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Toxoplasmosis

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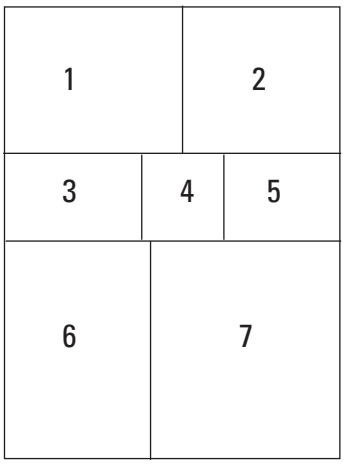

Cover. 1, Cat eating mouse, by Fabian Koster ⓒⓒⓘⓢ; 2, children play in Our Community Place sandbox, by Artaxerxes ⓒⓒⓘⓢ; 3, sea otter, by U.S. Fish and Wildlife Service; 4, transmission electron micrograph of a sporulated *Toxoplasma gondii* oocyst, Dubey and others, 1988; 5, English Leicester lambs, by Fernando de Sousa ⓒⓒⓘⓢ; 6, pregnant woman and cat, by Taylor Trimble; 7, kangaroos at Mambray Creek Camping Reserve, Mt Remarkable National Park, Australia, by Dragi Markovic & the Department of the Environment (Australia).

Toxoplasmosis

By Dolores E. Hill and J.P. Dubey

Edited by Rachel C. Abbott, Charles van Riper, III, and Elizabeth A. Enright

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U.S. Department of the Interior
SALLY JEWELL, Secretary

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Foreword

C. van Riper, III, R.C. Abbott, M. Friend, and C. Bunck

> *Let both sides seek to invoke the wonders of science instead of its terrors. Together let us explore the stars, conquer the deserts, eradicate disease, tap the ocean depths, and encourage the arts and commerce.*
> John F. Kennedy

Disease emergence in wildlife since the late 1900s has been of unprecedented scope relative to geographic areas of occurrence, wildlife species affected, and the variety of pathogens involved (Friend, 2006; Daszak and others, 2000). The emergence of many new zoonotic diseases in humans in recent years is a result of our densely populated, highly mobilized, and environmentally disrupted world. As towns and cities expand, and wildlife populations increase in numbers, the wildland-urban interface broadens, and human associations with wildlife become increasingly frequent. With geographic distance and isolation no longer meaningful barriers, the opportunities for once isolated diseases to spread have never been greater. Dealing with emerging diseases requires the ability to recognize pathogens when they first appear and to act appropriately. Since outbreaks often are evident in the nonhuman components of the environment before humans are affected, understanding our environment and associated "sentinel" wildlife is a prerequisite to protecting human health.

Increasingly, society is recognizing that parasitic zoonoses are an important component of emerging global infectious diseases (Daszak and Cunningham, 2002), not only for wildlife but for human populations. Because over 50 percent of the pathogens involved with human disease have had their origins in wild animal populations (Daszak and others, 2000; World Health Organization, 2004), there is more recognition than ever before of the need to better integrate the disciplines of human and animal health to address the phenomenon of infectious disease emergence and resurgence. Recognizing the need to document the present status of zoonotic diseases, the U.S. Geological Survey (USGS) instituted a series of USGS Circulars on emerging zoonotic diseases. This summary of toxoplasmosis is the sixth in the series.

Toxoplasmosis (*Toxoplasma gondii*), one of the better known and more widespread zoonotic diseases, originated in wildlife species and is now well established as a human malady. Food- and waterborne zoonoses, such as toxoplasmosis, are receiving increasing attention as components of disease emergence and resurgence (Slifko and others, 2000; Tauxe, 2002; Cotrovo and others, 2004). Toxoplasmosis is transmitted to humans via consumption of contaminated food or water, and the role of wildlife in this transmission process is becoming more clearly known and is outlined in this report. Currently, 9–40 percent of people in the United States and 50–80 percent in Europe are infected with *Toxoplasma gondii* (Dubey and Beattie, 1988). This zoonotic disease also causes problems in wildlife species across the globe as well as being a major cause of concern for human health. Future generations of humans will continue to be jeopardized by toxoplasmosis infections in addition to many of the other zoonotic diseases that have emerged during the past century. Through monitoring toxoplasmosis infection levels in wildlife populations, we will be better able to predict future human infection levels of this important zoonotic disease.

> *In examining disease, we gain wisdom about anatomy and physiology and biology. In examining the person with disease, we gain wisdom about life.*
> Oliver Sacks

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Conversion Factors and Abbreviations

Temperature in degrees Celsius (°C) may be converted to degrees Fahrenheit (°F) as follows:

°F=(1.8×°C)+32

AIDS	Acquired immunodeficiency syndrome
BMT	Bone marrow transplant
CDC	Centers for Disease Control and Prevention
CDV	Canine distemper virus
DAT	Direct agglutination test
DNA	Deoxyribonucleic acid
DT	Dye test
ELISA	Enzyme-linked immunosorbent assay
Gray (Gy)	The gray (Gy) is a measurement unit of the absorbed dose by matter of ionizing radiation
IAAT	Immunoabsorbent agglutination test
IFAT	Indirect fluorescent antibody test
Ig	Immunoglobulin
IHAT	Indirect hemagglutination test
IHC	Immunohistochemistry
KELA	Kinetic enzyme-linked immunosorbent assay
LAT	Latex agglutination test
MAT	Modified agglutination test
µm	The micrometer, or micron, is a measurement unit of linear distance equal to one millionth (10^{-6}) of a meter
NAHMS	National Animal Health Monitoring System
NOP	National Organic Program
PCR	Polymerase chain reaction
PASH	Periodic acid Schiff hematoxylin
RFLP	Restriction fragment length polymorphisms
TEM	Transmission electron micrograph
WB	Western blot test

Words in **bold** type in the text, the topic highlight boxes, and the tables are defined in the Glossary.

Toxoplasmosis

By Dolores E. Hill[1] and J.P. Dubey[1]

"Before the discovery of the oocyst in 1970, who would have thought that we would be living in a universe of *Toxoplasma*." (Dubey, 2010)

Synonyms

Litter box disease

Overview

Toxoplasmosis is a **zoonotic** protozoal **disease** of humans and animals caused by the **coccidian parasite**, *Toxoplasma gondii* (Nicole and Manceaux, 1909). Infection by *T. gondii* is widely **prevalent** in humans, and nearly one-third of humanity has been exposed to this parasite (Dubey and Beattie, 1988; Montoya and Liesenfeld, 2004; Dubey, 2010). Although *T. gondii* usually causes only mild disease or **asymptomatic infection** in **immune**-competent adults, it can cause devastating disease in congenitally infected children and in adults and children with depressed immunity. *T. gondii* utilizes **felids** as **definitive hosts** and has an unusually wide **intermediate host** range. Many species of **domestic** and wild animals, including **birds**, can be infected (table 1). This broad spectrum of intermediate hosts contributes to *T. gondii* being one of the most common **parasitic** infections of humans and other **warm-blooded** animals (Dubey and Beattie, 1988). *T. gondii* has been found worldwide from Alaska to Australia. Serologic surveys indicate that infections are common in wild pigs and **carnivores**, including bears, felids, foxes, raccoons, and skunks. **Clinical** and **subclinical** forms of toxoplasmosis have been reported in wild **cervids**, **ungulates**, **marsupials**, monkeys, and marine **mammals**. **Mortality** from toxoplasmosis has recently been reported in sea otter populations, and the disease is of growing concern as a sea otter mortality factor (Cole and others, 2000; Miller, Gardner, Kreuder, and others, 2000; Kreuder and others, 2003; Jessup and others, 2007; Thomas and others, 2007). Toxoplasmosis is a common cause of fetal death and abortion in sheep and goats. Adult goats and **swine** also are subject to serious illness. **Central nervous system** signs are common manifestations of *T. gondii* infection in cats and dogs (Dubey and Beattie, 1988).

Background

T. gondii is transmitted via the **fecal-oral route**, as well as through consumption of infected meat and by **transplacental** transfer from mother to fetus (Frenkel and others, 1970; Dubey and Beattie, 1988). Although cats are the definitive host for *T. gondii*, a wide range of other warm-blooded animals serves as intermediate hosts (Frenkel and others, 1970). These hosts often have **infective** stages of *T. gondii* in their tissues, thereby serving as sources for infection when their flesh is consumed raw or undercooked. Because *T. gondii* is one of the most common parasites of animals, consumption of infected meat contributes to the growing importance of toxoplasmosis as a zoonotic disease. For example, a recent study in the Slovak Republic found *T. gondii* infection to be common in wild boars, emphasizing the need to maintain high standards of hygiene during the handling of this important game species when it is prepared as food (Antolová and others, 2007).

T. gondii was initially discovered in the gundi (Nicolle and Manceaux, 1908), a small **rodent** that inhabits rocky areas on hills and mountains of the northern part of the African continent (Walker, 1964). About the same time, independent discovery of this parasite was made in a laboratory rabbit in São Paolo, Brazil (Splendore, 1908; Dubey, 2008). In retrospect, these widely geographically separated discoveries were somewhat of a bellwether relative to the broad geographic distribution of *T. gondii* and the variety of species it would be found to infect. It was another 30 years before *T. gondii* was found to cause disease in humans and an additional 47 years after that discovery before the full life cycle for this parasite was determined (box 1).

[1]U.S. Department of Agriculture, Agricultural Research Services, Animal Parasitic Diseases Laboratory.

Table 1. General summary of nonhuman species naturally infected with *T. gondii.*

Species type	Minimum number of species[1]
Domestic animals	
Livestock	8
Poultry	3
Cats	1
Dogs	1
Wild animals	
Ungulates	25
Carnivores	8
Felids	10
Marsupials	20
Marine mammals	10
Birds	11
Small mammals (rodents/lagomorphs)	9
Monkeys	18
Bears	3

[1]The number of species indicated is merely a minimum; more species may have been infected but have not been reported. The numbers should not be interpreted relative to each other, because some groups may be overrepresented due to higher susceptibility to clinical or fatal disease or both.

The name *Toxoplasma* (toxon = arc, plasma = form) is derived from the crescent shape of one of the three **infectious** stages of the parasite, the tachyzoite stage (fig. 1*A*). This stage rapidly multiplies in intermediate host cells and in nonintestinal **epithelial** cells of the definitive host. Bradyzoites (fig. 1*B*) and sporozoites (fig. 1*C*) are the other infectious stages. In contrast to tachyzoites, bradyzoites multiply slowly within a tissue **cyst**, while sporozoites are generated by sexual processes in the definitive host intestine and develop to maturity within oocysts released into the environment in cat feces (Dubey, 2008).

Past scientific literature suggests that different species of *Toxoplasma* have been identified over time (Keymer, 1981; Levine, 1985). However, modern technology has clarified the speciation of **protozoans** by replacing **morphology** and other physical characteristics with molecular descriptions. It is now generally accepted that there is only one species of *Toxoplasma*, *T. gondii*.

Strain differences exist that may affect the **pathogenicity** of the parasite in a given host (Dubey, 2010). Prior to the development of genetic markers, *T. gondii* **isolates** were grouped by their **virulence** to outbred mice. During the 1980s and 1990s, methods were developed to recognize genetic differences among *T. gondii* isolates from humans and animals (Pfefferkorn and Pfefferkorn, 1980; Dardé and others, 1988; Tibayrenc and others, 1991; Sibley and others,1992; Howe and Sibley, 1995; Dardé, 2008). Based on deoxyribonucleic acid (**DNA**) restriction fragment length polymorphisms (**RFLP**), Howe and Sibley (1995) classified *T. gondii* into three genetic Types (I, II, III) and linked virulence in mice to genetic type. They proposed that Type I isolates were 100 percent lethal to mice, irrespective of the dose, and that Types II and III generally were avirulent for mice (Howe and others, 1996). Circumstantial evidence suggests that certain genetic types of *T. gondii* may be associated with clinical **ocular** toxoplasmosis in humans (Khan and others, 2006). It has been suggested that Type I isolates or **recombinants** of Types I and III are more likely than Type II isolates to result in clinical toxoplasmosis, but genetic characterization has been limited essentially to isolates from patients ill with toxoplasmosis (Khan and others, 2005). There is very little information regarding the genetic diversity of *T. gondii* isolates circulating in the general human population. Therefore, we must be cautious in claiming a linkage between parasite **genotypes** and disease presentations without clear and discerning information regarding the parasite's biology in the human population and environment.

Acquisition of direct oral transmission by *T. gondii* appears to be a recent evolutionary change achieved by **recombination** between competing, distinct clonal lines of the parasite. This route of transmission has facilitated widespread distribution of *T. gondii* (Montoya and Liesenfeld, 2004).

100 Years of *Toxoplasma gondii* Box 1

DISCOVERY

1905 — *T. gondii* in gundi in Tunisia and a laboratory rabbit in Brazil are discovered simultaneously.

1910

1915

1920 — *T. gondii* is suspected in the eye of a child with **hydrocephalus**.

1925

1930

CONGENITAL TRANSMISSION

1935 — Congenital transmission is documented in humans.

DIAGNOSTIC TEST

1940

1945 — Clinical and parasitological aspects of congenital toxoplasmosis are characterized in humans.
Sabin and Feldman develop a dye test, the most specific serological test for *T. gondii*.

TRANSMITTAL BY MEAT

1950 — Transmission by raw and undercooked meat is suggested.

1955

1960 — The **resistance** of *T. gondii* from tissue cysts to proteolytic enzymes and its ability to survive to infect a host are demonstrated.

1965 — **Epidemiological** evidence that *T. gondii* is transmitted by ingestion of raw and undercooked meat is found.
T. gondii in feline feces is discovered.

ROLE OF CATS

1970 — The sexual phase of *T. gondii* in the small intestine of a cat is discovered.
Definitive and intermediate hosts are defined. The role of cats is confirmed from studies on remote islands.

1975

IMPACT ON AIDS

1980 — Toxoplasmic encephalitis is one of the leading causes of death in **AIDS** patients.

1985

FOOD-BORNE INFECTIONS

1990 — The high **prevalence** of *T. gondii* infection in pigs (approximately 20 percent) destined for human consumption is demonstrated.

1995 — Highly Active Anti-Retroviral Therapy (HAART) for AIDS patients is implemented, reducing the death rate from Toxoplasmic encephalitis.

2000 — Large-scale movement to confinement rearing by the swine industry results in significant reduction in *T. gondii* prevalence in pigs.

2005 — Food-borne infections from consumption of raw or undercooked meats of free-range domestic animals and wild game **reemerge**.

2010

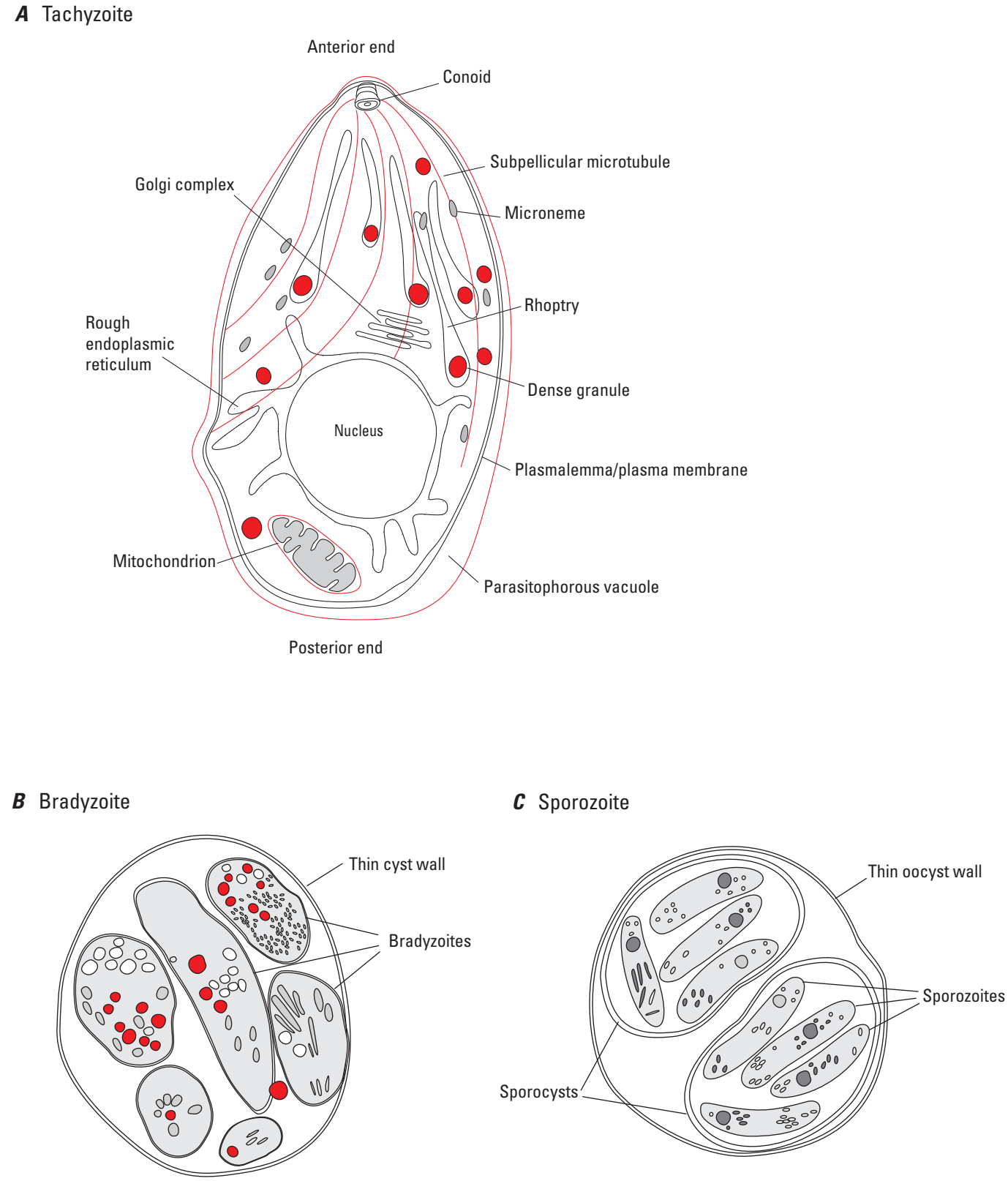


Figure 1. Infectious stages of *T. gondii*: *A*, tachyzoite, *B*, bradyzoite, and *C*, sporozoite. (*A*, Ajioka and others, 2001)

Causative Agent

T. gondii is a protozoan parasite belonging to the subclass Coccidiasina (Leuckart, 1879) (table 2). Coccidia, in general, have complicated life cycles (fig. 2), and *T. gondii* is no exception (box 2). As noted above, *T. gondii* has three infectious stages (fig. 3): the tachyzoites (in groups; fig. 3*A*), the bradyzoites (in tissue cysts; fig. 3*B*), and the sporozoites (in oocysts; fig. 3*C*).

The tachyzoite is often crescent shaped and is approximately the size of a red blood cell (fig. 4). The anterior end of the tachyzoite is pointed, and the posterior end is round. It has an outer covering, or pellicle, enclosing various **organelles**. Bradyzoites differ structurally only slightly from tachyzoites. They have a nucleus situated toward the posterior end, whereas the nucleus in tachyzoites is more centrally located. Bradyzoites are more slender than are tachyzoites and are less **susceptible** to destruction by **proteolytic enzymes** than are tachyzoites. Intact tissue cysts containing bradyzoites probably do not cause any harm and can persist for the life of the host.

Table 2. Taxonomic classification of the parasite causing toxoplasmosis.

Classification	Designation
Kingdom	Protista
Phylum	Apicomplexa
Class	Sporozoasida
Subclass	Coccidiasina
Order	Eucoccidioridia
Suborder	Eimeriorina
Family	Toxoplasmatidae
Genus	*Toxoplasma*
Species	*gondii*

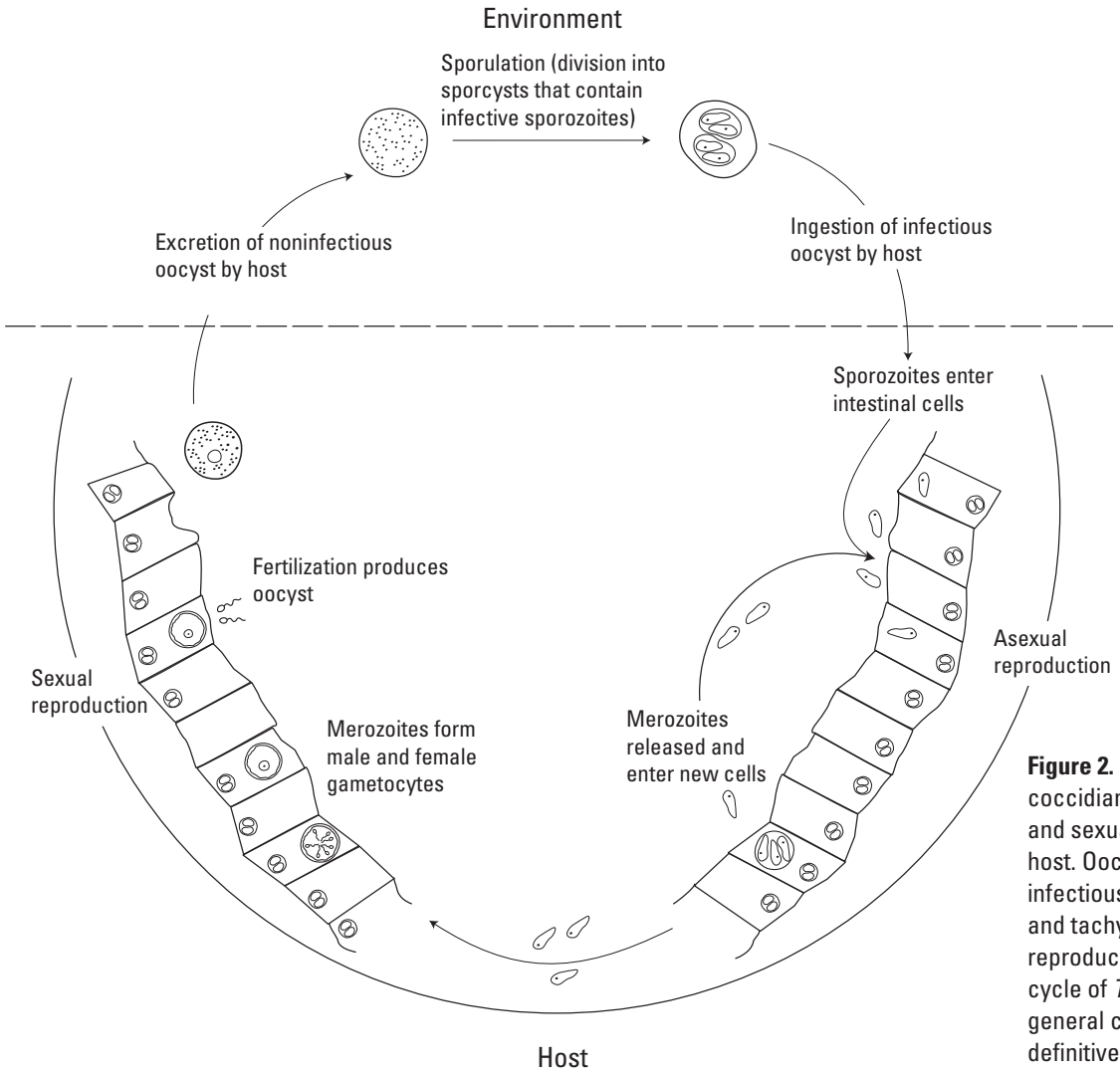
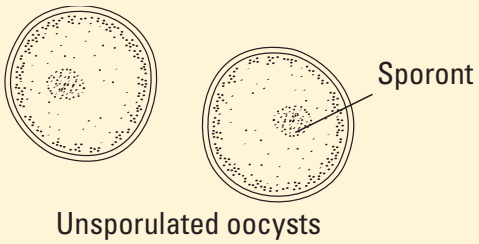


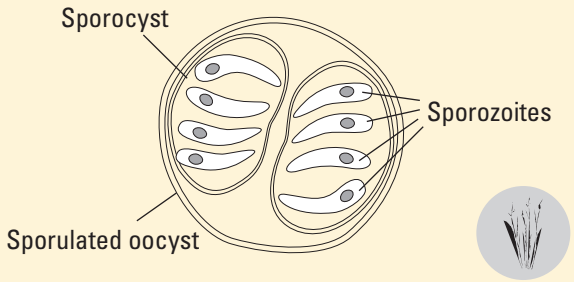
Figure 2. The life cycle of a typical coccidian parasite involves asexual and sexual reproduction within the host. Oocysts sporulate and become infectious in the environment, and tachyzoites and bradyzoites reproduce within the host. The life cycle of *T. gondii* is similar to this general coccidian life cycle in its definitive host, the cat.

Box 2 Life Cycle of *T. gondii*

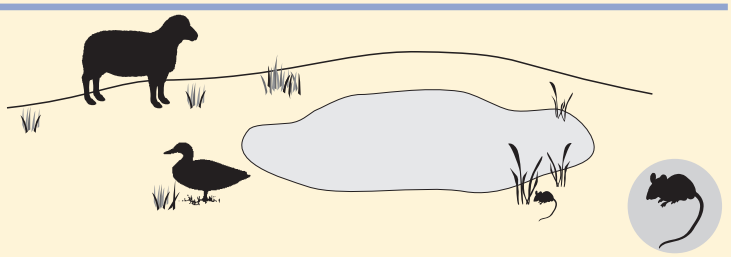
1.
Noninfective parasite oocysts (eggs) containing a single cell referred to as the sporont are passed by cats in their feces into the environment.



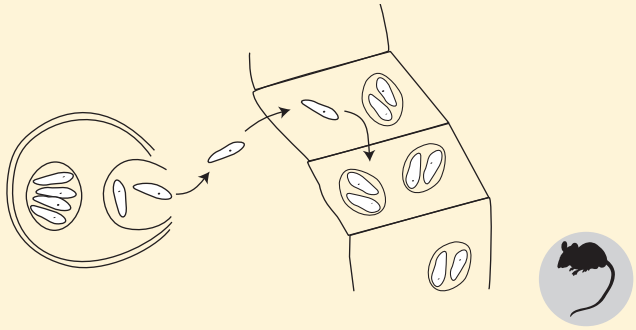
2.
Oocysts become infective within 1 to 5 days in the environment through sporulation (sporogony), which is a developmental process that results in the sporont dividing and forming two sporocysts, each containing four infective sporozoites.



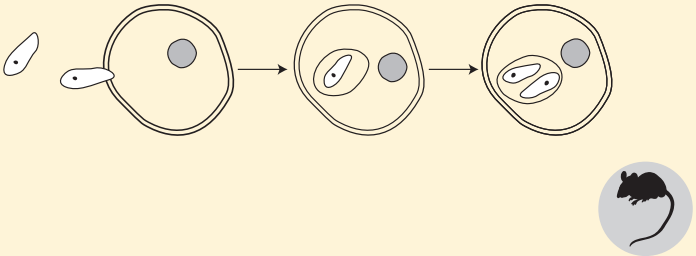
3.
Infective oocysts are ingested by intermediate hosts or humans in contaminated feed, water, soil, or other ingesta.



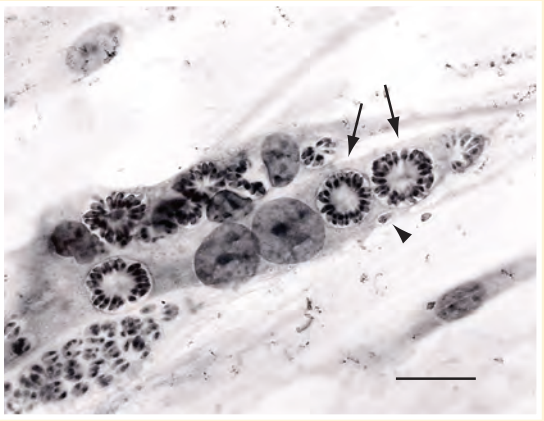
4.
In the small intestine, the sporozoites escape from the sporocysts and oocysts and enter the epithelial cells lining the internal surfaces of the intestine. The sporozoites multiply asexually by endodyogeny, resulting in the formation of tachyzoites.



5.
Tachyzoites can tilt, extend, and retract as they search for host cells, and they then enter the host cells by active penetration of the host cell membrane. After entering the host cell, the tachyzoite becomes ovoid in shape (*A*) and becomes surrounded by a **parasitophorous vacuole** in which it is protected from host defense mechanisms. Tachyzoites multiply asexually within the host cell by repeated divisions in which two progeny form within the parent parasite, consuming it.

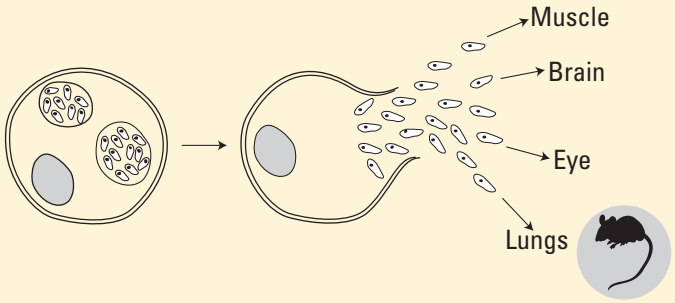


A. This photograph shows tachyzoites grown in cell culture in human foreskin fibroblasts. An ovoid tachyzoite can be seen surrounded by a vacuole (arrowhead). Also seen are groups of tachyzoites arranged in rosettes (arrows). Contrast was enhanced by the use of an immunohistochemical stain with a tachyzoite-specific polyclonal antibody. The scale bar is 20 micrometers (µm) in length. (Photo by Dr. J.P. Dubey)



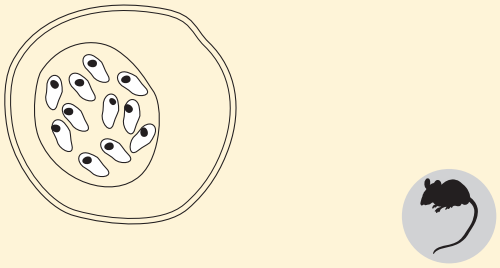
6.

Tachyzoites continue to divide until the host cell is filled with parasites. Tachyzoites accumulate in groups of 8 to 32 within the host cells and then break out of the host cell. The tachyzoites enter new host cells and undergo further divisions.

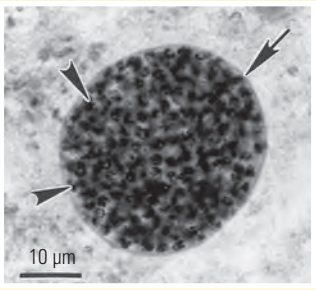


7.

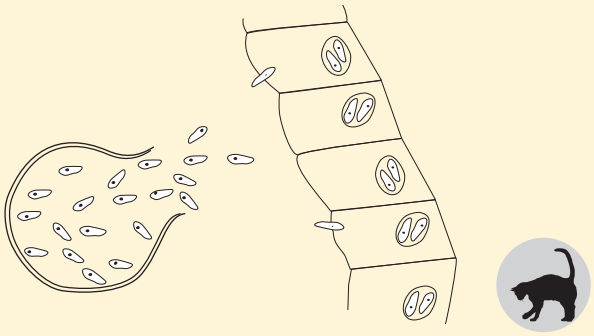
As infection becomes **chronic**, immunity builds up, and multiplication of tachyzoites slows. Bradyzoites, as they are now called, accumulate in large numbers within a host cell and become surrounded by a thin, elastic wall to form tissue cysts, which vary in size from 5 to 70 µm and remain **intracellular** (*B*). A tissue cyst may enclose hundreds of bradyzoites. Tissue cysts are most prevalent in muscular and neural tissues, including the brain, eye, skeletal, and cardiac muscle, but they may also occur in **visceral** organs, including lungs, liver, and kidneys.



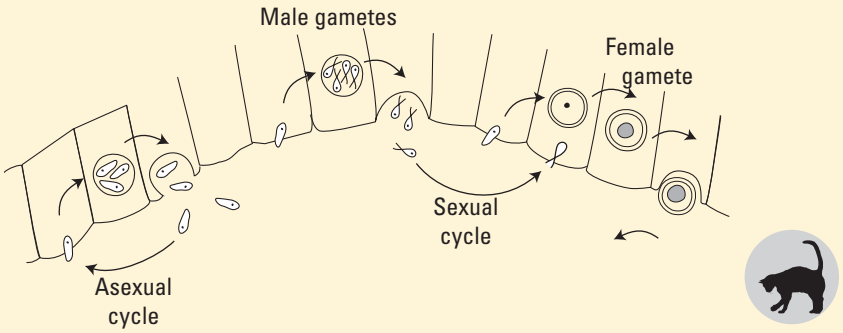
B. Tissue cyst in a section of mouse brain. Numerous bradyzoites (arrowheads) are surrounded by cyst wall (arrow). Contrast was enhanced by the use of periodic acid Schiff hematoxylin (PASH). The bradyzoites stain bright red with PAS but appear black in this photograph. The scale bar is 10 micrometers (µm) in length. (Photo from Dubey and others, 1998)



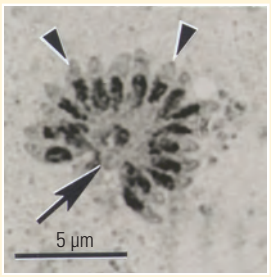
8.
After a cat ingests tissue cysts in meat, the tissue cyst wall is dissolved by proteolytic enzymes in the stomach and small intestine, thus releasing the bradyzoites. The bradyzoites then penetrate the epithelial cells of the small intestine and initiate development of numerous generations of asexual and sexual cycles of *T. gondii* (Dubey and Frenkel, 1972).



9.
Within cats, the bradyzoites multiply profusely in intestinal epithelial cells; this is known as the entero-epithelial cycle, and these stages are known as schizonts (*C*). Merozoites are released from schizonts and form male and female gametes. The male gamete (*D*) uses its two flagella to swim to the female gamete, which it then enters. After the female gamete is fertilized, oocyst wall formation begins. Three to 10 days after ingesting tissue cysts, oocysts are discharged into the intestinal lumen by the rupture of intestinal epithelial cells and released into the environment with the feces.



C. This image of an impression smear of infected cat intestine shows a schizont (arrow) with several merozoites (arrowheads) separating from the main mass. Contrast was enhanced by use of Giemsa stain. The scale bar is 5 micrometers (µm) in length. (Photo from Hill and others, 2005)



D. This image of an impression smear of infected cat intestine shows a male gamete with two flagella (arrows). Contrast was enhanced by use of Giemsa stain. The scale bar is 10 micrometers (µm) in length. (Photo from Hill and others, 2005)

10.
As the entero-epithelial cycle progresses, bradyzoites penetrate the lamina propria of the feline intestine and multiply as tachyzoites. Within a few hours after infection of cats, *T. gondii* may disseminate to extraintestinal tissues. *T. gondii* can persist in intestinal and extraintestinal tissues of cats for at least several months, and possibly for the life of the cat.

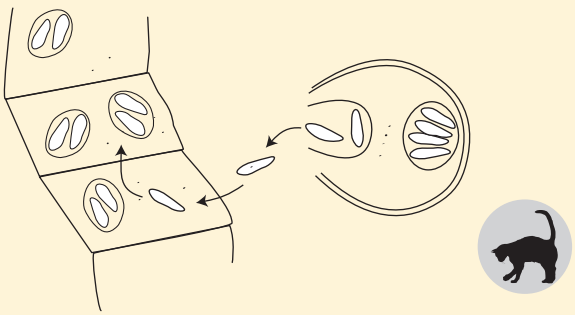


Figure 3. Stages of *Toxoplasma gondii*. *A*, Tachyzoites in an impression smear of lung. Note the crescent-shaped individual tachyzoites (arrows), dividing tachyzoites (arrowheads) compared with the size of host red blood cells and **leukocytes**. Giemsa staining was used to enhance contrast of the tissues. *B*, Tissue cyst in a section of muscle. The tissue cyst wall is very thin (arrow) and encloses many tiny bradyzoites (arrowheads). Hematoxylin and eosin staining was used to enhance contrast of the tissues. *C*, Transmission electron micrograph of a sporulated oocyst. Note the thin oocyst wall (large arrow), two sporocysts (arrowheads), and sporozoites, one of which is cut longitudinally (small arrows). (*A* and *B* from Hill and others, 2005; *C* from Dubey and others, 1998. µm, micrometer)

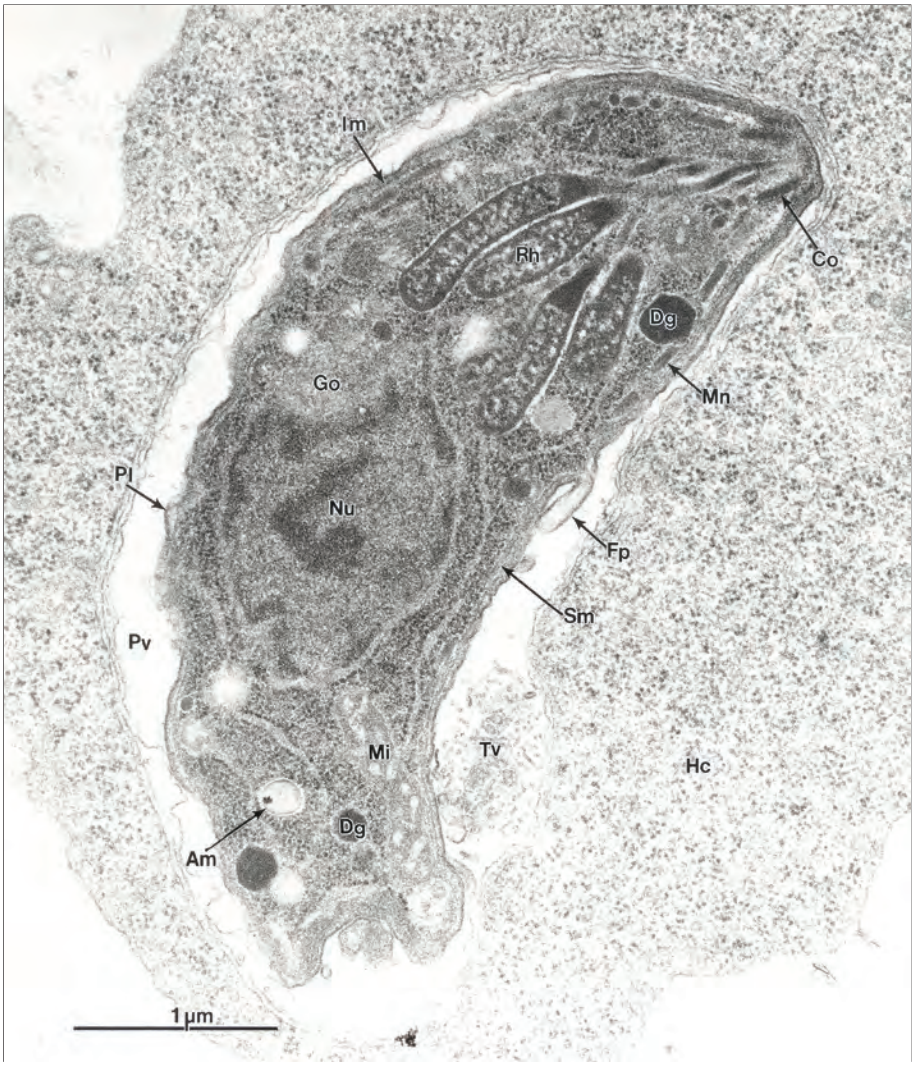


Figure 4. Transmission electron micrograph (TEM) of a tachyzoite of *T. gondii* in a mouse peritoneal exudate cell. Am (amylopectin granule), Co (conoid), Dg (electron-dense granule), Fp (finger-like projection of tachyzoite plasmalemma), Go (Golgi complex), Hc (host cell cytoplasm), Im (inner membrane complex), Mi (mitochondrion), Mn (microneme), Nu (nucleus), Pl (plasmalemma), Pv (parasitophorous vacuole), Rh (rhoptry), Sm (subpellicular microtubule), Tv (tubulovesicular membranes). (Photo courtesy of Dr. C.A. Speer, Montana State University, Bozeman, Mont. µm, micrometer)

Geographic Distribution

T. gondii infection is widespread in humans (fig. 5). Various **serologic** surveys have measured the prevalence of infection and have yielded highly variable results from place to place, in part due to differences in sampling techniques and various parameters associated with the sample **cohorts** (Dubey and Beattie, 1988; Dubey, 2010). In the United States and the United Kingdom, it is estimated that 10–40 percent of people are infected, whereas in Central and South America and continental Europe, estimates of infection range from 50 to 80 percent (Dubey and Beattie, 1988; Jones, Kruszon-Moran, and Wilson, 2007). A broader worldwide perspective of variation in the prevalence of human infection by *T. gondii* is provided by figure 5. Similar surveys have been conducted for major domestic animal species (Dubey and Beattie, 1988) and are aggregated here as a general reflection of variations in the geographic distribution of *T. gondii* in these species globally (fig. 6*A*–*E*) and between species (fig. 7).

The geographic distribution of *T. gondii* in nonhuman species is limited only by an absence of wild or domesticated felids or both to serve as definitive hosts for completion of the parasite's life cycle. Neither human nor domestic animal presence is needed for *T. gondii* to sustain itself among wild animal populations. In some instances, this parasite may be introduced into wildlife populations through the establishment of domestic animal populations in an area, or as a result of transient human presence accompanied by infected domestic cats, or both. For example, domestic cats now inhabit isolated oceanic land areas after being left behind by humans harvesting guano deposits from marine birds (Bailey and Niederach, 1951). Large-scale surveys have not been completed to determine the extent of infection in marine birds; however, fatal toxoplasmosis in endangered Hawaiian **corvids** and other native birds has been reported (Work and others, 2000; Work and others, 2002). A high percentage of domestic cats tested worldwide have **antibodies** to *T. gondii* (fig. 6*F*). Infection rates increase with the age of the cat and also are greater in **feral** cats than those in domestic cats (Dubey and Beattie, 1988). Serological evaluations of domestic dogs disclose worldwide prevalence of infection ranging from 1 to 58 percent (fig. 6*G*). The prevalence of infection by *T. gondii* in wildlife is noted elsewhere in this chapter (*see* Species Susceptibility).

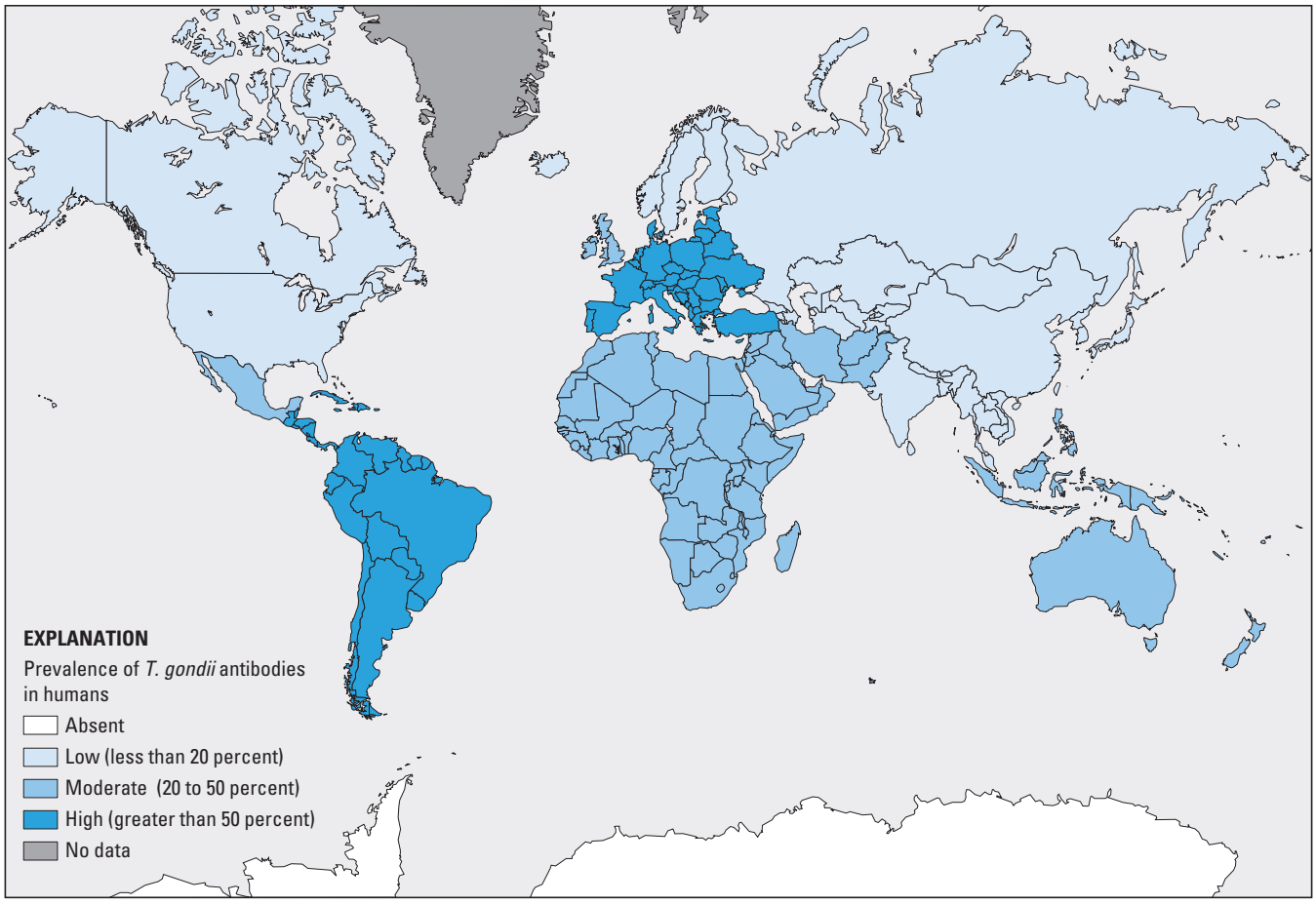


Figure 5. Geographic distribution of the prevalence of *T. gondii* antibodies in humans. (Data from Dubey and Beattie, 1988)

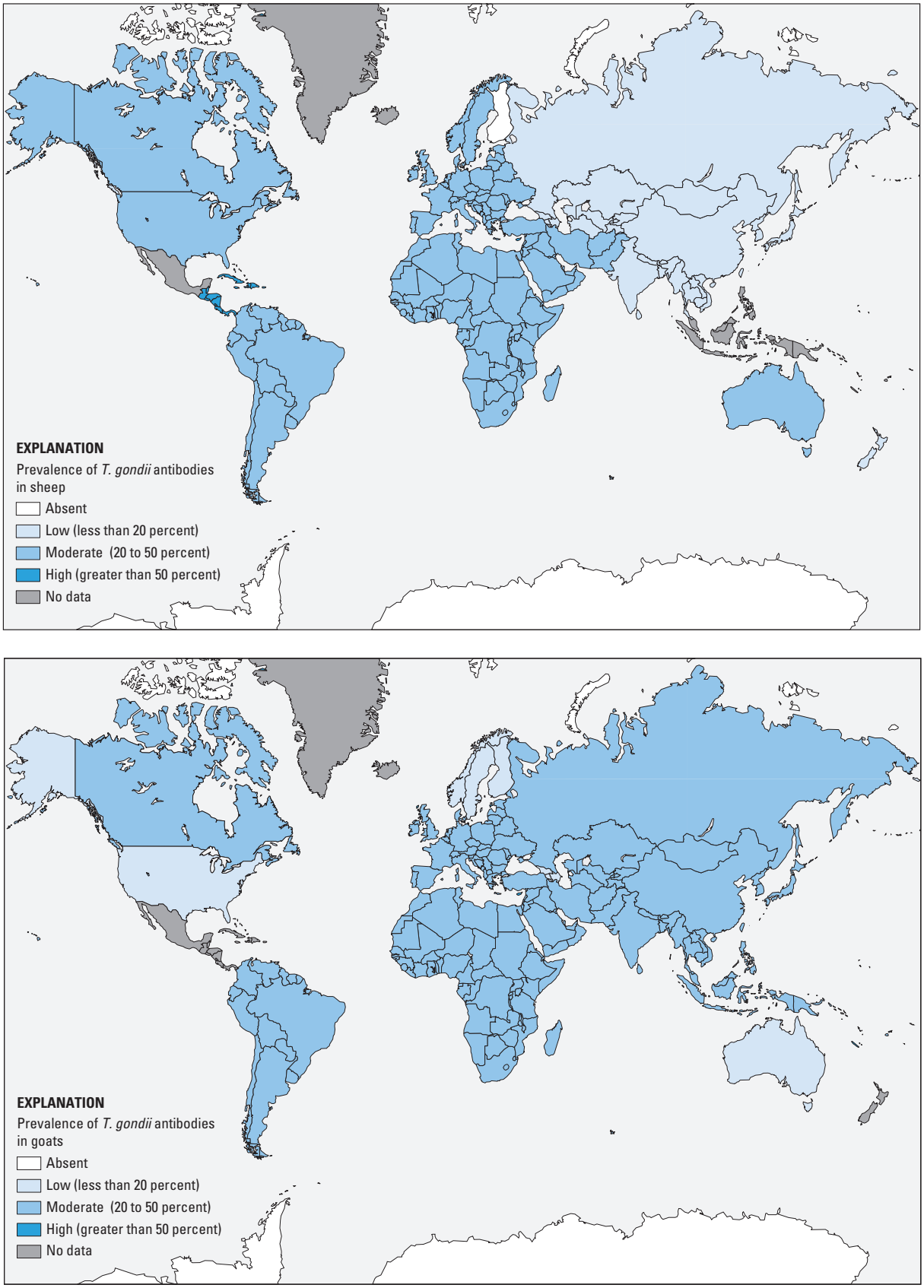


Figure 6. Geographic distribution of the prevalence of *T. gondii* antibodies in animals: *A*, sheep; *B*, goats. (Data from Dubey and Beattie, 1988)

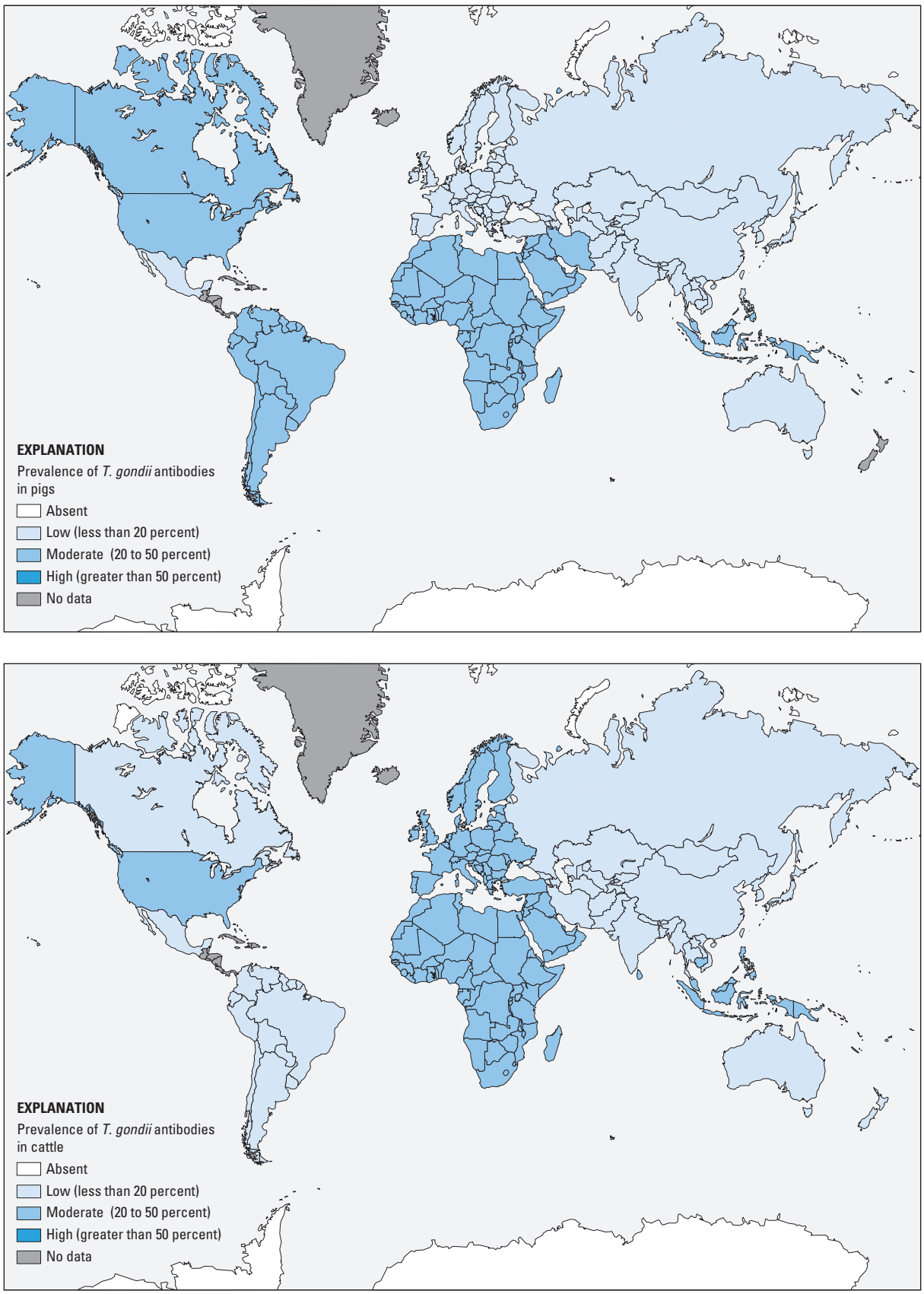


Figure 6. Geographic distribution of the prevalence of *T. gondii* antibodies in animals: *C*, pigs; *D*, cattle. (Data from Dubey and Beattie, 1988)

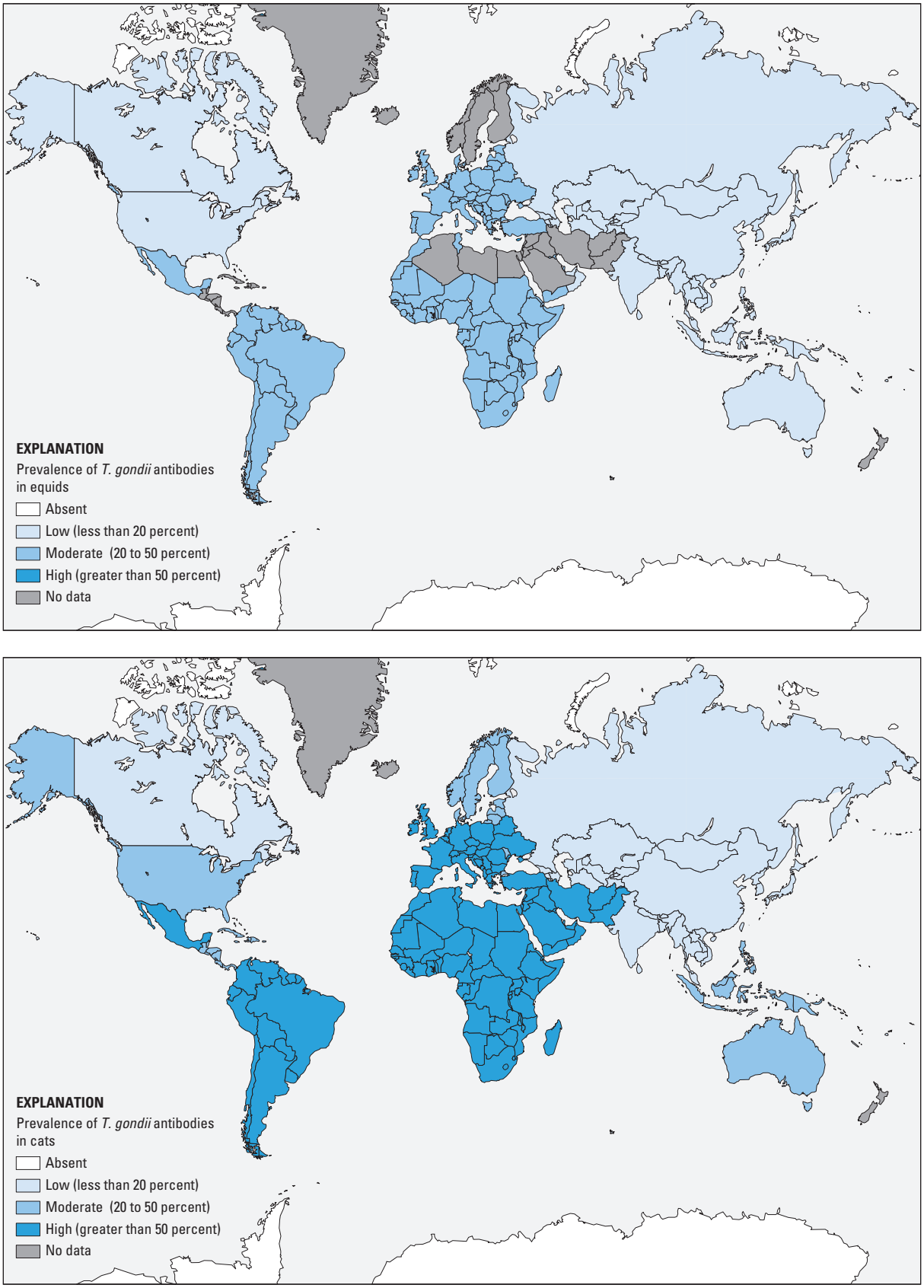


Figure 6. Geographic distribution of the prevalence of *T. gondii* antibodies in animals: *E*, **equids**; *F*, cats. (Data from Dubey and Beattie, 1988)

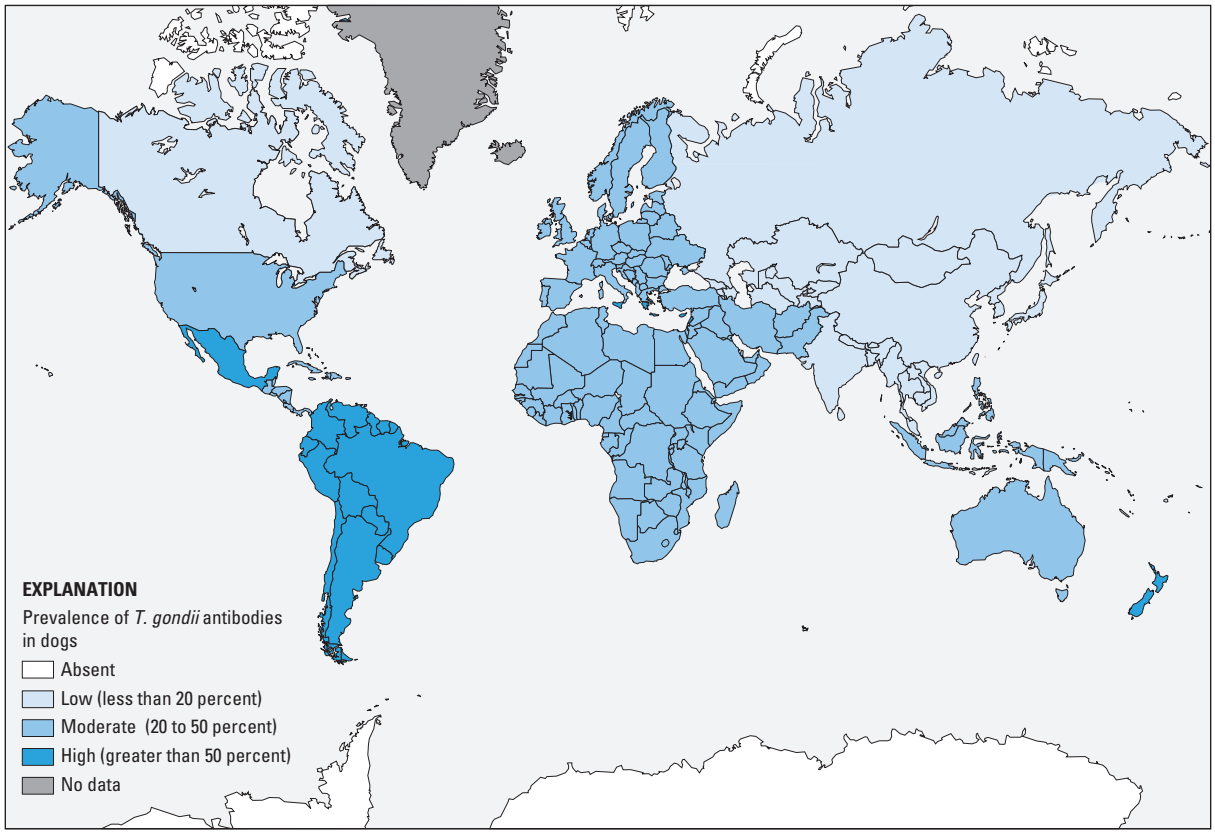


Figure 6. Geographic distribution of the prevalence of *T. gondii* antibodies in animals: *G*, dogs. (Data from Dubey and Beattie, 1988)

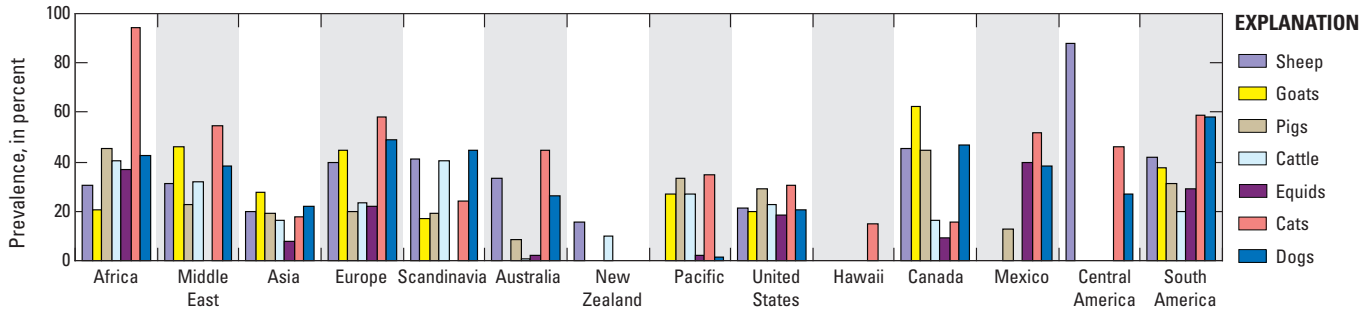


Figure 7. Prevalence of *T. gondii* in domestic animals by region. (Data from Dubey and Beattie, 1988)

Patterns and Trends

Toxoplasmosis is not a nationally reportable disease in the United States. Thus, there are no precise data for the number of cases diagnosed each year at the national level. An estimated 400–4,000 cases of **congenital** toxoplasmosis occur each year and 750 deaths are attributed to this disease annually (Hughes and Colley, 2000). The Centers for Disease Control and Prevention (CDC) considers **seroprevalence** data derived from the National Health and Nutrition Examination Survey to be the most reliable U.S. estimates of infection by *T. gondii*. The first of those evaluations involved samples from the period 1988–94 (Hughes and Colley, 2000; table 3), and the second involved samples from the period 1999–2000 (Jones and others, 2003). Surveys have demonstrated that seroprevalence remained stable at 23 percent from 1990 to 1998 (Jones and others, 2001). Recent surveys have demonstrated a significant decrease in seroprevalence to 10.8 percent over the last decade (Jones, Kruszon-Moran, and others, 2007). Earlier serological studies of military recruits sampled in the 1960s, and again in 1989, indicated a decline in prevalence (Hughes and Colley, 2000). Declines in human infection rates have also been reported from evaluations in France (Jeannel and others, 1988) and Sweden (Forsgren and others, 1991).

The prevalence of *T. gondii* antibodies in humans from earlier U.S. data ranges from 0 percent in a small sample of 21 Alaskan Eskimos to a high of 39 percent for a sample of 265 individuals from the State of Maryland (fig. 8) (Dubey and Beattie, 1988). Samples from 8 of the 21 data sets in that evaluation exceeded 25 percent prevalence. Worldwide, *T. gondii* is reported to infect up to one-third of the world's population (Montoya and Liesenfeld, 2004). Geographical comparisons in prevalence among these data sets are confounded by variables such as laboratory assays used, sample size (from 21 to 95,929), sex, ethnic composition of the population sampled, age distribution, and other considerations. For example, well-established global trends show a steady increase in the prevalence of *T. gondii* antibodies with age; the greater the prevalence of infection, the earlier the rise in the infection rate (fig. 9) (Dubey and Beattie, 1988). Nevertheless, causes for different infection rates in humans and animals in different geographic areas of a country remain to be elucidated (Dubey, 2010).

According to Jones and others (2003), "Predicting future trends in *T. gondii* prevalence in the United States is difficult because we do not have a national estimate of what proportion of *T. gondii* infections are attributed to undercooked meat exposure or to cat feces, soil, or water exposure." Other general factors that likely influence trends for infection and the geographic differences in infection rates include environmental conditions, cultural habits of the people, and animal fauna (Dubey and Beattie, 1988). All of these factors are associated with a continuum of change that is accelerating due to increased globalization of society, population increases, and shifts in human demographics that have consequences for landscape changes that impact interspecies relations.

Table 3. Selected reports of *T. gondii* seroprevalence in humans in the United States.

[From Dubey and Jones, 2008. DT, dye test; **ELISA**, enzyme-linked immunosorbent assay; MAT, modified agglutination test; NHANES, National Health and Nutrition Examination Survey]

Year sampled	Age group	Source of sera	Test	Number tested	Positive tests, in percent
1962	Young adult	Military recruits	DT	2,680	14
1989	Young adult	Military recruits	MAT	2,862	9.5
1988–94	Age-adjusted ≥12 years old	NHANES	ELISA[3]	17,658	22.5
1999–2000	Age-adjusted 12–49 years[1]	NHANES	ELISA[3]	4234	15.8
1999–2004	Age-adjusted 12–49 years[2]	NHANES	ELISA[3]	15,960	10.8

[1] Women 12–49 years, 14.9 percent.

[2] Women 15–44 years, 11 percent.

[3] *Platelia* Toxo-G enzyme immunoassay kit.

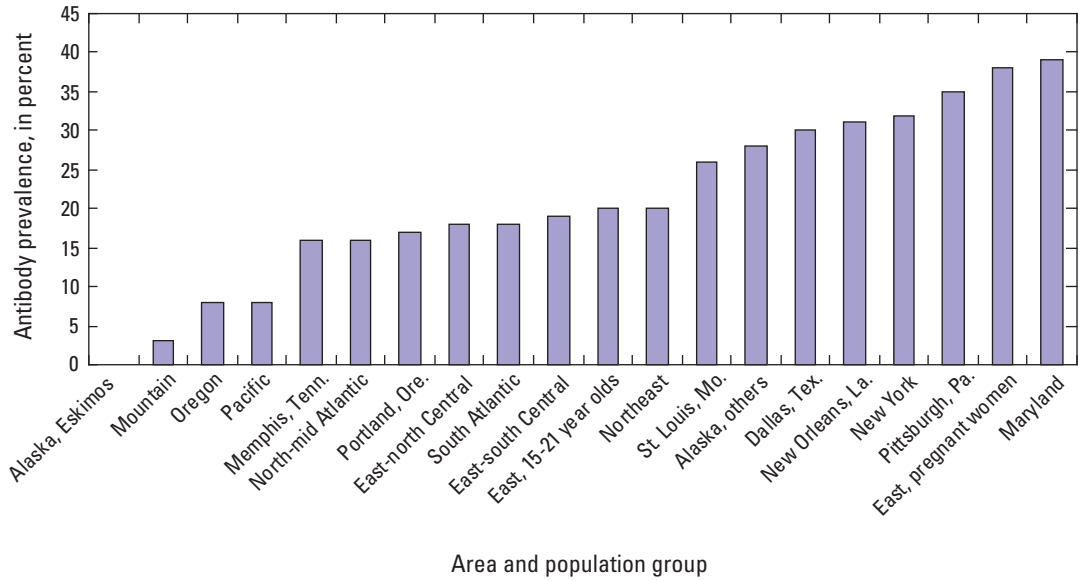


Figure 8. Prevalence of *T. gondii* antibodies in humans in the United States. (Data from Dubey and Beattie, 1988)

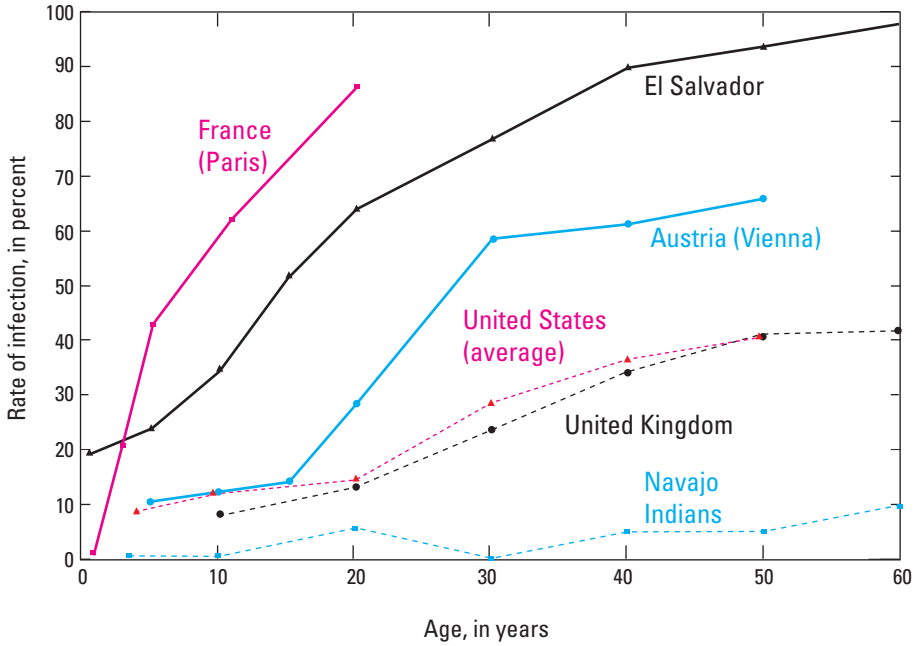


Figure 9. Prevalence of *T. gondii* infection with age in humans in various areas. (Reprinted from Dubey and Beattie, 1988, and published with permission)

Species Susceptibility

T. gondii usually parasitizes the host, definitive and intermediate, without producing clinical disease. Nevertheless, clinical cases, some of which are fatal, can occur. The clinical outcome is determined by the extent of injury to the intestine, **mesenteric lymph nodes**, and especially to vital organs such as the eye, brain, heart, and adrenal glands following invasion and intracellular growth of tachyzoites. Cellular impacts that can result include tissue **necrosis** followed by inflammation (Dubey and Beattie, 1988).

Pathogenicity of *T. gondii* is determined by the virulence of the **strain** and the **susceptibility** of the host species. For example, certain strains of mice are more susceptible than others to *T. gondii* infection, and the severity of infection in individuals within the same mouse strain may vary, though mice of any age are susceptible to clinical *T. gondii* infection. Adult rats do not become ill, but young rats can die of toxoplasmosis. Adult dogs, like adult rats, are resistant, whereas puppies are fully susceptible to clinical toxoplasmosis. Cattle and horses are among the hosts more resistant to clinical toxoplasmosis, whereas certain marsupials and **New World monkeys** are highly susceptible to *T. gondii* infection and disease (Dubey and Beattie, 1988). Little is known about the mechanisms associated with genetically determined susceptibility to clinical toxoplasmosis, including susceptibility in humans.

Human Infections

T. gondii infection is widespread in humans, though its prevalence varies widely from place to place. Most infections in humans are asymptomatic, but at times this parasite can produce debilitating disease. Infection may be congenitally or postnatally acquired. Congenital infection occurs when a woman becomes infected during pregnancy, though rare exceptions have occurred when women were infected just before pregnancy (Vogel and others, 1996). In addition, in **immunosuppressed** women reactivation of an infection acquired before pregnancy can lead to congenital toxoplasmosis (Minkoff and others, 1997; Mitchell and others, 1990; De Moura, 2006). Although the condition may be benign, its diagnosis is vital in pregnant women because of the risk to the fetus (box 3).

Postnatally acquired infection may be localized or generalized. Oocyst transmitted infections, or those directly associated with felid feces, may be more severe than tissue **cyst** induced infections, or those often associated with intermediate hosts (Teutsch and others, 1979; Benenson and others, 1982; Dubey and Beattie, 1988; Smith, 1993; Bowie and others, 1997; Burnett and others, 1998). Transfusion or organ transplantation from an infected person can also transmit the organism; transplant-associated toxoplasmosis is common enough that prophylactic treatment is standard procedure in

Table 4. Frequency of symptoms in **outbreaks** of toxoplasmosis in adult humans.

[From Dubey, 2010]

Symptom	Atlanta, Georgia, 35 patients	Panama Paraná, 35 patients	Brazil, 155 patients
	Patients with symptom in percent		
Fever	94	90	82
Enlarged **lymph nodes**	88	77	75
Headache	88	77	87
Muscle pain	63	68	80
Stiff neck	57	55	Not reported
Poor appetite	57	Not reported	69
Sore throat	46	Not reported	Not reported
Joint pain	26	29	61
Rash	23	0	7
Confusion	20	Not reported	Not reported
Earache	17	Not reported	Not reported
Nausea	17	36	38
Eye pain	14	26	Not reported
Abdominal pain	11	55	Not reported

Box 3 Pregnancy and Toxoplasmosis—A Disease of Concern

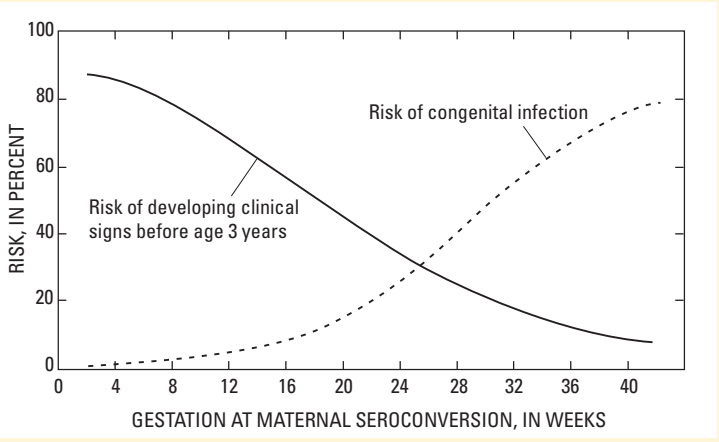
Although toxoplasmosis usually does not cause clinical illness in healthy people, it can cause debilitating disease in congenitally infected infants. Since 1937, when maternal-infant transmission of *T. gondii* was first documented by Wolf, Cowen, and Paige (1939), it has become recognized as a significant cause of disease in infants and children, as well as adults who may suffer from delayed conditions originating from congenital infections. In the United States, 400–4,000 infants are born each year with congenital toxoplasmosis (Hughes and Colley, 2000). Because about 90 percent of these infants usually do not show any signs of the disease at birth (Desmonts and Couvreur, 1974; Remington and others, 2006), the effects of the infection may not be recognized until later in childhood or adulthood. Early identification of infants at risk of infection or disease is vital to limiting the financial and social costs of people suffering the effects of the infection.

The organism is transmitted during gestation when the mother becomes infected for the first time. While the mother rarely has symptoms of infection, she does have parasites in the blood temporarily. Focal **lesions** develop in the placenta and the fetus may become infected. At first there is generalized infection in the fetus. Later, infection is cleared from the visceral tissues and may localize in the central nervous system.

The risk of congenital infection is lowest (10–15 percent) when maternal infection is during the first trimester and highest (60–90 percent) when infection is during the third trimester (Dunn and others 1999; Foulon and others, 1999; Remington and others, 2006). However, congenital infections acquired during the first trimester are more severe than those acquired during the second and third trimesters (Desmonts and Couvreur, 1974; Holliman, 1995; Remington and others, 1995; Remington and others, 2006).

A wide spectrum of pregnancy outcomes and clinical diseases can occur as a result of congenital toxoplasmosis, including spontaneous abortion and stillbirth, as well as birth defects in live born infants such as hydrocephalus (accumulation of cerebrospinal fluid in the brain), **microcephalus** (abnormally small brain for age), **intracerebral calcification**, **convulsions**, diminished vision, and **retinochoroiditis** (inflammation of the inner layers of the eye). Of these, hydrocephalus is the least common but most dramatic lesion of toxoplasmosis. By far, the most common condition resulting from congenital toxoplasmosis is ocular disease (Desmonts and Couvreur, 1974; Remington and others, 1995; Montoya and Liesenfeld, 2004; Remington and others, 2006). Retinochoroiditis and symptoms of central nervous system involvement can also develop later in life in apparently normal, but congenitally infected, children (Desmonts and Couvreur, 1974; Guerina and others, 1994; McAuley and others, 1994;

Photo courtesy of Taylor Trimble



Risk of congenital infection and development of clinical signs by duration of gestation at maternal seroconversion. (Modified from Dunn, 1999)

Dunn and others, 1999; McLeod and others, 2006; Remington and others, 2006; SYROCOT Study Group, 2007).

The socioeconomic impact of toxoplasmosis on human suffering and the cost of care of sick children, especially those with intellectual disability and blindness, are enormous (Roberts and Frenkel, 1990; Roberts and others, 1994). The testing of all pregnant women for *T. gondii* infection is compulsory in some European countries including France and Austria, which have high **incidence**s of toxoplasmosis during pregnancy (Thiebaut and others, 2007; Petersen, 2007). The goal of these screening programs is to prevent infection of **seronegative** women and to ensure early diagnosis and treatment of women infected during pregnancy to decrease transmission to their fetuses. Women are tested for antibodies prior to becoming pregnant. Seronegative women are then tested at regular intervals during pregnancy. If the tests show **seroconversion**, the woman is treated with the antibiotic spiramycin, which concentrates in the placenta, and testing of the fetus by amniocentesis or cordocentesis is performed to monitor the status of the fetus. If fetal infection is confirmed, the woman is additionally treated with the antimicrobial drugs pyrimethamine and sulfadiazine or sulfadoxine. Treatment of infected neonates begins at birth to reduce the potential complications of infection.

Seropositive women are not considered to be at risk of transmitting *T. gondii* to their fetuses unless they are **immunocompromised** (Montoya and Liesenfeld, 2004; Remington and others, 2006). Women who have given birth to an infant congenitally infected with *T. gondii* can be reassured that subsequent pregnancies will not result in infected babies except in very rare cases.

The cost benefits of mass screening are being debated in many other countries (Cortina-Borja and others, 2010; Remington and others, 1995; 2005; McLeod and others, 2009). Recently, Stillwaggon and others (2011) provided extensive guidelines for estimating costs of preventive maternal screening for and the social costs resulting from toxoplasmosis based on studies in Europe and the United States. While estimating these costs, it is important to consider the value of all resources used or lost, including the cost of medical and non-medical services, wages lost, cost of in-home care, indirect costs of psychological impacts borne by the family for lifetime care of a substantially cognitively impaired child, and the cost of a fetal death (estimated to be $5 million) (Olariu and others, 2011; Stillwaggon and others, 2011). Because the costs of these programs are high, such screening is not routinely done in most countries. In areas with low incidence of congenital toxoplasmosis, pregnant women are advised to practice careful hygiene during food preparation, gardening, and cat care to decrease the risk of becoming infected with *T. gondii* during pregnancy. This type of primary prevention has been shown to reduce the incidence of congenital transmission by up to 60 percent (Foulon and others, 1994). Routine newborn screening tests to increase early diagnosis and treatment of infected infants even if they are not showing any **clinical signs** is practiced in some countries/regions, including the United States. Other regions, such as Denmark, Poland, and Massachusetts, have begun including toxoplasmosis as part of routine newborn screening tests to increase early diagnosis and treatment of infected infants even if they are not showing any clinical signs (Guerina and others, 1994; Holliman, 1995; Foulon and others, 2000).

Although congenital toxoplasmosis can be a devastating and costly disease, women should not become alarmed. Practicing good hygiene during pregnancy is quite effective at decreasing the risk of infection and has no adverse effects for the mother or fetus.

Conditions resulting from congenital toxoplasmosis.

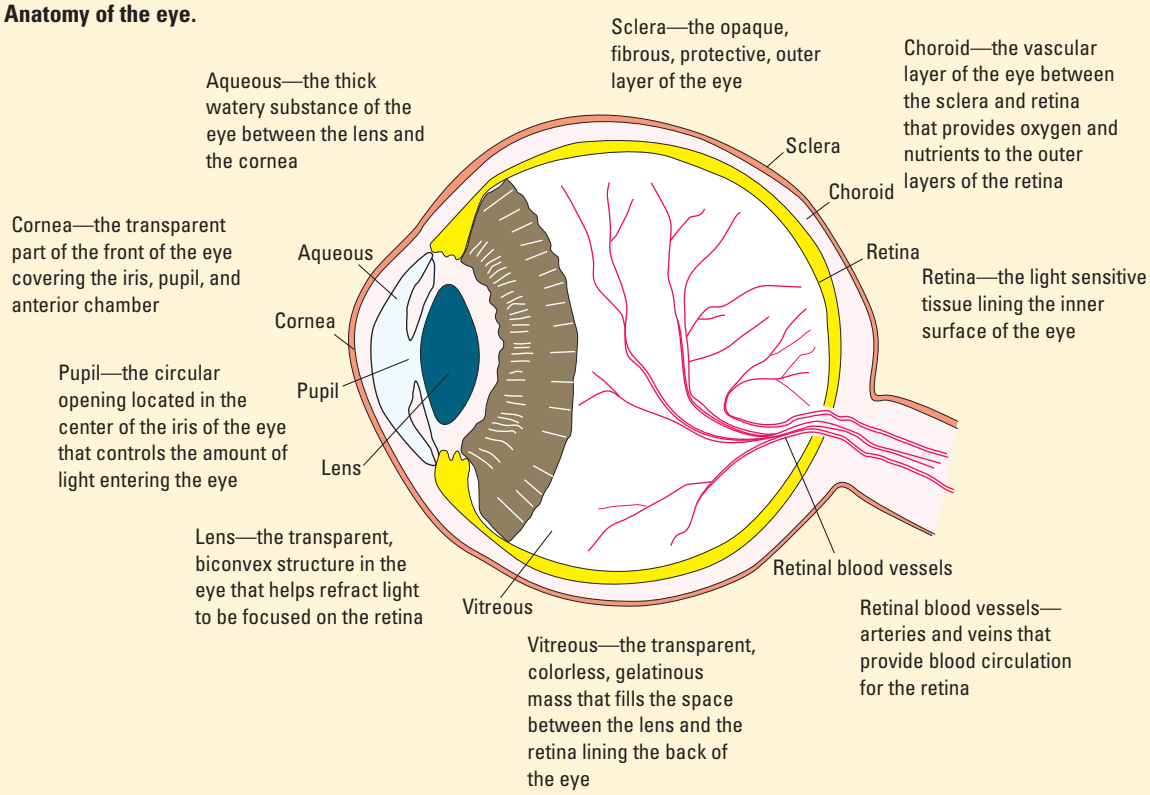
[Age distribution of patients: 119 patients 4 years old or less; 38 patients 5–9 years old; 19 patients 10–19 years old. From Feldman and Miller, 1956]

Condition	Patients with condition, in percent
Retinochoroiditis—an inflammation of the inner layers of the eye.	94
Intracerebral calcification—the accumulation of calcium in the cerebrum of the brain.	59
Psychomotor retardation—a slowing of thought and reduced movement.	45
Seizures.	39
Microphthalmia—a deformation resulting in abnormally small eyes.	36
Hydrocephalus—the accumulation of cerebrospinal fluid in the cavities of the brain.	22
Microcephaly—a significantly smaller head than the average for an individual's age and sex.	21

Box 4 Toxoplasmosis and Eye Disease

Based on an estimate that up to 2 percent of persons with *T. gondii* infection in the United States have ocular lesions (Holland, 2003), as many as 1.26 million persons in the United States may have ocular toxoplasmosis (based on the 2000 census, Holland, 2003). A higher percentage of infected persons has been documented to develop ocular disease in other parts of the world; 17.7 percent of infected individuals developed ocular lesions in one region of Southern Brazil, perhaps due to the virulence of the *T. gondii* types present there (Glasner and others, 1992; Dubey and others, 2012). A U.S. national survey of ophthalmologists produced estimates of 250,000 visits to ophthalmologists for active or inactive ocular toxoplasmosis during a 2-year period (Lum and others, 2005). In a British Columbia outbreak, of 100 people who were diagnosed with acute infection, 51 had **lymphadenopathy** (swollen, enlarged lymph nodes) and 20 had **retinitis** (inflamed **retina**) (Aramini and others, 1998; 1999). Most ocular toxoplasmosis is now believed to result from postnatally acquired disease (Holland, 1999; 2003), contrary to what was generally accepted prior to the 1990s, when almost all cases of this disease were believed to be a result of congenital infection (Perkins, 1973).

Anatomy of the eye.



"*T. gondii* remains the most common **pathogen** to infect the retina in otherwise healthy individuals" (Holland, 1999). In seven studies in the United States and Europe, toxoplasmosis was diagnosed as the most common cause of posterior **uveitis**, which involves inflammation of the retina and **choroid**; approximately 10 percent of uveitis cases were attributed to toxoplasmosis (Merrill and others, 1997). Toxoplasmic retinochoroiditis, from both congenital and postnatally acquired infection, results from acute infection or reactivation of previous lesions that had resolved, leaving residual retinochoroidal scars with variable amounts of pigmentation (Montoya and Remington, 1996; Holland, 1999; Holland 2004).

Patients suffering from retinochoroiditis, regardless of its cause, present with symptoms of eye pain, **sensitivity** to light or photophobia, tearing, blurred or diminished vision, and dark, floating spots in the visual field. These symptoms may appear acutely at the time of initial infection or may not occur until months to years later, when previous lesions become reactivated (Holland, 1999). The diagnosis of recurrent toxoplasmic retinochoroiditis may be made by an ophthalmologist by the appearance of "satellite" lesions at the border of preexisting retinochoroidal scars. The recurrence of toxoplasmic retinochoroiditis has been explained by reactivation of live tissue cysts at the edges of the scars. The multiplying parasites are released from the tissue cyst and cause inflammation and tissue damage, which lead to symptoms and signs of retionchoroiditis. In some cases, tissue cysts can exist in areas of the retina without obvious abnormalities (Holland, 1999).

The factors that lead to the reactivation of retinal tissue cysts remain unclear, but they may include age-related changes in the parasites themselves or host hormonal or **immunological** changes (Holland, 2003). Immunocompromised patients can have multiple active retinal lesions in both eyes in contrast to only one area of active disease in immunocompetent patients (Holland, 2004). Lesions tend to recur with progressive loss of vision over time, especially when the lesions are near the central structures of the eye (Holland, 2003). Because blindness may eventually result, prevention of recurrences is especially important; secondary prophylaxis with antimicrobials may be an effective management tool (Holland, 2004).

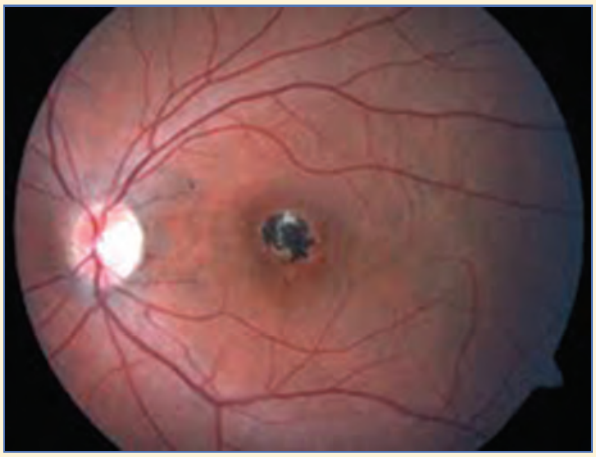


Photo of retinal scar (pigmented area) from acquired toxoplasmosis. (Photo courtesy of Steven M. Cohen, MD, Retina Vitreous Associates of Florida)

The most common treatment for ocular toxoplasmosis is a combination of the antimicrobial drugs pyrimethamine and sulfadiazine with prednisone, a corticosteroid used to treat inflammation. Although the effectiveness of this treatment method remains unproven in immunocompetent patients, it has inactivated chronically active ocular toxoplasmosis in patients with acquired immunodeficiency **syndrome** (AIDS) (Holland, 2004). Because corticosteroid treatment by itself does not prevent severe tissue destruction, concurrent use of antimicrobials is warranted (Holland, 2004). Because of the high numbers of people who develop ocular toxoplasmosis and the uncertainties in the pathogenesis and treatment of the disease, prevention of infection by *T. gondii* remains especially important in combating this disease.

Box 5 Toxoplasmosis in Immunocompromised People

Although *T. gondii* most often causes asymptomatic infection in healthy people, it has the potential to cause serious life-threatening disease in immunocompromised patients, in whom it may appear as an acute, **disseminated** infection or one involving only a single organ. The most common form of toxoplasmosis seen in immunocompromised patients is encephalitis, but the heart and lungs can also be infected (Ruskin and Remington, 1976; Emerson and others, 1981). Because **morbidity** and mortality from toxoplasmosis are so severe in immunocompromised patients, preventive measures and early diagnosis are crucial. Among those at increased risk of developing toxoplasmosis are people being treated with **immunosuppressive agents** for organ or bone marrow transplants and people suffering from the immunosuppressive effects of cancer or acquired immunodeficiency syndrome (AIDS).

Organ Transplants

Administration of immunosuppressive agents prior to organ transplantation is standard procedure for reducing the risk of organ rejection by the recipient. Unfortunately, this immunosuppression puts recipients at risk of becoming infected by agents carried by the donor. In the case of toxoplasmosis, the risk of developing serious disease is greatest for recipients who are seronegative for *T. gondii* and who are receiving organs from donors **seropositive** for *T. gondii* (Wreghitt and others, 1989; Renoult and others, 1997; Schaffner, 2001). For heart transplants, approximately 50 percent of seronegative recipients who are not given prophylactic medications acquire toxoplasmosis from seropositive donors (Schaffner, 2001). This rate is lower for patients undergoing liver and kidney transplants, 20 percent and less than 1 percent, respectively (Schaffner, 2001). Toxoplasmosis has also been transmitted to one patient by intestinal transplantation (Campbell and others, 2006). Disease results from the reactivation of **latent** cysts within the donor tissue. Patients may present with symptoms of fever, headache, confusion, and lethargy, as well as signs of **pneumonitis** (inflamed lung tissue), **myocarditis** (inflammation of the heart muscle), or both, usually within 3 months of the transplant (Renoult and others, 1997; Luft and others, 1983). Mortality rates are high; in one study, 64 percent of patients who developed toxoplasmosis after kidney transplant died (Renoult and others, 1997).

Bone Marrow Transplants

Although rare, toxoplasmosis can occur in patients receiving bone marrow transplants (BMT) as a result of reactivation of latent cysts in previously seropositive patients (Derouin and others, 1992; Martino, Maertens, and others, 2000; Mele and others, 2002). The incidence varies from less than 0.4 percent to 3 percent, according to levels of **endemicity** (Martino, Bretagne, and others, 2000). Most cases occur within 6 months of BMT (Derouin and others, 1992; Chandrasekar and others, 1997; Martino, Maertens, and others, 2000). The brain is the most common site of infection, followed by the heart and lung (Derouin and others, 1992; Chandrasekar and others, 1997; Martino, Maertens, and others, 2000; Mele and others, 2002). The disease is rapidly fatal, causing death in almost 90 percent of patients (Chandrasekar and others, 1997; Martino, Maertens, and others, 2000).

Cancer

Toxoplasmosis among cancer patients most often occurs in association with Hodgkin's disease, a form of lymphoma, but can also occur with other forms of lymphoma or leukemia (Israelski and Remington, 1993). Both the immune-related defects due to the cancer itself and the effects from immunosuppressive therapy probably put the patient at risk of developing toxoplasmosis. Patients with solid tumors, such as breast cancer, ovarian cancer, and lung carcinoma, may also develop toxoplasmosis as a result of bone-marrow suppression by treatment with drugs that inhibit the development of malignant cells (Ruskin and Remington, 1976; Israelski and Remington, 1993). Reactivation of latent cysts leads to clinical disease, most often seen as encephalitis (Vietzke and others, 1968; Ruskin and Remington, 1976; Israelski and Remington, 1993); fever, myocarditis, pneumonitis, and rash can also develop. Toxoplasmosis contributes to a high mortality among cancer patients not treated for the disease (Israelski and Remington, 1993).

AIDS

Toxoplasmosis ranks high on the list of diseases that lead to the death of patients with acquired immunodeficiency syndrome (AIDS); approximately 10 percent of AIDS patients in the United States and up to 30 percent in Europe are estimated to die from toxoplasmosis (Luft and Remington, 1992). Although in AIDS patients any organ may be involved, including the testes, dermis, and the spinal cord, toxoplasmic encephalitis is the most common clinical manifestation of toxoplasmosis in patients with AIDS-related immunosuppression (Dubey and Beattie, 1988; Luft and Remington, 1992; Jones and others, 1996).

Most AIDS patients suffering from toxoplasmic encephalitis have bilateral, severe, and persistent headache that responds poorly to analgesics. As the disease progresses, the headache may give way to a condition characterized by disorientation, lethargy, weakness, **hemiparesis** (partial **paralysis**), reflex changes, convulsions, **ataxia** (lack of muscle coordination), and coma. The predominant lesion in the brain is necrosis (death of cells or tissue), especially of the thalamus (Renold and others, 1992).

Around 1995, highly active antiretroviral therapy (HAART) became the treatment of choice for AIDS in western countries. Successful HAART in AIDS patients significantly reduced the incidence and deaths associated with toxoplasmic encephalitis (Jones and others, 1999; Kaplan and others 2000; Jones and others 2002; Hooshyar and others, 2007). Abgrall and others (2001) reported a decrease of 75 percent in the incidence of toxoplasmic encephalitis after the implementation of HAART in one French study. A similar decline in incidence was seen during the same time period in 11 U.S. cities (Kaplan and others, 2000).

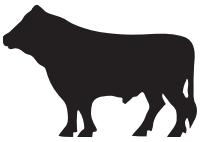
Because *T. gondii* can cause severe and often fatal disease in immunocompromised patients, early diagnosis and treatment is especially important. However, diagnosis by serological tests is often difficult and unreliable because of the altered immune function in these patients. Prior knowledge of the serostatus of a patient, and the donor in the case of organ transplants, is important for guiding decisions concerning prophylaxis and treatment. A high degree of suspicion for toxoplasmosis by physicians caring for immunocompromised patients at risk may increase early treatment, leading to decreased morbidity and mortality. In addition to medical treatment, reminders of basic hygiene to decrease primary infection by contact with cat feces and undercooked meat remain important for decreasing toxoplasmosis in this at-risk population.

heart and lung transplant recipients (Shulman and Appleman, 1991; Schaffner, 2001; Soave, 2001; Campbell and others, 2006). Enlarged lymph nodes are the most frequently observed clinical form of **acute** toxoplasmosis in humans (table 4), and this **symptom** may be associated with fever, fatigue, muscle pain, sore throat, and headache. However, ocular disease is a frequent and serious consequence of infection with *T. gondii* (box 4).

Immunocompromised individuals have a far greater risk for acquiring clinical disease from *T. gondii* than **immunocompetent** individuals, and the course of disease can be clinically severe (box 5). Recent studies have suggested a link between toxoplasmosis and **schizophrenia** (Torrey and Yolken, 2003; Yolken and Torrey, 2008). *T. gondii* infection in rodents leads to increased risk-taking behavior and loss of fear towards **feline predators** (Berdoy and others, 2000). Although numerous studies have demonstrated increased seroprevalence of anti-*T. gondii* antibodies in schizophrenic patients, a direct causal link has not been definitively shown.

Toxoplasmosis is an important food-borne disease in many countries. *T. gondii* is one of three pathogens (along with *Salmonella* and *Listeria*) that were believed to account for more than 75 percent of all deaths due to food-borne disease in the United States (Roberts and others, 1994; Buzby and Roberts, 1996; Mead and others, 1999; Scallan and others, 2011; Batz and others, 2012; Hoffmann and others, 2012; Jones and Dubey, 2012; see references below)**.** However, a recent nationwide study of fresh retail beef, pork, and chicken revealed that beef and chicken posed little risk to consumers as sources of *T. gondii*, while 0.4 percent of retail pork was infected with *T. gondii*, which could be transmitted to consumers (Dubey and others, 2005). Food- and water-borne sources of *T. gondii* infection can result in **epidemics** as well as individual infections (Bowie and others, 1997; Eng and others, 1999).

Animal Infections



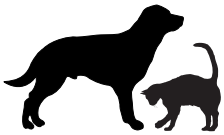
Livestock

T. gondii is capable of causing infection and severe disease in animals other than humans (Dubey and Beattie, 1988). *T. gondii* causes abortion and neonatal mortality in sheep worldwide and may cause embryonic death and resorption, fetal death and **mummification**, and stillbirth in these animals. Both isolated events and epidemics of abortion and neonatal mortality have been reported in U.S. sheep (Dubey and others, 1981; Dubey and Welcome, 1988). Congenitally infected lambs that survive the first week after birth usually grow normally, but they can be a source of infection for humans (Dubey, Sundar, and others, 2008). The prevalence of *T. gondii* in adult sheep and lambs in the United States is high; a recent survey of market lambs in the mid-Atlantic States demonstrated that 27 percent of the surveyed lambs were infected with *T. gondii*, thus posing a risk to consumers who handle or consume undercooked lamb (Dubey, Sundar, and others, 2008). Adult goats can develop clinical toxoplasmosis, and the disease is generally more severe in goats than in sheep. In contrast, cattle are considered a poor host for *T. gondii*. Although cattle can be experimentally infected with *T. gondii* oocysts, the parasite is eliminated or reduced to undetectable levels within a few weeks (Dubey, 1983a; 1986b). Antibodies to *T. gondii* were not found by enzyme-linked immunoabsorbent assay (ELISA) in meat juice from any of 2,049 samples of beef **bioassayed** during the National Retail Meat Survey for *T. gondii* (Dubey and others, 2005). Far less is known about *T. gondii* infections in other livestock species such as water buffalo, camels, and equids, though horses appear to be resistant to infection; **serology** and other assays have established infections in these species. However, there is little evidence that *T. gondii* is a recurring cause for clinical disease in any of these species (Dubey and Beattie, 1988).

Outbreaks of toxoplasmosis in pigs have been reported from several countries, especially Japan (Dubey, 1986a; Nogami and others, 1999). Mortality is more common in young pigs than adult pigs. Pneumonia, myocarditis, **encephalitis**, and placental necrosis have been reported in infected pigs. In the United States, serological **surveillance** of the national swine herd for *T. gondii* infection has been conducted at 5-year intervals since 1990 (*http://nahms.aphis.usda.gov/*). Results of the National Animal Health Monitoring System (NAHMS) surveys indicate that prevalence of *T. gondii* varies dramatically depending upon the age of the pigs surveyed Hill and others, 2010). For the 1990 survey, only sows were sampled. Grower/finisher and sow/breeder populations were surveyed concurrently in 1995 and 2000, and grower/finisher swine were targeted in 2006. The initial 1990 survey documented a nearly 20 percent seroprevalence of *T. gondii* antibodies in the U.S. sow population. Subsequent surveys have documented a decline in *T. gondii* seroprevalence in sows from 20 percent in 1990, to 15 percent in 1995, to 6 percent in 2000 (Patton and others, 1996; 1998; 2002). In contrast, seroprevalence in grower/finisher swine has remained somewhat stable over that period (1995, 3.2 percent; 2000, 0.9 percent; 2006, 2.6 percent; Hill and others, 2010).

Nonconfinement housing is a significant risk factor for *T. gondii* transmission (Assadi-Rad and others, 1995; Dubey, Weigel, and others, 1995; Weigel, Dubey, Siegel, Kitron, and others, 1995; Gamble and others, 1999; Lehmann and others, 2003; Hill and others, 2010). Pigs reared in nonconfinement management systems are at higher risk for *T. gondii* infection, because these animals have increased exposure to infected wildlife, organic material, and soil contaminated with cat feces containing infectious oocysts. Reduced seroprevalence in sow populations, as reported in the NAHMS surveys, likely resulted from the large-scale movement of the swine industry towards total confinement rearing of approximately 80 percent of the sow population and an emphasis on facility biosecurity, but the stable seroprevalence of approximately 2.0 percent in grower/finisher pigs may reflect gaps in adherence to good production practices known to prevent exposure to *T. gondii* in confinement-reared pigs.

Dogs and Cats



Serologic results provide worldwide evidence for domestic dogs commonly being infected with *T. gondii* (fig. 6*G*). Clinical toxoplasmosis is far less common than subclinical disease. Neonatal toxoplasmosis is rare in young dogs, and most cases are now considered to be due to a different parasite, *Neospora caninum* (Dubey, 2010).

The domestic cat is a definitive host for *T. gondii* and can be easily infected experimentally. Although serology indicates a high global prevalence of infection in this species (fig. 6*F*), clinical toxoplasmosis in cats is probably a rare occurrence. Nevertheless, **parenteral** inoculation with even a few *T. gondii* can kill cats. Newborn kittens can die from acute toxoplasmosis despite receiving passively transferred antibodies from the mother (Dubey and Beattie, 1988).

Wildlife

Clinical and subclinical forms of toxoplasmosis have been reported in many wild animal species. Serologic data (table 5) show that a significant number of feral pigs, bears, and cervids are exposed to *T. gondii* and suggest that *T. gondii* infections are common in carnivores (table 6).

Table 5. Serologic prevalence of *T. gondii* antibodies in wild game in the United States.

[LAT, latex agglutination test; MAT, modified agglutination test; IHAT, indirect hemagglutination test. Modified from Dubey and Odening, 2001; Hill and others, 2005]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Black bear	Alaska	LAT	40	15
	Pennsylvania	MAT	665	80
	Pennsylvania	MAT	28	79
	North Carolina	MAT	143	84
	Maryland	MAT	66	25.7
Grizzly bear	Alaska	LAT	480	18
	Alaska	MAT	892	25
White-tailed deer	Kansas	MAT	106	44
	Minnesota	MAT	1367	30
	Pennsylvania	MAT	593	60
	Alabama	MAT	16	44
Feral pig	Georgia	MAT	170	18.2
	California	IHAT	135	13
	North and South Carolina	MAT	257	34.2
	Florida	IHAT	457	2.6
Moose	Nova Scotia	IHAT	125	15
	Alaska	IHAT	110	23
Bison	Montana	MAT	93	2

Table 6. Serologic prevalence of *T. gondii* antibodies in wild **carnivorous** animals.

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Badger	Spain	MAT	29	75.9
Coyote	United States	MAT	222	59
	Georgia	LAT	17	18
		MAT	1	100
	Kansas	DT	13	62
	Texas	MAT	52	62
	Wisconsin	MAT	35	18
Foxes				
Arctic	Norway	MAT	594	43
Gray	United States	DT	4	25
		MAT	97	75.3
	Georgia	MAT	2	100
Island	California			
	Channel Islands	IHAT	194	29.9
Kit	California	IHAT	35	6
		NS	47	14.9
Red	Belgium	IFAT	123	100
	Spain	MAT	89	60.8
	United Kingdom	IHAT	549	20
	United States	MAT	283	85.9
	Georgia	MAT	1	100
	Kansas	MAT	2	50
		DT	10	90
Sand	Sharjah, United Arab Emirates	MAT	8	100
Skunk	Canada	MAT	64	15.6
	Connecticut	MAT	24	42
	Illinois	MAT	18	38.9
	Iowa	MAT	81	47
	Kansas	MAT	1	0
		DT	4	50
	Wisconsin	MAT	7	5

Table 6. Serologic prevalence of *T. gondii* antibodies in wild **carnivorous** animals. —Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Raccoon	Canada	MAT	91	27.5
	Connecticut	MAT	12	100
	Georgia	MAT	75	52
	Kansas	MAT	20	70
		DT	52	13
	Illinois	MAT	188	67
		MAT	379	48.5
	Iowa	MAT	14	29
		MAT	885	15
	Virginia	MAT	256	84
	Wisconsin	MAT	54	59.2
	Various U.S. areas	MAT	427	50.3
		MAT	99	46.5
Dingo	Australia	MAT	62	10
Wolves				
Canadian	Spain	MAT	27	48.1
	Alaska	MAT	125	9
Maned	Brazil	IFAT	59	74.6
Genet	Spain	MAT	18	66.6
Marten				
Pine	Spain	MAT	3	100
	Czech Republic	DT	6	17
Stone	Spain	MAT	14	92.8
Mink	Bulgaria	ELISA	156	12.2
	Poland	LAT	961	13.9
	Denmark	LAT	195	3
	United States	DT	24	54.2
Mongoose, Egyptian	Spain	MAT	22	59.1
Otter	Spain	MAT	5	100
Wolverine	Canada	MAT	41	41.5

Sporadic and widespread outbreaks of toxoplasmosis occur in rabbits, mink, birds and other domesticated wildlife. Animals that survive infection harbor tissue cysts, and can, therefore, transmit *T. gondii* infection to human consumers. Sporadic cases of clinical toxoplasmosis occur in rabbits (Dubey, Brown, and others, 1992a; Leland and others, 1992), squirrels (Soave and Lennette, 1959; Van Pelt and Dieterich, 1972; Dubey and others, 2006; Bangari and others, 2007), mink (Frank, 2001) and pet birds, especially in canaries and finches (Dubey, 2002; Dubey, Parnell, and others, 2004). An unusual clinical presentation of toxoplasmosis in canaries is blindness with nearly complete destruction of the eyes (Dubey, 2002). Toxoplasmosis in squirrels can simulate signs of rabies (Soave and Lennette, 1959).

Table 7. Summary of reports of clinical toxoplasmosis in selected species of carnivores.

[From Dubey, 2010. *Toxoplasma gondii* demonstrated by **histology** and presence of tachyzoites]

Species	Location	Living condition	Number of deaths	Remarks
Raccoon	Pennsylvania	Wild	1	Encephalitis, hepatitis.
Foxes				
Gray fox	Mississippi	Wild	1	Encephalitis, cerebral hematoma.
	Many U.S. states	Wild	6 of 157	Pneumonia, concurrent canine distemper virus.
	Virginia	Wild	1 of 35	Pneumonia, concurrent canine distemper virus.
Sand fox	Sharjah, United Arab Emirates	Captive	Not stated	Enteritis, hepatitis, myocarditis.
Arctic fox	Svalbard, Norway	Wild	Not stated	Hepatitis, pneumonia, encephalitis.
Red fox	Pennsylvania	Wild	1	Neurologic, disseminated toxoplasmosis, no canine distemper virus.
	Virginia	Captive	3 of 48	Pneumonia, concurrent canine distemper virus.
Fennec fox	Missouri	Captive	1	Disseminated toxoplasmosis.
Blanford's fox	Sharjah, United Arab Emirates	Captive	1	10 weeks old, visceral toxoplasmosis.
Mink	Wisconsin	Captive	Not stated	Abortion and neonatal mortality.
Ferrets				
Ferret	New Zealand	Not stated	Not stated	Neonatal mortality.
Black-footed ferrets	Kentucky	Captive	25	Disseminated toxoplasmosis.
Slender-tailed meerkat	Spain	Captive	7	Disseminated toxoplasmosis, pulmonary lesions predominant.
	Argentina	Captive	3	Disseminated toxoplasmosis, pulmonary lesions predominant.

Small Carnivores

Serologic surveys indicate that *T. gondii* infections are common in carnivores (table 6). Fur-bearing animals can often harbor live *T. gondii* without clinical signs, as evidenced by serologic prevalence and by isolation of *T. gondii* from apparently normal animals (Watson and Beverley, 1962; Walton and Walls, 1964; Bigalke and others, 1966; Dubey and others, 1993; Smith and Frenkel, 1995). Fatal toxoplasmosis has been diagnosed in carnivores submitted for **necropsy**, often for rabies examination. Most of the animals had concurrent distemper virus infection, which is immunosuppressive (Helmboldt and Jungherr, 1955). Clinical toxoplasmosis has been reported in carnivores (table 7), including chinchillas from the United States (Keagy, 1949; Gorham and Farrell, 1955), mink from Denmark (Momberg-Jørgensen, 1956) and Canada (Pridham and Belcher, 1958; Pridham, 1961), foxes from Denmark (Møller, 1952) and the United States, (Dubey, Hamir, and Rupprecht, 1990; Dubey and Lin, 1994), raccoons from the United States (Møller and Nielsen, 1964), a skunk from the United States (Dieters and Nielsen, 1978), and raccoon dogs from Japan (Hirato, 1939).

Bears

Serologic surveys indicate high numbers of bears are exposed to *T. gondii* (table 8). Jordan and others (1975) reported both *T. gondii* and *Trichinella spiralis* infections in a patient who consumed wild bear meat. *T. gondii* has been found in black bears (Dubey and others, 1994, 2013; Dubey, Humphreys, and Thulliez, 1995; Dunbar and others, 1998) in the United States. Clinical toxoplasmosis was diagnosed in a 6-day-old polar bear in Hungary (Kiss and Graf, 1989). Lesions were seen in the liver, skeletal muscles, and the brain. Kiupel and others (1987) reported toxoplasmosis in 9 young bears from Germany; 2 of 20 other bears that died had acute primary toxoplasmosis. However, in the light of the discovery of fatal sarcocystosis in black bears and polar bears in the United States (Zeman and others, 1993; Garner and others, 1997), the diagnosis is not definitive.

Table 8. Serologic prevalence of *T. gondii* antibodies in bears.

[From Dubey, 2010. ELISA, enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Location	Test	Number of animals tested	Animals tested positive, in percent
Black			
Canada	ELISA	38	34.2
Alaska	LAT	40	15
	MAT	143	43
Florida	LAT	66	56.1
North Carolina	MAT	143	84
Pennsylvania	MAT	665	80
	MAT	322	79.8
	MAT	28	78.6
	MAT	80	82.5
Grizzly			
Canada	ELISA	36	0
Alaska	LAT	480	18
	MAT	892	25
Polar			
Canada	ELISA	60	0
Norway[1]	MAT	83 cubs	3.6
	MAT	444 adults	21.4
Alaska	LAT	500	6

[1] Including Svalbard, Greenland, and Barents Sea.

Wild Cats

Serologic surveys indicate widespread infection in wild cats in North America and probably in other countries (table 9). Clinical toxoplasmosis has rarely been diagnosed in wild felids; most instances have occurred in zoo animals. Existing reports include two 4- to 6-month-old lions in captivity at the Jos Zoological Gardens, Nigeria (Ocholi and others, 1989); a 6-year-old captive Pallas cat from the Milwaukee County Zoo, Milwaukee, Wis., (Dubey, Gendron-Fitzpatrick, and others, 1988); a colony of pallas cats in a California zoo (Riemann and others, 1974); a 6-month-old bobcat from Georgia (Smith and others, 1995); a 1-week-old bobcat from Montana (Dubey and others, 1987); a 9-month-old cheetah from South Africa (Van Rensburg and Silkstone, 1984); a cheetah from a Texas zoo (Cannon, 1974); and sand cat kittens from a breeding center in the United Arab Emirates (Pas and Dubey, 2008). The bobcat from Montana was probably congenitally infected. The pallas cat from Wisconsin probably became infected by eating tissue cysts, because **enteroepithelial** stages (fig. 10) were found in sections of small intestine. An unusual finding in the pallas cat was severe *T. gondii*-associated **enteritis**.

Table 9. Serologic prevalence of *T. gondii a*ntibodies in large captive and wild cats.

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; KELA, kinetic enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Living condition	Test	Number of animals tested	Animals tested positive, in percent
Panthera spp.	Brazil	Captive	MAT	2	100
			IFAT	2	100
Tiger	Thailand	Captive	LAT	18	27.8
	Florida	Captive	ELISA	4	75
	Various U.S. areas	Captive	IFAT	11	63.6
Amur tiger	Midwestern U.S. zoos	Captive	MAT	18	27.8
Lion	Botswana	Wild	IFAT	53	92
	Brazil	Captive	MAT	27	51.8
			IFAT	3	100
	Southern Africa	Wild	IFAT	41	90.2
		Wild	ELISA	66	98
		Wild	IFAT	42	100
	Thailand	Captive	LAT	7	14.3
	Florida	Captive	ELISA	2	100
	Midwestern U.S. zoos	Captive	MAT	22	54.5
	Various U.S. areas	Captive	IFAT	10	80
	Zimbabwe	Wild	IFAT	21	100
		Wild	MAT	26	92.3

Table 9. Serologic prevalence of *T. gondii* antibodies in large captive and wild cats.—Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; KELA, kinetic enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Living condition	Test	Number of animals tested	Animals tested positive, in percent
Leopard	Brazil	Captive	MAT	3	100
	Botswana	Wild	IFAT	1	100
	Southern Africa	Captive	IFAT	4	75
	South Africa	Wild	IFAT	7	86
	Thailand	Captive	LAT	19	15.8
	California	Captive	IFAT	1	100
	Florida	Captive	ELISA	3	66.6
	Midwestern U.S. zoos	Captive	MAT	1	100
Jaguar	Brazil	Not stated	MAT	212	63.2
			IFAT	3	100
	French Guiana	Wild	MAT	1	100
	Thailand	Captive	LAT	3	33.3
	Various U.S. areas	Captive	IFAT	2	100
	Midwestern U.S. zoos	Captive	MAT	1	100
Snow leopard	Midwestern U.S. zoos	Captive	MAT	14	35.7
	Thailand	Captive	LAT	1	100
Bobcat	Quebec	Wild	MAT	10	40
	Mexico	Wild	LAT	6	66.6
	California	Wild	LAT	52	50
		Wild	LAT	25	88
		Wild	IFAT	3	100
	Georgia	Wild	MAT	6	83.3
	Kansas, Missouri	Wild	DT	2	50
	Pennsylvania	Wild	MAT	131	83
	Various U.S. areas	Captive	IFAT	3	333
Lynx	Quebec	Wild	MAT	106	44
	California	Captive	IFAT	1	100

Table 9. Serologic prevalence of *T. gondii a*ntibodies in large captive and wild cats.—Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; KELA, kinetic enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Living condition	Test	Number of animals tested	Animals tested positive, in percent
Eurasian lynx	Brazil	Captive	MAT	1	100
	Canada	Captive	ELISA	5	20
	Quebec	Wild	MAT	106	44
	Sweden	Wild	MAT	207	75
Iberian lynx, polecat	Spain	Wild	MAT	27	81.5
			MAT	26	80.7
		Wild	IHAT, LAT	57	44
Caracal	California	Captive	IFAT	1	100
	Midwestern U.S. zoos	Captive	MAT	4	50
Cheetah	Southern Africa	Captive	IFAT	23	43.4
	Thailand	Captive	LAT	1	100
	Various U.S. areas	Captive	IFAT	9	77.7
	Florida	Captive	IHAT	16	68.7
	Midwestern U.S. zoos	Captive	MAT	22	27.3
Cougar, puma, or Florida panther, mountain lion	Brazil	Captive	MAT	172	48.3
			IFAT	5	100
	Canada	Wild	LAT	23	34.8
		Captive	ELISA	15	7
	Central and South America	Wild	LAT	83	32.5
	Mexico	Wild	LAT	12	16.7
	United States	Wild	LAT	320	19.1
	California	Wild	LAT	36	58
		Captive	IFAT	42	25.5
		Wild	IFAT	26	92.3
	Florida	Wild	ELISA	38	9
		Captive	ELISA	6	83.3
	Midwestern U.S. zoos	Captive	MAT	8	62.5
	Various U.S. areas	Captive	IFAT	5	60
	Vancouver Island, Canada	Wild	IHAT	5	100
		Wild	MAT	12	92

Table 9. Serologic prevalence of *T. gondii a*ntibodies in large captive and wild cats.—Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hem-agglutination test; KELA, kinetic enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Living condition	Test	Number of animals tested	Animals tested positive, in percent
Jungle cat	Brazil	Captive	MAT	2	100
Amur leopard cat	Midwestern U.S. zoos	Captive	MAT	1	100
Sand cat	United Arab Emirates	Captive	MAT	6	100
		Captive	LAT	4	75
Pallas cat	Austria	Captive	MAT	8	100
	Colorado	Captive	LAT	4	100
	Midwestern U.S. zoos	Captive	MAT	5	20
	Ohio	Captive	ELISA	14	79
	Oklahoma	Captive	KELA	6	100
	Wisconsin	Captive	MAT	3	67
Lynx	Alaska	Wild	MAT	255	15.3
Wild cat	United Kingdom	Wild	IHAT	45	62
	Spain	Wild	MAT	6	50
Gordon's cat	United Arab Emirates	Captive	MAT	36	86.1
Serval	Brazil	Captive	MAT	2	100
	Florida	Captive	ELISA	2	50
	Various U.S. areas	Captive	IFAT	3	33.3
Asian golden cat, golden cat	Thailand		LAT	8	12.5
Fishing cat	Midwestern U.S. zoos	Captive	MAT	4	25
	Thailand	Captive	LAT	27	22.2
Geoffroy's cat	Bolivian Chaco	Wild	ELISA	8	25
	Brazil	Captive	MAT	12	75
Pampas cat	Brazil	Captive	MAT	8	12.5
Leopardus spp.	Bolivian Chaco	Wild	ELISA	10	100
Ocelot	Brazil	Captive	MAT	168	57.7
			IFAT	5	80
	Alabama	Captive	IFAT	1	100
Oncilla	Brazil	Captive	MAT	131	51.9
Margay	Brazil	Captive.	MAT	63	55.5

Table 9. Serologic prevalence of *T. gondii* antibodies in large captive and wild cats.—Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; KELA, kinetic enzyme-linked immunosorbent assay; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Living condition	Test	Number of animals tested	Animals tested positive, in percent
Clouded leopard	California	Captive	IFAT	2	50
	Midwestern U.S. zoos	Captive	MAT	7	14.3
	Thailand	Captive	LAT	16	12.5
Jaguarundi	Brazil	Captive	MAT	99	45.9
			IFAT	1	100
	Florida	Captive	ELISA	1	100

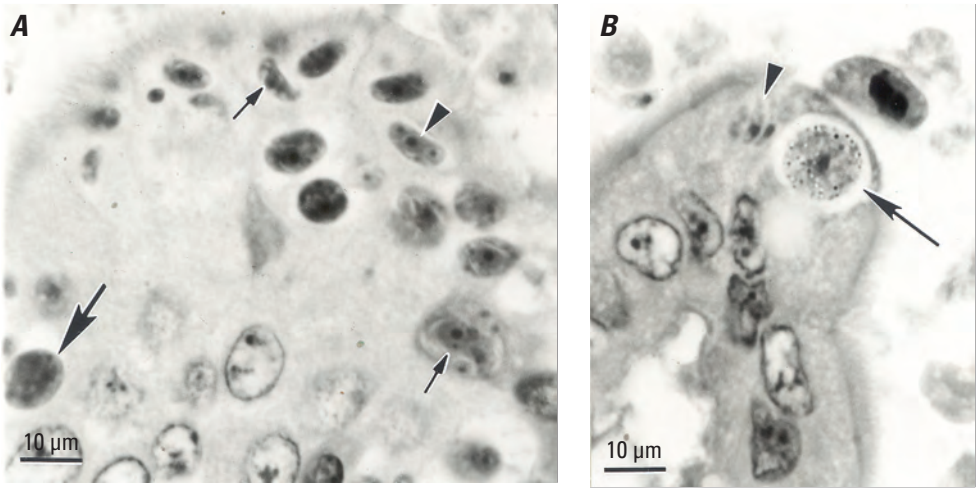


Figure 10. Enteroepithelial stages of *T. gondii* in the small intestine of a naturally infected Pallas cat. Hematoxylin and eosin stain was used to enhance contrast of the tissues. *A*, Numerous parasites at the tip of the villus. Note two immature female gamonts (small arrows), a schizont with three nuclei (arrowhead), and, probably, a microgamont (large arrow). *B*, An oocyst (large arrow) and merozoites (arrowhead) at the tip of the villus. (From Hill and others, 2005. µm, micrometer)

Small Mammals

Asymptomatic *T. gondii* infections are widely prevalent in many small mammals, including rats (Dubey and Frenkel, 1998), various species of mice (Dubey and Beattie, 1988; Brillhart and others, 1994; Smith and Frenkel, 1995; Dubey, Weigel, and others, 1995), and rabbits (Cox and others, 1981; Dubey and Beattie, 1988), but clinical toxoplasmosis is relatively rare. We are not aware of any reports of clinical toxoplasmosis in naturally infected rats and mice. **Epizootics** and individual cases of fatal toxoplasmosis have been reported for several small mammal species (table 10). Failure to find antibodies to *T. gondii* in 176 brown hares from Sweden, despite **epizootics** in this species, suggests that this animal is highly susceptible to *T. gondii*, and perhaps the disease is fatal in most of them (Gustafsson and Uggla, 1994).

Table 10. Summary of reports of clinical toxoplasmosis in squirrels, rabbits, hares, and other small mammals.

[From Dubey, 2010. *Toxoplasma gondii* demonstrated by histology and presence of tachyzoites or tissue cysts]

Species	Location	Living condition	Number of deaths	Remarks
American red squirrel	Indiana	Wild	2	Toxoplasmic pneumonia, polymerase chain reaction positive.
Grey squirrel	Louisiana Philadelphia	Captive Wild	2 1	Neurologic signs, disseminated toxoplasmosis.
Korean squirrel	Spain	Captive	4	Transported from People's Republic of China, died in Spain; transport stress, disseminated toxoplasmosis.
Red squirrel	Not stated	Captive Feral	1 1	Acute toxoplasmosis.
Rabbit	Georgia	Domestic	1	Visceral toxoplasmosis with severe splenitis.
	Massachusetts	Rabbitry	2	Disease outbreak among 50 Rhinelander and minilop breeds.
	Texas	Domestic	3	3 of 5 French lop rabbits from a private owner died, 1 rabbit necropsied. Visceral toxoplasmosis with severe lesions in spleen.
Brown hare Mountain hare	Sweden	Wild	39 8	Retrospective study of 388 wild brown hares and 202 wild mountain hares that died 1980–85.
Beaver	Connecticut	Wild	1	Disseminated toxoplasmosis, no evidence for paramyxal virus.
Woodchuck	New York	Wild	1	Encephalitis, polymerase chain reaction positive.
Porcupine	Costa Rica	Captive	1	Disseminated toxoplasmosis.
North American porcupine	New Jersey	Captive	2	Neurologic, concurrent infection with *Baylisascaris* spp.
Three-toed sloth	Pará, Brazil	Captive	1	Sudden death, visceral toxoplasmosis.
Guinea pig	England	Captive	1	Abortion, Caesarean section, tachyzoites in placenta and fetal liver.

Ungulates

Serological data (table 11) shows that a wide range of ungulate species are exposed to *T. gondii*. Clinical toxoplasmosis, including fatal infections, has been reported from ungulates and other nondomestic **ruminants** (table 12). Sacks and others (1983) reported acute toxoplasmosis in three deer hunters in the United States, probably acquired by ingesting undercooked venison. Wapiti (elk), pronghorn, bison, red deer, mule deer, and reindeer are also susceptible to *T. gondii* oral infection via oocysts. Although fatal toxoplasmosis has not been reported in the pronghorn, experimental exposure has shown the pronghorn to be highly susceptible to *T. gondii* infection as evidenced by generalized visceral toxoplasmosis in experimentally infected pronghorns (Dubey and others, 1982). Clinical toxoplasmosis has been reported in captive gazelle and gerenuk in the United States (Dubey, 2010).

Table 11. Serologic prevalence of *T. gondii* antibodies in wild ungulates.

[From Dubey, 2010. DT, dye test; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Alpaca	Peru	Immunoblot	356	0.8
	Germany	Immunoblot	12	33
	Peru	IFAT	278	34.5
African buffalo	Zimbabwe	MAT	18	5.6
American bison	Alaska	MAT	241	0.8
Barbary sheep	Spain	MAT	10	10
Barren-ground caribou	Canada	MAT	147	29.9
Bighorn sheep	California	MAT	697	3.6
		Not stated	178	12.3
		Not stated	998	21.6–25.0
Bushbuck	Zimbabwe	MAT	14	57.1
Caribou	Alaska	MAT	241	6
Dall sheep	Alaska	MAT	319	6.9
Deer				
Black-tailed	Washington	MAT	43	32.5
Fallow	Czech Republic	IFAT	143	17
	Spain	MAT	79	22.8
Marsh	Brazil	IHAT	66	27.2
Mule	California	LAT	[1]276	15
	Nebraska	MAT	89	34.8
Pampas	Brazil	IHAT	41	12.1
Red	Norway	MAT	571	7.7
	Spain	MAT	441	15.6
	Czech Republic	IFAT	377	45

Table 11. Serologic prevalence of *T. gondii* antibodies in wild ungulates.—Continued

[From Dubey, 2010. DT, dye test; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Roe	Austria	IHAT	40	12
	Czech Republic	DT	95	14
	Czech Republic	IFAT	79	24
	Norway	MAT	760	33.9
	Spain	MAT	278	39.2
	Spain	MAT	33	21.2
Sika	Czech Republic	IFAT	14	50
White-tailed	Alabama	MAT	19	21
	Iowa	MAT	84	64.2
	Minnesota	MAT	62	32.2
	Kansas	MAT	106	44
	Minnesota	MAT	1367	30
	Iowa	MAT	170	53.5
	Mississippi	MAT	73	46.5
	Ohio	MAT	147	44
	Pennsylvania	MAT	593	60
Eland	Zimbabwe	MAT	19	36.8
Elephant				
Indian	Thailand	MAT	156	45.5
		LAT	156	25.6
Asian	Sri Lanka	MAT	53	26.4
African	Zimbabwe	MAT	19	10.5
		MAT	20	10
Giraffe	Zimbabwe	MAT	10	10
Greater kudu	Zimbabwe	MAT	10	20
Llama	Argentina	IFAT	308	30
	Peru	IFAT	43	55.8
		Immunoblot	81	8.6
	United States	MAT	283	33.5
Moose	Canada	IHAT	125	15
	Norway	MAT	2,142	12.6
	Alaska	MAT	240	1.2

Table 11. Serologic prevalence of *T. gondii* antibodies in wild ungulates.—Continued

[From Dubey, 2010. DT, dye test; IFAT, indirect fluorescence antibody test; IHAT, indirect hemagglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Mouflon	Bulgaria	Ouchterlony test	60	0
	Czech Republic	IFAT	105	8.5
	Spain	MAT	27	14.8
Muskox	Canada	MAT	203	6.4
Nyala	Zimbabwe	MAT	10	90
Pronghorn	Kansas	MAT	63	4.7
Pyrenean chamois	Spain	MAT	10	20
Reindeer				
Fennoscandian reindeer	Norway and Finland	MAT	2,577	0.9
	Czech Republic	IFAT	2	50
Svalbard reindeer	Norway	MAT	866	1
		MAT	390	0
Rhinoceros				
Black	Zimbabwe	MAT	11	27.3
White	Zimbabwe	MAT	2	50
Sable	Zimbabwe	MAT	67	11.9
Spanish ibex	Spain	MAT	3	33
Vicuna	Peru	IFAT	200	5.5
	Peru	Immunoblot	103	2.9
Yak	Bulgaria	Ouchterlony test	267	6
Wildebeest	Zimbabwe	MAT	69	14.5

[1] Samples were from various species of deer, including mule deer.

Table 12. Examples of *T. gondii* infections in wild ungulates.

Species	Findings
Rhim gazelle	Fatal cases in a 1-year-old captive animal in Florida (Stover and others, 1990).
Gerenuk	2 captive animals in Florida (Stover and others, 1990).
Dama gazelle	Captive animal in Florida (Stover and others, 1990).
Mountain gazelle	4 captive animals in Germany (Stiglmair-Herb, 1987).
Cuvier's gazelle	Fatal disseminated infection in a captive animal in Missouri (Junge and others, 1992).
Red deer	Viable infectious stage in edible tissues in New Zealand (Collins, 1981).
Roe deer	Viable infectious stage in edible tissues in Germany (Entzeroth and others, 1981).
Pronghorn	Isolated from species in the United States (Dubey, 1981; Dubey and others, 1982)
Moose	Seropositive wild moose in Alaska (Kocan and others, 1986).
Mule deer	31 of 89 adult mule deer positive from Nebraska (Lindsay and others, 2005).
White-tailed deer	Isolated from the hearts of naturally infected deer in Alabama (Lindsay and others, 1991).
Saiga antelope	Fatal infections (Bulmer, 1971) Generalized visceral toxoplasmosis following natural infection (Ippen and others, 1981).
Reindeer	Generalized visceral toxoplasmosis following experimental infection (Oksanen and others, 1996). Fatal transplacental toxoplasmosis also diagnosed in a captive animal (Dubey, Lewis, and others, 2002).
Dik-dik	Fatal transplacental toxoplasmosis in a captive animal (Dubey, Tocidlowski, and others, 2002).
Unnamed deer	Fatal infections in two deer (Burgisser, 1960).

Marsupials

Toxoplasmosis is a serious disease of Australasian marsupials, and numerous deaths have been reported from zoos (table 13). Animals in the wild can also die of toxoplasmosis (Attwood and others, 1975; Obendorf and Munday, 1983, 1990). Animals can die suddenly, without clinical signs, or can exhibit neurological signs, loss of vision, diarrhea, and respiratory distress. Virtually any organ of the body can be affected. Clinical signs and necropsy findings in a variety of marsupials can be found in the report of Canfield and others (1990).

Antibodies to *T. gondii* were found in various species of marsupials (table 14). Attwood and others (1975) found dye test antibodies (1:4 to 1:4096) in 13 of 15 dasyurids. High levels of *T. gondii* antibodies were found in adult black-faced kangaroos in a zoo (Dubey, Ott-Joslin, and others, 1988), indicating that not all exposed wallabies die of toxoplasmosis. Jakob-Hoff and Dunsmore (1983) found antibodies in 2 of 25 Tammar wallabies, 0 of 26 black-flanked rock wallabies, and 0 of 3 bandicoots in the indirect hemagglutination test (IHAT). Because the IHAT is not sensitive for the diagnosis of toxoplasmosis in animals in general, it is not known if these differences in seroprevalence of *T. gondii* in various species of kangaroos were real.

Little is known of the **specificity** and sensitivity of different serologic tests for the detection of antibodies to *T. gondii* in kangaroos. Johnson and others (1988) evaluated the ELISA in naturally infected animals. They isolated *T. gondii* from the brains of 4 of 17 Tasmanian pademelons and 6 of 17 Bennett's wallabies and used sera from *T. gondii*-infected animals to standardize their ELISA. They found antibodies in 5 of 151 Bennett's wallabies and 15 of 85 Tasmanian pademelons. They further evaluated the **direct agglutination test** in experimentally infected animals (Johnson and others, 1989).

Little is known of treatment and prophylaxis in marsupials; there has not been any controlled study of treatment in marsupials (Dubey and Crutchly, 2008). Standard antitoxoplasmic therapy (sulfadiazine and pyrimethamine) had some success in treating clinical toxoplasmosis in zoo animals (Jensen and others, 1985). Vaccination with a live, modified, nonpersistent strain of *T. gondii* (S-48) was lethal in Tammar wallabies (Lynch and others, 1993). Some success was achieved in vaccination with the related coccidian *Hammondia hammondi* (Reddacliff, Hartley, and others, 1993; Reddacliff, Parker, and others, 1993). However, without a commercial vaccine, the best approach to preventing toxoplasmosis in marsupials may be to avoid contamination of food and water with *T. gondii* oocysts.

Table 13. Reports of clinical toxoplasmosis in captive Australian marsupials.

[From Dubey, 2010]

Species	Location	Remarks
Bennett's wallaby	Zoo in La Plata, Argentina	2 adults died suddenly with generalized toxoplasmosis. A 3-month-old joey was not infected. Viable *T. gondii* was isolated from brains of both adult females by bioassay in cell culture.
	Zoo in Lugo, Spain	3 of 5 wallabies died after relocation. 1 animal was necropsied; it died of visceral toxoplasmosis, including ulcerative gastritis. Disease was thought to be reactivation of chronic infection. *T. gondii*-like parasites were found in **biopsy** of ulcers in buccal **mucosa** of the second wallaby.
	Exotic animal farm in Missouri	6 of 18 adults and 1 joey died within 2 months. 2 adult wallabies were necropsied. Toxoplasmic myocarditis was the main lesion.
	Zoo in Illinois	Acute toxoplasmosis was diagnosed ante-mortem; tachyzoites were seen in peripheral leukocytes. The animal died of generalized toxoplasmosis in spite of therapy.
Western grey kangaroo	Animal farm in England	2 of 12 kangaroos died, 1 necropsied. Pulmonary toxoplasmosis was the main finding.
Black-faced kangaroo	Brookfield Zoo in Illinois	8 of 25 adults and 2 joeys died of acute toxoplasmosis. Serologic data on 6 animals were compared by different tests. Skeletal myositis was associated with tissue cysts found in 1 joey.
Common wombat	Australia	Histologically confirmed toxoplasmosis.

Table 14. Serological prevalence of *T. gondii* antibodies in wild marsupials from Australia.

[From Dubey, 2010. ELISA, Enzyme-linked immunosorbent assay; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Tasmanian pademelon	Tasmania	ELISA	85	17.7
Bennett's wallaby	Tasmania	ELISA	151	3.3
Eastern barred bandicoot	Tasmania	MAT	150	6.7
Western grey kangaroo	Perth area	ELISA	219	15.5
Wombat	New South Wales	MAT	23	21

Nonhuman Primates

New World monkeys are highly susceptible to clinical toxoplasmosis (table 15), whereas Old World monkeys are resistant (Dubey and Beattie, 1988; Cunningham and others, 1992; Dietz and others, 1997). Acute toxoplasmosis has led to deaths of captive ring-tailed lemurs (Borst and van Knapen, 1984; Dubey and others, 1985; Spencer and others, 2004).

Table 15. Summary of reports of clinical toxoplasmosis in captive nonhuman primates.

[From Dubey, 2010. *Toxoplasma gondii* demonstrated by histology and presence of tachyzoites]

Species	Location	Number of deaths	Remarks
Squirrel monkey	Zoo in Japan	2	Visceral toxoplasmosis.
	Zoo in Hungary	21	Disseminated toxoplasmosis.
	Zoo in Mexico	16	Visceral toxoplasmosis.
	Zoo in Israel	6	Disseminated toxoplasmosis.
	Zoo in United Kingdom	6	Disseminated toxoplasmosis.
	Laboratory in French Guiana	47	Sudden death.
Ring tailed lemur	Zoo in United States	1	Pneumonia, visceral toxoplasmosis.
New world primates	Zoos in Brazil	[1]33	Disseminated toxoplasmosis.
	Zoo in Germany	[2]32	Myocarditis, pneumonia.
	Zoo in Denmark	[3]7	Confirmed disseminated toxoplasmosis.
Golden lion tamarin	Zoo in United States	5	Visceral toxoplasmosis.
Woolly monkey	Zoo in United States	2	Disseminated toxoplasmosis.

[1] 3 squirrel monkeys, 7 golden-headed lion tamarins, 3 emperor marmosets, 1 yellow handed marmoset, 1 black marmoset, 5 woolly monkeys, 1 black ear-tufted marmoset,1 night monkey,1 black lion tamarin, 2 golden lion tamarins, 6 howler monkeys, and 2 white ear-tufted marmosets.

[2] 17 ring-tailed lemurs, 15 squirrel monkeys.

[3] 1 common marmoset, 2 cotton-top tamarins, 1 red-bellied white-lipped tamarin, 2 golden lion tamarins, 1 pale-headed saki.

Marine Mammals

A variety of marine mammals have been found to be infected by *T. gondii* (tables 16–17), suggesting contamination of coastal waters and survival of *T. gondii* oocysts in seawater on the Atlantic and Pacific coasts of North America (Lindsay and others, 2001; Miller, Gardner, Kreuder, and others, 2002; Conrad and others, 2005). *T. gondii* is considered a significant cause of encephalitis in sea otters (Cole and others, 2000; Lindsay and others, 2001; Conrad and others, 2005) (box 6). Two new *T. gondii* genotypes (Types A and X) have been characterized from sea otters collected in California and Washington States (Miller and others, 2004; Conrad and others, 2005; Sundar and others, 2008).

Table 16. Examples of clinical toxoplasmosis in marine mammals.

[From Dubey, 2010. Confirmed by histology, tachyzoites, or cysts]

Species	Location	Living condition	Remarks
Elephant seal	California	Wild	Encephalitis.
Northern fur seal	California	Wild	Encephalitis.
Pacific harbor seal	Cold Bay, Alaska	Wild	1 day old, 11.5 kilograms, hepatitis.
Monk seal	Hawaii	Wild	1 adult male, good nutritional condition, lymphadenitis.
Sea lion	Pennsylvania	Captive	10 days old, disseminated.
	California	Captive	Adult, myocarditis.
	Florida	Captive	9-year-old male, disseminated, myocarditis.
Atlantic bottlenose dolphin	Florida	Wild	1 adult and her calf, disseminated.
	Florida	Wild	Young male, hepatitis, adrenalitis.
	Tuscany	Wild	2 adults.
	United States	Wild	1 of 97 stranded adults.
	Canada	Captive	2 with encephalitis.
Striped dolphin	Spain	Wild	Lymphadenitis, encephalitis, 4 of 110 stranded animals.
	Tuscany	Wild	4 wild adults, encephalitis with coinfection with morbillivirus.
Indo-Pacific bottlenose dolphin	United States	Wild	Late term fetus, myocarditis, encephalitis.
Spinner dolphin	Hawaii	Wild	Adrenalitis.
Risso's dolphin	Spain	Wild	Adult and her fetus, disseminated.
	Italy	Wild	1 adult.
Tucuxi dolphin	Rio de Janeiro	Wild	1 adult, lymphadenitis.
Indo-Pacific humpbacked dolphin	Queensland	Wild	4 of 4 stranded adults.
Walrus	Canada	Wild	Seizure.[1]
West Indian manatee	Florida	Wild	Encephalitis.
	Georgetown, Guyana	Wild	Myocarditis.
Beluga whale	Quebec	Wild	6 months old, encephalitis.
	Spain	Wild	31 years old, disseminated.

[1] *T. gondii* found by polymerase chain reaction.

Table 17. Serologic prevalence of *T. gondii* antibodies in marine mammals.

[From Dubey, 2010. DT, dye test: ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; IHAT, indirect hemagglutination test; LAT, latex agglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Sea otter	California; live	IFAT	80	36
	Dead	IFAT	77	61
	Dead	MAT	100	82
	Dead	MAT	25	52
	Washington; live	IFAT	21	38
	Dead	MAT	10	100
Walrus	Alaska	MAT	53	5.6
Monk seal	Hawaii	MAT	117	1.7
Sea lion	Alaska	MAT	27	29.6
	California	MAT	18	61.1
Harbor seal	Washington	MAT	380	7.6
	Alaska	MAT	311	16.4
	Canada	MAT	34	9
Grey seal	Canada	MAT	122	9
Hooded seal	Canada	MAT	60	1.6
Ringed seal	Alaska	MAT	32	15.6
Bearded seal	Alaska	MAT	8	50
Spotted seal	Alaska	MAT	9	11.1
Bottlenose dolphin	California	MAT	94	96.8
	Florida	MAT	47	100
	Florida; South Carolina	MAT	146	100
	South Carolina	MAT	49	53
	Canada	MAT	8	100
	Japan; Solomon Island	LAT	[1] 58	13.7
	Japan	IHAT	59	10.1
	Spain	MAT	7	57.1
Common dolphin	United Kingdom	DT	21	28.6
	Spain	MAT	4	50
Striped dolphin	Spain	MAT	36	11.1
Humpback whale	United Kingdom	DT	1	100
Harbor porpoise	United Kingdom	DT	70	1.4
	Spain	MAT	1	100
Steller sea lion	Russia	ELISA	189	13.8

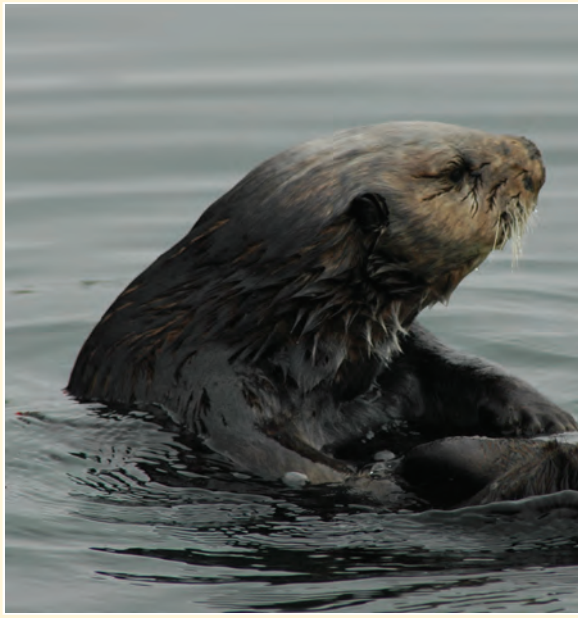
[1] Pacific bottlenose dolphin (*Tursiops aduncus*).

Box 6 Toxoplasmosis and Sea Otters

Photo courtesy of Milton Friend, U.S. Geological Survey

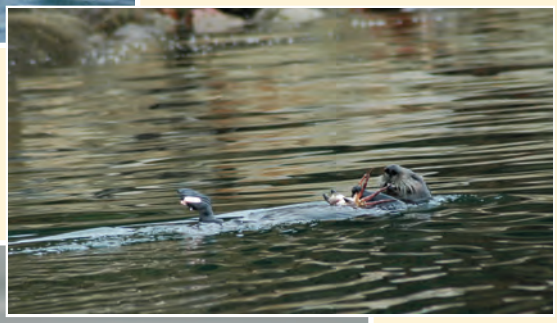
Most people who sit on the California coastline watching sea otters frolicking in the water or floating lazily on the surface don't realize how interconnected their lives are. Southern sea otters make their homes in the nearshore marine habitat alongside more than one-half of the population of Californians who reside in coastal communities (Conrad and others, 2005). The coastal waters have become the collection point of runoff from human activities containing various types of pollutants, including bacterial and protozoal organisms that are having a serious impact on the health of southern sea otters and their ability to survive as a species.

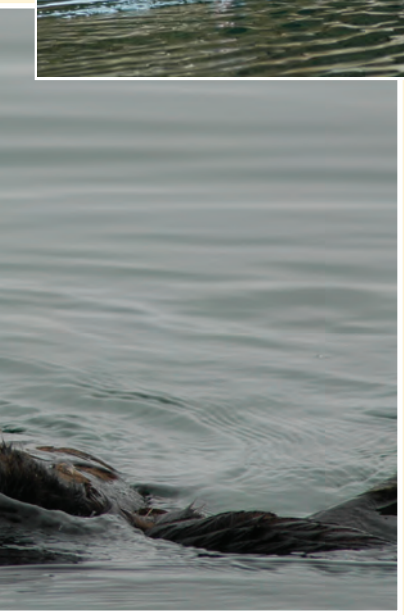
In the late 1800s, the southern sea otter was hunted to near extinction by the fur industry. After gaining federal protection in 1911, their numbers began to recover. However, the species was listed as "threatened" in 1977 under the U.S. Endangered Species Act, and in the 1990s, the recovery appeared to be much slower than expected. Research determined that an increased mortality rate was responsible for this slow recovery rather than a decreased birth rate or migration (Estes and others, 2003; Kreuder and others, 2003; Gerber and others, 2004). In 1992, **pathologist**s at the U.S. Geological Survey's National Wildlife Health Center in Madison, Wis., determined that infectious diseases were responsible for 38.5 percent of sea otter deaths (Thomas and Cole, 1996). Since then, protozoal **meningoencephalitis** (inflammation of the brain and membranes covering the spinal cord and brain) caused by *T. gondii* has been shown to be a primary cause of 16.2 percent of otter deaths and a contributing factor in 11.4 percent of deaths (Kreuder and others, 2003). Encephalitis caused by *T. gondii* may cause abnormal behavior of otters, making them more susceptible to shark attacks (Kreuder and others, 2003). Most of these mortalities have been subadults and prime-age adults, thus these deaths have a direct impact on potential population growth and recovery. In addition to their value as a tourist attraction, sea otters play a crucial role in the health of the nearcoastal ecosystem. They act as a "**keystone**" species to help maintain the health of coastal kelp forests by feeding on sea urchins, which can destroy kelp forests by their herbivorous behavior if left unchecked. Kelp forests are important habitats for many marine species and also act to protect the coastline from erosion.



Center and bottom photos courtesy of Tania Larson, U.S. Geological Survey

Studies have revealed an important connection between the land environment adjacent to marine coastal habitats and the high mortality rate among sea otters (Miller, Gardner, Kreuder, and others, 2002; Fayer and others, 2004). Coastal freshwater runoff contaminated with *T. gondii* oocysts has been strongly associ-





ated with infections of *T. gondii* in southern sea otters; 42–62 percent of southern sea otters in areas along the Pacific coast have been shown to be infected by *T. gondii* (Miller, Gardner, Kreuder, and others, 2002; Miller, and others, 2008). Because felines are the only definitive host for this parasite, infective oocysts must have come from terrestrial habitats. Sea otters may ingest these oocysts directly from contaminated water or indirectly by feeding on mussels, snails, and other shellfish, which can concentrate protozoans during filter-feeding (Cole and others, 2000; Arkush and others, 2003; Lindsay and others, 2003; Miller and others, 2008; Johnson and others, 2009). Northern anchovies may also serve as a source of infection for marine mammals (American Society for Microbiology, 2008). Because toxoplasmosis is a zoonotic disease, humans recreating in these waters or eating uncooked shellfish harvested in these areas are also at risk of becoming infected by *T. gondii*.

Southern sea otters are acting as a sentinel species for the detection of the presence of *T. gondii* in the marine environment. This contamination is a reflection of the level of contamination in the terrestrial environment. Although the proportion of infected cats that shed oocysts is generally low (Dabritz and others, 2007), each infected cat may shed 1 million oocysts after initial infection. Because of the large number of outdoor cats, both owned and feral, this level of shedding could lead to substantial environmental contamination and a high risk for both human and animal infection (Dabritz and others, 2006).

Cat owners may be unaware of the consequences of allowing their cats to defecate outdoors and their role in maintaining the health of their immediate environment as well as more distant habitats that may be connected by water runoff patterns. Disposing of feline feces by flushing them down the toilet may not be a safe method of disposal, because water treatment methods may not totally inactivate *T. gondii* oocysts, thus making sewage discharges into ocean waters a possible source of contamination and subsequent infection for sea otters. Where precautions exist to prevent environmental contamination from landfills, bagging cat feces and disposing of them in landfills may be preferable to flushing them down the toilet. By becoming aware of the interconnectedness of the habitats of humans, domestic animals, and wildlife, people can alter their behaviors in simple ways that can have much larger impacts on the health of both animals and humans.

Cold-Blooded Wildlife

Modern technology, such as DNA analysis, has facilitated greater capability to differentiate among various forms of protozoan parasites. The application of this technology has resulted in previous reports of *T. gondii* infections in **cold-blooded** species becoming invalid. Natural infections may be possible under certain environmental conditions in horned toads and other types of lizards (Stone and Manwell, 1969). Cold-blooded species that have ingested *T. gondii* but have not become infected may passively transfer *T. gondii* to other species.

Birds

Many species of birds can act as intermediate hosts for *T. gondii*, apparently with or without clinical signs (Dubey, 2002) (table 18). Cases of fatal toxoplasmosis have been reported in birds, including pigeons, canaries, Hawaiian crows, psittacines, and wild turkeys (Dubey, 2002). Canaries may develop blindness in severe cases of toxoplasmosis (Dubey, 2002). Recently, toxoplasmosis has been documented for the first time in several species of birds. An outbreak of fatal toxoplasmosis occurred in captive kakarikis, a New Zealand parakeet (Hartley and others, 2008). A bald eagle died of **necrotizing** myocarditis caused by *T. gondii* (Szabo and others, 2004). In addition, a red-bellied woodpecker died of neural toxoplasmosis (Gerhold and Yabsley, 2007). Fatal toxoplasmosis has also been reported in a penguin (Mason and others, 1991).

Table 18. Serologic prevalence of *T. gondii* antibodies in wild birds.

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; IHAT, indirect hemagglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Ostrich	Canada	MAT	973	2.9
Cattle egret	United States	IHAT	40	2.5
Goshawk	Poland	MAT	28	100
Duck	Czech Republic	IFAT	360	14
	Japan	ELISA	221	21.17
Wood duck	United States	IHAT	16	6
Goose	Czech Republic	IFAT	178	43
	Japan	ELISA	123	14.63
	Russia	ELISA	74	18.92
Magpie goose	United States	MAT	11	10.8
Barnacle goose	Norway	MAT	149	7
White-backed vulture	Nigeria	MAT	240	64.8
Turkey vulture	United States	IHAT	2	50
Eurasian buzzard	France	MAT	14	79
Wild turkey	United States	MAT	130	10
		MAT	16	71
American coot	United States	IHAT	38	3
Ring-billed gull	United States	IHAT	13	15.3
Laughing gull	United States	IHAT	33	6

Table 18. Serologic prevalence of *T. gondii* antibodies in wild birds.—Continued

[From Dubey, 2010. DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; IHAT, indirect hemagglutination test; MAT, modified agglutination test]

Species	Location	Test	Number of animals tested	Animals tested positive, in percent
Rock pigeon	Belgium	MAT	220	3.18
	Germany	DT	49	2
	Israel	MAT	495	4
	Italy	DT	108	3
	Poland	MAT	230	74.8
	Poland	MAT	695	4.6
	South Africa	IHAT	16	100
	Taiwan	LAT	665	4.7
	United States	DT	20	10
		DT	15	6
		DT	80	8.7
		MAT	34	5.9
		IHAT	322	8.6
Spotted dove	United States	DT	134	8.2
Ruddy ground-dove	Panama	MAT	79	12.6
Barn owl	United States	MAT	38	27.3
		MAT	80	22.5
		MAT	28	10.7
	France	MAT	18	11
Tawny owl	France	MAT	12	50
Plain wren	Panama	MAT	1	100
Northern mockingbird	United States	IHAT	133	0.75
American robin	United States	IHAT	23	8.6
		IHAT	20	5
Clay-colored robin	Panama	MAT	12	16.6
Crimson-backed tanager	Panama	MAT	8	12.5
Blue-gray tanager	Panama	MAT	15	33
Palm tanager	Panama	MAT	3	33
Common grackle	United States	IHAT	27	37
Great-tailed grackle	Panama	MAT	33	33
Red-winged blackbird	United States	IHAT	31	6.4
Brewer’s blackbird	United States	IHAT	4	25
House sparrow	Czech Republic	IFAT	227	12.3
Eurasian tree sparrow	Czech Republic	IFAT	41	4.9
European starling	United States	IHAT	563	4.8
American crow	United States	IHAT	74	14

Obtaining a Diagnosis

Clinical signs of toxoplasmosis are nonspecific and are not sufficiently characteristic for a definite diagnosis. Toxoplasmosis, in fact, mimics several other infectious diseases. Thus, biologic, serologic, or **histologic** methods, or some combination of these methods, are used to obtain a diagnosis (Dubey, Thulliez, and Powell, 1995). Detection of *T. gondii* antibody in patients may aid diagnosis. Numerous serologic procedures are available for detection of **humoral** antibodies; these include the Sabin-Feldman dye test, the indirect hemagglutination assay, the indirect fluorescent antibody test (IFAT), the direct agglutination test, the latex agglutination test, ELISA, and the immunoabsorbent agglutination test (IAAT). The IFAT, IAAT, and ELISA have been modified to detect immunoglobulin (Ig) M antibodies (Frenkel and others, 1970; Remington and others, 1995), which appear sooner after infection than the IgG antibodies and disappear faster than IgG antibodies after recovery (Remington and others, 1995).

Disease Ecology

The environmentally resistant stage (oocyst) is part of the life cycle of all coccidian parasites. Oocysts of *T. gondii* are formed only in cats, probably in all members of the family Felidae (fig. 11). Cats shed oocysts after ingesting any of the three infectious stages of *T. gondii*, that is, tachyzoites, bradyzoites, and sporozoites (Dubey and Frenkel, 1972, 1976; Dubey, 1996). The time to the shedding of oocysts after initial infection (prepatent period) and the frequency of oocyst shedding vary according to the stage of *T. gondii* ingested. This time period ranges from 3 to 10 days after ingestion of tissue cysts and 18 days or more after ingestion of tachyzoites or oocysts (Dubey and Frenkel, 1972, 1976; Dubey, 1996; Dubey, 2010). Less than 50 percent of cats shed oocysts after ingesting tachyzoites or oocysts, whereas nearly all cats shed oocysts after ingesting tissue cysts (Dubey and Frenkel, 1976). In freshly passed feces, oocysts are noninfective subspherical to spherical forms (fig. 11). These oocysts become infectious (sporulate) outside the cat within 1–5 days depending upon aeration and temperature. Sporulated oocysts contain two ellipsoidal sporocysts (fig. 11), each of which contains four sporozoites (box 7).

Toxoplasmosis may be acquired by **congenital** infection, by ingestion of tissue-inhabiting stages of the parasite, or by ingestion of oocysts in the environment (fig. 12). Most natural infections are probably acquired by ingestion of tissue cysts in infected meat or ingestion of oocysts in food or water contaminated by cat feces (fig. 13). Oocysts of *T. gondii* have been reported from the feces of naturally infected Iriomote cats (Akuzawa and others, 1987), jaguar and ocelots (Patton, Rabinowitz, and others, 1986), cheetah and bobcats, (Marchiondo and others, 1976), and Canadian cougars (Aramini and others, 1998). An outbreak of acute toxoplasmosis in humans was attributed to contamination of a Canadian water reservoir by oocysts shed by domestic and feral cats, as well as cougars (Bell and others, 1995; Bowie and others, 1997).

The bradyzoites from the tissue cysts or the sporozoites from the oocyst penetrate host intestinal epithelial cells and multiply in the intestine as tachyzoites within 24 hours of infection. *T. gondii* may spread first to mesenteric lymph nodes and then to distant organs by invasion of **lymphatics** and blood. *T. gondii* can multiply in virtually any cell in the body. All extracellular forms of the parasite are directly affected by antibody, but intracellular forms are not. It is believed that the cellular **immune system**, including **lymphocytes** and **macrophages**, is more important than humoral factors, such as antibodies, in immune-mediated destruction of *T. gondii* (Renold and others, 1992). However, how *T. gondii* is destroyed in immune cells is not completely known (Renold and others, 1992).

Immunity does not **eradicate** infection. *T. gondii* tissue cysts persist several years after acute infection. The fate of tissue cysts is not fully known. For example, it is not known whether or not bradyzoites can form new tissue cysts directly without transforming into tachyzoites. It has been proposed that tissue cysts may at times rupture during the life of the host. The released bradyzoites may be destroyed by the host's immune responses, or there may be formation of new tissue cysts.

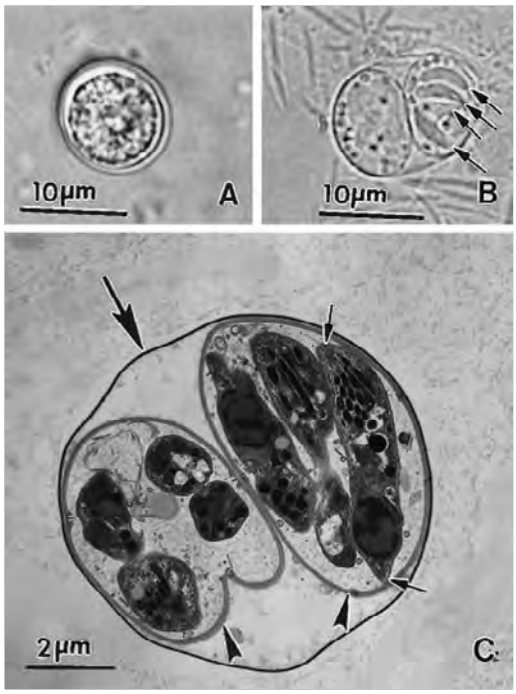


Figure 11. Oocysts of *T. gondii*. *A*, Unsporulated oocyst. Note the central mass (sporont) occupying most of the oocyst. *B*, Sporulated oocyst with two sporocysts. Four sporozoites (arrows) are visible in one of the sporocysts. *C*, Transmission electron micrograph of a sporulated oocyst. Note the thin oocyst wall (large arrow), two sporocysts (arrowheads), and sporozoites, one of which is cut longitudinally (small arrows). (From Hill and others, 2005. µm, micrometer)

In immunosuppressed patients, such as those given large doses of immunosuppressive agents in preparation for organ transplants and in those with acquired immunodeficiency syndrome (AIDS), rupture of a tissue cyst may result in transformation of bradyzoites into tachyzoites and renewed multiplication. The immunosuppressed host may die from toxoplasmosis unless treated. It is not known how **corticosteroids** cause relapse, but it is unlikely that they directly cause rupture of the tissue cysts.

Domestic cats, rather than wild felids, probably act as the primary source of infective oocysts in the environment, leading to infections in humans and animals, both domestic and wild. Food-borne transmission of the parasite is an important route of infection, particularly for people eating undercooked meat (box 8). Numerous wild species of animals may be infected with *T. gondii* (see Species Susceptibility) without showing clinical signs, thus presenting risks to humans who eat wild game or who skin animals for their fur. Because of the presence of viable organisms in subclinically infected animals (Dubey, 1982, 1983b; Dietz and others, 1993), careful and hygienic pelting practices may mitigate the risk of infection with *T. gondii*. In Texas, discussions are underway to regulate the translocation of feral hogs for hunting purposes because of the possible spread of porcine diseases (Leggett, 2008). If the hogs are also infected with *T. gondii*, they may spread the parasite to new areas and present a risk to the hunters consuming them.

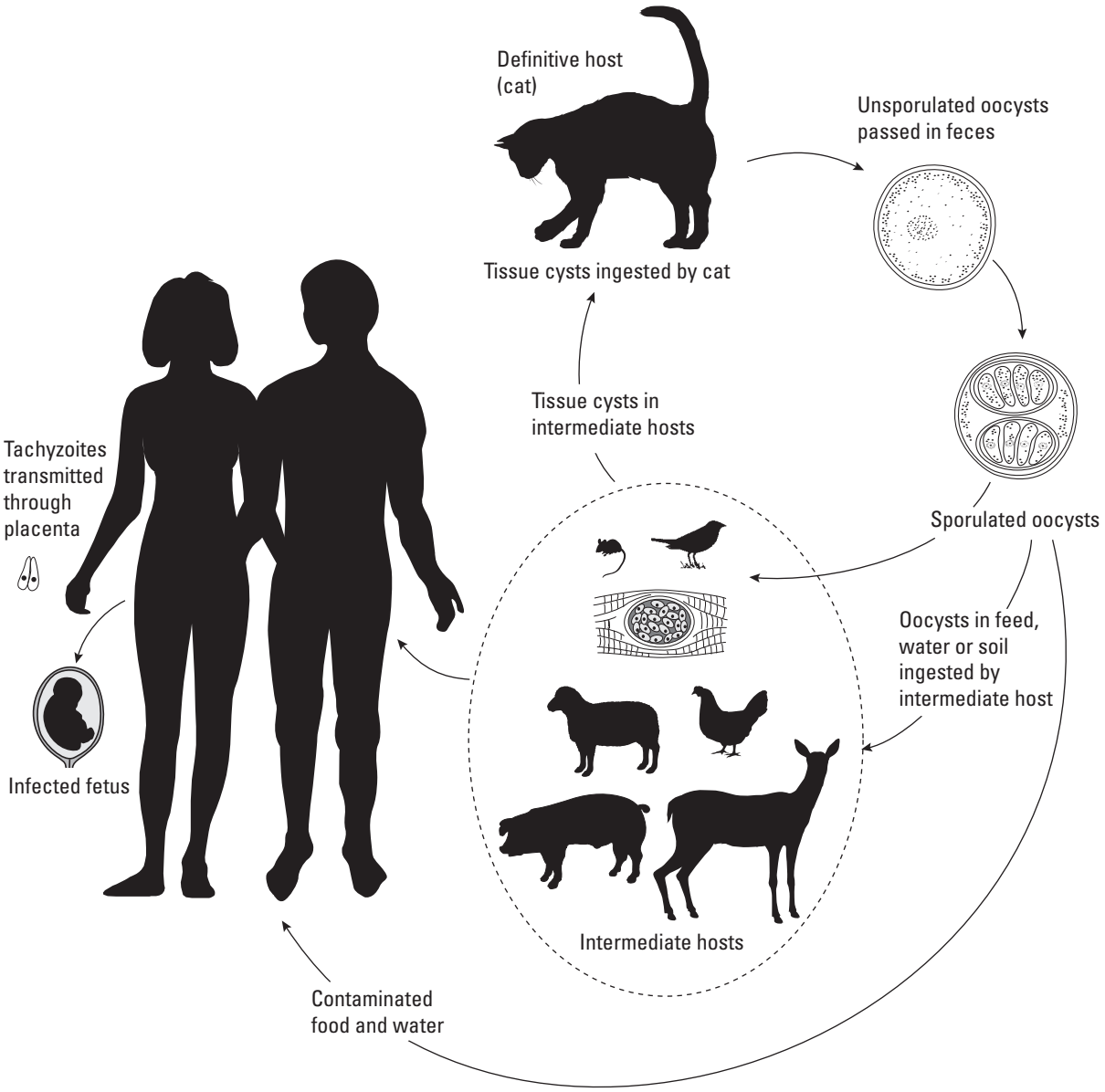


Figure 12. Life cycle of *T. gondii*. (From Dubey and Beattie, 1988)

Box 7 Cats and Toxoplasmosis

Fact or fiction: "The most efficient way to get toxoplasmosis from a cat is to eat the cat undercooked!" (Colville and Berryhill, 2007)

Cats are the most common pet in the United States according to the American Veterinary Medical Association (2007). Thirty-three percent of U.S. households own at least 1 cat, totaling 81 million owned cats (Conrad and others, 2005; Robertson, 2008). Large numbers of pet cats also exist in other countries. There are nearly as many feral cats (approximately 73 million) as there are household cats in the United States; both populations contribute to contamination of the environment with oocysts of *T. gondii* (Dabritz and others, 2006, 2007). Domestic cats, rather than wild species, are probably the major source of contamination because of the large number of owned and feral cats and because oocyst formation is greatest in domestic cats. Cats may excrete millions of oocysts after ingesting as few as one bradyzoite or one tissue cyst, resulting in widespread contamination of the environment (Frenkel and others, 1970; Dubey, 2001).

Countries with the largest numbers of pet cats.

[From Maps of World]

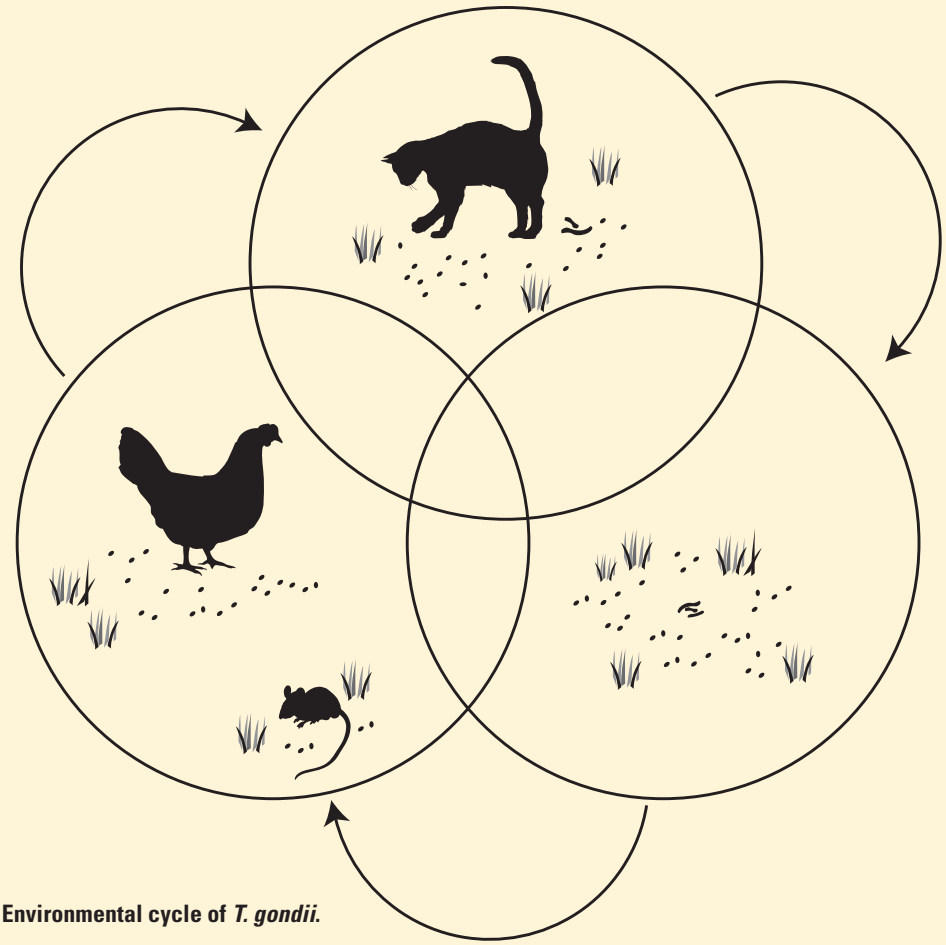
Country	Number of pet cats
United States	76,430,000
China	53,100,000
Russia	12,700,000
Brazil	12,466,000
France	9,600,000
Italy	9,400,000
United Kingdom	7,700,000
Germany	7,700,000
Ukraine	7,350,000
Japan	7,300,000

There are some who feel that the domestic cat is unjustly maligned relative to its role in the **maintenance** and spread of toxoplasmosis in humans (Colville and Berryhill, 2007). That feeling is reflected by the quotation highlighting this highlight box, yet the quotation and the belief are, in fact, untrue. Transmission by oocysts in cat feces is the most efficient means of transmission. Because felids are the only definitive host for *T. gondii*, this parasite could not complete its life cycle without cat species, and, thus, would cease to exist. Sporulated oocysts survive for long periods under most ordinary environmental conditions and for months even in harsh environments. They can survive in moist soil, for example, for months and even years (Dubey and Beattie, 1988). Oocysts in soil can be mechanically transmitted by **invertebrates** such as flies, cockroaches, dung beetles, and earthworms, which can spread oocysts onto human food and animal feeds.

Infection rates in cats are determined by the rate of infection in local avian and rodent populations, because cats are thought to become infected by eating these animals. The more oocysts there are in the environment, the more likely it is that prey animals will be infected, and this in turn will increase the infection rate in cats. Infection among food animals increases the risk of transmission to humans.

In certain areas of Brazil, approximately 60 percent of 6–8-year-old children have antibodies to *T. gondii* linked to the ingestion of oocysts from the environment, which is heavily contaminated with *T. gondii* oocysts (Bahia-Oliveira and others, 2003). The largest recorded outbreak of clinical toxoplasmosis in humans was epidemiologically linked to drinking water from a municipal water reservoir in British Columbia, Canada (Aramini and others, 1998; 1999). This water reservoir was thought to be contaminated with *T. gondii* oocysts excreted by cougars. Although attempts to recover *T. gondii* oocysts from water samples in the British Columbia outbreak were unsuccessful, methods to detect oocysts were reported (Isaac-Renton and others, 1998). An outbreak of toxoplasmosis at a riding stable in Atlanta, Ga., was attributed to either ingestion or inhalation of oocysts from dust in the stable contaminated with cat feces (Teutsch, and others, 1979).

Cat feces contaminated with oocysts are not only an outdoor risk—they are an indoor risk to cat owners who are unaware of the risk of acquiring toxoplasmosis from indoor cats. A diet of only high quality commercial pet food—never raw or undercooked meats—and clean, fresh drinking water will decrease the chance of a pet cat becoming infected. Hunting by pet cats, and their ingestion of infected prey, can be limited by keeping them indoors. Daily cleaning of litter boxes limits the time for newly defecated oocysts to become infectious. Pregnant women and immunocompromised people can decrease their risk of infection by asking other, immunocompetent people to clean litter boxes. Basic rules of hygiene, including washing hands after emptying litter boxes, after handling cats, and before eating can decrease the risk of infection. With proper precautions and education, cat ownership need not be detrimental to one's health or to the environment.



Environmental cycle of *T. gondii*.

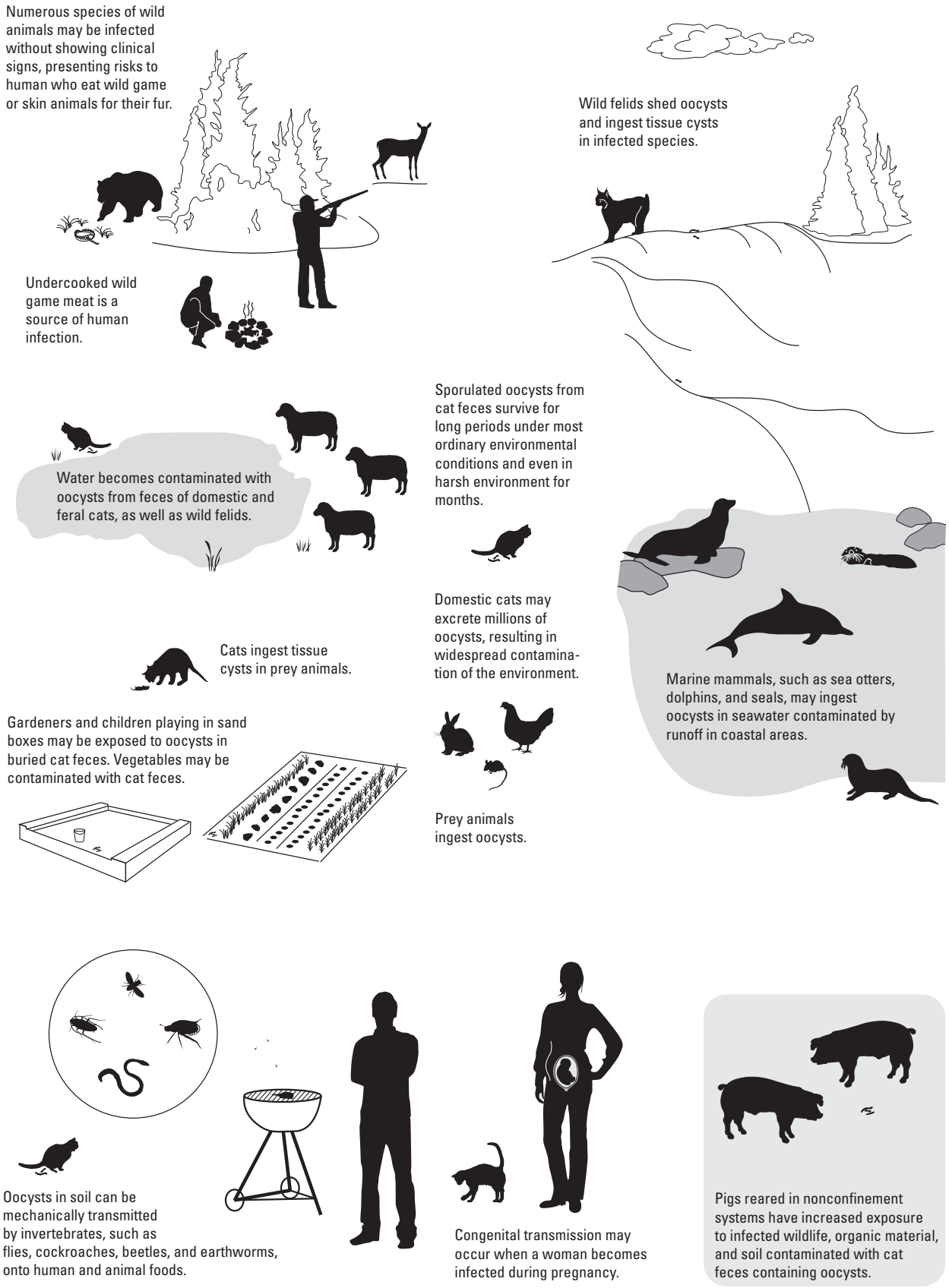


Figure 13. General pathways for infection with *T. gondii*.

Points to Ponder

Toxoplasma gondii is perhaps the most widespread protozoan parasite affecting humans. Despite a significant reduction in the infection rate in the United States, based on a decrease in seroprevalence from 23 percent to 10.8 percent during the last decade (Jones, Kruszon-Moran, and others, 2007), food-borne infections remain a substantial threat due to greater demands for food products raised in animal-friendly systems. However, these systems allow domestic livestock access to the outdoors and wildlife, which is a major risk factor for infection and has led to the reemergence of *T. gondii* infections in pigs (Kijlstra and others, 2004; van der Giessen and others, 2007; Gebreyes and others, 2008). Other food animals, such as sheep, goats, and horses, require outdoor access for grazing, making confinement systems of production impractical. The presence of tissue cysts of *T. gondii* in food animals varies by species with pigs, sheep, and goats having the highest levels followed by poultry, rabbits, dogs, and horses (Tenter, 2009). Cattle and buffalo rarely develop tissue cysts despite having high rates of seroprevalence (Dubey and others, 2005; Tenter, 2009) (fig. 14). Because of the high level of tissue cysts in pigs, sheep, and goats, it can be assumed that meat from these animals will contain cysts if the animals are seropositive (Tenter and others, 2000; Dubey and Jones, 2008).

Outbreaks of acute toxoplasmosis have occurred in various countries as a result of people consuming raw or undercooked meat (table 19). Different cultural and ethnic preferences for meats present varying risks of *T. gondii* infection for people. This may alter the dynamics of toxoplasmosis in the United States as its society continues to incorporate diverse dietary and food preferences. In addition, the increased globalization of society results in travelers to new regions experimenting with novel foods. These explorations may increase food-borne illness risks from sources that are not significant in their home countries. It is prudent for travelers to be aware of the regional differences that exist in the prevalence of *T. gondii* infection in food animals. In Norway, for example, 18 percent of sheep are infected with *T. gondii* compared to 3 percent of pigs (Skjerve and others, 1996, 1998). Consumption of raw or undercooked lamb in that country

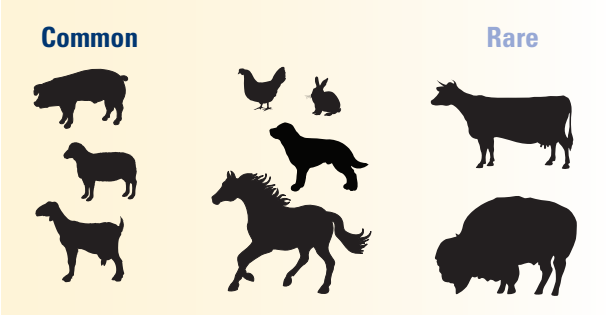


Figure 14. Relative occurrence of tissue cysts in food animals. (Data from Dubey and others, 2005; Tenter, 2009)

is an important risk factor for toxoplasmosis (Kapperud and others, 1996), whereas, in Poland pork presents the primary risk to consumers (Paul, 1998), because 36 percent of pigs are infected (Bartoszcze and others, 1991).

Consumption of wild game meats is regaining popularity, and exotic game meats are becoming more readily available in various parts of the world, often putting consumers at risk of infection due to inadequate quality standards and veterinary inspections. Kangaroo meat, which can be easily purchased over the Internet, is a lean meat that is usually eaten rare. Because more than 20 percent of kangaroos are potentially infected (Kijlstra and Jongert, 2008), consumption of this meat presents a significant risk for people. Many species of wild game in the United States have been found to be seropositive (table 8). Viable tissue cysts have been detected in 17–28 percent of deer in parts of the United States, making consumption of raw or undercooked venison and bear meat risk factors for infection (Sacks and others, 1983; Ross and others, 2001; Dubey and Jones, 2008).

Because prevention of infection by *T. gondii* is not easily accomplished in animals raised outdoors or in wild game, education of consumers of these meat products about proper food handling and preparation can decrease their risk of becoming infected. In addition, suppliers of potentially infected meats can decrease the risk to consumers by adequately treating meat prior to sale.

Table 19. Outbreaks of food-borne acute toxoplasmosis in humans.

Country	Meat consumed
Canada	Raw caribou, dried seal meat, seal liver (McDonald and others, 1990; Pekeles and others, 1991).
Australia	Rare kangaroo, undercooked lamb (Robson and others, 1995).
Brazil	Raw mutton (Bonametti and others, 1997).
Kenya	Raw spleen, liver of wild boar and pig (Choi and others, 1997).

Box 8 Toxoplasmosis as a Food-Borne Infection

In the United States, infection in humans was thought to most commonly result from ingestion of tissue cysts contained in undercooked meat (Dubey and Beattie, 1988; Roghmann and others, 1999; Lopez and others, 2000), though the exact contribution of food-borne toxoplasmosis rather than oocyst-induced toxoplasmosis to human infection is currently unknown. *T. gondii* infection is common in many animals used for food, including sheep, pigs, goats, and rabbits.

In one study, viable *T. gondii* tissue cysts were isolated from 17 percent of 1,000 adult pigs (sows) from a slaughter plant in Iowa (Dubey, Weigel, and others, 1995). Serological surveys of pigs from Illinois pig farms indicated an infection rate of about 3 percent in market weight animals and 20 percent for breeding pigs, suggesting that age is a factor for pigs acquiring *T. gondii* infection (Weigel, Dubey, Siegel, Hoefling, and others, 1995). A seroprevalence of 47.4 percent was reported in swine production facilities in the northeastern United States, where pigs are managed largely in nonconfinement systems (Gamble and others, 1999); viable *T. gondii* was isolated from 51 of 55 finisher pigs from a farm in Massachusetts (Dubey, Gamble, and others, 2002). Another study documented persistent *T. gondii* infection in confinement reared grower/finisher swine on farms that followed good production practices but that did not practice barn-only boot hygiene (H.R.Gamble, J.P. Dubey, and D.E. Hill, unpub. data, 2008); changes to barn-only boot usage eliminated *T. gondii* infection on previously seropositive farms. Poor adherence to biosecurity practices may allow peridomestic species, such as rodents and cats, access to confined pigs, resulting in infection. Rodents serve as **reservoirs** of infection for *T. gondii* as well as other swine diseases (Dubey, Weigel, and others, 1995; Weigel, Dubey, Siegel, Kitron, and others, 1995). Cats, though frequently used for rodent control, are not effective for rodent control in swine production facilities (Timm and others, 1987), and are the only source of infectious oocysts for the environmental contamination that can lead to *T. gondii* infection in swine. An upsurge in consumer demand for "organically raised," "humanely raised," and "free range" pork products has resulted in increasing numbers of hogs being raised in nonconfinement systems (Honeyman and others, 2006) to fulfill a consumer demand that has increased 20 percent per year in sales since 1990 (Dimitri and Greene, 2002). National Organic Program (NOP) standards (*www.ams.usda.gov/nop/*) require that all organically raised animals must have access to the outdoors, including access to pasture for ruminants. Although "humanely raised" and "free range" products have standards that are less stringently defined, outdoor access is also considered a requirement for labeling. These practices substantially increase the risk of exposure of pigs to *T. gondii*.

In a nationwide survey of retail chicken, beef, and pork in the United States, only pork was found to harbor viable *T. gondii* tissue cysts; viable tissue cysts were isolated from 0.38 percent of pork samples, and 0.57 percent of samples had antibodies to *T. gondii* (Dubey and others, 2005). In this study, the northeastern United States had a higher number of positive pork samples than other regions of the country, reflecting the higher risk of pig infection due to regional management practices. Panama serves as an example of this relationship by having one of the highest infection rates in swine, up to 60 percent in some regions (Correa and others, 2008). This high prevalence emphasizes the importance of good farming practices and safe food preparation to eliminate the parasite. In a recent study of lambs destined for human consumption in the United States, 27 percent of lambs were seropositive for antibodies to *T. gondii* (Dubey, Sundar, and others, 2008). Viable *T. gondii* was not found in a nationwide survey of retail chicken in the United States, though 1.5 percent of samples were seropositive (Dubey and others, 2005); however, most chicken in the United States is cooled to near freezing or is completely frozen at the packing plant (Chan and others, 2001), which would kill organisms in tissue cysts (Kotula and others, 1991).

Infection in cattle is less prevalent than is infection in sheep or pigs in the United States. However, recent surveys in several European countries using serology and polymerase chain reaction (**PCR**) to detect parasite deoxyribonucleic acid (DNA) have shown that infection rates in pigs and horses are negligible, but infection in sheep and cattle ranges from 1 to 6 percent (Tenter and others, 2000;

Wyss and others, 2000). Serological surveys in eastern Poland revealed that 53 percent of cattle, 15 percent of pigs, and 0–6 percent of chickens, ducks, and turkeys were positive for *T. gondii* infection; nearly 50 percent of the people in the region were also serologically positive for *T. gondii* infection (Sroka, 2001). Dubey, Graham, and others (2002) isolated Type I strains of *T. gondii* from 30 percent of 82 domestic free range chickens from rural areas of Brazil, the first report of isolation of predominantly Type I strains of *T. gondii* from a food animal.

T. gondii infection is also prevalent in game animals. Among wild game in the United States, *T. gondii* infection is most prevalent in black bears and in white-tailed deer. Serological surveys of white-tailed deer in the United States have demonstrated seropositivity of 30–60 percent (Lindsay and others, 1991; Humphreys, and others, 1995; Vanek and others, 1996). One study reported the occurrence of clinical toxoplasmosis and necrotizing retinitis (severe inflammation of retina leading to tissue death) in deer hunters with a history of consuming undercooked or raw venison (Ross and others, 2001). Approximately 80 percent of black bears are infected in the United States (Dubey, Humphreys, and Thulliez, 1995), and about 60 percent of raccoons have antibodies to *T. gondii* (Dubey, Hamir, and others, 1992; Dubey and Odening, 2001). Because raccoons and bears **scavenge** for their food, infection in these animals is a good indicator of the prevalence of *T. gondii* in the environment. Edelhofer and others (1996) used the indirect hemagglutination test (IHAT) to detect *T. gondii* in wild pigs in Austria and found antibodies in 19 percent of 264 wild pigs; Dubey and others (1997) found similar prevalence (18 percent) in 170 feral pigs from the United States. Choi and others (1997) reported an outbreak of toxoplasmosis in humans in Korea acquired by eating meat from a feral pig. Thus, the potential for human infections after eating undercooked wild game appears to be high.

Virtually all edible portions of an animal can harbor viable *T. gondii* tissue cysts, and tissue cysts can survive in food animals for years. The number of *T. gondii* tissue cysts in meat from food animals is very low. It is estimated that as few as one tissue cyst may be present in 100 grams of meat. Because it is not practical to detect this low level of *T. gondii* infection in meat samples, digestion of meat samples in trypsin or pepsin is used to concentrate *T. gondii* tissue cysts for detection (Dubey, 1988). This process ruptures the *T. gondii* tissue cyst wall, releasing hundreds of bradyzoites. The bradyzoites survive in the digests for several hours. Even in the digested samples, only a few *T. gondii* are present and their identification by direct microscopic examination is not practical. Therefore, the digested material is bioassayed in mice (Dubey, 1988). The mice **inoculated** with digested material have to be kept for 6–8 weeks before *T. gondii* infection can be detected reliably—this procedure is not practical for mass scale samples. The detection of *T. gondii* DNA in meat samples by PCR has been reported (Warnekulasuriya and others, 1998; Jauregui and others, 2001); however, comparison of PCR methods of *T. gondii* detection to existing serological methods and bioassays demonstrated the relative insensitivity of PCR in detecting *T. gondii* in tissues (Hill and others, 2006).

Cultural habits of people may affect the acquisition of *T. gondii* infection (Cook and others, 2000). For example, in France the prevalence of antibody to *T. gondii* is very high in humans. Although 84 percent of pregnant women in Paris have antibodies to *T. gondii*, only 32 percent in New York City and 22 percent in London have such antibodies (Dubey and Beattie, 1988). The high incidence of *T. gondii* infection in humans in France appears to be related in part to the French habit of eating some meat products undercooked or uncooked. In contrast, the high prevalence of *T. gondii* infection in Central and South America is probably due to high levels of contamination of the environment with oocysts (Dubey and Beattie, 1988; Glasner and others, 1992; Neto and others, 2000). This said, however, it should be noted that the relative frequency of acquisition of toxoplasmosis from eating raw meat and that due to ingestion of food or water contaminated by oocysts from cat feces is very difficult to determine and, as a result, statements on the subject are at best controversial. There are no tests at the present time to determine the source of infection in a given person.

There is little, if any, danger of *T. gondii* infection by drinking cow's milk and, in any case, cow's milk is generally pasteurized or even boiled, but infection has followed drinking unboiled goat's milk (Dubey and Beattie, 1988). Raw hens' eggs, though an important source of *Salmonella* infection, are extremely unlikely to transmit *T. gondii* infection.

Disease Prevention and Control

Human Infections

The stages of *T. gondii* present in meat are killed by contact with soap and water (Dubey and Beattie, 1988; Lopez and others, 2000). To prevent infection of humans by *T. gondii*, thorough washing of hands after handling raw meat is essential. Thorough washing with soap and water of all cutting boards, sink tops, knives, and other materials coming in contact with uncooked meat will kill *T. gondii* transferred from the meat to those materials. *T. gondii* organisms in meat can also be killed by exposure to extreme cold or heat. Tissue cysts in meat are killed by heating the meat throughout to 67 °C for at least 4 minutes (Dubey, Kotula, and others, 1990). *T. gondii* in meat is killed by cooling to -13 °C for 3 days (Kotula and others, 1991) and by exposure to 400 grays of gamma **irradiation** (Dubey and Thayer, 1994). It is recommended that the meat of any animal is cooked to 67 °C before consumption and that tasting meat while cooking or seasoning is avoided. Because of the risk associated with *T. gondii* stages in meat and oocyst contamination of soil, pregnant women can decrease the risk of exposure to *T. gondii* by avoiding contact with cats, soil, and raw meat.

Cats are the key to the transmission of *T. gondii*. Adherence to good hygienic measures appears to be the most practical and effective method available to minimize transmission of *T. gondii* to humans. A diet of only dry, canned, or cooked food will prevent the infection of pet cats and the subsequent excretion of oocysts. Daily emptying of the cat litter box, preferably not by a pregnant woman, will rid the litter box of excreted oocysts. The use of gardening gloves will prevent hand exposure to oocysts in buried cat feces. Thorough washing of vegetables before eating will clean them of contamination with cat feces. Education of expectant mothers will increase their awareness of the dangers of toxoplasmosis (Foulon and others, 1994, 2000). At present no commercial reagents are available for detecting *T. gondii* oocysts in the environment.

Animal Infections

Control of toxoplasmosis in animals depends on preventing infection of cats and limiting the contact between cats and other species. Cat populations on farms can be controlled by spaying and neutering programs. Systems of confinement rearing for domestic animals, such as pigs, can be implemented to limit contact with cats and the contamination of local environments with infective oocysts. Infection of pregnant sheep and goats can be prevented by sequestering them from cats.

Infection of zoo animals with *T. gondii* can be prevented by housing cats, including all wild Felidae, in separate buildings from other animals, particularly marsupials and New World monkeys. As with domestic cats, the risk of infection of wild, captive felids will be decreased if they are not fed uncooked meat, unless frozen. Proper disinfection and autoclaving of equipment used to clean cat cages and enclosures will kill oocysts. Control of feral cats within zoos is also important.

Prevention of toxoplasmosis within domestic poultry operations requires management practices that eliminate the source of infective tachyzoites and oocysts transmitted by rodents, **coprophagic arthropods**, and cats. Disinfection procedures include the use of ammonia followed by drying and a temperature of 55 °C (Springer, 1991). Similar disinfection procedures decrease the risk of infection of captive-reared game and wild birds; other preventive measures include the avoidance of crowding and **cannibalism** and the use of serologic testing and postmortem evaluations to determine causes of mortality in birds that die (Sanger, 1971).

Unfortunately, *T. gondii* oocysts are ubiquitous in the environment, are quite resistant to environmental insults, and can persist for years (Dabritz and others, 2006, 2007). The enormous (60–80 million) uncontrolled population of feral cats in the United States makes elimination of oocysts from the environment virtually impossible.

Currently, a noncyst forming isolate of *T. gondii* (S48) is used as a vaccine to prevent abortion in sheep (Buxton and Innes, 1995), and several recombinant vaccines and transformed bacteria expressing *T. gondii* surface **antigens** have been described (Innes and Vermeulen, 2006; Gatkowska and others, 2008; Jongert and others, 2008). None are available for use in humans.

Periodic monitoring of harvested free-ranging wildlife populations for the prevalence of *T. gondii* and the use of information acquired from monitoring can be used, where warranted, in hunter education programs. Doing so in a manner that provides context relative to human health risks can help to minimize risks associated with processing game in the field and in preparing game for consumption. Care taken when dressing game animals can prevent exposure to raw meat and tissue fluids.

Treatment

Sulfadiazine and pyrimethamine (Daraprim) are two drugs widely used for treatment of toxoplasmosis (Guerina and others, 1994; Chirgwin and others, 2002). Although these drugs have a beneficial action during the acute stage of the disease process when the parasite is actively multiplying, they will not usually eradicate infection. It is believed that these drugs have little effect on subclinical infections, but the growth of tissue cysts in mice has been restrained with sulfonamides. Certain other drugs, diaminodiphenylsulfone, atovaquone, spiramycin, and clindamycin, are also used to treat toxoplasmosis in difficult cases.

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Glossary

A

acute Sharp or severe, such as an illness with a sudden onset and a relatively short course.

antibody A protein formed in the body of a vertebrate that is used by the immune system to identify and neutralize the effects of foreign invading proteins, called antigens, such as bacteria and viruses.

antigen Any foreign substance (generally proteins) to which the body reacts by producing antibodies. Antigens may be soluble substances such as toxins, particulate matter such as pollen, or microorganisms such as bacteria or viruses.

arthropod A member of the phylum Arthropoda, invertebrate animals that have exoskeletons, segmented bodies, and jointed legs, including insects, crabs, spiders, etc. (*See also* invertebrates.)

asymptomatic infection An infection that is present in an organism but that does not show the symptoms that usually appear with it. (*See also* infection, symptom, *and* subclinical.)

ataxia Lack of coordination.

autoimmunodeficiency syndrome (AIDS) An infectious disease complex resulting from infection with human immunodeficiency virus (HIV). (*See also* disease *and* infectious.)

B

bioassay A test performed to measure the effects of a substance on a living organism, including determination of the presence of a disease-causing agent by injection of material into a susceptible host. (*See also* disease *and* host.)

biopsy A test involving the removal of cells or tissues from a living subject for examination, usually by microscope, to determine the presence or extent of a disease. (*See also* disease.)

bird A warm-blooded vertebrate, belonging to the Class Aves, with wings and feathers (although the wings are poorly developed for some flightless species like ostriches).

C

calcification The hardening of a tissue by deposition of calcium salts.

cannibalism The act of eating the flesh of one's own species.

carnivore A mammal with teeth and other body adaptations for feeding on flesh; primarily species belonging to the Order Carnivora (for example, wolves, bears, raccoons, weasels, civets, hyenas, and tigers).

carnivorous Eating flesh.

central nervous system (CNS) The brain and spinal cord.

cervid A mammal within the Family Cervidae, for example, deer, elk, moose, and caribou.

choroid The tissue layer of the eye between the sclera and retina containing numerous blood vessels.

chronic Persisting for a relatively long time.

clinical signs Readily observable indications of a disease or injury. (*See also* disease *and* symptom.)

coccidian Pertaining to Coccidia, an order of protozoa of the subphylum Sporozoa, containing organisms commonly parasitic in the epithelial cells of the intestinal tract, but also found in the liver and other organs. (*See also* parasite *and* parasitic.)

cohort A group of individuals possessing a common characteristic and studied over time.

cold-blooded Species such as fishes and reptiles, which have blood that varies in temperature to approximately that of the surrounding environment. (*See also* fish *and* reptile.)

congenital Acquired during fetal development and present at birth.

convulsion A seizure or violent involuntary contraction or series of contractions of the voluntary muscles.

coprophagic Ingesting dung or feces.

corticosteroid A class of steroid hormones that are produced in the adrenal cortex or synthetically and are used as anti-inflammatory agents and to suppress the immune response. (*See also* inflammatory.)

corvid A bird within the Family Corvidae (for example, crows, ravens, rooks, jackdaws, jays, and magpies). (*See also* bird.)

cyst A stage in the life cycle of certain parasites, during which they are enclosed within a protective wall.

D

dedifferentiation A loss of differentiation of cells and of their orientation to one another.

definitive host An organism in which sexually mature stages of a parasite occur. (*See also* host *and* parasite.)

direct agglutination test A laboratory test in which whole organisms are mixed with serum to detect specific antibodies against the organism; clumping within the solution indicates a positive result. (*See also* sera/serum.)

disease An abnormal condition of an animal or plant that causes specific signs or symptoms. (*See* symptom.)

disseminated Distributed throughout, such as the body.

DNA (deoxyribonucleic acid) The carrier of genetic information for all organisms except the ribonucleic acid (RNA) viruses and found in all living cells.

domestic Pertaining to an environment managed by humans.

E

ELISA (enzyme-linked immunosorbent assay) A molecular-based test used to detect the presence of either antigen or antibody in a sample.

emergence Referring to the new appearance and(or) increase in frequency of occurrence within the past three decades of infectious diseases, or the threat of an increase in the near future relative to populations affected, geographic distribution, or magnitude of effects. (*See also* disease, infectious, *and* reemergence.)

encapsulated Enclosed within a protective cover.

encephalitis Inflammation of the brain.

endemic A disease that is commonly present within a human population or a geographical area or having the quality of being constantly present in a human population in a specific location. (*See also* disease.)

enteritis Inflammation of the intestine.

enteroepithelial Pertaining to the cells lining the intestine, in which the sexual cycle of *Toxoplasma gondii* takes place in the cat.

enzyme A substance, usually a protein, produced by the body to facilitate a chemical reaction.

epidemic An outbreak of disease affecting a disproportionately large number of humans within a population, community, or region during a period of time. (*See also* disease *and* outbreak.)

epidemiological Pertaining to epidemiology. (*See also* epidemiology.)

epidemiology The study of the causes, occurrence, and control of disease in populations. (*See also* disease.)

epithelial Pertaining to the cells that cover the external and internal surfaces of the body. (*See also* epithelium.)

epithelium The cellular covering of the internal and external surfaces of the body and organs.

epizootic An outbreak of disease affecting a greater number of animals than normal; typically involving many animals in the same region at the same time. (*See also* disease *and* outbreak.)

equid A mammal within the Family Equidae (for example, horses, wild horses, donkeys, and zebras), which has legs ending in single, hoof-covered toes. (*See also* mammal.)

eradicate To completely eliminate.

F

fecal-oral route/transmission Passing a disease from one individual to another via direct or indirect ingestion of feces containing pathogens. (*See also* disease *and* pathogen.)

felids Mammals within the Family Felidae (for example, domestic cats, lion, tiger, leopard, lynx, cheetah, and many other wild cats). (*See also* mammal.)

feline Pertaining to members of the cat family (Felidae).

feral Typically, animals that have descended from tame stock and are now sustaining themselves in nature.

fish Refers to finfish (for example, cold-blooded strictly aquatic vertebrates with a well differentiated skull and a bony skeleton), in contrast to shellfish (invertebrates) and jawless fishes. (*See also* vertebrates.)

G

genotype The genetic makeup of a cell, an organism, or an individual usually with reference to a specific character under consideration.

H

hemiparesis Muscular weakness or partial paralysis affecting one side of the body. (*See also* paralysis.)

histologic Pertaining to histology. (*See also* histology.)

histology The study of the microscopic structure of tissues.

host An organism that harbors or nourishes microbes, viruses, and parasites. (*See also* parasite.)

humoral Pertaining to or derived from a body fluid, especially serum. (*See also* sera/serum.)

hydrocephalus An abnormal expansion of the cavities (ventricles) within the brain that is caused by the accumulation of cerebrospinal fluid and is characterized by enlargement of the head and deterioration of the brain.

I

immunoblotting A laboratory test in which proteins are separated by their lengths using gel electrophoresis. The proteins are then transferred to cellulose sheets and identified by staining with specific antibodies. The test can also be used to identify the presence of antibodies to specific antigens in serum samples. Also known as Western blotting. (*See also* antibody *and* sera/serum.)

immune Protected against a particular disease by biological defenses that prevent pathogens from causing disease in the organism. (*See also* disease *and* pathogen.)

immune system A system of biological structures and processes within an organism that protect it from disease. (*See also* disease.)

immunocompetent Capable of developing a normal immune response. (*See also* immune.)

immunocompromised Incapable of developing a normal immune response, usually as a result of disease, malnutrition, or immunosuppressive therapy. (*See also* disease, immune, *and* immunosuppressive agents.)

immunological Relating to immunology, the study of the structure and function of the immune system. (*See also* immune system.)

immunosuppressed Incapable of developing a normal immune response, usually as a result of disease, malnutrition, or immunosuppressive therapy. (*See also* disease *and* immune.)

immunosuppressive agents Drugs that are used to inhibit or prevent activity of the immune system in patients undergoing organ transplantation and treatment of autoimmune diseases. (*See also* disease *and* immune system.)

incidence The probability of a new case of a specific disease developing in a population at risk during a specified time period. (*See also* disease.)

infection The invasion by and reproduction of microorganisms in body tissues.

infectious Capable of causing infection, of being passed to another organism by pathogens that enter the body, or the condition of suffering from a disease that can be passed to another organism. (*See also* disease *and* pathogen.)

infective Capable of producing infection. (*See also* infection.)

inflammatory Pertaining to or characterized by inflammation, the reaction of tissue to injury or infection that is characterized by redness, pain, swelling, and heat. (*See also* infection.)

inoculate To inject material suspected of containing a disease-causing agent into an animal; development of disease in the animal confirms the presence of the pathogen in the sample. (*See also* disease *and* pathogen.)

intermediate host An organism in or on which the larval stage of a parasite develops but does not sexually reproduce. (*See also* host, larva/larval, *and* parasite.)

intracellular Within a cell or cells.

intracerebral calcification Hardening of the brain tissue by infiltration of cells by calcium or calcium salts.

invertebrates Animals lacking a spinal column (for example, insects, crustaceans). (*See also* vertebrates.)

irradiation The use of gamma rays, X-rays, or electron beams to sterilize food.

isolate A bacterial or fungal strain that has been separated as a pure strain from a mixed bacterial or fungal culture. (*See also* strain.)

J

K

keystone species A species that significantly influences its ecosystem in ways that are not entirely replicated by other species and are disproportionate to its abundance (for example, beaver, sea otter, prairie dog).

L

larva/larval The immature, early form of an organism that at birth or hatching is not like its parent and has to undergo a series of form and size changes before assuming adult features.

latent In a dormant or hidden stage.

lesion An abnormal change in tissue or an organ due to disease or injury. (*See also* disease.)

leukocytes White blood cells that act to protect the body from disease-causing viruses, bacteria, toxins, parasites, and tumor cells. (*See also* disease *and* parasite.)

lymph nodes Rounded, encapsulated aggregations of lymphoid tissue distributed throughout the body along the lymphatic system. These nodes filter the flow of lymph circulating within the body. (*See also* encapsulated *and* lymphatics.)

lymphadenopathy Disease (infection, autoimmune disease, or tumor) of the lymph nodes, often used synonymously with "swollen/enlarged lymph nodes." (*See also* disease, infection, *and* lymph nodes.)

lymphatics The lymphatic system, a vast network of tubes transporting lymph, a clear, watery, sometimes faintly yellowish fluid derived from body tissues that contains white blood cells and acts to remove bacteria and certain proteins from the tissues, transport fat from the small intestine, and supply mature lymphocytes to the blood.

lymphocyte A type of white blood cell important for producing antibodies (B-lymphcytes) and cellular immunity (T-lymphocytes). (*See also* antibody.)

M

macrophage A type of white blood cell found in tissues that acts to engulf and digest cellular debris and pathogens and to stimulate the immune system in response to foreign material. (*See also* immune system *and* pathogen.)

maintenance The act of continuing or keeping in existence, for example, a maintenance host—an organism that keeps a disease agent in existence in nature and is a source of infection for susceptible hosts. (*See also* disease, host, *and* infection.)

mammal **A** warm-blooded vertebrate animal that possesses hair during some part of its life and suckles its young. (*See also* vertebrates *and* warm-blooded.)

marsupials Mammalian species having an external abdominal pouch (marsupium) enclosing the mammary glands for carrying their young until their development is complete; young of these species are born in a very underdeveloped state and must be carried and nourished for a prolonged period of time (for example, opossums, kangaroos, koala, and wombats). (*See also* mammal.)

meningoencephalitis Inflammation of the meninges (the layer of tissue covering the brain and spinal cord) and the brain.

mesenteric Pertaining to the mesentery, the double layer of tissue that suspends the intestine from the abdominal wall.

microcephalus An abnormally small head.

morbidity A diseased condition, or the severity or incidence of a disease. (*See also* disease.)

morphology The study of the form and structure of an organism or its parts.

mortality Susceptible to death; death.

mucosa The thin layer of tissue that lines body cavities and passages; mucous membrane.

mummification The drying and shriveling of an organism or dead fetus within the uterus.

myocarditis Inflammation of the heart muscle.

N

necropsy Examination of a dead animal body to determine the cause of death.

necrosis Cell or tissue death.

necrotizing Causing the death of cells or tissues.

neuritis Inflammation of a nerve or group of nerves that is characterized by pain, loss of reflexes, and atrophy of the affected muscles.

New World monkeys Members of the four families of primates that are found in Central and South America: Cebidae, Aotidae, Pitheciidae and Atelidae. New World monkeys have flatter noses than Old World monkeys,

with side facing nostrils. Most have long tails that are often prehensile and almost all live in trees. The group includes marmosets, tamarins, capuchins, squirrel, owl, howler, and spider monkeys.

O

ocular Pertaining to the eye.

Old World monkeys Members of the primate family Cercopithecidae native to Africa and Asia. Old World monkeys have downward facing nostrils and non-prehensile tails. The group includes macaques, baboons, colobus monkeys, langurs, vervet monkey, and proboscis monkey.

organelle A specialized, membrane-bound structure within a cell with a particular function, such as mitochondrion, nucleus, ribosome, and flagellum.

outbreak The occurrence of a specific disease within a small, localized group of people or organisms. (*See also* disease.)

P

paralysis The loss of muscle function resulting in the inability to move affected body parts.

parasite An organism that lives in or on another organism of a different species from which it derives nutrients and shelter.

parasitic Pertaining to parasites. (*See also* parasite).

parasitophorous vacuole A small membrane-bound space within a host cell in which a *T. gondii* tachyzoite develops. (*See also* host.)

parenteral Located outside the digestive tract.

pathogen Typically, microorganisms capable of inducing disease, but broadly includes all disease-inducing agents. (*See also* disease.)

pathogenic Able of producing disease. (*See also* disease.)

pathogenicity The ability of an agent to produce disease in another organism. (*See also* disease.)

pathologist A person who studies pathology (*See also* pathology.)

pathology The study of the structural and functional effects of disease. (*See also* disease.)

PCR (polymerase chain reaction) A laboratory technique used to amplify exponentially a single or few copies of a selected sequence of DNA using enzymatic replication of the DNA in order to generate millions or more copies of a particular DNA sequence. (*See also* DNA.)

pneumonitis Inflammation of the lung.

predator An animal that preys on other animals as a source of food.

prevalence The total number of cases of a disease divided by the number of hosts examined in a population at a given time. (*See also* disease *and* host.)

prevalent Widely or commonly occurring.

proteolytic Relating to substances that break down proteins.

protozoan One-celled organisms with recognizable nucleus, cytoplasm, and cytoplasmic structures, such as amoebas, ciliates, flagellates, and sporozoans.

Q

R

recombinant Formed by recombination. (*See also* recombination.)

recombination The process in which strands of DNA are rearranged by breakage and reattachment to create new arrangements of genes within the chromosomes, either naturally or artificially. (*See also* DNA.)

reemergence Referring to the reappeaance of infectious diseases that were once major health problems, but that had declined dramatically and have again become major health problems. (*See also* disease, infectious, *and* emergence.)

reptile A vertebrate of the Class Reptilia that breathes by means of lungs and has external coverings of scales or bony plates; includes snakes, lizards, crocodiles, turtles, and dinosaurs. (*See also* vertebrates.)

reservoir The host population that maintains the disease agent in nature and provides a source of infection to susceptible hosts. (*See also* disease *and* host.)

resistance The ability of an organism to remain either uninfected or disease-free despite becoming infected by a disease-causing organism; the ability of an organism, a tissue, or a cell to tolerate the effects of a harmful physical or environmental condition. (*See also* disease.)

retina The thin layer of neural cells lining the back of the eye.

retinitis Inflammation of the retina. (*See also* retina.)

retinochoroiditis Inflammation of both the retina and choroid. (*See also* retina *and* choroid.)

rodent A member of the Order Rodentia, a diverse group of mammals characterized by incisor teeth that grow throughout life and must be worn away by cutting and gnawing hard materials; includes squirrels, mice, rats, voles, chipmunks, gophers, lemmings, beaver, porcupines, and many others. (*See also* mammal.)

ruminant An even-toed, hoofed mammal that has a complex four-chambered stomach and chews the cud; includes cattle, sheep, goats, and deer. (*See also* mammal.)

S

scavenge To feed on dead or decaying matter.

schizophrenia A psychiatric disorder marked by severely impaired thinking, emotions, and behavior.

sensitivity The proportion of individuals with a disease who are correctly diagnosed by a particular test. (*See also* disease.)

sera/serum The pale fluid that remains after blood has clotted.

seroconversion Development of detectable specific antibodies in the blood serum in response to immunization or infection. (*See also* antibody, infection, *and* sera/serum.)

serologic Pertaining to serology. (*See also* serology.)

serology Laboratory evaluations of the serum portion of blood for the purpose of detecting and measuring host antibody response to infectious agents and other antigens. (*See also* antibody, antigen, *and* infectious.)

seronegative The absence of a specific antibody in the blood as determined by a laboratory test. (*See also* antibody.)

seropositive The presence of a specific antibody in the blood as determined by a laboratory test. (*See also* antibody.)

seroprevalence The total number of cases of a disease based on the results of serologic tests divided by the number of hosts examined in a population at a given time. (*See also* disease, serologic, *and* host.)

specificity The proportion of individuals without a disease who are correctly identified as negative by a particular test. (*See also* disease.)

strain A genetically or biochemically distinguishable subtype of a microorganism.

subclinical Pertaining to an inapparent, asymptomatic infection. (*See also* asymptomatic infection *and* clinical signs.)

surveillance The systematic collection, analysis, and interpretation of data pertaining to the occurrence of specific diseases for the purpose of monitoring morbidity and mortality trends. (*See also* disease, morbidity, *and* mortality.)

susceptibility/susceptible Pertaining to the ability of an animal to become infected by a disease-causing organism or to develop disease after becoming infected. (*See also* disease.)

swine Pigs, hogs, and boars.

symptom A subjective indication of a disorder or disease perceived by the patient, such as pain, nausea, fatigue, or weakness. (*See also* disease.)

syndrome A group of signs and symptoms characterizing a specific disease or condition. (*See also* disease *and* symptom.)

T

taxonomic Pertaining to taxonomy. (*See also* taxonomy.)

taxonomy The systematic principles and procedures of grouping and arranging organisms into a hierarchical order.

transplacental Through the placenta, the organ in mammals that allows exchange of nutrients and wastes between the mother and fetus. (*See also* mammal.)

U

ungulate A mammal that has hooves. The even-toed hoofed species (Artiodactyla) include deer, antelope, cattle, and sheep; the odd-toed hoofed mammals (Perissodactyla) include horses, tapirs, and rhinoceroses. (*See also* mammal.)

uveitis Inflammation of the uvea, the middle layer of the eye that lies between the sclera and retina and is divided into the anterior uvea (iris and ciliary body) and the posterior uvea (choroid). (*See also* choroid *and* retina.)

V

vertebrates Animals that have spinal columns, including mammals, birds, reptiles, amphibians, and fish. (*See also* bird, fish, mammal, *and* reptile.)

virulence The degree or ability of a pathogenic organism to cause disease. (*See also* disease *and* pathogenic.)

viscera The internal organs, particularly of the thoracic and abdominal cavities, such as heart, lungs, liver, kidneys, and intestines.

visceral Pertaining to viscera (*See also* viscera).

viviparous Bearing living offspring rather than laying eggs.

W

warm-blooded Species, such as birds and mammals, that have a constant body temperature, independent of the surrounding environment. (*See also* bird *and* mammal.)

X

Y

Z

zoologist A person who studies animals.

zoonosis Infectious disease transmissible between animals and humans, and vice versa. (*See also* disease.)

zoonotic Transmissible between animals and humans, and vice versa.

Appendix 1. Common and Scientific Names for Species Cited

Common name	Scientific name
Mammals	
Arctic fox	*Vulpes lagopus*
Arctic hare	*Lepus arcticus*
Badger (European)	*Meles meles*
Bandicoot (Eastern barred)	*Perameles gunnii*
Bear	*Ursus* spp.
Bearded seal	*Erignathus barbatus*
Beluga whale	*Delphinapterus leucas*
Bennett's wallaby (red-necked wallaby)	*Macropus rufogriseus*
Bison	*Bison bison*
Black bear	*Ursus americanus*
Black-faced kangaroo (western grey)	*Macropus fuliginosus*
Black-flanked rock wallaby	*Petrogale lateralis*
Bobcat	*Lynx rufus*
Bottlenose dolphin	*Tursiops truncatus*
Brown bear	*Ursus arctos arctos*
Brown hare	*Lepus europaeus*
California sea lion	*Zalophus californianus*
Camel	*Camelus* spp.
Canadian cougar (North American)	*Puma concolor cougar*
Caribou	*Rangifer tarandus*
Cat (domestic)	*Felis catus*
Cattle (domestic)	*Bos taurus*
Cheetah	*Acinonyx jubatus*
Chinchilla	*Chinchilla* spp.
Common dolphin	*Delphinus delphis*
Cougar	*Puma concolor*
Coyote	*Canis latrans*
Cuvier's gazelle	*Gazella cuvieri*
Dama gazelle	*Gazella dama*
Dasyurid (kowari and mulgara)	*Dasyuroides byrnei* and *Dasycercus cristicauda*

Common name	Scientific name
Mammals—Continued	
Dik-dik	*Madoqua guentheri*
Dog (domestic)	*Canis lupus familiaris*
Duck (mallard)	*Anas platyrhynchos*
Eastern barred bandicoot	*Perameles gunnii*
European lynx (Eurasian lynx)	*Lynx lynx*
Ferret (European polecat)	*Mustela putorius furo*
Fox	*Vulpes* spp.
Gerenuk	*Litocranius walleri*
Goat (domestic)	*Capra hircus*
Gray fox	*Urocyon cinereoargenteus*
Greenland seal (harp seal)	*Pagophilus groenlandicus*
Gray seal	*Halichoerus grypus*
Grizzly bear	*Ursus arctos horribilis*
Gundi	*Ctenodactylus gundi*
Harbor seal	*Phoca vitulina*
Harp seal	*Pagophilus groenlandicus*
Hawaiian monk seal	*Monachus schauinslandi*
Hooded seal	*Cystophora cristata*
Horse (domestic)	*Equus caballus*
Human	*Homo sapiens*
Indo-Pacific bottlenose dolphin	*Tursiops aduncus*
Iriomote cat	*Prionailurus iriomotensis*
Jackal (golden)	*Canis aureus*
Jaguar	*Panthera onca*
Koala	*Phascolarctos cinereus*
Lion	*Panthera leo*
Lynx (Canadian)	*Lynx canadensis*
Mink (American)	*Neovison vison*
Moose	*Alces alces*
Mountain gazelle	*Gazella gazella*
Mule deer	*Odocoileus hemionus*
Narwhal	*Monodon monoceros*
Northern fur seal	*Callorhinus ursinus*
Ocelot	*Felis pardalis*
Opossum (Virginia)	*Didelphis virginiana*

Common name	Scientific name
Mammals—Continued	
Pacific walrus	*Odobenus rosamarus*
Pallas cat	*Felis manul*
Pig (domestic)	*Sus scrofa*
Polar bear	*Ursus maritimus*
Pronghorn	*Antilocapra americana*
Rabbit	family Leporidae
Raccoon dog	*Nyctereutes procyonoides*
Raccoon	*Procyon lotor*
Rat	*Rattus* spp.
Red deer (elk, wapiti)	*Cervus elaphus*
Red fox	*Vulpes vulpes*
Red kangaroo	*Macropus rufus*
Reindeer	*Rangifer tarandus*
Rhim gazelle	*Gazella leptoceros*
Ribbon seal	*Histriophoca fasciata*
Ringed seal	*Pusa hispida*
Risso's dolphin	*Grampus griseus*
Roe deer (western)	*Capreolus capreolus*
Saiga antelope	*Saiga tatarica*
Sand cat	*Felis margarita*
Sea lion (California)	*Zalophus californianus*
Sheep (domestic)	*Ovis aries*
Skunk	*Spilogale putorius, Mephitis mephitis*
Southern sea otter	*Enhydra lutris nereis*
Spinner dolphin	*Stenella longirostris*
Spotted seal	*Phoca largha*
Squirrel	*Sciurus carolensis, Sciurus griseus, Spermophilus tridecemlineatus, Tamiasciurus hudsonicus*
Striped dolphin	*Stenella coeruleoalba*
Tammar wallaby	*Macropus eugenii*
Tasmanian pademelon	*Thylogale billardierii*
Walrus	*Odobenus rosmarus*
Wapiti (elk)	*Cervus elaphus*
Warthog	*Phacochoerus aethiopicus, Phacochoerus africanus*
Water buffalo	*Bubalus bubalis*

Common name	Scientific name
Mammals—Continued	
White-tailed deer	*Odocoileus virginianus*
Wild boar	*Sus scrofa*
Wolf	*Canis lupus*
Wombat (common)	*Vombatus ursinus*
Yellow-footed rock wallaby	*Petrogale xanthopus*
Birds	
American coot	*Fulica americana*
American crow	*Corvus brachyrhynchos*
Blue-gray tanager	*Thraupis episcopus*
Brewer's blackbird	*Euphagus cyanocephalus*
Canary	*Serinus canaria*
Cattle egret	*Bubulcus ibis*
Chicken	*Gallus gallus*
Clay-colored robin	*Turdus grayi*
Common barn owl	*Tyto alba*
Common grackle	*Quiscalus quiscula*
Crimson-backed tanager	*Ramphocelus dimidiatus*
Finch	*Carduelis* spp.
Great-tailed grackle	*Quiscalus mexicanus*
Hawaiian crow	*Corvus hawaiiensis*
House sparrow	*Passer domesticus*
Laughing gull	*Larus atricilla*
Little penguin (fairy penguin)	*Eudyptula minor*
Mockingbird (northern)	*Mimus polyglottos*
Ostrich	*Struthio camelus*
Palm tanager	*Thraupis palmarum*
Plain wren	*Thryothorus modestus*
Red-winged blackbird	*Agelaius phoeniceus*
Ring-billed gull	*Larus delawarensis*
Robin (American)	*Turdus migratorius*
Rock dove	*Columba livia*
Ruddy ground dove	*Columbina talpacoti*
Spotted dove	*Streptopelia chinensis*
Starling (common)	*Sturnus vulgaris*
Tree sparrow (Eurasian)	*Passer montanus*

Common name	Scientific name
Birds—Continued	
Turkey vulture	*Cathartes aura*
Turkey	*Meleagris* spp.
Turkey (wild)	*Meleagris gallopavo*
Vulture (white-backed)	*Gyps africanus*
Wood duck	*Aix sponsa*
Cold-blooded animals	
Carp (common)	*Cyprinus carpio*
Crocodile (Nile)	*Crocodylus niloticus*
Horned toad (horned lizard)	*Phrynosoma* spp.
Monitor lizard	*Varanus exanthematicus*
Northern anchovy	*Engraulis mordax*
Saltwater crocodile	*Crocodylus porosus*

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