

## RISK FACTORS FOR *TOXOPLASMA GONDII* INFECTION IN WILD RODENTS FROM CENTRAL COASTAL CALIFORNIA AND A REVIEW OF *T. GONDII* PREVALENCE IN RODENTS

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**ABSTRACT:** Sera from 523 wild rodents were tested for *Toxoplasma gondii* antibodies using either an indirect fluorescent antibody test (IFAT) (rats and mice, with titer  $\geq 80$  considered positive) or a latex agglutination test (LAT) (voles, squirrels, and pocket mice, with titer  $\geq 32$  considered positive). Seventeen percent (88/523) of the rodents, including 26% (85/328) of the *Peromyscus* sp. and 8% (3/37) of *Spermophilus beecheyi*, were seropositive. Fourteen percent (23/161) of rodents captured in trap sites next to Morro Bay (California) and 15% (16/109) of rodents from sites adjacent to riparian habitats had antibodies to *T. gondii*, compared to 19% (49/253) of rodents captured in habitats not associated with water; this difference was not statistically significant ( $P = 0.32$ ). Significantly fewer rodents were captured  $< 200$  m from residential housing compared to locations further away (11% vs. 30%, respectively). Factors associated with an increased risk for *T. gondii* seropositivity in rodents were capture location  $\geq 200$  m from residential housing and adult age.

The protozoan parasite *T. gondii* infects most species of warm-blooded vertebrates, including rodents. Like humans, rodents serve as intermediate hosts for *T. gondii*. Parasites form a latent cyst stage in the tissues of intermediate hosts, notably in the striated muscle and brain. The definitive hosts for *T. gondii* are domestic and feral cats (*Felis catus*) and other members of the Felidae (Dubey et al., 1970; Aramini et al., 1999; Kenny et al., 2002). Cats also serve as intermediate hosts. *Toxoplasma gondii* undergoes sexual reproduction in the feline intestine, resulting in the production of millions of environmentally resistant oocysts that are shed in cat feces (Dubey et al., 1970). Rodents become infected after ingesting soil, vegetation, or water contaminated with *T. gondii* oocysts. *Toxoplasma gondii* can be transmitted horizontally between warm-blooded animals, including humans, when they consume the raw or undercooked flesh of intermediate or definitive hosts (Tenter et al., 2000). Vertical transmission also occurs and is of particular concern for humans, because infection in utero can cause abortion or congenital defects in the fetus (Jones et al., 2003). Both modes of transmission appear to be important to ensure the survival of *T. gondii* in nature.

Small rodents play an important role in the life cycle of *T. gondii*, because they are believed to represent the main source of infection for domestic and feral cats. Studies of cat predation on wildlife suggest that rodents comprise about two-thirds of the prey consumed, although this may vary according to season, rodent abundance, and the availability of other prey (McMurry and Sperry, 1941; Hubbs, 1951; Eberhard, 1954; Molsher et al., 1999). On islands where cats were not present, *T. gondii* in intermediate hosts was rare or absent (Wallace, 1969; Garcelon

et al., 1992; Dubey, Rollor et al., 1997). Conversely, on U.S. swine farms where cats were abundant, the prevalence of *T. gondii* infection in mice was significantly associated with a higher mean density of cats on the premises (Smith et al., 1992; Dubey, Weigel et al., 1995).

There is a paucity of data on *T. gondii* infection in small mammals native to California. The most abundant rodent species in coastal California ecosystems south of San Francisco Bay are *Peromyscus* spp., including *P. maniculatus* (deer mice), *Microtus* spp. (voles), and *Perognathus* spp. (pocket mice) (Jameson and Peeters, 1988). It was not known which species may be important in maintaining *T. gondii* infection in near shore-dwelling wild carnivores and domestic cats, so a variety of small mammals was included in the sampling protocol. In addition to constituting an important prey item for domestic cats, rodents were also suitable for study because they are easily captured, and tissues harvested at necropsy can later be tested for the presence of parasites by immunohistochemistry or polymerase chain reaction (PCR).

The Morro Bay area of central coastal California was selected for study because of the high proportion of southern sea otters (*Enhydra lutris nereis*) with evidence of *T. gondii* infection (Miller et al., 2002; Conrad et al., 2005) off the adjacent coastline. In an updated analysis of the risk for *T. gondii* exposure in 562 live or dead otters (Conrad et al., 2005), otters sampled between San Simeon and Morro Bay were 5 times more likely to be infected with *T. gondii* than were otters living along the more remote and rocky Big Sur coastline. An epidemiologic investigation of *T. gondii* infection prevalence in owned and feral cats was being concurrently conducted in the Morro Bay area (Dabritz et al., 2007). The objectives of this study were to determine demographic and habitat-related risk factors for *T. gondii* seropositivity in wild rodents and to obtain tissue samples for future *T. gondii* genotyping. We hypothesized that there would be a greater risk for *T. gondii* seropositivity in wild rodents captured near residential housing, because of the association with owned domestic cats. A previous study had shown that *T. gondii* seropositivity in southern sea otters was associated with high freshwater outflow (Miller et al., 2002), so we also hypothesized that rodents captured near bodies of water (Morro Bay) or water channels would be more likely to be serologically positive.

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## MATERIALS AND METHODS

### Rodent trapping

Trapping was conducted at 18 different locations in the Morro Bay area between July 2004 and February 2006. Study sites were selected to be either <200 m from residential housing or feral cat colonies, where cat densities were expected to be high ( $n = 13$ ), or situated on public lands  $\geq 200$  m from private residences ( $n = 5$ ). Sites were classified as being adjacent to Morro Bay ( $n = 3$ ), adjacent to riparian habitats ( $n = 6$ ), or neither ( $n = 9$ ). Trapping occurred twice in each wet (December–May) and dry (June–November) season. Each site was trapped for a minimum of 2 nights. In the first trapping session of 2004 (July–August), only mice were sampled; other species were released. Subsequently, sampled species were expanded to include rats and voles. Trapping of *S. beecheyi* (California ground squirrels) began in 2005 and was conducted at 4 sites. At 2 of these sites, ground squirrels were the predominant species trapped. Ten to 60 traps were set per site, depending on the space available. For nocturnal rodents, kangaroo rat–sized folding live traps (HB Sherman, Tallahassee, Florida) were set in a transect layout 5–15 m apart within 2 hr of sunset and baited with hamster/rat feed (Radco, San Jose, California). If temperatures were expected to fall below 12 C, traps were bedded and over-baited. Traps were collected within 3 hr of sunrise. At 4 sites, Tomahawk 48 × 15 × 15 cm live traps (Tomahawk Live Trap, Tomahawk, Wisconsin) were set during daylight hours and monitored for 2–3 hr for the presence of ground squirrels. Rodents were identified to species using a key (Jameson and Peeters, 1988). The location of each captured animal was recorded on a handheld Garmin GPS locator (Olathe, Kansas). All animals were humanely handled in accordance with an animal use protocol approved by the Institutional Animal Care and Use Committee at the University of California (UC) Davis, which is accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International. In 2004 and 2005, captured rodents were killed using CO<sub>2</sub> asphyxiation and immediately bled by intracardiac puncture. In 2006 only, animals trapped on nights 1 and 2 were bled via the retro-orbital sinus, ear-tagged, and released; rodents captured on the third night were killed and bled via intracardiac puncture. After collection, whole blood was allowed to clot and was centrifuged, and serum was aliquoted into separate vials. Serum was either frozen and transported to UC Davis on dry ice for testing or refrigerated until transport and shipped in coolers at 4 C. Serum was frozen at –20 C until tested.

### IFAT for *Peromyscus* spp., *Mus musculus*, *Reithrodontomys megalotis*, *Neotoma fuscipes*, and *Rattus* spp.

Sera were diluted in phosphate-buffered saline (PBS), pH 7.4 (Diamedix, Miami, Florida) in 2-fold serial dilutions from 1:40 to 1:160. Titer was characterized as the inverse of the dilution of the last well, with distinctive fluorescence outlining formalin-fixed tachyzoites. Ten  $\mu$ l of serum was incubated at 37 C for 1 hr on 12-well slides (Erie Scientific, St. Louis, Missouri) coated with formalin-fixed *T. gondii* tachyzoites derived from tissue culture, as previously described (Miller et al., 2001). Slides were washed 3 times for 5 min each in PBS, and 10  $\mu$ l of secondary fluorescein isothiocyanate (FITC)–conjugated antibody was added to detect the presence of antibodies bound to *T. gondii* tachyzoites. Secondary conjugates were applied as follows: for *Peromyscus* spp. (*P. californicus*, *P. boylii*, *P. maniculatus*, and *P. truei*; California or parasitic mouse, brush mouse, deer mouse, and Pinon mouse, respectively), goat anti-*P. leucopus* immunoglobulin G (IgG) with FITC (Kirkegaard & Perry Laboratories Inc., Gaithersburg, Maryland) diluted 1:100; for *N. fuscipes* (dusky-footed wood rat) and *Rattus* spp. (*R. norvegicus* and *R. rattus*, Norway and roof or black rat, respectively), goat anti-rat IgG with Alexa Fluor 488 (Invitrogen Molecular Probes, Carlsbad, California) diluted 1:200; and for *M. musculus* (house mouse) and *Rattus megalotis* (western harvest mouse), goat anti-mouse IgG with FITC (Jackson ImmunoResearch Laboratories, West Grove, Pennsylvania) diluted 1:100. After addition of the appropriate secondary antibody, slides were incubated for 1 hr at 37 C, were washed 3 times in PBS, and 10  $\mu$ l of buffered pH 8.5 glycerol was added to each well; the slides were examined at  $\times 200$  on an immunofluorescent microscope (Karl Zeiss Optical, Inc., Chester, Virginia). Titers  $\geq 80$  were considered seropositive (Tenter, 1987). Positive controls consisted of sera from experimentally infected *P. californicus* mice or Wistar rats experimentally infected with *T. gondii*. Negative controls were sera col-

lected from mice and rats before experimentally induced *T. gondii* infection, or from wild-caught *Peromyscus* spp. or *N. fuscipes* that had previously tested seronegative for *T. gondii*.

### LAT for *Spermophilus beecheyi*, *Microtus californicus* (California meadow vole), and *Perognathus californicus* (California pocket mouse)

Sera were tested with a commercial kit (Toxotest, Tanabe USA Inc., San Diego, California) according to the manufacturer's instructions, with the following modifications to reduce the amount of serum needed for the test. A 1:8 dilution of serum to the manufacturer's LAT buffer was made in a total volume of 50  $\mu$ l. Two-fold serial dilutions up to 1:64 were aliquoted in a microtiter plate, using 25  $\mu$ l of LAT buffer from the first well, and 25  $\mu$ l of the diluted serum was discarded from the last well. Twenty-five microliters of the manufacturer's LAT bead solution was added to each well and incubated in a covered container for 12 hr at room temperature. Titers  $\geq 32$  were considered positive (Tsubota et al., 1977). Positive control serum was supplied with the kit, and the negative control was LAT buffer in place of serum.

### Data analysis

Wild rodent trapping success was compared for sites near residences to sites with low human impact using a chi-square test. Risk factors for *T. gondii* infection in wild-caught rodents were assessed using a mixed logistic regression model with the %glimmix macro in SAS version 9.1 (Cary, North Carolina). Rodents were considered infected if they had an IFAT titer  $\geq 80$  or LAT titer  $\geq 32$ , and were otherwise considered uninfected. Fixed effects considered in the model were rodent age class (adult or juvenile), sex, the site characteristics defined above, and season (wet or dry). Capture location was modeled as a random effect. The variables were first assessed singly, and those significant at  $P = 0.20$  were selected for inclusion in a multivariable model. Different models were compared using the likelihood ratio test. Fit of the selected model was assessed by examining the deviance residuals and Hosmer–Lemeshow goodness-of-fit statistic. Statistical tests were considered significant if the  $P$  value was  $< 0.05$ .

## RESULTS

In total, 623 wild rodents were trapped in 3,682 trap-nights (including rodents released without sampling and those captured more than once in 2006), for an overall trap success rate of 16.9%. Of these, 523 individuals were serologically tested for *T. gondii* infection and 479 were necropsied. Eighty-eight

TABLE I. *Toxoplasma gondii* seroprevalence by species for rodents trapped in the Morro Bay area of California.

Species	No. tested	No. seropositive* (%)
<i>Peromyscus maniculatus</i>	74	25 (34)
<i>Peromyscus boylii</i>	29	9 (31)
<i>Peromyscus californicus</i>	214	50 (23)
<i>Peromyscus truei</i>	11	1 (9)
<i>Spermophilus beecheyi</i>	37	3 (8)
<i>Mus musculus</i>	15	0 (0)
<i>Microtus californicus</i>	25	0 (0)
<i>Neotoma fuscipes</i>	50	0 (0)
<i>Perognathus californicus</i>	14	0 (0)
<i>Reithrodontomys megalotis</i>	44	0 (0)
<i>Rattus rattus</i>	10	0 (0)
Total	523	88 (17)

\* For *Peromyscus* spp., *Rattus* spp., *N. fuscipes*, *R. megalotis*, and *M. musculus*, a positive test was defined as an IFAT titer  $\geq 80$ ; for voles, squirrels, and *P. californicus*, a positive test was defined as an LAT titer  $\geq 32$ .

TABLE II. Univariable logistic regression analysis of risk factors for *T. gondii* seropositivity in rodents from the Morro Bay area of California.

Risk factor	Description	Odds ratio	95% CI	<i>P</i> value
Proximity to residential housing	Far	1.00		
	Near	0.63	0.39–1.01	0.055
Sex	Male	1.00		
	Female	1.06	0.67–1.67	0.82
Age class	Juvenile	1.00		
	Adult	3.04	1.07–8.61	0.037
Season	Dry	1.00		
	Wet	0.82	0.52–1.30	0.39
Site proximity to water	Not	1.00		
	Next to bay	0.68	0.39–1.16	0.16
	Riparian	0.72	0.39–1.33	0.30

(17%) of the 523 rodents with serum available for testing had antibodies to *T. gondii*. The proportion of rodents seropositive by species is shown in Table I. *Peromyscus californicus* comprised the majority (40%) of the species captured. For the 3 habitat classifications, 14% (23/161) of rodents from sites adjacent to Morro Bay and 15% (16/109) of rodents from riparian sites tested seropositive, compared to 19% (49/253) of rodents captured at all other locations. There was no significant difference in the proportion of seropositive rodents amongst habitat types ( $P = 0.32$ ). At 3 sites adjacent to residences, no rodents were captured. Sites located near residential housing had significantly lower trap success (11% vs. 30%) compared with sites remote from residential housing ( $P < 0.001$ ).

The univariable analysis of risk factors for *T. gondii* seropositivity in wild rodents is shown in Table II. Only a multiple logistic regression model incorporating main effects was employed, because the random effect could not be estimated. Two risk factors were significant in the multiple logistic regression analysis (Table III): adult age and capture in locations  $\geq 200$  m from residential housing (sites assumed to have low impact due to domestic cats). In this model, the odds ratio for being seropositive was 3.10 (95% CI = 1.09–8.83,  $P = 0.034$ ) for adults compared to juveniles, and 0.62 (95% CI = 0.39–0.99,  $P = 0.049$ ) for capture in sites  $< 200$  m from residential housing, compared to sites  $\geq 200$  m away from residences. Check of model fit using the Hosmer–Lemeshow goodness-of-fit test ( $P = 0.93$ ) and deviance residuals indicated that the model fit was adequate.

## DISCUSSION

Seventeen percent of the wild rodents had antibodies to *T. gondii*. There was considerable variability in the proportion of

rodents infected among different species, as has been reported in other studies worldwide (see summary in Table IV). The prevalence of 26% in *Peromyscus* spp. was higher than in any previous U.S. reports for *Peromyscus* spp. since 1985 (range 0–15.5%, typically  $< 7\%$ , under subheading *Peromyscus* spp., Table IV). Detecting *T. gondii* antibodies only in *Peromyscus* spp. and California ground squirrels could be attributable to variable test sensitivity or the duration of immunity to *T. gondii*. The LAT used to test pocket mouse, ground squirrel, and western harvest mouse sera has not been validated in these species and has been shown to be less sensitive for detecting *T. gondii* antibodies in pigs and cats (Dubey and Thulliez, 1989; Lappin and Powell, 1991; Dubey, Thulliez et al., 1995). Therefore, seroprevalence in these species could have been underestimated. For practical purposes, the LAT was the only test available, since secondary antibodies for these rodent species were not commercially available. Furthermore, duration of immunity may vary, depending on the species. For example, in congenitally infected rats, antibody titers wane over time and may not be detectable by 87 days postinoculation (Dubey, Shen et al., 1997).

In the present study, the odds of *T. gondii* seropositivity were about 3-fold greater for adults compared to juveniles, suggesting that the risk for acquiring toxoplasmosis is related to a higher probability for being exposed to oocysts in the environment over the animal's lifetime. A similar relationship between age and infection has been detected in sea otters (Miller et al., 2002), as well as in humans (Jones et al., 2001). The greater odds for seropositivity in adult mice in the present study may be related to the sensitivity of the different methodologies for detection of *T. gondii* infection, since congenitally infected mice can test seronegative while harboring parasites in their

TABLE III. Multivariable logistic regression model of risk factors associated with *T. gondii* seropositivity in rodents from the Morro Bay area of California.

Risk factor	Description	Parameter estimate (SE)	Adjusted odds		
			ratio	95% CI	<i>P</i> value
Proximity to residential housing	Far		1.00		
	Near	−0.4749 (0.2417)	0.62	0.39–0.99	0.049
Age class	Juvenile		1.00		
	Adult	1.1322 (0.5335)	3.10	1.09–8.83	0.034
Intercept		2.4353 (0.5260)			

TABLE IV. Prevalence of *T. gondii* in wild-caught rodents worldwide reported from 1985 to 2006.

Location	No. positive/no. tested	% Positive	Test (cutoff)*	Reference
<i>Peromyscus</i> spp.				
Iowa pig farms, U.S.	0/21	0.0	MAT (32)	Smith et al., 1992
Kansas, U.S.	5/171	2.9	MAT (25)	Brillhart et al., 1994
Missouri, U.S.	1/15	6.7	DT (8)	Smith and Frenkel, 1995
Illinois, U.S.	3/61	4.9	MAT (25)	Dubey, Weigel et al., 1995
Humpback Reservoir, British Columbia, Canada	5/80 (>3 km from residence) 11/71 (<3 km from residences)	6.3 15.5	MAT (25)	Aramini et al., 1999
Washington Co., Rhode Island, U.S.	4/391 4/53 at a single site	1.0 7.5	MAT (25)	DeFeo et al., 2002
New England, U.S.	2/33	6.1	MAT (10)†	Lehmann et al., 2003
House mice ( <i>M. musculus</i> )				
Iowa pig farms, U.S.	2/588	0.3	MAT (32)	Smith et al., 1992
Missouri, U.S.	0/17	0.0	DT (8)	Smith and Frenkel, 1995
Kansas, U.S.	0/11	0.0	MAT (25)	Brillhart et al., 1994
Illinois pig farms, U.S.	26/1,243	2.1	MAT (25)	Dubey, Weigel et al., 1995
Panama City, Costa Rica	2/571	0.04	MAT (NS)‡	Frenkel et al., 1995
Czech Republic	9/934	1.0	DT (4)	Hejlicek et al., 1997
Illinois pig farms, U.S.	7/465§ 6/390	1.5 1.5	MAT (25) Bioassay	Mateus-Pinilla et al., 1999
New England, U.S.	0/2	0.0	MAT (10)	Lehmann et al., 2003
Manchester, U.K.	118/200	59.0	SAG1 PCR	Marshall et al., 2004
Manchester, U.K.	53/100	53.0	SAG1 PCR	Hughes et al., 2006
Rats				
India (northern)	Bandicoot rat 18/186	9.7	IHAT (64)	Chhabra et al., 1985
Baltimore, Maryland, U.S.	<i>Rattus norvegicus</i> 54/109	49.5	IFAT (32)	Childs and Seegar, 1986
Strathblane, Scotland	<i>R. norvegicus</i> 5/65	7.7	DT (10)	Jackson et al., 1986
Kobe Zoo, Japan	<i>R. norvegicus</i> , <i>R. rattus</i> 0/55	0.0	LAT (64)	Murata, 1989
Guangdong, P. Rep. China	Rats unspecified spp. 9/955	1.0	IHAT (64)	Lin et al., 1990
Mantova, Italy	<i>R. norvegicus</i> 14/20	70.0	IFAT (40)	Genchi et al., 1991
Iowa pig farms, U.S.	<i>R. norvegicus</i> 0/9	0.0	MAT (32)	Smith et al., 1992
Kansas, U.S.	<i>Neotoma floridana</i> 2/28 <i>Sigmodon hispidus</i> 0/62	7.0 0.0	MAT (25)	Brillhart et al., 1994
U.K. farms	<i>R. norvegicus</i> 84/235	35.7	LAT (10)	Webster, 1994
Illinois pig farms, U.S.	<i>R. norvegicus</i> 6/95	6.3	MAT (25)	Dubey, Weigel et al., 1995
Missouri, U.S.	<i>S. hispidus</i> , <i>R. norvegicus</i> 0/5	0.0	DT (8)	Smith and Frenkel, 1995
Panama City, Costa Rica	<i>R. norvegicus</i> 52/226	23.0	MAT (NS)‡	Frenkel et al., 1995
Czech Republic	<i>R. norvegicus</i> 1/84	1.0	DT (4)	Hejlicek et al., 1997
Taiwan	Rat 1/13	7.7	LAT (32)	Fan et al., 1998
New England, U.S.	<i>R. norvegicus</i> 1/2	50.0	MAT (10)†	Lehmann et al., 2003
Manchester, U.K.	<i>R. norvegicus</i> 19/45	42.2	SAG1 PCR	Hughes et al., 2006
Voles				
Strathblane, Scotland	<i>Clethrionomys glareolus</i> 6/68	8.8	DT (10)	Jackson et al., 1986
Kansas, U.S.	<i>Microtus ochrogaster</i> 1/2	50.0	MAT (25)	Brillhart et al., 1994
Czech Republic	<i>Clethrionomys glareolus</i> 3/304	1.0	DT (4)	Hejlicek et al., 1997
Czech Republic	<i>Arvicola terrestris</i> 0/10	0.0	DT (4)	Hejlicek et al., 1997
Czech Republic	<i>Microtus</i> spp. 15/1,643	0.9	DT (4)	Hejlicek et al., 1997
New England, U.S.	<i>Microtus pennsylvanicus</i> 1/8	12.5	MAT (25)†	Lehmann et al., 2003
Hunan Province, P. Rep. China	<i>Microtus fortis</i> 36/124	29.0	MAT (20)	Zhang et al., 2004
Miscellaneous spp.				
Strathblane, Scotland	<i>Apodemus sylvaticus</i> 10/98	10.2	DT (10)	Jackson et al., 1986
Kansas, U.S.	<i>Dipodomys ordii</i> (kangaroo rat) 0/15	0.0	MAT (25)	Brillhart et al., 1994
Kansas, U.S.	<i>Reithrodontomys megalotis</i> (harvest mouse) 1/2	50.0	MAT (25)	Brillhart et al., 1994
Kansas, U.S.	<i>Ondatra zibethicus</i> (muskrat) 7/42	17.0	DT (8)	Smith and Frenkel, 1995
Czech Republic	<i>Apodemus</i> spp. 24/2,165	1.1	DT (4)	Hejlicek et al., 1997
Czech Republic	<i>O. zibethicus</i> 105/437	24.0	DT (4)	Hejlicek et al., 1997
Korea	<i>Apodemus agrarius</i> 15/1,008	1.5	ELISA (0.18)	Jeon and Yong, 2000

TABLE IV. Continued.

Location	No. positive/no. tested	% Positive	Test (cutoff)*	Reference
Washington Co., Rhode Island, U.S.	Wild rodents, miscellaneous spp. 6/758#	0.8	MAT (25)	DeFeo et al., 2002
New England, U.S.	<i>Blarina brevicauda</i> (shrew) 0/2	0.0	MAT (10)†	Lehmann et al., 2003
New England, U.S.	<i>Zapus hudsonius</i> (jumping mouse) 0/1	0.0	MAT (10)†	Lehmann et al., 2003
Sao Paulo, Brazil	<i>Hydrochoeris hydrochoeris</i> (capybara) 63/149	42.3	MAT (25)‡	Canon-Franco et al., 2003
French Guiana	<i>Myoprocta acouchy</i> (acouchy) 1/26	4.0	MAT (40)	De Thoisy et al., 2003
French Guiana	<i>Dasyprocta agouti</i> (gouti) 8/45	18.0	MAT (40)	De Thoisy et al., 2003
French Guiana	<i>Coendou prehensilis</i> (porcupine) 0/19	0.0	MAT (40)	De Thoisy et al., 2003
French Guiana	<i>Agouti paca</i> (paca) 22/37	60.0	MAT (40)	De Thoisy et al., 2003
Squirrels				
Kansas, U.S.	<i>Sciurus</i> spp.** 2/11	18.0	DT (8)	Smith and Frenkel, 1995
Czech Republic	<i>Sciurus vulgaris</i> 0/3	0.0	DT (4)	Hejlíček et al., 1997

\* DT, dye test; ELISA, enzyme-linked immunosorbent assay; IFAT, indirect fluorescent antibody test; LAT, latex agglutination test; MAT, modified agglutination test; SAG1 PCR, polymerase chain reaction amplification of *T. gondii* surface antigen gene DNA confirmed by sequencing.

† All seropositive rodents had titers  $\geq 40$ .

‡ Cutoff for a positive MAT was not specified for rodents.

§ Seroprevalence varied by year after a *T. gondii* vaccination program for cats on the farms was initiated.

|| Inoculation of homogenates of heart and brain tissue into mice for bioassay.

# Two of the 6 positive were *Microtus* spp. (voles); 4 were *Peromyscus* spp.

¶ 104/149 (69.8%) seropositive by IFAT using 1:16 as the cutoff for a positive test.

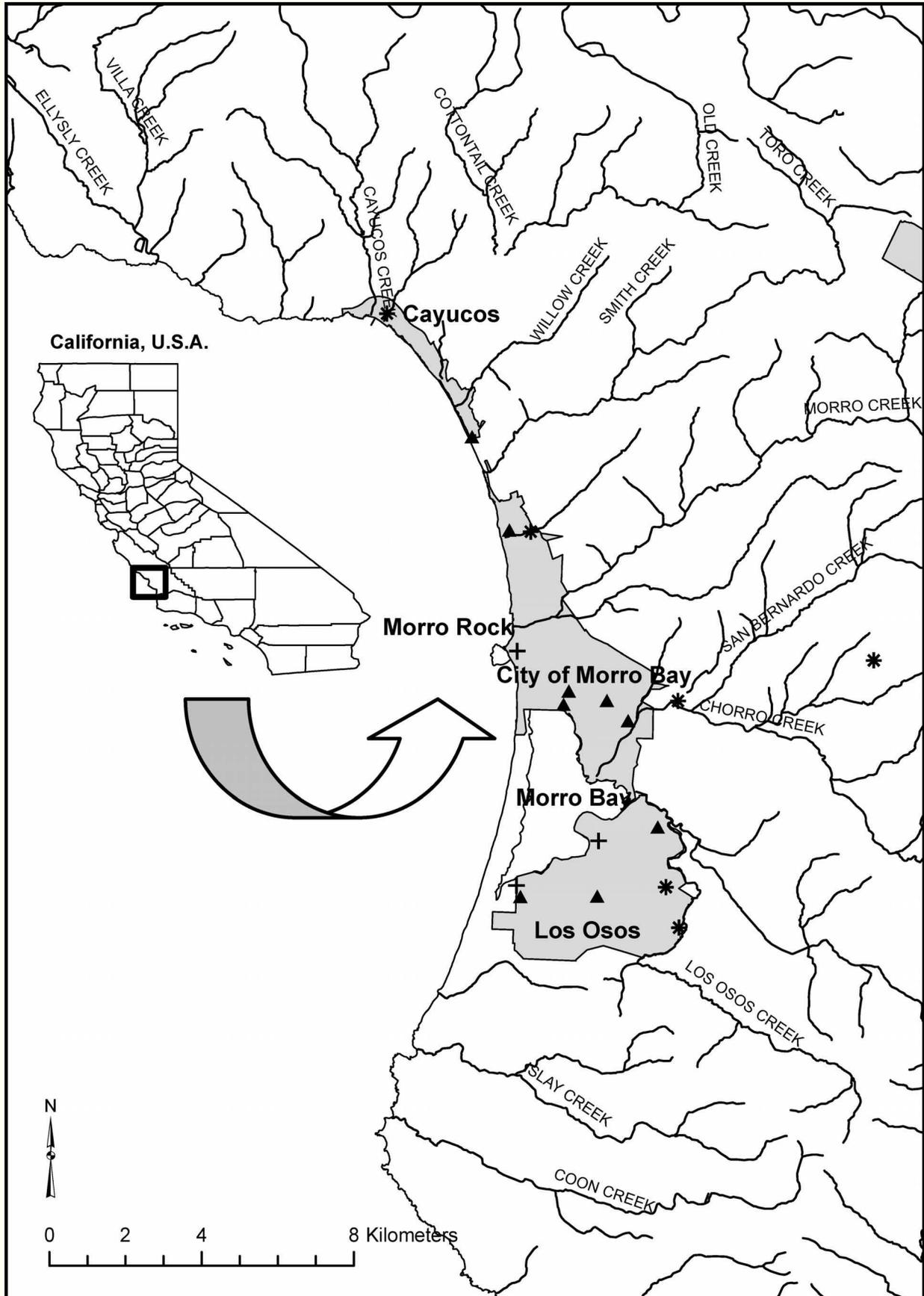
\*\* Five gray squirrels, 5 fox squirrels, and 1 thirteen-lined ground squirrel.

tissues (Jacobs, 1964; Owen and Trees, 1998). Alternatively, the higher risk for infection in adults compared to juveniles in the present study suggests that congenital transmission is rare or fatal in California mice. Nonetheless, congenital transmission based on the presence of tissue cysts in the brain was documented in 2 of 4 pups born to an experimentally infected *P. maniculatus* (Dubey, 1983). High frequencies of congenital transmission have also been detected by PCR in urban populations of naturally infected mice, but the earlier study did not address pup survival since DNA amplification was performed on fetuses (Marshall et al., 2004). Studies of congenitally infected mice suggest that *T. gondii* infection results in motor and behavioral deficits that would make them more susceptible to predation in the wild (Hay et al., 1983, 1984, 1985; Vyas et al., 2007). To our knowledge, other than the aforementioned experimental infection of *P. maniculatus* (Dubey, 1983), there have been no other published studies in *Peromyscus* spp. of transmission and survival probability for pups born to mice with primary or chronic *T. gondii* infection. Experimental studies in *P. californicus* or other *Peromyscus* spp. would be helpful to assess the impact of congenital transmission on behavior and survival in these species.

Three (13%) of the 23 ground squirrels trapped at Morro Rock were seropositive. A 30- to 40-member feral cat colony had been removed from this site 10 yr earlier. The squirrels at this site live in rocks adjacent to the mouth of Morro Bay in a tidal zone and are fed items such as peanuts and bread by the public, which the squirrels retrieve from the ground. Oocysts transported via water and deposited in the tidal zone or disseminated in the soil by cats defecating in the area could serve as a source of infection for these squirrels. California ground squirrels could also become infected with *T. gondii* if they scavenge dead carrion, as has been reported for this species (Jameson and Peeters, 1988). The *T. gondii* seroprevalence in squirrels detected in the present study is comparable to the 18%

seroprevalence in squirrels from Kansas (Smith and Frenkel, 1995). However, only 2 studies of *T. gondii* seroprevalence in squirrels have been conducted since 1985 (Table IV).

In the present study, there was no association of *T. gondii* seropositivity with rodents sampled near riparian habitats or the inlet of Morro Bay, despite the fact that one hypothesized route of transmission of *T. gondii* oocysts to sea otters is via freshwater outflow (Miller et al., 2002). The results of the present study suggest that transport mechanisms other than, or in addition to, freshwater outflow may be involved. *Toxoplasma gondii* oocysts could reach coastal waters in non-point-source runoff from storm drains or infiltrate aquifers connected with the intertidal zone. The lack of association of *T. gondii*-infected rodents with streams and water bodies could also be related to the fact that rodents consume most of the water they require from their food (Jameson and Peeters, 1988) and that water courses transport oocysts to the near-shore marine environment with little dissemination to the surrounding habitat. When *T. gondii* seroprevalence in *P. maniculatus* around the Humpback Reservoir, British Columbia, Canada, was studied following a human waterborne toxoplasmosis outbreak in 1995 (Aramini et al., 1999), the overall seroprevalence in mice sampled in riparian habitats was 10.6% (subheading *Peromyscus* spp., Table IV). This is comparable to the 15% seroprevalence for rodents sampled near streams in the present study. However, all rodents in the British Columbia investigation were sampled in riparian environments, so no comparison with non-riparian habitats was possible. The association of bodies of water and streams with rodent infection may require a closer association with water than that of the land-dwelling rodents in the present study. Rodents that swim in water (muskrats or pacas) or wade through low-lying semiaquatic environments (capibaras) appear to have higher infection prevalences (ranging from 17 to 60%) than do rodents inhabiting more arid environments (see subheading Miscellaneous spp., Table IV). Oocysts can survive for long



periods in water and moist soil, especially if they are protected from ultraviolet light (Yilmaz and Hopkins, 1972; Dubey, 1998; Lindsay et al., 2003).

Rodents in the present study were about 40% less likely to be seropositive for *T. gondii* (OR 0.62) if sampled near residential housing. This finding was unexpected, because the greater density of cats in peridomestic habitats is likely to increase the potential for contamination of the soil with *T. gondii* oocysts. The finding of a lower risk for *T. gondii* infection in rodents sampled near residential housing differed from findings of rodent surveys in British Columbia, Canada (Aramini et al., 1999), and the United Kingdom, where 59% of mice living in urban environments were infected with *T. gondii* compared to 46% of mice captured in rural habitats (D. Thomasson, pers. comm.). In the British Columbia study, deer mice sampled <3 km from residences were more likely to be infected with *T. gondii* than were deer mice sampled elsewhere (subheading *Peromyscus* spp., Table IV). The prevalence of *T. gondii* (tested by PCR) in urban house mice in Manchester, U.K. (59%) was one of the highest prevalences ever reported for mice (subheading house mice, Table IV). Reasons for the lower seroprevalence in mice sampled from peridomestic environments in the present study may be related to detection bias and the close proximity (<3 km) of all but one site to large areas of residential housing. The only site that was >3 km from the community boundaries (the easternmost site in Fig. 1) was home to an owned domestic cat with a high *T. gondii* titer. There were no sites where the presence of cats or wild felids could be completely ruled out, and all sites were potentially located within the home range of owned and feral domestic cats.

Trap success was significantly lower at sites impacted by human populations or close to feral cat colonies. Low trap success may be a result of homeowner rodent extermination efforts and/or cat predation. Experimental studies have documented behavioral changes in *T. gondii*-infected rodents that may make them more susceptible to predation (Hutchison et al., 1980; Berdoy et al., 1995, 2000; Vyas et al., 2007). Infected mice spend more time moving around in familiar environments, demonstrate impaired motor performance, and are less reactive to novel stimuli than are uninfected controls (Hutchison et al., 1980). Compared to their uninfected counterparts, *T. gondii*-infected rats approach novel stimuli more readily, exhibit greater physical activity, and lose their aversion for cat urine (Berdoy et al., 1995, 2000; Vyas et al., 2007). *Toxoplasma gondii*-infected rodents may, therefore, be more easily captured by cats compared with *T. gondii*-free rodents, and could be less likely to be detected in sites where cats are abundant.

*Toxoplasma gondii* infection in rodents probably involves the interaction of complex ecological elements that include abiotic factors such as climate and oocyst survival, and biotic factors such as differing susceptibility of rodent species to *T. gondii* infection and genotype, different patterns of congenital transmission in mice and rats, and changes in host behavior that make infected hosts more susceptible to predation. It may, therefore, be difficult to elucidate risk factors that are strongly

associated with *T. gondii* infection in rodents, even when sentinel species such as sea otters along the adjacent coastline suggest high levels of *T. gondii* transmission in coastal terrestrial fauna.

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## LITERATURE CITED

- ARAMINI, J. J., C. STEPHEN, J. P. DUBEY, C. ENGELSTOFT, H. SCHWANTJE, AND C. S. RIBBLE. 1999. Potential contamination of drinking water with *Toxoplasma gondii* oocysts. *Epidemiology and Infection* **122**: 305–315.
- BERDOY, M., J. P. WEBSTER, AND D. W. MACDONALD. 1995. Parasite-altered behaviour: Is the effect of *Toxoplasma gondii* on *Rattus norvegicus* specific? *Parasitology* **111**: 403–409.
- , ———, AND ———. 2000. Fatal attraction in rats infected with *Toxoplasma gondii*. *Proceedings of the Royal Society of London B* **267**: 1591–1594.
- BRILLHART, D. B., L. B. FOX, J. P. DUBEY, AND S. J. UPTON. 1994. Seroprevalence of *Toxoplasma gondii* in wild mammals in Kansas. *Journal of the Helminthological Society Washington* **61**: 117–121.
- CANON-FRANCO, W. A., L. E. YAI, A. M. JOPPERT, C. E. SOUZA, S. R. N. D'AURIA, J. P. DUBEY, AND S. M. GENNARI. 2003. Seroprevalence of *Toxoplasma gondii* antibodies in the rodent *Capybara* (*Hydrochoeris hydrochoeris*) from Brazil. *Journal of Parasitology* **89**: 850.
- CHHABRA, M. B., S. L. GUPTA, AND O. P. GAUTAM. 1985. *Toxoplasma* seroprevalence in animals in northern India. *International Journal of Zoonoses* **12**: 136–142.
- CHILDS, J. E., AND W. S. SEEGAR. 1986. Epidemiologic observations on infection with *Toxoplasma gondii* in three species of urban mammals from Baltimore, Maryland, USA. *International Journal of Zoonoses* **13**: 249–261.
- CONRAD, P. A., M. A. MILLER, C. KREUDER, E. R. JAMES, J. K. MAZET, H. DABRITZ, D. A. JESSUP, F. GULLAND, AND M. E. GRIGG. 2005. Transmission of *Toxoplasma*: Clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. *International Journal of Parasitology* **35**: 1155–1168.
- DABRITZ, H. A., I. A. GARDNER, M. A. MILLER, M. R. LAPPIN, E. R. ATWILL, A. E. PACKHAM, A. C. MELLI, AND P. A. CONRAD. 2007. Evaluation of two *Toxoplasma gondii* serologic tests used in a serosurvey of domestic cats in California. *Journal of Parasitology* **93**: 806–816.
- DE THOISY, B., M. DEMAR, C. AZNAR, AND B. CARMÉ. 2003. Ecologic correlates of *Toxoplasma gondii* exposure in free-ranging neotropical mammals. *Journal of Wildlife Diseases* **39**: 456–459.
- DEFEO, M. L., J. P. DUBEY, T. N. MATHER, AND R. C. RHODES III. 2002. Epidemiologic investigation of seroprevalence of antibodies to *Toxoplasma gondii* in cats and rodents. *American Journal of Veterinary Research* **63**: 1714–1717.
- DUBEY, J. P. 1983. *Toxoplasma gondii* infection in rodents and insectivores from Montana. *Journal of Wildlife Diseases* **19**: 149–150.
- . 1998. *Toxoplasma gondii* oocyst survival under defined temperatures. *Journal of Parasitology* **84**: 862–865.
- , N. L. MILLER, AND J. K. FRENKEL. 1970. The *Toxoplasma gondii* oocyst from cat feces. *Journal of Experimental Medicine* **132**: 636–662.
- , E. A. ROLLOR, K. SMITH, O. C. KWOK, AND P. THULLIEZ. 1997.

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FIGURE 1. Rodent trapping sites (2004–2006) in the Morro Bay area, California. Communities are designated by shaded areas. Trapping site adjacent to Morro Bay (+), adjacent to riparian habitat (\*), or adjacent to neither (▲).

- Low seroprevalence of *Toxoplasma gondii* in feral pigs from a remote island lacking cats. *Journal of Parasitology* **83**: 839–841.
- , S. K. SHEN, O. C. KWOK, AND P. THULLIEZ. 1997. Toxoplasmosis in rats (*Rattus norvegicus*): Congenital transmission to first and second generation offspring and isolation of *Toxoplasma gondii* from seronegative rats. *Parasitology* **115**: 9–14.
- , AND P. THULLIEZ. 1989. Serologic diagnosis of toxoplasmosis in cats fed *Toxoplasma gondii* tissue cysts. *Journal of the American Veterinary Medical Association* **194**: 1297–1299.
- , P. THULLIEZ, R. M. WEIGEL, C. D. ANDREWS, P. LIND, AND E. C. POWELL. 1995. Sensitivity and specificity of various serologic tests for detection of *Toxoplasma gondii* infection in naturally infected sows. *American Journal of Veterinary Research* **56**: 1030–1036.
- , R. M. WEIGEL, A. M. SIEGEL, P. THULLIEZ, U. D. KITRON, M. A. MITCHELL, A. MANNELLI, N. E. MATEUS-PINILLA, S. K. SHEN, AND O. C. KWOK. 1995. Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. *Journal of Parasitology* **81**: 723–729.
- EBERHARD, T. 1954. Food habits of Pennsylvania house cats. *Journal of Wildlife Management* **18**: 284–286.
- FAN, C. K., K. E. SU, W. C. CHUNG, Y. J. TSAI, H. Y. CHIOL, C. F. LIN, C. T. SU, AND P. H. CHAO. 1998. Seroepidemiology of *Toxoplasma gondii* infection among Atayal aborigines and local animals in Nanao district, Ilan county and Jen-ai district, Nan-tou county, Taiwan. *Kaohsiung Journal of Medical Sciences* **14**: 762–769.
- FRENKEL, J. K., K. M. HASSANEIN, R. S. HASSANEIN, E. BROWN, P. THULLIEZ, AND R. QUINTERO-NUNEZ. 1995. Transmission of *Toxoplasma gondii* in Panama City, Panama: A five-year prospective cohort study of children, cats, rodents, birds, and soil. *American Journal of Tropical Medicine and Hygiene* **53**: 458–468.
- GARCELON, D. K., R. K. WAYNE, AND B. J. GONZALES. 1992. A serologic survey of the island fox (*Urocyon littoralis*) on the Channel Islands, California. *Journal of Wildlife Diseases* **28**: 223–229.
- GENCHI, G., G. A. POLIDORI, L. ZAGHINI, AND P. LANFRANCHI. 1991. Aspetti epidemiologici della toxoplasmosi nell'allevamento intensivo del suino. *Archivio Veterinario Italiano* **42**: 105–111.
- HAY, J., P. P. AITKEN, AND M. A. ARNOTT. 1985. The influence of congenital *Toxoplasma* infection on the spontaneous running activity of mice. *Zeitschrift für Parasitenkunde* **71**: 459–462.
- , D. M. HAIR, W. M. HUTCHISON, AND D. I. GRAHAM. 1983. The effect of congenital and adult-acquired *Toxoplasma* infections on the motor performance of mice. *Annals of Tropical Medicine and Parasitology* **77**: 261–277.
- , W. M. HUTCHISON, AND D. I. GRAHAM. 1984. The effect of congenital *Toxoplasma* infection on mouse activity and relative preference for exposed areas over a series of trials. *Annals of Tropical Medicine and Parasitology* **78**: 611–618.
- HEJLICEK, K., I. LITERAK, AND J. NEZVAL. 1997. Toxoplasmosis in wild mammals from the Czech Republic. *Journal of Wildlife Diseases* **33**: 480–485.
- HUBBS, E. L. 1951. Food habits of feral house cats in the Sacramento valley. *California Fish and Game* **37**: 177–189.
- HUGHES, J. M., R. H. WILLIAMS, E. K. MORLEY, D. A. N. COOK, R. S. TERRY, R. G. MURPHY, J. E. SMITH, AND G. HIDE. 2006. The prevalence of *Neospora caninum* and co-infection with *Toxoplasma gondii* by PCR analysis in naturally occurring mammal populations. *Parasitology* **132**: 1–8.
- HUTCHISON, W. M., M. S. BRADLEY, B. CHEYNE, B. W. P. WELLS, AND J. HAY. 1980. Behavioural abnormalities in *Toxoplasma*-infected mice. *Annals of Tropical Medicine and Parasitology* **74**: 337–345.
- JACKSON, M. H., W. M. HUTCHISON, AND J. C. SIM. 1986. Toxoplasmosis in a wild rodent population of central Scotland and a possible explanation of the mode of transmission. *Journal of Zoology London (A)* **209**: 549–557.
- JACOBS, L. 1964. The occurrence of *Toxoplasma* infection in the absence of demonstrable antibodies. In *First International Congress of Parasitology*, Vol. 1, A. Caorradetti (ed.). Pergamon Press, Roma, Italy, p. 176–177.
- JAMESON, JR., E. W., AND H. J. PEETERS. 1988. California mammals. University of California Press, Berkeley, California, 403 p.
- JEON, S. H., AND T. S. YONG. 2000. Serologic observation of *Toxoplasma gondii* prevalence in *Apodemus agrarius*, a dominant species of field rodents in Korea. *Yonsei Medical Journal* **41**: 491–496.
- JONES, J. L., D. KRUSZON-MORAN, M. WILSON, G. MCQUILLAN, T. NAVIN, AND J. B. MCAULEY. 2001. *Toxoplasma gondii* infection in the United States: Seroprevalence and risk factors. *American Journal of Epidemiology* **154**: 357–365.
- , A. LOPEZ, AND M. WILSON. 2003. Congenital toxoplasmosis. *American Family Physician* **67**: 2131–2138.
- KENNY, D. E., M. R. LAPPIN, F. KNIGHTLY, J. BALER, M. BREWER, AND D. M. GETZY. 2002. Toxoplasmosis in Pallas' cats (*Otocolobus felis manul*) at the Denver Zoological Gardens. *Journal of Zoo and Wildlife Medicine* **33**: 131–138.
- LAPPIN, M. R., AND C. C. POWELL. 1991. Comparison of latex agglutination, indirect hemagglutination, and ELISA techniques for the detection of *Toxoplasma gondii*-specific antibodies in the serum of cats. *Journal of Veterinary Internal Medicine* **5**: 299–301.
- LEHMANN, T., D. H. GRAHAM, E. DAHL, C. SREEKUMAR, F. LAUNER, J. L. CORN, H. R. GAMBLE, AND J. P. DUBEY. 2003. Transmission dynamics of *Toxoplasma gondii* on a pig farm. *Infection Genetics and Evolution* **3**: 135–141.
- LIN, S., Z. C. LING, B. C. ZENG, AND H. Y. YANG. 1990. Prevalence of *Toxoplasma gondii* infection in man and animals in Guangdong, Peoples Republic of China. *Veterinary Parasitology* **34**: 357–360.
- LINDSAY, D. S., M. V. COLLINS, S. M. MITCHELL, R. A. COLE, G. J. FLICK, C. N. WETCH, A. LINDQUIST, AND J. P. DUBEY. 2003. Sporulation and survival of *Toxoplasma gondii* oocysts in seawater. *Journal of Eukaryotic Microbiology* **50**: 687–688.
- MARSHALL, P. A., J. M. HUGHES, R. H. WILLIAMS, J. E. SMITH, R. G. MURPHY, AND G. HIDE. 2004. Detection of high levels of congenital transmission of *Toxoplasma gondii* in natural urban populations of *Mus domesticus*. *Parasitology* **128**: 39–42.
- MATEUS-PINILLA, N. E., J. P. DUBEY, L. CHOROMANSKI, AND R. M. WEIGEL. 1999. A field trial of the effectiveness of a feline *Toxoplasma gondii* vaccine in reducing *T. gondii* exposure for swine. *Journal of Parasitology* **85**: 855–860.
- MCMURRY, F. B., AND C. C. SPERRY. 1941. Food of feral house cats in Oklahoma, a progress report. *Journal of Mammalogy* **22**: 184–190.
- MILLER, M. A., I. A. GARDNER, C. KREUDER, D. M. PARADIES, K. R. WORCESTER, D. A. JESSUP, E. DODD, M. D. HARRIS, J. A. AMES, A. E. PACKHAM, ET AL. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondii* infection of southern sea otters (*Enhydra lutris nereis*). *International Journal for Parasitology* **32**: 997–1006.
- , K. SVERLOW, P. R. CROSBIE, B. C. BARR, L. J. LOWENSTINE, F. M. GULLAND, A. E. PACKHAM, AND P. A. CONRAD. 2001. Isolation and characterization of two parasitic protozoa from a Pacific harbor seal (*Phoca vitulina richardsi*) with meningoencephalomyelitis. *Journal of Parasitology* **87**: 816–822.
- MOLSHER, R., A. NEWSOME, AND C. DICKMAN. 1999. Feeding ecology and population dynamics of the feral cat (*Felis catus*) in relation to the availability of prey in central-eastern New South Wales. *Wildlife Research* **26**: 593–607.
- MURATA, K. 1989. A serological survey of *Toxoplasma gondii* infection in zoo animals and other animals. *Japanese Journal of Veterinary Science* **51**: 935–940.
- OWEN, M. R., AND A. J. TREES. 1998. Vertical transmission of *Toxoplasma gondii* from chronically infected house (*Mus musculus*) and field (*Apodemus sylvaticus*) mice determined by polymerase chain reaction. *Parasitology* **116**: 299–304.
- SMITH, D. D., AND J. K. FRENKEL. 1995. Prevalence of antibodies to *Toxoplasma gondii* in wild mammals of Missouri and east central Kansas: Biologic and ecologic considerations of transmission. *Journal of Wildlife Diseases* **31**: 15–21.
- SMITH, K. E., J. J. ZIMMERMAN, S. PATTON, G. W. BERAN, AND H. T. HILL. 1992. The epidemiology of toxoplasmosis on Iowa swine farms with an emphasis on the roles of free-living mammals. *Veterinary Parasitology* **42**: 199–211.
- TENTER, A. 1987. Comparison of enzyme-linked immunosorbent assay and indirect fluorescent antibody test for the detection of IgG antibodies to *Sarcocystis muris*. *Zentralblatt für Bakteriologie und Hygiene (A)* **267**: 259–271.
- , A. R. HECKEROTH, AND L. M. WEISS. 2000. *Toxoplasma gondii*: From animals to humans. *International Journal for Parasitology* **30**: 1217–1258.

- TSUBOTA, N., S. OHSHIMA, AND M. HOSHINO. 1977. Studies on latex agglutination test for toxoplasmosis. Evaluation of the microtiter test as a serologic test for toxoplasmosis in some animals. *Japanese Journal of Parasitology* **4**: 291–298.
- VYAS, A., S. K. KIM, N. GIACOMINI, J. C. BOOTHROYD, AND R. M. SAPPOLSKY. 2007. Behavioral changes induced by *Toxoplasma* infection of rodents are highly specific to aversion of cat odors. *Proceedings of the National Academy of Sciences U S A* **104**: 6442–6447.
- WALLACE, G. D. 1969. Serologic and epidemiologic observations on toxoplasmosis on three Pacific atolls. *American Journal of Epidemiology* **90**: 103–111.
- WEBSTER, J. P. 1994. Prevalence and transmission of *Toxoplasma gondii* in wild brown rats, *Rattus norvegicus*. *Parasitology* **108**: 407–411.
- YILMAZ, S. M., AND S. H. HOPKINS. 1972. Effects of different conditions on duration of infectivity of *Toxoplasma gondii* oocysts. *Journal of Parasitology* **58**: 938–939.
- ZHANG, S. Y., S. F. JIANG, Y. Y. HE, C. E. PAN, M. ZHU, AND M. X. WEI. 2004. Serologic prevalence of *Toxoplasma gondii* in field mice, *Microtus fortis*, from Yuanjiang, Hunan Province, People's Republic of China. *Journal of Parasitology* **90**: 437–438.