Assessment of reproductive tract disease in cats at risk for *Tritrichomonas foetus* infection

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Objective—To determine whether *Tritrichomonas foetus* infection resides in reproductive tract tissues from cats housed for breeding and for which a high prevalence of colonic *T foetus* infection has been reported.

Animals—61 purebred cats in 36 catteries undergoing elective ovariohysterectomy or castration and for which reproductive tract tissues, feces, and a reproductive history were obtained.

Procedures—Reproductive tract tissues were examined for *T foetus* via light microscopy, immunohistochemical analysis, and PCR assay. History of reproductive tract disease was examined to detect statistical associations with identified or reported exposure to colonic *T foetus* infection.

Results—15 of 61 (25%) cats and 22 of 33 (67%) catteries were identified with active or reported *T foetus* infection. Light microscopic, immunohistochemical, or molecular evidence of *T foetus* infection of the reproductive tract was not detected in any cats, including 15 cats with colonic *T foetus* infection, 29 cats residing in a cattery in which *T foetus*–infected cats were identified, and 8 cats for which gross or light microscopic evidence of reproductive tract disease was identified. There were no differences in total number of litters, number of litters per breeding, kitten mortality rate, or birth defects between cats or catteries infected with *T foetus* and those for which *T foetus* infection was not identified.

Conclusions and Clinical Relevance—No evidence of reproductive tract colonization by *T foetus* was detected in this study. Accordingly, it is unlikely that reproductive tract infection with *T foetus* plays an important role in overall disease transmission. (*Am J Vet Res* 2010;71:76–81)

Tritrichomonas foetus was initially described as a venereal pathogen of cattle in the late 1800s. The first case of *T foetus* infection in the United States was reported in 1932.[1] In infected bulls, *T foetus* resides in epithelial secretions and crypts of the penis and prepuce. Trichomonads are transferred to the cranial portion of the vagina during breeding and colonize the uterus at the time of estrus. Infection of female cattle results in early fetal death and, less often, abortion or pyometra.

In 2003, *T foetus* was identified in cats as a pathogen of the gastrointestinal tract.[2] In cats, *T foetus* resides in epithelial secretions and crypts of the colon, where they cause lymphocytic, plasmacytic inflammation and chronic diarrhea.[3,4] The infection is common in purebred cats,[5] in which high-density housing conditions promote the presumptive fecal-oral transmission of the parasite. The origins of *T foetus* infections in cats are unknown.[5] There is no evidence that proximity to agricultural species is a risk factor for infection in cats.[5] In fact, recent studies[6,7] of cross-species transmission of *T foetus* between the reproductive tract of cattle and bowel of cats revealed biological and pathogenic differences in disease outcome, which suggests that bovine and feline isolates are host adapted. Nevertheless, whether *T foetus* infection in cats is restricted to the gastrointestinal tract or also can involve the reproductive tract is unknown. Venereal transmission of *T foetus* infection in cats could be an alternate explanation for the high prevalence of infection in catteries[5] in which the breeding of cats is common. Infection of the reproductive tract of cats with *T foetus* could further serve as a reservoir for persistent infection akin to that observed in chronically infected bulls. Finally, *T foetus* may play a role in the prevalence of reproductive tract disease in breeding catteries, which is a problem for which little quantitative information is currently available.

The purpose of the study reported here was to determine whether *T foetus* infection could be detected in reproductive tract tissues of cats housed for intense

Abbreviation

GAPDH Glyceraldehyde phosphate dehydrogenase

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breeding and for which a high prevalence of *T foetus* infection of the colon has been identified. We further sought to establish whether a *T foetus* infection of the colon would be associated with clinical signs or pathological evidence of reproductive tract disease in cats or catteries in which *T foetus* infection is identified.

Materials and Methods

Animals—To be eligible for participation in the study, cats had to be purebred and residing in a cattery in the United States in which an active breeding program (defined as kittens born within the past 3 years) was declared by the owner. For each cattery, any number of adult cats and up to 2 kittens (≤ 12 months old) were permitted to enter the study. The number of kittens was limited for logistic reasons.

Cats were recruited by posting announcements of the study on cat fancier e-mail list serves and posting information on the Web site of one of the authors (JLG). Announcements were also sent by facsimile to all veterinarians who had previously submitted a fecal sample to the authors' laboratory for commercial *T foetus* diagnostic testing. As an incentive for participation in the study, free PCR testing for the detection of *T foetus* was performed on feces from each cat enrolled in the study, each cattery owner received a certificate of appreciation suitable for framing, and costs for shipment of samples were paid for through the study. Written consent was obtained from all cattery owners who allowed their cats to participate in the study.

Sample collection—Samples required for the study included a reproductive tract specimen (which was obtained during elective castration or ovariohysterectomy), a fresh fecal sample, and a swab specimen obtained from the penis and prepuce (males) or vestibule (females). Participating veterinarians were provided forms for written consent of owners whose cats participated in the study, a questionnaire to be filled out by each cattery owner, and detailed instructions for sample handling and shipment. Cats were excluded if they had received antimicrobials within 1 week prior to sample collection.

For reproductive tract specimens of male cats, aseptic surgical castration was performed. One testis and vas deferens was placed in a sealed container filled with sterile saline (0.9% NaCl) solution, and the other testis and vas deferens was placed in a sealed container filled with neutral-buffered 10% formalin. A sterile cotton-tipped applicator was used to swab the prepuce and penis; the applicator was subsequently inserted into a red-top, evacuated glass tube that contained just enough isopropyl alcohol to keep the swab moist. Reproductive tract specimens of female cats were obtained via routine ovariohysterectomy. After surgery, the reproductive tract was divided longitudinally. One ovary, uterine horn, and half of the uterus were placed in a sealed container filled with sterile saline solution, and the other ovary, uterine horn, and half of the uterus were placed in a sealed container filled with neutral-buffered 10% formalin. A sterile cotton-tipped applicator was used to swab the vestibule; the applicator was subsequently inserted into a red-top, evacuated glass tube that contained just enough isopropyl alcohol to keep the swab moist.

For all cats, a 1-g fecal specimen was collected directly from the colon at the time of hospitalization. The specimen was preserved with isopropyl alcohol in a sealed red-top evacuated glass tube. Samples were shipped via overnight carrier to the authors' laboratory.

Extraction of DNA from reproductive tract samples—Extraction of DNA was performed on samples obtained from the reproductive tract specimens submitted in sterile saline solution. A scalpel blade was used to obtain ovarian or testicular tissue from the interior of each gonad and then to mince the tissue. The mucosal lining of the vas deferens or uterine body and uterine horn was scraped from the seromuscular layer with a sterile scalpel blade. The epididymis was sharply dissected from the testis, minced, and combined with tissue obtained from the vas deferens. Each sample was stored at –20°C prior to DNA extraction. Lysis buffer and proteinase K were added to samples (25 mg) of gonadal and reproductive tract tissues; tissue samples were allowed to incubate overnight at 56°C with frequent vortexing. Then DNA extractions were performed by use of a commercially available kit[a] in accordance with the tissue protocol. For swab specimens from the reproductive tract, isopropyl alcohol was removed by vacuum drying[b] of each swab. The DNA extractions were performed by use of a commercially available kit[a] in accordance with the buccal swab centrifuge protocol. Extracted DNA was stored at –20°C for up to 2 months before evaluation via PCR assays.

PCR assay of reproductive tract samples—Before PCR assay for *T foetus*, all samples of DNA extracted from reproductive tract specimens were subjected to PCR amplification of an approximately 400-bp gene sequence of feline GAPDH DNA. Reaction conditions for GAPDH gene amplification consisted of a 50-µL reaction volume of PCR buffer containing 1.25 U of *Taq* polymerase,[c] 100 pmol of each primer (forward, 5′–CCTTCATTGACCTCAACTACAT–3′; and reverse, 5′–CCAAAGTTGTCATGGATGACC–3′), 200µM of each deoxynucleoside triphosphate, and 5 µL of DNA template. Amplification of DNA was performed with an initial denaturation at 95°C for 5 minutes; 40 cycles of denaturation at 95°C for 45 seconds, annealing at 55°C for 45 seconds, and extension at 72°C for 45 seconds; and a final extension at 72°C for 5 minutes. The PCR assay for *T foetus* was performed as described elsewhere.[8] Because a PCR assay for the GAPDH gene was performed, the possibility that a negative PCR result for *T foetus* could be attributed to an insufficient amount of DNA or endogenous PCR inhibitors in the extracted DNA was ruled out for each sample.

DNA extraction and PCR assay of fecal samples—For each cat, DNA was extracted from 50 mg of feces by use of a commercially available kit[9,d] in accordance with manufacturer instructions. The DNA was stored at –20°C for ≤ 1 week prior to use in PCR assays. Each DNA sample was subjected to PCR amplification of an 876-bp gene sequence of bacterial 16S rRNA and 208-bp gene sequence of the partial *T foetus* rRNA gene

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unit, as described elsewhere.[8,10] Because a PCR assay for the bacterial 16S rRNA gene was performed, the possibility that a negative PCR assay result for *T foetus* could be attributed to endogenous PCR inhibitors in the extracted DNA was ruled out for each sample.

Light microscopy—After ≥ 24 hours of fixation, formalin-preserved reproductive tract specimens were transferred to 70% ethanol and stored for up to 1 year prior to processing. Representative samples of ovary, uterine horn, and uterus (females) or testis, epididymis, and vas deferens (males) from each cat were embedded in paraffin, sectioned at a thickness of 5 µm, and stained with H&E. Each slide was examined by a board-certified veterinary pathologist (SAH) for histologic abnormalities and trichomonads.

Immunohistochemical analysis—Immunohistochemical analysis was used to detect *T foetus* organisms in reproductive tract tissues in cats that had *T foetus* infection identified on the basis of PCR assay performed on DNA extracted from feces, in cats residing in a cattery in which a *T foetus*–infected cat was identified or a history of *T foetus* infection was reported by the cattery owner, and in cats in which gross or histologic abnormalities of the reproductive tract were identified. Tissue sections were deparaffinized in xylene (2 washes; 10 min/wash) and rehydrated by incubation in a graded series of alcohol solutions (100%, 95%, 70%, and 50% ethanol; 5 min/incubation). After tissue sections were rinsed in tap water for 5 minutes, antigen retrieval was performed by incubating tissue sections in 0.4% pepsin in 0.01N HCl for 30 minutes at 37°C. Endogenous peroxidase activity was quenched by incubation in 0.3% H_2O_2 in deionized water for 30 minutes followed by blockade of binding sites for endogenous avidin and biotin by use of a commercial kit.[e] Tissue sections were incubated in blocking serum for 20 minutes to 12 hours prior to treatment with primary antibodies. For each tissue specimen, 1 serial section was incubated with antibodies against a surface antigen of *T foetus* (polyclonal rabbit antibody[f] [diluted 1:200 in blocking serum]), whereas the other section was treated with rabbit isotype control antibodies.[g] Immunostaining was performed with a commercially available kit[h] and a peroxidase substrate.[i] Sections were rinsed in tap water, counterstained with hematoxylin or methyl green, dehydrated in a graded series of alcohol solutions, and immersed in xylene; a cover slip was then applied by use of commercial mounting medium.[j] Positive control slides mounted with sections of *T foetus*–infected feline colon were included in all assays.

Reproductive history—The owner of each cat participating in the study was required to complete a brief questionnaire.[k] The questionnaire was designed to quantify the reproductive history of the cat and cattery of that owner during the preceding 3 years.

Statistical analysis—Statistical analyses were performed by use of a commercially available software package.[l] The Fischer exact test was used for the analysis of categorical data. Median and mean ± SD were calculated when appropriate. All other variables were assessed by use of a Mann-Whitney rank sum test. Values of $P \leq 0.05$ were considered significant.

Results

Animals—Sixty-one cats from 36 catteries met the criteria for inclusion and participated in the study. There were 40 females and 21 males. The females comprised 34 adults (median age, 3 years; range, 1 to 7.5 years) and 6 kittens (median age, 5.25 months; range, 3 to 5.5 months). Males comprised 10 adults (median age, 2.8 years; range, 1.25 to 11 years) and 11 kittens (median age, 6 months; range, 4 to 11 months). Breeds represented included Abyssinian (n = 15), Bengal (12), Ragdoll (11), Tonkinese (4), Birman (3), Norwegian Forest Cat (3), Oriental Shorthair (3), Bombay (2), Burmese (2), Chartreux (2), Japanese Bobtail (2), Russian Blue (1), and Siamese (1).

Intestinal infection with *T foetus*—Bacterial 16S rRNA gene products were amplified from DNA extracted from the feces of each cat participating in the study. *Tritrichomonas foetus* rRNA gene products were amplified from DNA extracted from the feces of 15 of 61 (25%) cats. Among female cats, 9 of 34 (26%) adult and 0 of 6 kittens were identified with *T foetus* infection. Among the male cats, 3 of 10 adults and 3 of 11 kittens were identified with *T foetus* infection. Infection with *T foetus* was significantly ($P = 0.009$) more likely to be diagnosed in male kittens than in female kittens. Two females (1 adult and 1 kitten) were reported by their owners to have received ronidazole for treatment of *T foetus* infection; both of these cats had negative test results for *T foetus*. Four males were reported by their owners to have received ronidazole for treatment of *T foetus* infection; 2 of these cats (1 adult and 1 kitten) had positive test results for *T foetus*, whereas the other 2 (both kittens) had negative test results for *T foetus*. A current problem with diarrhea was reported for 15 of 60 (25%) cats. There was no significant difference in the prevalence of diarrhea in cats infected with *T foetus*, compared with that for cats in which *T foetus* infection was not detected.

Reproductive tract infection with *T foetus*—The GAPDH gene products were amplified from DNA extracted from reproductive tract tissues and swab specimens obtained from each cat in the study. However, *T foetus* rRNA gene products were not amplified from DNA extracted from reproductive tract tissues or swab specimens from any cats in the study.

Histologic examination of reproductive tract tissues—Microscopic abnormalities were identified in reproductive tract tissues obtained from 8 of 34 (24%) adult female cats. In 4 of these cats, gross evidence of pyometra or cystic distention of the uterus was observed. Abnormal histopathologic findings ranged from mild periglandular lymphocytic inflammation to severe endometritis characterized by gland distention with sloughed necrotic epithelial cells and large numbers of neutrophils accompanied by multifocal loss or rupture of endometrial glands and superficial epithelium resulting in large amounts of necrotic debris in the uterine lumen. The endometrial stroma was diffusely expanded by numerous neutrophils and aggregates of lympho-

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cytes and plasma cells. Histopathologic abnormalities were not observed in male cats or female kittens. Trichomonads were not identified in any of the cats. There was no significant difference in the prevalence of histologic abnormalities of the reproductive tract between cats infected or exposed to *T foetus*, compared with the prevalence for cats in which *T foetus* infection or exposure was not identified.

Immunohistochemical analysis for *T foetus* in reproductive tract tissues—Immunohistochemical analysis was performed on reproductive tract tissues obtained from 52 cats. Trichomonads were not observed by use of immunohistochemical analysis in any of these reproductive tract specimens. In 15 cats for which immunohistochemical analysis was performed, *T foetus* infection was identified on the basis of PCR assays performed on DNA extracted from feces. Twenty-nine additional cats resided in a cattery in which a *T foetus*–infected cat was identified or for which a history of *T foetus* infection in the cat or cattery was reported. Five cats were included solely because of gross or microscopic abnormalities of the reproductive tract. In contrast to results for the reproductive tract specimens, trichomonads were consistently identified by immunohistochemical analysis in sections of colonic mucosa obtained from a cat with naturally occurring *T foetus* infection that were included as positive control samples.

History of reproductive tract disease in cats with and without *T foetus* infection—A history of breeding activity was not reported for any of the kittens in the study. Adult female cats were bred 0 to 8 times (mean ± SD, 3.1 ± 2.4 breedings; median, 2 breedings) during the past 3 years and gave birth to 0 to 8 litters (mean, 2.6 ± 2.2 litters; median, 2 litters). Calculated percentage for litters per breeding ranged from 17% to 100% (mean, 87 ± 23%; median, 100%). There was no significant difference in total number of litters or number of litters per breeding between cats infected with *T foetus* and cats for which *T foetus* infection was not identified. The proportion of adult female cats for which a history of reproductive problems was reported was 19 of 34 (56%). Reproductive problems reported were death of kittens of undetermined cause at < 4 weeks of age (n = 10 cats), stillborn kittens (7), small litter size (3), need for cesarean section (2), insufficient milk production (2), pyometra (1), premature birth of kittens (1), and bloody vaginal discharge (1). Five cats gave birth to kittens with defects described as a flat chest, cleft palate, lobster claw paw, or unspecified. There was no significant difference in the prevalence or type of reproductive problems reported for female cats infected with *T foetus*, compared with results for female cats in which *T foetus* infection was not identified.

Adult male cats were allowed to breed 2 to 20 times (mean ± SD, 7.8 ± 7.2 breedings; median, 6 breedings) during the past 3 years, which resulted in the birth of 2 to 20 litters (mean, 6.8 ± 6.4 litters; median, 5 litters). Calculated median percentage for litters per breeding ranged from 65% to 100% (mean ± SD, 91 ± 14%; median, 100%). There was no significant difference in total number of litters or number of litters per breeding between male cats infected with *T foetus* and those for male cats in which *T foetus* infection was not identified. The proportion of adult male cats for which a history of reproductive problems was reported was 3 of 9. Reproductive history was unavailable for 1 male cat. Reproductive problems reported were death of kittens of undetermined cause at < 4 weeks of age (n = 3 cats), stillborn kittens (2), and pyometra in a female to which the male was bred (1). Reproductive problems were not reported for any male cat with *T foetus* infection.

History of reproductive tract disease in catteries with and without *T foetus* infection—A complete history of reproductive tract disease was available for 33 of the 36 catteries. An owner-reported history of *T foetus* infection or presence in the cattery of a cat with *T foetus* infection was identified in 22 of 33 (67%) catteries. In 8 of these catteries, no prior history of *T foetus* infection was reported by the owner. Catteries contained 5 to 34 cats (mean ± SD, 14 ± 7 cats; median, 14 cats), which included both adult cats and kittens. Cattery owners reported that breeding was performed 5 to 70 times (mean, 22 ± 17 breedings; median, 16 breedings) during the past 3 years, which resulted in the birth of 4 to 59 litters (mean ± SD, 16 ± 12 litters; median, 12 litters) and 16 to 210 kittens (mean ± SD, 57 ± 42 kittens; median, 43 kittens). Calculated percentage for litters per breeding ranged from 21% to 100% (mean, 83 ± 19%; median, 89.4%), and number of kittens per litter ranged from 1 to 5 (mean ± SD, 3.6 ± 0.92 kittens/litter; median, 3.6 kittens/litter). Cattery owners reported that 0 to 28 kittens (mean, 7.3 ± 6.6 kittens; median, 5 kittens) were stillborn or died within 4 weeks after birth during the past 3 years. Causes given for death of kittens were birth defects (n = 10 catteries), pneumonia or septicemia (10), undetermined (8), and stillborn (6). Birth defects were described as a flat chest, umbilical hernia, cleft palate, diaphragmatic hernia, heart defects, and craniofacial deformity.

The number of female cats per cattery for which a vaginal discharge was reported by the owner during the past 3 years ranged from 0 to 10 (mean ± SD, 1.8 ± 2.2 female cats/cattery; median, 1 female cat/cattery). Only 2 cattery owners reported a male cat with penile discharge during the past 3 years. Cattery owners reported contacting a veterinarian because of a difficult birth 0 to 7 times (mean, 1.5 ± 1.9 difficult births; median, 1 difficult birth) during the past 3 years. There were no significant differences in reproductive history between catteries without *T foetus* infection and catteries in which *T foetus* infection was reported or identified.

Discussion

Tritrichomonas foetus is recognized as a sexually transmitted reproductive pathogen of cattle. In domestic cats, *T foetus* infection is presumably confined to the distal portions of the gastrointestinal tract[3,4]; however, to our knowledge, studies to examine the reproductive tract of infected cats have not been performed. In the study reported here, reproductive tract specimens, from cats housed for intense breeding and for which a high risk of *T foetus* infection has been reported,[5] were examined for light microscopic, immunohistochemical, and molecular evidence of *T foetus* infection. Within this

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study population, 25% of the cats and 67% of the catteries were identified with active or historical *T foetus* infection. The prevalence of *T foetus* infection among catteries in this study is larger than that reported in another study.[5] This presumably reflected our successful use of incentives to participate in the study (free PCR testing of fecal samples to detect *T foetus*) that would appeal to cattery owners aware of or concerned about *T foetus* infection. It is also likely that we underestimated the true number of infected catteries in this study because a lack of infection was based on awareness of the owner and not on results of comprehensive diagnostic testing.

Light microscopic, immunohistochemical, or molecular evidence of *T foetus* infection of the reproductive tract was not detected in any cats in this study. This was despite the inclusion of 15 cats with active *T foetus* infection of the colon, 29 cats residing in a breeding cattery in which a *T foetus*–infected cat was identified or for which a history of *T foetus* infection was reported, and 8 cats for which gross or light microscopic evidence of reproductive tract disease was identified.

In addition to the lack of infection in the reproductive tract, failure to detect *T foetus* in reproductive tract specimens obtained from male cats in this study could have been attributed to age, conservative means of sampling the penis and prepuce, presence of low numbers of organisms, or a low prevalence of infection. In bulls, the *T foetus* infection rate increases with age and is attributed to a deepening of the epithelial crypts of the penis and prepuce.[11–14] The preferred method for detecting the organism in bulls is by repeated and vigorous scraping of the fornix with a sterile, 18-inch, plastic insemination pipette. Whether the penis and prepuce of cats of any age are hospitable to colonization by *T foetus* is unknown. We chose to conservatively obtain swab specimens rather than to scrape the penis and prepuce of cats in this study on the basis of reluctance to cause harm. Nevertheless, all preputial swab specimens contained sufficient cellular material to detect feline GAPDH DNA, whereas *T foetus* DNA was not identified. Although we did not obtain samples from the urethra or prostate, *T foetus* has not been detected in these locations nor in the epididymides of infected bulls.[11] Our decision to examine additional portions of the feline reproductive tract that are not known to be infected in bulls (eg, vas deferens and gonads) was based on an assumption that the infection would not necessarily behave the same in each of these species and the fact that the specimens could be easily obtained. In this study, *T foetus* infection appeared to be more common in male kittens than in female kittens. Similar findings were not observed in adult cats. The clinical importance of this observation remains unknown because the number of kittens identified with *T foetus* infection in this study was small.

In addition to the lack of infection of the reproductive tract, failure to detect *T foetus* in reproductive tract specimens obtained from female cats in this study may have been attributable to the timing of sample procurement, presence of few organisms, or a low prevalence of infection. In female cattle, *T foetus* is deposited in the proximal portion of the vagina by an infected bull and enters the lumen of the uterus at the time of estrus. Female cattle reportedly are able to eliminate the infection between 1 and 4 months after exposure.[15,16] In female cattle, topical application of *T foetus* organisms to the external aspect of the vulva fails to cause infection, in contrast to results after deposition of organisms in the cranial portion of the vagina.[17] Whether topical or vaginal deposition of *T foetus* results in colonization of the uterus in cats is unknown. In contrast to female cattle, the vaginal lumen of cats is long, narrow, and indistensible,[18] and the cervix is a major barrier to penetration by spermatozoa, except during a 48-hour period of peak serum estrogen concentrations during estrus.[19]

We are aware of a single case report[20] in which *T foetus* organisms were observed by use of light microscopic examination of uterine contents obtained during ovariohysterectomy in a cat with pyometra. Interestingly, the cat had clinical signs of hypersexuality and was administered medroxyprogesterone acetate for treatment of presumptive hyperestrogenemia. It is possible that hyperestrogenemia predisposed the reproductive tract of this cat to infection by *T foetus*. Unfortunately, histologic examination of uterine tissues of this cat was not performed.

No evidence of infection of the reproductive tract by *T foetus* was detected in the 40 female cats examined in this study. This was despite the inclusion of 4 cats with severe endometritis and pyometra, 2 of which had a concurrent infection of the colon with *T foetus* and 1 of which resided in a cattery in which *T foetus*–infected cats were identified. In heifers and cows, *T foetus* organisms are found on the surface of the endometrium and within the lumen of endometrial glands.[15,21–23] It is believed that pyometra and abortion occur in < 5% of *T foetus*–infected cattle. It is interesting that in cattle, trichomonads have been identified in the esophagus, abomasum, and intestines of late-term fetuses aborted as a result of *T foetus* infection.[24] These fetuses have been described as having necrotizing enteritis with tissue invasion of trichomonads,[25] which is similar to that observed in some cats with enteric infection.[4] On the basis of this observation, examination of the gastrointestinal tract of aborted or stillborn kittens for evidence of *T foetus* may be worth investigating. It is important to recognize that the reproductive histories of the catteries described in this report were derived from information for individual cats whose infection status at the time they were bred or gave birth was unknown. Accordingly, it is worthwhile to continue to gather prospective data on *T foetus* infection in individual cats with active reproductive tract disease.

The primary impact of *T foetus* infection in cattle is prolongation of the calving interval as a result of early fetal death. In the present study, there appeared to be no difference in the total number of litters, number of litters per breeding, kitten mortality rate, or birth defects between cats or catteries infected with *T foetus* and those for which *T foetus* infection was not identified. Importantly, these variables vary considerably among breeds of cats,[26] thereby making it difficult to make a cross-sectional comparison of these findings. Also, these data were generated on the basis of owner memory. However, our historical reproductive findings are generally similar to those reported and summarized by other investigtors.[26]

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In the study reported here, there was no light microscopic, immunohistochemical, or molecular evidence that the reproductive tract of cats was colonized by *T foetus*, despite the inclusion of cats housed for intense breeding and for which a high risk of *T foetus* infection and reproductive tract disease was identified. Accordingly, it would appear unlikely that infection of the reproductive tract with *T foetus* plays an important role in transmission of the disease or in pathological changes to the reproductive tract of cats in breeding catteries.

a. QIAmp DNA Mini Kit, Qiagen, Valencia, Calif.
b. Eppendorf Vacufuge concentrator 5301, Brinkmann Instruments Inc, Westbury, NY.
c. AmpliTaq Gold DNA polymerase, Perkin-Elmer, Foster City, Calif.
d. ZR fecal DNA kit, Zymo Research, Orange, Calif.
e. Avidin/Biotin blocking kit, Zymed Laboratories Inc, San Francisco, Calif.
f. Anti-TF1.15 antibody, provided by Dr. Lynette B. Corbeil, Department of Pathology, School of Medicine, University of California, San Diego, Calif.
g. Zymed Laboratories Inc, San Francisco, Calif.
h. Vectastain Universal Elite ABC Kit, Vector Laboratories, Burlingame, Calif.
i. Vector NovaRED substrate kit for peroxidase, Vector Laboratories, Burlingame, Calif.
j. VectaMount mounting medium, Vector Laboratories, Burlingame, Calif.
k. Copies of the questionnaire available from the author on request.
l. SigmaStat, Jandell Scientific, San Rafael, Calif.

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