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RISK FACTORS FOR TRANSMISSION OF *TOXOPLASMA GONDII* ON SWINE FARMS IN ILLINOIS

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ABSTRACT: Two epidemiologic studies of risk factors for transmission of *Toxoplasma gondii* to swine were conducted for farms in Illinois. The first study was a cross-sectional survey of swine farms from the state of Illinois pseudorabies testing program, in which farm owners or managers were interviewed by telephone regarding presence of risk factors for transmission of *T. gondii* on the farm. There were 123 farms surveyed that provided blood samples for at least 30 sows. The mean sow seroprevalence was 19.5% (median = 10.0%). Multiple regression analysis of the association of sow seroprevalence with outdoor housing of sows, cat access to sow areas, number of sows, open feed storage and water delivery, delayed removal of carcasses, and presence of rodents on the farm indicated that higher sow seroprevalence was associated with cat access to sows ($P = 0.009$) and fewer sows in the herd ($P = 0.05$). The second study was a field investigation of 47 swine farms (37 from the cross-sectional study). Data collection included obtaining blood samples from swine, cats, and rodents, and fecal samples from cats, heart and brain tissue from rodents, and feed, water, and soil samples for *T. gondii* examination. The risk of *T. gondii* transmission from cats and rodents to sows and finishing pigs was evaluated, taking into account housing conditions and herd size. Multiple regression analysis indicated that *T. gondii* seroprevalence in finishing pigs increased with more seropositive juvenile cats on the farm ($P < 0.0001$) and higher seroprevalence in house mice ($P = 0.0023$). For sows, the only risk factor associated with increased *T. gondii* seroprevalence was a higher number of seropositive juvenile cats on the farm ($P = 0.0008$). Housing swine outdoors was not associated with a higher *T. gondii* seroprevalence. These results identify *T. gondii* infection in cats (particularly juveniles) and house mice as indicators of increased risk of transmission to swine.

Infection of swine with *Toxoplasma gondii* creates a public health concern because the parasite can be transmitted to humans through the handling and consumption of raw or undercooked pork containing tissue cysts (Dubey and Beattie, 1988). If the risk of transmission of *T. gondii* to humans from consumption of pork is to be reduced, risk factors for acquisition of infection in swine need to be identified.

Several studies on swine farms have identified cats (Smith et al., 1992; Assadi-Rad et al., 1995) and rodents (Lubroth et al., 1983; Assadi-Rad et al., 1995) as playing a role in the transmission of *T. gondii*. In contrast, Smith et al. (1992) conclude that rodents are not an important source of infection for swine. Farm characteristics may modify the risk of *T. gondii* transmission from other species. Housing swine outdoors has been associated with an increased risk of *T. gondii* infection (Lubroth et al., 1983; Zimmerman et al., 1990; Smith et al., 1992; Assadi-Rad et al., 1995). Smaller swine herds have also been shown to be at increased risk (Zimmerman et al., 1990; Assadi-Rad et al., 1995).

There are several unresolved issues regarding *T. gondii* transmission on swine farms that need further investigation. Assadi-Rad et al. (1995) identified the presence of cats on a farm with increasing swine seroprevalence but had no *T. gondii* diagnostic data on cats to identify them as a source of infection. Smith et al. (1992) found a high *T. gondii* seroprevalence in cats but did not find an association with seroprevalence in swine. No previous study has examined the direct sources of *T. gondii* infection (oocysts and tissue cysts) as risk factors for infection in swine. In addition, several herd management factors that may modify the risk of *T. gondii* transmission to swine have not been studied previously. Delivery of feed in open bins or storage in open-sided buildings may increase the opportunity for cats to defecate in feed. Likewise, if water delivery is in open troughs or bowls, cats may deposit oocysts at these sites. Delayed removal of swine carcasses that are infected with *T. gondii* serves as a source of infection for cats.

Reported below are 2 studies examining risk factors for *T. gondii* infection on swine farms in Illinois. The first study was a cross-sectional survey of 123 farms from a serological survey of *T. gondii* infection in swine (Weigel et al., 1995), designed to identify demographic, housing, management, and environmental characteristics of farms associated with an increased risk of *T. gondii* transmission. The hypotheses tested in this study are that higher seroprevalence of antibodies to *T. gondii* in sows is associated with housing sows outside, access of cats to swine pens and lots, fewer sows in the herd, feed delivery in open bins, feed storage in open-sided buildings, water delivery in open troughs or bowls, delayed removal of swine carcasses, presence of mice on the farm, and presence of rats on the farm. The second investigation was an intensive field investigation of 47 swine farms (Dubey et al., 1995). The hypotheses to be tested in this study are that higher seroprevalence of antibodies to *T. gondii* in sows and finishing pigs is associated with increased exposure to cats, increased exposure to rodents, housing hogs outside, and smaller herd size.

MATERIALS AND METHODS

Cross-sectional survey

Farm selection: The sampling frame was comprised of the 179 swine farms sampled for a survey of the seroprevalence of antibodies to *T. gondii* (Weigel et al., 1995). Sample selection occurred from January to August 1992 at the Illinois Animal Disease Laboratories (Galesburg and Centralia, Illinois), from farms that submitted blood samples for the

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state pseudorabies testing program. An introductory letter describing the purpose of the project was sent to each producer in the serological survey. A subsequent attempt was made to contact by telephone the owners or managers of each of the herds in order to conduct an interview on farm characteristics and management practices believed to be relevant for ascertaining risk for *T. gondii* transmission. Consent for conducting the telephone interview was obtained for 140 herds (a 78% success rate). Interviews were conducted from February to September 1992.

Interview: Upon achieving telephone contact, the producer was given the results of the serological survey for this farm. A standardized interview was then conducted in which questions were asked on herd demographics, type of housing, the method of feed storage and delivery, the method of water delivery, the method of dead pig disposal, the presence of rodents and other wildlife on the farm, and the presence of cats inside and outside swine facilities.

Serological testing: Sera collected for the serological survey were forwarded to the Parasite Biology and Epidemiology Laboratory of the USDA Agricultural Research Service in Beltsville, Maryland. Detection of antibodies to *T. gondii* was conducted as described in the companion paper (Dubey et al., 1995).

Statistical methods: The unit of analysis was the farm. The pseudorabies testing involves mostly sows. Therefore, the outcome analyzed was the seroprevalence of antibodies to *T. gondii* in sows. A farm was included in the analysis only if there were test results for at least 30 sows. This sample size provides a 95% probability of detecting seropositivity in a herd if the true seroprevalence is at least 10% (Beal, 1983). Of the 140 farms interviewed, 123 met this criterion and thus were included in the analysis. The predictors analyzed for their association with *T. gondii* seroprevalence were: (1) sows housed outdoors (for farrowing or gestation), (2) cat access to sows (either allowed inside or, when hogs are kept outside, cats seen outside), (3) number of sows in the herd, (4) feed delivery in open bins or storage in open-sided buildings, (5) water delivery in gestation or farrowing areas in open troughs or bowls, (6) time until removal of swine carcasses, (7) presence of mice on the farm, and (8) presence of rats on the farm.

The bivariate association of sow *T. gondii* seroprevalence with the dichotomous predictors (all except 3 and 6, above) was analyzed using the Mann–Whitney *U*-test (Siegel, 1956). A nonparametric test was used in these comparisons because of the positive skewness in the distribution of *T. gondii* seroprevalence values. For the interval level variables (number of sows, time to removal of swine carcasses), the bivariate association with swine seroprevalence was examined using Pearson's linear correlation coefficient. The independent association of *T. gondii* seroprevalence in sows with potential risk factors was analyzed using a multiple linear regression model. Variable selection was by initial forced entry of the selected predictors, with backward elimination of nonsignificant variables ($P \geq 0.05$). In all statistical analyses, one-tailed probabilities were calculated, reflecting the testing of directional hypotheses.

Field investigation

Methods: The methods of farm selection, sample collection, and serologic testing were described in the companion paper (Dubey et al., 1995). For the 10 farms sampled in 1992 that were not part of the cross-sectional survey (above), the same telephone interview of risk factors was conducted.

Statistical analysis: The risk factors for swine *T. gondii* seroprevalence analyzed were exposure to cats, exposure to rodents, outdoor housing, and herd size. Risk of exposure to cats was indicated by one of the following: cat access to swine pens or lots, number of cats trapped on the farm, *T. gondii* seroprevalence in cats, number of cats seropositive for *T. gondii*, or detection of oocysts on the farm. If finishing pigs were maintained outside, or if cats were allowed access to indoor finishing areas, then cat access to finishing stages was present. If sows were housed outside, or if cats were allowed access to indoor sows areas, then cat access to sow facilities was present. Evidence from previous studies (Dubey and Beattie, 1988; Dubey et al., 1995) has indicated that cats acquire *T. gondii* infection primarily during the juvenile period, at which time they are most likely to shed oocysts. Therefore, the cat trapping variables were examined for all cats on the farm and separately for juvenile cats only. There was detection of oocysts on the farm when oocysts were isolated from cat feces, pig feed, soil, or water samples taken from the farm. Risk of exposure to rodents was indicated by 1 of the following: number of house mice (*Mus musculus*) trapped on the farm, *T. gondii* seroprevalence in house mice, number of house mice seropositive for *T. gondii*, isolation of *T. gondii* from any rodents on the farm, or evidence of *T. gondii* infection in any rodents on the farm. There was isolation of *T. gondii* from rodents when bioassay in mice recovered *T. gondii* from samples of rodent (*Mus, Peromyscus,* or rat) heart and brain tissues (Dubey et al., 1995) collected on the farm. Evidence for *T. gondii* infection in rodents was indicated by either successful bioassay or positive serology. Outdoor housing of finishing pigs was present when any finishing stage (farrowing, nursery, grower, finisher) was housed outside. Outdoor housing of sows occurred when either the gestation or farrowing stage was housed outside. Herd size was represented by either the number of finishing pigs marketed per year, or the number of sows in the herd, depending upon the class of swine analyzed. Herd size data were obtained from the risk factor interview.

The bivariate association of *T. gondii* seroprevalence with the dichotomous risk factor variables (swine housed outside, cat access to swine, oocysts detected on farm, isolation from rodents, and evidence of infection in rodents) was analyzed by comparing the distribution of seroprevalences when the factor was present and absent using the Mann–Whitney *U*-test (Siegel, 1956). For the interval level risk factor variables (cat and mouse numbers, seropositives, seroprevalences), the bivariate association with swine seroprevalence was examined using Pearson's linear correlation coefficient. The linear correlation of each categorical risk factor with *T. gondii* seroprevalence (point biserial correlation coefficient [Cohen and Cohen, 1983]) also was calculated for decisions regarding selection of variables representing each risk factor in the subsequent multivariate analysis.

The independent association of *T. gondii* seroprevalence in swine with potential risk factors was analyzed using a multiple linear regression model. The unit of analysis was the farm. Separate analyses were conducted for finishing pigs and sows. The basic regression model was the following:

$$\hat{Y} = \beta_0 + \beta_1[\text{cat exposure}] + \beta_2[\text{rodent exposure}] + \beta_3[\text{outdoor housing}] + \beta_4[\text{herd size}]$$

where $\hat{Y}$ = predicted *T. gondii* seroprevalence for sows or finishing pigs on a farm.

The screening of the cat and mice exposure variables for inclusion in the initial regression model proceeded as follows. The cat exposure variables—cat access to swine facilities, number of cats trapped, number of seropositive cats, *T. gondii* seroprevalence in cats, number of juvenile cats trapped, number of seropositive juvenile cats, *T. gondii* seroprevalence in juvenile cats, and detection of oocysts on the farm—were each examined for their bivariate linear correlation with *T. gondii* seroprevalence in sows and finishing pigs. Likewise, the rodent exposure variables—number of house mice trapped, number of seropositive house mice, *T. gondii* seroprevalence in house mice, isolation of *T. gondii* from any rodent captured on the farm, and evidence for *T. gondii* infection in rodents on the farm—were also each examined for their bivariate linear correlation with *T. gondii* seroprevalence in sows and finishing pigs. In each case, the cat or mouse exposure variable with the highest correlation with *T. gondii* seroprevalence was retained for the multiple regression analysis. These cat and rodent exposure variables were included with outdoor housing and herd size in the initial regression model. Thereafter the analysis proceeded with backward stepwise elimination of nonsignificant variables ($P \geq 0.05$). In all statistical analyses, one-tailed probabilities were calculated, reflecting the testing of directional hypotheses.

RESULTS

Cross-sectional survey

The mean sow *T. gondii* seroprevalence among the 123 farms sampled was 19.5% (median = 10.0%). Farm characteristics with respect to analytic variables were the following: 87 (70.7%) of the farms had sows housed outside in either farrowing or gestation; 34 (27.6%) had feed storage in open bins or open-

TABLE I. Results for the final regression model evaluating risk factors for the percentage of sows on a farm that were seropositive in the cross-sectional survey.

Model variable	Regression coefficient	Proportion of variance*	t value	1-tailed P value
Cat access to sows	0.12	0.045	2.40	0.009
Number of sows in herd	−0.000154	0.022	−1.65	0.05

* Squared semipartial correlation coefficient. Multiple $R^2 = 0.231$; $F(2,43) = 6.45$; $P = 0.0036$.

sided buildings; 40 (32.5%) had water delivery in open troughs or bowls; 73 (59.3%) identified mice and 34 (27.6%) identified rats as being present on the farm; 90 (73.2%) had facilities where cats had access to sow housing. The median time until disposal of swine carcasses was 12 hr. The mean number of sows in the herd was 247 (median = 190).

The only risk factor having a significant association with sow seroprevalence in the bivariate analysis was cat access ($P = 0.03$, Mann–Whitney U-test), with a median sow seroprevalence of 10.6% when cats had access to swine and 6.7% when access was not present. In the multiple regression analysis (Table I), *T. gondii* seroprevalence in sows was higher when cats had access to swine ($P = 0.009$) and decreased with more sows in the herd ($P = 0.05$).

Field investigation

Characteristics of the farms studied are displayed in Table II. Most had farrow-to-finish production. Most of the farms had swine housed outside at least some of the time, with sows more likely to be housed outside than finishing pigs. The herds visited in 1993 were more likely to have had swine housed outside. Cats had access to swine on most farms. The herds visited in 1992 tended to be larger (number of sows: mean = 446 [1992] vs. 253 [1993], median = 300 [1992] vs. 160 [1993]; number of finishing pigs marketed/yr: mean = 7,133 [1992] vs. 3,213 [1993], median = 6,000 [1992] vs. 2,350 [1993]). The mean *T. gondii* seroprevalence among farms was 2.3% for finishing pigs (1.8% in 1992, 2.7% in 1993) and 15.3% for sows (14.7% in 1992, 15.9% in 1993).

TABLE II. Characteristics of farms in the field investigation.

Herd characteristics	1992 n	1992 %	1993 n	1993 %
Farrow-to-finish	19	82.6	23	95.8
Type of housing:				
Total confinement	9	39.1	6	25.1
Partial confinement	12	52.2	15	62.5
Pasture	2	8.7	2	8.3
Sows outside	12	52.2	19	79.2
Finishing stages outside	9	40.9	14	58.3
Cat access to sows	18	78.3	17	70.8
Cat access to finishing stages	18	78.3	16	66.7

Table III shows the bivariate association of each categorical risk factor with *T. gondii* seroprevalence, as determined from the Mann-Whitney U-test. Detection of oocysts on the farm was associated with higher seroprevalence in swine, with the risk more apparent for sows ($P < 0.01$) than finishing pigs ($P = 0.02$). Evidence for higher risk of *T. gondii* infection with cat access to swine was equivocal. The general indicators of *T. gondii* infection in rodents by isolation alone or by isolation and serology together were not associated with increased risk of swine infection. Housing swine outdoors was also not associated with increased *T. gondii* seroprevalence in swine.

Table IV presents the linear correlations of *T. gondii* seroprevalence with the risk factor variables. For the quantitative variables, *T. gondii* seroprevalence in both finishing pigs and sows was correlated with the number of cats trapped, the number of seropositive cats, the number of juvenile cats trapped, the number of seropositive juvenile cats, and the house mouse seroprevalence.

Examination of the linear correlations in Table IV led to the selection of variables for the multiple regression analyses. For both finishing pigs and sows, the cat exposure variable with the highest linear correlation with *T. gondii* seroprevalence in swine was the number of seropositive juvenile cats trapped, and the rodent exposure variable with the highest linear correlation was the house mouse seroprevalence. These variables were retained for regression modeling, along with housing swine outside and herd size.

In the multiple regression analysis for finishing pigs, the risk factors associated with an increased *T. gondii* seroprevalence (Table V) were more seropositive juvenile cats on the farm ($P < 0.0001$) and higher *T. gondii* seroprevalence in house mice ($P = 0.0023$). In the multiple regression analysis for sows, the only risk factor associated with an increased *T. gondii* seroprevalence was more seropositive juvenile cats on the farm ($r^2 = 0.21$; $\beta = 0.045$, $P < 0.001$).

DISCUSSION

The role of the cat in increasing the risk of *T. gondii* infection for swine has been confirmed by the studies reported here. In the larger cross-sectional survey, cat access to sow housing was identified as increasing the risk of *T. gondii* infection. In the intensive field investigation where cat infection with *T. gondii* was determined by serologic diagnosis and detection of oocysts, a more direct link with transmission was implicated by the higher seroprevalence for both finishing pigs and sows when more seropositive cats were trapped or when oocysts were detected on the farm. The risk of swine infection with *T. gondii* was most apparent when there was an increase in the number of seropositive juvenile cats on the farm. The previous study (Dubey et al., 1995) indicated that most cats acquire *T. gondii* infection as juveniles, and thus these recently infected cats are most likely to shed oocysts.

Because transmission of *T. gondii* from cats to swine without an intermediate host occurs only via oocysts, presence of oocysts on the farm alone is responsible for the risk of transmission. The association between detection of oocysts on the farm and swine seroprevalence, despite the difficulty in detecting them, suggests that the true association is stronger than identified here. Possible modes of transmission of oocysts to swine were iden-

TABLE III. Percentage of finishing pigs and sows that were seropositive for *Toxoplasma gondii* (seroprevalence) at each level of the categorical risk factors.

Risk factor	Level	Finishing pigs seroprevalence n	Mean %	Median %	Mann–Whitney test *P* value	Sows seroprevalence n	Mean %	Median %	Mann–Whitney test *P* value
Swine housed outdoors	Yes	23	2.5	1.1	ns	31	14.9	6.8	ns*
	No	24	2.0	1.6		15	16.1	6.7	
Cat access to swine facilities	Yes	34	2.8	1.2	0.05	34	16.5	5.9	ns
	No	13	0.9	0.0		12	11.9	6.7	
Oocysts detected	Yes	6	5.3	3.7	0.02	6	37.1	31.4	<0.01
	No	41	1.8	1.1		40	12.0	4.0	
Isolation from rodents	Yes	8	1.9	0.0	ns	8	15.9	11.8	ns
	No	39	2.3	1.1		38	15.2	6.7	
Infection in rodents	Yes	22	1.1	3.1	ns	22	18.8	9.5	ns
	No	25	1.1	1.5		24	12.1	5.2	

* ns, Not significant.

tified in the companion study (Dubey et al., 1995). Oocysts were detected in pig feed and soil, confirming these sources as a direct mode of transmission of *T. gondii* from cats to swine.

Oocysts are scattered throughout the farm and detection of their presence and abundance is difficult. Therefore, risk of transmission from cats may be estimated more accurately from infection detected by serological testing. The number of seropositive cats (particularly juveniles) is probably a better indicator of exposure risk than is cat seroprevalence because the environmental load of oocysts is more directly determined by the number than the proportion of infected cats.

The cross-sectional study conducted here is in agreement with a cross-sectional survey of 107 swine farms in Tennessee (Assadi-Rad et al., 1995), which identified the presence of cats on the farms as a risk factor for *T. gondii* seropositivity in swine. Seropositivity in cats or shedding of oocysts was not investigated in the Tennessee study. In a study of 20 Iowa swine farms, Smith et al. (1992) were not able to identify a statistical association between *T. gondii* seroprevalence in cats and sows. The small sample size limited identification of these relationships, had they existed. Nevertheless, due to high seroprevalence, it was suggested that cats were involved in the transmission of *T. gondii* to swine. The current study identifies presence of oocysts on the farm and the number of seropositive cats on the farm as factors associated with an increased risk of infection in swine, rather than cat seroprevalence, which in the current study was

TABLE IV. Linear correlation with swine *Toxoplasma gondii* seroprevalence for risk factor variables evaluated for inclusion in the multiple regression analyses.*

Risk factor variable	Linear correlation with: Finishing pig seroprevalence *r*	*P*	Sow seroprevalence *r*	*P*
Cat exposure variables				
Number of cats trapped	0.40	0.006	0.31	0.04
Number of seropositive cats	0.45	0.002	0.40	0.006
Cat *T. gondii* seroprevalence	0.05	0.75	0.23	0.13
Number of juvenile cats trapped	0.47	<0.001	0.31	0.03
Number of seropositive juvenile cats	0.66	<0.001	0.46	0.002
Juvenile cat *T. gondii* seroprevalence	0.36	0.07	0.30	0.15
Oocysts detected on the farm[a]	0.30	0.04	0.45	0.02
Cat access to finishing pig/sow housing[a]	0.22	0.13	0.11	0.48
Rodent exposure variables				
Number of house mice trapped	−0.27	0.07	−0.18	0.23
Number of seropositive house mice	0.07	0.65	0.08	0.61
House mouse *T. gondii* seroprevalence	0.58	<0.001	0.29	0.05
Isolation of *T. gondii* from rodents[a]	−0.04	0.81	0.02	0.92
Evidence of *T. gondii* infection in rodents[a]	0.20	0.18	0.17	0.25
Finishing pigs/sows housed outside[a]	0.07	0.63	−0.03	0.85
Number of finishing pigs/sows in herd	−0.22	0.13	−0.23	0.13

* All correlation values are Pearson's correlation coefficients, except for variables marked with '[a]', where correlations are point biserial correlation coefficients.

TABLE V. Results for the final regression model evaluating risk factors for the percentage of finishing pigs on a farm that were seropositive in the field investigation.

Model variable	Regression coefficient	Proportion of variance*	*t* value	1-tailed *P* value
Number of seropositive juvenile cats on the farm	0.010	0.201	4.34	<0.0001
Seroprevalence in house mice	0.342	0.095	2.99	0.0023

* Squared semipartial correlation coefficient. Multiple $R^2 = 0.532$; $F(2,44) = 24.96$; $P < 0.0001$.

also not correlated with swine seroprevalence. As discussed above, number of seropositive cats is probably a better indicator of *T. gondii* exposure risk than is cat seroprevalence.

The field investigation conducted here also identifies an increased risk of *T. gondii* infection in swine due to exposure to rodents. This is consistent with the previous conclusions of Lubroth et al. (1983) and Assadi-Rad et al. (1995). In the current study, the increased risk of *T. gondii* infection due to rodent exposure was identified clearly for finishing pigs, with higher seroprevalence in mice (9.5% of variance accounted for) most clearly indicating this increased risk. It is not clear why house mouse seroprevalence is a better indicator of exposure risk than is the number of infected house mice. Although the simple correlation of sow seroprevalence with house mouse seroprevalence indicated rodent exposure as a risk for sows, in the multiple regression analysis the independent effect of rodent exposure was not apparent. The risk of infection due to rodent exposure may not be as apparent in sows because their seropositivity may reflect past exposure, prior to the time of infection of the current generation of mice.

Although the current study identifies house mouse *T. gondii* seroprevalence as a predictor of infection in swine, it remains to be demonstrated whether direct transmission from mice to swine via consumption of infected tissue is common, or whether rodents serve primarily as a reservoir for infection of previously uninfected cats. Nevertheless, the possibility of direct transmission of *T. gondii* from rodents to swine cannot be ruled out and given the observed association should be investigated further. Although the current study found a risk of swine infection due only to house mouse infection, the small sample sizes for rats and white-footed mice precluded identifying these rodents as involved in transmission risk. However, the observation that 47% of the farms had identified *T. gondii* infection in rodents demonstrates that rodents are a reservoir for infection and have the potential for direct transmission of *T. gondii* to swine. Even if direct transmission from rodents to swine is rare, a rodent reservoir of infection increases the risk of cat exposure to *T. gondii*. As discussed in the companion paper (Dubey et al., 1995), the percentage of farms with rodent reservoirs of infection is probably underestimated.

Housing swine outdoors was not identified as increasing their risk of exposure to *T. gondii* in either the cross-sectional survey or the field investigation. This contradicts the conclusions of previous studies (Lubroth et al., 1983; Zimmerman et al., 1990; Smith et al., 1992; Assadi-Rad et al., 1995), which suggest that confinement protects swine from exposure. A closer examination of their analyses indicates their conclusions were not fully supported by their data. The study of Lubroth et al. (1983) compared only 2 herds and, therefore, did not take sampling variation into account. Zimmerman et al. (1990) excluded noninfected farms and partial confinement herds in their analysis; thus, sampling bias may have been introduced. The statistical associations identified were not strong, i.e., they found no association with housing type for sows and found only a trend ($P = 0.09$) toward lower seroprevalence in confined finishing pigs. Smith et al. (1992) compared the seroprevalence of sows housed in total confinement with sows not housed in total confinement, finding a lower seroprevalence in the confined group. This analysis also excluded partial confinement herds. Assadi-Rad et al. (1995) did pairwise comparisons of seropositivity rates among sows housed in total and partial confinement and on pasture, finding the greatest difference in risk between sows with outdoor access versus sows in total confinement. Hogs are grouped on farms, and thus in using hogs (rather than farms) as the unit of analysis, the Iowa and Tennessee studies ignored unidentified farm effects; their analyses also violate the assumption of independence of observations in tests of differences between proportions (Hays, 1988). Using hogs as the unit of analysis also artificially increases sample size and inflates statistical power, allowing weak associations to be statistically significant. In the current cross-sectional and field investigations, the unit of analysis was the farm, and farms with all seroprevalence levels and housing types were examined. These analyses indicate that total confinement does not significantly reduce risk of swine exposure to *T. gondii*. Swine housed in total confinement may still be exposed to *T. gondii* because cats are either kept indoors intentionally to reduce the rodent population, or uninvited cats enter the facilities through open windows and doors. The results of the cross-sectional survey indicate that cat access to swine is more important than housing type. Even if cats do not have access to swine, feed contaminated previously with oocysts can be brought inside, and humans can track in oocysts on their shoes. In addition, infected house mice can serve as a source of infection for swine. On the other hand, housing swine outdoors does not guarantee exposure to *T. gondii*. Cat or rodent reservoirs of infection are prerequisites for transmission to swine. In the current study, housing swine outdoors was not correlated with either the number of seropositive cats or seroprevalence in mice, the 2 more direct risk exposures identified. Perhaps different associations exist between exposure to *T. gondii* and outdoor housing in other geographic regions.

The cross-sectional survey identified a weak association indicating that small herd size increases the risk of *T. gondii* exposure in swine. These results are in agreement with the findings of Zimmerman et al. (1990) and Assadi-Rad et al. (1995). However, it is not clear what management factors associated with smaller herd size increase the risk of *T. gondii* infection for swine. The producer interview investigated feed storage and

delivery, and water delivery, as well as disposal of dead swine, to assess their role in affecting risk of *T. gondii* transmission. None of these factors were associated with either *T. gondii* seroprevalence in swine or herd size. However, a post-hoc analysis suggests that small herd size is associated with higher *T. gondii* seroprevalence in swine because the risk of *T. gondii* exposure per hog is greater. In the field investigation, the estimated *T. gondii* exposure from cats was independent of herd size (no correlation found with the number of seropositive cats, either including all ages or juveniles only). However, if the number of seropositive cats was divided by a measure of herd size (number of sows + number of finishing pigs marketed per year) to create a ratio indicating the seropositive cat exposure rate per hog, there was a strong positive correlation between this ratio and seroprevalence in both finishing pigs ($r = 0.49$, $P = 0.0005$ for all cats; $r = 0.73$, $P < 0.0001$ for juvenile cats) and sows ($r = 0.34$, $P = 0.022$ for all cats; $r = 0.35$, $P = 0.019$ for juvenile cats). Thus, swine on smaller farms may be at increased risk of *T. gondii* infection primarily because the cat exposure risk, which is independent of herd size, is distributed over fewer hogs.

The identification of exposure to cats and rodents as factors increasing the risk of *T. gondii* infection for swine suggests targets for interventions designed to reduce swine exposure. Swine exposure to oocysts needs to be reduced by prevention of cat access to swine housing and feed. With these interventions it would be appropriate to target juvenile cats. Preventing female cats from raising litters in swine barns or obtaining only neutered adult cats for rodent control could be first simple steps toward decreasing *T. gondii* exposure risk in swine. Rodent control as a means of reducing *T. gondii* transmission to swine should also be considered.

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