This article may seem an odd combination of protozoal parasites to some readers; however, these two protozoal parasites are similar in their origins and both are causative agents of diarrhea in dogs and cats.\textsuperscript{1,2} \textit{Giardia} spp and \textit{Tritrichomonas foetus} are both flagellated Protists and because of their close association with host mucus membranes, they are considered to be muscoflagellates. Both live, feed, and disrupt the intestinal tract of dogs and cats. \textit{Giardia} spp causes diarrhea in both dogs and cats and although \textit{Tritrichomonas foetus} has rarely been found in the diarrheic feces of dogs, it is now considered to be the cause of an emerging infectious diarrheal disease of cats.\textsuperscript{1} The in-depth 2007 review article in \textit{Science} highlights several similarities of these two parasites at the molecular level, including metabolic and genetic traits, and suggests that they are of sister lineages.\textsuperscript{4} These two protozoal parasites are different, yet interestingly similar in their biology and control (Fig. 1).

Diarrhea is a common clinical entity in small animal veterinary practice, and has many possible causes including stress, disturbances in water balance, nutritional and immune status, dietary indiscretion, neoplasia, inflammatory disease, and bacterial, parasitic, or viral pathogens or coinfections with any combination of these.\textsuperscript{5,6} Any disruption of the normal intestinal flora and function can lead to an abnormal altered pH within the milieu, resulting in the overpopulation of opportunistic pathogens. Stress has an effect on normal function and the immunologic integrity of the gut.\textsuperscript{7} Giardiasis and intestinal tritrichomoniasis are more common in animals housed in stressful situations, pet stores, puppy mills, shelters, and catteries.\textsuperscript{8–10} The host-parasite relationships that cause diarrhea are complex and may be affected by many factors.\textsuperscript{11,12}

Special concern for clients who are immunocompromised must be given in regard to proper diagnosis and sanitation measures when their pet has diarrhea.\textsuperscript{13} Many
species of Giardia occur worldwide in many hosts and some do have the potential to be zoonotic; T foetus occurs in cattle, pigs, dogs, and cats, and has not been considered to be zoonotic.\(^1\) However, humans may be infected with the venereal trichomand species, Trichomonas vaginalis. Practical diagnoses of the underlying parasitologic cause of diarrheal infections in all animals are based on host, site specificity, direct observation, and molecular techniques. Efforts to determine a specific diagnosis are highly recommended. The zoonotic potential of diarrhea of dogs and cats, regardless of causative agent, is possible, and sanitation measures and treatment of all animals in the household when indicated cannot be overemphasized to clients.

GIARDIASIS

Giardiasis is caused by infections with Giardia spp parasites and occurs in many animal species including humans, cattle, sheep, goats, dogs, cat, rodents, birds, and amphibians. This cosmopolitan parasite causes a malabsorption syndrome in many of the humans and animals that it parasitizes.\(^14\) The species of this genus is specific in some animals and intertwined in others. Many species and genotypes have been described, and it is recognized that some differ in host range but many are restricted to one host. The prevalence of each of the 7 genetic assemblies varies considerably from country to country.\(^15\)
**GENUS GIARDIA KUNSTLER 1882**

Giardia spp are in the Order Diplomonadida, whose key characteristics include cuplike depressions on the nuclear surface in front of one basal body, three basal body-associated microtubular fibers, and no Golgi, mitochondria, hydrogenosomes, or axostyle. Giardia spp are in the suborder Diplomonadina, family Hexamitidae. These organisms have two karyomastigonts (nuclei and associated flagella) that are arranged in binary symmetry.²

There are at least 41 species of Giardia that have been described, which live in vertebrates and are distributed in three morphologic groups corresponding to the species Giardia intestinalis from man and other animals.² However, there are several differing opinions concerning the nomenclature of members of this genus.¹,¹⁵ Over the years, the species infecting humans has had several names, several from the scientists who first described the organism or the location within the host, including Giardia lamblia, Giardia duodenalis, Giardia intestinalis, and most recently Giardia enterica.

Antonie van Leeuwenhoek first described and sketched the trophozoites and cyst forms in 1681. The Czech physician, Vilem Lambl, has been credited with the actual discovery of Giardia spp parasites in 1850 when he observed the organisms in the stools of children with diarrhea. He named these organisms Cermomonas intestinalis. Raphael Anatole Émile Blanchard renamed the organism Lamblia intestinalis in 1888.¹⁶ Charles Wardell Stiles changed the name to G lamblia in 1915 in honor of Professor A. Giard of Paris and Dr F. Lambl of Prague.¹⁷ Some of the species that have remained consistently associated with one host are Giardia muris in mice, Giardia agilis in amphibians, and Giardia psittaci in birds.¹

The Giardia species that is found in humans is considered as a species complex with members discussed in the terms of assemblages that are based on genotypes, determined by various molecular techniques.¹,¹⁵,¹⁸ The polymerase chain reaction (PCR) techniques that have been used to define the members of the assemblages include glutamate dehydrogenase (GDH), elongation factor 1-α (ef1-α), triphosphate isomerase (TPI), and rDNA.⁶,¹⁹,²⁰ The 7 genetic assemblages are lettered and most, but not all are species specific.⁹,¹⁵,²⁰ Assemblages A and B are found in both humans and animals; assemblages C to G are usually host specific with assemblages C and D in canines, assemblage E in hoof stock such as cattle, sheep, goats, pigs, and water buffaloes, and assemblages F in cats and G in rats. It is generally agreed that the genetics of parasites in this genus are still not clearly defined, and because there is now a slight possibility of meiosis and genetic exchange, the nomenclature of this genus, the population genetics, and host specificity are still under intense investigation.⁴,¹⁵,²¹

**MORPHOLOGY AND LIFE CYCLE**

There are two life stages, the feeding trophozoite and the environmentally stable cyst. Giardia spp trophozoites feed in the jejunum and ileum of the small intestine whereas other intestinal flagellates are found in the cecum and colon.¹ The cysts are environmentally stable and dormant yet “spring loaded for action” to excyst on ingestion.²²

After ingestion of infective cysts, the acidic conditions in the stomach stimulate the relatively quick excystation process that involves changes in the mRNA expression and cell ultrastructure. When the excysting parasites reach the alkaline environment of the small intestine they are exposed to digestive enzymes and bile salts, which enables the completion of the excystation process. The resulting trophozoites (after excystation the four nucleated stage divides in to two trophozoites that each have
two nuclei) that emerge from each cyst adhere to enterocytes by means of the ventral disk, start feeding, and establish an infection. The trophozoites, 12 to 17 by 7 to 10 \( \mu m \) in size, contain two slender axonemes located inside of the trophozoites, and basal bodies. The four pairs of flagella are located between the two endosome (prominent nucleolus)-filled nuclei in the middle of the cell. \( Giardia \) spp trophozoites are teardrop-shaped, and have been described as split pears with a flattened ventral surface occupied by the ventral adhesive disk tapering posteriorly to a tail.

The trophozoites are teardrop-shaped, and have been described as split pears with a flattened ventral surface occupied by the ventral adhesive disk tapering posteriorly to a tail. The adhesive disk is uniquely adapted for attachment to the mucous epithelial cells lining the intestine. The parasite alternates between attachment and free-swimming phases. The complexities of division and formation of new functional adhesive disks is not fully understood. This information is critical to the understanding of the pathogenesis and treatment protocols for giardiasis because the number of feeding parasites dictates the severity of disease.

Encystment is an adaptation for survival outside of the host, “packing its bags” so to speak, as it folds in on itself and forms a protective coat around the flagella and internal structures, ready to complete its journey through the host and pass out into the environment. The transformation of the trophozoite to the cyst occurs when the surrounding internal environment changes and the organism is stressed. This stress may be due to water reabsorption, or chemical and enzyme clues as the organism passes down through the intestinal tract. Details of this fascinating process can be found in the newly publish article by Midlej and Benchimol. The signals for encystment have yet been fully identified; however, a reduction in the concentration of free cholesterol may be the first molecular signal. During the encystment process, encystment proteins are released from vesicles and are the basis of \( Giardia \) spp fecal antigen tests. The infective cysts (9–13 \( \times \) 7–9 \( \mu m \)) are passed into the environment in the feces, and the cyst is considered to be the diagnostic stage of the parasite.

Cyst excretion from the host is intermittent. The excreted cysts contain a mitotically arrested trophozoite that can remain infectious for months in cool, wet environments. Knowledge of the biochemical composition and functional properties of the complex outer membranous system have been described in detail, and just how tough these cysts are in the environment are starting to be understood. Cyst environmental survival is a major factor for the high prevalence of giardiasis worldwide.

Trophozoites are rarely passed directly into the environment. This situation may occur if the intestinal motility is extremely fast and the resulting diarrhea is very liquid. These trophozoites will soon perish outside of the host and will not be infective to other animals or humans.

**EPIDEMIOLOGY**

The reported incidence and prevalence of giardiasis in humans and animals has been documented worldwide, but varies considerably among populations and geographic locations. Few studies have compared \( Giardia \) spp isolates from humans and animals living in the same locality or household. \( Giardia \) spp are transmitted to humans and animals via the fecal oral route. \( Giardia \) spp cysts are shed in the feces intermittently and are immediately infective. Cysts survive in moist environments and are resistant to most disinfectants, are able to survive water treatment disinfection, and can pass through physical barriers such as filters.

Waterborne outbreaks in human populations have been devastating in both rural and urban communities. The largest waterborne acute giardiasis outbreak described to date occurred in Norway in the fall of 2004, with more than 1500 people
affected. Continuous *Giardia* spp infections, due to poor sanitation, occur in developing countries, however, people living in urban environments are not without risk where *Giardia* is the most common parasite of humans. Hand washing and other common sanitation practices in daycare facilities and food service operations are important to prevent person-to-person spread of the infective cysts to susceptible hosts in these situations.

The incidence of giardiasis in animals is greatest in populations of dogs and cats in confined breeding facilities and animal shelters with poor sanitation and crowded conditions. Animals in unsanitary confined quarters are easily reinfected by grooming infective cysts from their own hair coat or from others. Inanimate objects, such as food bowls and cages in catteries and kennels, may serve as reservoirs of infective cysts.

Risk factors for people include age, location, lifestyle, and immune status; for animals risk factors include being young and housed in a stressful, unsanitary situation.

**PATHOGENIC PROCESS**

Most dogs and cats are able to ingest infective *Giardia* spp cysts with no adverse effects. Others develop varying degrees of illness and clinical signs. The numbers of cysts ingested plays a role in the resulting pathogenesis. In humans, 10 to 100 cysts are required to establish an infection. It makes sense that the severity of the pathogenesis is related to the dose of infective cysts. If an otherwise healthy individual person or animal ingests a large number of cysts from a contaminated source, the immune system could be overwhelmed and disease would follow.

The host-parasite interaction and resulting pathogenesis at the intestinal villi has been studied intensively in both humans and animals. Initial stress on the animal has been shown to jump-start this pathologic process with *Giardia* spp as well as other organisms and causative agents of diarrhea. T-lymphocyte–mediated pathogenesis is common to this and a variety of other enteropathies. A series of cascading events occurs, starting with the loss of the microvillus brush border after parasite attachment. These events result in disaccharidase insufficiencies and malabsorption of electrolytes, nutrients, and water. The enteroctytic injury is mediated by activated host T lymphocytes resulting from the parasite disrupting the epithelial tight junctions, increasing intestinal permeability and destruction of enterocytes. It has also been shown that goblet cells in *Giardia* infected dogs become hyperplasic and generate gates, allowing tissue invasion by the trophozoites. Hyper excretion of chloride ions has also been reported. Different strains of *Giardia* parasites have been shown to vary in their ability to cause enterocyte apoptosis. The total effects of parasite attachment and disruption of the intestinal integrity may eventually lead to the development of severe chronic intestinal disorders including inflammatory bowel disease, Crohn disease, and food allergies. Further research using *Giardia* spp as the test model may actually result in new therapeutic targets for these devastating chronic diseases in people and animals.

**CLINICAL SIGNS**

Infections with *Giardia* are common, but most animals and people remain asymptomatic. The severity of clinical signs varies with age, stress level, immune and nutritional status, as well as species of animal host and strain of parasite. The resultant small bowel diarrhea is usually self limiting. However, the clinical signs range from slight abdominal discomfort to severe abdominal pain and cramping, explosive watery, foul-smelling diarrhea, with malabsorption and possible physical growth arrest.
Acute giardiasis develops after an incubation period of 1 to 14 days (average, 7 days) in people and usually lasts 1 to 3 weeks. The prepatent period in dogs is usually 1 to 2 weeks and can last for 24 hours to months.

DIAGNOSIS

Giardiasis is often a diagnostic dilemma. *Giardia* spp are one of the most commonly misdiagnosed, underdiagnosed, and overdiagnosed parasites in veterinary practices today. The gold standard technique is fecal flotation with centrifugation in zinc sulfate, stained with Lugol iodine. Other in-clinic techniques include the saline direct fecal smear and the SNAP *Giardia* antigen test (IDEXX Laboratories). Reference laboratories provide additional diagnostic techniques including immunofluorescent assays (IFA) (eg, the MeriFluor *Cryptosporidium/Giardia*) and PCR. The Companion Animal Parasite Council recommends “testing symptomatic (intermittently or consistently diarrheic) dogs and cats with a combination of direct smear, fecal flotation with centrifugation, and a sensitive, specific fecal ELISA optimized for use in companion animals. Repeat testing performed over several (usually alternating) days may be necessary to identify infection.”

There are many reasons why fecal flotation is challenging for private practitioners, including poor or no equipment including centrifuges, microscopes, micrometers, availability of proper flotation solutions, and inability to correctly identify the small delicate cysts. Cysts are shed intermittently, and repeated fecal analyses may be needed before cysts are recovered in a sample. Many pseudoparasites, such as yeasts, plant remnants, and debris, have been mistaken for these tiny organisms.

In many clinics the only diagnostic technique used is the direct fecal smear; however, trophozoites are fragile and are often found only in very fresh, diarrheic feces, and can be confused with other flagellates or anything that moves. Trophozoites are rarely seen in direct fecal smears unless the sample is taken directly from the rectum and the feces are diarrheic. Mobile trophozoites have a tumbling or falling-leaf motion. Cysts are difficult to identify in wet mounts, and the sample size is usually inadequate for diagnosis.

The SNAP *Giardia* Test for dogs and cats (IDEXX Laboratories) is available to veterinarians, is easy to use, and reliably identifies *Giardia* spp cyst wall protein shed in dog or cat feces. The enzyme-linked immunosorbent assay (ELISA)-based technology of the SNAP *Giardia* antigen test uses antibodies specific to *Giardia* cyst wall proteins released into the feces during the encystation process. The lateral flow technology allows a blue color to be visualized when antibody binds *Giardia* cyst wall antigen.

There have been several population surveys and comparison studies completed, with interesting results. Some incongruent results have come from the evaluation of various testing techniques (direct smear, fecal flotation, ELISA), where none of the three methods consistently agreed with the others nor did any one method prove to be superior in a particular group of animals. None of the current methods for diagnosing *Giardia* as the cause of diarrhea in dogs and cats is 100% reliable. Flotation and antigen testing can be used in combination, and if more than one fecal sample is analyzed, a solid accurate diagnosis can be made.

TREATMENT AND CONTROL

There is a strong argument for not treating asymptomatic people and animals for giardiasis. *Giardia* cysts are ubiquitous in the environment, and most people and animals will be exposed to cysts but most will not become ill. On the other hand, treatment of
Giardia in dogs and cats, ill or asymptomatic, has been strongly recommended because of the possible zoonotic risk. In the animal with diarrhea, medical treatment should definitely be initiated. Fenbendazole (50 mg/kg once daily for 3 or 5 days) or the combination product Drontal Plus (febantel-pyrantel-praziquantel, 37.8 mg/kg, 7.56 mg/kg, 7.56 mg/kg, respectively) (febantel is metabolized to fenbendazole) is the most current treatment recommendation for giardiasis in dogs and cats. Fenbendazole, a benzimidazole anthelmintic, binds to the α-tubulin cytoskeleton of trophozoites. Energy metabolism is thus inhibited by lack of glucose uptake.

Veterinarians have routinely treated giardiasis in dogs and cats with metronidazole (22 mg/kg orally twice daily for 5 days). This compound is an effective therapy for diarrhea in dogs and cats regardless of cause, and may definitely be used in combination with fenbendazole to relieve clinical signs and eliminate parasites. Metronidazole is in the nitroimidazole class of agents. Once the drug enters the parasite it becomes activated by the reduction of the nitro group and binds covalently to DNA molecules, resulting in irreversible helical damage and death of the organism. This drug should not be used in higher doses in small animals due to the adverse side effects. Tinidazole (Tindamax or Fasigyn) is a second-generation nitroimidazole that is closely related to metronidazole. It has recently been approved in the United States for the treatment of giardiasis in people. The mechanism of action is not clearly understood. Ronidazole (Ridzol) is also in the same class of drugs as tinidazole, has been used for treatment of Blackhead in turkeys, and was recently tried for treatment of T. foetus in cats. There are several other compounds including furazolidone, quinacrine, albendazole, and oxfendazole that have been used for treatment of giardiasis but are not recommended at this time.

Unfortunately, there are many cases of giardiasis in humans and animals that do not respond to initial treatment efforts. It is the authors’ opinion that reinfection is the most common cause of treatment failure. A thorough review of the treatment protocol including treatment of all contact animals, bathing after treatment, and sanitation of the environment should be performed before resistance to the medications is considered. Increasing dosages of medications, especially metronidazole, may result in irreversible side effects.

Because immune status of the host is a primary factor in treatment success, giardiasis in debilitated young animals is definitely much harder to eliminate than in a mature, healthy, well-nourished animal. When animals are in stressful situations such as confinement in animal shelters, pet shops, kennels, or catteries, the added stress will compound the pathologic process and complicate treatment attempts, and adversely affect success.

A Giardia vaccine for dogs and cats is available commercially (GiardiaVax, Fort Dodge Animal Health, Overland Park, Kansas) but has not proven to prevent infection in dogs or cats. The Giardia vaccine is entered in the “not recommended category” in The 2006 American Animal Hospital Association canine vaccine guidelines. Sanitation measures should include thorough cleaning of all surfaces with detergent and hot, soapy water. Chemical disinfectants have been recommended and evaluated, but there is no substitute for cleanliness. There has been recent interest in the use of ultraviolet light to eliminate Giardia cysts from water sources in kennel situations.

PUBLIC HEALTH

In his presentation to an Academy in 1915, Charles Atwood Kofoid raised the question of the zoonotic potential of Giardia spp and the possible contamination of human food
by the cyst-infected feces of vermin such as mice, rats, and cats. He started the discussion on the multiple biologic problems of host specificity and transformation by the environment that has not yet been resolved.66

Is Giardia spp zoonotic? This key question is often asked by practicing veterinarians but has not been answered with data. There is no fast and easy way to determine which assemblage the parasite found in a dog or cat stool belongs to, or if in fact the Giardia spp cysts seen actually pose a zoonotic threat to the owner of the animal, the veterinarian and his staff, or the researcher and the caretakers. There is an increasing number of water-related Giardia spp epidemics in human populations worldwide, and the significance of nonhuman hosts in these occurrences is still an unresolved issue.9,12,15,20,35,65 Overlapping of transmission cycles of humans and animals may result in zoonotic transfer. Once the taxonomy issues are resolved, veterinarians may have a better understanding of the risks of and links between the interaction between animal and humans that enables the zoonotic transfer of infection. Currently there is little epidemiologic evidence that strongly supports the importance of zoonotic transmission.9,20,67,68

The question that logically follows the previous one concerns treatment options. If Giardia spp cysts are found on fecal flotation or the Giardia spp cyst antigen is positive, should the animals and all of their housemates be treated? What if only one test is positive? What if the tests are positive and the stool is normal? Should these animals be treated with medications and the animals quarantined? Until all of these questions can be answered definitely, all animals with diarrhea that test positive for Giardia spp parasites on fecal flotation or the antigen test should be treated as well as all of their housemates, and bathed on the last day of treatment. If the animal does not have diarrhea, testing for Giardia spp antigen is not advised on a routine basis. These decisions can have major consequences if the puppy is one of many in a pet shop, in a group situation in an animal shelter, or in a cohort purpose bred for research.

TRITRICHOMONIASIS

Beginning in 1996, reports of large numbers of trichomonads in feline feces have been reported in the literature.10,69,70 Thanks to a few determined researchers and their laboratory teams, it is now known that some of these organisms are actually T foetus, the same organism that is known to cause early abortions and infertility in naturally bred cattle. Tritrichomoniasis is prevalent among cats in shelters and purebred show cats, and is significantly associated with the history of diarrhea within the cattery.71 T foetus is now the cause of an emerging infectious diarrheal disease of cats worldwide (United States, Britain, Norway, Australia, and Italy).

GENUS TRICHOMONAS KOFOID 1920

Parabasalids are anaerobic flagellates without mitochondria. Most of these organisms live as parasites in the alimentary or urogenital tract of vertebrates and invertebrates.2 Parabasalia are characteristically pear-shaped with one nucleus, and have a rodlike axostyle. Trichomonads do not have a cyst stage. Organisms in the genus Tritrichomonas are small flagellates (8–22 μm) with three free anterior flagella and a recurrent one, forming a well-developed undulating membrane. The recurrent flagellum is free posteriorly. There are 20 described species that live in the intestinal tract of nonhuman primates, rodents, swine, birds, reptiles, and amphibians.2 Tritrichomonas suis lives in the nasal cavity, stomach, and intestines of pigs, and is now considered to be identical to T foetus.72 T foetus has been recognized for many years as an important venereal transmitted pathogen of bovines that causes infertility and early abortion in naturally
bred cattle. More recently, *T foetus* has been recognized as an intestinal pathogen in cats, causing chronic large bowel diarrhea, and has also been found in the feline uterus and, rarely, in the intestinal tract of dogs. *Trichomonas vaginalis* commonly occurs as a venereal disease in people. Men are asymptomatic carriers and women suffer from vaginitis. Because it is difficult to determine the specific genera of trichomonads based on morphology alone, molecular techniques have been developed to identify trichomonads. Diagnosis is usually based on host site specificity and the number of anterior flagella.

**MORPHOLOGY AND LIFE CYCLE**

*T foetus* is a flagellated protozoan parasite that measures 6 to 11 by 3 to 4 mm. The organisms reproduce by binary fission within the intestine of the host. It is presumed that cats are infected by direct contact because there is no cyst stage. A recent report found *T foetus* in the uterus of a cat with pyometra. This animal did live in a house with other cats that were diagnosed with enteric tritrichomonads but the route of transmission is unknown.

It is now known that isolates of *T foetus* from cattle are infectious for cats, and that isolates of *T foetus* from cats are infectious for cows. Isolates of *T foetus* from cats do not seem to be as pathogenic for cattle as are cattle isolates.

**EPIDEMIOLOGY**

The origin and prevalence of *T foetus* in the feline colon is unknown. It has been reported in this species in the United States as well as other countries including Britain, Switzerland, Norway, and Australia. Three epidemiologic studies have been completed and reported in the United States and Britain over the last several years, which agree that the disease is usually found in densely housed young cats whereby fecal-oral transmission may readily occur.

The epidemiologic study that was conducted by Gookin and colleagues in 2004 also included data concerning *Giardia* spp infection. It was concluded that there was a high prevalence of *T foetus* infection in purebred domestic show cats. The clearest and most preventable risk factor for infection was a high density (low number of square feet of facility area per cat) of cats housed within a facility.

The British survey was conducted in 2007 with fecal samples from 111 United Kingdom cats with diarrhea. The assessment of *T foetus* infection was determined by PCR. Sixteen (14.4%) samples were found to be positive. In agreement with studies from the United States, infected cats were predominantly of pedigree breed and under 1 year old. The investigators noted that Siamese and Bengal cats specifically were overrepresented in this population.

A more recent study was conducted in pet cats in the United States. There were 173 feline fecal samples analyzed, with 17 (10%) both culture- and PCR-positive. No correlation was found between breed and sex. All positive samples were diarrheic.

From the results of these studies, one cannot help but wonder what effect stress has on the predisposition for disease with this organism.

**PATHOGENIC PROCESS**

There is little information regarding the pathologic process in naturally infected animals. However, a detailed report of the pathology of experimentally infected cats was presented in 2004. Forty-three sections of colon were evaluated from seven cats with chronic diarrhea and *T foetus* infection. Following experimentally induced
infection, *T. foetus* organisms colonize the feline ileum, cecum, and colon, resulting in diarrhea. The presence of organisms was associated with multiple changes within the lining of the intestine including infiltration of lymphocytes and neutrophils, loss of goblet cells, and other changes in the mucosa surface. Trichomonads were most commonly found in close proximity to the surface of the mucosa and less frequently compressed within the lumen of the colonic crypts. The investigators concluded that the number of factors mediating pathogenicity of the organism is limited. Identified mechanisms included the possibilities of alterations in the normal intestinal flora, adherence to the epithelium, and elaboration of cytokines and enzymes. These possibilities were extrapolated from the vast array of studies on the pathology of venereal *T. foetus* in cattle.

**CLINICAL SIGNS**

Cats infected with *T. foetus* present in good body condition and appetite, with chronic large bowel diarrhea, associated with blood, mucus, flatulence, tenesmus, and anal irritation. Owners report that the cats pass cow-pie like stools that are malodorous. Cases are usually diagnosed with trichomoniasis after it becomes apparent that the diarrhea is nonresponsive to routine therapies.

Trichomonads are usually commensal organisms causing no clinical signs in their host. Some cats with *T. foetus* infection are asymptomatic.

**DIAGNOSIS**

The diagnosis is made by direct observation of the flagellates in fresh or cultured feces. Flotation solutions will destroy trophozoites. The trophozoites are difficult to distinguish from those of *Giardia* spp and other nonpathogenic intestinal trichomonads such as *Pentatrichomonas hominis*. Trophozoites of *T. foetus* and *Giardia* spp are about the same size but they move differently. *Giardia* spp organisms have been said to have motility that resembles the fall of a leaf, whereas trichomonads move erratically. *T. foetus* cannot be reliably distinguished from the nonpathogenic *P. hominis*. Cultivation of feline feces in the commercially available transport and test system (InPouch TF-Feline, Biomed Diagnostics Inc, San Jose, California) has been recommended and is now considered to be the gold standard diagnostic test for *T. foetus* in felines. As with other causes of diarrhea, bacterial, viral, other parasites, and nutritional problems need to be ruled out before a diagnosis of trichomoniasis can be made.

**TREATMENT AND CONTROL**

There is no approved treatment for *T. foetus* in cats. Treatment of infected animals is difficult, and although many medications have been suggested and used alone or in combination, success is limited. The medications that have been evaluated include paromomycin, metronidazole, sulfamethoxine, fenbendazole, furazolidine, enrofloxacine, gentamycin, and cephalaxin. Diarrhea did improve during the treatment of the animals but none of the antimicrobials were effective in resolution of clinical signs. More recently, tinidazole was also found to be relatively ineffective. However, ronidazole was shown to be effective (30 mg/kg once a day for 10 days) in cats that were experimentally infected. Ronidazole is not readily available but may be obtained through compounding pharmacies in the United States. This drug must be used with caution because it will cause neurologic side effects. Other routine measures...
to relieve diarrheal symptoms such as dietary changes have also failed to help resolve symptoms.

Once again, sanitation of the environment and the animals in a cattery is critical, with animals shedding trichomonads into the environment and constant grooming activities of themselves and their kittens after defecation. These organisms do not survive for any length of time outside the host, but cats are fastidious and will definitely reingest these parasites readily.

PUBLIC HEALTH

The possibility of cat to human transmission has been alluded to, but has not been suspected or proved.81

SUMMARY

There is a vast amount of information available for *Giardia* spp in pets and humans, but the investigations of *T foetus* in cats is still new and information relatively sparse. The most obvious reason for this disparity is that *Giardia* is a historic and well-known human pathogen and *T foetus* is not. The one obvious common denominator in the incidence of these two parasites in pets is being housed in densely populated areas such as breeding kennels and catteries, and therefore most likely to be under stress.

These two protozoal parasites are different, yet interestingly similar in their biology and control. The host-parasite relationships of these two parasites are complex and may be affected by many factors. The zoonotic potential of diarrhea of dogs and cats, regardless of causative agent, is possible, and sanitation measures and treatment of all animals in the household cannot be overemphasized to clients.

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