ABSTRACT: Field studies were conducted on 47 swine farms in Illinois during 1992 and 1993 to identify sources and reservoirs of Toxoplasma gondii infection. Blood samples were obtained from swine and from trapped wildlife. Serum antibodies to T. gondii were determined using the modified agglutination test, incorporating mercaptoethanol. Antibodies to T. gondii (titer ≥ 25) were found in 97 of 4,252 (2.3%) finishing pigs, 395 of 2,617 (15.1%) sows, 267 of 391 (68.3%) cats, 126 of 188 (67.0%) raccoons, 7 of 18 (38.9%) skunks, 29 of 128 opossums (22.7%), 6 of 95 (6.3%) rats, 3 of 61 (4.9%) white-footed mice (Peromyscus sp.), and 26 of 1,243 (2.1%) house mice (Mus musculus). Brains and hearts of rodents trapped on the farm were bioassayed in mice for the presence of T. gondii. Toxoplasma gondii was recovered from tissues of 7 of 1,502 (0.5%) house mice, 2 of 67 (3.0%) white-footed mice, and 1 of 107 (0.9%) rats. Feces of 274 cats trapped on the farms and samples of feed, water, and soil were bioassayed in mice for the presence of T. gondii oocysts. Toxoplasma gondii was isolated from 2 of 491 (0.4%) feed samples, 1 of 79 (1.3%) soil samples, and 5 of 274 (1.8%) samples of cat feces. All mammalian species examined were reservoirs of T. gondii infection. All farms had evidence of T. gondii infection either by detection of antibodies in swine or other mammalian species, or by detection of oocysts, or by recovery from rodent by bioassay. The possibility of transmission of T. gondii to swine via consumption of rodents, feed, and soil was confirmed.

Humans become infected with Toxoplasma gondii usually by ingesting oocysts in food and water contaminated by cat feces or by consuming tissue cysts in undercooked meat (Dubey and Beattie, 1988). Pork is considered to be the most important meat source of T. gondii infection in the U.S. (Dubey, 1986). There are potentially serious consequences of T. gondii infection in humans. Exposure of women to T. gondii for the first time during pregnancy can result in perinatal mortality and birth defects (Alford et al., 1974; Wilson et al., 1980; Dubey and Beattie, 1988; Frenkel, 1990). Infection of immunocompromised humans, e.g., cancer and AIDS patients can result in encephalitis, blindness, and death (Dubey and Beattie, 1988; Frenkel, 1990).

Infection of swine with T. gondii is widespread. In a national survey in 1983–1984, antibodies to T. gondii were found in 23% of 11,229 market-aged <7 mo and 41.4% of 613 adult swine (Dubey et al., 1991). Recently, viable T. gondii was isolated from 17% of 1,000 sows from Iowa (Dubey, Thulliez, and Powell, 1995). There are several potential reservoirs of T. gondii on swine farms. Cats are the definitive host; the shedding of T. gondii oocysts in cat feces may contaminate feed, water, and soil that can be ingested by swine. Transmission to swine may also occur by the consumption of tissues of animals such as rodents and birds infected with T. gondii tissue cysts and by cannibalism (Penkert, 1973; Dubey and Beattie, 1988).

In order to develop control strategies to reduce or eliminate T. gondii in the nation’s swine herds, it is imperative to obtain detailed information on the reservoirs of infection and the modes of transmission of T. gondii to swine. Epidemiologic investigations of the reservoirs of T. gondii on swine farms have been limited. Lubroth et al. (1983) in a study of 2 swine farms in Georgia found serological evidence of T. gondii infection in house mice, white-footed mice, and raccoons, and rats, as well as recovering T. gondii by bioassay of rodent tissue in mice; they concluded that rodents were a major source of T. gondii infection for swine. Smith et al. (1992) trapped wildlife on 19 swine farms in 3 counties in central Iowa, and found serologic evidence for T. gondii infection in cats, rodents, raccoons, skunks, and opossums; cats were implicated as the primary source of T. gondii infection for swine. Neither of these studies attempted detection of T. gondii oocysts on swine farms.

We have conducted extensive epidemiological investigations on T. gondii infection on swine farms in Illinois (Weigel, Dubey, Siegel, Hoefling et al., 1995). In the first paper of this series, we report the results of an investigation of sources and reservoirs of T. gondii infection on 47 swine farms in Illinois. The companion paper (Weigel, Dubey, Siegel, Kitron et al. 1995) examines risk factors for transmission of T. gondii to swine.

MATERIALS AND METHODS

Field methods

Selection of farms: The epidemiologic investigations of T. gondii infection on swine farms was conducted during the spring and summer of 1992 and 1993. In each year, 24 farms were selected. In 1992, 14 of the farms were selected during recruitment of farms for the cross-sectional survey described in the companion paper (Weigel, Dubey, Siegel, Kitron et al. 1995). In addition, 10 farms used previously for research and clinical training were recruited. The sample of 24 farms was selected to represent the diversity of Illinois swine operations in herd size, type of confinement, and geographic location.

In 1993, a sample of 24 farms was selected from the sampling frame of farms with completed risk factor interviews that had not been selected in 1992. Selection was by stratified random sampling, with geographic region (3 levels: north, central, south), type of housing (4 levels: total confinement, partial confinement with sows outside, partial confinement with growing or finishing pigs outside, pasture), and herd size (2 levels: above median breeding herd size [= 167.5 sows], below median breeding herd size) as strata. No distinction was made between swine housed outside on dirt or on concrete. However, on the farms selected, concrete lots were covered with mud. Toxoplasma gondii serologic prevalence was not a factor taken into account in farm selection.
One pasture operation studied in 1992 was reselected for the 1993 study because the farm had become a farrow-to-finish production unit. Thus, there were 47 farms in the 2-yr sample. Each farm in the study was visited 3 times between late March and early October.

Survey of swine for T. gondii: On each visit, both blood sample was collected and captured. Approximately 0.5 ml of blood was obtained from weight pigs (or the largest finishing pigs on the premises). In 1992, an additional sample of blood from 30 sows was taken on the first of the 3 visits. In 1993, blood was obtained from 30 sows on each of the 3 visits. A sample of 30 sows provides a 95% confidence of detecting a T. gondii seroprevalence of at least 10%; a sample of 90 sows or finishing pigs provides a 99% confidence of detecting a seroprevalence of at least 5% (Beal, 1983).

Trapping of animals: On each visit, overnight live-trapping of small and medium-size mammals was conducted. Traps were set for mice indoors (at a density of 1 per 150 m² of building floor space, with additional traps set in areas where sightings of mice or mouse feces confirmed their presence) and outdoors (at 30-m intervals along perimeters of swine buildings and lots). These traps were designed to trap small rodents; however, occasionally small birds were also trapped. A total of 32 traps were set for rats, both indoors and outdoors. These were mostly distributed evenly throughout the indoor facilities except when the farm personnel identified an area where rats were sighted, in which case more traps were placed in these areas. The bait for rodent traps was bread with peanut butter. A total of 20 medium-sized mammal traps baited with sarines were set outdoors. Their primary purpose was to trap cats, although raccoons, opossums, and other medium-sized mammals were also trapped. These traps were dispersed over the farm, with 10 placed near swine buildings and lots and 10 placed in fields, woods, and along ponds and streams (within 0.8 km of the farm). In addition, whenever possible, additional cats were caught by hand.

Animal processing: Blood samples were obtained from each animal captured. All animals except pet cats and rats were anesthetized by injection with a ketamine HCl and acepromazine maleate (Aveco Co., Inc., Fort Dodge, Iowa) cocktail. Rats were anesthetized with ether. Rodents and birds were necropsied in the field, and the hearts and brains were collected. All other live-trapped animals were released. Feces from cats were obtained from anesthetized cats by inserting a latex-gloved finger into the anus, or by abdominal massage, or by a water enema, and examined microscopically for T. gondii tissue cysts (Dubey and Desmonts, 1980). Oocysts or tissue cysts were demonstrated in their tissues and antibodies to T. gondii were assayed in MAT. All mice, irrespective of their serologic status, were killed 2 mo after inoculation, and a 2-4-mm portion of their cerebrum was crushed between a glass slide and coverslip and examined microscopically for T. gondii tissue cysts (Dubey and Beattie, 1988). Mice were considered T. gondii positive only when ta- chyozytes or tissue cysts were demonstrated in their tissues and antibodies in the serum.

Bioassay in laboratory mice to identify T. gondii infection: All mice used for bioassays were Swiss-Webster albino females weighing approximately 20-25 g. Each sample of rodent tissue, feed, water, and soil were inoculated subcutaneously (s.c.) into 2 mice. The processed samples of cat feces were inoculated orally or s.c. into 2-4 mice. The mice were examined for T. gondii infection. Infection of the brain of the mice that died after inoculation with test material were examined microscopically for T. gondii tachyzoites (lungs) or tissue cysts (brain). Survivors were bled from the orbital sinus 2 mo after inoculation with test material; a 1:50 dilution of each mouse serum was tested for anti-T. gondii antibodies using the MAT. All mice, irrespective of their serologic status, were killed 2 mo after inoculation, and a 2-4 mm portion of their cerebrum was crushed between a glass slide and coverslip and examined microscopically for T. gondii tissue cysts (Dubey and Beattie, 1988). Mice were considered T. gondii positive only when tachyzoites or tissue cysts were demonstrated in their tissues and antibodies in the serum.

Brains of mice containing tissue cysts of the isolates from feed, water, cat feces, or rodent tissues were fed directly to T. gondii-free cats or were first cultivated in T. gondii-free laboratory mice and then fed to T. gondii-free cats. These procedures were undertaken to exclude Haemoplasma hammondi infection. Toxoplasma gondii and H. hammondi are structurally similar coccidians, however, H. hammondi cannot be maintained by mouse to mouse passage (Frenkel and Dubey, 1975). Oocysts obtained from experimental cats were orally inoculated into mice to complete the transmission cycle. The cats were from the T. gondii cat colony at the Beltsville Agricultural Research Center, Beltsville, Maryland (Dubey, 1995). Antibodies to T. gondii were not found in 1:25 dilution of the preinoculation serum of any of these cats when examined in MAT.

RESULTS

Table I presents a summary of the trapping and serological testing results by species and year for all 47 farms in the study.
Table II shows the variation among farms in serological testing results. Seroprevalence is defined as the percentage of samples that tested positive at the 1:25 dilution. Overall, the *Toxoplasma gondii* seroprevalence was highest in cats (68.3%) and raccoons (67.0%). Adult cats had a higher seroprevalence (75.6%) than juvenile cats (45.8%). Cats were found on all farms except 1, with seropositive cats on 44 farms (93.6%). The median cat seroprevalence among farms was 66.7%. Except for house mice, relatively few rodents were trapped. The overall seroprevalence among house mice was low (2.1%). House mice were trapped on all 47 farms, with 14 farms (29.8%) having at least 1 seropositive mouse. The highest seroprevalence in *Mus musculus* among farms was 14.3%. With respect to swine, 15.1% of sow samples were seropositive. The overall seroprevalence among house mice was low (2.1%). House mice were trapped on all 47 farms, with 14 farms (29.8%) having at least 1 seropositive mouse. The highest seroprevalence in *M. musculus* among farms was 14.3%. With respect to swine, 15.1% of sow samples were seropositive. There was a wide, positively skewed distribution of sow seroprevalence rates among farms (0% to 93.9%; median = 6.7%), with 37 farms (78.7%) having at least 1 seropositive sow. The seroprevalence among finishing pigs was 2.3%. Among farms, the median seroprevalence was 1.1% (maximum = 16.7%), with 26 farms (55.3%) having seropositive finishing pigs.

*Toxoplasma gondii* was isolated from the tissues of 7 of 1,502 house mice (0.5% of samples) on 5 farms (10.6% of farms), 2 of 67 (3.0%) white-footed mice on 2 farms (4.3%), and 1 of 107 (0.9%) rats (Table III); no *T. gondii* was isolated from tissue samples of 8 voles (*Microtus* spp.), 3 ground squirrels, 2 moles, 2 shrews, and 21 sparrows. It is noteworthy that of the 9 infected rodents identified, 6 of them had no detectable antibodies to *T. gondii* in their sera (Table III).

*Toxoplasma gondii* was isolated by bioassay in mice from 5 of 274 (1.8%) cat fecal samples on 4 farms (8.5%), 2 of 491 (0.4%) feed samples on 2 farms (4.3%), and 1 of 79 (1.3%) soil samples (Table IV). Experimental cats fed tissue cysts of all strains isolated from rodents, soil, and feed shed *T. gondii* oocysts in their feces. Two of the *T. gondii* strains isolated from house mice were lethal to mice on their original passage (Table III). Oocysts of most isolates from Illinois were lethal to mice. When considering both the serological testing and *T. gondii* isolation from tissues, evidence for the presence of *T. gondii* infection in rodents (either *Mus, Peromyscus*, or rats) was present for 22 farms (46.8%). When considering the serological test-
### Table II. Variation among farms in Toxoplasma gondii seroprevalence by species.

<table>
<thead>
<tr>
<th>Species</th>
<th>Year</th>
<th>No. tested per farm</th>
<th>Farms positive</th>
<th>Seroprevalence by farm (%)</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Farms positive</td>
<td>No. %</td>
</tr>
<tr>
<td><strong>Cats</strong></td>
<td>1992</td>
<td>23</td>
<td>22</td>
<td>95.7</td>
</tr>
<tr>
<td></td>
<td>1993</td>
<td>23</td>
<td>22</td>
<td>91.7</td>
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<tr>
<td></td>
<td>Both</td>
<td>46</td>
<td>44</td>
<td>93.6</td>
</tr>
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<td><strong>Raccoons</strong></td>
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<td>18</td>
<td>15</td>
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</tr>
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<td></td>
<td>1993</td>
<td>16</td>
<td>14</td>
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</tr>
<tr>
<td></td>
<td>Both</td>
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<td>29</td>
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<tr>
<td><strong>Opossums</strong></td>
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<tr>
<td></td>
<td>1993</td>
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<td>8</td>
<td>54.2</td>
</tr>
<tr>
<td></td>
<td>Both</td>
<td>35</td>
<td>17</td>
<td>52.3</td>
</tr>
<tr>
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<td>22</td>
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<td></td>
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<td><strong>Rats</strong></td>
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<td>10</td>
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<td></td>
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<td>Both</td>
<td>47</td>
<td>14</td>
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#### Discussion

As expected, there was a high seroprevalence for cats (68.3%), indicating a high rate of prior exposure to *T. gondii* and probably also a high degree of past oocyst shedding. Seroprevalence rates from the 1992 (66.5%) and the 1993 (70.7%) samples were comparable, suggesting the absence of sampling bias. The 46% seroprevalence for juvenile cats indicates that approximately half of the farm cats were exposed to *T. gondii* before reaching maturity.

Compared with the high prevalence of serum antibodies to *T. gondii* in the current survey, it was possible to isolate viable...
oocysts from only 1.8% (5 of 274) of cat fecal samples. These 5 cats represent 4 farms. All 5 cats that were shedding *T. gondii* oocysts in feces had a relatively high (≥200) titer for *T. gondii* antibodies, using the MAT. The most likely explanation for this low isolation rate is that most of the cats (75%) caught were adults. Cats shed oocysts in feces usually for 1 wk after infection, and most cats are infected as juveniles (Dubey and Beattie, 1988). The 46% seroprevalence rate for juvenile cats in this study confirms this observation. Thus, most of the cats caught had presumably shed oocysts in the past. Another factor contributing to the failure to isolate oocysts is the small sample size of feces (usually <1 g) obtainable by massage or digital insertion. Use of an enema in the second year of the study increased the size of fecal samples.

Finding of *T. gondii* oocysts in feces of only 1.8% of cats does not diminish the importance of oocysts in maintaining *T. gondii* in the environment. A cat may excrete millions of oocysts in a day and the oocysts can survive in the environment for >1 yr. The infection may have been derived congenitally (Jacobs, 1964). When infection had been recent, antibodies may not have developed yet. In cases of long-term infection, antibody concentration may have dropped below the level of detection. This indicates that the use of a serum dilution of 1:25 for the detection of *T. gondii* antibodies in the present study may have underestimated the prevalence of *T. gondii* infection in mice. On the other hand, bioassay will not always be successful in isolating *T. gondii* from infected animals. Although concentration of *T. gondii* tissue cysts in either the heart or the brain is expected, it is possible for infection to occur without invasion of these organs. With respect to the bioassay itself, inoculation with infected tissue may not always be successful in establishing infection in the host’s brain or heart.

The prevalence of infection in rodents may have been underestimated. The small number of house mice (median n = 14) and the even smaller number of white-footed mice and rats captured on some farms is insufficient to detect low prevalence of infection where it exists. In addition, less than perfect diagnostic sensitivity contributes to underestimation. In the present study, 5 of the 7 mice that had viable *T. gondii* in their tissues were seronegative. There are several explanations for this phenomenon. The infection may have been derived congenitally (Jacobs, 1964). When infection had been recent, antibodies may not have developed yet. In cases of long-term infection, antibody concentration may have dropped below the level of detection. This indicates that the use of a serum dilution of 1:25 for the detection of *T. gondii* antibodies in the present study may have underestimated the prevalence of *T. gondii* infection in mice. On the other hand, bioassay will not always be successful in isolating *T. gondii* from infected animals. Although concentration of *T. gondii* tissue cysts in either the heart or the brain is expected, it is possible for infection to occur without invasion of these organs. With respect to the bioassay itself, inoculation with infected tissue may not always be successful in establishing infection in the host’s brain or heart.

Sampling bias in trapping also may result in underestimation of the prevalence of infection. Mice are generally more susceptible to *T. gondii* infection than other hosts and the ingestion of a few oocysts can produce clinical toxoplasmosis in mice, which will make them more subject to predation by cats (Dubey and Beattie, 1988). It is also likely that some mice die of acute toxoplasmosis. Both of these factors contribute to underrepresentation of infected rodents in trapping surveys.

Thus, small sample sizes, imperfect diagnostic sensitivity, and sampling bias may have resulted in the underestimation of the prevalence of *T. gondii* infection in rodents on swine farms. It is very likely that >50% of the farms studied had rodent reservoirs of *T. gondii* infection.

The relative abundance of different species of rodents on the swine farms sampled was not estimated precisely. The larger number of mice trapped reflects in part the greater number of mouse traps set. Rats may also be more trap shy than mice, which contributes further to their underrepresentation. Thus, the relative sizes of *T. gondii* reservoirs in these species remains unclear. However, the existence of seropositive test results and the successful isolation of *T. gondii* by bioassay indicates that on swine farms in Illinois, at least 3 rodent species (house mice, white-footed mice, and rats) are a reservoir and potential source of *T. gondii* infection.

In a study of 2 swine farms in Georgia, Lubroth et al. (1983) found 60% (n = 20) of house mice, 100% (n = 3) of white-footed mice, 50% (n = 6) of cotton rats, and 100% (n = 2) of...
Norway rats seropositive for *T. gondii*, using the indirect immunofluorescent antibody test. These higher seroprevalences suggest that *T. gondii* infection in rodents is higher in Georgia than in Illinois. In contrast, in the current study by Smith et al. (1992) of 19 Iowa farms, the house mouse seropositivity was 0.3% (2 positive out of 858 sampled), with 10% of farms (2 of 20) having seropositive mice; serologic testing of 21 white-footed mice and 9 Norway rats yielded negative results. It appears the Iowa farms have a lower house mouse seropositivity because the mean number of samples per farm (29) was comparable to the current study, and the narrower range (21–36) would have allowed better detection of seropositive mice on each farm than existed for the current study.

How rodents become infected in nature is not known. Carnivorism, ingestion of oocysts from contaminated feed, and congenital transmission are all efficient routes of infection. *Toxoplasma gondii* can be repeatedly transmitted congenitally in mice and several infected litters may be produced by chronically infected, clinically normal mice (Beverley, 1959). However, epidemiologic data on remote islands suggests that *T. gondii* is not maintained in the environment without oocysts (Wallace, 1969; Munday, 1972).

The seroprevalence for raccoons (67.0%) in the current study was almost the same as that estimated for cats. Previously, Dubey et al. (1992) estimated the seroprevalence among raccoons sampled from 6 states to be 50%. As omnivores, raccoons feed on carrion and vegetation, and are considered as good monitors of environmental contamination with *T. gondii*. However, despite the high prevalence, raccoons are unlikely to serve as a primary source of *T. gondii* infection for swine because they do not excrete the *T. gondii* oocysts (Dubey et al., 1993), and swine are unlikely to consume raccoon flesh.

Comparative data on the prevalence of infection in mammalian and avian reservoirs of *T. gondii* infection are available from several studies in the United States. In a recent survey (Brillhart et al., 1994), 2 of 115 white-footed mice from Kansas had *T. gondii* antibodies. In the survey from Montana (Dubey, 1983), *T. gondii* was not isolated from tissues of 500 ground squirrels (*Spermophilus richardsoni*), 99 deer mice (*Peromyscus maniculatus*), 84 muskrats (*Ondatra zibethicus*), and 52 meadow voles (*Microtus pennsylvanicus*). The isolation of *T. gondii* from 7 of 1,502 house mice, 2 of 67 *Peromyscus* sp., and 1 of 107 rats in the present study, combined with the serologic evidence from swine farms in Georgia and Iowa, suggests that *T. gondii* is more concentrated on swine farms, most likely due to the higher concentration of cats.

The seroprevalence of *T. gondii* in swine was lower in the present study than in the 1983–1984 national survey that also used the MAT (Dubey et al., 1991), where 23% of 11,229 finishing pigs and 41.4% of 613 breeder hogs had *T. gondii* antibodies at a serum dilution of 1:25. In the national survey, *T. gondii* antibodies were found in 24.2% of 1,330 hogs from Illinois (Dubey et al., 1991). Although the proportion of finishing versus breeding swine from Illinois was not recorded, the majority (>95%) of the hogs surveyed in the entire sample were finishing pigs. The swine seroprevalence estimates in the current field study (15.1% for 2,617 sows, 2.3% for 4,252 finishing pigs) were slightly lower than the 1992 statewide Illinois serological survey of pseudorabies testing samples from a state diagnostic laboratory, where 20.8% of 5,080 breeding hogs and 3.1% of 1,885 finishing pigs were seropositive at 1:25 dilution (Weigel, Dubey, Siegel, Hoefling et al., 1995). These differences probably represent sampling variation. Overall, estimates of swine seroprevalence from the 2 recent Illinois studies have high precision because of the large sample sizes.

Sera for all 3 Illinois surveys were examined in 1 laboratory at Beltsville using the MAT. Overall, the *T. gondii* seroprevalence estimates for swine in the 2 recent surveys from Illinois are substantially lower than the national survey conducted 8–10 yr ago. Without comparative data on herd characteristics, the reasons for this decline in seroprevalence are not clear, but changes in management and housing are potentially important factors. Confinement housing can reduce exposure of pigs to cats and rodents and the use of rodenticides can affect the numbers of rodents on the swine premises.

In a study of Iowa swine, Smith et al. (1992), using the MAT with a titer of 32 as the cutoff for seropositivity, estimated the *T. gondii* seroprevalence for sows to be 14.3% (n = 273); this estimate was similar to the field study conducted here in Illinois. It is not possible to compare the results of the present study with other surveys using other serologic tests.

When the serologic testing for *T. gondii* antibodies in all species, the bioassay of rodent tissues, and the detection of oocysts are considered as evidence of *T. gondii* infection on swine farms, all 47 farms had evidence of *T. gondii* infection. Thus, the risk of transmission of *T. gondii* to swine is a potential problem for all swine farms.

The current investigation has identified all mammalian species captured in large numbers on swine farms in Illinois (cats, raccoons, opossums, skunks, house mice, white-footed mice, rats) as reservoirs of *T. gondii* infection. Rodents are probably the only direct animal source of *T. gondii* infection for swine. Rodents also assist in maintaining the infection in the cat population. The shedding of oocysts by cats on swine farms has been verified, and confirmation has been provided that pig feed and soil are 2 direct sources of oocysts that can infect swine.

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**LITERATURE CITED**


