

Toxoplasmosis in wild and domestic animals

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6.1 Introduction

Toxoplasma gondii is widely distributed in wild and domestic animals. The present chapter reviews toxoplasmosis in wild and domestic animals. Coverage in wild animal species is limited to confirmed cases of toxoplasmosis, cases with parasite isolation, cases with parasite detection by polymerase chain reaction (PCR), and experimental infection studies (Figs. 6.1–6.3). Studies concerning serological prevalence have not been included for the majority of host species. This was done because many serological tests, e.g. latex agglutination (LAT), indirect fluorescent antibody (IFAT), and indirect hemagglutination, have been demonstrated to underestimate the prevalence of *T. gondii*.

6.2 Toxoplasmosis in wildlife

6.2.1 Felids

Congenital toxoplasmosis has been reported in bobcats (*Felis rufus*) kits (Dubey et al., 1987). Toxoplasmic meningoencephalitis has been observed in a 6-month-old bobcat (Smith et al., 1995). *T. gondii* has been isolated from the tissues of adult bobcats (Lindsay et al., 1997b; Dubey et al., 2004b). Bobcats are important in maintaining *T. gondii* in wild herbivores in many areas of the United States (Fig. 6.1). Oocysts excreted by cougars (*Felis concolor*) were thought to be the source of a large water borne outbreak of human toxoplasmosis in Victoria, British Columbia, Canada, and oocysts were isolated from the feces of cougars

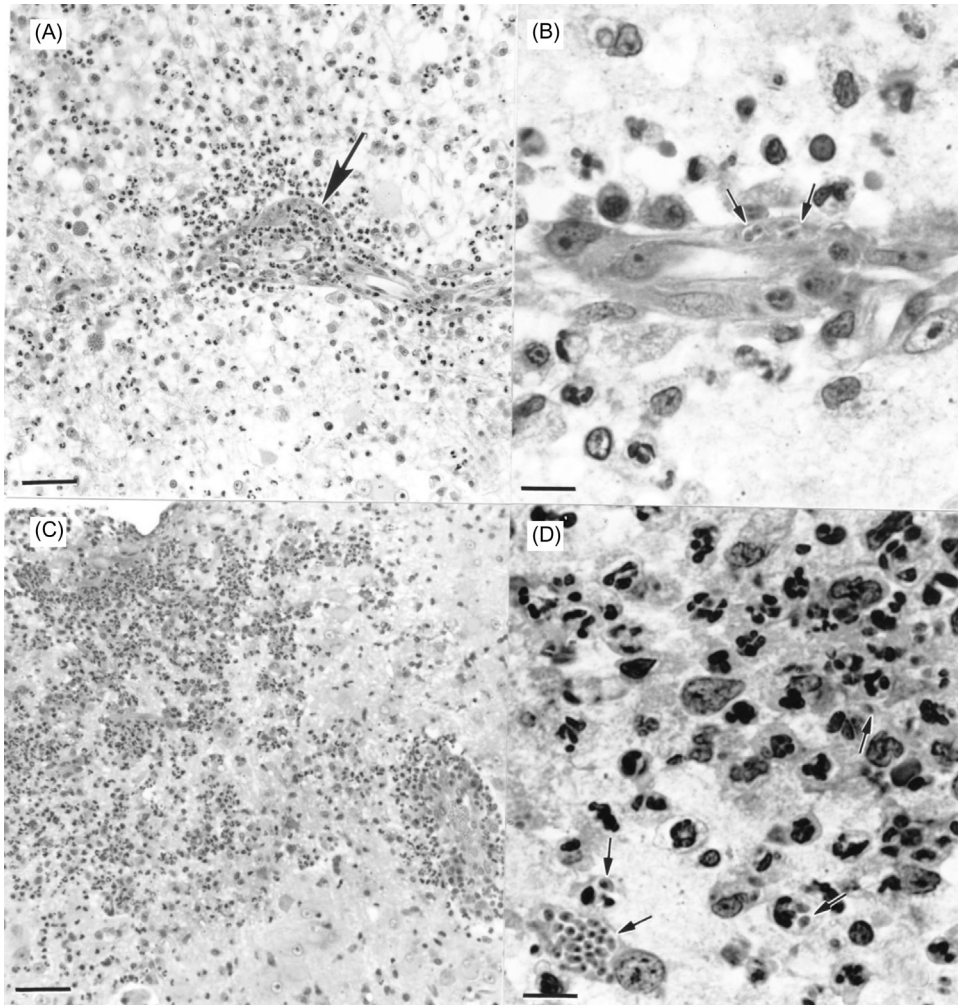


FIGURE 6.1 Fatal toxoplasmic encephalitis in a naturally infected bobcat. H&E stain. (A) Necrosis and inflammation of a blood vessel (*arrow*). Bar = 50 μm . (B) Tachyzoites (*arrows*) in a capillary. Bar = 10 μm . (C) Vasculitis and suppurative encephalitis. Bar = 100 μm . (D) An abscess with degenerating neutrophils and tachyzoites (*arrows*). Bar = 10 μm .

collected around the water shed (Aramini et al., 1998).

Acute toxoplasmosis was reported in a 16-week-old juvenile cheetah (*Acinonyx jubatus*) that was privately owned in Dubai, United Arab Emirates (Lloyd and Stidworthy, 2007). It was housed with three domestic cats and had been with its present owner for 3 weeks and was fed beef and quail. The cub died suddenly

with signs rapidly progressive pyrexia, tachypnea, abdominal effusion, and hepatomegaly (Lloyd and Stidworthy, 2007). *T. gondii* stages were demonstrated in multiple tissues using immunohistochemistry and PCR.

T. gondii has been isolated by bioassay in mice from a jaguarundi (*Puma yagouaroundi*) (Pena et al., 2011), a Jaguar (*Panthera onca*) (Demar et al., 2008), Cougar (*Puma concolor*)

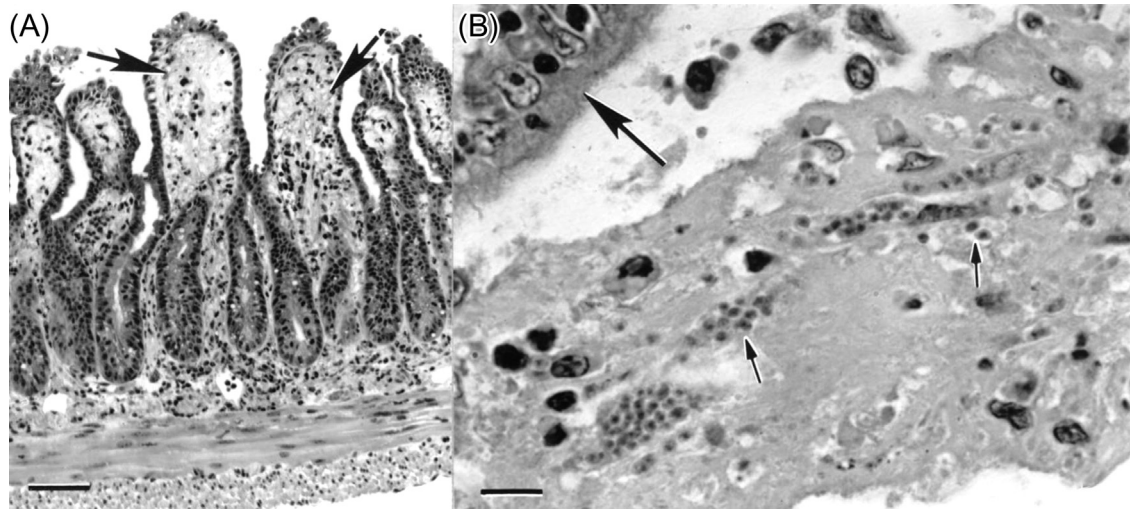


FIGURE 6.2 Necrosis associated with *Toxoplasma gondii* in small intestine. H&E stain. (A) Necrosis of lamina propria (arrows) of villi 7 days after feeding oocysts to a mouse. The surface epithelium is not affected. Numerous tachyzoites are present in lesions but are not visible at this magnification. Bar = 100 μ m. (B) Necrosis of the lamina propria cells including blood vessels in a naturally infected Pallas's cat. Numerous tachyzoites (small arrows) are present. The surface epithelium (large arrow) was not affected. Bar = 10 μ m.

(Dubey et al., 2008a,b), and sand cat (*Felis margarita*) (Dubey et al., 2010). Experimental infections resulting in oocyst excretion have been demonstrated in jaguarundi (*P. yagouaroundi*), ocelot (*Furcifer pardalis*), bobcats (*Lynx rufus*), and cheetah (*A. jubatus*) (Jewell et al., 1972; Miller et al., 1972). In general, these felids are not as efficient at producing oocysts as are domestic cats. Congenital toxoplasmosis is a major factor hindering breeding programs for endangered Pallas's cats (*Otocolobus manul*) and sand cats (*F. margarita*) in zoos worldwide (see next).

6.2.2 Canids

Acute toxoplasmosis has been reported in arctic foxes (*Alopex lagopus*) (Sorensen et al., 2005), Fennec foxes (*Fennecus zerda*) (Kottwitz et al., 2004), gray foxes (*Urocyon cinereoargenteus*) (Davidson et al., 1992; Dubey and Lin, 1994; Kelly and Sleeman, 2003), red foxes

(*Vulpes vulpes*) (Reed and Turek, 1985; Dubey et al., 1990; Kelly and Sleeman, 2003), and sand foxes (*Vulpes rueppellii*) (Pas and Dubey 2008c). Coinfection with canine distemper virus is often associated with clinical toxoplasmosis in gray (Davidson et al., 1992; Kelly and Sleeman, 2003) and red foxes (Reed and Turek, 1985). Clinical toxoplasmosis has not been documented in wolves, coyotes, hyenas, or dingos. *T. gondii* has been isolated from arctic foxes (Dubey et al., 2011b), red foxes (Smith and Frenkel, 1995; Dubey et al., 2004b, 2011b), gray foxes (Dubey et al., 2004b), and coyotes (Lindsay et al., 1997b; Dubey et al., 2004b). Aubert et al. (2010) found modified agglutination test (MAT) antibodies in 14 of 19 (74%) red foxes from France and isolated *T. gondii* from the hearts of 9 (69%) of 13 seropositive red foxes. The isolates were all genotype Type II.

Herrmann et al. (2012) used serology (immunoblot) and PCR to examine the prevalence of *T. gondii* in red foxes and rodents from the German Federal States of Brandenburg and

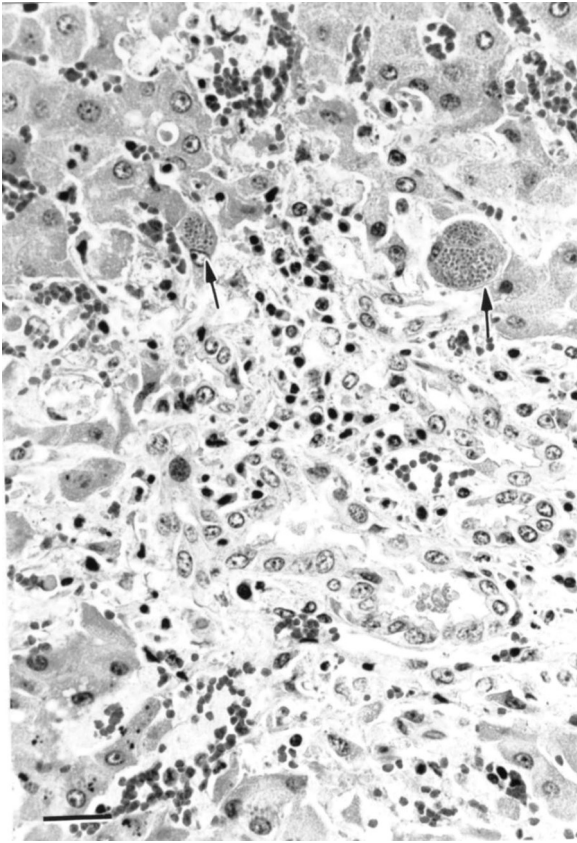


FIGURE 6.3 Section of liver from a gazelle with toxoplasmosis showing a central area of hepatitis. Note *Toxoplasma gondii* (arrows) in hepatocytes at the periphery of the lesion. H&E stain. Bar = 25 μ m.

Saxony-Anhalt. They found 152/204 (74.5%) and 149/176 (84.7%) of red foxes in Brandenburg and Saxony-Anhalt were immunoblot positive, respectively, but none of 72 rodents (69 common voles *Microtus arvalis*, 2 Mediterranean water shrews *Neomys anomalus*, and 1 striped field mouse *Apodemus agrarius*) had antibodies to *T. gondii*. PCR was conducted on heart tissue from seropositive red fox tissues and 28/152 (18%) and 20/149 (13%) of seropositive foxes from Brandenburg and Saxony-Anhalt, respectively, were positive (Herrmann et al., 2012). PCR was done on heart and lung samples from the 72 rodents and none tested positive (Herrmann et al., 2012).

6.2.3 Bears

Clinical toxoplasmosis has not been reported from bears. Viable *T. gondii* has been isolated from black bears (*Ursus americanus*) (Dubey et al., 1995a) and brown bears (*Ursus arctos horribilis*) (Dubey et al., 2011b). The prevalence of *T. gondii* in black bears in the United States is the highest of any hosts for *T. gondii* worldwide. In a recent survey, *T. gondii* antibodies were found in 4% of dams and 5% of their nursing cubs while in dens; the study concluded that there is no transplacental transmission of *T. gondii* in bears and that 50% of bears acquire infection postnatally by their 10 months of age (Dubey et al., 2016). Compared with black bears, the prevalence of *T. gondii* in brown bears from Alaska is about half (44%) of black bears (Ramey et al., 2019). The prevalence of *T. gondii* in 527 polar bears (*Ursus maritimus*) was 3.6% in cubs still with their dam and 21.4% for subadults and adults from Svalbard and the Barents Sea and East Greenland (Oksanen et al., 2009) and 6% of 500 polar bears from the Beaufort and Chukchi sea areas of the Arctic Ocean (Rah et al., 2005). The prevalence in polar bears was 23.9% (33) of 105 animals from southern Beaufort Sea (Atwood et al., 2017) and in grizzly bears (*U. arctos*) (Chomel et al., 1995). Meat from any species of bear should be considered a potential source of *T. gondii*.

6.2.4 Raccoons

Many serosurveys indicate that *T. gondii* is highly prevalent in raccoons (*Procyon lotor*) (reviewed by Hancock et al., 2005). Encysted *T. gondii* has been isolated from the tissues of naturally infected raccoons (Lindsay et al., 1997b; Dubey et al., 2004c, 2011b). Clinical toxoplasmosis has not been reported from raccoons and they are resistant to experimental infection (Dubey et al., 1993b).

6.2.5 Squirrels

Acute toxoplasmosis has been reported in gray squirrels (*Sciurus carolinensis*) (Dubey et al., 2006a), eastern fox squirrels (*Sciurus niger*) (Kumar et al., 2018), American red squirrels (*Tamiasciurus hudsonicus*) (Bangari et al., 2007), 13-lined ground squirrels (*Citellus tridecemlineatus*) (van Pelt and Dieterich, 1972), Eurasian red squirrels (*Sciurus vulgaris*) (Jokelainen and Nylund, 2012), Swinhoe's striped squirrel (*Tamias swinhoi*) (Fayyad et al., 2016), and Korean squirrels (*Tamias sibericus*) (Carrasco et al., 2006). *T. gondii* has been isolated from gray squirrels (Smith and Frenkel, 1995) and Formosan giant flying squirrels (*Petaurista petaurista grandis*) (Cross et al., 1969).

6.2.6 Rabbits and hares

Fatal toxoplasmosis has been reported from three domestic (*Oryctolagus cuniculus*) rabbits from two different sources in the United States (Dubey et al., 1992a). The rabbits died after an acute illness characterized by fever, lethargy, and diarrhea in one rabbit and no clinical signs in the other two rabbits. The most striking lesion in all three rabbits was foci of necrosis of the spleen and liver associated with massive presence of multiplying tachyzoites (Dubey et al., 1992a). Similar findings were present in 2–18-month-old domestic rabbits from 15 flocks in Germany. Necropsy examinations of 49 rabbits revealed lesions of a generalized granulomatous-necrotizing toxoplasmosis within the spleen, liver, lungs, and lymph nodes (Bergmann et al., 1980). Both authors of the current chapter (DSL and JPD) have inoculated domestic rabbits orally and subcutaneously with *T. gondii* oocysts (usually 10,000/rabbit) to generate immune serum for immunohistochemistry. All inoculated rabbits have or would have developed fatal toxoplasmosis had they not been euthanized for humane reasons. Viable *T. gondii* has been isolated from

domestic rabbits (*O. cuniculus*) from Brazil (Dubey et al., 2011a).

Brown hares (*Lepus europaeus*) develop fatal toxoplasmosis after experimental infection with as few as 10 oocysts and all inoculated hares died within 8–19 days after ingesting oocysts (Sedlak et al., 2000). The typical pathological finding in hares is hemorrhagic enteritis, enlargement and hyperemia of mesenteric lymph nodes, splenomegaly, and multiple necrotic lesions in the parenchyma of the liver and other organs (Sedlak et al., 2000). Mountain hare (*Lepus timidus*) experimentally inoculated with 50 *T. gondii* oocysts and examined 7 days later had gross lesions in the mesenteric lymph nodes and liver (Gustafsson et al., 1997). Histologically, the hares had extensive necrotic areas in the small intestine, mesenteric lymph nodes and liver, and less prominent foci of necrosis in various other organs (Gustafsson et al., 1997). Recent retrospective studies in Finland (Jokelainen et al., 2011) have documented natural toxoplasmosis in hares similar to these experimental reports. Acute generalized toxoplasmosis was demonstrated immunohistochemically, and *T. gondii* was confirmed as the cause of death in 14 (8%) of 173 European brown hares (*L. europaeus*) and 4 (3%) of 148 mountain hares (*L. timidus*) from Finland (Jokelainen et al., 2011). Aubert et al. (2010) demonstrated that 3 (13%) of 23 European brown hares (*L. europaeus*) from France were positive in the MAT but were not able to isolate *T. gondii* from the hearts of two seropositive animals.

6.2.7 Skunks and fisher

T. gondii genotype III was isolated from three of six asymptomatic striped skunks (*Mephitis mephitis*) from Mississippi (Dubey et al., 2004d). Two of the three isolated were mouse pathogenic even though they were molecularly consistent with the mouse avirulent genotype III. Lesions of toxoplasmosis and *T. gondii* parasites were not observed at necropsy of 37 striped

skunks from Illinois (Gehrt et al., 2010). This population was serologically 60% positive for exposure to *T. gondii* (Gehrt et al., 2010).

T. gondii was detected by PCR from brain and skeletal muscle of a free-ranging juvenile fisher (*Martes pennanti*) from Maryland (Gerhold et al., 2005). Clinically this animal had encephalitis, but it was not associated with the *T. gondii* infection. *T. gondii* antibodies were found using MAT in 100% of 38 and using IFAT in 71% of 45 fisher from Pennsylvania (Larkin et al., 2011).

6.2.8 Beavers

T. gondii has been isolated from beaver (*Castor canadensis*) tissue (Dubey, 1983; Smith and Frenkel, 1995). Fatal systematic toxoplasmosis was seen in a 5-month-old beaver that was in a rehabilitation center in Connecticut (Forzán and Frasca, 2004). Histologic lesions contained *T. gondii* positive stages by immunohistochemistry and consisted of lymphohistocytic encephalitis, myocarditis, and interstitial pneumonia with multinucleated cells (Forzán and Frasca, 2004).

6.2.9 Woodchuck and other large rodents

Central nervous system toxoplasmosis has been observed in a woodchuck (*Marmota monax*) (Bangari et al., 2007) from New York. The woodchuck was euthanized because of progressive clinical signs of head tilt, circling, and rapid weight loss. The brain and heart were positive for *T. gondii* by immunohistochemistry and PCR (Bangari et al., 2007).

Clinical toxoplasmosis has not been reported in capybara (*Hydrochaeris hydrochaeris*) or nutria (*Myocastor coypus*). However, the parasite has been isolated from capybara from Brazil (Yai et al., 2009) and *T. gondii* DNA has

been detected by PCR in nutria from Italy (Nardoni et al., 2011).

6.2.10 Insectivores

Little is known about toxoplasmosis in insectivores. The prevalence of *T. gondii* using the Sabin–Feldman dye test was <1% in 578 insectivores from the Czech Republic (Hejlíček et al., 1997). Fatal toxoplasmosis was diagnosed in a juvenile male common mole (*Talpa europaea*) from Germany (Geisel et al., 1995). None of 70 *T. europaea* from the Netherlands were serologically positive using the latex agglutination test (LAT), but *T. gondii* DNA was detected by real-time PCR in the brain of two of these common moles (Krijger et al., 2014). The brains and/or hearts from 3 of 22 white-toothed shrews (*Crocidura russula*) from organic pig farms in the Netherlands were positive for *T. gondii* by PCR (Kijlstra et al., 2008). In another study from organic pig farms from the Netherlands, none of the brains from 9 common shrews (*Sorex araneus*) and 2 (2%) brains from 102 white-toothed shrews (*C. russula*) were positive by PCR for *T. gondii* (Meerburg et al., 2012). None of two Mediterranean water shrews (*N. anomalus*) from Germany were positive by serology or PCR (Herrmann et al., 2012). *T. gondii* DNA was detected in the heart of 1 of 578 striped field mice (*A. agrarius*) from North Korea (Hong et al., 2014).

6.2.11 Bats

Acute toxoplasmosis has been observed in a juvenile spectacled flying-fox (*Pteropus conspicillatus*) and a juvenile little red flying fox (*Pteropus scapulatus*) from Australia (Sangster et al., 2012). One was a captive born member of a colony, and the other was undergoing rehabilitation at a wildlife hospital. Severe, acute interstitial pneumonia with varying combinations of neutrophils, large foamy macrophages,

and fibrin present within alveoli were seen in the lungs and *T. gondii* confirmed using immunohistochemistry. Lesions in the CNS consisted of multiple foci of gliosis, including gemistocytic astrocytes, at all levels of the cerebrum, cerebellum, and brainstem of the bats (Sangster et al., 2012). The bats are arboreal in nature, and it was suggested that the *T. gondii* infections might have been acquired in captivity by food accidentally contaminated with oocysts (Sangster et al., 2012). Isolation of *T. gondii* was reported from pipistrelle bats *Vespertilio pipistrellus* and the red night bat *Nyctalus noctula* from Alma-Ata, Kazakhstan, USSR (Galuzo et al., 1970). Inoculation of RH *T. gondii* did not induce clinical disease in red night bats in these studies (Galuzo et al., 1970). *T. gondii* is widely prevalent in bats, and the seropositivity in noncarnivorous bats is intriguing. In a study from Brazil antibodies to *T. gondii* were found in 22 species of bats (Cabral et al., 2014).

6.2.12 White-tailed and mule deer

T. gondii is prevalent in deer from North America. Consumption of venison has been linked with clinical toxoplasmosis in humans (Sacks et al., 1983; Ross et al., 2001). Clinical toxoplasmosis has not been described from naturally infected deer in North America. *T. gondii* has been isolated from the tissues of white-tailed deer (*Odocoileus virginianus*) (Lindsay et al., 1991a,b, 1997b; Dubey et al., 2004b; Gerhold et al., 2017) and mule deer (*Odocoileus hemionus*) (Dubey, 1982). Viable *T. gondii* was isolated from 6 of 61 white-tailed deer fetuses from dams, which were in early pregnancy (45–85 days of gestation) from Iowa and 9 of 27 white-tailed deer fetuses from Minnesota dams which were in mid-gestation (130–150 days) of a gestational period of 7 months (Dubey et al., 2008b). The fetuses from *T. gondii* positive white-tailed and mule deer were negative for *T. gondii* antibodies in one

study, suggesting that seropositive dams do not transmit the infection to their fetuses (Lindsay et al., 2006) unless an acute infection occurs during pregnancy. Acute toxoplasmosis and death can occur in mule deer experimentally inoculated with *T. gondii* oocysts (Dubey et al., 1982).

6.2.13 Other deer

Congenital toxoplasmosis has been observed in reindeer (*Rangifer tarandus*) from a private collection in the United States (Dubey et al., 2002a). Yearling reindeer may develop enteritis and die after experimental oral infection with *T. gondii* oocysts (Oksanen et al., 1996).

Aubert et al. (2010) demonstrated that 36 (60%) of 60 roe deer (*Capreolus capreolus*) from France were positive in the MAT and obtained 12 isolates from the hearts (38%) of 33 MAT positive roe deer. They also reported that *T. gondii* antibodies were present in 4 (17%) of 24 red deer (*Cervus elaphus*) (Aubert et al., 2010). One (25%) of four fallow deer (*Dama dama*) from France examined by Aubert et al. (2010) was positive (MAT titer 1:25), but attempts to isolate *T. gondii* by bioassay in mice were not successful.

6.2.14 Other wild ruminants

Elk (*Cervus canadensis*) are resistant to clinical disease following oral infection with oocysts, but *T. gondii* can be isolated from many of their tissues indicating that elk are a potential source of infection for humans (Dubey et al., 1980). Antibodies to *T. gondii* were detected in sera of 221 of 317 (69.7%) elk from Pennsylvania collected during 2013–16 hunting season by the MAT and hearts from 2 of 20 elk were positive by bioassay (Dubey et al., 2017). Eighty of 142 (56.3%) elk from Kentucky were seropositive for *T. gondii* (Cox et al., 2017).

T. gondii was as isolated by bioassay in mice from one of seven naturally infected moose (*Alces alces*) from Montana (Dubey, 1981). The isolate was not pathogenic for mice.

Antibodies against *T. gondii* were detected in 8 of 79 (10%) moose from Minnesota tested by MAT (Verma et al., 2016). The parasite was isolate by bioassay of hearts from 3 of 68 of these moose. Two samples were from adults, and one was from a 3-week-old calve. Congenital toxoplasmosis was observed in a child from Alaska whose mother had consumed PCR positive moose meat (Colosimo et al., 2013).

T. gondii was been isolated by bioassay in mice from 1 of 21 naturally infected pronghorn antelope (*Antilocapra americana*) from Montana (Dubey, 1981). The isolate was not pathogenic for mice. Acute toxoplasmosis and death can occur in pronghorn antelopes experimentally inoculated with *T. gondii* oocysts (Dubey et al., 1982). Toxoplasmic encephalitis has been observed in a 4-month-old Rocky Mountain bighorn sheep (*Ovis canadensis canadensis*) (Baszler et al., 2000). Aubert et al. (2010) demonstrated MAT antibodies in 7 (23%) of 31 mouflons (*Ovis gmelini musimon*) from France and isolated the parasite from 1 (25%) of 4 hearts from seropositive mouflons.

6.2.15 Sea otters and other marine mammals

Toxoplasmosis was first recognized as a significant cause of mortality in southern sea otters (*Enhydra lutris nereis*) in the early 1990s (Cole et al., 2000). Encephalitis is the primary cause of *T. gondii* associated death in these sea otters (Cole et al., 2000). This was unexpected as sea otters do not ingest the usual intermediate hosts of *T. gondii* and their location in seawater keeps them segregated from cats. Definitive proof that *T. gondii* was killing the sea otters came when viable *T. gondii* was

isolated from the tissues of sea otters (Cole et al., 2000; Lindsay et al., 2001a), and isolated parasites from sea otters were shown to retain the ability to make oocysts when fed to cats (Cole et al., 2000). Initial isolates were all type II genotypes of *T. gondii* (Cole et al., 2000). It has been postulated that *T. gondii* oocysts excreted in the feces of feral cats living along the Pacific coast enter the marine environment and are ingested by sea otters when they feed on paratenic hosts (Cole et al., 2000), and this is supported by the fact that coastal freshwater runoff is a risk factor for *T. gondii* infection in southern sea otters (Miller et al., 2002). *T. gondii* oocysts will sporulate in seawater (Lindsay et al., 2003) and remain infectious for 1.5 years at room temperature and for at least 2 years at 4°C (Lindsay and Dubey, 2009), and viable *T. gondii* and *T. gondii* DNA can be recovered from experimentally inoculated bivalves (Lindsay et al., 2001b, 2004; Arkush et al., 2003), further supporting these assumptions. In addition, two species of filter feeding fish, northern anchovies (*Engraulis mordax*) and Pacific sardines (*Sardinops sagax*), have been shown to be able to remove *T. gondii* oocysts from seawater and can potentially serve as biotic vectors for *T. gondii* within the marine environment (Massie et al., 2010). *T. gondii* also cause deaths in other marine mammals off the Pacific coast of the United States often in the same areas as the sea otters (Miller et al., 2001), and it has also been isolated from Pacific harbor seal (*Phoca vitulina*) and California sea lion (*Zalophus californianus*).

Toxoplasmosis is frequently reported from dolphins worldwide. Congenital toxoplasmosis has been reported in bottlenose dolphin (*Tursiops aduncus*) (Jardine and Dubey, 2002). Disseminated toxoplasmosis with transplacental fetal infection has been seen in a pregnant Risso's dolphin (*Grampus griseus*) (Resendes et al., 2002). Acute cases of toxoplasmosis have been seen in humpbacked dolphins (*Sousa chinensis*) (Bowater et al., 2003), spinner dolphins

(*Stenella longirostris*) (Migaki et al., 1990), striped dolphins (*Stenella coeruleoalba*) (DiGuardo et al., 2010), and Atlantic bottlenosed dolphins (*Tursiops truncatus*) (Inskoop et al., 1990). *T. gondii* has been isolated from the hearts of 3 of 52 bottlenose dolphins (*T. aduncus*) from the eastern United States by mouse bioassay (Dubey et al., 2008a). *T. gondii* was isolated from the brain of a stranded female striped dolphin (*Stenella coeruleoalba*) from Costa Rica that died from non-*T. gondii* related causes (Dubey et al., 2007a).

Toxoplasmosis has been reported from several additional species of marine mammals such as beluga whales (*Delphinapterus leucas*) (Mikaelian et al., 2000), Mediterranean fin whale (*Balaenoptera physalus*) (Mazzariol et al., 2012), California sea lion (*Z. californianus*) (Migaki et al., 1977), northern fur seal (*Callorhinus ursinus*) (Holshuh et al., 1985), elephant seal (*Mirounga angustirostris*) (Dubey et al., 2004a), Hawaiian monk seal (*Monachus schauinslandi*) (Honnold et al., 2005), Antillean manatee (*Trichechus manatus manatus*) (Dubey et al., 2003; Bossart et al., 2012), and West Indian manatee (*T. manatus*) (Burgelt and Bonde, 1983). Experimental infection of gray seals (*Halichoerus grypus*) with up to 10,000 *T. gondii* oocysts did not induce overt clinical disease (Gajadhar et al., 2004). Mild behavioral changes were the only adverse effects, and *T. gondii* was isolated from brain and muscles of the experimentally infected seals.

6.2.16 New world monkeys

Toxoplasmosis can be a problem in exhibited new world monkeys (Table 6.1). Many reports of acute disease have come from squirrel monkeys (*Saimiri sciureus*) (Cedillo-Peláez et al., 2011; Epiphanio et al., 2003) and golden lion tamarins (*Leontopithecus rosalia*) (Dietz et al., 1997; Pertz et al., 1997; Juan-Salles et al., 1998; Epiphanio et al., 2003). Squirrel monkeys and

TABLE 6.1 Summary of host species reports of clinical toxoplasmosis in New World primates.

Cotton-top tamarin (<i>Saguinus oedipus</i>)
Yellow-handed marmoset (<i>Saguinus midas midas</i>)
Black marmoset (<i>Saguinus midas niger</i>)
Emperor marmoset (<i>Saguinus imperator</i>)
Red-bellied white-lipped tamarin (<i>Saguinus labiatus</i>)
Black lion tamarin (<i>Leontopithecus chrysopygus</i>)
Golden-headed lion tamarins (<i>Leontopithecus chrysomelas</i>)
Golden lion tamarins (<i>Leontopithecus rosalia</i>)
Squirrel monkeys (<i>Saimiri sciureus</i>)
Pygmy marmoset (<i>Callithrix pygmaea</i>)
Common marmoset (<i>Callithrix jacchus</i>)
Black ear-tufted marmoset (<i>Callithrix penicillata</i>)
Pale-headed saki (<i>Pithecia pithecia</i>)
Night monkey (<i>Aotus trivirgatus</i>)
Howler monkey (<i>Alouatta fusca</i>)
Woolly monkey (<i>Lagothrix lagotricha</i>)

Dietz, H.H., Henriksen, P., Bille-Hansen, V., Henriksen, S.A., 1997. Toxoplasmosis in a colony of New World monkeys. *Vet. Parasitol.* 68, 299–304; Bouer, A., Werther, K., Catao-Dias, J.L., Nunes, A.L., 1999. Outbreak of toxoplasmosis in *Lagothrix lagotricha*. *Folia Primatol.* (Basel) 70, 282–285; Epiphanio, S., Sa, L.R., Teixeira, R.H., Catao-Dias, J.L., 2001. Toxoplasmosis in a wild-caught black lion tamarin (*Leontopithecus chrysopygus*). *Vet. Rec.* 149, 627–628; Epiphanio, S., Sinhorini, I.L., Catao-Dias, J.L., 2003. Pathology of toxoplasmosis in captive new world primates. *J. Comp. Pathol.* 129, 196–204; Dubey, J.P., 2010. *Toxoplasmosis of Animals and Humans*, second ed. CRC Press, Boca Raton, FL, pp. 1–313; Cedillo-Peláez, C., Rico-Torres, C.P., Sales-Garrido C.G., Correa, D., 2011. Acute toxoplasmosis in squirrel monkeys (*Saimiri sciureus*) in Mexico. *Vet. Parasitol.* 180, 368–371; Pena, H.F.J., Marvulo, M.F.V., Horta, M.C., Silva, M.A., Silva, J.C.R., Siqueira, D.B., et al., 2011. Isolation and genetic characterisation of *Toxoplasma gondii* from a red-handed howler monkey (*Alouatta belzebul*), a jaguarundi (*Puma yagouaroundi*), and a black-eared opossum (*Didelphis aurita*) from Brazil. *J. Parasitol.* 175, 377–381.

Panamanian night monkeys (*Aotus lemurinus*) are highly susceptible to oral tissue cyst inoculation and develop acute fatal disease (Harper et al., 1985; Escajadillo and Frenkel, 1991; Furuta et al., 2001). Pena et al. (2011) isolated *T. gondii* by bioassay in mice of its heart and brain of a

young male red-handed howler monkey (*Alouatta belzebul*) with suspected toxoplasmosis from a zoo in Brazil.

6.2.17 Old world monkeys

Toxoplasmosis is reported infrequently in old world monkeys. A case of concurrent central nervous system toxoplasmosis and simian immunodeficiency virus-induced AIDS encephalomyelitis was seen in a Barbary macaque (*Macaca sylvana*) (Sasseville et al., 1995). Rhesus monkeys (*Macaca mulatta*) and stump-tailed macaques (*Macaca arctoides*) have been used as experimental models for human congenital toxoplasmosis (Wong et al., 1979; Schoondermark-Van de Ven et al., 1993), and cynomolgus monkeys (*Macaca fascicularis*) have been used as a model for recurrent toxoplasmic retinochoroiditis (Holland et al., 1988).

6.2.18 American marsupials

Clinical toxoplasmosis has not been reported from marsupials from the Americas. The parasite has been isolated from opossums (*Didelphis virginiana*) from Georgia (Dubey et al., 2011b) and Kansas (Smith and Frenkel, 1995) in the United States, and black-eared opossums (*Didelphis aurita*) from Brazil (Pena et al., 2011) have been examined and proven to be positive by bioassay in mice.

6.2.19 Australian marsupials

T. gondii infection is usually life ending in marsupials from Australia or New Zealand. Outbreaks of toxoplasmosis often occur in these animals when housed in zoos (see next). These animals evolved in the absence of cats and *T. gondii*, and this may be why they are so highly susceptible.

Canfield et al. (1990) summarized clinical signs, necropsy findings and histopathological

changes are summarized for 43 macropods (species not given), 2 common wombats (*Vombatus ursinus*), 2 koalas (*Phascolarctos cinereus*), 6 possums (species not given), 15 dasyurids (species not given), 2 numbats (*Myrmecobius fasciatus*), 8 bandicoots (species not given), and 1 bilby (*Macrotis lagotis*). The animals either died suddenly without clinical signs or exhibited signs associated with respiratory, neurological, or enteric disease. At necropsy, many had no visible lesions. Common necropsy findings included pulmonary congestion, edema and consolidation, adrenal enlargement and reddening, hemorrhage and ulceration of stomach and small intestine, and lymphadenomegaly and splenomegaly (Canfield et al., 1990). Congenital toxoplasmosis apparently occurs in black-faced kangaroos (*Macropus fuliginosus melanops*) based on the finding of *T. gondii* in the tissues of a 82 day-old joey that died from toxoplasmosis (Dubey et al., 1988b). *T. gondii* was seen in the heart, kidney, liver, lung, lymph node, spleen, small intestine, and stomach from two koalas (*P. cinereus*) that died suddenly in a fauna park in Sydney, Australia (Hartley et al., 1990).

Experimental studies support the assumption that Australian marsupials are highly susceptible to toxoplasmosis. Eastern barred bandicoots (*Perameles gunnii*) develop acute toxoplasmosis when fed 100 *T. gondii* oocysts and died 15 and 17 days postinfection (Bettli et al., 2000). Lesions consistent with acute toxoplasmosis were present in their tissues. The authors indicated that *T. gondii* may be a cause for a reduction in wild populations of eastern barred bandicoots. Tammar wallabies (*Macropus eugenii*) fed 500, 1000, or 10,000 *T. gondii* oocysts died of acute toxoplasmosis 9–15 days after challenge (Reddacliff et al., 1993). The lesions consisted of foci of necrosis and inflammation in the intestines, lymphoid tissue, adrenal cortex, heart, skeletal muscle and brain, and severe generalized pulmonary congestion and edema.

6.2.20 African wildlife

Surprisingly little is known about *T. gondii* and toxoplasmosis from African mammals. Clinical disease has not been reported from free-ranging elephants, hippopotamus, rhinos, giraffes, gazelle, wildebeests, impalas, chimpanzees, baboons, orangutans, and gorillas. *T. gondii* has not been isolated from these species that were naturally infected. Antibodies to *T. gondii* have been demonstrated in elephants, hippopotamus, rhinos, giraffes, wildebeests, and chimpanzees.

Early after the life cycle in cats was discovered, an experimental study was done in two female chimpanzees fed *T. gondii* oocysts of the Beverly strain (Draper et al., 1971). One chimpanzee (female 1) was Sabin–Feldman dye test negative and the other (female 2) was dye test positive (1:250) before the study was initiated. *T. gondii* was isolated by bioassay in mice from the blood (sampled 1 week PI), inguinal lymph node (sampled 11 weeks PI), and thigh muscle (sampled 11 weeks PI) of female 1 fed 2.5×10^6 *T. gondii* oocysts [but not cerebral spinal fluid (taken 11 weeks PI) (Draper et al., 1971)]. Clinically, female 1 became slightly anorexic, developed enlarged superficial lymph nodes, and seroconverted in the dye test to 1:8192 at 30 days PI. Female 2 was fed 1.5×10^6 *T. gondii* oocysts and did not demonstrate clinical signs or an increase in antibody titer nor was *T. gondii* isolated from blood samples by bioassay in mice (Draper et al., 1971).

6.2.21 Wild rodents

A detailed discussion of *T. gondii* prevalence in wild rodents (mice and rats) is beyond the scope of this chapter. *T. gondii* has been isolated from the tissues from wild rodents worldwide (Dubey, 2010). Genotypes of these isolates are similar to isolates from other animals in the same geographic area.

Dabritz et al. (2008) has recently reviewed what was known about the global serological

prevalence of *T. gondii* in wild rodents. Until large-scale studies are conducted using bioassay or molecular detection methods, the role of wild rodents in maintaining *T. gondii* in the environment will not be fully understood. Properly conducted serological studies usually indicate that few (<10%) wild mice or rats are usually found to be seropositive. For example, 2 (0.8%) of 238 rats (*Rattus norvegicus*) from Grenada, West Indies, were found to be serologically positive using the MAT (Dubey et al., 2006b). When the brains and hearts of all 238 rats were examined by bioassay in mice, *T. gondii* was isolated from only 1 of the 238 rats with the positive rat being one of the 2 serologically positive animals. This clearly demonstrates a low prevalence of *T. gondii* infection in this rat population.

6.2.22 Wild birds

Table 6.2 lists the wild avian hosts from which viable *T. gondii* has been isolated, and Table 6.3 lists the avian species that have been reported to suffer from clinical toxoplasmosis.

T. gondii is readily isolated from the hearts and breast muscles of raptors (Lindsay et al., 1993; Dubey et al., 2011b; Table 6.2). Necrotizing myocarditis caused by *T. gondii* has been observed in a bald eagle (*Haliaeetus leucocephalus*) from New Hampshire (Szabo et al., 2004). Severe toxoplasmic hepatitis was seen in an adult barred owl (*Strix varia*) from Quebec, Canada (Mikaelian et al., 1997). No clinical signs were seen in three red-tailed hawks (*Buteo jamaicensis*) fed *T. gondii* tissue cysts (Lindsay et al., 1991a,b). *T. gondii* was isolated from all three red-tailed hawks. No clinical signs were seen in great horned owls (*Bubo virginianus*), barred owls (*S. varia*), or screech owls (*Asio otus*) fed *T. gondii* tissue cysts (Dubey et al., 1992b), but parasites were isolated from the tissues of the owls at necropsy. *T. gondii* was not reisolated from a sparrow hawk (*Falco sparverius*) that had been experimentally infected (Miller et al., 1972).

TABLE 6.2 Host records for *Toxoplasma gondii* isolation from wild birds.**Anseriformes**

Mallards (*Anas platyrhynchos*)
 Pochard (*Aythya ferrina*)
 Tufted ducks (*Aythya fuligula*)
 Pintail (*Anas acuta*)
 Gadwall (*Anas strepera*)
 Canada goose (*Branta canadensis*)

Accipitriformes

Goshawk (*Accipiter gentilis*)
 Cooper's hawk (*Accipiter cooperi*)
 Common buzzard (*Buteo buteo*)
 Kestrel (*Falco tinnunculus*)
 American kestrel (*Falco sparverius*)
 Pallid harrier (*Circus macrourus*)
 Bald eagle (*Haliaeetus leucocephalus*)
 Black vulture (*Aegypius monachus*)
 Red-tailed hawk (*Buteo jamaicensis*)
 Red-shouldered hawk (*Buteo lineatus*)

Galliformes

Partridge (*Perdix perdix*)
 Pheasant (*Phasianus colchicus*)
 Wild turkey (*Meleagris gallopavo*)

Gruiformes

Coot (*Fulica atra*)

Charadriiformes

Blackheaded gull (*Larus ridibundus*)
 Common tern (*Sterna hirundo*)

Columbiformes

Collared dove (*Streptopelia decaocto*)
 Laughing dove (*Streptopelia senegalensis*)
 Woodpigeon (*Columba palumbus*)

Pigeon (*Columba livia*)

Ruddy ground dove (*Columbina talpacoti*)

Strigiformes

Ferruginous pygmy owl (*Glauclidium brasilianum*)
 Little owl (*Athene noctua*)
 Great horned owl (*Bubo virginianus*)
 Barred owl (*Strix varia*)

Passeriformes

Great gray shrike (*Lanius excubitor*)
 Yellowhammer (*Emberiza citrinella*)
 Chaffinch (*Fringilla coelebs*)
 House sparrow (*Passer domesticus*)
 Tree sparrow (*Passer montanus*)
 Jay (*Garrulus glandarius*)
 Starling (*Sturnus vulgaris*)
 Palm tanager (*Thraupis palmarum*)
 Blackbird (*Turdus merula*)
 Mistle thrush (*Turdus viscivorus*)
 Song thrush (*Turdus philomelos*)
 Robin (*Erithacus rubecula*)
 Great tit (*Parus major*)
 Nuthatch (*Sitta europaea*)
 Treecreeper (*Certhia familiaris*)
 Greenfinch (*Chloris chloris*)
 American crow (*Corvus brachyrhynchos*)
 Carrion crow (*Corvus corone*)
 Jackdaw (*Corvus monedula*)
 Rook (*Corvus frugilegus*)

Dubey, J.P., 2002. A review of toxoplasmosis in wild birds. *Vet. Parasitol.* 106, 121–153; Dubey, J.P., 2010. *Toxoplasmosis of Animals and Humans, second ed.* CRC Press, Boca Raton, FL, pp. 1–313; Lindsay, D. S., Smith, P.C., Hoerr, F.J., Blagburn, B.L., 1993. Prevalence of encysted *Toxoplasma gondii* in raptors from Alabama. *J. Parasitol.* 79, 870–873.

TABLE 6.3 List of avian species in which clinical toxoplasmosis has been reported.

Columbiformes
Common pigeon (<i>Columba livia</i>)
Crown pigeons (<i>Goura</i> sp.)
Torres Strait pigeon (<i>Ducula spilorrhoa</i>)
Wonga pigeon (<i>Leucosarcia melanoleuca</i>)
Bleeding-heart dove (<i>Gallicolumba luzonica</i>)
Nicobar pigeon (<i>Caloenas nicobarica</i>)
Luzon bleeding-heart pigeon (<i>Gallicolumba luzonica</i>)
Orange-breasted green pigeon (<i>Treron bicincta</i>)
Crested wood partridge (<i>Rolulus rouloul</i>)
Yellow-headed rockfowl (<i>Picathartes gymnocephalus</i>)
Passeriformes
Canaries (<i>Serinus canarius</i>)
Greenfinches (<i>Carduelis chloris</i>)
Goldfinches (<i>Carduelis carduelis</i>)
Sirkins (<i>Carduelis spinus</i>)
Linnets (<i>Carduelis cannabina</i>)
Bullfinches (<i>Pyrrhula pyrrhula</i>)
Hawaiian crow (<i>Corvus hawaiiensis</i>)
Satin bowerbird (<i>Ptilonorhynchus violaceus</i>)
Regent bowerbird (<i>Sericulus chrysocephalus</i>)
Red-whiskered bulbul (<i>Pycnonotus jocosus</i>)
Psittaciformes
Budgerigars (<i>Melopsittacus undulatus</i>)
Regent parrot (<i>Polytelis anthopeplus</i>)
Superb parrot (<i>Polytelis swansonii</i>)
Red lory (<i>Eos bornea</i>)
Swainson's lorikeet (<i>Trichoglossus moluccanus</i>)
Crimson rosella (<i>Platyercus elegans</i>)
Strigiformes
barred owl (<i>S. varia</i>)

Galliformes

Wild turkeys (*Meleagris gallopavo*)
 Partridges (*Perdix perdix*)
 Capercaillie (*Tetrao urogallus*)
 Erckel's francolin (*Francolinus erckelii*)
 Guinea fowl (*Numida meleagris*)

Anseriformes

Maggie geese (*Anseranas semipalmata*)
 Hawaiian nene goose (*Nesochen sandicensis*)

Sphenisciformes

Humboldt penguin (*Spheniscus humboldti*)
 Megellanic penguin (*Spheniscus magellanicus*)
 Black-footed penguin (*Spheniscus demersus*)
 Little penguin (*Eudyptula minor*)
 Indian pangolin (*Manis crassicaudato*)

Pelecaniformes

Red-footed booby (*Sula sula*)

Dubey, J.P., Lewis, B., Beam, K., Abbitt, B., 2002a. Transplacental toxoplasmosis in a reindeer (*Rangifer tarandus*) fetus. *Vet. Parasitol.* 110, 131–135; Dubey, J.P., Tociłowski, M.E., Abbitt, B., Llizo, S.Y., 2002b. Acute visceral toxoplasmosis in captive dik-dik (*Madoqua guentheri smithi*). *J. Parasitol.* 88, 638–641; Dubey, J.P., 2010. *Toxoplasmosis of Animals and Humans*, second ed. CRC Press, Boca Raton, FL, pp. 1–313.

Viable *T. gondii* was isolated from the hearts of 8 of 16 wild turkeys (*Meleagris gallopavo*) from Alabama (Lindsay et al., 1994). Fatal systemic toxoplasmosis has been seen in wild turkeys from Georgia (Howerth and Rodenroth, 1985) and West Virginia (Quist et al., 1995).

6.3 Toxoplasmosis in zoos

Toxoplasmosis is a zoo management problem because wild felids can excrete *T. gondii* oocysts in their feces (Jewell et al., 1972; Miller et al., 1972; Lukesova and Literak, 1998) and

feral cats occur in zoos (Gorman et al., 1986). Oocysts excreted by these felids can make their way into highly susceptible species.

Mammalian species that frequently develop toxoplasmosis in zoos include Australian marsupials (Portas, 2010), New World and arboreal monkeys (Dietz et al., 1997; Pertz et al., 1997; Juan-Salles et al., 1998; Epiphany et al., 2000), lemurs (Dubey et al., 1985), and Pallas's cats (*O. manul*) (Riemann et al., 1974; Dubey et al., 1988a, 2002a,b; Basso et al., 2005) (Fig. 6.2). Lesions in these animals are consistent with acute toxoplasmosis and are usually most severe in visceral tissues such as the lungs, liver, and spleen.

Toxoplasmosis is common in lemurs exhibited in zoo worldwide (Dubey et al., 1985). A female ring-tailed lemur (*Lemur catta*) died of toxoplasmosis in a zoo in Spain 1 week after the delivery of 4 stillborn offspring which all had disseminated toxoplasmosis (Juan-Sallés et al., 2011). *T. gondii* was isolated from the tissues of a 3-year-old secundiparous female ring-tailed lemur from a zoo in Alabama that died of acute toxoplasmosis (Spencer et al., 2004). The isolate was not pathogenic for mice and was genetically a Type II isolate. This case points out the difficulty in preventing toxoplasmosis in highly susceptible animals because this lemur was housed in a group on an island in the zoo (Spencer et al., 2004), making it easier to prevent contact with feral cats. Oocysts on the lemur's food (fruit etc.) or carried in by black birds were considered likely sources of infection in this case (Spencer et al., 2004).

Sporadic cases of acute toxoplasmosis have been reported in exhibited dik-dik (*Madoqua guentheri smithi*) (Dubey et al., 2002b), slender-tailed meerkats (*Suricata suricatta*) (Juan-Salles et al., 1997), African crested porcupines (*Hystrix cristata*) (Harrison et al., 2007), New World porcupines (Erethizontidae sp.) (Fayyad et al., 2016), and Brazilian prehensile-tailed porcupines (*Coendou mexicanus*) (Morales et al., 1996). Fatal disseminated toxoplasmosis in three captive slender-tailed meerkats (*S. suricatta*) in a

zoo in La Plata, Argentina, was found to be caused by the normally nonpathogenic genotype Type III isolate of the parasite suggesting that meerkats are highly susceptible to infection (Basso et al., 2009). A case of abortion due to *T. gondii* has been reported in a Greenland muskox (*Ovibos moshatatus wardi*) (Crawford et al., 2000).

Fatal toxoplasmosis was reported in a 7-year-old giant panda (*Ailuropoda melanoleuca*) in a zoo in China (Ma et al., 2015). The animal had acute gastrointestinal and respiratory signs and *T. gondii* was seen in lung lesions. It was anorexic and lethargic and died 2 days after it signs developed despite supportive treatment with intramuscular cephalosporin and intravenous infusion of glucose solution. Parasite DNA was detected in the liver, spleen, lung, kidney, and intestines using PCR. Antibodies to *T. gondii* were detected in sera from 7 of 19 giant pandas in the breeding program at the Chengdu Research Base of Giant Panda Breeding in Sichuan, China (Loeffler et al., 2007).

Abortion and neonatal death have been observed in captive nilgais (*Boselaphus tragocamelus*). *T. gondii* DNA was demonstrated in the tissues of the nilgais using PCR (Sedlak et al., 2004). Fatal toxoplasmosis was diagnosed in a captive, adult female saiga antelope (*Saiga tatarica*). *T. gondii* was detected in the liver, lung, spleen, kidney, and intestine and confirmed by PCR (Sedlak et al., 2004). Acute toxoplasmosis has been seen in captive Cuvier's gazelle (*Gazella cuvieri*), slender-horned gazelle (*Gazella leptoceros*), dama gazelle (*Gazella dama*), and gerenuk (*Litocranius walleri*) housed in North American Zoos (Stover et al., 1990; Junge et al., 1992). These infections are disseminated and most lesions are in the liver (Fig. 6.3), lungs, lymph nodes, adrenal glands, spleen, intestines, and brain.

Outbreaks of toxoplasmosis also occur in avian species exhibited in zoos (Poelma and Zwart, 1972; Hubbard et al., 1986). Toxoplasmosis in canaries has been reported from aviaries worldwide (reviewed by Dubey, 2002). *T. gondii*

genotype III was isolated from five of five black-winged lorys (*Eos cyanogenia*) from an acute toxoplasmosis outbreak in an aviary in South Carolina (Dubey et al., 2004d). Acute systemic toxoplasmosis was reported to be the cause of death of 3 of 10 Nicobar pigeons (*Caloenas nicobarica*) in an aviary collection in South Africa (Las and Shivaprasad, 2008). Feral cats were a known management problem and lesions were consistent with oocyst-acquired infection. Three 1–3-month-old black-footed penguin chicks (*Spheniscus demersus*) died from acute toxoplasmosis within 24 hours of showing central nervous signs (Ploeg et al., 2011). The birds were housed in a baby penguin crèche in a zoo in the Netherlands. A cat with a litter of kittens had recently been observed feeding on fish intended for the penguins in the zoo, and the cat was suspected as the source of infection (Ploeg et al., 2011).

Management and husbandry programs can be designed to help achieve prevention of toxoplasmosis in highly susceptible species in zoos and aviaries. Felids should never be fed fresh unfrozen meats because of the possibility contamination with *T. gondii* tissue cysts. Meat that has been frozen solid and then thawed can be safely fed because freezing kills *T. gondii* tissue cysts (Kotula et al., 1991). Feral cats should be actively controlled in zoos to prevent them from shedding oocysts. Highly susceptible species should not be housed near felids.

Outdoor aviaries are at risk because of oocysts excreted by domestic cats. Aviaries should be designed to exclude cat feces and transport hosts (flies, roaches, etc.) that may bring in *T. gondii* on or in their bodies.

6.4 *Toxoplasma gondii* and endangered species

Toxoplasmosis can adversely affect endangered avian and mammalian species. The ‘Alala (Hawaiian crow, *Corvus hawaiiensis*) is an

endangered species, and only about 25 were left in captivity and the wild in 2000 (Work et al., 2000). Tragically, these birds are highly susceptible to fatal toxoplasmosis and develop disease after being introduced back in to the wild. Toxoplasmosis appears to pose a significant threat and management challenge to reintroduction programs for ‘Alala in Hawaii (Work et al., 2000).

Captive breeding groups of golden lion tamarins (*L. rosalia*) have developed acute toxoplasmosis and suffered many fatalities both in North American and European zoos (Pertz et al., 1997; Juan-Salles et al., 1998). These arboreal monkeys are endangered and attempts to breed them in captivity for eventual release in the wild are hammered, because it is difficult to keep them from being exposed to *T. gondii*.

Repeated transplacental transmission of *T. gondii* by Pallas’s cats maybe responsible for the high rate of impact of this disease on the Pallas’s cat population in zoos. Efforts by North American zoos to establish genetically viable captive populations of Pallas’s cats (*O. manul*) have been compromised by high newborn kitten mortality due to toxoplasmosis (Brown et al., 2005). In their natural environment, Pallas’s cats generally have little exposure to *T. gondii*, and it is believed that they acquire *T. gondii* infection after captivity (Brown et al., 2005). The mortality rate for toxoplasmosis of Pallas’s cat kittens born in Zoos in the United States is 35%–60% (Kenny et al., 2002; Brown et al., 2005).

Sand cats (*F. margarita*) housed at the Breeding Centre for Endangered Arabian Wildlife in the United Arab Emirates and Al Wabra Wildlife Preservation, Qatar, have been reported to suffer from congenital (Pas and Dubey 2008a) and acquired toxoplasmosis (Dubey et al., 2010). Serological examination of endangered Gordon’s wildcat (*Felis silvestris gordonii*) kept at the same institution (Pas and Dubey 2008b) indicated that seropositive Gordon’s wildcats were present but no clinical

history consistent with toxoplasmosis has been reported in these animals. Unlike domestic cats, Sand cat (*F. margarita*) queens will repeatedly infect litters of kittens making it very difficult to keep up numbers of healthy kittens in breeding programs. Fortunately, Gordon's wildcats appear to behave like domestic cats in their responses to *T. gondii* infection.

6.5 Toxoplasmosis in pets

6.5.1 Cats

Most cats are asymptomatic during a primary *T. gondii* infection. Fever (40.0°C–41.7°C) is present in many cats with clinical toxoplasmosis. Clinical signs of dyspnea, polypnea, icterus, and signs of abdominal discomfort were the most frequent findings in 100 cats with histologically confirmed toxoplasmosis (Dubey and Carpenter, 1993). Uveitis and retinochoroiditis are also common clinical signs in cats with toxoplasmosis. Gross and microscopic lesions are found in many organs but are most common in the lungs. Gross lesions in the lungs consist of edema and congestion, failure to collapse, and multifocal areas of firm, white to yellow, discoloration. Pericardial and abdominal effusions may be present. The liver is the most frequently affected abdominal organ and diffuse necrotizing hepatitis may be visible grossly. Gross lesions associated with necrosis can also be observed in the mesenteric lymph nodes and pancreas.

All ages, sexes, and breeds of domestic cats are susceptible to *T. gondii* infection (Dubey et al., 1977). Transplacentally or lactogenically infected kittens will excrete oocysts, but the prepatent period is usually 3 weeks or more, because the kittens are infected with tachyzoites (Dubey et al., 1995b). Domestic cats under 1 year of age produce the most numbers of *T. gondii* oocysts. Cats that are born and raised outdoors usually become infected with

T. gondii shortly after they are weaned and begin to hunt. *T. gondii* naive adult domestic cats will excrete oocysts if fed tissue cysts, but they usually will excrete fewer numbers of oocysts and excrete oocysts for a shorter period of time than recently weaned kittens.

Intestinal immunity to *T. gondii* is strong in cats that have excreted oocysts (Dubey, 1995). Primary *T. gondii* infection in cats does not cause immunosuppression (Lappin et al., 1992; Davis and Dubey, 1995). Serum antibody does not play a significant role in resistance to intestinal infection and intestinal immunity is most likely cell mediated. Oocysts begin to be excreted in the feces before IgM, IgG, or IgA antibodies are present in the serum (Lappin et al., 1989). Partial development of the enteroepithelial stages occur in the intestines of immune cats, but oocyst production is prevented (Davis and Dubey, 1995). Most cats that have excreted oocysts once do not reexcrete oocysts if challenged within 6 months to 1 year. Intestinal immunity will last up to 6 years in about 55% of cats (Dubey, 1995).

Vaccination of cats against intestinal *T. gondii* infection has been successfully achieved using a mutant strain (T-263) of the parasite (Frenkel et al., 1991; Freyre et al., 1993). Oral administration of strain T-263 bradyzoites results in intestinal infection but does not result in oocyst production in cats. These vaccinated cats do not excrete oocysts when challenged with oocyst producing strains of *T. gondii*. The T-263 strain is safe to use in healthy cats. It is not recommended for use in pregnant cats or FeLV positive cats or immunocompromised cats (Choromanski et al., 1994, 1995). It has only limited ability to persist in the tissues of cats and cannot survive more than 3 back-passages in cats. No reversion to oocyst excretion or increase in virulence has been observed in over 200 inoculated cats. The T-263 strain is rapidly cleared from the mouth of inoculated cats.

It is logical to assume that cat owners and veterinarians would be at a greater risk for

developing toxoplasmosis; however, serological studies do not confirm this assumption. In one study in AIDS patients, it was conclusively shown that owning cats did not increase the risk of developing toxoplasmosis (Wallace et al., 1993). The role of cat ownership and exposure to *T. gondii* is, however, not completely clear at present. Many studies have been conducted to determine the association between cat ownership or cat exposure and the prevalence of *T. gondii* infection in humans. Many studies do not find a positive relationship while many find a positive relationship. It must be stressed that preventing exposure to cats is not the same as preventing exposure to *T. gondii* oocysts. Pregnant women or immunocompromised individuals should not change the cat's litter box. If feces are removed daily, this will also help prevent exposure by removing oocysts before they can sporulate. *T. gondii* oocyst can survive in the soil for years and can be disseminated from the original site of deposition by erosion, other mechanical means, and by phoretic vectors. Inhalation of oocysts stirred up in the dust by horses has been associated with an outbreak of human toxoplasmosis at a riding stable (Teutsch et al., 1979). Oocysts are not likely to remain in the air for extended periods of time. Washing fruits and vegetables and wearing gloves while gardening are means of preventing exposure to oocysts.

T. gondii oocysts were not isolated from the fur of oocyst-excreting cats (Dubey, 1995). Therefore it is unlikely that infection can be obtained by petting a cat. Tachyzoites are not likely to be present in the oral cavity of cats with active *T. gondii* infection, and none would be in a chronic infection; therefore it is unlikely that a cat bite would transmit *T. gondii* infection. Cat scratches are also unlikely to transmit *T. gondii* infection.

T. gondii has been isolated from the tissues from domestic cats worldwide (Dubey, 2010). Genotypes of feline isolates are similar to

isolates from other animals in the same geographic area.

6.5.2 Dogs

T. gondii was once confused with *Neospora caninum* as a cause of disease in dogs, and many reports of toxoplasmosis in dogs are actually neosporosis (Dubey and Lindsay, 1996; Lindsay and Dubey, 2000). True toxoplasmosis does occur in dogs (Dubey et al., 1989). Clinical toxoplasmosis in dogs is often associated with immunosuppression induced by canine distemper virus infection. Clinical signs are usually most apparent in the respiratory and hepatic systems and probably result from reactivation of latent infections (Dubey et al., 1989). Transplacental infections have not yet been confirmed in naturally infected dogs. Dogs are resistant to experimental infection with tissue cysts and oocysts (Lindsay et al., 1996, 1997a).

A role for dogs in the transmission of *T. gondii* to humans has been postulated based on serological surveys and observations that dogs ingest cat feces and often role in cat feces and other foul smelling substances (Frenkel et al., 2003). It is believed that dogs can bring oocysts to a home after ingesting them and deposit them in or around the home when they defecate. Experimentally infective *T. gondii* oocysts can be found in dog feces for up to 2 days after they ingest oocysts (Lindsay et al., 1997a). *T. gondii* oocysts will not sporulate when placed on dog fur (Lindsay et al., 1997a). Schares et al. (2005) found viable *T. gondii* oocysts in 2 of 24,089 dogs in Germany. The role of dogs as potential transport hosts for *T. gondii* needs further examination. *T. gondii* has been isolated from the tissues from domestic dogs worldwide (Dubey, 2010). Genotypes of feline isolates are similar to isolates from other animals in the same geographic area. A related

Apicomplexan parasitic protozoa *N. caninum* is present in dogs.

6.5.3 Ferrets

Congenital toxoplasmosis has been observed in farmed raised ferrets (*Mustela putorius furo*) from New Zealand (Thornton and Cook, 1986). Thirty percent of the kits on the farm died acutely and had lesions of disseminated toxoplasmosis. An epizootic of toxoplasmosis occurred among a population of endangered black-footed ferrets (*Mustela nigripes*) at a zoo in the United States (Burns et al., 2003). Twenty-two adults and 30 kits died from acute toxoplasmosis and an additional 13 adults died from chronic toxoplasmosis after the initial outbreak.

6.6 Domestic farm animals

6.6.1 Mink

Acute toxoplasmosis with abortions has been reported in farmed mink (*Mustela vison*) from Europe and the United States (Frank, 2001; Smielewska-Los and Turniak, 2004). The practice of feeding nonfrozen slaughter offal was blamed for acute toxoplasmosis in one report (Smielewska-Los and Turniak, 2004). Toxoplasmosis was diagnosed using PCR and immunohistochemistry in a young free-ranging mink (*M. vison*) that had signs of left hind limb lameness, ataxia, head tremors, and bilateral blindness and was found on a college campus in Michigan (Jones et al., 2006). *T. gondii* has been isolated from wild mink from the United States (Smith and Frenkel, 1995).

6.6.2 Horses

Horses are resistant to experimental infection with 1×10^4 or 1×10^5 oocysts. *T. gondii* can persist in edible tissues of horses for up to

476 days (Dubey, 1985). Although *T. gondii* has been isolated from tissues of horses, there is no confirmed report of clinical toxoplasmosis in horses (Al-Khalidi and Dubey, 1979). A related Apicomplexan parasitic protozoa *Neospora hughesi* is present in horses.

6.6.3 Swine

Abortion in sows is the most common sign of toxoplasmosis in swine. Sows only abort once. Abortions are rare in most pork producing regions of the world with the exception of Taiwan. Pigs raised on dirt are more likely to have *T. gondii* in their tissues. Diagnosis of *T. gondii* abortion in sows is best done by examining fetal fluids for antibodies using the modified agglutination test. Undercooked pork is a source of human infection, and viable tissue cysts can remain in pork for up to 865 days (Dubey, 1988). *T. gondii* has been isolated from the tissues from domestic pigs worldwide (Dubey, 2010). Genotypes of pig isolates are similar to isolates from other animals in the same geographic area.

6.6.4 Cattle

Clinical toxoplasmosis in cattle is rare, and abortions are uncommon. Many reports of bovine abortion due to *T. gondii* are actually due to *N. caninum* (Dubey and Lindsay, 1996). Attempts to isolate *T. gondii* from seropositive cattle are often unsuccessful indicating that beef may not be a significant source of human infection in the United States (Dubey et al., 2005). For example, no *T. gondii* was isolated from 2094 samples of beef obtained from retail markets in the United States (Dubey et al., 2005). However, viable tissue cysts can remain in cattle for up to 1191 days (Dubey and Thulliez, 1993). Additional studies are needed to full document these experimental findings.

6.6.5 Sheep

T. gondii is a common cause of abortion in ewes and an important production problem. Multiple abortions can occur in a flock indicating a common oocyst source for ewes. Ewes develop solid immunity after aborting *T. gondii* infected fetuses. A vaccine to prevent abortion in ewes is available in several countries (Buxton and Innes, 1995). Diagnosis of *T. gondii* abortion in ewes is best done by examining fetal fluids for antibodies using the modified agglutination test. Undercooked lamb and mutton is a source of human infection. *T. gondii* has been isolated from the tissues from domestic sheep worldwide (Dubey, 2010). Genotypes of sheep isolates are similar to isolates from other animals in the same geographic area.

6.6.6 Goats

T. gondii is a common cause of abortion in does. Multiple abortions can occur in a flock indicating a common oocyst source for does. Does develop immunity after aborting *T. gondii* infected fetuses but repeat abort can occur. Diagnosis of *T. gondii* abortion in goats is best done by examining fetal fluids for antibodies using the modified agglutination test. Undercooked goat meat is a source of human infection. *T. gondii* has been isolated from the tissues from domestic goat worldwide (Dubey, 2010). Genotypes of goat isolates are similar to isolates from other animals in the same geographic area. Drinking raw not pasteurized goats milk is a potential source of *T. gondii* for humans and the parasite can survive for up to 7 days in refrigerated goat milk (Walsh et al., 1999).

6.6.7 Buffalos

Naturally occurring clinical toxoplasmosis has not been observed in buffalos (*Bison bison*, *Bubalus bubalis*, *Syncerus caffer*) and viable

T. gondii has not been isolated from buffaloes. Serological surveys indicate that buffalos are exposed to the parasite.

6.6.8 Camels

Acute toxoplasmosis was observed in a 6-year-old camel (*Camelus dromedarius*) (Hagemoser et al., 1990). Dyspnea was the main clinical sign and many tachyzoites were found in its lungs and plural exudates. *T. gondii* has been isolated from camel meat using cat bioassays (Hilali et al., 1995).

6.6.9 Llamas, alpaca, and vicunas

Experimental studies indicate that llamas (*Lama glama*) are resistant to clinical toxoplasmosis even if challenged during pregnancy (Jarvinen et al., 1999). Naturally occurring toxoplasmosis has not been reported in llamas, alpacas (*Lama pacos*), or vicunas (*Lama vicugna*).

6.6.10 Chickens

Chickens (*Gallus domesticus*) that are raised on the ground are a potential source of *T. gondii* due to high level of exposure to oocysts. Chickens usually do not develop clinical signs even after oral inoculation of large numbers of oocysts (Dubey et al., 1993c; Kaneto et al., 1997). Egg production may adversely be affected in laying hens fed large numbers of oocysts, but *T. gondii* is not readily transmitted to the eggs of these hens (Biancifiori et al., 1986). Clinical toxoplasmosis does not occur on modern chicken farms where birds are raised indoors. Chickens raised in modern production facilities in confinement indoors are not likely to have viable *T. gondii* in their tissues. None of 2094 samples from commercial chickens in retail markets from the United States contained viable *T. gondii* in a survey from the United States (Dubey et al., 2005).

However, *T. gondii* has been isolated from the tissues from chickens worldwide (Dubey, 2010). Genotypes of chicken isolates are similar to isolates from other animals in the same geographic area. The prevalence of isolation is dependent on the methods used to raise the chickens with chickens raised outside having a higher prevalence of infection.

6.6.11 Turkeys

Domestic turkeys (*M. gallopavo*) fed *T. gondii* oocysts remained clinically normal except a few that develop pneumonia associated with *Aspergillus*-like fungi (Dubey et al., 1993a). Tissue cysts are present in breast and leg muscles of inoculated turkeys. Clinical toxoplasmosis does not occur on modern turkey farms. *T. gondii* has been isolated from the tissues from turkeys worldwide (Dubey, 2010). Genotypes of turkey isolates are similar to isolates from other animals in the same geographic area. The prevalence of isolation is dependent on the methods used to raise the turkeys with turkeys raised outside having a higher prevalence of infection.

6.6.12 Ducks and geese

Domestic ducks (*Anas platyrhynchos*) fed *T. gondii* oocysts do not develop clinical toxoplasmosis (Sedlak et al., 2004). Viable *T. gondii* has been isolated from the tissues of a naturally infected domestic ducks (Dubey et al., 2003) and from a domestic goose (*Anser anser*) (Dubey et al., 2007b).

6.7 Fish, reptiles, and amphibians

Toxoplasmosis does not occur in fish, reptiles, or amphibians. Reports of natural infections in these animals in nature are erroneous. Fish and reptiles can be manipulated to make

them susceptible to *T. gondii*, but they have to be experimentally infected and kept at temperatures of around 37°C–40°C. For example, zebrafish (*Danio rerio*) adapted to 37°C from 28°C were able to be infected intraperitoneally with 10 tissue cysts of the Me49 (genotype II) or VEG (genotype III) strains of *T. gondii*. Clinical signs in zebrafish included bilateral exophthalmia, swollen abdomens, whirling swimming behavior, and generalized subdermal hemorrhaging. Tachyzoites were present in tissue sections of parasites developing in muscle, heart, liver, spleen, kidney, pancreas, reproductive organs, eyes, and brain (Sanders et al., 2015).

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