *T cati* infection was significantly associated with outdoor access and age, with prevalence being high-

Table 1—Prevalence of infection with or exposure to various retroviral, bacterial, and protozoal organisms in feral and pet domestic cats from a rural county in North Carolina.

Organism	Feral cats	Pet cats
FIV	5/100 (5)	3/76 (4)
FeLV	4/100 (4)	1/76 (1)
<i>Bartonella henselae</i>	93/100 (93)	57/76 (75)*
<i>Toxoplasma gondii</i>	63/100 (63)	26/76 (34)*
<i>Cryptosporidium</i> spp	6/87 (7)	4/66 (6)
<i>Giardia</i> spp	5/87 (6)	3/66 (5)
<i>Toxocara cati</i>	18/87 (21)	12/66 (18)

Data are given as number positive/number tested (%).
*Significantly ($P < 0.05$) different from percentage of feral cats.

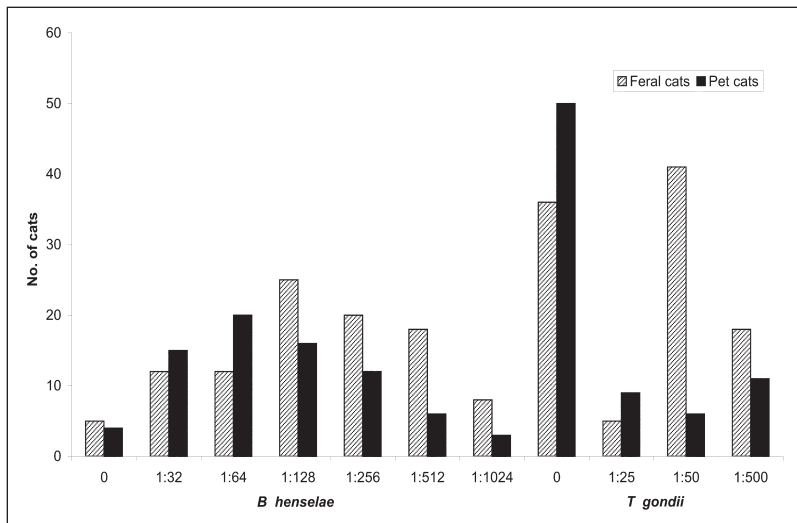


Figure 1—Serum titers of antibodies against *Bartonella henselae* and *Toxoplasma gondii* among 100 feral and 76 pet domestic cats from a rural county in North Carolina.

er in pet cats with outdoor access ($P = 0.03$) and in younger cats ($P = 0.005$). Although examination of the data suggested that pet cats with outdoor access might have had a higher prevalence of *Giardia* spp and that younger cats might have had a higher prevalence of *Cryptosporidium* spp, differences were not found to be significant. Prevalence of *T. cati* infection was not significantly associated with whether cats received anthelmintic treatment.

Discussion

Feral cats included in the present study were from a limited geographic area in a rural county in North Carolina, and pet cats were drawn from a single clinic in the same county. Overall, our results suggest that these feral and pet domestic cats had similar baseline health status, as reflected by results of hematologic and serum biochemical testing and similar prevalences of infection with FeLV, FIV, *Cryptosporidium* spp, *Giardia* spp, and *T. cati*. Feral cats did have higher seroprevalences of antibodies against *B. henselae* and *T. gondii* than did pet cats, but this likely was related to greater exposure to vectors of these organisms. Our results, therefore, conflict with the common portrayal of feral cats as disease ridden and in poor health and suggest that the health risk to humans through association with feral cats should be expected to vary with the environment (eg, temperature, humidity, and wildlife density) and management protocols the cats experience.

Infection with FeLV and FIV was evaluated as a possible contributing factor to the presence of other infectious diseases in this study, but the overall prevalences of these retroviral infections were too low to permit proper evaluation of possible associations with coinfection.

Domestic cats are considered the major reservoir for *B. henselae*, and infection in cats in the United States is widespread and common.¹⁰⁻¹³ Approximately half of cats seropositive for antibodies against *B. henselae* are also bacteremic.^{12,14,15} As in the present study, previous studies^{10,12,15-18} have also documented higher

seroprevalences in feral, stray, and shelter cats than in pet cats. Fleas^{19,20} and, to a lesser extent, ticks¹¹ are implicated in the transmission of *B. henselae*, and both seropositivity and bacteremia are associated with flea infestation.^{12,15,21} There is also a significant association between age, seropositivity, and bacteremia, with cats < 1 year old more commonly infected,^{10,12,16} as was the case for pet cats in the present study. Although feral cats included in the present study were from managed colonies, they did not receive any ectoparasite control. In addition, feral cats were judged to be between 6 months and approximately 2 years old, whereas pet cats had a median age of 4 years. Thus, it is possible that the higher seroprevalence of antibodies against

B. henselae in feral cats in the present study could at least partly be explained by the younger age and greater exposure to fleas and ticks for feral cats, compared with pet cats.

The seroprevalence of antibodies against *T. gondii* and the median antibody titer were significantly higher in feral cats than in pet cats in the present study. This is consistent with the assumed greater opportunity for feral cats to prey on intermediate hosts or become infected through contaminated soil or water. A higher seroprevalence in feral versus pet cats has been reported previously,²²⁻²⁴ although some studies^{25,26} have found no difference in seroprevalence between the 2 populations. In the present study, pet cats with outdoor access had a higher seroprevalence than did those without outdoor access, which is again consistent with greater potential for exposure to infective prey and contaminated soil and water. Thus, specific life history parameters should be considered as more important risk factors for *T. gondii* infection than broad categorization of domestic cats as feral or pet.

The overall prevalence of *Cryptosporidium* spp in feces from cats in the present study was 6.5% and did not differ between feral and pet cats. This is consistent with findings from previous studies,²⁷⁻²⁹ which reported prevalences ranging from 3.8% to 8.1%. One of these studies²⁹ also found no difference between feral and pet cat populations. Prevalence was not significantly higher among pet cats with outdoor access than among pet cats without outdoor access in the present study; however, the P value (0.06) was close to the cutoff for significance, and it is likely that the small sample size and low prevalence limited the power of this comparison. Because the diagnostic test used in this study has been shown to react with *Cryptosporidium parvum* and *Cryptosporidium felis*, it is not known which *Cryptosporidium* species was detected. Furthermore, it is difficult to speculate about the potential role of cats as reservoirs for human infection,³⁰ as cross-infection studies have produced conflicting results. The recent recognition of the 2 genotypes of *C. parvum* and the identification of *C. felis* as a distinct

species raise the possibility that investigators have been working with different genotypes or species.^{31,32} Dog ownership, not cat ownership, has been recognized as a risk factor for *Cryptosporidium* infection in HIV-positive people, but it is still sensible to consider cats and particularly kittens as a possible reservoir for human *Cryptosporidium* infection.³³

The overall prevalence of *Giardia* spp in feces from cats in the present study was 5.2% and did not differ between feral and pet cats. Given the current knowledge about *Giardia duodenalis* host range and cross-transmission and that the prevalences of infections in humans and domestic cats in the United States are similar (typically ranging between 2.4% and 7.3%), it is unclear whether cats are reservoirs for human infection or vice versa.^{27,34-36} As with *Cryptosporidium* spp, it is reasonable to consider cats as potential sources for human infection. This is particularly prudent in that a recent study³⁷ found 32 of 40 (80%) cats positive for *G duodenalis* infection by use of a polymerase chain reaction assay, suggesting that for many cats tested by means of microscopy and staining techniques, results might be false negative because of low numbers of cysts in the sample.

The overall prevalence of *T cati* in feces from cats in the present study was 19.6% and did not differ between feral and pet cats. This is consistent with recent reports^{27,34} in which prevalence ranged between 3.9% and 32.7% for cats in the United States. The association between outdoor access and prevalence among pet cats in the present study likely was related to a greater exposure to contaminated soils or paratenic hosts. Both feral and pet cats can be sources of human infection through contamination of the environment with *T cati* ova, which can survive for months to years depending on climatic conditions.³⁸

Regional differences in the prevalences of diseases in feral cats have been reported.³⁸ Thus, it is important to include appropriate control populations of pet animals when collecting data to assess the zoonotic risk posed by feral or wild carnivores. Simply reporting the prevalence in feral cat populations could potentially erroneously inflate the implied risk of exposure to zoonotic organisms posed by feral cats and inappropriately affect policy decisions made regarding feral cat management and control.

^aSNAP, Idexx Laboratories, Portland, Me.

^bMerifluor, Meridian Diagnostics, Cincinnati, Ohio.

^cBaker System 9110+, BioChem ImmunoSystems, Allentown, Pa.

^dDiff-Quik, Dade-Behring Inc, Deerfield, Ill.

^eVetScan Diagnostic Profile Plus, Abaxis Inc, Union City, Calif.

^fStatView 5, SAS Institute Inc, Cary, NC.

References

- Gross EM, Hoida G, Sadeh T. Opposition to trap-sterilize-release programs for feral cats (lett). *J Am Vet Med Assoc* 1996;208:1380-1381.
- Hughes JE. Feral cats (lett). *J Am Vet Med Assoc* 1993;203:1256-1257.
- Johnson K, Lewellen L, Lewellen J. National Pet Alliance's survey report on Santa Clara County's pet population. *Cat Fancier's Almanac* 1994;Jan:71-77.
- Yamaguchi N, Macdonald DW, Passanisi WC, et al. Parasite prevalence in free-ranging farm cats, *Felis silvestris catus*. *Epidemiol Infect* 1995;116:217-223.
- Zaubrecher KI, Smith RE. Neutering of feral cats as an alternative to eradication programs. *J Am Vet Med Assoc* 1993;203:449-452.
- August JR, Chase TM. Toxoplasmosis. *Vet Clin North Am Small Anim Pract* 1988;17:55-71.
- Kordick DL, Breitschwerdt EB. Relapsing bacteremia after blood transmission of *Bartonella henselae* to cats. *Am J Vet Res* 1997;58:492-497.
- Dubey JP, Desmonts G. Serological responses of equids fed *Toxoplasma gondii* oocysts. *Equine Vet J* 1987;19:337-339.
- Aiello SE, Mays A. *Merck veterinary manual*. 8th ed. Whitehouse Station, NJ: Merial Ltd, 1998;1291.
- Koehler JE, Glaser CA, Tappero JW. *Rochalimaea henselae* infection: a new zoonosis with the domestic cat as reservoir. *J Am Vet Med Assoc* 1994;271:531-535.
- Breitschwerdt EB, Kordick DL. *Bartonella* infection in animals: carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. *Clin Microbiol Rev* 2000;13:428-438.
- Chomel BB, Abbott RC, Kasten RW, et al. *Bartonella henselae* prevalence in domestic cats in California: risk factors and association between bacteremia and antibody titers. *J Clin Microbiol* 1995;33:2445-2450.
- Jameson P, Greene C, Regnery R, et al. Prevalence of *Bartonella henselae* antibodies in pet cats throughout regions of North America. *J Infect Dis* 1995;172:1145-1149.
- Chomel BB, Boulouis HJ, Petersen H, et al. Prevalence of *Bartonella* infection in domestic cats in Denmark. *Vet Res* 2002;33:205-213.
- Gurfield AN, Boulouis HJ, Chomel BB, et al. Epidemiology of *Bartonella* infection in domestic cats in France. *Vet Microbiol* 2001;80:185-198.
- Childs JE, Rooney JA, Cooper JL, et al. Epidemiologic observations on infection with *Rochalimaea* species among cats living in Baltimore, Md. *J Am Vet Med Assoc* 1994;204:1775-1778.
- Allenberger F, Schonbauer M, Dierich MP. Prevalence of antibody to *Rochalimaea henselae* among Austrian cats. *Eur J Pediatr* 1995;154:165.
- Baneth G, Kordick DL, Hegarty BC, et al. Comparative seroreactivity to *Bartonella henselae* and *Bartonella quintana* among cats from Israel and North Carolina. *Vet Microbiol* 1996;50:95-103.
- Chomel B, Kasten RW, Floyd-Hawkins K, et al. Experimental transmission of *Bartonella henselae* by the cat flea. *J Clin Microbiol* 1996;34:1952-1956.
- Foil L, Andress E, Freeland RL, et al. Experimental infection of domestic cats with *Bartonella henselae* by inoculation of *Ctenocephalides felis* (Siphonaptera: Pulicidae) feces. *J Med Entomol* 1998;35:625-628.
- Maruyama S, Kabeya H, Nakao R, et al. Seroprevalence of *Bartonella henselae*, *Toxoplasma gondii*, FIV and FeLV infections in domestic cats in Japan. *Microbiol Immunol* 2003;47:147-153.
- Dubey JP. Toxoplasmosis. *J Am Vet Med Assoc* 1994;205:1593-1598.
- Dubey JP, Weigel RM, Siegel AM, et al. Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. *J Parasitol* 1995;81:723-729.
- Dubey JP, Saville JA, Stanek JF, et al. Prevalence of *Toxoplasma gondii* antibodies in domestic cats from rural Ohio. *J Parasitol* 2003;88:802-803.
- DeFeo ML, Dubey JP, Mather TN, et al. Epidemiologic investigation of seroprevalence of antibodies to *Toxoplasma gondii* in cats and rodents. *Am J Vet Res* 2002;63:1714-1717.
- Smielewska-Los E, Pacon J. *Toxoplasma gondii* infection of cats in epizootiological and clinical aspects. *Pol J Vet Sci* 2002;5:227-230.
- Spain CV, Scarlett JM, Wade SE, et al. 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.
- Dubey JP. Duration of immunity to shedding of *Toxoplasma gondii* oocysts in cats. *J Parasitol* 1995;81:410-415.
- Mtambo MMA, Nash AS, Blewett DA, et al. *Cryptosporidium* infection in cats: prevalence of infection in domestic and feral cats in the Glasgow area. *Vet Rec* 1991;129:502-504.
- Caccio S, Pinter E, Fantini R, et al. Human infection with

Cryptosporidium felis: case report and literature review. *Emerg Infect Dis* 2002;8:85–86.

31. Asahi H, Koyama T, Arai H, et al. Biological nature of *Cryptosporidium* sp isolated from a cat. *Parasitol Res* 1991;77:237–240.

32. Mtambo MM, Wright SE, Blewett DA. Infectivity of a *Cryptosporidium* species isolated from a domestic cat (*Felis domesticus*) in lambs and mice. *Res Vet Sci* 1996;60:61–63.

33. Glaser C, Safrin S, Reingold A, et al. Association between *Cryptosporidium* infection and animal exposure in HIV-infected individuals. *J AIDS* 1998;17:79–82.

34. Hill SL, Cheney JM, Taton-Allen GF, et al. Prevalence of enteric zoonotic organisms in cats. *J Am Vet Med Assoc* 2000;216:687–692.

35. Kappus KD, Lundgren RG Jr, Juranek DD, et al. Intestinal parasitism in the United States: update on a continuing problem. *Am J Trop Med Hyg* 1994;50:705–713.

36. Adam RD. The biology of *Giardia* spp. *Microbiol Rev* 1991;55:706–732.

37. McGlade TR, Robertson ID, Elliot AD, et al. High prevalence of *Giardia* detected in cats by PCR. *Vet Parasitol* 2003;110:197–205.

38. Luria BJ, Levy JK, Lappin MR, et al. Prevalence of infectious diseases in feral cats in Northern Florida. *J Vet Intern Med* 2003;17:42–47.



Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Investigation of the transmission of *Mycobacterium bovis* from deer to cattle through indirect contact

Mitchell V. Palmer et al

Objective—To investigate the infection of calves with *Mycobacterium bovis* through oral exposure and transmission of *M bovis* from experimentally infected white-tailed deer to uninfected cattle through indirect contact.

Animals—24 11-month-old white-tailed deer and 28 6-month-old crossbred calves.

Procedure—In the oral exposure experiment, doses of 4.3×10^8 CFUs (high dose) or 5×10^7 CFUs (low dose) of *M bovis* were each administered orally to 4 calves; as positive controls, 2 calves received *M bovis* (1.7×10^8 CFUs) via tonsillar instillation. Calves were euthanatized and examined 133 days after exposure. Deer-to-cattle transmission was assessed in 2 phases (involving 9 uninfected calves and 12 deer each); deer were inoculated with 4×10^8 (phase I) or 7×10^8 (phase II) CFUs of *M bovis*. Calves and deer exchanged pens (phase I; 90 days' duration) or calves received uneaten feed from deer pens (phase II; 140 days' duration) daily. At completion, animals were euthanatized, and tissues were collected for bacteriologic culture and histologic examination.

Results—In the low- and high-dose groups, 3 of 4 calves and 1 of 4 calves developed tuberculosis, respectively. In phases I and II, 9 of 9 calves and 4 of 9 calves developed tuberculosis, respectively.

Conclusions and Clinical Relevance—Results indicated that experimentally infected deer can transmit *M bovis* to cattle through sharing of feed. In areas where tuberculosis is endemic in free-ranging white-tailed deer, management practices to prevent access of wildlife to feed intended for livestock should be implemented. (*Am J Vet Res* 2004;65:1483–1489)



See the midmonth issues of JAVMA for the expanded table of contents for the AJVR or log onto www.avma.org for access to all the abstracts.