

Authors

H. Ray Gamble, USDA/ARS
Sharon Patton, Univ. of TN, Veterinary Medicine

Toxoplasma

Originally published as a National Pork Board/
American Meat Science Association Fact Sheet.

Reviewer

Lawrence E. Miller, USDA/APHIS

Introduction

Toxoplasma gondii is a protozoan (single-celled) parasite found in muscle and other tissues of many warm-blooded animals including pigs and people. Cats and other felids are the only hosts in which the parasite can complete its entire life cycle (Figure 1), and the only animals that excrete the environmentally resistant and infectious stage called the oocyst (*eggs*) in the feces. Infection occurs when pigs, and other animals, accidentally ingest oocysts in soil or water or eat tissues of rodents, wildlife, or meat containing cysts. Ingested oocysts or tissue cysts enter the intestine and release sporozoites or bradyzoites, respectively. These stages penetrate intestinal epithelial cells and transform into rapidly dividing tachyzoites. Tachyzoites are dispersed throughout the body by the circulatory and lymphatic systems, eventually entering and encysting as bradyzoites (tissue cysts) in skeletal muscle and other organs of the body (brain, heart, liver). These cysts remain alive in the body for the lifetime of the animal, and are infective when eaten by other hosts, such as humans. Once tissue cysts have formed, most animals are resistant to a second infection. In the cat, a series of asexual stages in the intestine is followed by sexual reproduction of the parasite with the development of gamonts, fertilization, formation of zygotes, and the production of oocysts that are passed in the feces. Cats may shed more than 10 million oocysts per day for 3-10 days after infection. Oocysts must mature (sporulate) in the environment for 1-5 days to become infective for a new host. Transplacental transmission of infection can occur in some hosts, including humans, who become infected during pregnancy.

Toxoplasmosis in humans

Human infection with *Toxoplasma gondii* is quite high relative to most other diseases. Serological surveys report population infection rates of nearly 100% in some countries. Prevalence rates in France, for example, are reported to range from 42-84% of the population. In the United States, the rate of infection with *T. gondii* appears to be declining. In the most recent serological survey (National Health and Nutrition Examination Survey) involving over 17,000 people, 23% were positive for *T. gondii* antibodies, indicating infection with the parasite. Of the women of child bearing age that were tested, 14% were positive for *T. gondii*. Prevalence of infection was lower in a younger population (military recruits), declining from 14.4% in a 1962 study, to 9.5% in a similar 1989 study.

Exposure of healthy adults to *Toxoplasma gondii* generally results in either an asymptomatic infection or a mild *flu-like* illness. A major health problem associated with *T. gondii* is transmission of the parasite from a pregnant woman to her unborn baby. At risk are women who acquire a primary infection either during,

or shortly before, pregnancy. Women who have previously been exposed to *T. gondii* and who have a healthy immune system, have minimal risk of transmitting the parasite to the fetus. Transplacental infection can result in miscarriage, stillbirth, or live birth with congenital infection. Infant mortality may be as high as 12%, and 30% may have severe birth defects, including mental retardation. Congenital toxoplasmosis may be expressed as either a neonatal disease (in approximately 15% of cases), or appear later during infancy, childhood, adolescence or adulthood (approximately 85% of cases). Typical consequences of neonatal disease include hydrocephalus, intracranial calcification, and chorioretinitis.

Most postnatal cases of toxoplasmosis are mild with only a *flu-like* illness for several days before immunity occurs. The disease can be reactivated in immunosuppressed people, and can result in severe illness and death. Toxoplasmic encephalitis, a disease caused by parasites multiplying in the brain, results from either acute infection or reactivation of a latent infection. It is the second most common opportunistic infection of the central nervous system in AIDS patients. In healthy adults, most infections with *T. gondii* are subclinical; however, acute disease occurs occasionally and includes lymphadenopathy, chorioretinitis, or central nervous system infection. Human toxoplasmosis in the United States is estimated to cost \$5.26 billion annually in medical costs, losses in personal productivity, and costs of special education and residential care. An additional \$100 million is attributed to medical costs of toxoplasmic encephalitis in AIDS cases.

Humans become infected with *T. gondii* in three ways: 1) accidental ingestion of the oocysts excreted by cats in their feces, 2) ingestion of tissue cysts by consumption or improper handling of undercooked or raw meat from infected animals, and 3) congenitally, in the case of previously unexposed women who become infected around and during pregnancy. The oocysts excreted by cats are very resistant to environmental fluctuations and survive for years in soil, even in adverse conditions. Humans can be exposed to oocysts deposited in cat feces through gardening activities, eating unwashed fruits and vegetables, drinking water contaminated with cat feces, cleaning a cat litter box, contact with cat feces in sandboxes, etc. Once infection has occurred, it is not possible, based on tools currently available, to determine whether a person was infected by ingestion of oocysts or by consumption of contaminated and undercooked meat. The Centers for Disease Control estimates that 50% of all human exposures in the U.S. are foodborne and that *T. gondii* is responsible for approximately 20% of all deaths attributed to foodborne pathogens. Of the major meat animal species investigated thus far, pigs are the only species shown to frequently harbor the parasite. Uncooked pork was identified as the source of two outbreaks in healthy adults in Korea resulting in chorioretinitis (3 patients) and lymphadenopathy (5 patients). In a cross-sectional study of adults, a group known to avoid eating meat (Seventh Day Adventists) had a significantly lower prevalence rate of infection (18%) as compared with the non-Seventh Day Adventists in the study (40%); however, cat ownership and association with oocyst-contaminated environments were not investigated.

Toxoplasma and pork

Most species of livestock, including sheep, goats, and pigs, are susceptible to infection with *Toxoplasma gondii*; however, animals exposed to *T. gondii* rarely show signs of infection. Animals are infected in a similar manner to humans: ingestion of oocysts from the environment; consumption of infected animals such as mice, birds, and other wildlife; consumption of undercooked meat scraps; and in some species, through in utero transmission.

Year	Location	# tested	% positive	Comment
1983-1984	National sample	11,229	42	Sows
		613	23	Market hogs
1990	National sample	3,479	20	Sows
1991-1992	Tennessee	3,841	36	Sows
1992	Illinois	5,080	20.8	Breeding swine
		1,885	3.1	Market hogs
1995	National sample	3,236	15	Sows
		4,712	3.2	Finishers
1997-1998	Illinois	3,498	20.7	Sows
	Indiana	3,646	43.2	PRV samples
	New Jersey	1,504	33.1	PRV samples
	Pennsylvania	4,800	0.92	Market hogs
	Iowa	8,326	0.14	Market hogs
2000	National sample	8,086	6	Sows
		5,720	0.9	Market hogs

Table 1. Prevalence of *Toxoplasma gondii* in pigs in the United States.

Prevalence of *Toxoplasma gondii* in pigs varies, but generally exceeds 10-20% in most countries. Infection rates are higher in breeding populations than in market pigs, reflecting that length of exposure is a factor in acquiring *T. gondii* infection. A number of serologic surveys for *T. gondii* have been conducted in the U.S. (Table 1). Infection was estimated at 23.9% of pigs in 1983-1984 with higher rates in breeders (42%) than in market pigs (23%). When pigs from these same areas were tested in 1992, the percentage had dropped to 20.8% of breeders and 3.1% of finisher pigs. Prevalence of *T. gondii* was 20% in sows tested in the 1990 National Animal Health Monitoring System (NAHMS) swine survey. Using sera from the NAHMS swine survey conducted in 1995, sow prevalence had fallen to 15.0% and finisher pigs had a seroprevalence of 3.2%. These results suggest that the frequency of *T. gondii* infection in pigs is declining, and that there is a clear difference in prevalence rates between breeding animals (sows) and market hogs.

Epidemiology

Transmission of *T. gondii* to pigs on the farm occurs by various means. Risk factors for transmission include exposure to live or dead rodents and other wildlife, as well as deliberate or inadvertent feeding of raw or undercooked meat scraps containing infective stages of the parasite. More importantly, pigs can acquire *T. gondii* from ingesting the environmentally resistant oocyst stage shed by cats. In several studies of management factors, outside housing of swine, access of cats to swine, infection in cats and mice, and small herd size were positively correlated with *T. gondii* infection. Once established, *T. gondii* can rapidly spread through an entire herd via cannibalism.

In a study of 47 farms in Illinois with typical rates of *Toxoplasma gondii* infection (15.1% in sows and 2.3% in finishers), a variety of reservoir hosts were found, including cats (68.3% infected), raccoons (67% infected), skunks (38.9% infected), opossums (22.7% infected), rats (6.3% infected) and mice (2.2% infected). In this same study, oocysts were found in samples of feed, soil, and cat feces. Oocysts can be found virtually anywhere, including pig feed and pig barns where cats are resident. An infected cat can shed millions of oocysts each day for up to one week, and these stages can survive in most climates for several years. Avoiding environmental contamination is a major hurdle in reducing pig exposure to *T. gondii*.

Control: Slaughter testing

There are no programs for the slaughter inspection of pigs for toxoplasmosis. It is not possible to detect the microscopic tissue cysts by visual inspection. Methods for testing pigs include serology and bioassay. Serological assays include various forms of agglutination tests and ELISA. The most sensitive and specific test method is the modified agglutination test using preserved whole tachyzoites. This test, however, is not suitable for use in the slaughterhouse or in the field due to the length of time required to obtain a result. The availability of an ELISA that is both sensitive and specific would allow wider use of serologic testing. The most definitive method for the detection of *T. gondii* infection is by bioassay, in which portions of tissue are inoculated into mice or cats. This procedure requires several weeks to determine the presence of parasites and is therefore not suitable for testing slaughtered animals.

Processing

Currently, no regulations require that pork be inspected for *T. gondii* and no further processing is mandated to inactivate the parasite. However, many of the methods that are in place for processing pork for inactivation of *Trichinella spiralis* (*trichinae*) are also effective for the inactivation of *T. gondii*. The following discussion summarizes the inactivation of *T. gondii* by processing methods.

Cooking - Thermal death curves for the interaction of temperatures and times required to kill *Toxoplasma gondii* in meat have been generated. From these data, we know that *T. gondii* is killed in 336 seconds at 49°C, in 44 seconds at 55°C, and in 6 seconds at 61°C. These times and temperatures apply only when the product reaches and maintains temperatures evenly distributed throughout the meat. The temperatures reported to kill *T. gondii* are lower than those required for *T. spiralis* (see *Trichinae* Fact Sheet). Thus methods prescribed for the destruction of trichinae in the USDA's Code of Federal Regulations are effective for the destruction of *T. gondii* also. The use of microwaves is not effective in killing *T. gondii*, probably because of uneven heating throughout the meat (see *Trichinae* Fact Sheet).

Freezing - Thermal death curves establishing the effect of cold on the viability of *T. gondii* in pork have been generated. Although tissue cysts remained viable at temperatures slightly below freezing (11.2 days

at -6.7°C and 22.4 days at -3.9 and -1.0°C) parasites were inactivated almost instantaneously at temperatures of -9.4°C and lower. Based on the data, the predicted times required to kill *T. gondii* are shorter than those required to kill *trichinae* (see Trichinae Fact Sheet); thus, processing times for pork prescribed by the USDA's Code of Federal Regulations to kill *trichinae* will also be effective for *T. gondii*. There is no evidence that there are strains of *T. gondii* with different freezing susceptibilities.

Curing – Our knowledge of the effect of various curing processes on *T. gondii* is limited and additional studies are needed to determine the effectiveness of curing for the destruction of *T. gondii* in pork and pork products.

Irradiation - *T. gondii* tissue cysts were rendered non-infectious by treatment with 40-50 krad of cesium-137, indicating that irradiation is a suitable method for eliminating the risk of this parasite in pork products.

Prevention of infection

Despite the widespread distribution of *T. gondii* in wildlife and the opportunity for cats to contaminate the environment with the resistant oocyst stage, it is possible to raise pigs free from *T. gondii* infection, as evidenced by many negative swine production sites found in recent seroprevalence studies. Prevention of infection in swine is accomplished through on-farm adherence to good production practices (GPPs), which include: 1) adopting an effective rodent control program to minimize mouse populations, 2) creating a level of biosecurity which reduces or eliminates exposure of swine to wildlife, 3) eliminating feral cats or securing feed and swine areas from access by cats, 4) prompt removal of dead pigs, and, 5) changing or thoroughly washing boots before entering barns to avoid tracking in oocysts.

The contribution of cats to the spread of *Toxoplasma gondii* infection in pigs cannot be overemphasized. Since it takes only one oocyst to infect a pig, protection of pigs from environmental contamination, contamination of feed, and transport of oocysts on boots is vital to control. In prevalence studies, from 41.9-70.7% of farm cats were seropositive for *T. gondii* infection. Cats only shed oocysts for 1–3 weeks; however, in one study, 1.8% of farm cats tested were actively shedding oocysts. Even more important was the finding of viable oocysts in soil and feed samples from these farms, suggesting that oocysts shed by cats become widely dispersed in the environment. Risk analysis of management factors associated with positive pigs showed that infection correlated with the presence of infected juvenile cats (sources of oocysts) and *T. gondii* infected mice. Thus, a high level of biosecurity and good production practices which take into account the possible sources of environmental and feed contamination are necessary to assure the raising of pigs free from *Toxoplasma gondii* infection.

Selected References and Additional Reading

Assadi-Rad, A.M., New, J.C. and Patton, S. 1995. Risk factors associated with transmission of *Toxoplasma gondii* to sows kept in difference management systems in Tennessee. *Vet. Parasitol.* 57: 289-297.

Dubey J.P. 1990. Status of toxoplasmosis in pigs in the United States. *J. Am. Vet. Med. Assoc.* 196: 270-274.

Dubey J.P., Leighty J.C., Beal V.C., Anderson W.R., Andrews C.D. and Thulliez, Ph. 1991. National seroprevalence of *Toxoplasma gondii* in pigs. *J. Parasitol.* 77: 517-521.

Dubey J.P., Kotula A.W., Sharar A., Andrews C.D., and Lindsay D.S. 1990. Effect of high temperature on infectivity of *Toxoplasma gondii* tissue cysts in pork. *J. Parasitol.* 76: 201-204.

Dubey J.P., Weigel R.M., Siegel A.M., Thulliez P., Kitron U.D., Mitchell M.A., Mannelli A., Mateus-Pinilla N.E., Shen S.K., Kwok O.C.H. and Todd K.S. 1995. Sources and reservoirs of *Toxoplasma gondii* infection on 47 swine farms in Illinois. *J. Parasitol.* 81: 723-729.

Dubey, J.P., Murrell, K.D., Hanbury, R.D., Anderson, W.R., Doby, P.B. and Miller, H.O. 1986. Epidemiologic findings on a swine farm with enzootic toxoplasmosis. *J. Am. Vet. Med. Assoc.* 189: 55-56.

Gamble, H.R., Brady, R.C. and Dubey, J.P. 1999. Prevalence of *Toxoplasma gondii* infection in domestic pigs in the New England states. *Vet. Parasitol.* 82: 129-136.

Kotula A.W., Dubey J.P., Sharar A.K., Andrews C.D., Shen S.K. and Lindsay D.S. 1991. Effect of freezing on infectivity of *Toxoplasma gondii* tissue cysts in pork. *J. Food Prot.* 54: 687-690.

Lopez, A., Dietz, V.J., Wilson, M., and Jones, J.L. 2000. Preventing congenital toxoplasmosis. *MMWR* 49: 59 - 75.

Mead, P.S., Slutsker, L., Dietz, V., McCaig, L.F., Bresee, J.S., Shapiro, C., Griffin, P.M. and Tauxe, R.V. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.*, 5(5): 1999 Sept.-Oct., available from: URL:<http://www.cdc.gov/ncidod/EID/eid.htm>

Patton, S., Zimmerman, J., Roberts, T., Faulkner, C., Diderrich, V., Assadi-Rad, A., Davies, P. and Kliebenstein, J. 1996. Seroprevalence of *Toxoplasma gon-*

dii in hogs in the National Animal Health Monitoring System (NAHMS). *J. Eucaryotic Microbiol.* 43: 1215.

Roberts T., Weiss, M., Southard, L. 1993. Issues in pork safety: cost control and incentives. *Agricultural Outlook AO-201*, USDA-ERS, October: 28-32.

Roghamm, M-C., Faulkner, C.T., Lefkowitz, A., Patton, S., Zimmerman, J. and Morris, J.G. 1999. Decreased seroprevalence for *Toxoplasma gondii* in Seventh Day Adventists in Maryland. *Am. J. Trop. Med. Hyg.* 60: 790-792.

Smith K.E., Zimmerman J.J., Patton S., Beran G.W. and Hill H.T. 1992. The epidemiology of toxoplasmosis of Iowa swine farms with an emphasis on the roles of free-living mammals. *Vet. Parasitol.* 42: 199-211.

Smith, J.L. 1997. Long-term consequences of foodborne toxoplasmosis: effects on the unborn, the immunocompromised, the elderly and the immunocompetent. *J. Food Protect.* 60: 1595-1611.

Smith K.E., Zimmerman J.J., Patton S., Beran G.W. and Hill H.T. 1992. The epidemiology of toxoplasmosis of Iowa swine farms with an emphasis on the roles of free-living mammals. *Vet. Parasitol.* 42: 199-211.

Smith K.L., Wilson M., Hightower A.W., Kelley P.W., Struewing J.P., Juraneck D.D. and McAuley J.B. 1996. Prevalence of *Toxoplasma gondii* antibodies in U.S. military recruits in 1989: comparison with data published in 1965. *Clin. Infect. Dis.* 23: 1182-1183.

Weigel R.M., Dubey J.P., Siegel A.M., Kitron U.D., Mannelli A., Mitchell M.A., Mateus-Pinilla N.E., Thulliez P., Shen S.K., Kwok O.C.H. and Todd K.S. 1995. Risk factors for transmission of *Toxoplasma gondii* on swine farms in Illinois. *J. Parasitol.* 81, 736-741.

Weigel R.M., Dubey J.P., Siegel A.M., Hoefling D., Reynolds D., Herr L., Kitron U.D., Shen S.K., Thulliez P., Fayer R. and Todd K.S. 1995. Prevalence of antibodies to *Toxoplasma gondii* in swine in Illinois in 1992. *Am. J. Vet. Med. Assoc.* 206: 1747-1751.

Reference to products in this publication is not intended to be an endorsement to the exclusion of others which may be similar. Persons using such products assume responsibility for their use in accordance with current directions of the manufacturer. The information represented herein is believed to be accurate but is in no way guaranteed. The authors, reviewers, and publishers assume no liability in connection with any use for the products discussed and make no warranty, expressed or implied, in that respect, nor can it be assumed that all safety measures are indicated herein or that additional measures may be required. The user therefore, must assume full responsibility, both as to persons and as to property, for the use of these materials including any which might be covered by patent.
This material may be available in alternative formats.

Information developed for the Pork Information Gateway, a project of the U.S. Pork Center of Excellence supported fully by USDA/Agricultural Research Service, USDA/Cooperative State Research, Education, and Extension Service, Pork Checkoff, NPPC, state pork associations from Iowa, Kentucky, Missouri, Mississippi, Tennessee, Pennsylvania, and Utah, and the Extension Services from several cooperating Land-Grant Institutions including Iowa State University, North Carolina State University, University of Minnesota, University of Illinois, University of Missouri, University of Nebraska, Purdue University, The Ohio State University, South Dakota State University, Kansas State University, Michigan State University, University of Wisconsin, Texas A & M University, Virginia Tech University, University of Tennessee, North Dakota State University, University of Georgia, University of Arkansas, and Colorado State University.