

**FELINE ZOONOSES GUIDELINES FROM THE AMERICAN ASSOCIATION
OF FELINE PRACTITIONERS**



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INTRODUCTION

Zoonotic diseases are defined as being common to, shared by, or naturally transmitted between humans and other vertebrate animals.¹ Transmission of zoonotic agents from animals to people can potentially occur by direct contact with the animal, indirect contact with secretions or excretions from the animal, and contact with vehicles like water, food or fomites that were contaminated by the animal. For many agents, infection of the animal and human occurs from a shared vector or environmental exposure.

Most zoonotic agents can infect anyone regardless of their immune status. However, when immunosuppressed people are infected the clinical illness is often more severe. For example, primary *Toxoplasma gondii* infection of an immunocompetent person is usually inapparent whereas infection in an immunosuppressed person can cause life-threatening disease. Examples of immunosuppressed individuals include those with acquired immune deficiency syndrome (AIDS), those on immune suppressive drugs for immune-mediated disease, cancer, or organ transplantation, the fetus or other young people without fully developed immune systems, and older individuals with decremental deterioration of the immune system.

When immunodeficiency is detected or suspected in a family, it is often recommended that cat ownership be discontinued due to potential health risks.^{2,3} Because there are many infectious agents that infect both cats and humans, it is sometimes assumed that zoonotic diseases are commonly acquired from cat contact. In actuality, humans are unlikely to acquire infectious diseases from healthy, adult, parasite free,

indoor cats.^{2,4-7} In many instances of cat-associated zoonoses, people are more commonly infected than cats and so it is more likely one human being will become infected from contact with another human being or with the contaminated environment (Examples-*Cryptosporidium* spp., *Giardia* spp., *Salmonella* spp.). The Centers for Disease Control in the United States mention in their online publication, *Preventing Infections from Pets; A Guide for People with HIV Infection*, "You do not have to give up your pet".^a

Pet (including cat) ownership provides many health benefits, including increased happiness and decreased depression.^{5,8} All human or animal care givers should provide accurate information to clients concerning the risks and benefits of pet ownership so that an informed decision about acquiring and keeping pets can be made. However, information provided to clients often varies among health care providers. For example, in a recent study, responses of veterinarians and physicians varied dramatically when queried about zoonoses.⁹ Veterinarians were more likely than physicians to encounter or discuss zoonoses in their practices. Most physicians did not feel comfortable counseling clients about zoonoses and felt that veterinarians should provide information for patients and physicians. However, there was an almost total lack of communication about the issues between the veterinarians and the physicians.

There are multiple infectious agents capable of zoonotic transfer. The most common or important zoonoses associated with cats are listed by agent in Table 1.

^a www.cdc.gov/hiv/pubs/brochure/oi_pets.htm

Table 1. Feline zoonotic agents

Organism	Clinical presentation	Source of infection	Relative human risk from cats
Bacteria			
<i>Bacillus anthracis</i> ^a	Cat-subacute to chronic; carbuncular lesions of jowl and tongue, swelling of lips, head and throat Human-cutaneous ulcer with necrotic center, pneumonia, bloody diarrhea, hematemesis, meningitis	Cat-wounds, inhalation, ingestion Human-wounds, inhalation, ingestion	Extremely rare; not associated with cats to date
<i>Bartonella</i> spp.	Cat-subclinical, uveitis, fever, neurologic signs, gingivitis Human-lymphadenopathy, fever, malaise, bacillary angiomatosis, bacillary peliosis, et al.	Cat-fleas, bites or scratches ?? Human-bites, scratches, fleas	Common; mostly in areas with fleas; most important direct feline zoonosis
<i>Bordetella bronchiseptica</i>	Cat-subclinical, upper respiratory and rarely pneumonia Human-pneumonia in immunosuppressed	Cat-aerosolization Human-aerosolization	Extremely rare
<i>Borrelia burgdorferi</i>	Cat-subclinical Human-rash, polyarthritis, myocarditis, and neurologic disease	Cat- <i>Ixodes</i> spp. Human- <i>Ixodes</i> spp.	Rare; Northeast USA, north central USA and northern California; shared vector
<i>Campylobacter jejuni</i>	Cat-subclinical, gastroenteritis Human-subclinical, bacteremia, gastroenteritis, myalgia, arthralgia polyradiculoneuritis?	Cat-fecal contamination, poultry products, carnivorism Human-fecal contamination, poultry products	Rare; occasionally associated with cat contact
<i>Capnocytophaga canimorsus</i>	Cat-subclinical Human-bacteremia; keratitis	Cat-normal oral flora Human-Bite wounds, possibly scratches	Extremely rare; occasionally transmitted by cat bites
<i>Corynebacterium diphtheriae</i>	Cat-subclinical, membrane covering larynx, enlarged kidneys, paralysis Human-fever, pharyngitis, diphtheritic membrane, cervical lymphadenopathy,	Cat-Inhalation, contact with secretions Human-Inhalation, contact with secretions	Extremely rare; not associated with cats to date
<i>Francisella tularensis</i>	Cat-septicemia, pneumonia Human-ulceroglandular, glandular oculoglandular, pneumonic, or typhoidal (depending on route of infection)	Cat-blood sucking arthropods, ingestion of contaminated meat (rabbits) Human-blood sucking arthropods, contaminated meat or water, inhalation, cat bites	Rare; occasionally transmitted by cat bites
<i>Helicobacter</i> spp.	Cat-subclinical, rare vomiting Human-subclinical, gastric ulcer	Cat-fecal or oral contamination? Human-fecal or oral contamination?	Rare; while common in people; transmission from cats unlikely; reverse zoonosis likely
<i>Listeria monocytogenes</i>	Cat-subclinical intestinal carrier Human-abortion, stillbirth, septicemia neonatal death, meningoenzephalitis, uveitis, aseptic meningitis	Cat-contaminated soil or water Human-human carriers, contaminated soil, water, vegetation, or silage	Not associated with cat contact to date
<i>Leptospira</i> spp.	Cat-subclinical, fever, nephritis, hepatitis Human-fever, malaise, acute Inflammatory renal or hepatic disease, uveitis, CNS disease	Cat-direct contact with urine, ingestion of contaminated meat Human-direct contact with urine, ingestion of contaminated meat, bite wounds	Regional variation in human endemicity; not associated with cat contact to date
<i>Mycoplasma felis</i>	Cat-chronic draining tracts, polyarthritis Human-cellulitis, polyarthritis	Cat-normal flora Human-cat bite	Extremely rare; only 2 cat associated cases reported
<i>Mycobacterium</i> spp.	Cat-cutaneous lesions predominant Human-respiratory disease	Cat-ingestion, contact, inhalation Human-inhalation primary	Cats are not a source of human infection

Organism	Clinical presentation	Source of infection	Relative human risk from cats
<i>Salmonella</i> spp.	Cat-subclinical, mixed or large bowel diarrhea, bacteremia, abortion Human-Subclinical, gastroenteritis, bacteremia, abscesses	Cats-fecal contamination, poultry products, camivorisim, "songbird fever" Human-fecal contamination, poultry products	Common human infection; rare from cat contact
<i>Streptococcus</i> group A	Cat-subclinical, transient carrier (if at all) Human-strep throat, septicemia, skin infections, otitis, toxic shock, syndrome, glomerulonephritis, et etc.	Cats-aerosol Human-aerosol	Extremely rare (if ever) from cat contact; reverse zoonosis theoretically possible
<i>Yersinia enterocolitica</i>	Cat-subclinical Human-gastroenteritis	Cat-fecal contamination Human-fecal contamination	Extremely rare; not reported from cat contact
<i>Yersinia pestis</i>	Cat-bubonic, bacteremic, or pneumonic Human-bubonic, bacteremic, or pneumonic	Cat-Ingestion of bacteremic rodents; rodent fleas Human-rodent fleas, cat bites, aerosol, contact with exudates	Southwest region, occasionally associated with cat contact
<i>Yersinia pseudotuberculosis</i>	Cat-Anorexia, gastroenteritis, abdominal pain, icterus Human-lymphadenopathy, ileitis, arthralgia, septicemia, cutaneous swellings	Cat-fecal contamination Human-ingestion, inhalation	Extremely rare; not reported from cat contact
Cestodes			
<i>Dipylidium caninum</i>	Cat-subclinical Human-subclinical, pruritis ani, abdominal pain	Cat-ingestion of flea Human-ingestion of flea	Extremely rare; shared vector
<i>Echinococcus multilocularis</i>	Cat-subclinical Human-hepatic and pulmonary disease	Cat-ingestion of rodent Human-ingestion of eggs	Extremely rare; north central USA and Canada; not definitively linked to cat contact
Ectoparasites			
<i>Cheyletiella</i>	Cat-pruritic skin disease Human-pruritic skin disease	Cat-direct contact Human-direct contact	Occasional
<i>Sarcoptes scabiei</i>	Cat-pruritic skin disease Human-pruritic skin disease	Cat-direct contact Human-direct contact	Rare
Fungi			
Dermatophytes	Cat-subclinical, superficial dermatologic disease Human-superficial dermatologic disease	Cat-direct contact Human-direct contact	Common
<i>Sporothrix schenckii</i>	Cat-chronic draining cutaneous tracts Human-chronic draining cutaneous tracts feline exudate contact	Cat-wound contamination from soil Human-wound contamination from soil;	Rare; not geographically defined; cats have large numbers of organisms in exudates
Nematodes			
<i>Ancylostoma braziliense</i>	Cat-subclinical, hemorrhagic diarrhea, blood loss anemia Human-Pruritic skin disease (cutaneous larva migrans)	Cat-ingestion of transport host; trans-mammary, egg ingestion, skin penetration Human-Skin penetration by larva after > 3 days in environment	Rare; exposure from contaminated environment
<i>Ancylostoma tubaeforme</i>	As for <i>A. braziliense</i>	As for <i>A. braziliense</i>	As for <i>A. braziliense</i>

Organism	Clinical presentation	Source of infection	Relative human risk from cats
<i>Dirofilaria immitis</i>	Cat-subclinical; rarely cough, vomiting or sudden death Human-subclinical pulmonary mass	Cat-mosquito Human-mosquito	Extremely rare; shared vector
<i>Strongyloides stercoralis</i>	Cat-subclinical, hemorrhagic diarrhea Human-Pruritic skin disease; diarrhea; disseminated disease in immunosuppressed	Cat-fecal oral Human-skin penetration	Rare; exposure from contaminated environment
<i>Toxocara cati</i>	Cat-subclinical, vomiting, failure to thrive Human-subclinical; cough, ocular disease	Cat-ingestion of transport host, egg ingestion Human-ingestion of larvated eggs after 1-3 weeks in environment or ingestion of larvae and adults	Rare; exposure from contaminated environment
<i>Uncinaria stenocephala</i>	As for <i>A. braziliense</i>	As for <i>A. braziliense</i>	As for <i>A. braziliense</i>
Protozoans			
<i>Cryptosporidium parvum</i>	Cat-subclinical or small bowel diarrhea Human-subclinical or small bowel diarrhea	Cat-fecal contamination, carnivorous Human-fecal contamination	Rare; common in people but rarely directly linked to cats; potential reverse zoonosis
<i>Entamoeba histolytica</i>	Cat-hemorrhagic diarrhea Human-hemorrhagic diarrhea	Cat-ingestion of cysts Human-ingestion of cysts	Extremely rare; immediately infectious and common in people of some countries; but not definitely linked to cats; potential reverse zoonosis
<i>Giardia</i> spp.	Cat-subclinical or small bowel diarrhea Human-subclinical or small bowel diarrhea	Cat-fecal contamination, carnivorous Human-fecal contamination	Extremely rare; immediately infectious and common in people of some countries; but rarely directly linked to cats; potential reverse zoonosis
<i>Toxoplasma gondii</i>	Cat-subclinical, fever, uveitis, muscle pain, hepatic inflammation, pancreatitis Human-subclinical, lymphadenopathy, abortion, stillbirth, encephalitis	Cat-ingestion of transport host, ingestion of oocysts after 1-5 days of sporulation, transplacental Human-ingestion of undercooked meat, transplacental ingestion of oocysts after 1-5 days of sporulation,	Rare; common in people but not usually associated with individual cats because of short term oocyst shedding and sporulation period
Rickettsiae and chlamydiae			
<i>Chlamydia felis</i>	Cat-conjunctivitis, mild upper respiratory Human-conjunctivitis, pneumonia, endocarditis, glomerulonephritis	Cat-direct contact, aerosol Human-direct contact, aerosol?	Extremely rare; direct contact with cats occasionally
<i>Coxiella burnetii</i>	Cat-subclinical, abortion, or stillbirth Human-fever, pneumonitis, myalgia, lymphadenopathy, arthritis, hepatitis, endocarditis	Cat-blood sucking arthropods, ingestion of contaminated tissues Human-blood sucking arthropods, aerosol from infected tissues	Extremely rare; distribution unknown; multiple cat point source outbreaks
<i>Rickettsia felis</i>	Cat-subclinical Human-fever, lymphadenopathy	Cat-fleas Human-fleas	Rare; shared vector
Viruses			
Cowpox	Cat-Circumscribed, ulcerative and pruritic skin lesions and mild conjunctivitis Human-Papulovesicular skin disease	Cat-direct contact Human-direct contact	Extremely rare
Rabies	Cat-progressive CNS disease Human-progressive CNS disease	Cat-animal bites, ingestion, inhalation Human-animal bites, ingestion, inhalation	Regional; direct transmission from cats can occur

³For more information concerning this organism, please see the AAFP Newsletter, December, 2001.

The following is a brief description of the most common cat-associated illnesses that are encountered in small animal practice grouped by route of transmission. Recommendations to minimize dangers associated with cat ownership and to those providing cat health care are included by section and the majority are summarized in Tables 2 and 3. Many of

the recommendations were adapted from those utilized by the Centers for Disease Control.^{a,b}

^awww.cdc.gov/hiv/pubs/brochure/oi_pets.htm

^bwww.cdc.gov/ncidod/diseases/index.htm

Table 2. General guidelines for veterinarians to aid in the management of zoonotic diseases of cats

<ul style="list-style-type: none"> • Familiarize yourself and your staff with zoonotic issues. • Take an active role in discussing the health risks and benefits of cat ownership with clients so that logical decisions concerning ownership and management of individual cats can be made. • Make it clear to your clients that you and your staff understand conditions associated with human immune deficiency, are discreet, and are willing to help. • Provide information concerning veterinary or public health aspects of zoonoses to cat owners, but do not diagnose or treat diseases in people or make recommendations about these issues. • Refer clinically ill cat owners to a physician for additional information and treatment. • Since veterinarians and physicians have different experiences concerning zoonoses, veterinarians should volunteer to speak to the cat owner's physician to clarify zoonotic issues when indicated. • When public health related advice is offered, it should be documented in the medical record. • When reportable zoonotic diseases are diagnosed, appropriate public health officials should be contacted. • Vaccinate all cats for rabies. • Routinely administer anthelmintics to kittens as early as 3, 5, 7, and 9 weeks of age to aid in control of hookworms and roundworms. • In <i>D. immitis</i> endemic areas, monthly heartworm preventatives that control hookworms and roundworms should be used. • Test all cats for gastrointestinal parasites at least once yearly. • Offer diagnostic plans to assess for presence of organisms with zoonotic potential, particularly if the cat is clinically ill. • Consider the following minimal diagnostic plan for cats with diarrhea of > 1-2 days duration and for all cats in the home of immunosuppressed people: <ul style="list-style-type: none"> ○ Zinc sulfate centrifugation and microscopic examination for oocysts, cysts and eggs. ○ Fecal wet mount to evaluate for trophozoites of <i>Giardia</i> and <i>Tritrichomonas</i>. ○ Rectal cytology to observe for white blood cells and spirochetes consistent with <i>Campylobacter</i> spp.. ○ <i>Cryptosporidium</i> spp. screening by IFA, antigen ELISA or acid fast stain. ○ Fecal culture for <i>Salmonella</i> spp. and <i>Campylobacter</i> spp.. • Periodically (monthly in <i>Echinococcus multilocularis</i> endemic areas) administer taeniocides, particularly in cats allowed outdoors. • Maintain flea and tick control at all times. • Do not allow clients to restrain cats and do not attempt to pull cats from their carriers. • Train staff members in how to avoid bites and scratches. • Provide rabies vaccination for all staff members that handle animals. • Reevaluate rabies antibody titers of staff members that handle animals every two years. • Follow biosecurity measures for small animal hospitals.
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Table 3. General guidelines for cat owners to avoid zoonotic transfer of disease

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- If a new cat is to be adopted, the cat least likely to be a zoonotic risk is a clinically normal, arthropod-free, adult animal from a private family.
 - Once the cat to be adopted is identified, it should be quarantined from any immunocompromised person until a thorough physical examination and zoonoses risk assessment is performed by a veterinarian.
 - Immediate veterinary care should be sought for all unhealthy cats.
 - Veterinary care should be sought at least once or twice yearly for a physical examination, fecal examination, deworming recommendations, and vaccine needs assessment.
 - Get cats for cats vaccinated for rabies at appropriate intervals.
 - Avoid handling unhealthy cats, particularly those with gastrointestinal, respiratory, skin, neurological, or reproductive disease.
 - Do not handle cats with which you are unfamiliar.
 - Do not allow cats to drink from the toilet.
 - Wash hands after handling cats.
 - Remove fecal material from the home environment daily.
 - If possible, do not have immunocompromised people clean the litterbox. If immunocompromised people must clean the litter box, they should wear gloves and wash hands thoroughly when finished.
 - Use litterbox liners and periodically clean the litterbox with scalding water and detergent.
 - Wear gloves when gardening and wash hands thoroughly when finished.
 - Cover children's sandboxes to lessen fecal contamination by outdoor cats.
 - Only feed cats cooked or commercially processed food.
 - Control potential transport hosts like flies and cockroaches that may bring zoonotic agents into the home.
 - Filter or boil water from sources in the environment.
 - Housing cats indoors may lessen their exposure to other animals that may carry zoonotic agents, to excrement of other animals, and to fleas and ticks.
 - Seek veterinary advice concerning flea and tick control.
 - Do not share food utensils with your cat.
 - Avoid being licked on the face by your cat.
 - Have your cat's claws clipped frequently to lessen the risk of skin penetration; nail caps or declawing could be considered in some cases.
 - Consider behavior modification for cats prone to biting or scratching.
 - Do not tease cats or attempt to pull them from their carriers.
 - If bitten or scratched by a cat, seek medical attention.
 - Cook meat for human consumption to 80 C for 15 minutes minimum (medium-well).
 - Wear gloves when handling meat and wash hands thoroughly with soap and water when finished.
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ENTERIC ZOONOTIC AGENTS

There are multiple enteric agents capable of infecting human beings and cats (Table 1). Some of these infections are common in cats.¹⁰⁻¹³ For example, enteric agents with zoonotic potential were detected in feces of 13.1% of cats tested in north-central Colorado¹² and in 40.7% of the kittens tested in central New York State.¹³ Some infectious agents like *Giardia* spp., *Cryptosporidium* spp., *Salmonella* spp., and *Campylobacter* spp. are immediately infectious and could be acquired from contact with individual cats. Other infectious agents like *Ancylostoma braziliense*, *Toxocara cati*, and *Toxoplasma gondii* require a period of time outside the host to become infectious. It is more likely for people to develop infection by feline enteric pathogens from contact with the environment than by direct cat contact. General guidelines for prevention of enteric zoonoses are included in Tables 2 and 3. The morphologic characteristics of enteric parasites are listed in Table 4.

Cestodes. Cats and people can be infected with adult *Dipylidium caninum*, acquired by the ingestion of fleas which harbor cysticercoids. *Dipylidium caninum*, while rare in people, is usually seen in children. It can cause abdominal pain, diarrhea and pruritus ani or be relatively asymptomatic and recognized only because proglottids are passed per rectum. Cats can bring infected fleas into the human environment. This organism can also be classified with the shared vector zoonoses.

Cats, dogs, and foxes are definitive hosts of *Echinococcus multilocularis*. These animals become infected by ingesting intermediate hosts (rodents). Definitive hosts of this cestode are subclinically infected but pass infective eggs (Table 4) into the environment.^{14,15}

Table 4. Morphologic characteristics of enteric zoonotic parasites of cats

Organism	Life stage and description
Nematodes	
<i>Toxocara cati</i>	Egg; 65-75 μ
<i>Ancylostoma cati</i>	Egg; 55-65 μ x 34-45 μ
<i>Ancylostoma braziliense</i>	Egg; 55-76 μ x 35-45 μ
<i>Uncinaria stenocephalia</i>	Egg; 60-75 μ x 33-50 μ
<i>Strongyloides stercoralis</i>	Egg; 55 μ x 30 μ ; larvated Larvae; rhabditiform 1 st stage larva Egg; 30-33 μ x 45-55 μ ; larvated
Cestodes	
<i>Dipylidium caninum</i>	Proglottid; double pored Egg packet; each egg is 25-40 μ x 30-45 μ
<i>Echinococcus multilocularis</i>	Egg; 37 μ x 32 μ
Coccidians	
<i>Toxoplasma gondii</i>	Oocyst; 10 μ X 12 μ
<i>Cryptosporidium</i> spp.	Oocyst; 4-6 μ x 4-7 μ
Flagellates	
<i>Giardia</i> spp.	Cyst; 7-10 μ x 8-12 μ Trophozoite; 10-12 μ x 15-18 μ

Following human ingestion of eggs, *E. multilocularis* oncospheres enter the portal circulation and are distributed to the liver and other tissues. Larval or metacestode forms then develop in infected tissues as tumor-like masses. The liver, lung, and brain are most commonly infected. The larval tumors are multilocular and grow rapidly (alveolar echinococcosis). A combination of surgical excision and anthelmintic treatment is used to treat the syndrome in people but the disease often has a poor prognosis. *Echinococcus multilocularis* is most common in the northern and central parts of North America but seems to be spreading with the fox population (the most common definitive host). It is also present in parts of Europe and Asia. It is rare in human beings in North America but, to reduce the incidence further, cats in endemic areas should not be allowed to hunt. Taeniocides should be administered monthly to cats that live in endemic areas and are allowed to hunt (Table 5).

Table 5. Drugs used in the management of feline zoonotic diseases

Drug	Dose and Route of Administration	Organisms
Amoxicillin	10-22 mg/kg, PO, q12hr	<i>Streptococcus</i> group A
Amoxicillin clavulanate	15 mg/kg, PO, q12hr	<i>Bartonella</i> spp., <i>Bordetella bronchiseptica</i> <i>Pasteurella multocida</i>
Ampicillin	22 mg/kg, IV, q8hr	<i>Leptospira</i> spp.
Azithromycin	7.5-10 mg/kg, PO, q12-72hr	<i>Cryptosporidium</i> spp., <i>Bartonella</i> spp.
Clarithromycin	7.5 mg/kg, PO, q12-24hr	<i>Helicobacter</i> spp.
Clindamycin	10-12 mg/kg, PO, q12hr	<i>Toxoplasma gondii</i>
Doxycycline	5-10 mg/kg, PO, q12-24hr	<i>B. bronchiseptica</i> , <i>Bartonella</i> spp. <i>Chlamydomphila felis</i> , <i>Ehrlichia</i> spp. <i>Mycoplasma felis</i>
Enrofloxacin	5-10 mg/kg, PO, q24hr	<i>Bartonella</i> spp., <i>Campylobacter</i> spp. <i>Mycoplasma felis</i> , <i>Yersinia pestis</i>
Enrofloxacin	5-10 mg/kg, IM, IV, q24hr	<i>Salmonella</i> spp. bacteremia
Erythromycin	10 mg/kg, PO, q8hr	<i>Bartonella</i> spp., <i>Campylobacter</i> spp.
Fenbendazole	50 mg/kg, PO, q24hr	<i>Ancylostoma</i> spp., <i>Giardia</i> , <i>Strongyloides stercoralis</i> , <i>Toxocara cati</i>
Fipronil	7.5-15 mg/kg topical 0.25% spray and 10% spot-on	Ticks, fleas
Fipronil/methoprene	7.5-15 mg/kg, topical spot-on	Ticks, fleas
Fluconazole	50 mg, PO, q12-24hr	Dermatophytes, <i>Sporothrix schenckii</i>
Griseofulvin (microsize)	25 mg/kg, PO, q12hr	Dermatophytes
Griseofulvin (ultramicrosize)	5-10 mg/kg, PO, q24hr	Dermatophytes
Imidocloprid	10-20 mg/kg, topical spot-on	Fleas
Itraconazole	5 mg/kg, PO, q12hr for 4 days and then 5 mg/kg, PO, q24hr	Dermatophytes, <i>Sporothrix schenckii</i>
Ivermectin	24 µg/kg, PO, monthly 200-300 µg/kg, PO, weekly	<i>Dirofilaria immitis</i> , hookworms <i>Cheyletiella</i> , <i>Sarcoptes scabiei</i>
Lufenuron	80-100 mg/kg, PO, q2weeks 30 mg/kg, PO, q30days 10 mg/kg, SQ, q180days	Dermatophytes Fleas Fleas
Lime-sulfur	Dip every 5-7 days	Dermatophytes
Metronidazole	25 mg/kg, PO, q12hr	<i>Entamoeba histolytica</i> , <i>Giardia</i>
Miconazole and 2% chlorhexidine	Dip every 3-4 days	Dermatophytes
Milbemycin	0.5 – 0.99 mg/kg, PO, monthly	<i>Dirofilaria immitis</i> , hookworms Roundworms
Paromomycin	150 mg/kg, PO, q12hr X 5 days	<i>Cryptosporidium</i> spp.
Praziquantel	5 mg/kg, PO, SC or IM, once	<i>Dipylidium caninum</i> , <i>Echinococcus multilocularis</i>
Pyrantel	20 mg/kg, PO, once; repeat in 3 weeks	<i>Ancylostoma</i> spp., <i>Strongyloides stercoralis</i> <i>Toxocara cati</i>
Pyrantel plus praziquantel	72.6 mg pyrantel and 18.2 mg praziquantel, 1 tab/cat, PO	Hookworms, roundworms, and cestodes
Selamectin	6 mg/kg, topically once a month	Hookworms, roundworms, fleas
Terbinafine	20 mg/kg, PO, q24-48hr	Dermatophytes
Tylosin	10-15 mg/kg, PO, q12hr	<i>Cryptosporidium</i> spp.

Nematodes. Cats and people can be infected with *Toxocara cati*. Visceral (including neural) larva migrans (VLM) and ocular larva migrans (OLM) are the syndromes associated with human toxocariasis. Most cases of VLM and OLM are thought to be caused by *Toxocara canis* infection but the same syndromes can occur following infection with *T. cati*.^{14,16,17} Human infection with *Toxascaris leonina* has not been reported. Visceral larva migrans is most common in children < 6 years of age and ocular larva migrans is most common in older children and young adults. Infected cats pass eggs into the human environment. In warm weather, after 3 to 4 weeks, the eggs larvate and then are infectious. People are infected by ingestion of larvated eggs that release infective larvae in the gastrointestinal tract. The larvae penetrate the mucosa of the small intestine and migrate to the liver, lungs, and other organs (visceral larva migrans). The inflammatory reaction against the larvae can result in clinical signs of disease. Manifestations include eosinophilia, abdominal pain, anorexia, nausea, vomiting, fever, cough, hepatomegaly, myocarditis, and encephalitis. Larvae (usually only one) that migrate to the eye can cause severe intraocular inflammation.

Adult *T. cati* have been passed in the vomitus or per rectum in some infected children. The affected children generally have no evidence of VLM and probably ingested advanced larval stages or adult worms passed by infected cats.

Toxocara eggs are environmentally resistant, so when an area is contaminated, the potential for infection will persist for months or years. In the United States, the seroprevalence of antibodies against *Toxocara* is 2.8% in the general human population and from 4.6% to 7.3% in children 1 to 11 years of age.¹⁴ Thus, exposure to infective roundworms is still

common. Cats can be the definitive host for *Ancylostoma braziliense*, *A. tubaeforme*, *Uncinaria stenocephala*, and *Strongyloides stercoralis*. Eggs are passed into the environment where they larvate after several days in warm, humid conditions. Infective larvae penetrate human skin by direct contact. Pruritic, serpiginous, erythematous tracts occur as the larvae migrate in the epidermis (cutaneous larva migrans). While *A. caninum* has been linked with eosinophilic enteritis in people, this syndrome has not been described with hookworms that infect cats.¹⁸

Risk of hookworm and roundworm infections are lessened by reducing exposure to animal excrement and routine administration of anthelmintics to cats (Tables 2 and 3). Direct skin contact with moist, potentially infected soil should be avoided. The children's sandbox should be covered when not in use and fecal material should be removed immediately. Geophagia and ingestion of untreated surface water should be discouraged. In areas where nematodes are common, 3 doses of an anthelmintic can be administered every 2 weeks to kittens beginning as early as 3 weeks of age to lessen potential clinical disease and environmental contamination with eggs (Table 5).^c The queens should be treated concurrently because they often have patent infections while nursing. Fecal flotation should also be done once or twice yearly on feces from all cats and more frequently for cats that go outdoors. Ivermectin-containing heartworm preventatives aid in the control of hookworms and both selamectin and milbemycin heartworm preventatives aid in the control of hookworms and roundworms.¹⁹ However, fecal flotation is still indicated at least yearly for cats on heartworm preventatives since there are other important parasites that the drugs do not control.

^cwww.cdc.gov/ncidod/dpd/parasites/ascaris/prevention.htm

Protozoans. Cats and human beings can be infected with *Entamoeba histolytica*, *Cryptosporidium parvum*, *C. felis*, *Toxoplasma gondii* and *Giardia* spp. (Table 1). *Entamoeba histolytica* infection is only rarely described in cats and so is not likely to be a significant zoonosis.²⁰ *Balantidium coli* has not been isolated from cats.²¹ While trichomoniasis of cats may be common, *Tritrichomonas foetus*.^{22,23} transmission from a cat to a person has never been documented.

Cryptosporidiosis. *Cryptosporidium parvum* is a coccidian that commonly infects people and can result in severe gastrointestinal disease. The organism frequently causes diarrhea outbreaks in daycare centers,²⁴ approximately 300,000 people in Milwaukee developed cryptosporidiosis when a water purification system malfunctioned,²⁵ and nearly 10-20% of AIDS patients are infected with *C. parvum* at some time during their lives.²⁶ Many individuals require hospitalization for intravenous fluid therapy. Infection of immunosuppressed individuals may be life-threatening. People coinfecting with AIDS may never be cured.

Cryptosporidium spp. oocysts or antigens have been documented in feces of many domestic cats with or without diarrhea in the United States, Japan, Scotland, Australia, and Spain.^{12,13,27-36} Presence of serum antibodies can be used to estimate numbers of individuals exposed to *C. parvum*. An enzyme-linked immunosorbent assay for detection of *C. parvum* IgG was developed and applied to serum of cats.³⁵ Using this assay, the seroprevalences of *C. parvum* antibodies in serum of cats in Colorado and the United States are 15.3% and 8.3%, respectively.^{35,37} Oocysts or antigens of *C. parvum* were detected in feces of 5.4% of cats tested in north-central Colorado¹² and in 3.8% of the kittens tested in central New York State.¹³

While the source of most *C. parvum* infections in people is unknown; contaminated water is one likely source.³⁸ Cryptosporidiosis has been documented in people and cats in the same environment suggesting the possibility for interspecies transmission or acquisition from a common source.³⁹⁻⁴² Oocysts are passed sporulated and infectious so there is potential for direct zoonotic transfer.

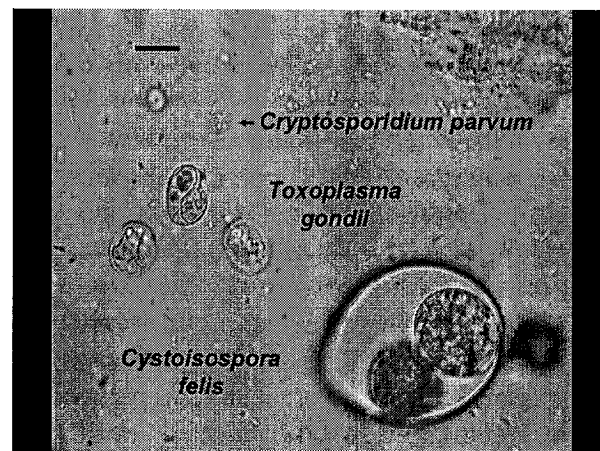


Figure 1. *Cystoisospora felis* oocysts, sporulated *Toxoplasma gondii* oocysts, and *Cryptosporidium parvum* oocysts in a feline fecal sample. Bar = 10 μ m.

There have been limited cross-infection studies performed with *C. parvum* isolates from cats or human beings. A feline isolate failed to cross-infect mice, rats, guinea pigs, or dogs,⁴³ but another isolate from a cat cross-infected lambs.⁴⁴ *Cryptosporidium hominis*, a human parasite, does not infect cats.⁴⁵ An alternative to cross-infection studies is comparison of isolates genetically. A feline genotype (*C. felis*) that varies considerably from human and cattle genotypes has been identified.⁴⁶ *C. felis* has been documented in infected human beings and cows suggesting the genotype can infect other mammals.⁴⁷⁻⁵⁰ However, in a study of HIV-infected people with cryptosporidiosis, there was no statistical association with cat ownership, suggesting that cat contact is an uncommon way to acquire cryptosporidiosis.⁵¹ While cats are commonly infected with

Cryptosporidium spp.^{12,13} and can shed oocysts for extended periods of time,⁴³ only small numbers of oocysts per gram of feces are shed.²⁸ This may decrease the risk of transmission from cats to people.

It is impossible to determine zoonotic strains of *Cryptosporidium* by microscopic examination. Thus, it seems prudent to assume feces from all cats infected with *Cryptosporidium* spp. are a potential human health risk. Techniques for the detection of *Cryptosporidium* spp. should be included in the diagnostic evaluation of all cats with diarrhea and all cats in the homes of immunosuppressed individuals. Only a few *Cryptosporidium* spp. oocysts are generally shed by infected cats and they are extremely small (approximately 5 microns), so acid-fast or immunofluorescent antibody staining of feces will aid in their identification.⁵² Fecal antigen ELISAs are also available; at this time it is unknown whether immunofluorescent antibody assays (IFA) or fecal antigen ELISAs developed for the detection of *C. parvum* will consistently detect *C. felis*. Recently, a polymerase chain reaction assay has been used to amplify *Cryptosporidium* DNA from feline feces and was more sensitive than an IFA assay.⁵³

It is unknown where cats acquire cryptosporidiosis, but because rodents⁵⁴ are commonly infected, acquisition may be acquired by carnivorousness. It is possible that administration of paromomycin, tylosin, or azithromycin (Table 5) can lessen oocyst shedding from infected cats but data is limited and it is unknown whether treated cats are cured.^{33,34} Paromomycin should not be prescribed to cats with bloody diarrhea because absorption is enhanced which can result in acute renal failure in some cats.⁵⁵ Reinfection is also likely (see Followup testing recommendations, page 17). *Cryptosporidium* spp. can be removed from

contaminated surface water by boiling or filtration. Hands should be washed after handling fecally contaminated material, e.g., soil, even if gloves were worn (Table 2).

Giardiasis. *Giardia* is a flagellate with worldwide distribution that causes significant gastrointestinal disease in dogs, cats, and people. The organism is thought to have a wide host range. Prevalence in cats varies by the region; 3.9% and 1.9% of client-owned cats with or without diarrhea, respectively, were infected in a study performed in north central Colorado.¹² In a study of kittens < 1 yr of age in central New York State, the organism was identified in 6.1% and 8.1% of client-owned and shelter cats, respectively.¹³

The organism is immediately infectious when passed as cysts in stool, so there is potential for direct zoonotic transfer. There have been varying results concerning cross-infection potential of *Giardia* spp.. In one study, *Giardia* spp. from human beings were inoculated into cats; the cats were relatively resistant to infection.⁵⁶ In contrast, evaluation of human and feline *Giardia* spp. isolates by isoenzyme electrophoresis suggests that cats could serve as a reservoir for human infections.⁵⁷

Recent genetic analysis has revealed 2 major genotypes in people. Assemblage A has been found in infected human beings and many other mammals including dogs and cats.⁵⁸ Assemblage B has been found in infected human beings and dogs, but not cats.⁵⁸ It appears that there is also a specific genotype of *Giardia* that infects cats, but not people.⁵⁸

To date, there has not been a documented case of human giardiasis acquired from a cat. However, since potentially zoonotic strains have been detected in cats and it is impossible to determine zoonotic strains of *Giardia* spp. by microscopic examination, it seems prudent

to assume feces from all cats infected with *Giardia* spp. are a potential human health risk.

Giardia is a common enteric pathogen and can be detected in feces of cats with and without diarrhea.

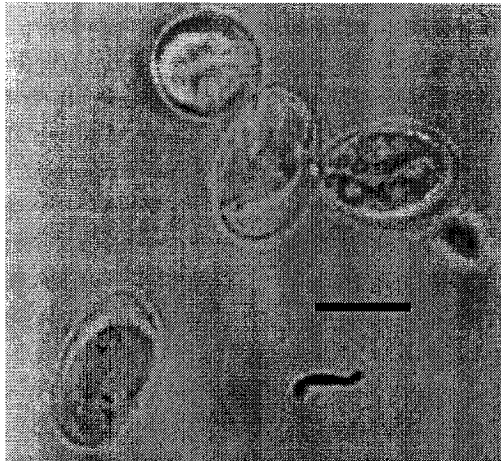


Figure 2. *Giardia* cysts. Bar = 10 μ m.

Fecal examination should be performed on all cats at least yearly and treatment with anti-*Giardia* drugs (Table 5) should be administered if indicated. Zinc sulfate centrifugation is considered the optimal fecal flotation technique by most parasitologists (Table 6). If fresh stool is available from cats with diarrhea, examination of a wet mount to detect the motile trophozoites may improve sensitivity and can also be used to detect *Tritrichomonas foetus* infection. While monoclonal antibody based immunofluorescent antibody tests and fecal antigen tests are available, limited studies of sensitivity and specificity for feline *Giardia* isolates have been performed. These techniques should be used in addition to, not in lieu of, fecal flotation that can also reveal other parasites.

Table 6. Zinc sulfate centrifugation

1.	Place 1 gram fecal material in a 15 ml conical centrifuge tube
2.	Add 8 drops of Lugol iodine and mix well
3.	Add 7 to 8 ml of ZnSO ₄ (1.18 specific gravity)* solution and mix well
4.	Add ZnSO ₄ solution until there is a slight positive meniscus
5.	Cover the top of the tube with a coverslip
6.	Centrifuge at 1500-2000 rpm for 5 minutes
7.	Remove the coverslip and place on a clean microscope slide for microscopic examination
8.	Examine the entire area under the coverslip for the presence of eggs, cysts, oocysts, or larvae at 100X.

*Add 330 g ZnSO₄ to 670 ml of distilled water
Fisher Scientific, Hanover Park, Illinois

A *Giardia* vaccine was recently licensed but is not currently recommended for routine prophylactic use in cats.⁵⁹ Vaccination against *Giardia* could be considered in cats with recurrent infection and is being evaluated as a therapeutic agent.⁶⁰ However, administration of the vaccine three times to cats with giardiasis was ineffective as a treatment in one experimental study.⁶¹ Prevention of zoonotic giardiasis includes boiling or filtering surface water for drinking. Hands should be washed after handling fecally contaminated material, even if gloves were worn (Table 2). It is unknown whether treated cats are cured and it is likely that if a treated cat is exposed again it will be reinfected (see Followup testing recommendations, page 18).

Toxoplasmosis. *Toxoplasma gondii* is one of the most common of the feline zoonoses; approximately 30-40% of human adults in the world are seropositive, suggesting previous or current infection.⁶² People are usually infected congenitally, after ingestion of sporulated oocysts, or ingestion of tissue cysts in undercooked meat. Clinical disease is generally mild following primary infection in immunocompetent people. Self-limiting fever, malaise, and lymphadenomegaly are the most common clinical abnormalities and the majority of people never realize when their acute *T. gondii* infection occurs. The

disease can be confused with infectious mononucleosis. Clinical disease is usually more severe in immunodeficient individuals, including people with AIDS and people treated with immunosuppressive agents (e.g. cancer chemotherapy). *Toxoplasma gondii* is a common opportunistic CNS infection of people with AIDS; as T-helper cell counts decline, toxoplasmic encephalitis can result from activation of bradyzoites in tissue cysts. Stillbirth, CNS disease, and ocular disease are common clinical manifestations in the fetus if a woman contracts an acute *T. gondii* infection during pregnancy.⁶³

Cats (wild and domestic) are the only known definitive hosts for *T. gondii*. They pass unsporulated (non-infectious) oocysts into the environment.⁶⁴ Once passed into the environment, sporulation occurs in 1-5 days; sporulated (infectious) oocysts survive for months to years. While ingestion of tissue cysts in undercooked meat is a common way for people to acquire *T. gondii* infection, it is likely that some people acquire toxoplasmosis from ingestion of sporulated oocysts in contaminated soil or drinking water. Clinical toxoplasmosis developed in a group of people following a common exposure in a riding stable,⁶⁵ in a group of soldiers drinking contaminated water in Panama,⁶⁶ and from an oocyst-contaminated municipal water supply.⁶⁷

Cats only shed oocysts for days (after tissue cyst ingestion) to several weeks (after sporulated oocyst ingestion). Thus, an individual cat will be passing oocysts into the human environment for only a small fraction of its entire life span. Because oocysts are passed unsporulated and non-infectious, contact with fresh feline feces (< 1 day old) is not a risk. Most cats are fastidious and do not leave feces on their fur long enough for sporulation to occur. For example, bioassay failed to detect oocysts on the fur of cats

seven days after they were shedding millions of oocysts in feces.⁶⁸ These findings suggest that touching individual cats is an unlikely way to acquire toxoplasmosis; this hypothesis is supported by epidemiologic studies as well. In general, veterinary health care providers are no more likely than the general population to be seropositive for *T. gondii* infection. In one case control study of pregnant women, there was no association between primary toxoplasmosis and having a cat or kitten at home, litterbox cleaning, or owning a cat that hunts.⁶⁹ People with HIV infection who owned cats were no more likely to acquire toxoplasmosis during their illness than people with HIV infection who did not have cat contact.⁷⁰ When CNS toxoplasmosis occurs concurrently with AIDS, it is thought to be reactivation of chronic infection rather than a primary infection in most cases.

Following primary inoculation of cats, it is difficult to induce repeat oocyst shedding. Superinfection with *Isospora* led to oocyst shedding in some *T. gondii* infected cats.⁶⁴ Prednisolone administered at 10-80 mg/kg, PO or methylprednisolone administered at 10-80 mg/kg, IM will induce repeat oocyst shedding in some cats with toxoplasmosis but the level and duration of shedding is much lower and shorter than with primary infection. However, these doses are greater than those used in clinical practice. Administration of methylprednisolone acetate administered at 5 mg/kg, weekly for 4 to 6 weeks to cats infected with *T. gondii* for 14 weeks or 14 months failed to induce oocyst shedding.⁶⁴ Cats infected with *T. gondii* were given feline immunodeficiency virus (FIV) followed by feline leukemia virus (FeLV) and developed immunodeficiency associated syndromes (Lappin MR; unpublished data), but repeat *T. gondii* oocyst shedding could not be demonstrated. Cats with FIV or FeLV infections have been inoculated with *T. gondii*; oocyst shedding periods and number

of oocysts shed were similar to those for cats without FIV or FeLV infections.^{64,71} It has been shown that gut immunity to *T. gondii* in cats is not permanent; 4 of 9 cats inoculated 6 years after primary inoculation shed few to 1.25×10^6 oocysts for 6 to 10 days even though each had high serum antibody titers.⁶⁸ However, *T. gondii* infected cats with and without FIV infection failed to repeat oocyst shedding when reinfected with *T. gondii* 16 months after primary inoculation.⁷¹ Thus, cats that are exposed to *T. gondii* repeatedly probably do not shed large numbers of oocysts after the first infection and are of minimal public health risk.

There is no serologic assay that accurately indicates when a cat shed *T. gondii* oocysts in the past. Most cats that are shedding oocysts are seronegative;⁷² and most seropositive cats (IgM or IgG) have completed the oocyst shedding period, are unlikely to repeat shedding and are unlikely to be a source of human infection. Most seronegative cats would shed the organism if infected, so they should not be fed raw meat or allowed to hunt. Because people are not commonly infected with *T. gondii* from contact with individual cats and because serologic test results cannot accurately predict the oocyst shedding status of cats, testing healthy cats for *T. gondii* antibodies has little public health application and is not recommended.^{5,72} While fecal examination can determine if an individual cat is actively shedding oocysts, it is not very useful for public health purposes because the oocyst shedding period is so short. Finding oocysts has limited clinical relevance because most cats are subclinically infected at that time. If people are concerned that they may have toxoplasmosis, they should see their doctor for serologic testing.

The primary way to avoid contracting *T. gondii* infection is to avoid ingestion of the organism in undercooked meat. Meats

(particularly pork in the United States) should be cooked to medium-well (160°F; 80°C) to inactivate tissue cysts. Gloves should be worn when handling raw meats (including field dressing) and hands should be cleansed thoroughly afterwards. Freezing meat at -12°C for several days will kill most tissue cysts. Ingestion of raw goat's milk can also result in human toxoplasmosis.

Surface water collected directly from the environment should be boiled or filtered prior to drinking (Table 2). Gloves should be worn when contacting fecally contaminated material, e.g., soil, and hands should be washed afterwards. Produce from the garden should be washed carefully prior to ingestion. The children's sandbox should be covered when not in use. The litterbox should be cleaned daily; oocysts require 1-5 days to sporulate. Immunosuppressed or pregnant clients should not clean the litterbox. Sporulated oocysts are extremely resistant to most disinfectants; cleaning with scalding water or steam is most effective but can lead to burns. Use of disposable litter pans may be worth considering.

Oocysts measuring 10 X 12 μ in a cat fecal sample could be *T. gondii*. *Hammondia hammondi* and *Besnoitia darlingi* are morphologically similar coccidians passed by cats but are not human pathogens.⁶⁴ Differentiation of these parasites from *T. gondii* can be made by laboratory animal inoculation. Alternately, if the infected cat develops *T. gondii* serum antibodies it was likely infected with *T. gondii*. If a cat is found to be shedding oocysts morphologically consistent with *T. gondii*, the feces should be disposed of daily until the oocyst shedding period is complete; administration of clindamycin, sulfonamides, or pyrimethamine can reduce levels of oocyst shedding (Table 5).

In summary, because people are unlikely to contract *T. gondii* infection from direct contact with their personal cats, patients need not be advised to part with their cats or to have them tested for toxoplasmosis.^{73,a}

Bacterial diseases. *Salmonella* spp., *Campylobacter* spp., *Escherichia coli*, *Helicobacter* spp. and *Yersinia enterocolitica* infect cats and can cause disease in people. *Yersinia enterocolitica* is probably a commensal agent in cats but can induce fever, abdominal pain, bacteremia, and chronic polyarthritis in people.

Campylobacteriosis. *Campylobacter jejuni*, *C. coli*, *C. helveticus*, and *C. upsalensis* infections can be subclinical or result in anorexia, vomiting, and large bowel diarrhea in human beings and cats.⁷⁴⁻⁷⁷ Disease in cats is uncommon.⁷⁴ People are usually infected by ingesting contaminated food or water. The organism is directly infectious in feces; infection of human beings has been linked to cats in several reports.⁷⁸⁻⁸¹ In previous studies, it was reported that up to 60% of the pets from crowded environments were infected.^{1,74} *Campylobacter* spp. were cultured from feces of 47 of 227 commercially reared cats.⁷⁵ However, the incidence in client-owned cats may be lower. In 2 recent studies in north central Colorado¹² and central New York state,¹³ *Campylobacter* spp. were cultured from the stool of 0.0% and 1.8% of client-owned cats and 1.6% and 0.0% of shelter source cats, respectively. Diagnosis is based on culture. Several antibiotics including erythromycin, chloramphenicol, quinolones, and second generation cephalosporins are effective for treatment (Table 5). At this time, optimal repeat testing intervals are unknown but reinfection should be prevented by keeping cats indoors and feeding them cooked or commercially processed food (Table 2; see Followup testing recommendations, page 18).

Helicobacteriosis. Cats are infected by *H. felis*, *H. pametensis*, *H. pylori*, and “*H. heilmanni*”.⁸²⁻⁸⁴ *Helicobacter pylori* causes ulcers in people and has been isolated from a colony of research cats but not stray cats. *H. pylori* is rarely found in naturally exposed cats, and human infection probably does not originate from cats.⁸⁵ However, an infected person and his cat were infected with a genetically identical “*H. heilmanni*”.⁸⁶ In cats, the prevalence of *Helicobacter*-like organisms in gastric tissues ranges from 41-100% of healthy cats and 57-100% of vomiting cats.⁸³ In one study of farm workers with helicobacteriosis, an association was made with cat contact,⁸⁷ but in 3 other studies, including one of veterinarians, there was no epidemiologic association of cat contact with human helicobacteriosis.⁸⁸⁻⁹⁰ Based on these reports, it appears that human beings are unlikely to acquire *Helicobacter* spp. infection from contact with cats. However, people should avoid being licked on the face and should not share food utensils with cats (Table 2).

Salmonellosis. *Salmonella enteritidis* has more than 2,000 variants.⁹¹ The organism is infectious when passed in feces and can be a direct zoonosis. However, it appears that most infections occur from indirect contact. *Salmonella* infection in cats is often subclinical. Approximately 50% of clinically affected cats have gastroenteritis; others are presented with abortion, stillbirth, neonatal death, or signs of bacteremia.⁹²⁻⁹⁴ Neutropenia and neutrophils on rectal cytology are common findings in acute salmonellosis. Songbird fever is a clinical syndrome noted in some cats following the ingestion of infected birds.⁹² The incidence of salmonellosis varies by the region and husbandry. It was reported that *Salmonella* was cultured from 1% to 18% of cats.⁹⁵ However, the incidence in client-owned cats may be lower. In 2 recent studies in north

central Colorado¹² and central New York State,¹³ *Salmonella* was cultured from the stool of 0.8% and 0.9% of client owned cats and 1.3% and 0.7% of shelter source cats, respectively.

Diagnosis of salmonellosis is made by culture of stool. Prevention of salmonellosis is based on sanitation and control of exposure to feces, including that of prey species. Insect control should be maintained as well; flies trapped in greyhound kennels were recently shown to carry *Salmonella* spp..⁹⁶ Antibiotic therapy with drugs like quinolones can control clinical signs of disease but should not be administered to subclinical *Salmonella* carriers due to risk of developing antibiotic resistance. Several cats have been reported with multiple antibiotic resistant *Salmonella* infections.⁹⁷⁻⁹⁹ In bacteremic cats, parenterally administered quinolones (Table 5) are usually effective at controlling clinical signs of disease. At this time, optimal repeat testing intervals are unknown but reinfection should be prevented by keeping the cat indoors and feeding it cooked or commercially processed food (Table 2; see Followup testing recommendations that follow.

Followup testing recommendations and maintenance of cats with enteric zoonotic infections. For the majority of the enteric zoonotic agents of cats, it is unknown whether treatment eliminates infection. Repeat infection and shedding can occur with most enteric zoonotic agents after treatment. Diagnostic test results can be falsely negative or transiently negative and so it can be difficult to prove cure. Thus, with the information currently available, it is difficult

to make definitive recommendations concerning followup testing of cats known to be infected with an agent with zoonotic potential. The following are general recommendations for long-term management of cats known to have harbored an enteric zoonotic agent.

If a positive cat is detected, feces should be removed from the litterbox daily and disposed of properly while treatment is administered (if indicated). The litterbox should be disinfected or cleaned with scalding water and detergent, preferably by someone other than an immunosuppressed person with care taken to avoid burns. Probable sources of the primary infection should be removed if possible. For example, the cat should be housed indoors to minimize exposure to transport hosts, contaminated food or water, and other cats, and only processed foods should be fed. If the source of reinfection is likely to have been removed, it is indicated to repeat the appropriate fecal test at least once within 2 to 4 weeks of discontinuing treatment. However, the client should be advised that a single negative test result does not document elimination of infection. For cats that become chronic carriers of an enteric zoonotic agent, the clients should be advised of the public health risk. That risk may be unacceptable if very young children or immunocompromised people will be exposed. If the clients choose to keep the cat, they should exercise meticulous hygiene and sanitation, with emphasis placed on frequent hand washing, particularly after handling the cat, contacting potentially contaminated surfaces or materials, and before eating. They should be advised to seek medical care if they develop diarrhea or unexplained fever.

BITES AND SCRATCHES

Several infectious agents have been transmitted from cats to people via bites or scratches, including *Bartonella* spp., *Capnocytophaga* spp., *Mycoplasma felis*, *Pasteurella* spp., *Fransicella tularensis*, rabies, and *Yersinia pestis*. *Yersinia pestis* is discussed with the respiratory diseases. Guidelines for prevention of zoonoses transmitted by bites and scratches are summarized in Table 2.

Bartonellosis. Cats can be infected with *Bartonella henselae*, *B. clarridgeiae*, *B. koehlerae*, and *B. weissii*.¹⁰⁰⁻¹⁰⁴ *Bartonella henselae* and *B. clarridgeiae* have been associated with cat scratch disease in human beings.^{105,106} *B. henselae* causes bacillary angiomatosis and bacillary peliosis in immunosuppressed people. There are 2 genetic variants of *Bartonella henselae*, Type I and Type II. Both variants can be detected in infected cats and people.¹⁰⁷⁻¹⁰⁸ *Bartonella* spp. infection is the most common direct zoonosis associated with cats. In Japan, 35 of 233 (15.0%) veterinary health care providers were seropositive suggesting previous or current infection.¹⁰⁹

People with cat scratch disease develop a variety of clinical signs such as lymphadenopathy, fever, malaise, weight loss, uveitis, myalgia, headache, conjunctivitis, skin eruptions, and arthralgia. The disease is self-limited but may take several months to completely resolve. The incubation period is approximately 3 weeks. Most cases are associated with kitten contact. There are approximately 25,000 cases of cat scratch disease diagnosed in the USA every year resulting in at least 12.5 million dollars in health care costs.

As many as 54.6%-81% of cats in some geographical areas of the United States are

Bartonella spp. seropositive and so presumably were infected at one time.^{110,111} *Bartonella* spp. infection is more common in flea-infested cats from catteries.¹¹² *Bartonella henselae* replicates in fleas and can survive in flea feces for days.^{113,114} *Bartonella henselae* has been cultured from the blood of many naturally-exposed cats, cats infected with the organism by inoculation intradermally, subcutaneously, intravenously, or intramuscularly, and cats infected by fleas.^{111,115-119} Intravenous, intramuscular, and intradermal inoculation has resulted in fever, lethargy, lymphadenopathy, and neurologic diseases in some cats.¹¹⁸⁻¹²¹ In some naturally infected cats, uveitis and other clinical signs of disease including stomatitis, fever, and lymphadenopathy have been reported.¹²²⁻¹²⁵

Blood culture is the optimal test to prove the presence of current *Bartonella* spp. infection. However, bacteremia can be intermittent, and false-negative results might occur. Polymerase chain reaction can be used to document presence of *Bartonella* spp. DNA but there are occasional false negative results and positive results do not necessarily indicate that the organism is alive.¹²⁶ Serologic testing can be used to determine whether an individual cat has been exposed but both seropositive and seronegative cats can be bacteremic, limiting the diagnostic utility of serologic testing.¹²⁷ Thus, testing healthy cats for *Bartonella* spp. infection is not currently recommended.¹²⁸ Testing should be reserved for cats with suspected clinical bartonellosis.

Administration of doxycycline, tetracycline, erythromycin, amoxicillin-clavulanate, or enrofloxacin (Table 5) limit bacteremia but does not cure infection in all cats.^{115,116,118} Thus, antibiotic treatment of healthy bacteremic cats is controversial and not

currently recommended. Treatment should be reserved for cats with suspected clinical bartonellosis. Doxycycline was used successfully in the management of *Bartonella* spp. uveitis in a cat.¹²³ Administration of azithromycin decreased lymph node volume, but did not change the clinical outcome in children with cat scratch disease.¹²⁹

There are several precautions that can be taken to lessen the potential to develop bartonellosis (Table 2). These guidelines should be emphasized to immunosuppressed people. If a new cat is to be adopted, an adult cat without history of flea infestation is least likely to be infected. Flea control (Table 5) should be maintained continually and cats housed indoors to lessen the potential for exposure. Flea feces should be removed from the kitten and the environment. Kittens should be avoided by immunosuppressed people.

***Capnocytophaga* spp., *Mycoplasma felis*, and *Pasteurella* spp.** Approximately 300,000 emergency room visits per year are made by people bitten by animals in the United States.¹³⁰ Most of the aerobic and anaerobic bacteria associated with bite or scratch wounds cause only local infection in immunocompetent individuals. However, 28% to 80% of cat bites become infected and severe sequelae including meningitis, endocarditis, septic arthritis, osteoarthritis and septic shock can occur.¹³⁰

Immunocompromised people or individuals exposed to *Pasteurella* spp. or *Capnocytophaga canimorsus* (DF-2) are more likely to develop systemic clinical illness than when exposed to other bacteria associated with animal bites.^{131,132} Local cellulitis is noted initially, followed by evidence of deeper tissue infection. Osteomyelitis underlying the bite wound is often associated with *P. multocida* infection. Bacteremia and

the associated clinical signs of fever, malaise, and weakness are common and death can occur from either of these 2 genera, particularly in splenectomized individuals. *Pasteurella multocida* from a cat was cultured from the lungs of a man with AIDS who had only passive contact with the cat.¹³³ *Mycoplasma* spp. infections of people associated with cat bites, one with cellulitis and one with septic arthritis, have been reported.^{134,135}

Diagnosis of bacterial infections is confirmed by culture. Treatment of infected bite wounds in people includes local wound drainage and systemic antibiotic therapy. Penicillin derivatives are very effective against most *Pasteurella* infections. Penicillins and cephalosporins are effective against *Capnocytophaga in vitro*. People with bites and scratches should seek immediate medical attention. To avoid bites and scratches, cats should not be teased and appropriate restraint techniques should be utilized (Table 2).

Rabies. Cats are highly susceptible to rabies. They are usually infected with the enzootic strain that predominates in terrestrial animals locally. For example, along the Atlantic coast in the U.S., cats are most likely to be infected with the raccoon strain of rabies, in the Midwest, with a skunk strain. In Germany, cats became spillover hosts for the strain in foxes. There is no feline adapted strain of rabies anywhere in the world among wild or domestic cats, i.e., felids usually get infected from other animal species but do not maintain the infection within their own species. Despite the prevalence of rabies in bats in the United States and the likelihood that a cat would be attracted to and would attack a bat floundering on the ground, rabies from bat origin rarely occurs in cats. Perhaps this is because the cats are adept at avoiding getting bitten when they attack a bat, and bats, with

their tiny teeth, may have a hard time penetrating feline fur and skin.

Since 1980, more cases of rabies have been reported in cats than in dogs in the USA. In 2001, 270 cases of feline rabies were reported versus 89 cases of canine rabies.¹³⁶ Feline rabies is a major, potentially lethal, occupational health hazard for those commonly working with cats with unknown vaccination status including veterinary staff as well as humane shelter and rescue group employees. Preexposure vaccination should be offered to veterinarians and others who work with cats in rabies enzootic areas.¹³⁷ In a recent survey, 85.1% of veterinary medical association members and managers of animal shelters or wildlife rehabilitation centers had been vaccinated versus only 17.5% of staff members.¹³⁸ The pre-exposure series consists of three injections given on days 0, 7, and 21 or 28. Vaccinated individuals should have their titers checked every two years and a booster administered once the titer drops below an acceptable level.¹³⁷ Rabies vaccines are interchangeable. If a properly immunized person is exposed to rabies he or she should get two booster doses IM three days apart.¹³⁷

Cats should be administered their first rabies vaccine in accordance with the vaccine label, local ordinances, and published guidelines.¹³⁹ The second rabies vaccine should be administered one year later, and thereafter boosters are given annually or tri-annually as indicated for the specific vaccine product. Currently approved vaccines cannot induce rabies as occurred when modified live vaccines were used. While all approved vaccines have a very high level of efficacy, rabies has occurred in cats that were vaccinated. Some of those breakthrough cases could have occurred with use of outdated, improperly stored or improperly administered vaccine. Feline rabies

vaccination should be mandatory as it is for dogs in most communities. This should include indoor cats because they occasionally get outdoors and because rabid animals such as bats and raccoons can enter houses.

While rabies vaccination results in soft tissue sarcomas in between 1:1,000 and 1:10,000 cats, vaccination should be required in all cats due to the public health risks.¹³⁹⁻¹⁴³

The clinical signs in cats have been extensively reviewed.¹⁴⁴ Rabid cats can present with classical furious or dumb rabies but clinical signs can also be subtle, including hind leg lameness, increased vocalization with a change in pitch of voice, lethargy, anorexia, trembling, vomiting, and aggressiveness. It is possible that various strains of rabies could cause a different spectrum of illness. Rabies should always be considered in the differential diagnosis of a cat with these and other neurological symptoms that are not otherwise explained, or that becomes ill following an injury compatible with a bite.

In theory, cats can transmit rabies by scratch as well as bites because they lick their paws. A cat that has bitten or scratched a person or another animal should be confined and observed daily for ten days.¹³⁹ It should not receive rabies vaccine during that time. If it shows signs suggestive of rabies it should be euthanized, the local health department should be notified, and the head submitted (refrigerated, not frozen) for rabies examination at an approved laboratory. If it remains healthy then there is no risk that rabies transmission occurred, and it can be vaccinated and released from the quarantine at the end of the ten-day period.

If a properly, currently vaccinated cat is bitten by a proven or suspect rabid animal, it should receive a booster immediately and be

observed for 45 days. If it remains well through that time, it can be released from quarantine. If signs suggestive of rabies develop, it should be euthanatized and examined for rabies at an approved laboratory.

If a cat that is not currently vaccinated is bitten by a proven or suspect rabid animal, it should be euthanatized immediately. If the owner is not willing to have this done the cat should be kept in strict isolation for 6 months and vaccinated one month before release from quarantine. If signs of rabies develop during the quarantine period, it should be euthanatized and examined for rabies at an approved laboratory.

Cats that are rabies suspects should be strictly isolated and access to them limited to personnel that are currently immunized. Appropriate measures should be taken to reduce any possibility of the staff being injured by these animals during the quarantine period. Public health officials should be notified immediately about possible exposures to rabies. Individuals exposed to potentially rabid animals should be urgently referred to a physician.

Feline retroviruses. There has been concern that the feline retroviruses, feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), and feline foamy virus (FeFV) can infect people.¹⁴⁵ This has been a particular concern with FeLV because subtypes B and C can replicate in human cell lines.^{146,147} Several studies have been performed over the years to assess the risk. To date, people have not been shown to be infected with feline retroviruses. In a recent study, 204

veterinarians and others potentially exposed to feline retroviruses were assessed for antibodies against FIV and FeFV, FeLV p27 antigen, and FeLV provirus.¹⁴⁵ There was no serologic or molecular evidence of infection of any individual by any of the 3 retroviruses. At this time, there is no known risk of human infection with feline retroviruses. Whether infection of a cat with a retrovirus increases human risk for other zoonoses is undetermined.

Tularemia. Tularemia is caused by *Francisella tularensis*, a gram-negative bacillus, widely endemic in the continental United States and Europe. *Dermacentor variabilis*, *D. andersoni*, and *Amblyomma americanum* are vectors.^{148,149} Tularemia can be transmitted to people by ingestion, aerosol from water, soil or other fomites, from tick bite or from contact with infected animals including cats. Cats are infected most frequently by tick bites or by ingesting infected rabbits or rodents. Infected cats exhibit generalized lymphadenopathy and abscess formation in organs such as the liver and spleen which lead to fever, anorexia, icterus, and death.¹⁵⁰⁻¹⁵² Ulceroglandular, oculoglandular, glandular, oropharyngeal, pneumonic, and typhoidal forms occur in people, depending on the route of exposure. Cat-associated tularemia in human beings has occurred most frequently via bites but also has been associated with exposure to infected cat tissues.^{149,153} Cultures and documentation of increasing antibody titers can be used to confirm the diagnosis in cats and people. To lessen risk of exposure, ectoparasite control should be maintained and cats should not be allowed to hunt. This disease is an uncommon zoonosis.

RESPIRATORY EXPOSURE

A number of agents carried by cats can infect people by exposure to respiratory secretions. These include *Yersinia pestis*, *Bordetella bronchiseptica*, *Staphylococcus* spp., and potentially, *Chlamydomphila felis*. *Coxiella burnetii* infects people by inhalation, but is covered in the genitourinary section because it is passed in parturient secretions. People can develop respiratory disease by inhaling *Francisella tularensis*, but this agent is discussed in the bites and scratches section because this is a more common route of transmission from cats.

Bordetellosis. *Bordetella bronchiseptica* is a common primary pathogen in dogs resulting in infectious tracheobronchitis. Many cats have serologic evidence of exposure or are culture positive, particularly in crowded environments.^{154,155} Cats may acquire infection from contact with infected dogs.¹⁵⁶ In one study, *B. bronchiseptica* was isolated from 82 of 740 cats sampled.¹⁵⁵ While exposure is common, the infection is usually subclinical in cats. Clinically affected cats have fever, mucopurulent nasal discharge, and cough.^{157,158} By 1998, 39 cases of *B. bronchiseptica* infection in people had been reported; many were immunodeficient.¹⁵⁹⁻¹⁶² Association with a cat has only been reported once, in an HIV and *B. bronchiseptica* coinfecting person.¹⁶² Because cats are commonly exposed but people are rarely infected, it appears that *B. bronchiseptica* infection of people from contact with cats is uncommon. However, in households with immunosuppressed family members, a diagnostic workup and antimicrobial therapy should be considered for cats with respiratory disease. The organism is easily cultured. Tetracycline derivatives, amoxicillin-clavulanate, and quinolones are effective in controlling clinical signs of disease but

treated cats can be culture positive for months (Table 5).

Chlamydiosis. *Chlamydomphila felis* (formerly feline *Chlamydia psittaci*) commonly causes conjunctival disease and can cause rhinitis in cats.¹⁶³ The prevalence rates of antibodies against an isolate of *Chlamydomphila felis* in Japan were 51.1% in stray cats, 15.0% in pet cats, 3.1% in the general human population and 5.0% in small animal clinic veterinarians, suggesting that transfer between cats and people may occur.¹⁶⁴ This agent is thought to cause conjunctivitis in people following direct contact with ocular discharges from cats.¹⁶⁵⁻¹⁶⁸ Feline *Chlamydia* was indirectly associated with atypical pneumonia in an apparently immunocompetent 48 year old man,¹⁶⁹ with malaise and cough in an immunosuppressed woman,¹⁷⁰ and with endocarditis and glomerulonephritis in a 40 year old woman.¹⁷¹ Care should be made to avoid direct conjunctival contact with discharges from the respiratory or ocular secretions of cats, especially by immunosuppressed persons (Table 2). Tetracycline derivatives topically or orally are effective for the treatment of infected cats.¹⁶³

Group A Streptococcus. Human beings are the natural hosts for group A *Streptococcus pyogenes*, the principal cause of “strep throat” in people. It is theoretically possible that cats in close contact with infected people could develop colonization of pharyngeal tissues which could lead to the infection of people.¹⁷²⁻¹⁷⁵ However, this is poorly documented and is unlikely. Veterinarians may be consulted about treating the cats of a family with chronic or recurrent strep throat. Culture of the tonsillar crypts with Lancefield group serologic testing should be used to confirm carriage. Without serotyping, other

beta hemolytic streptococci, not *S. pyogenes*, found in cats could be isolated and erroneously designated as the source of human infection. Penicillin derivatives should be effective at clearing any possible carrier state in cats.

Feline plague. Feline plague is caused by *Yersinia pestis*, a gram-negative coccobacillus found most commonly in the U.S., in mid- and far-western states; it is also found in many Asian, African and Latin American countries.^{176,177} Rodents are the natural hosts for this bacterium; cats are most commonly infected by ingesting bacteremic rodents or lagomorphs or by being bitten by *Yersinia* infected rodent fleas.¹⁷⁷ People are most commonly infected by rodent flea bites, but there have been many documented cases of transmission by exposure to wild animals and domestic cats. From 1977 to 1998, 23 cases of human plague (7.7% of the total cases) resulted from contact with infected cats.¹⁷⁸ Human beings can be infected by inhalation of respiratory secretions of cats with pneumonic plague, by bite, or by contaminating mucous membranes or abraded skin with secretions or exudates. Bubonic, septicemic, and pneumonic plague can develop in cats and people; each form has accompanying fever, headache, weakness, and malaise.¹⁷⁷ Suppurative lymphadenitis (buboes) of the cervical and submandibular lymph nodes is the most common clinical manifestation in cats. Exudates from cats with lymphadenomegaly should be examined cytologically for the characteristic bipolar rods. The diagnosis is confirmed by culture of exudates, tonsillar area, and saliva, by fluorescent antibody staining of exudates, and by documentation of increasing antibody titers. Cats in enzootic areas with suppurative lymphadenitis should be considered plague suspects and extreme caution should be exercised when handling exudates or treating draining wounds. People that are exposed to

infected cats should be urgently referred to physicians for antimicrobial therapy and public health officials alerted. Aminoglycosides, chloramphenicol, enrofloxacin (Lappin MR, unpublished data) and tetracyclines can be used successfully for the treatment of feline plague. Dogs are more resistant to *Yersinia* infection than cats. Cats are not considered to be a zoonotic risk to people after 4 days of antibiotic treatment. Guidelines for handling hospitalized plague suspects are listed in Table 7.

Table 7. Plague control procedures

In endemic areas, from April through October, cats with clinical evidence of submandibular or retropharyngeal lymphadenopathy or abscessation, clinical signs of bacteremia, or coughing should be considered plague suspects.

All plague suspects should be placed in strict isolation and the door clearly marked as housing a plague-infected animal.

Number of staff exposures to the cat for treatments or cleaning should be minimized.

Cats with submandibular abscessation should be handled with care; gloves, surgical mask (preferably a N-95 type respirator), and gown should be worn while aspirating the mass.

Coughing cats that require transoral tracheal aspiration should be handled as plague suspects; the procedures should be completed while wearing gloves, gown and surgical mask.

Specimens should be collected, bagged, clearly labeled as plague suspect, and transported to the appropriate Diagnostic Laboratory.

Antemortem samples should only be submitted from client-owned cats and include abscess material smeared and dried on a slide for FA; abscess biopsy; lymph node biopsy; tracheal wash fluid. Fresh tissues or fluids can be submitted for culture or mouse inoculation.

Postmortem samples vary by the clinical signs; appropriate tissues include abscess material, spleen, liver, or lung.

Surfaces contaminated by contact with fluids from infected cats should be cleaned with quaternary ammonium disinfectants.

Bedding and waste should be incinerated.

Affected cats, the home environment, and the veterinary hospital should be treated for fleas.

The clients and all other individuals in contact with the infected cat should be urgently advised to consult a physician for prophylactic antibiotic treatment.

Companion animals of infected cats should be treated prophylactically with tetracyclines for 7 days.

County and the State Department of Health officials should be notified.

CUTANEOUS OR EXUDATE EXPOSURE

Dermatophytosis. There are several dermatophytes shared between cats and human beings; *Microsporum canis* is thought to be the most common. Approximately 50% of the people exposed and most people living in households with dermatophyte-infected cats become infected themselves.¹⁷⁹ Cats can be subclinical carriers or develop superficial dermatologic disease characterized by broken haired alopecia, crusts, and scale.^{180,181} Infected people develop characteristic red, raised, circular, pruritic lesions at infection sites. Invasive infection can occur in immunocompromised people.¹⁸² Microconidia may be noted within hair shafts on cytologic examination and some cutaneous fungi fluoresce under black light illumination. Definitive diagnosis can be made by culture of hair but false negative and false positive results can occur. Risk to people is greatest from kittens from shelters with known history of infection and from pet cats exposed to large numbers of other animals. The age of both the person and the cat also influence risk; children and kittens are most likely to be infected.¹⁸³ To lessen the risk for zoonotic transmission, affected areas should be carefully shaved (which may worsen the lesion locally) and topical treatment combined with systemic treatment (Table 5). A vaccine is available that is not recommended by most as a preventative.⁵⁹ When used as a treatment, vaccination may result in the development of a subclinical carrier state. To be considered ringworm free, a previously infected cat should be shown to be negative by culture 3 times, 3 weeks apart.¹⁷⁹

Ectoparasites. In addition to being the vector or reservoir of some zoonotic agents (see shared vector zoonoses), ectoparasites can also induce disease primarily.

Ctenocephalides felis, *Cheyletiella*, *Sarcoptes scabiei*, *Notedres cati*, and a variety of ticks will parasitize both cats and people. Pruritic skin disease is most common with ectoparasites other than ticks. Diagnosis is based on visualization of the organism grossly (*C. felis*, ticks) or during microscopic examination of skin scrapings (*S. scabiei*; *Notedres cati*; *Cheyletiella*), combing or tape test (*Cheyletiella*). Topical and systemic treatments are available (Table 5).

Sporotrichosis. *Sporothrix schenckii* is a saprophytic fungus common to soils throughout the world. Multiple cases have been reported in cats.¹⁸⁴⁻¹⁸⁶ Infection of cats and human beings usually occurs after the organism contaminates broken skin. Cats are thought to be infected by scratches from contaminated claws of other cats; infection is most common in outdoor males.¹⁸⁵ Infection of both cats and people is characterized by ulcerative cutaneous lesions usually with a mucopurulent discharge. In cats, lesions are most common on the limbs, head, and tailbase. Many cats develop systemic infection of lymph nodes and lymphatics. Humans often have nodular lymphadenitis advancing centripetally from the site of inoculation. In cats, the organism replicates readily and large numbers are passed in the exudates, potentially resulting in human infection.¹⁸⁶ The organism is round, oval or cigar shaped and can be extracellular or intracellular after being engulfed by macrophages. The presumptive diagnosis is based on cytologic demonstration; definitive diagnosis is confirmed by culture. Long term antifungal treatment is usually required. Direct skin contact with exudates should be avoided.

GENITOURINARY EXPOSURE

Coxiellosis. *Coxiella burnetii* is the rickettsial agent found throughout the world including North America that causes Q fever in human beings. Cats, cattle, sheep, and goats are usually subclinically infected and pass the organism into the environment in urine, feces, milk, and parturient discharges. Infection of cats most commonly occurs following tick exposure, ingestion of contaminated carcasses, or aerosolization from a contaminated environment. The true incidence of infection in cats has not been determined; 20% of the cats tested from a humane society in southern California and in Maritime Canada were seropositive suggesting exposure is common.^{187,188} The organism was grown from the vagina of healthy cats in Japan.¹⁸⁹ People are infected by aerosol exposure to the organism passed by normally parturient or aborting cats. Acute clinical signs in people include fever,

malaise, headache, interstitial pneumonitis, myalgia, and arthralgia.¹⁹⁰⁻¹⁹⁴ In cat-associated infections, clinical signs develop 4 to 30 days after contact. In approximately 1% of human cases, chronic Q fever can develop years after primary infection and can manifest as hepatic inflammation or valvular endocarditis. Tetracyclines, chloramphenicol, and quinolones are usually effective therapeutic agents in people. Gloves and masks should be worn when attending to parturient or aborting cats.

Leptospirosis. Cats can be infected with *Leptospira interrogans*, but the disease is usually subclinical even though organisms can be detected in urine, blood, and tissues.¹⁹⁵ Ascites due to infection may have occurred in one cat.¹⁹⁶ To our knowledge, infection of human beings from cat contact has not been reported.

SHARED VECTOR ZOOSES

There are many zoonotic organisms transmitted by vectors. Those transmitted by fleas and ticks are potentially of the greatest significance because cats can bring those vectors into the human environment. Those transmitted by mosquitoes, like *Dirofilaria immitis* and West Nile virus, are not directly related to cats in any fashion.

Anaplasma phagocytophilum. DNA of *A. phagocytophilum* (previously *Ehrlichia equi* and human granulocytic ehrlichial agent)¹⁹⁷ has been amplified from the blood of cats in the United States, Sweden, Ireland, Denmark, and Mexico.¹⁹⁸⁻²⁰² Several of the cats were clinically ill and responded to administration of tetracycline therapy suggesting the organism was associated with the clinical disease.^{198,199} Several of the cats were infested by *Ixodes* spp. ticks that are known to

be the vector in humans. It is unknown, but unlikely that direct contact with infected cats would result in human infection.

Bartonella spp. *Bartonella henselae* is transmitted between cats by fleas and lives for at least days in flea feces.^{113,114,117} Thus, it is possible fleas or their excrement are associated with human infection. See the bite and scratch zoonoses for a further discussion of this organism.

Borrelia burgdorferi. *Ixodes* spp. ticks are the vectors for *B. burgdorferi*. The organism is endemic to the northeastern and north central United States as well as northern California.²⁰³ Significant clinical syndromes in some infected people include rash, arthritis, cranial neuropathies, and myocardial disease. While *B. burgdorferi* antibodies have been

detected in the serum of cats and experimental infections have been produced, there is no compelling evidence to suggest that naturally-infected cats are clinically affected.^{204,205} There is no evidence that human borreliosis is associated with cat contact. It is unlikely that the organism reaches infectious levels in cat urine. However, since *Ixodes* spp. will feed on cats, it is possible for cats to bring infected ticks into the human environment. Thus, tick control should be maintained (Table 2).

***Ehrlichia* spp.** Based on the presence of morulae in mononuclear cells and the presence of antibodies that seroreact with *E. canis* or *Neorickettsia risticii* (previously *E. risticii*), ehrlichiosis has been suspected in multiple cats around the world.²⁰⁶⁻²¹³ To date, *E. canis*-like DNA has been amplified from EDTA blood from three cats in North America and two cats in France.^{212,213} Whether these *Ehrlichia* will also infect human beings is unknown, and it is unlikely

that direct zoonotic transfer occurs. Tick control should be maintained.

***Rickettsia felis*.** In human beings, louse-borne or epidemic typhus is caused by *Rickettsia prowazekii*. In southern Texas and California, opossums serve as a reservoir and the organism is transmitted by *Ctenocephalides felis*. Utilizing PCR and restriction fragment length polymorphism, *Rickettsia felis* was discovered in a person with clinical signs similar to typhus.²¹⁴⁻²¹⁵ Subsequently, *R. felis* has been isolated from *C. felis* in multiple states including California, Florida, Georgia, Louisiana, New York, North Carolina, Oklahoma, Texas, and Tennessee as well as France.²¹⁶ The organism is passed tran-stadially and trans-ovarially in the flea. Experimentally inoculated cats are subclinically infected but seroconvert. It is unknown if cats are clinically affected. However, flea control should be maintained.

SHARED ENVIRONMENT ZONNOSES

A number of infectious agents infect people and cats from the same environment but are not usually transmissible between species. Examples include *Histoplasma capsulatum*, *Blastomyces dermatitidis*, *Coccidioides*

immitis, *Cryptococcus neoformans*, *Mycobacterium avium*, and *Aspergillus* spp.. Cats infected with these organisms can be sentinels, warning of environmental risk to people.

BIOTERRORISM

Yersinia pestis, *Francisella tularensis*, *Bacillus anthracis* and *Coxiella burnetii* are some of the potential agents of bioterrorism. Cats could be coincidental victims of such an attack and could be sentinels of human exposure. They could also maintain the infection in the environment for some period of time after the initial attack. Veterinarians

should promptly report cases of these infections to their state departments of animal and public health and should do so with particular urgency if cases occur with unusual frequency or geographic distribution. Further discussion of bioterrorism is beyond the scope of this monograph. Links to important resources are available for review.^d

^dwww.avma.org/pubhlth/biosecurity/resources.asp

RECOMMENDATIONS FOR VETERINARIANS

Veterinarians should familiarize themselves with zoonotic issues and take an active role in discussion of the health risks and benefits of pet ownership with clients so that logical decisions concerning ownership and management of individual animals can be made (Tables 2 and 3). Attempts should be made to show that the staff of the veterinary hospital understands immunodeficiency, is discreet, and is willing to help. Veterinarians should contact appropriate public health officials when reportable zoonotic diseases are diagnosed. Information concerning veterinary or public health aspects of zoonoses should be provided to clients as

indicated or requested, but veterinarians should not diagnose or treat diseases in people or make recommendations about those issues. The client should always be referred to a human health care provider for additional information and treatment. The veterinarian should always document in the medical record that public health related advice was offered. Failure to provide information concerning zoonoses may have legal implications.²¹⁷ Biosecurity procedures should be followed to lessen potential for infectious disease spread within the hospital (Table 8).

BIOSECURITY PROCEDURES FOR SMALL ANIMAL HOSPITALS^e

General biosecurity guidelines. Contaminated hands are the most common source of infectious disease transmission in the hospital environment. Fingernails of personnel having patient contact should be cut short. Hands should be washed prior to and after attending to each individual animal. Hands should be washed as follows: collect clean paper towels and use to turn on water faucets; wash hands for 30 seconds with antiseptic soap, being sure to clean under fingernails; rinse hands thoroughly; use the paper towel to dry hands; and use paper towel to turn off the water faucets. Antiseptic hand lotions should be made available. Personnel should not touch patients, clients, food, door knobs, drawer or cabinet handles or contents, equipment, or medical records with soiled hands or gloves.

All employees should wear an outer garment like a smock or scrub suit when attending to patients. Footwear should be protective, clean, and cleanable, or disposable shoe covers used appropriately. A minimum of 2 sets of outer garments should always be

available, and should be changed immediately after contamination with feces, secretions, or exudates. Equipment such as stethoscopes, pen lights, thermometers, bandage scissors, lead ropes, percussion hammers, and clipper blades can be fomites and should be cleaned and disinfected with 0.5% chlorhexidine solution after each use. Disposable thermometer covers or disposable thermometers should be used.

To avoid zoonotic transfer of infectious diseases, food or drink should not be consumed in areas where cat care is provided. Food and beverages should not be kept in refrigerators used for storing laboratory specimens. All areas where cats are examined or treated should be cleaned and disinfected immediately after use, irrespective of infectious disease status of the individual animal.

Patient evaluation. Recognition of zoonotic diseases starts with the front desk personnel. Staff should be trained to be alert for public health problems and to direct these issues to

the appropriate person. Cats with cutaneous, gastrointestinal or respiratory diseases are the most likely to be contagious. Infectious gastrointestinal disease is possible in all cats with small or large bowel diarrhea, whether the signs are acute or chronic. The index of suspicion for infectious diseases is increased for cats with acute disease and fever, particularly if the animal is from a crowded environment like a breeding facility, boarding facility, or humane society. Front desk personnel should indicate clearly on the hospital record that gastrointestinal or respiratory disease is occurring. If the presenting complaint is known prior to admission into the hospital, it is optimal to meet the client in the parking area to determine the infectious disease risk prior to entering the hospital. If infectious gastrointestinal or respiratory disease is suspected, the cat should be transported (ie; not allowed to walk on the premises) to an examination room or the isolation facility. If a cat with acute gastrointestinal or respiratory disease is brought directly to the reception desk, the receptionist should contact the receiving clinician or technician immediately and coordinate placement of the animal in an examination room to minimize hospital contamination. If hospitalization is required, the cat should be transported to the appropriate housing area by the shortest route possible, preferably using a carrier to lessen hospital contamination.

Hospitalized patients. If possible, all cats with suspected zoonotic diseases such as *Salmonella* spp., *Campylobacter* spp., rabies, or plague should be housed in an isolated area of the hospital. The number of staff members entering the isolation area should be kept to a minimum. Upon entry into the isolation area, outerwear should be removed and surgical booties or other disposable shoe covers should be placed over the shoes. Alternately, a foot-bath filled with

disinfectant should be placed by the exit and used when leaving the area. A disposable gown (or smock designated for the patient) and latex gloves should be put on. A surgical mask (preferably a type N95 particulate respiratory) should be worn when attending cats with plague. Separate equipment and disinfectant supplies should be used in the isolation area.

All biological materials submitted to the clinical pathology laboratories or diagnostic laboratories from animals with suspected or proven infectious diseases should be clearly marked as such. Fecal material should be placed in a plastic, screw-capped cup using a tongue depressor, while wearing gloves. Place the cup in a clean area and put the lid on with a clean gloved hand. Remove the used gloves and place the cup in a second bag clearly marked with the name of the zoonotic disease suspected. The outer surface of the bag should be disinfected prior to leaving the isolation area.

Disposable materials should be placed in plastic bags in the isolation area. The external surfaces of the bags should be sprayed with a disinfectant prior to being removed from the isolation area. After attending to the patient, contaminated equipment and surfaces should be cleaned and disinfected, and contaminated outer garments and shoe covers should be removed. Hands should be washed after discarding the contaminated outerwear. Disposable dishes and litterpans should be used or dishes and litterpans should be cleansed thoroughly with detergent before returning them to the central supply area. Optimally, materials like outerwear and equipment to be returned to central supply should be placed in plastic bags and sprayed with a disinfectant prior to transport. Procedures requiring general hospital facilities like surgery and radiology should be postponed to the end of the day, if possible,

and the contaminated areas disinfected prior to use with other animals. Cats should be discharged using the shortest possible route to the parking lot.

Basic disinfection protocols. Cats should not be moved from cage to cage if possible. Cage papers and litterpans soiled by feces, urine, blood, exudates, or respiratory secretions should be removed and placed in trash receptacles. Bulk fecal material should also be placed in trash receptacles.

Many agents are resistant to disinfectants or require prolonged contact time to be inactivated.²¹⁸ Contaminated surfaces including the cage or run floor, walls, ceiling, door, and door latch should be wetted thoroughly with a disinfectant which is then blotted with clean paper towels or mops. Surfaces should be in contact with the disinfectant for 10 minutes if possible, particularly if known infectious agents are present. Soiled paper towels should be placed in trash receptacles. If zoonotic diseases are suspected, the trash bags should be sealed, the surface of the bag sprayed with a disinfectant, and the trash bags discarded.

Contaminated surfaces in examination rooms should be cleaned to remove hair, blood, feces, and exudates. Examination tables, countertops, floors, canister lids, and water taps should be saturated with disinfectant for 10 minutes if possible.

Surfaces should be blotted with paper towels until dry, and the soiled towels placed in a trash receptacle. Urine or feces on the floor should be contained with paper towels, blotted, and placed in trash receptacles. The soiled area of the floor should be mopped with disinfectant.

Disinfectants are relatively effective for viral and bacterial agents, but require high concentrations and long contact times to kill parasite eggs, cysts, and oocysts. Cleanliness is the key to lessening hospital-borne infection with these agents; detergent or steam cleaning inactivates most. Litterpans and dishes should be thoroughly cleaned with detergent and scalding water.

More frequent cleaning is suggested for areas where hospital acquired infections are more common, like surgical suites and critical care units. In these areas, periodic closure for extensive cleaning is indicated. If hospital borne infections occur frequently, environmental cultures should be used to attempt to identify a source and so assess cleaning and disinfection protocols.

^cThese guidelines were written initially for the Biosecurity Standard Operating Procedures at Colorado State University and then adapted for use here. <http://www.vth.colostate.edu/vth/biosecurity/biosecurity.html>

Table 8. General hospital biosecurity guidelines

Wash hands before and after each cat contact.

Wear gloves when handling cats when zoonotic diseases are on the differential list of diagnoses.

Minimize contact with hospital materials (instruments, records, door handles, etc.) while hands or gloves are contaminated.

Always wear an outer garment like a smock or scrub shirt when handling cats.

Change outer garments when soiled by feces, secretions, or exudates.

Clean and disinfect equipment (stethoscopes, thermometers, bandage scissors, etc.) with 0.5% chlorhexidine solution after each use.

Do not consume fluid or drink in areas where cat care is provided.

Clean and disinfect examination tables and cages after each use.

Clean and disinfect litter boxes and dishes after each use.

Place cats with suspected infectious diseases immediately into an examination room or an isolation area upon admission into the hospital.

When possible, postpone until the end of the day any procedures using general hospital facilities like surgery and radiology.

REFERENCES

1. Evans RH. Public health and important zoonoses in feline populations. In, August JR (ed), *Consultations in Feline Internal Medicine*. Third edition, WB Saunders Co., Philadelphia, 1997, pp. 611-629.
2. Burton B. Pets and PWAs: Claims of health risk exaggerated. *AIDS Patient Care* February, 1989, pp. 34-37.
3. Spencer L. Study explores health risks and the human animal bond. *J Am Vet Med Assoc* 1992;201:1669.
4. Kravetz JD, Federman DG. Cat-associated zoonoses. *Arch Intern Med* 2002;162:1945-1952.
5. Angulo FJ, Glaser CA, Juranek DD, et al. Caring for pets of immunocompromised persons. *J Am Vet Med Assoc* 1994;205:1711-18.
6. Glaser CA, Angulo FJ, Rooney JA. Animal associated opportunistic infections among persons infected with the human immunodeficiency virus. *Clin Infect Dis* 1994;18:14-24.
7. Greene CE. Immunocompromised people and pets. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 710-717.
8. Carmack B. The role of companion animals for persons with AIDS/HIV. *Hol Nurs Pract* 1991;5:24-31.
9. Grant S, Olsen CW. Preventing zoonotic diseases in immunocompromised persons: the role of physicians and veterinarians. *Emerg Inf Dis* 1999;5:159-163.
10. Kirkpatrick CE. Epizootiology of endoparasitic infections in pet dogs and cats presented to a veterinary teaching hospital. *Vet Parasitol* 1988;30:113-124.
11. Nolan TJ and Smith G. Time series analysis of the prevalence of endoparasitic infections in cats and dogs presented to a veterinary teaching hospital. *Vet Parasitol* 1995; 59: 87-96.

12. Hill S, Lappin MR, Cheney J, et al. Prevalence of enteric zoonotic agents in cats. *J Am Vet Med Assoc.* 2000;216:687-692.
13. Spain CV, Scarlett JM, Wade SE, McDonough P. Prevalence of enteric zoonotic agents in cats less than 1 year old in central New York State. *J Vet Int Med* 2001;15:33-38.
14. Blagburn BL, Conboy G, Jutras P, et al. Strategic control of intestinal parasites: diminishing the risk of zoonotic disease. *Comp Cont Ed Pract Vet* 1997(S);19:4-20.
15. Marcus LC. Medical aspects of visceral and cutaneous larva migrans and hydatid disease in humans. *Comp Cont Ed Pract Vet* 2001;23(S);11-17.
16. Overgaauw PAM. Aspects of *Toxocara* epidemiology: Toxocarosis in dogs and cats. *Crit Rev in Microbio* 1997; 23: 233-251.
17. Fisher, M. *Toxocara cati*; an underestimated zoonotic agent. *Trends Parasitol* 2003;19:167-170.
18. Prociv R, Croese J. Human enteric infection with *Ancylostoma caninum*: hookworms reappraised in the light of a “new” zoonosis. *Act Tropica* 1996;62:23-44.
19. McTier TL, Shanks DJ, Wren JA, et al. Efficacy of selamectin against experimentally induced and naturally acquired infections of *Toxocara cati* and *Ancylostoma tubaeforme* in cats. *Vet Parasitol* 2000;91:311-319.
20. Shimada A, Muraki Y, Awakura T, et al. Necrotic colitis associated with *Entamoeba histolytica* infection in a cat. *J Comp Path* 1992;106:195-199.
21. Nkauchi K. The prevalence of *Balantidium coli* infection in fifty six mammalian species. *J Vet Med Sci* 1999;61:63-65.
22. Gookin JL, Breitschwerdt EB, Levy MG, et al. Diarrhea associated with trichomonosis in cats. *J Am Vet Med Assoc* 1999;215:1450-1454.
23. Gookin JL, Levy MG, Law JM, et al. Experimental infection of cats with *Tritrichomonas foetus*. *Am J Vet Res* 2001;62:1690-1697.
24. Diers J, McCallister GL. Occurrence of *Cryptosporidium* in home daycare centers in west-central Ohio. *J Parasitol* 1989;75:637-638.
25. MacKenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *N Engl J Med* 1994;331:161-167.
26. Beneson A. S. 1990. Cryptosporidiosis. In, *Control of Communicable Diseases in Man*. 15th edition, American Public Health Association, pp 112-114.
27. Arai H, Fukuda Y, Hara T, et al. Prevalence of *Cryptosporidium* infection among domestic cats in the Tokyo metropolitan district, Japan. *Jap J Med Sci* 1990;43:7-14.
28. Uga S, Matsumura T, Ishibashi K. Cryptosporidiosis in dogs and cats in Hyogo Prefecture, Japan. *Jap J Parasitol* 1989;38:139-143.
29. Goodwin MA, Barsanti JA. Intractable diarrhea associated with intestinal cryptosporidiosis in a domestic cat also infected with feline leukemia virus. *J Am Anim Hosp Assoc* 1990;26:365-368.
30. 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.
31. Lent SF, Burkhardt JE, Bolka D. Coincident enteric cryptosporidiosis and lymphosarcoma in a cat with diarrhea. *J Am Anim Hosp Assoc* 1993;29:492-496.
32. Nash AS, Mtambo MMA, Gibbs HA. *Cryptosporidium* infection in farm cats in the Glasgow area. *Vet Rec* 1993;133:576-577.

33. Barr SC, Jamrosz GF, Hornbuckle WE, et al. Use of paromomycin for treatment of cryptosporidiosis in a cat. *J Am Vet Med Assoc* 1994;205:1742-1743.
34. Lappin MR, Dowers K, Edsell D, et al. Cryptosporidiosis and inflammatory bowel disease in a cat. *Fel Pract* 1997;3:10-13.
35. Lappin MR, Ungar B, Brown-Hahn B, et al. Enzyme-linked immunosorbent assay for the detection of *Cryptosporidium* spp. IgG in the serum of cats. *J Parasitol* 1997;83:957-960.
36. Sargent KD, Morgan UM, Elliot A, et al. Morphological and genetic characterisation of *Cryptosporidium* oocysts from domestic cats. *Vet Parasitol* 1998;77:221-227.
37. McReynolds C, Lappin MR, McReynolds L, et al. Regional seroprevalence of *Cryptosporidium parvum* IgG specific antibodies of cats in the United States. *Vet Parasitol* 1998;80:187-195.
38. Juranek DD. Cryptosporidiosis: sources of infection and guidelines for prevention. *Clin Infect Dis* 1995;21(S);57-61.
39. Egger M, Nguyen X, Schaad UB, et al. Intestinal cryptosporidiosis acquired from a cat. *Infection* 1990;18:177-178.
40. Bennett MD, Baxby D, Blundell N, et al. Cryptosporidiosis in the domestic cat. *Vet Rec* 1985;116:73-74.
41. Edelman M J, Oldfield EC. Severe cryptosporidiosis in an immunocompetent host. *Arch Int Med* 1988;148:1873-1874.
42. Koch KL, Shandey TV, Weinstein GS, et al. Cryptosporidiosis in a patient with hemophilia, common variable hypogammaglobulinemia, and the acquired immunodeficiency syndrome. *Annals Int Med* 1983;99:337-340.
43. Asahi H, Koyama T, Arai H, et al. Biological nature of *Cryptosporidium* sp. isolated from a cat. *Parasitol Res* 1991;77:237-240.
44. Mtambo MMA, Wright E, Nash AS, et al. Infectivity of a *Cryptosporidium* species isolated from a domestic cat (*Felis domestica*) in lambs and mice. *Res Vet Sci* 1996;60:61-63.
45. Morgan-Ryan, U.M. *et al.*: *Cryptosporidium hominis* n. sp. (Apicomplexa: Cryptosporidiidae) from *Homo sapiens*. *J. Eukaryot. Microbiol.* 49:433-440; 2002.
46. Morgan UM, Constantine CC, Forbes DA, et al. Differentiation between human and animal isolates of *Cryptosporidium parvum* using rDNA sequencing and direct PCR analysis. *J Parasitol* 1997;83:825-830.
47. Pieniazek NJ, Bornay-Llinares FJ, Slemenda SB, et al. New *Cryptosporidium* genotypes in HIV-infected persons. *Emerg Inf Dis* 1999;5:444-449.
48. Caccio S, Pinter E, Rantini R, et al. Human infection with *Cryptosporidium felis*: case report and literature review. *Emerg Inf Dis* 2002;8:85-86.
49. Morgan U, Weber R, Xiao L, et al. Molecular characterization of *Cryptosporidium* isolates obtained from human immunodeficiency virus-infected individuals living in Switzerland, Kenya, and the United States. *J Clin Microbiol* 2000;38:1180-1183.
50. Bornay-Llinares FJ, da Silva AJ, Moura INS, et al. Identification of *Cryptosporidium felis* in a cow by morphologic and molecular methods. *Applied Environ Microbiol* 1999;65:1455-1458.
51. Glaser CA, Safrin S, Reingold A, et al. Association between *Cryptosporidium* infection and animal exposure in HIV-infected individuals. *J AIDS* 1998;17:79-82.
52. Mtambo MMA, Nash AS, Blewett DA, et al. Comparison of staining and concentration techniques for detection of *Cryptosporidium* oocysts in cat faecal specimens. *Vet Parasitol* 1992;45:49-57.

53. Scorza AV, Brewer MM, Lappin MR. Polymerase chain reaction for the detection of *Cryptosporidium* spp. in cat feces. *J Parasitol* 2003;89:423-426.
54. Chalmers RM, Sturdee AP, Bull SA, et al. The prevalence of *Cryptosporidium parvum* and *C. muris* in *Mus domesticus*, *Apodemus sylvaticus*, and *Clethrionomys glareolus* in an agricultural system. *Parasitol Res* 1997;83:478-482.
55. Gookin JL, Riviere JE, Gilger BC, et al. Acute renal failure in four cats treated with paromomycin. *J Am Vet Med Assoc* 1999;215:1821-1823.
56. Kirkpatrick CE and Green GA. Susceptibility of domestic cats to infections with *Giardia lamblia* cysts and trophozoites from human sources. *J Clin Microbiol* 1985; 21:678-680.
57. Meloni BP, Lymbery AJ, Thompson RCA. Isoenzyme electrophoresis of 30 isolates of *Giardia* from humans and felines. *Am J Trop Med Hyg* 1988;38:65-73.
58. Thompson RCA, Hopkins RM, Homan WL. Nomenclature and genetic groupings of *Giardia* infecting mammals. *Parasitology Today* 2000;16:210-213.
59. Richards J, Rodan I, Elston T, et al. Feline vaccine selection and administration. *Compend Cont Ed Pract Vet* 2001;23:71-80.
60. Olson ME, Ceri H, Morch DW. *Giardia* vaccination. *Parasitology Today* 2000;16:213-217.
61. Stein JE, Radecki SV, Lappin MR. Efficacy of *Giardia* vaccination for treatment of giardiasis in cats. *J Am Vet Med Assoc* 2003;222:1548-1551.
62. Dubey JP, Beattie CP. *Toxoplasmosis of Animals and Man*. Boca Raton, FL, CRC Press, 1988, pp 1-220.
63. Jones JL, Lopez A, Wilson M, et al. Congenital toxoplasmosis: a review. *Obstet Gynecol Surv* 2001;56:296-305.
64. Dubey JP, Lappin MR. Toxoplasmosis and neosporosis. In, Greene CE (ed), *Infectious diseases of the dog and cat*. WB Saunders Co, 2nd edition, Philadelphia, 1998, pp 493-503.
65. Teutsch SM, Juranek DD, Sulzer A, et al. Epidemic toxoplasmosis associated with infected cats. *New Eng J Med* 1979;300:695-699.
66. Benenson MW, Takafuji ET, Lemon SM, et al. Oocyst-transmitted toxoplasmosis associated with ingestion of contaminated water. *New Eng J Med* 1982; 307:666-669.
67. Aramini JJ, Stephen C, Dubey JP, et al. Potential contamination of drinking water with *Toxoplasma gondii* oocysts. *Epidem Infect* 1999;122:305-315.
68. Dubey JP. Duration of immunity to shedding *Toxoplasma gondii* oocysts by cats. *J Parasitol* 1995;81:410-415.
69. Cook AJ, Gilbert RE, Buffolano W, et al. Sources of *Toxoplasma* infection in pregnant women: European multicentre case-control study. *BMJ* 2000;321:142-147.
70. Wallace MR, Rossetti RJ, and Olson PE. Cats and toxoplasmosis risk in HIV-infected adults. *J Am Med Assoc* 1993; 269:76-77.
71. Lappin MR, George JW, Pedersen NC, et al. Primary and secondary *Toxoplasma gondii* infection in normal and feline immunodeficiency virus infected cats. *J Parasitol* 1996;82:733-742.
72. Lappin MR. Feline toxoplasmosis: interpretation of diagnostic test results. *Seminars Vet Med Surg* 1996;11:154-160.
73. Morbidity and Mortality Weekly Report. August 20, 1999, 48:1-59. RR10.
74. Fox JG. *Campylobacter* infections. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 226-229.

75. Shen Z, Feng Y, Dewhirst FE, et al. Coinfection of enteric *Helicobacter* spp. and *Campylobacter* spp. in cats. *J Clin Microbiol* 2001;39:2166-2172.
76. Baker J, Barton MD, Lanser J. *Campylobacter* species in cats and dogs in South Australia. *Aust Vet J* 1999;77:662-6.
77. Hald B, Madsen M. Healthy puppies and kittens as carriers of *Campylobacter* spp. with special reference to *Campylobacter upsaliensis*. *J Clin Microbiol* 1997;35:3351-3352.
78. Holt PE. The role of dogs and cats in the epidemiology of human campylobacter enterocolitis. *J Sm An Pract* 1981;22:681-685.
79. Hopkins RS, Olmsted R, and Istre GR. Endemic *Campylobacter jejuni* infection in Colorado: Identified risk factors. *Am J Pub Hlth* 1984;74:249-250.
80. Deming MS, Tauxe RV, Blake PA, et al. *Campylobacter* enteritis at a university: transmission from eating chickens and from cats. *Am J Epidem* 1987;126:526-534.
81. Gurgan T, Diker KS. Abortion associated with *Campylobacter upsaliensis*. *J Clin Microbiol* 1994;32:3093-3094.
82. Neiger R, Simpson KW. *Helicobacter* infection in dogs and cats: facts and fiction. *J Vet Int Med* 2000;14:125-133.
83. Simpson K, Neiger R, DeNovo R, et al. The relationship of *Helicobacter* spp. infection to gastric disease in dogs and cats. *J Vet Int Med* 2000;14:223-227.
84. Handt LK, Fox JG, Dewhirst FE, et al. *Helicobacter pylori* isolated from the domestic cat: public health implications. *Infect Immun* 1994;62:2367-2374.
85. El-Zaatari FAK, Woo JS, Badr A., et al. Failure to isolate *Helicobacter pylori* from stray cats indicated that *H. pylori* in cats may be an anthroponosis-an animal infection with a human pathogen. *J Med Microbiol* 1997;46:372-376.
86. Dieterich C, Wiesel P, Neiger R, et al. Presence of multiple "*Helicobacter heilmannii*" strains in an individual suffering from ulcers and his two cats. *J Clin Microbiol* 1998;36:1366-1370.
87. Thomas DR, Salmon RL, Meadows D, et al. Incidence of *Helicobacter pylori* in farmworkers and the role of zoonotic spread. *Gut* 1995;37(S):A24 (abstract).
88. Ansorg R, Heintschel von Heinnegg E, et al. Cat owners' risk of acquiring a *Helicobacter pylori* infection. *Zentralbl Bakteriol* 1995;283:122-126.
89. Neiger R, Schmassmann A, Seidel KE. Antibodies against *Helicobacter pylori* and *Helicobacter felis* in veterinarians. *Gastroentrol Int* 1998;11:127 (abstract).
90. Webb PM, Knight T, Elder J, et al. Is *Helicobacter pylori* transmitted from cats to humans? *Helicobacter* 1996;1:79-81.
91. Tan J. Human zoonotic infections transmitted by dogs and cats. *Arch Intern Med* 1997;157:1933-1943.
92. Tauni MA, Osterlund A. Outbreak of *Salmonella typhimurium* in cats and humans associated with infection in wild birds. *J Small Anim Pract* 2000;41:339-341.
93. Dow SW, Jones RL, Henik RA, et al. Clinical features of salmonellosis in cats: Six cases (1981-1986). *J Am Vet Med Assoc* 1989;194:1464-1466.
94. Foley JE, Orgad U, Hirsh DC, et al. Outbreak of fatal salmonellosis in cats following use of a high-titer modified-live panleukopenia virus vaccine. *J Am Vet Med Assoc* 1999;214:67-70.
95. Greene CE. Salmonellosis. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 235-240.
96. Urban JE, Broce A. Flies and their bacterial loads in greyhound dog kennels in Kansas. *Current Microbiol* 1998;36:164-170.

97. Low JC, Tennant B, and Munro D. Multiresistant *Salmonella typhimurium* DT104 in cats. *Lancet* 1996;348:1391-1392.
98. Wall PG, Davis S, Threlfall EJ, et al. Chronic carriage of multidrug resistant *Salmonella typhimurium* in a cat. *J Sm An Pract* 1995;36:279-281.
99. Wall PG, Threlfall EJ, Ward LR, et al. Multiresistant *Salmonella typhimurium* DT104 in cats: A public health risk. *Lancet* 1996;348:471-472.
100. Regnery RL, Anderson BE, Clarridge JE III, et al. Characterization of a novel *Rochalimaea species*, *R. henselae* sp. nov., isolated from blood of a febrile, human immunodeficiency virus-positive patient. *J Clin Microbiol* 1992;30:265-274.
101. Clarridge JE, Raich TJ, Pirwani D, et al. Strategy to detect and identify *Bartonella* species in routine clinical laboratory yields *Bartonella henselae* from human immunodeficiency virus - positive patient and unique *Bartonella* strain from his cat. *J Clin Microbiol* 1995;33:2107-2113.
102. Dehio C and Sander A. *Bartonella* as emerging pathogens. *Trends Microbiol* 1999;7:226-228.
103. Droz S, Chi B, Horn E, et al. *Bartonella koehlerae* sp. nov., isolated from cats. *J Clin Microbiol* 1999;37:1117-22.
104. Pretorius AM, Kelly PJ. An update on human bartonellosis. *Cent Afr J Med* 2000;46:194-200.
105. Breitschwerdt EB, Kordick DL. *Bartonella* infection in animals: carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. *Clin Microbiol Rev* 2000;13:428-38.
106. Kordick DL, Hilyard EJ, Hadfield TL, et al. *Bartonella clarridgeiae*, a newly recognized zoonotic pathogen causing inoculation papules, fever, and lymphadenopathy (cat scratch disease). *J Clin Microbiol* 1997;35:1813-1818.
107. Heller R, Artois M, Xemar V, et al. Prevalence of *Bartonella henselae* and *Bartonella clarridgeiae* in stray cats. *J Clin Microbiol* 1997;35:1327-1331.
108. Bergmans AMC, Schellekens JFP, van Embden JDA, et al. Predominance of two *Bartonella henselae* variants among cat-scratch disease patients in the Netherlands. *J Clin Microbiol* 1996;34:254-260.
109. Kumasaka K, Arashima Y, Yanai M, et al. Survey of veterinary professionals for antibodies to *Bartonella henselae* in Japan. *Japan J Clin Pathol* 2001;49:906-910.
110. Jameson PH, Greene CE, Regnery RL, et al. Prevalence of *Bartonella henselae* antibodies in pet cats throughout regions of North America. *J Infect Dis* 1995;172:1145-114.
111. 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.
112. Foley JE, Chomel B, Kikuchi Y, et al. Seroprevalence of *Bartonella henselae* in cattery cats: Association with cattery hygiene and flea infestation. *Vet Qrtly* 1998;20:1-5.
113. Higgins JA, Radulovic S, Jaworski DC, et al. Acquisition of the cat scratch disease agent *Bartonella henselae* by cat fleas (*Siphonaptera: Pulicidae*). *J Med Entomol* 1996;33:490-495.
114. Finkelstein JL, Brown TP, O'Reilly KL, et al. Studies on the growth of *Bartonella henselae* in the cat flea (*Siphonaptera: Pulicidae*). *J Med Entomol* 2002;39:915-919.

115. Regnery RL, Rooney JA, Johnson AM, et al. Experimentally induced *Bartonella henselae* infections followed by challenge exposure and antimicrobial therapy in cats. *Am J Vet Res* 1996;57:1714-1719.
116. Greene CE, McDermott M, Jameson PH, et al. *Bartonella henselae* infection in cats: evaluation during primary infection, treatment, and rechallenge infection. *J Clin Microbiol* 1996;34:1682-1685.
117. Chomel BB, Kasten RW, Floyd-Hawkins K, et al. Experimental transmission of *Bartonella henselae* by the cat flea. *J Clin Microbiol* 1996;34:1952-1956.
118. Kordick DL, Papich MG, and Breitschwerdt EB. Efficacy of enrofloxacin or doxycycline for treatment of *Bartonella henselae* or *Bartonella clarridgeiae* infection in cats. *Antimicrob Ag & Chemo* 1997;41:2448-2455.
119. Guptill L, Slater L, Ching-Ching W, et al. Experimental infection of young specific pathogen-free cats with *Bartonella henselae*. *J Inf Dis* 1997;176:206-216.
120. Mikolajczyk MG, O'Reilly KL. Clinical disease in kittens inoculated with a pathogenic strain of *Bartonella henselae*. *Am J Vet Res* 2000;61:375-9
121. O'Reilly KL, Bauer RW, Freeland RL, et al. Acute clinical disease in cats following infection with a pathogenic strain of *Bartonella henselae* (LSU16). *Infect Immun* 1999;67:3066-72
122. Ueno J, Hohdatsu T, Muramatsu Y, et al. Does coinfection of *Bartonella henselae* and FIV induce clinical disorders in cats? *Microbiol Immunol* 1996;40:617-620.
123. Lappin MR, Black JC. *Bartonella* spp. associated uveitis in a cat. *J Am Vet Med Assoc* 1999;214:1205-1207.
124. Lappin MR, Jensen W, Kordick DL, et al. *Bartonella* spp. antibodies and DNA in aqueous humor of cats. *Fel Med Surg* 2000;2:61-68.
125. Lappin MR. Infectious causes of fever in cats. *J Vet Int Med* 2002;16:366.
126. Jensen WA, Fall MZ, Rooney J, et al. Rapid identification and differentiation of *Bartonella* species using a single-step PCR assay. *J Clin Microbiol* 2000;38:1717-22.
127. Pretorius AM, Kelly PJ, Birtles RJ, Raoult D. Isolation of *Bartonella henselae* from a serologically negative cat in Bloemfontein, South Africa. *J S Afr Vet Assoc* 1999;70:154-155.
128. Morbidity and Mortality Weekly Report, August 20, 1999, 48 (RR10);1-59.
129. Bass JW, Freitas BC, Freitas AD, et al. Prospective randomized double blind placebo-controlled evaluation of azithromycin for treatment of cat-scratch disease. *Pediatr Inf Dis J* 1998;17:1059-1061.
130. Talan DA, Citron DM, Abrahamian FM, et al. Bacteriologic analysis of infected dog and cat bites. *New Eng J Med* 1999;340:84-92.
131. Carpenter PD, Heppner BT, Gnann JW. DF-2 bacteremia following cat bites. Report of two cases. *Am J Med* 1987;82:621.
132. Valtonen M, Lauhio A, Carlson P, et al. *Capnocytophaga canimorsus* septicemia: Fifth report of a cat-associated infection and five other cases. *Eur J Clin Microbiol Infect Dis* 1995;14:520-523.
133. Drabick JJ, Gasser RA, Saunders NB, et al. *Pasteurella multocida* pneumonia in a man with AIDS and nontraumatic feline exposure. *Chest* 1993;103:7-11.
134. Bonilla HF, Chenoworth CE, Tully JG, et al. *Mycoplasma felis* septic arthritis in a patient with hypogammaglobulinemia. *Clin Infect Dis* 1997; 24: 222-225.
135. McCabe SJ, Murray JF, Ruhnke, HL, et al. *Mycoplasma* infection of the hand acquired from a cat. *J Hand Surg* 1987;12:1085-1088.

136. Krebs JW, Noll HR, Rupprecht CE. Rabies surveillance in the United States during 2001. *J Am Vet Med Assoc* 2002;221:1690-1701.
137. Centers for Disease Control and Prevention (CDC). Human rabies prevention—United States, 1999. Recommendations of the Advisory Committee on Immunization Practices (ACIP). *Morb Mortal Wkly Rep* 1999;48(RR-1)1-21.
138. Trevejo RT. Rabies Preexposure vaccination among veterinarians and at-risk staff. *J Am Vet Med Assoc* 2000;217:1647-1650.
139. Jenkins SR, Auslander M, Conti L, et al. Compendium of Animal Rabies Prevention and Control. *J Am Vet Med Assoc* 2003;222:156-161.
140. Hendrick MJ, Goldschmidt MH: Do injection site reactions induce fibrosarcomas in cats? *J Am Vet Med Assoc* 199;968, 1991.
141. Hendrick MJ, Goldschmidt MH, Shofer F, et al: Postvaccinal sarcomas in the cat: Epidemiology and electron probe microanalytical identification of aluminum. *Cancer Res* 52:5391-5394, 1992.
142. Hendrick MJ, Shofer FS, Goldschmidt MH, et al: Comparison of fibrosarcomas that developed at vaccination sites and at nonvaccination sites in cats: 239 cases (1991-1992). *J Am Vet Med Assoc* 205:1425-1429, 1994.
143. Kass PH, Barnes WG, Spangler WL, et al: Epidemiologic evidence for a causal relation between vaccination and fibrosarcoma tumorigenesis in cats. *J Am Vet Med Assoc* 193;203:396-405.
144. Greene CE, Dressen DW. Rabies. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 114-126.
145. Butera ST, Brown J, Callahan ME, et al. Survey of veterinary conference attendees for evidence of zoonotic infection by feline retroviruses. *J Am Vet Med Assoc* 2000;217:1475-1479.
146. Morgan RA, Dornsife RE, Anderson WF, et al. In vitro infection of human bone marrow by feline leukemia viruses. *Virology* 1993;193:439-442.
147. Sarma PS, Huebner RJ, Basker JF et al. Feline leukemia and sarcoma viruses susceptibility of human cells to infections. *Science* 1970;168:1098-1100.
148. Markowitz LE, Hynes NA, de la Cruz P, et al. Tick-borne tularemia: An outbreak of lymphadenopathy in children. *J Am Med Assoc* 1985;254:2922-2925.
149. Rohrbach BW. Tularemia. *J Am Vet Med Assoc* 1988;193:428-432.
150. Baldwin CJ, Panciera RJ, Morton RJ, et al. Acute tularemia in three domestic cats. *J Am Vet Med Assoc* 1991;199:1602-1605.
151. Rhyan JC, Gahagan T, and Fales WH. Tularemia in a cat. *J Vet Diag Invest* 1990;2:239-241.
152. Woods JP, Crystal MA, Morton RJ, et al. Tularemia in two cats. *J Am Vet Med Assoc* 1998;212:81-83.
153. Capellan J, Fong IW. Tularemia from a cat bite: Case report and review of feline-associated tularemia. *Clin Infect Dis* 1993;16:472-475.
154. Hoskins JD, Williams J, Roy AF, et al. Isolation and characterization of *Bordetella bronchiseptica* from cats in southern Louisiana. *Vet Immunol Immunopathol* 1998;65:173-176.
155. Binns SH, Dawson S, Speakman AJ, et al. Prevalence and risk factors for feline *Bordetella bronchiseptica* infection. *Vet Rec* 1999;144:575-580.
156. Dawson S, Jones D, McCracken CM, et al. *Bordetella bronchiseptica* infection in cats following contact with infected dogs. *Vet Rec* 2000;146:46-48.

157. Coutts AJ, Dawson S, Binns S, et al. Studies on natural transmission of *Bordetella bronchiseptica* in cats. *Vet Microbiol* 1996;48:19-27.
158. Welsh RD. *Bordetella bronchiseptica* infections in cats. *J Am Anim Hosp Assoc* 1996;32:153-158.
159. Stefanelli P, Mastrantonio P, Hausman SZ, et al. Molecular characterization of two *Bordetella bronchiseptica* strains isolated from children with coughs. *J Clin Microbiol* 1997;35:1550-1555.
160. Garcia San Miguel L, Quereda C, Martinez M, et al. *Bordetella bronchiseptica* cavitory pneumonia in a patient with AIDS. *Eur J Clin Microbiol Infect Dis* 1998;17:675-676
161. Gomez L, Graziutti M, Sumoza D, et al. Bacterial pneumonia due to *Bordetella bronchiseptica* in a patient with acute leukemia. *Clin Infect Dis* 1998;26:1002-1003.
162. Dworkin MS, Sullivan PS, Buskin SE, et al. *Bordetella bronchiseptica* infection in human immunodeficiency virus - infected patients. *Clin Infect Dis* 1999;28:1095-1099.
163. Sykes JE. Feline upper respiratory tract pathogens: *Chlamydophila felis*. *Compend Cont Ed Pract Vet* 2001;23:231-241.
164. Yan C, Fukushi H, Matsudate H, et al. Seroepidemiological investigation of feline chlamydiosis in cats and humans in Japan. *Microbiol Immunol* 2000;44:155-160.
165. Bialasiewicz AA, Jahn GJ. Ocular findings in *Chlamydia psittaci*-induced keratoconjunctivitis in the human. *Fortschr Ophthalmol* 1986;83:629-631.
166. Schmeer N, Jahn GJ, Bialasiewicz AA, et al. The cat as a possible source for *Chlamydia psittaci*-induced keratoconjunctivitis in the human. *Tierarztl Prax* 1987;15:201-204.
167. Hartley JC, Stevenson S, Robinson AJ, et al. Conjunctivitis due to *Chlamydophila felis* (*Chlamydia psittaci* feline pneumonitis agent) acquired from a cat: case report with molecular characterization of isolates from the patient and cat. *J Inf* 2001;43:7-11.
168. Ostler HB, Schacter J, Dawson R. Acute follicular conjunctivitis of epizootic origin. *Arch Ophthalmol* 1969;82:587-591.
169. Cotton MM and Partridge MR. Infection with feline *Chlamydia psittaci*. *Thorax* 1998;53:75-76.
170. Griffins PD, Lechler RI, Treharne JD. Unusual chlamydial infection in a human renal allograft recipient. *Br Med J* 1978;277:1264-1265.
171. Regan RJ, Dathan JRE, Treharne JD. Infective endocarditis with glomerulonephritis associated with cat chlamydia (*C. psittaci*) infection. *Br Heart J* 1979;42:349-352.
172. Cooperman SM. Cherchez le chien-household pets as reservoirs of persistent or recurrent streptococcal sore throats in children. *NY State J Med* 1982;82:1685-1687.
173. Crowder HR, Dorn CR, Smith RE. Group A streptococcus in pets and group A streptococcal diseases in man *Int J Zoonoses* 1978;5:45-54.
174. Greene CE and Prescott JF. Streptococcal and other gram-positive bacterial infections. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 205-214.
175. Mayer G, Van Ore S. Recurrent pharyngitis in family of four. *Postgrad Med* 1982;74:277-279.
176. Eidson M, Thilsted JP, and Rollag OJ. Clinical, clinicopathologic and pathologic features of plague in cats: 119 cases (1977-1988). *J Am Vet Med Assoc* 1991;199:1191-1197.

177. Macy DW. Plague. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 295-300.
178. Gage KL, Dennis DT, Orloski KA, et al. Cases of cat-associated human plague in the Western US, 1977-1998. *Clin Infect Dis* 2000;30:893-900.
179. Foil CS. Dermatophytosis. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 362-370.
180. Woodgyer AJ. Asymptomatic carriage of dermatophytes by cats. *NZ Vet J* 1977;25:67-69.
181. Romano R, Valenti L, and Barbara R. Dermatophytes isolated from asymptomatic stray cats. *Mycoses* 1997; 40: 471-472.
182. King D, Cheever LW, Hood A, et al. Primary invasive cutaneous *Microsporium canis* infections in immunocompromised patients. *J Clin Microbiol* 1996;34:460-462.
183. Morriello KA, DeBoer DJ. Feline dermatophytosis: recent advances and recommendations for therapy. *Vet Clin North Am Small Animal Pract* 1995;25:901-921.
184. Davies C and Troy GC. Deep mycotic infections in cats. *J Am An Hosp Assoc* 1996; 32: 380-391.
185. Rosser EJ, Dunstan RW. Sporotrichosis. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 399-402.
186. Dunston RW, Langham RF, Reimann DA, et al. Feline sporotrichosis: a report of five cases with transmission to humans. *J Am. Acad Dermatol* 1986;15:37.
187. Randhawa AS, Dieterich WH, Jolley WB, et al. Coxiellosis in pound cats. *Feline Pract* 1974;4:37-38.
188. Higgins D and Marrie TJ. Seroepidemiology of Q fever among cats in New Brunswick and Prince Edward Island. *Ann NY Acad Sci* 1988:271-274.
189. Nagaoka H, Sugieda M, Akiyama M, et al. Isolation of *Coxiella burnetii* from the vagina of feline clients at veterinary clinics. *J Vet Med Sci* 1998;60:251-252.
190. Marrie TJ. *Coxiella burnetii* (Q Fever) pneumonia. *Clin Infect Dis* 1995;21:S253-S264.
191. Marrie TJ, Durant H, Williams JC, et al. Exposure to parturient cats: A risk factor for acquisition of Q fever in maritime Canada. *J Infect Dis* 1988;158:101-108.
192. Marrie TJ, Langille D, Papukna V, et al. Truckin' pneumonia - an outbreak of Q fever in a truck repair plant probably due to aerosols from clothing contaminated by contact with newborn kittens. *Epidem & Infect* 1989;102:119-127.
193. Marrie TJ, MacDonald A, Durant H, et al. An outbreak of Q fever probably due to contact with a parturient cat. *Chest* 1988b;93:98-103.
194. Pinsky RL, Fishbein DB, Greene CR, et al. An outbreak of cat-associated Q fever in the United States. *J Infect Dis* 1991;164:202-204.
195. Greene CE, Miller MA, Brown CA. Leptospirosis. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 272-281.
196. Agunloye CA and Nash AS. Investigation of possible leptospiral infection in cats in Scotland. *J Sm An Pract* 1996;37:126-129.
197. Dumler JS, Barbet AF, Bekker CP, et al. Reorganization of genera in the families *Rickettsiaceae* and *Anaplasmataceae* in the order *Rickettsiales*: unification of some species of *Ehrlichia* with *Anaplasma*, *Cowdria* with *Ehrlichia* and *Ehrlichia* with *Neorickettsia*, descriptions of six new species combinations and designation of *Ehrlichia*

- equi* and 'HE agent' as subjective synonyms of *Ehrlichia phagocytophila*. Int J Syst Evol Microbiol 2001;51:2145-2165.
198. Bjoersdorff A, Svendenius L, Owens JH, et al. Feline granulocytic ehrlichiosis - a report of a new clinical entity and characterisation of the new infectious agent. J Sm An Pract 1999;40:20-24.
 199. Lappin MR, Breitschwerdt EB, Jensen WA. Molecular and serological evidence of *Anaplasma phagocytophilum* infection of cats in North America. J Am Vet Med Assoc In press, 2003.
 200. Prause LC, Hawley JR, Jensen WA, et al. Prevalence of select infectious agents in dogs and cats from villages of Quintano Roo, Mexico. J Vet Int Med 2003;17:425.
 201. Shaw SE, Kenny MJ, Lerga AI, et al. A PCR-based survey of tick-borne infections in Danish cats and dogs. Proceedings of the 18th Conference of World Association for Advancement of Veterinary Parasitology, Stresa, Italy, August, 2001.
 202. Shaw SE, Kenny MJ, Lerga AI. PCR-based survey of tick-borne diseases in the UK/Ireland. European Society for Veterinary Internal Medicine, September, 2001.
 203. Greene CE, Appel MJG, Straubinger RK. Lyme borreliosis. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 282-293.
 204. Magnarelli LA, Anderson JF, Levine HR, et al. Tick parasitism and antibodies to *Borrelia burgdorferi* in cats. J Am Vet Med Assoc 1990;197:63-66.
 205. Levy SA, O'Connor TP, Hanscom JL, et al. Evaluation of a canine C6 ELISA Lyme Disease test for the determination of the infection status of cats naturally exposed to *Borrelia burgdorferi*. Vet Therapeutics 2003;4:172-177.
 206. Charpentier F, Groulade P. Probable case of ehrlichiosis in a cat. Bull Acad Vet France 1986;59:287-290.
 207. Beaufils JP, Marin-Granel J, Jumelle P. Ehrlichiosise feline: a propos de deux cas. Bull Aca Vet France 1997;70:73-80.
 208. Beaufils JP: Ehrlichiosis: clinical aspects in dogs and cats. Comp Cont Ed Pract Vet 19S:57-61, 1997.
 209. Alimony NRP, de Almeida LE, Moreira NS, et al. Ehrlichiose clinica em gato (*Felis catus*). R Bras Ci Vet 1998;5:82-83.
 210. Lappin MR. Feline ehrlichiosis. In, Greene CE (ed), Infectious Diseases of the Dog and Cat. Second edition, WB Saunders Co., Philadelphia, 1998, pp. 149-154.
 211. Stubbs CJ, Holland CJ, Reif JS, et al. Feline ehrlichiosis; literature review and serologic survey. Comp Cont Ed Pract Vet 2000;22:307-317.
 212. Breitschwerdt E, Abrams-Ogg A, Hancock S, et al. 2002. Molecular evidence of *Ehrlichia canis*-like infection in cats. J Vet Int Med 16:642-649.
 213. Beaufils JP, Breitschwerdt E, Hancock SI, et al. Ehrlichiose feline: Identification genetique de l'agent chez deux chats. Prat Med Chir Anim Comp 2002;27:235-238.
 214. Azad AF, Radulovic S, Higgins JA, et al. Flea-borne rickettsioses: Ecologic considerations. Em Infect Dis 1997; 3: 319-327.
 215. Higgins JA, Radulovic S, Schriefer ME, et al. *Rickettsia felis*: A new species of pathogenic rickettsia isolated from cat fleas. J Clin Microbiol 1996; 34: 671-674.
 216. Rolain JM, France M, Davoust B, et al. Molecular detection of *Bartonella quintana*, *B. koehlerae*, *B. henselae*, *B. clarridgeiae*, *Rickettsia felis*, and *Wolbachia pipientis* in cats fleas, France. Emerg Inf Dis 2003;9:338-342.
 217. Tannenbaum J. Medical-legal aspects of veterinary public health in private practice. Sem Vet Med Surg 1991;6:175-185.

218. Greene CE. Environmental factors in infectious disease. In, Greene CE (ed), *Infectious Diseases of the Dog and Cat*. WB Saunders Co., 2nd edition, Philadelphia, 1998, pp 673-683.