RESEARCH REPORT
PORK SAFETY

Title: *Toxoplasma gondii I*nfection in Sows and Market-Weight Pigs in the United States and its Potential Impact on Consumer demand for Pork
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Abstract

The objectives of this research were to assess and compare the prevalence of *Toxoplasma gondii* in sows and market-weight pigs (finisher pigs) in the United States using serum collected from sows in 1990, 1995, and 2000 and from finisher pigs in 1995 and 2000. Serum for this study was obtained from a survey of swine herds conducted by the National Animal Health Monitoring System (NAHMS). Participating herds were from 17 of the major pork producing states: Arkansas, Colorado, Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Carolina, Ohio, Oklahoma, Pennsylvania, South Dakota, Texas, and Wisconsin. Serum samples from 505 herds were analyzed for *T. gondii* antibodies by the modified agglutination test (MAT) using formalin fixed tachyzoites as antigen. Titers of 32 or greater were considered positive. A positive titer means that the pig was infected with *T. gondii* at some time during its life and that it has *T. gondii* cysts in the muscles and/or organs. Positive animals were in 16/17 states. Of swine tested, 536/13,835 (4 %) were positive. Sows in the study (487/8086, 6 %) were 7 times more likely to be infected than finishers (49/5720, 0.9%). The prevalence in sows decreased between 1990 and 2000. Sows in 1990 were 4 times and sows in 1995 were 3 times more likely to be infected than sows in 2000. The prevalence in finishers decreased from 3.2 % to 0.9% between 1995 and than in 2000 with finishers in 1995, 4 times more likely to be infected. Of 505 herds, 25 % had at least one positive animal: sow herds were 10 times more likely to harbor a positive animal than finisher herds although the percentage of positive sow herds decreased from 49% in 1990 to 30 % in 2000. The percentage of positive finisher herds decreased also with herds in 1995, 3 times more likely to be infected than herds in 2000.

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Introduction

Based on national serologic data collected between 1988 and 1994 National Health and Nutritional Assessment Survey (NHANES III), approximately 22% of the adult population in the United States has had toxoplasmosis, a disease caused by the parasite *Toxoplasma gondii*.[1,2] The natural route of transmission is from animals and contaminated soil to humans by way of ingestion.[3-5] People, other mammals, and birds are usually infected with *T. gondii* by eating (1) tissue cysts in undercooked meat, (2) oocysts (eggs) from cat feces during gardening or other activities involving soil contact, or (3) food and water contaminated with oocysts. Animals develop immunity to additional infection as cysts form in their tissues after infection. These cysts remain alive in the body for the lifetime of the animal, and, in undercooked meat, the cysts will infect animals that eat them. Sheep, goats, pigs, chickens, and wild game all may have infective *T. gondii* cyst in edible portions of meat.[3-5] There is some evidence that handling raw pork or eating undercooked pork is a common route of infection for people in the United States. It is assumed by many researchers that 50% of all toxoplasmosis is foodborne.[6,7] In one study, Seventh Day Adventists had a significantly decreased risk of infection compared with non-Seventh Day Adventist. While the basis for this decrease was not determined, one possible protective factor is their general adherence to a meatless diet.[8]

If a woman is infected with *T. gondii* for the first time during pregnancy, spontaneous abortion or infection of the fetus may occur. Infant mortality may be as high as 12%, and 30% will suffer severe birth defects, including mental retardation.[4,5] It is estimated that 4100 of the 4.1 million babies born annually in the United States have congenital infection. The estimated human illness costs of congenital toxoplasmosis is about $5.4 billion annually, with one half of that being food related.[4,5]

Most postnatal cases of toxoplasmosis are mild with only a flu-like illness for several days before immunity occurs.[2-5] The disease can be reactivated in immunosuppressed people, and result in severe illness and death. Toxoplasmic encephalitis with the parasite multiplying in the brain is the second most common AIDS-related opportunistic infection of the central nervous system. The cost of treating toxoplasmosis in AIDS-patients in the United States ranges between 23 and 106 million dollars each year. [6-8] The Centers for Disease Control.[1] listed *Toxoplasma gondii* as one of three major pathogens responsible for foodborne illnesses and death (21% of total) with an estimated 12,100 people developing chronic disease due to toxoplasmosis. There is a need to explore the accuracy of these estimates and to assess the relationship between meat consumption and *T. gondii* infection. Despite its widespread prevalence, the relative contributions of various routes of infection are unknown.

Over the past 15 years we, and other researchers, have conducted seroprevalence surveys of *T. gondii* antibodies in breeding hogs and market-weight pigs throughout the country.[10-20] Prevalence has ranged from 20-42% in sows and from 3-23% in market-weight pigs. In 1996, we reported an apparent decline in the number of sows seropositive for *T. gondii* antibodies in the US based on titers of sera collected in 1990 as a part of the NAHMS survey of 412 randomly selected swine herds in 17 states. [12,13] Using the modified agglutination test (MAT) 679/3472 (20%) of the hogs tested were positive for *T. gondii* IgG antibodies. A previous national seroprevalence survey of sows in 1983-84 reported 42% positive using the MAT. [18] We analyzed blood samples collected in the 1995 NAHMS and documented a continued decrease in sows.[13-15] Samples tested were from 285 farms and 16 states. Of the total swine tested, 8% (644/7979) were positive for T. gondii IgG; 15% (488/3236) of sows were positive and 3.2% (153/4712) of finisher pigs were positive. Prevalence in sows was significantly higher than the market-weight pigs. In fact, sows were 5 times more likely

to be infected than finisher pigs. The prevalence of *T. gondii* in sows decreased between 1990 and 1995. Sows in 1990 were 1.4 times more likely to be infected than sows in 1995.

Roberts[6] indicated that farm-level interventions are the most effective control strategies for parasites that originate on the farm, because parasites, unlike bacteria, do not multiply in food products once the animal is slaughtered. Over the past 10 years, our investigations on farms in Tennessee[11] and Iowa[10] and other investigations in Illinois[19,20] have studied the epidemiology of *T. gondii* and the risk factors associated with transmission of *T. gondii* in swine herds. In Tennessee,[11] we found that sows associated with cats were 2. 6 times more likely to be infected than sows not associated with cats; sows raised on small farms were 4.5 times more likely to be infected than sows raised on large farms; sows kept outdoors were 23 times more likely to be infected than sows raised indoors. In the two studies funded in part by NPPC,[12-15,] we also measured the farm management relationships associated with transmission of *T. gondii* in sows in the 1990,1995 NAHMS survey and finishers in the 1995 survey. The results were similar to the TN study with fewer infected sows and pigs in herds that (1) were raised in total confinement operations, (2) excluded cats and used bait for rodent control, and (3) denied cats, dogs, or birds access to the facilities. Two of our earlier studies[11,16] also indicated that the percentage of market-weight pigs infected with *T. gondii* is considerably lower than sows and lower than the 23% previously reported[18] in a survey of market-weight pigs at slaughter.

The purpose of this research was to compare the seroprevalence of *T. gondii* in sows and market-weight pigs in the NAHMS survey in 2000 to the 1995 survey, and possibly document a continued low percentage of infected market-weight pigs. This is particularly important because the majority of the fresh commercial cuts of pork are derived from market animals rather than breeding swine. *Toxoplasma gondii* has been isolated from fresh pork tissues used for human consumption;[23] however, the prevalence in the finishing swine in our previous studies was very low.

Objectives

1. Compare the prevalence of *Toxoplasma gondii* in sows to that of market weight pigs sampled for NAHMS 2000
2. Assess any change in prevalence of *T. gondii* in the United States by comparing:

–Sows tested in 2000 with those tested previously in the 1990 and 1995 NAHMS Surveys

–Market weight pigs tested in 2000 with those tested in the 1995 NAHMS Survey

Procedures

Serum samples were obtained from swine participating in the National Animal Health Monitoring System in 2000. NAHMS Swine 2000 was designed to provide both participants and the industry with information on approximately 94 % of the United States swine herds on operations with 100 or more pigs.[22] Participating herds were from 17 of the major pork producing states: Arkansas, Colorado, Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Carolina, Ohio, Oklahoma, Pennsylvania, South Dakota, Texas, and Wisconsin.[22] Blood was collected from sows and finishers with approximately 15 samples/herd collected from finishers and 30 samples/herd from sows.

Serum was frozen, shipped to the University of Tennessee, College of Veterinary Medicine and there tested for *Toxoplasma gondii* antibodies with the MAT using formalin-fixed tachyzoites as antigen as described previously.[11] Numerous studies have demonstrated MAT to be the most sensitive and specific test for detecting *T. gondii* antibodies in animals.[21] MAT was used for the 1990 and 1995 NAHMS samples and as the serologic test of most recent investigators. Titers of greater than or equal to

32 were considered positive. A herd was considered positive if one animal tested positive for antibodies against *T. gondii.* A positive titer means that the pig was infected with *T. gondii* at some time during its life and that it has *T. gondii* cysts in the muscles and/or organs. The prevalence of *T. gondii* in sows and market-weight pigs was calculated and compared to the results of the 1990 and 1995 studies. The hypotheses were tested using standard statistical techniques appropriate for nominal and ordinal scale data. Serum from all pigs sampled was not included in the sample set received at the University of Tennessee. Also some samples were of poor quality and could not be assayed. Serologic test data were stored in MS Excel Datasets. Descriptive data analysis was done with Epi Info 2000, and MS Access. Statistical analysis of selected variables was based on *Chi-Square* and *Odds Ratio* Methods (GraphPad Instat Software).

Results and Discussion.

Serum samples from 505 swineherds were available for analysis. A total of 14,359 tubes were received, and 13,835 were suitable for analysis: 8086/8461 samples from breeders (sows/gilts), 5720/5868 from finisher pigs, and 29/30 from animals of unknown age/status. As shown in Table 1, 3.9% of the swine tested were positive (536/13,835) for *T. gondii* IgG antibodies: 6.02 % of the sows and 0.86 % of finishers. The prevalence in sows was significantly greater than in finisher pigs. Sows in the study were 7 times more likely to be infected than finishers (odds ratio 7.417 derived from Chi-square analysis).

The prevalence of *T. gondii* in sows decreased between 1990 and 2000 (Table 2). Sows in 1990 were 1.4 times more likely to be infected than sows in 1995 (odds ratio 1.37 derived from Chi-square analysis); however, sows in 1990 were 4 times more likely to be infected than sows in 2000 (odds ratio 3.784 derived from Chi-square analysis), and sows in 1995 were 3 times more likely to positive than sows tested in 2000 (odds ratio 2.771). The prevalence of *T. gondii* in finishers also decreased between 1995 and 2000 (Table 3). Finishers in 1995 were 4 times more likely to be infected than those in 2000 (odds ratio 3.884 derived from Chi-square analysis).

Positive animals were present in 16 of the 17 states included in the study. None of the animals tested in Colorado were positive. In the other 16 states prevalence ranged from 14% in Wisconsin to less than 1% in North Carolina (0.26%). No finisher pigs were tested in Arkansas and Oklahoma, but positive finishers were found in 9/15 states tested: Illinois, Iowa, Kansas, Minnesota, Missouri, Nebraska, Ohio, Pennsylvania, and Wisconsin with prevalence ranging from 0.12% in Iowa to 7.41% in Wisconsin. Prevalence in sows ranged from no positives in Colorado to 13.62% in Iowa and 18.23% in Wisconsin. (Table 4). Prevalence differences between states is probably more related to husbandry practices than the environment. For example, in North Carolina large finisher operations feed pelleted feed that has little chance of contamination by cats. Other states feed corn that is at times stored in areas exposed to the outside and possible contamination by *T. gondii* oocysts. Our previous studies[10-16] have shown that large confinement operations, good biosecurity, traps and baits for rodent control instead of cats all contribute to decreased *T. gondii* infection rates.

Using herds as the unit of analysis, 25% (128/505) had a least one positive animal in the herd. The percentage of positive herds changes when sows and finishers are examined separately. In finisher operations, 4% (16/403) had at least one positive finisher pig in the herd, and in sow herds, 30% (118/392) had at least one positive sow (Tables 5). There were significantly more positive sow herds than positive finisher herd. Sow herds were 10 times more likely to have a positive animal than finisher herds (odds ratio 10.417 derived from Chi-square analysis) (Table 8).

In the 1990 NAHMS study, 49% of the sow herds had at least one positive sow

as compared to 56% of the herds in 1995 (Table 6). This difference was not statistically significant; however, there was a significant decrease in the number of positive sow herds in 2000 (Table 8). Sow herds in 1990 were twice as likely to include an infected animal than herds in 2000 (odds ratio 2.255 derived from Chi-square analysis), and sow herds in 1995 were 3 times more likely to be infected than herds in 2000 (odds ratio 2.926, Table 8). A similar decrease was seen in the finisher herds (Table 7). Finisher herds in 1995 were six times more likely to include an infected animal than herds in 2000 (odds ratio 5.598, Table 8).

Table 1. Seroprevalence of *Toxoplasma gondii* in Swine in the National Animal Health Monitoring System – 2000

Pig Type	Number Positive	Number Tested	Percent Positive
Sows	487	8086	6.0%
Finishers	49	5720	0.9%
Unknown	0	29	0.0%
Total	**536**	**13,835**	**3.9 %**

Table 2. Seroprevalence of *Toxoplasma gondii* in Sows in the National Animal Health Monitoring System: 1990, 1995, 2000

Year	Number Positive	Number Tested	Percent Positive
Sows 2000	487	8086	6%
Sows 1995	488	3236	15%
Sows 1990	679	3479	20%

Table 3. Seroprevalence of *Toxoplasma gondii* in Finishers in the National Animal Health Monitoring System: 1995, 2000

Year	Number Positive	Number Tested	Percent Positive
Finishers 2000	49	5720	0.9%
Finishers 1995	153	4712	3.2%

Table 4. Seroprevalence of *Toxoplasma gondii* in Sows* and Finishing Pigs* in the National Animal Health Monitoring System, 2000, by State

State	Number of Positive Sows/Total Number of Sows (% +)		Number of Positive Finishers/ Total Number of Finishers* (% positive)		Number of Positive Animals/Total Tested*** (% Positive)	
Arkansas	12/920	(01.3%)	0/00	(0.0%)	12/920	(01.3 %)
Colorado	0/123	(00.0%)	0/59	(0.0%)	0/182	(00.0%)
Illinois	43/642	(06.7%)	4/594	(0.7%)	47/1236	(03.8%)
Indiana	44/748	(05.9%)	0/493	(0.0%)	44/1241	(03.6%)
Iowa	126/925	(13.6%)	1/804	(0.1%)	127/1729	(07.4%)
Kansas	13/311	(04.2%)	6/276	(2.2%)	19/587	(03.2%)
Michigan	50/589	(08.5%)	0/269	(0.0%)	50/858	(05.8%)
Minnesota	16/418	(03.8%)	2/634	(0.3%)	18/1066**	(01.7%)
Missouri	16/668	(02.4%)	2/306	(0.7%)	18/974	(01.9%)
Nebraska	28/689	(04.1%)	10/565	(1.8%)	38/1254	(03.0%)
North Carolina	2/268	(00.8%)	0/490	(0.0%)	2/758	(00.3%)
Ohio	28/593	(04.7%)	4/368	(1.1%)	32/961	(03.3%)
Oklahoma	4/137	(02.9%)	0/00	(0.0%)	4/137	(02.9%)
Pennsylvania	20/211	(09.5%)	1/326	(0.3%)	21/537	(03.9%)
South Dakota	6/199	(03.0%)	0/128	(0.0%)	6/327	(01.8%)
Texas	3/111	(02.7%)	0/60	(0.0%)	3/171	(01.8%)
Wisconsin	70/384	(18.2%)	18/243	(7.4%)	88/627	(14.0%)
Total	**481/7936**		**48/5615**		**529/13,565**	

* 1 positive finisher sample had no valid state code

** 29 samples had no age/sex recorded: 14 were from MN

*** 270 animals had no valid state code recorded

Table 5. Herds in the National Animal Health Monitoring System Positive for *Toxoplasma gondii – 2000**

Herd Type	Number Positive	Number Tested	Percent Positive
Sow	118	392	30%
Finisher	16	403	4%

* Many farms have both sow and finisher herds

Table 6. Sow Herds with at Least One Sow Positive for *Toxoplasma gondii* in the National Animal Health Monitoring System - 1990, 1995, 2000

Year	Number Positive	Number Tested	Percent Positive
2000	118	392	30%
1995	126	226	56%
1990	203	412	49%

Table 7. Finisher Herds with at Least One Finisher Positive for *Toxoplasma gondii* in the National Animal Health Monitoring System - 1995, 2000

Year	Number Positive	Number Tested	Percent Positive
2000	16	403	4%
1995	53	282	19%

Table 8. Statistical Analysis of Seroprevalence of *Toxoplasma gondii* in Swine and Swine Herds in the National Animal Health Monitoring System, 2000

Comparison	Toxoplasma Serologic Status		Probability of Non Random Association	Odds Ratio	95 % Confidence Interval	
	Positive	Negative			UpperLimit	Lower Limit
Sows 2000	487	7599	P < 0.01	7.417	9.970	5.518
Finishers 2000	49	5671				
Sows 1990	679	2800	P < 0.01	3.784	4.284	3.342
Sows 2000	487	7599				
Sows 1995	488	2748	P < 0.01	2.771	3.165	2.426
Sows 2000	91	2997				
Finishers 1995	153	4559	P < 0.01	3.8841	5.371	2.809
Finishers 2000	49	5671				
Sow herds 2000	118	274	P < 0.01	10.417	17.960	6.042
Finisher herds 2000	16	387				
Sow herds 1990	203	209	P = 0.7709*	0.7709	1.068	0.5564
Sow herds 1995	126	100	P < 0.01			
Sow herds 1990	203	209	P < 0.01	2.255	3.013	1.688
Sow herds 2000	118	274				
Sow herds 1995	126	100	P < 0.01	2.926	4.110	2.083
Sow herds 2000	118	274				
Finisher herds 1995	53	229	P < 0.01	5.598	10.024	3.126
Finisher herds 2000	16	387				

* Not Statistically significant

Summary

1. Of the sows tested, 6 % tested positive for *Toxoplasma gondii*; 94 % were negative.
2. The prevalence of *T. gondii* in sows decreased between 1990 and 2000. These results show a continuing decline in the prevalence of *T. gondii* on swine farms in the major swine producing states in the United States. In the 1990 NAHMS survey, 20% of the breeding hogs tested were positive for *T. gondii* antibodies;[12-13] 15% were positive in 1995[14,15] and 6 % in 2000. A national seroprevalence survey in 1983-84[18] reported a 42% seroprevalence in breeding hogs. All four surveys used the same method of analysis (MAT).
3. The prevalence in the finisher pigs was 0.9%, with 99.1% of the animals negative for *T. gondii*. This is a significant decrease from 3.2% for finishers in 1995.
4. On the farms tested, 4% of the finisher herds had at least one animal in the herd infected with *T. gondii*. This was a significant decrease from the 19% infected herds in 1995.
5. On the farms tested, 30% of the sow herds had at least one animal in the herd infected with *T. gondii,* a significant decline from 56 and 49% in 1995 and 1990.
6. The prevalence in breeding animals was significantly higher than in finishing swine with sows 7 times more likely to be infected.
7. The prevalence in finisher pigs is less than the 1-3 % seroprevalence in the finishing swine tested from North Carolina and Tennessee in 1995.[11,16] In another study[19,20] 3% of finishing swine in Illinois were seropositive for *T. gondii* antibodies. The 0.9% seroprevalence in finisher pigs is considerably lower than the 6% for hogs. (Breeders are 7 times more likely to be infected.) Perhaps this is related to the age of the animals and the husbandry practices employed on the farms. Sows are older and have had more time to become infected, and are more likely to be exposed to the outside at sometime during their lives than finishers.

Conclusion

This study documents a continuing decline in the number of swine in the United States that are infected with *Toxoplasma gondii*. This decline is likely the result of good husbandry practiced by the swine industry. More breeding animals are infected than finishing pigs. Humans can become infected with *T. gondii* if they eat the tissue stages in poorly cooked meat, including pork. The breeding animals probably do not play a significant role in the transmission of *T. gondii* to humans. These animals are older when sold and are made into processed meats, which are usually highly seasoned, smoked, heated, or frozen. They are not sold as cuts of meat and, therefore, are only a danger to individuals in the processing plant handling the raw meat. The market-weight animals are sold as commercial cuts of pork and Dubey[23] isolated *T. gondii* from pork tissues used for human consumption. However, the prevalence in the finishing swine in this study was 0.9% and in a previous study of 2312 finishing swine on 13 farms in North Carolina, was also less than 1%.

Although this decrease is encouraging, the industry should continue to reduce the number of positive sows and finishing swine. As stated previously, the results of this research directly relate to USDA priority areas of food safety and market development. *Toxoplasma gondii* has the potential to dramatically impact consumer demand for pork. Consumer confidence in the safety of pork is vital to continued and enhanced demand for pork, both domestically and internationally (NPPC Annual Report, 1995). Our data argues against pork consumption as the

chief contributor to human infection with *Toxoplasma gondii* in the United States. Similar studies should be conducted with other consumer products like beef and chicken.

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