Cats and Toxoplasma: Implications for Public Health

H. A. Dabritz¹ and P. A. Conrad²

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

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

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; Rognmann 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.
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 them 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., 2007). 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 Yolk, 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.mapofworld.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 aetiological 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 (Shares et al., 2008). For the latter series, total faecal production was calculated by multiplying the estimated
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) (Dubey 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 *Toxoplasma gondii* oocyst loading (*D*) in the environment that could be applied to cat populations in the USA and other locations is presented below:

\[
D = \frac{([O \times \rho_0] + E) \times \omega \times \rho_T \times K}{A}
\]

where *O* = owned cat population size; \(\rho_0\) = proportion of owned cats defecating outside 100% of the time; *E* = feral cat population size; \(\omega\) = annual faecal production per cat of 14 600 g; \(\rho_T\) = proportion of cat faeces containing *T. gondii* oocysts; *K* = concentration of *T. gondii* oocysts in cat faeces (e.g. 1.56 \(\times\) 10\(^7\) 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 \(\geq 250,000\) inhabitants) and all areas in adjacent counties where \(\geq 25\%\) 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
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

<table>
<thead>
<tr>
<th>Location</th>
<th>Study population</th>
<th>% (No. positive/No. tested)</th>
<th>Method* (cutoff for a positive test)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central/South America/Caribbean</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Argentina Pregnant women</td>
<td>59 (1796/3049)</td>
<td>IFAT</td>
<td>Fuente et al., 1997</td>
<td></td>
</tr>
<tr>
<td>Bolivia Children 2–14 year</td>
<td>43 (311/727)</td>
<td>DAT (1 : 8)</td>
<td>del Castillo and Herruzo, 1998</td>
<td></td>
</tr>
<tr>
<td>Brazil Adult &amp; child residents</td>
<td>26 (34/132)</td>
<td>ELISA (&gt;2 SDs of background)</td>
<td>Cerqueira et al., 1998</td>
<td></td>
</tr>
<tr>
<td>Brazil (Santo Inacio)</td>
<td>66 (72/110)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brazil (Jaguapita) Rural residents</td>
<td>66 (227/345)</td>
<td>IFAT (1 : 16)</td>
<td>Garcia et al., 1999</td>
<td></td>
</tr>
<tr>
<td>Brazil (Fortaleza) Women &amp; children</td>
<td>53 (529/997)</td>
<td>EIA (26 IU/ml)</td>
<td>Rey and Ramalho, 1999</td>
<td></td>
</tr>
<tr>
<td>Brazil (Recife) Blood donors</td>
<td>75 (120/160)</td>
<td>ELISA (OD/0.149 ≥ 1.0)</td>
<td>Coelho et al., 1999</td>
<td></td>
</tr>
<tr>
<td>Brazil (Campos dos Goytacazes)</td>
<td>57 (822/1436)</td>
<td>Micro-ELISA (commercial kit)</td>
<td>Bahia-Oliveira et al., 2003</td>
<td></td>
</tr>
<tr>
<td>Brazil (Natali) Students 5–21 year</td>
<td>46 (441/959)</td>
<td>Micro-ELISA (≥3 IU)</td>
<td>de Amorim Garcia et al., 2004</td>
<td></td>
</tr>
<tr>
<td>Brazil (Goiania-GO) Women of childbearing age</td>
<td>51 (1148/2242)</td>
<td>IFAT (1 : 20)</td>
<td>Avelino et al., 2004</td>
<td></td>
</tr>
<tr>
<td>Brazil (Mato Gross, Para St.) Amazon basin tribes</td>
<td>46 (472/1018)³</td>
<td>IFAT (1 : 16)</td>
<td>Sobral et al., 2005</td>
<td></td>
</tr>
<tr>
<td>Brazil (Londrina) Butchers</td>
<td>60 (28/47)</td>
<td>IFAT (1 : 16)</td>
<td>Dias et al., 2005</td>
<td></td>
</tr>
<tr>
<td>Brazil (Rio Grande do Sul)</td>
<td>74 (1583/2126)</td>
<td>IFAT (cutoff not specified)</td>
<td>Spading et al., 2005</td>
<td></td>
</tr>
<tr>
<td>Brazil (Santa Isabel do Ivai) Adult volunteers</td>
<td>51 (1255/2460)²</td>
<td>ELISA (commercial kit)</td>
<td>de Moura et al., 2006</td>
<td></td>
</tr>
<tr>
<td>Brazil (Jardim Sao Remo)</td>
<td>32 (110/339)</td>
<td>IFAT (1 : 16)</td>
<td>Francisco et al., 2006</td>
<td></td>
</tr>
<tr>
<td>Brazil (Rondonia State) Farm residents</td>
<td>73 (195/266)</td>
<td>MAT (1 : 25)</td>
<td>Cavalcante et al., 2006a</td>
<td></td>
</tr>
<tr>
<td>Brazil (Cascavel, Ceará St) Pregnant women 14–43 year</td>
<td>70 (161/231)</td>
<td>ELISA (commercial kit)</td>
<td>Heukelbach et al., 2007</td>
<td></td>
</tr>
<tr>
<td>Chile Healthy adults &amp; children</td>
<td>37 (28 124/76 317)</td>
<td>IHAT (1 : 16)</td>
<td>Contreras et al., 1996</td>
<td></td>
</tr>
<tr>
<td>Chile (Santiago) Adults &lt;30 year</td>
<td>25 (138/560)</td>
<td>ELISA (&gt;2 SDs of background)</td>
<td>Abarca et al., 1997</td>
<td></td>
</tr>
<tr>
<td>Chile (Osorno region) Blood donors (n = 160), STD patients (n = 145)</td>
<td>20 (62/305)</td>
<td>IHAT (1 : 32)</td>
<td>Zamorano et al., 1999</td>
<td></td>
</tr>
<tr>
<td>Colombia, (Quindio) Pregnant women</td>
<td>61 (569/937)</td>
<td>IFAT (1 : 16)</td>
<td>Gomez-Marin et al., 1997</td>
<td></td>
</tr>
<tr>
<td>Colombia (Cali) Pregnant women</td>
<td>46 (437/955)¹</td>
<td>MEIA (commercial kit)</td>
<td>Rosso et al., 2008</td>
<td></td>
</tr>
<tr>
<td>Colombia (northwest) Adults (n = 130 women, n = 10 men)</td>
<td>53 (74/140)</td>
<td>Immunoassay (commercial kit, &gt;1 IU)</td>
<td>Pordeus et al., 2008</td>
<td></td>
</tr>
<tr>
<td>Costa Rica Children &amp; adults</td>
<td>76 (938/1234)</td>
<td>IFAT (1 : 16)</td>
<td>Arias et al., 1996</td>
<td></td>
</tr>
<tr>
<td>Cuba Children &amp; adults</td>
<td>30 (2632/8863)</td>
<td>ELISA</td>
<td>Machin Sanchez et al., 1993</td>
<td></td>
</tr>
<tr>
<td>Cuba (Havana Province) Pregnant women</td>
<td>71 (257/362)</td>
<td>Micro-ELISA</td>
<td>Martinez Sanchez et al., 1994</td>
<td></td>
</tr>
<tr>
<td>Cuba (Havana) Pregnant women</td>
<td>71 (3931/5537)</td>
<td>ELISA</td>
<td>Gonzalez-Morales et al., 1995</td>
<td></td>
</tr>
<tr>
<td>Grenada, West Indies Pregnant women</td>
<td>57 (304/534)</td>
<td>ELISA</td>
<td>Asthana et al., 2006</td>
<td></td>
</tr>
<tr>
<td>Guatemala Children 2 month-2 year</td>
<td>12 (66/532)</td>
<td>EIA (commercial kit)</td>
<td>Jones et al., 2005</td>
<td></td>
</tr>
<tr>
<td>Children 3–10 year</td>
<td>38 (189/500)</td>
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</tbody>
</table>
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 paten 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>
<thead>
<tr>
<th>Location</th>
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<th>% (No. positive/ No. tested)</th>
<th>Method* (cutoff for a positive test)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jamaica</td>
<td>Pregnant women</td>
<td>57 (911/1604)</td>
<td>ELISA (commercial kit)</td>
<td>Prabhakar et al., 1991</td>
</tr>
<tr>
<td>Trinidad</td>
<td>Pregnant women</td>
<td>43 (129/300)</td>
<td>ELISA</td>
<td>Orrett, 1993</td>
</tr>
<tr>
<td>Venezuela (Amazonas St.)</td>
<td>Guajibo Amerindians</td>
<td>88 (106/121)</td>
<td>ELISA (OD &gt; 0.3)</td>
<td>de la Rosa et al., 1999</td>
</tr>
<tr>
<td>Venezuela (Zulia State)</td>
<td>Bazi Amerindians</td>
<td>49 (221/447)</td>
<td>IHAT (1 : 64)</td>
<td>Chacin-Bonilla et al., 2001</td>
</tr>
<tr>
<td>Venezuela (Sierra de Perija)</td>
<td>Yucpa Amerindians</td>
<td>63 (59/94)</td>
<td>IHAT (1 : 64)</td>
<td>Diaz-Suarez et al., 2003</td>
</tr>
<tr>
<td><strong>North America</strong></td>
<td></td>
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<tr>
<td>Canada</td>
<td>Cree hunters</td>
<td>10 (5/50)</td>
<td>ELISA (≥2 IU/ml)</td>
<td>Levesque et al., 2007</td>
</tr>
<tr>
<td>Canada (Ontario)</td>
<td>Veterinary staff</td>
<td>14 (20/141)</td>
<td>ELISA</td>
<td>Shubaiber et al., 2003</td>
</tr>
<tr>
<td>Canada (Halifax)</td>
<td>Children 7 months-17 years</td>
<td>3 (33/998)</td>
<td>IHAT (1 : 64)</td>
<td>Pereira et al., 1992</td>
</tr>
<tr>
<td>Canada (Montreal)</td>
<td>Caucasian adults</td>
<td>30 (56/189)</td>
<td>ELISA (0.25 above mean of negative sera)</td>
<td>Frappier-Davignon et al., 1990</td>
</tr>
<tr>
<td>Mexico</td>
<td>Women with high-risk pregnancies</td>
<td>35 (122/350)</td>
<td>ELISA</td>
<td>Galván Ramirez et al., 1995</td>
</tr>
<tr>
<td>Mexico</td>
<td>Cat owners</td>
<td>64 (38/59)</td>
<td>ELISA (OD &gt; 0.2)</td>
<td>Galván Ramirez et al., 1999</td>
</tr>
<tr>
<td>Mexico (Durango)</td>
<td>Pregnant women</td>
<td>6 (21/343)</td>
<td>EIA (commercial kit)</td>
<td>Alvarado-Esquivel et al., 2006a</td>
</tr>
<tr>
<td>Mexico (Durango)</td>
<td>Psychiatric patients</td>
<td>18 (25/137)</td>
<td>EIA (commercial kit)</td>
<td>Alvarado-Esquivel et al., 2006b</td>
</tr>
<tr>
<td>Mexico (Durango)</td>
<td>Blood donors</td>
<td>7 (32/432)</td>
<td>EIA (commercial kit)</td>
<td>Alvarado-Esquivel et al., 2007</td>
</tr>
<tr>
<td>Mexico (Durango)</td>
<td>Waste pickers Waste workers</td>
<td>21 (19/90)</td>
<td>EIA (commercial kit)</td>
<td>Alvarado-Esquivel et al., 2008a</td>
</tr>
<tr>
<td>Mexico (Durango)</td>
<td>Adult rural residents</td>
<td>24 (110/463)</td>
<td>EIA (commercial kit)</td>
<td>Alvarado-Esquivel et al., 2008b</td>
</tr>
<tr>
<td>USA (Illinois)</td>
<td>Swine farm workers</td>
<td>31 (54/174)</td>
<td>MAT (1 : 25)</td>
<td>Weigel et al., 1999</td>
</tr>
<tr>
<td>USA (Maryland)</td>
<td>7th-day adventists &amp; blood donors</td>
<td>31 (78/251)</td>
<td>MAT (1 : 32)</td>
<td>Roghmann et al., 1999</td>
</tr>
<tr>
<td>USA</td>
<td>NHANES, 1988–94</td>
<td>23 (3974/17 658)</td>
<td>EIA (&gt;6 IU)</td>
<td>Jones et al., 2001b</td>
</tr>
<tr>
<td>USA (Illinois)</td>
<td>HIV+HIV- women with high STD risk</td>
<td>15 (380/2525)</td>
<td>EIA (commercial kit)</td>
<td>Falusi et al., 2002</td>
</tr>
<tr>
<td>USA</td>
<td>NHANES, 1999–2000</td>
<td>16 (669/4234)</td>
<td>EIA (≥10 IU)</td>
<td>Jones et al., 2003b</td>
</tr>
<tr>
<td>USA</td>
<td>NHANES, 1988–94, participants with ethnicity data</td>
<td>25 (3787/14909)</td>
<td>EIA (commercial kit)</td>
<td>Kruzon-Moran and McQuillan, 2005</td>
</tr>
<tr>
<td>USA</td>
<td>NHANES, 1999–2004, 6–49 year</td>
<td>11 (1712/15960)</td>
<td>EIA (commercial kit)</td>
<td>Jones et al., 2007</td>
</tr>
</tbody>
</table>

*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.

1Dat calculated from number and percentage with positive results.

2Age-adjusted.

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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

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</thead>
<tbody>
<tr>
<td>Central/South America/Caribbean</td>
<td>Brazil (Jaguapita) Rural cats</td>
<td>73 (119/163)</td>
<td>IFAT (1 : 16)</td>
<td>Garcia et al., 1999</td>
</tr>
<tr>
<td></td>
<td>Brazil (Sao Paulo) Patients at vet teaching hospital</td>
<td>18 (44/248)</td>
<td>IFAT (1 : 16)</td>
<td>Lucas et al., 1999</td>
</tr>
<tr>
<td></td>
<td>Brazil (Guarulhos, Sao Paulo) Strays (n = 470), breeder (n = 32)</td>
<td>26 (132/502)</td>
<td>MAT (1 : 20 or 1 : 25)</td>
<td>Silva et al., 2002</td>
</tr>
<tr>
<td></td>
<td>Brazil (Parana) Free-roaming cats</td>
<td>84 (49/58)</td>
<td>MAT (1 : 20)</td>
<td>Dubey et al., 2004</td>
</tr>
<tr>
<td></td>
<td>Brazil (Sao Paulo metro area) Strays housed at IUPA</td>
<td>40 (40/100)</td>
<td>ELISA (OD &gt; 2.0)</td>
<td>Meireles et al., 2004</td>
</tr>
<tr>
<td></td>
<td>Brazil (Sao Paulo State) Stray cats</td>
<td>35 (84/237)</td>
<td>MAT (1 : 25)</td>
<td>Pena et al., 2006</td>
</tr>
<tr>
<td></td>
<td>Brazil (Rondonia State) Free-roaming cats</td>
<td>87 (55/63)</td>
<td>MAT (1 : 25) &amp; IFAT (1 : 25)</td>
<td>Cavalcante et al., 2006b</td>
</tr>
<tr>
<td></td>
<td>Brazil (Sao Paulo) Relinquished to Center of Zoonosis Control</td>
<td>25 (100/400)</td>
<td>IFAT (1 : 64)</td>
<td>Bresciai et al., 2007</td>
</tr>
<tr>
<td></td>
<td>Colombia (Bogota &amp; Armenia) Unwanted cats</td>
<td>32 (52/170)</td>
<td>MAT (1 : 20)</td>
<td>Dubey et al., 2006</td>
</tr>
<tr>
<td></td>
<td>Grenada, West Indies Domestic cats</td>
<td>35 (14/40)</td>
<td>MAT (1 : 25)</td>
<td>Asthana et al., 2006</td>
</tr>
<tr>
<td></td>
<td>Panama (Panama City) Free-roaming cats</td>
<td>46 (110/241)</td>
<td>DAT (not reported)</td>
<td>Frenkel et al., 1995</td>
</tr>
<tr>
<td></td>
<td>Puerto Rico (Monia Island) Feral cats</td>
<td>84 (16/19)</td>
<td>MAT (1 : 10)</td>
<td>Dubey et al., 2007</td>
</tr>
<tr>
<td></td>
<td>St Kitts, West Indies Domestic cats</td>
<td>85 (90/106)</td>
<td>MAT (1 : 20)</td>
<td>Moura et al., 2007</td>
</tr>
<tr>
<td>North America</td>
<td>Canada (Victoria) Cats at veterinary clinics</td>
<td>22 (16/73)</td>
<td>MAT (1 : 25)</td>
<td>Aramin et al., 1999</td>
</tr>
<tr>
<td></td>
<td>Canada (British Columbia) Owned cats</td>
<td>23 (50/221)</td>
<td>IFAT (not reported)</td>
<td>Aramin et al., 1999</td>
</tr>
<tr>
<td></td>
<td>Mexico (Mexico City) Feral cats</td>
<td>67 (4/6)</td>
<td>CF (not reported)</td>
<td>Suzan and Ceballos, 2005</td>
</tr>
<tr>
<td></td>
<td>USA (Georgia, Florida, Ohio) Cats with uveitis</td>
<td>41 (51/124)</td>
<td>ELISA(^1) IgG (1 : 64)</td>
<td>Lappin et al., 1992</td>
</tr>
<tr>
<td></td>
<td>USA (Iowa) Swine farm cats</td>
<td>42 (31/74)</td>
<td>MAT (1 : 32)</td>
<td>Smith et al., 1992</td>
</tr>
<tr>
<td></td>
<td>USA (Illinois) Swine farm cats</td>
<td>76 (223/295)</td>
<td>MAT (1 : 25)</td>
<td>Dubey et al., 1995</td>
</tr>
<tr>
<td></td>
<td>USA (Iowa) Free-ranging cats</td>
<td>80 (16/20)</td>
<td>MAT (1 : 32)</td>
<td>Hill et al., 1998</td>
</tr>
<tr>
<td></td>
<td>USA (Rhode Island) Veterinary clinics (n = 116), animal shelters (n = 84)</td>
<td>42 (64/200)</td>
<td>MAT (1 : 25)</td>
<td>DeFeo et al., 2002</td>
</tr>
<tr>
<td></td>
<td>USA (Ohio) Outdoor/feral (n = 252), unknown (n = 23)</td>
<td>48 (133/275)</td>
<td>MAT (1 : 25)</td>
<td>Dubey et al., 2002</td>
</tr>
<tr>
<td></td>
<td>USA (Florida) Feral cats at spay/neuter clinic</td>
<td>9 (49/53)</td>
<td>ELISA(^1) IgG (1 : 64)</td>
<td>Luria et al., 2004</td>
</tr>
<tr>
<td></td>
<td>USA Clinically ill cats</td>
<td>29 (3619/12 628)</td>
<td>ELISA(^1) IgG (1 : 64)</td>
<td>Vollaire et al., 2005</td>
</tr>
<tr>
<td></td>
<td>USA Feral cats</td>
<td>34 (31/92)</td>
<td>IFAT (1 : 640)</td>
<td>Clifford et al., 2006</td>
</tr>
<tr>
<td></td>
<td>USA (Channel Islds, California) Owned (n = 43), stray at shelter (n = 80)</td>
<td>11 (13/123)</td>
<td>ELISA(^1) IgG (1 : 64)</td>
<td>Dabritz et al., 2007b</td>
</tr>
<tr>
<td></td>
<td>USA (San Luis Obispo County, California) Owned</td>
<td>20 (25/122)</td>
<td>MAT (1 : 25)</td>
<td>Dubey et al., 2008</td>
</tr>
</tbody>
</table>

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

\(^1\)To facilitate comparison, only IgG-positive results are summarized.
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 stray reports 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 Toxoplasma 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 Ivaí, 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 Erechim, 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
Toxoplasma 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/m²) would be >10 times lower than that of Sousa et al. (1988), if the latter estimate of 193–774 oocysts/m², 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/m² 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/m² 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, Franc e 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; Dumètère 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). Dumètère 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 (Dumètère 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

<table>
<thead>
<tr>
<th>Organization</th>
<th>Location</th>
<th>Website</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVMA</td>
<td>Schaumburg, IL</td>
<td><a href="http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure.pdf">http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure.pdf</a> (English)</td>
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<tr>
<td></td>
<td></td>
<td><a href="http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure_spanish.pdf">http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure_spanish.pdf</a> (Spanish)</td>
</tr>
<tr>
<td>Control and Prevention</td>
<td></td>
<td><a href="http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure.pdf">http://www.avma.org/animal_health/brochures/toxoplasmosis/toxoplasmosis_brochure.pdf</a> (English)</td>
</tr>
<tr>
<td>University of California</td>
<td>Davis, CA</td>
<td><a href="http://www.seaotterresearch.org/Facts_about_Toxo_and_Cat_poop.pdf">http://www.seaotterresearch.org/Facts_about_Toxo_and_Cat_poop.pdf</a> (English)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><a href="http://www.seaotterresearch.org/Toxo_gondii_lo_que.todos.debemos.saber.pdf">http://www.seaotterresearch.org/Toxo_gondii_lo_que.todos.debemos.saber.pdf</a> (Spanish)</td>
</tr>
</tbody>
</table>
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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