REVIEW ARTICLE

Cats and *Toxoplasma*: Implications for Public Health

H. A. Dabritz[1] and P. A. Conrad[2]

[1] Infant Botulism Treatment and Prevention Program, California Department of Public Health, Richmond, CA, USA
[2] Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California, Davis, CA, USA

Impacts

- Cat owners who allow their pets outdoors should be made aware that their free-roaming cats can acquire and faecally shed the protozoan parasite, *Toxoplasma gondii*.
- Cat owners should be encouraged to keep their pets indoors and collect cat faeces in litter boxes destined for disposal in sanitary landfills.
- Persons who work with soil or garden regularly should wear gloves to protect themselves from pathogens in soil, such as *Toxoplasma gondii*, that are spread by owned and feral free-roaming cats.

Keywords:
Faecal pollution; cats; *Toxoplasma gondii;* zoonosis; toxoplasmosis

Correspondence:
H. A. Dabritz. Infant Botulism Treatment and Prevention Program, California Department of Public Health, 850 Marina Bay Pkwy, E-361, Richmond, CA 94804, USA. Tel.: +1 510 231 7603; Fax: +1 510 231 7609;
E-mail: haydee.dabritz@cdph.ca.gov

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Summary

Cats are popular as pets worldwide because they are easy to care for and provide companionship that enriches the lives of human beings. Little attention has been focused on their potential to contaminate the environment with zoonotic pathogens. One such pathogen, the protozoan parasite *Toxoplasma gondii*, rarely causes clinical manifestations in cats or immunocompetent humans; however, it can have serious adverse effects on human foetuses and immunocompromised patients. Many human infections are believed to be acquired from eating undercooked or raw meat, such as pork and lamb (Tenter et al. *Int. J. Parasitol*., 30, 2000, 1217; Dubey et al. *J. Parasitol.* 91, 2005, 1082). However, the prevalence of *T. gondii* infection in human populations that do not consume meat or eat it well-cooked suggests that the acquisition of infection from the environment, via oocysts in soil, water or on uncooked vegetables, is also important (Rawal. *Trans. Royal Soc. Trop. Med. Hyg.*, 53, 1959, 61; Roghmann et al. *Am. J. Trop. Med. Hyg.*, 60, 1999, 790; Chacin-Bonilla et al. *Am. J. Trop. Med. Hyg.*, 65, 2001, 131). In the past 20 years, two changes occurred that significantly increased the size of the cat population in the USA. Pet cat ownership grew from 50 million to 90 million animals, and animal welfare activists created feeding stations for abandoned and free-roaming cats. As many cat owners allow their cats to deposit faeces outside and cats maintained in colonies always defecate outside, ample opportunity exists for *T. gondii* oocysts to enter the environment and be transmitted to humans. Prevention efforts should focus on educating cat owners about the importance of collecting cat faeces in litter boxes, spaying owned cats to reduce overpopulation, reducing the numbers of feral cats and promoting rigorous hand hygiene after gardening or soil contact.

Introduction

The domestic cat (*Felis catus*) has a long and mostly favourable history in its association with human beings. When humans began to cultivate crops and store grain, cats were valued for their predatory abilities in controlling rodents, thereby protecting stored food from harm. In modern times, cats are appreciated for their ability to provide companionship (Patronek et al., 1996; Castelli et al., 2001; Neidhart and Boyd, 2002) as well as for

deterring rodents from invading the home (Coleman and Temple, 1993; American Pet Products Manufacturers Association (APPMA, 2005). For men with AIDS in San Francisco who lived alone, cats were important sources of companionship (Castelli et al., 2001). Forty-one per cent of US cat owners considered cats to be family members (American Veterinary Medical Association AVMA, 2007) and 18% of cat owners who had adopted a cat in the past year considered their the cat to be part of the family or felt love for their cat (Neidhart and Boyd, 2002).

With the recognition of acquired immune deficiency syndrome (AIDS) in the early 1980s, a spectrum of diseases was reported in individuals with HIV (Levy et al., 1985; Jones et al., 1999). One of these diseases was toxoplasmosis (Luft and Remington, 1992; Mamidi et al., 2002). It was estimated that 10–50% of AIDS patients with latent toxoplasmosis would develop toxoplasmic encephalitis (Luft and Remington, 1992; Porter and Sande, 1992), and at least 10% of all AIDS patients would die as a result (Jones et al., 1999). Nonetheless, the risk of acquiring *Toxoplasma gondii* from cat faeces collected in litter boxes is negligible when protective hygiene measures are employed (Wallace et al., 1993; Centers for Disease Control and Prevention, 1999). Prior to the advent of AIDS, the most commonly recognized risk for developing clinical toxoplasmosis was infection with *T. gondii* before birth, if the woman was infected with the parasite for the first time during pregnancy (Jones et al., 2001a). There is a 20–50% chance of transmitting *T. gondii* to the foetus during pregnancy (Carter and Frank, 1986; Jones et al., 2001a; Mombro et al., 2003). Several studies have also suggested that latent toxoplasmosis has detrimental neurological and behavioural effects on humans (reviewed by McAllister, 2005). Individuals latently infected with toxoplasmosis were more likely to be involved in automotive accidents (Flegr et al., 2002), to suffer from schizophrenia (Torrey and Yolken, 2003) and to score differently on tests of personality profiles (Flegr et al., 1996).

For these reasons, health professionals have raised concern about cats and their potential to contaminate the environment with *T. gondii* oocysts. The most recent estimates of the number of cats in the USA are 82 million (AVMA, 2007) and 90 million (APPMA, 2005). In Europe, estimates carried out in 1994 placed the owned cat population at 41 million animals, with the highest percentage of cat-owning households in Austria, Belgium, France, the Netherlands and Switzerland (Nott, 1996). In 2006, cats were estimated to number 21.7 million animals in France, Italy and Germany alone (MapXL Inc., http://www.mapsofworld.com/world-top-ten/countries-with-most-pet-cat-population.html; accessed 12/27/2007). The UK cat population was estimated to be 8.0 million animals in 2009, with 20% of households owning a cat (Pet Food Manufacturers' Association, http://www.pfma.org.uk/images/stories/PFma_annual_report_2009.pdf; accessed 5/8/2009). In the USA, 50–59% of cat owners in one study kept their cats indoors all the time, but the remainder allowed their pets to spend some or all of their time outside (APPMA, 2005). These estimates are supported by other studies in the USA estimating that 40–86% of owned cats are allowed outdoors, where they have ample time to prey on birds and rodents and defecate outside (Johnson and Lewellen, 1993, 1994; Luke, 1996; Patronek et al., 1996; DeFeo et al., 2002; Clancy et al., 2003; Dabritz et al., 2006, 2007a,b). About 50% of cat owners cited that one of the benefits of owning cats was their ability to catch or scare away rodents (APPMA, 2005). In a survey of rural cat owners in Wisconsin, 23% of residents on farms indicated that they kept cats for pest control (Coleman and Temple, 1993). Predation by cats on wildlife not only affects bird and rodent populations (Coleman and Temple, 1996; Hawkins et al., 1999; Jessup, 2004), but also exposes cats to parasites and pathogens maintained in wildlife. Pathogens of concern because of their zoonotic potential include rabies virus, *Yersinia pestis* (the aetiologic agent of plague) and some species of *Salmonella, Campylobacter, Giardia, Cryptosporidium*, as well as *T. gondii*.

Methods

The data presented in Tables 1–2 and 4–5 were assembled by searching PubMed (including 'Related Articles' when relevant references were located), by looking up references cited in published papers and by searching the authors' personal archives (approximately 1100 references) maintained in an EndNote database. In PubMed, the search terms 'Toxoplasma and oocyst and cat' and 'Toxoplasma and oocyst shedding' in the years of interest were used to identify studies of *T. gondii* oocyst shedding prevalence in cats; and 'Toxoplasma and soil' or 'Toxoplasma and environmental contamination' to identify studies relating to toxoplasmosis and soil contact. For the seroprevalence studies in humans and cats from Central/South and North America, PubMed search terms included 'Toxoplasma and human (or cat) and USA (or Canada or South America) and seroprevalence (or prevalence)'.

The quantity of oocysts shed by cats during a single *T. gondii* infection was estimated by summarizing data from five experimental studies of 44 cats that produced oocysts following experimental infection (Dubey, 1976, 1995, 2001, 2002, 2005), six cats experimentally infected at the University of California, Davis, and 23 naturally infected cats whose faecal results were confirmed by PCR (Schares et al., 2008). For the latter series, total faecal production was calculated by multiplying the estimated

Table 1. Studies investigating an association between *Toxoplasma gondii* infection or seropositivity and soil contact, 1990–2006

Location	Description of soil exposure	Study population (size)	OR* or RR† (95% CI)	Reference
Brazil (Rio Grande do Sul)	Contact with soil	Pregnant women (2126)	9.1%/6.8%‡ of the variance for seroprevalence	Spalding et al., 2005
Brazil (Erechim)	Gardening	Adults & children (241)	2.4* (1.3–4.3)	Jones et al., 2006
European cities§	Contact with soil	Prenatally screened women (1110)	1.8* (1.2–2.7)	Cook et al., 2000
Jordan	Soil contact (worked on land)	Pregnant women (280)	2.8* (P = 0.022)	Jumaian, 2005
Mexico (Durango City)	Lives in house with a soil floor	Pregnant women (343)	7.2* (1.4–36.8)	Alvarado-Esquivel et al., 2006a
Nigeria (Jos)	Contact with garden or soil	Healthy individuals (144)	21% (higher in soil-exposed)	Uneke et al., 2005
Panama (Panama City)	Playing in soil	Children <5 year (571)	2.8† (1.5–5.0)	Frenkel et al., 1995
Serbia	Exposure to soil	Women 15–49 year (2936)	10.3† (2.7–38.6)¶**	Bobic et al., 2003
USA (Illinois)	Gardening	Workers/residents on farms (174)	2.2* (1.1–4.5)	Weigel et al., 1999
USA	Soil-related occupation	NHANES ≥12 year (12566)	1.4* (1.1–1.9)	Jones et al., 2001b
Yugoslavia (Belgrade)	Exposure to soil (farming/gardening)	Women 15–45 year (1157)	1.4† (1.1–2.0)**	Bobic et al., 1998

*Odds ratio (rounded to 1 decimal place).
†Relative risk (rounded to 1 decimal place).
‡Most influential risk factor; % of variance for urban residents/% for rural residents.
§Copenhagen, Denmark; Brussels, Belgium; Milan and Naples, Italy; Lausanne, Suisse; Oslo, Norway.
¶Adjusted for age, location, period between diagnosis and interview, and 17 other variables.
**Statistically significant for women aged 15–19 years only.

concentration of oocysts/g by the amount of faeces produced over 8 days, i.e. 320 g based on defecation of 40 g/day (Fig. 1) (Dabritz et al., 2006). Inclusion of these cats did not change the estimate of the median total oocyst production, despite the presence of five extreme values (Fig. 1). Data from 50 cats that shed oocysts, reported by Dubey et al. (2002b), were excluded because the inoculation dose of cysts in heart or tongue from naturally infected pigs may have been higher and resulted in greater oocyst production (also presented in Fig. 1).

An equation to estimate the density of *T. gondii* oocyst loading (D) in the environment that could be applied to cat populations in the USA and other locations is presented below:

$$D = \langle [(O \times \rho_O) + E] \times \omega \times \rho_T \times K \rangle / A$$

where O = owned cat population size; ρ_O=proportion of owned cats defecating outside 100% of the time; E = feral cat population size; ω = annual faecal production per cat of 14 600 g; ρ_T=proportion of cat faeces containing *T. gondii* oocysts; K = concentration of *T. gondii* oocysts in cat faeces (e.g. 1.56×10^5 oocysts/g for infections producing 50 million oocysts shed for 8 days) and A = land area. Parameters needed for the equation that could be estimated from local survey data are the owned and feral cat population size, the proportion of owned cats defecating outside and the proportion of cats shedding *T. gondii* oocysts. Land area should incorporate urban and suburban areas of residential housing and thus Metropolitan Statistical Areas (MSAs) were used as the unit of measure for the USA. MSAs are defined as urban, suburban and rural areas adjacent to a Core-based Statistical Area (a large population centre with at least one urbanized cluster of ≥50 000 inhabitants) and all areas in adjacent counties where ≥25% of the population work in the Core-based Statistical Area.

The role of the cat in the life cycle of *Toxoplasma gondii*

Cats play an important role in maintaining *T. gondii* in nature because they are the definitive hosts for this protozoan parasite and rarely develop clinical disease as a result of infection. *Toxoplasma gondii* undergoes sexual reproduction in the felid intestine, resulting in the production of millions of environmentally resistant oocysts. There is a wide variability in the quantity of oocysts produced, varying from 3 to 810 million oocysts (and, occasionally, none) per cat infection (Dubey, 1976, 2001, 2002, 2005; Dubey et al., 2002b). Oocysts may survive for months in soil and water, thereby enhancing the probability of transmission to intermediate hosts such as birds, rodents and

Table 2. Prevalence of *Toxoplasma gondii* (or *T. gondii*-like) oocyst shedding in domestic cat faeces reported between 1985 and 2008

Location	No. tested	No. positive	%	Detection method	Reference
Australia (Adelaide)	115	0	0	Sucrose flotation	Rothe et al., 1985
Australia (Kimberley)	33	5	15	$ZnSO_4$ flotation	Meloni et al., 1993
Brazil (Sao Paulo)	237	3	1.3	Mouse bioassay*	Pena et al., 2006
Belgium	30	0	0	NaCl flotation	Vanparijs et al., 1991
Canada (Victoria)	26	0	0	Mouse bioassay	Aramini et al., 1999
Chile (Santiago)	230	10	4.3	Burrows' technique	Lopez et al., 2006
Colombia	170	0	0	Mouse bioassay*	Dubey et al., 2006
Czech Republic (Brno)	620	8	1.3	Sucrose flotation	Svobodova and Svoboda, 1986
Europe (16 countries)	24 106	26	0.11	$ZnCl_2$–NaCl flotation confirmed by PCR	Schares et al., 2008
France (Lyon)	322	0	0	Iodomercurate potassium flotation	Afonso et al., 2006
Germany (Cottbus)	264	0	0	ZnCl flotation	Knaus and Fehler, 1989
Germany (south)	100†	12	12	$ZnSO_4$ flotation†	Beelitz et al., 1992
Germany (north)	1147	7	0.6	$ZnSO_4$ flotation	Epe et al., 1993
Germany (Freiburg)	3167	35	1.1	Flotation	Barutzki and Schaper, 2003
Netherlands	305	1	0.3	Flotation	Robben et al., 2004
Panama (Panama City)	383	2	0.5	Mouse bioassay*	Frenkel et al., 1995
Puerto Rico (Mona Island)	6	0	0	Mouse bioassay*	Dubey et al., 2007
Qatar	170	57	34	10% formalin flotation	Abu-Madi 2008 (unpublished data)
Spain (La Rioja & Madrid)	382	0	0	Telemann method	Miro et al., 2004
UK (Bristol)	51	0	0	McMaster slide technique	Gethings et al., 1987
USA (Illinois)	274	5	1.8	Mouse bioassay*	Dubey et al., 1995
USA (Colorado)	206	0	0	$ZnSO_4$ flotation	Hill et al., 2000
USA (California)	326	3	0.9	$ZnSO_4$ flotation	Dabritz et al., 2007a
USA (Midwest)	12	0	0	Mouse bioassay*	de Camps et al., 2008

*Sucrose flotation followed by mouse bioassay.
†Unit of interest was faeces from a litter of kittens. None of the faeces from the 30 litters whose queens were housed exclusively indoors contained *T. gondii* oocysts.

Table 3. Estimates* of the annual quantity of *Toxoplasma gondii* oocysts entering the environment in the USA, with two assumptions for the feral cat population size

Quantity of oocysts shed per feline infection	Ownership status	Cat population size $\times 10^7$	Annual faecal production (t) $\times 10^6$	% faeces defecated outside	Annual quantity of *T. gondii* laden faeces† (t)	No. oocysts deposited outside‡	Density (oocyst/ km^2 [m^2]) in MSAs§
50 million¶	Owned	8.2	1.2	36	4310	7.39×10^{14}	4.0×10^8 (404)
	Feral**	2.7	0.4	100	3991	6.84×10^{14}	3.7×10^8 (374)
	Total	10.9	1.6		8301	1.42×10^{15}	7.8×10^8 (779)
	Owned	8.2	1.3	36	4310	7.39×10^{14}	4.0×10^8 (404)
	Feral††	7.4	1.1	100	10 804	1.69×10^{15}	9.2×10^8 (923)
	Total	15.6	2.4		15 114	2.43×10^{15}	1.3×10^9 (1328)

*Based on the equation and assumptions presented in the *Methods* section.
†Annual amount of *T. gondii*-laden faeces = annual production × % deposited outside × % containing *T. gondii* oocysts (1%).
‡Total oocysts deposited outside = annual amount of *T. gondii*-infected faeces (col. 6) × concentration (oocysts/g) × 1 million (t to g conversion).
§Metropolitan Statistical Areas = 1.83×10^6 km^2 (U.S. Census Bureau, 2000).
¶To obtain estimates based on a total of 1 million oocysts, divide the result by 50.
**Feral cats represent 25% of the total cat population.
††Feral cats represent 45% of the total cat population.

humans (Yilmaz and Hopkins, 1972; Frenkel et al., 1975). Cats shed oocysts in their faeces for 3 to 5 days after initial infection with *T. gondii,* and the shedding period lasts for a median of 8 days, although it may be as long as 3 weeks (Dubey, 1976, 2001, 2002, 2005). Duration of immunity to *T. gondii* appears to be lifelong, and shed-

Table 4. Estimates of *Toxoplasma gondii* seroprevalence in humans published since 1990 from Central/South America, the Caribbean and North America

Location	Study population	% (No. positive/ No. tested)	Method* (cutoff for a positive test)	Reference
Central/South America/Caribbean				
Argentina	Pregnant women	59 (1796/3049)	IFAT	Fuente et al., 1997
Bolivia	Children 2–14 year	43 (311/727)	DAT (1 : 8)	del Castillo and Herruzo, 1998
Brazil (Santo Inacio)	Adult & child residents	26 (34/132)	ELISA (>2 SDs of background)	Cerqueira et al., 1998
Brazil (Iraquara)		66 (72/110)		
Brazil (Jaguapita)	Rural residents	66 (227/345)	IFAT (1 : 16)	Garcia et al., 1999
Brazil (Fortaleza)	Women & children	53 (529/997)	EIA (≥6 IU/ml)	Rey and Ramalho, 1999
Brazil (Recife)	Blood donors	75 (120/160)	ELISA (OD/0.149 ≥ 1.0)	Coelho et al., 2003
Brazil (Campos dos Goytacazes)	Population-based sample of children & adults	57 (822/1436)	Micro-ELISA (commercial kit)	Bahia-Oliveira et al., 2003
Brazil (Natal)	Students 5–21 year	46 (441/959)	Micro-ELISA (≥3 IU)	de Amorim Garcia et al., 2004
Brazil (Goiania-GO)	Women of childbearing age	51 (1148/2242)	IFAT (1 : 20)	Avelino et al., 2004
Brazil (Mato Gross, Para St.)	Amazon basin tribes	46 (472/1018)†	IFAT (1 : 16)	Sobral et al., 2005
Brazil (Londrina)	Butchers	60 (28/47)	IFAT (1 : 16)	Dias et al., 2005
Brazil (Rio Grande do Sul)	Pregnant women 12–48 year	74 (1583/2126)	IFAT (cutoff not specified)	Spalding et al., 2005
Brazil (Santa Isabel do Ivai)	Adult volunteers	51 (1255/2460)‡	ELISA (commercial kit)	de Moura et al., 2006
Brazil (Jardim Sao Remo)	Children aged 1–15 year of low SES	32 (110/339)	IFAT (1 : 16)	Francisco et al., 2006
Brazil (Rondonia State)	Farm residents	73 (195/266)	MAT (1 : 25)	Cavalcante et al., 2006a
Brazil (Cascavel, Ceará St)	Pregnant women 14–43 year	70 (161/231)	ELISA (commercial kit)	Heukelbach et al., 2007
Chile	Healthy adults & children	37 (28 124/76 317)	IHAT (1 : 16)	Contreras et al., 1996
Chile (Santiago)	Adults <30 year	25 (138/560)	ELISA (>2 SDs of background)	Abarca et al., 1997
Chile (Osorno region)	Blood donors (n = 160), STD patients (n = 145)	20 (62/305)	IHAT (1 : 32)	Zamorano et al., 1999
Colombia, (Quindio)	Pregnant women	61 (569/937)	IFAT (1 : 16)	Gomez-Marin et al., 1997
Colombia (Cali)	Pregnant women	46 (437/955)‡	MEIA (commercial kit)	Rosso et al., 2008
Colombia (northwest)	Adults (n = 130 women, n = 10 men)	53 (74/140)	Immunoassay (commercial kit, >1 IU)	Pordeus et al., 2008
Costa Rica	Children & adults	76 (938/1234)	IFAT (1 : 16)	Arias et al., 1996
Cuba	Children & adults	30 (2632/8863)	ELISA	Machin Sanchez et al., 1993
Cuba (Havana Province)	Pregnant women	71 (257/362)	Micro-ELISA	Martinez Sanchez et al., 1994
Cuba (Havana)	Pregnant women	71 (3931/5537)	ELISA	Gonzalez-Morales et al., 1995
Grenada, West Indies	Pregnant women	57 (304/534)	ELISA	Asthana et al., 2006
Guatemala	Children 2 month-2 year Children 3–10 year	12 (66/532) 38 (189/500)	EIA (commercial kit)	Jones et al., 2005

Table 4. Continued

Location	Study population	% (No. positive/ No. tested)	Method* (cutoff for a positive test)	Reference
Jamaica	Pregnant women	57 (911/1604)	ELISA (commercial kit)	Prabhakar et al., 1991
Trinidad	Pregnant women	43 (129/300)	ELISA	Orrett, 1993
Venezuela (Amazonas St.)	Guajibo Amerindians	88 (106/121)	ELISA (OD > 0.3)	de la Rosa et al., 1999
Venezuela (Zulia State)	Bazi Amerindians	49 (221/447)	IHAT (1 : 64)	Chacin-Bonilla et al., 2001
Venezuela (Sierra de Perija)	Yucpa Amerindians	63 (59/94)	IHAT (1 : 64)	Diaz-Suarez et al., 2003
North America				
Canada	Cree hunters	10 (5/50)	ELISA (≥3 IU/ml)	Levesque et al., 2007
Canada (Ontario)	Veterinary staff	14 (20/141)	ELISA	Shubaiber et al., 2003
Canada (Halifax)	Children 7 months-17 years	3 (33/998)	IHAT (1 : 64)	Pereira et al., 1992
Canada (Montreal)	Caucasian adults	30 (56/189)	ELISA (0.25 above mean of negative sera)	Frappier-Davignon et al., 1990
Mexico	Women with high-risk pregnancies	35 (122/350)	ELISA	Galván Ramirez et al., 1995
Mexico	Cat owners	64 (38/59)	ELISA (OD > 0.2)	Galván Ramirez et al., 1999
Mexico (Durango)	Pregnant women	6 (21/343)	EIA (commercial kit)	Alvarado-Esquivel et al., 2006a
Mexico (Durango)	Psychiatric patients	18 (25/137)	EIA (commercial kit)	Alvarado-Esquivel et al., 2006b
Mexico (Durango)	Blood donors	7 (32/432)	EIA (commercial kit)	Alvarado-Esquivel et al., 2007
Mexico (Durango)	Waste pickers Waste workers	21 (19/90) 8 (7/83)	EIA (commercial kit)	Alvarado-Esquivel et al., 2008a
Mexico (Durango)	Adult rural residents	24 (110/463)	EIA (commercial kit)	Alvarado-Esquivel et al., 2008b
USA (Illinois)	Swine farm workers	31 (54/174)	MAT (1 : 25)	Weigel et al., 1999
USA (Maryland)	7th-day adventists & blood donors	31 (78/251)	MAT (1 : 32)	Roghmann et al., 1999
USA	NHANES, 1988–94	23 (3974/17 658)§	EIA (>6 IU)	Jones et al., 2001b
USA (Illinois)	HIV+/HIV– women with high STD risk	15 (380/2525)	EIA (commercial kit)	Falusi et al., 2002
USA	NHANES, 1999–2000	16 (669/4234)§	EIA (≥10 IU)	Jones et al., 2003b
USA	NHANES, 1988–94, participants with ethnicity data	25 (3787/14909)§	EIA (commercial kit)	Kruszon-Moran and McQuillan, 2005
USA	NHANES, 1999–2004, 6–49 year	11 (1712/15960)§	EIA (commercial kit)	Jones et al., 2007

*DAT, direct agglutination test; EIA, Enzyme immunoassay; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; IHAT, indirect haemagglutination test; MAT, modified agglutination test; MEIA, microparticle enzyme immunoassay.
†By ELISA (>2 SDs above mean background), 539/1018 (53%) tested positive.
‡Denominator calculated from number and percentage with positive results.
§Age-adjusted.

ding of oocysts by cats is unlikely following re-infection with *T. gondii*, unless the cat is exposed >6 years after initial infection (Dubey, 1995), given high doses of corticosteroids (Dubey, 1995), significantly undernourished (Ruiz and Frenkel, 1980a) or superinfected with other coccidian parasites (Dubey, 1976). Despite the short patent period for oocyst shedding and its singular occurrence in the cat's lifetime, the number of cats spending time outside and their propensity for hunting are likely to ensure a steady supply of susceptible definitive hosts capable of acquiring *T. gondii* parasites.

Feral cat populations

Owned cats represent only one component of the population potential for the domestic cat to contaminate the

Table 5. Estimates of *Toxoplasma gondii* seroprevalence in domestic cats (*Felis catus*) published since 1990 from Central/South America, the Caribbean and North America

Location	Study population	% (No. positive/ no. tested)	Method* (cutoff for a positive test)	Reference
Central/South America/Caribbean				
Brazil (Jaguapita)	Rural cats	73 (119/163)	IFAT (1 : 16)	Garcia et al., 1999
Brazil (Sao Paulo)	Patients at vet teaching hospital	18 (44/248)	IFAT (1 : 16)	Lucas et al., 1999
Brazil (Guarulhos, Sao Paulo)	Strays (n = 470), breeder (n = 32)	26 (132/502)	MAT (1 : 20 or 1 : 25)	Silva et al., 2002
Brazil (Parana)	Free-roaming cats	84 (49/58)†	MAT (1 : 20)	Dubey et al., 2004
Brazil (Sao Paulo metro area)	Strays housed at IUPA	40 (40/100)	ELISA (OD > 2.0)	Meireles et al., 2004
Brazil (Sao Paulo State)	Stray cats	35 (84/237)	MAT (1 : 25)	Pena et al., 2006
Brazil (Rondonia State)	Free-roaming cats	87 (55/63)	MAT (1 : 25) & IFAT (1 : 25)	Cavalcante et al., 2006b
Brazil (Sao Paulo)	Relinquished to Center of Zoonosis Control	25 (100/400)	IFAT (1 : 64)	Bresciani et al., 2007
Colombia (Bogota & Armenia)	Unwanted cats	32 (52/170)	MAT (1 : 20)	Dubey et al., 2006
Grenada, West Indies	Domestic cats	35 (14/40)	MAT (1 : 25)	Asthana et al., 2006
Panama (Panama City)	Free-roaming cats	46 (110/241)	DAT (not reported)	Frenkel et al., 1995
Puerto Rico (Mona Island)	Feral cats	84 (16/19)	MAT (1 : 10)	Dubey et al., 2007
St Kitts, West Indies	Domestic cats	85 (90/106)	MAT (1 : 20)	Moura et al., 2007
North America				
Canada (Victoria)	Cats at veterinary clinics	22 (16/73)	MAT (1 : 25)	Aramini et al., 1999
Canada (British Columbia)	Owned cats	23 (50/221)	IFAT (not reported)	Aramini et al., 1999
Mexico (Mexico City)	Feral cats	67 (4/6)	CF (not reported)	Suzan and Ceballos, 2005
USA (Georgia, Florida, Ohio)	Cats with uveitis	41 (51/124)	ELISA‡ IgG (1 : 64)	Lappin et al., 1992
USA (Iowa)	Swine farm cats	42 (31/74)	MAT (1 : 32)	Smith et al., 1992
USA (Illinois)	Swine farm cats	76 (223/295)	MAT (1 : 25)	Dubey et al., 1995
USA (Iowa)	Free-ranging cats	80 (16/20)	MAT (1 : 32)	Hill et al., 1998
USA (Rhode Island)	Veterinary clinics (n = 116), animal shelters (n = 84)	42 (84/200)	MAT (1 : 25)	DeFeo et al., 2002
USA (Ohio)	Outdoor/feral (n = 252), unknown (n = 23)	48 (133/275)	MAT (1 : 25)	Dubey et al., 2002
USA (Florida)	Feral cats at spay/neuter clinic	9 (49/533)	ELISA‡ IgG (1 : 64)	Luria et al., 2004
USA	Clinically ill cats	29 (3619/12 628)	ELISA‡ IgG (1 : 64)	Vollaire et al., 2005
USA (Channel Islds, California)	Feral cats	34 (31/92)	IFAT (1 : 640)	Clifford et al., 2006
USA (San Luis Obispo County, California)	Owned (n = 43), stray at shelter (n = 80)	11 (13/123)	ELISA‡ IgG (1 : 64)	Dabritz et al., 2007b
USA (Pennsylvania)	Owned	20 (25/122)	MAT (1 : 25)	Dubey et al., 2008

*CF, complement fixation; DAT, direct agglutination test; ELISA, enzyme-linked immuno-sorbent assay; IFAT, indirect fluorescent antibody test; MAT, modified agglutination test.

†*Toxoplasma gondii* parasites were isolated from 37/54 cats by bioassay in laboratory-reared cats or mice.

‡To facilitate comparison, only IgG-positive results are summarized.

environment with *T. gondii* oocysts. There are large populations of unowned cats worldwide. Cats that have grown up with little or no human contact and will not socialize with humans are termed 'feral' (Levy and Crawford, 2004). Feral cats, by definition, cannot be adopted and will spend their entire lives wandering freely outdoors. Animals lost or abandoned by prior owners are usually adoptable, and such cats may fluctuate between the unowned and owned cat populations (Patronek, 1998). It is estimated that between 14% and 36% of cats are acquired as strays (Rochlitz, 2000), suggesting that large numbers of cats are separated from their original owners. In the early 1990s, feral cats became the focus of rescue efforts by cat lovers and activists in Europe, the

UK and the USA. Feral cat activists began vigorous efforts to protect abandoned and stray cats from euthanasia by animal control agencies, and as a result large numbers of feral cats now live in the USA. (Levy and Crawford, 2004).

Many feral cats are free-living in colonies of 10 or more animals, maintained by caretakers who provide food and some veterinary care, including vaccination and spaying or neutering to control colony size (Centonze and Levy, 2002; Stoskopf and Nutter, 2004; Winter, 2004). The total number of cat colonies in the USA and Europe is not known. Population control in feral cat colonies has had variable success, and may be hampered by the inability to trap all the sexually mature animals (Nutter et al., 2004), lack of money for ongoing spay/neuter programmes, lack of community support for the cat colony and influx of non-resident cats into the colony (Mahlow and Slater, 1996; Winter, 2004). There is a wide variability in estimates of the size of the feral cat population, depending on the region of USA where surveys are implemented and methods used to make calculations. Some estimates place the number as high as 25 to 40 million feral cats, with feral cats representing >50% of the total cat population (Patronek and Rowan, 1995). Other studies from several states in the USA, based on the number of strays reportedly fed by householders, suggest more conservative estimates, with feral cats representing 25–45% of the cat population (Johnson and Lewellen, 1993, 1994; Levy and Crawford, 2004; Dabritz et al., 2006). However, the latter method of calculation does not account for feral cats that visit >1 household, which could result in an overestimate of the feral cat population size. Alternatively, the presence of managed cat colonies could lead to an underestimation of the number of feral cats.

While advocates of protection for feral cats in colonies claim that these cats have negligible impacts on the environment and wildlife, lower reproductive capacity than reported and short lifespan, there is a growing body of scientific evidence to show that feral cats cause negative effects through disturbance to ecological communities and predation on wildlife (Jessup, 2004; Winter, 2004). Feral and owned free-roaming cats also help spread pathogens such as *T. gondii* to humans and livestock, as well as maintaining wildlife reservoirs (Frenkel et al., 1995; Weigel et al., 1995; Lehmann et al., 2003). Weigel et al. (1995) found that *T. gondii* seroprevalence in finishing pigs increased when more seropositive juvenile cats were present on the farm. Lehmann et al. (2003) also demonstrated that *T. gondii* infection of wildlife was four times as likely within 50 m of pig sties compared with that further away, suggesting that oocysts from cats were disseminated into the surrounding habitat. *Toxoplasma gondii* oocysts may spread from the original defecation site via the action of water, earthworms and insects (Wallace, 1972; Ruiz and Frenkel, 1980b; Saitoh and Itagaki, 1990; Bowie et al., 1997).

Areas of high freshwater outflow were recognized as a risk factor for *T. gondii* infection in the southern sea otter (*Enhydra lutris nereis*) (Miller et al., 2002), and human outbreaks of toxoplasmosis have been linked to drinking water contaminated with land runoff (Bowie et al., 1997; de Moura et al., 2006; Palanisamy et al., 2006). Evidence collected from these outbreaks included a 3-fold higher risk for toxoplasmosis in women who lived in residences receiving water from the Humpback Reservoir in the 1995 British Columbia (Canada) outbreak versus residents outside the distribution system (Bowie et al., 1997); isolation of *T. gondii* parasites from stored water during the 2001 outbreak in Santa Isabel do Ivai, Brazil (de Moura et al., 2006) and the observation that the majority of persons affected in the 2004 Coimbatore (India) outbreak received water from a single reservoir (Palanisamy et al., 2006). Several epidemiological studies have also identified soil contact and frequent gardening as risk factors for *T. gondii* infection (summarized in Table 1). Cook et al. (2000) reported that pregnant women with acute *T. gondii* infections were almost twice as likely to have had soil contact compared with uninfected controls (see Table 1), and a case-control study conducted in Erecheim, Brazil (Jones et al., 2006), demonstrated that adults and children who worked in the garden were 2.4 times more likely to be diagnosed with acute toxoplasmosis than those who did not garden. The 1988–1994 US National Health and Nutrition Examination Survey (NHANES) determined that *T. gondii* seroprevalence was higher in non-Hispanic white and Mexican–American persons who worked in soil-related occupations than those of the same ethnicity who did not have occupational exposure (37% versus 24% and 34% versus 24%, respectively) (Jones et al., 2001b). Unfortunately, most people are unaware that they can acquire toxoplasmosis from soil or water (Jones et al., 2003a; Humane Society of the United States, 2005).

Estimates of the quantity of *Toxoplasma gondii* oocysts in the environment

There are few estimates for the amount of cat faeces produced and where it ends up in the environment. In San Francisco, pet faeces make up nearly 4% of all residential waste (Jones, 2006). Every day, a cat generates approximately 40 g of faecal waste (Dabritz et al., 2006), suggesting that the annual faecal production for the 82–90 million owned cats in the USA amounts to 1.2–1.3 million tonnes (t). In addition to the amount of outdoor faecal deposition by cats, estimation of the quantity of

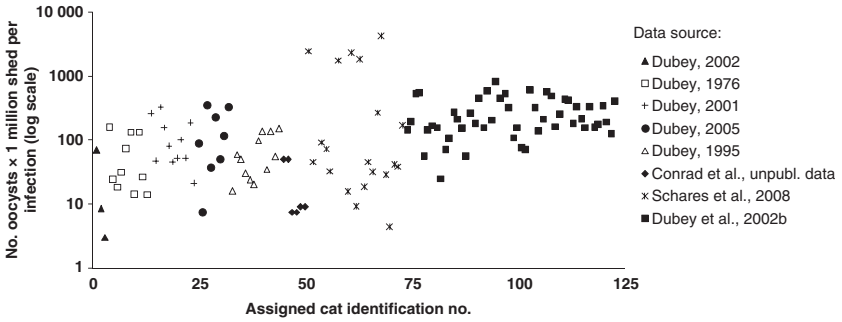

Fig. 1. The quantity of *Toxoplasma gondii* oocytes shed by experimentally or naturally infected cats fed p.o. tachyzoites or bradyzoites in pig or murine tissues, or peritoneal exudates from mice. Mean quantity shed including Dubey et al., 2002b: 253 million oocysts, median: 125 million oocysts. Mean quantity shed excluding Dubey et al., 2002b: 244 million oocysts, median: 50 million oocysts.

T. gondii soil contamination requires knowledge of the annual incidence of *T. gondii* in cats from the area of interest. Such studies are impractical, because they would require repeated sampling of faeces or serum from a large population of cats for a year or more. The only published study to date that measured *T. gondii* incidence in cats occurred at a feral cat colony in France, where *T. gondii* incidence was calculated to be 0.17 infections per cat-year (Afonso et al., 2006). The same study also demonstrated that *T. gondii* antibody titres in naturally infected individual cats were stable over the 10-year study period, and these data are supported by an earlier experimental study (Dubey, 1995). A study from coastal California estimated a somewhat lower incidence than the study by Afonso et al. (2006) for owned cats of 0.04 infections per cat-year, based on an age-adjusted seroprevalence of 29.6%, a median survival of 7 years and a population of 7284 owned cats representing 50 988 cat-years at risk (Dabritz et al., 2007b). In the absence of longitudinal studies, estimates of *T. gondii* soil contamination by cats may be based on observational studies of the prevalence of *T. gondii* oocyst shedding in cat faeces. These studies are hampered by a number of factors: the poor sensitivity of oocyst detection methods (Dabritz et al., 2007a); the inability of microscopy to distinguish *T. gondii* oocysts from the oocysts of the related feline coccidian species, *Hammondia hammondi* and *Besnoitia darlingi* (Dubey and Sreekumar, 2003); the difficulty of obtaining adequately fresh faecal samples from cat owners (collected <24 h of defecation) and the inability of the owners of outdoor (high-risk) cats to locate their pet's faeces (Dabritz et al., 2007a). Finally, while mouse bioassay is the most sensitive method currently available to detect *T. gondii* oocysts in cat faeces, it is both expensive and time-consuming (Ruiz and Frenkel, 1980a; Rothe et al., 1985). The estimates generated from observational studies of oocyst shedding prevalence are, therefore, likely to be conservative.

With these caveats, a summary of surveys to detect oocyst shedding in domestic cats from 1985 to 2008 is presented in Table 2. Prevalence in these surveys ranged from 0% to 34%, but was typically ≤1%. Fourteen of the 22 surveys used centrifugation–flotation methods to determine oocyst shedding prevalence, which has an analytical sensitivity of 250–300 oocysts/g of faeces (Rothe et al., 1985; Dabritz et al., 2007a), and only five used mouse bioassay. The surveys employing mouse bioassay did not necessarily detect higher oocyst shedding prevalence than those utilizing flotation and microscopy. The largest and most recent study (Schares et al., 2008) encompassed 16 countries in Europe and confirmed microscopical identification using PCR, finding a mere 0.11% of cats that were shedding *T. gondii* oocysts. Low prevalence may be related to the management of the cat populations studied, as the risk for exposure to *T. gondii* parasites is greatest in cats that prey on wildlife and live outdoors or on farms (Weigel et al., 1995; Dubey et al., 2002a; Lehmann et al., 2003; Clifford et al., 2006). Kittens may also be highly susceptible to infection and shed greater quantities of oocysts (Dubey et al., 1977; Dubey, 2002), although Schares et al. (2008) found no significant difference in the concentration of oocysts in the faeces of kittens versus adult cats. The three studies in Table 2 with the highest oocyst shedding prevalence, which was not confirmed by molecular methods, were undertaken in high-risk populations: litters of farm kittens whose faeces were tested in pools (Beelitz et al., 1992) and free-roaming cats in rural Aboriginal communities (Meloni et al., 1993) and Qatar (Abu-Madi M.A., unpublished data). Similar to the findings of the southern German study (Beelitz et al., 1992), Ruiz and Frenkel (1980a) found that approximately 50% of kittens in Costa Rica, defined as animals weighing <600 g or approximately 2 months old, were shedding *T. gondii* oocysts. In an earlier study of unwanted or stray cats brought to a humane society

in Hawaii, four of six cats found shedding oocysts were aged 4–6 months (Wallace, 1971).

Despite the putative single oocyst shedding period for cats, free-roaming cats may be capable of disseminating large quantities of oocysts into the environment. The output of such a calculation for the USA is presented in Table 3, assuming that feral cats comprise either 25% or 45% of the total cat population. This estimate (779–1728 oocysts/m^2) would be >10 times lower than that of Sousa et al. (1988), if the latter estimate of 193–774 oocysts/m^2, which was based on production of 1 million oocysts per feline infection, was multiplied by 50 for comparison purposes (i.e. 9650–38 700 oocysts/m^2 for the 50–200 cats that lived in this 7.7-ha rural Panamanian community). The estimate in Table 3 is also approximately four times lower than the 4671 oocysts/m^2 environmental burden reported for three coastal communities in California with approximately 10 000 owned and feral cats, using the same estimate (50 million oocysts) for the total oocyst production per feline *T. gondii* infection (Dabritz et al., 2007a). This difference is probably related to the larger land area represented by MSAs, which constitute approximately 20% of the US land area. The actual oocyst loading could be more concentrated around feral cat colonies or residences with high proportions of cat ownership and outdoor cats (Dabritz et al., 2006; Afonso et al., 2008).

In Central and South America, ≥80% of cats are free-roaming and defecate outside (Ruiz and Frenkel, 1980a), whereas 8–17% are kept exclusively outdoors in the USA (Johnson and Lewellen, 1993, 1994; Luke, 1996; Patronek et al., 1997; Clancy et al., 2003). We found similar proportions in the Morro Bay area, California, with 10% of cats from a population-based telephone survey and 5% of cats from a *T. gondii* faecal survey maintained outside all the time. Given the different life styles of cats in these two regions, it is likely that soils in Central and South America are more highly contaminated with *T. gondii* oocysts than those in North America, as suggested by the estimate of *T. gondii* loading from Panama (Sousa et al., 1988). Studies of *T. gondii* seroprevalence in humans from Central and South America (summarized in Table 4) also provide evidence to suggest that environmental contamination is higher in this region than in North America. Human seroprevalence in North American countries ranged from 3% to 64%, while in Central and South American countries it was 12–88%, with the lowest prevalence in studies that included children and young adults (Abarca et al., 1997; Jones et al., 2005; Francisco et al., 2006). Seroprevalence data for humans and animals in Europe and many other countries may be found in a comprehensive review by Tenter et al. (2000). In Central and South America, seroprevalence in cats (Table 5) was 18–87%, with only three of 10 studies in Table 5 reporting seroprevalences ≥73%. *Toxoplasma gondii* seroprevalence estimates in North American cats range between 9% and 80%, with the highest seroprevalences reported for farm cats (Smith et al., 1992; Dubey et al., 1995), feral or outdoor cats (Hill et al., 1998; Dubey et al., 2002a; Clifford et al., 2006) and cats with uveitis (Lappin et al., 1992). The prevalence of *T. gondii* in cats from the two regions may be similar, so the greater contamination of the environment in Central and South American countries is most likely related to management of cat faeces. Forty per cent of cat owners in a study from the USA (Dabritz et al., 2006) reported collecting all of their cat's faeces in litter boxes, but most households in Central and South America do not (Ruiz and Frenkel, 1977, 1980a).

The risk of acquiring *Toxoplasma gondii* infection from the environment

Given the estimated level of contamination in the US soils (Table 3), what is the risk of a human being acquiring *T. gondii* infection through contact with soil or surface water? Although *T. gondii* oocysts were not recovered from soil in two environmentally implicated human outbreaks of toxoplasmosis in the USA (Teutsch et al., 1979; Stagno et al., 1980), contaminated soil was strongly suspected as the source. Mice and/or cats infected with *T. gondii* were found living at both locations. Epidemiological studies from European countries, the Middle East, South America and the USA have also demonstrated significant associations between soil contact and *T. gondii* seropositivity in humans (Table 1) (Frenkel et al., 1995; Weigel et al., 1999; Cook et al., 2000; Jones et al., 2001b, 2006; Nimri et al., 2004; Jumaian, 2005; Nash et al., 2005). Soil-related risk factors included frequent gardening and occupations that involved regular contact with soil, such as farming. In Brazil, Costa Rica, France and Japan, *T. gondii* oocysts have been detected in soil using flotation–centrifugation, followed by inoculation into mice or PCR (Ruiz et al., 1973; Ito et al., 1975; Coutinho et al., 1982; Afonso et al., 2008). Human outbreaks of toxoplasmosis have also been linked to water contaminated by runoff from the surrounding terrain (Benenson et al., 1982; Bowie et al., 1997; de Moura et al., 2006; Palanisamy et al., 2006). However, no *T. gondii* oocyst was found in water samples taken during the investigation of the 1995 British Columbia outbreak (Isaac-Renton et al., 1998). The outbreak occurred following a week of heavy rain, and *T. gondii* oocysts were detected in two of 23 scats or faeces collected from necropsied cougars (*F. concolor vancouverensis*) (Aramini et al., 1998). This finding suggested that infected faeces from wild cougars

were washed into the reservoir. In a more recent toxoplasmosis outbreak involving contaminated drinking water in Santa Isabel do Ivai, Brazil (2001–02), bioassays of stored reservoir water in pigs and chickens at two different laboratories confirmed the presence of *T. gondii* oocysts (de Moura et al., 2006). During the investigation of the same outbreak, a queen and her kittens were found living on top of a cistern that provided residential drinking water. Oocysts have also been detected in underground and raw surface water by PCR methods in France (Villena et al., 2004) and in underground (well) water from farms in Poland (Sroka et al., 2006). These reports implicate feline faeces as an important source of environmental contamination for *T. gondii* oocysts.

Detection of oocysts in environmental samples is hampered by the low concentration of oocysts in soil and water, and by the lack of techniques with sufficient sensitivity to detect their presence (Rothe et al., 1985; Isaac-Renton et al., 1998; Kourenti and Karanis, 2006; Dabritz et al., 2007a). The minimal infectious dose for pigs and mice is as low as a single oocyst (Dubey et al., 1996, 1997), and is believed to be of similar magnitude for humans. Therefore, the ability to detect low quantities of oocysts present in environmental samples is a critical requirement for studies of *T. gondii* pathogen pollution and human health risk. Many advances have been made with regard to isolation of *T. gondii* oocysts from the sample matrix (Kourenti et al., 2003; Dumètre and Dardé, 2005; Kourenti and Karanis, 2006), but these methods are time-consuming and expensive. Schwab and McDevitt (2003) developed a PCR-enzyme immunoassay capable of detecting DNA when slightly fewer than 50 oocysts were present (Schwab and McDevitt, 2003). Dumètre and Dardé (2005) produced monoclonal antibodies (mAbs) to the *T. gondii* oocyst wall, one of which (4B6) was used in combination with filtration and immunomagnetic separation to detect *T. gondii* oocysts in drinking and surface water (Dumètre and Dardé, 2007). However, this mAb cross-reacted with the oocysts of three other coccidian species and the recovered oocysts had to be confirmed by *T. gondii*-specific PCR. In the same study, oocyst recoveries of 14–34% were achieved in drinking water containing 10 oocysts/l, but were unsuccessful at the same concentration in surface water. More sensitive techniques are needed to detect *T. gondii* oocysts occurring at low concentrations in environmental samples, and mAbs specific to the *T. gondii* oocyst outer wall antigens are needed to separate oocysts from debris in the surface water matrix. Until better techniques are readily available, detection of *T. gondii* in environmental samples will continue to present challenges.

Mitigating the environmental impact of cats

In the absence of widely available methods to test for the presence of *T. gondii* oocysts in the environment and thereby assess the potential health hazard in specific areas, public health efforts should be directed towards educating the public and reducing the environmental impact of cats. Simple hygiene measures, such as wearing disposable gloves when gardening or working in soil and thorough hand washing after soil contact, would prevent many human *T. gondii* infections. Most cat owners are probably unaware that allowing their pets outside may facilitate transmission of *T. gondii* between cats and rodents, particularly if their cat is using the outdoors as a litter box. Collecting cat faeces in indoor litter boxes and disposing of them in garbage destined for landfills, where runoff is controlled, will prevent the spread of *T. gondii* to wildlife and unsuspecting humans. On farms, cats should be excluded from barns housing animals whose meat is destined for the human food chain. Higher numbers of free-roaming cats on farms were associated with an increased risk for *T. gondii* infection in swine (Weigel et al., 1995).

Veterinarians should continue to educate cat owners about the importance of spaying and neutering their cats to control the size of the owned cat population. Four websites with online educational material about toxoplasmosis are listed in Table 6. Responsible cat ownership

Table 6. Online educational resources about toxoplasmosis for the public and cat owners

Organization	Location	Website
AVMA	Schaumburg, IL	http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure.pdf (English) http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure_spanish.pdf (Spanish)
Centers for Disease Control and Prevention	Atlanta, GA	http://www.cdc.gov/toxoplasmosis/pdfs/toxo_cat_owners_8_2004.pdf
Cigna	Philadelphia, PA	http://www.cigna.com/healthinfo/tn7481.html
University of California	Davis, CA	http://www.seaotterresearch.org/Facts_about_Toxo_and_Cat_poop.pdf (English) http://www.seaotterresearch.org/Toxo_gondii_Lo_que_todos_debemos_saber.pdf (Spanish)

should also be encouraged. This includes measures such as keeping cats indoors and collecting faeces in litter boxes for ultimate disposal in garbage destined for landfills, which are designed to prevent waste materials leaking into groundwater. In addition, cat faeces should not be disposed of in toilets, because experimental evidence suggests *T. gondii* oocysts survive chemical and physical inactivation treatments at levels four to six times higher than those used to treat raw sewage (Wainwright et al., 2007a,b). California became the first US state to enact legislation (AB2485, summary available at http://www.seaotters.org/CurrentIssues/index.cfm?DocID = 319; accessed 5/8/2009) aimed at protecting surface waters and ultimately wildlife from exposure to *T. gondii* oocysts in cat faeces. The bill requires manufacturers of cat litter to label their packaging with an advisory cautioning against cat litter disposal in toilets.

Other measures to reduce environmental contamination with *T. gondii* oocysts involve feral and unowned cats. Free-roaming cats, including animals abandoned by their owners, should be placed in shelters or surrendered to rescue groups where they have an opportunity to be adopted, rather than left to live outside without provisions. Cats lacking socialization with humans, particularly those born and raised in the wild, are likely to become feral. Discussions of the options for feral cat management are hotly debated and have been published elsewhere (Jessup, 2004; Stoskopf and Nutter, 2004; Winter, 2004). Feral cat colonies should be located away from public parks, areas known to be inhabited by wildlife susceptible to cat predation, and water sources, including reservoirs, streams, lakes, estuaries and tidal bays.

To protect the public health, similar to what occurred with regard to canine rabies in the USA, efforts to develop a *T. gondii* vaccine for cats should be renewed. Universal vaccination of owned and feral cats against *T. gondii* would prevent or decrease the production of *T. gondii* oocysts, and could ultimately improve public health by reducing the quantity of oocysts in the environment. While potential vaccine candidates were identified in the past, one product required a strict cold chain and could cause clinical toxoplasmosis in inoculated cats co-infected with FeLV (Hermentin and Aspock, 1988; Choromanski et al., 1995). Furthermore, it consisted of attenuated live bradyzoites derived from mice, so that mass production would have been labour-intensive and unprofitable. Efforts to develop different *T. gondii* vaccines are underway (Mishima et al., 2002; Milt McAllister, personal communication), but production of a commercially available vaccine is years away. In the interim, measures to mitigate the environmental impact of cats, if implemented consistently nationwide, could reduce the extent of soil contamination with *T. gondii* oocysts and the risk to human health.

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