Toxoplasma

Introduction

*Toxoplasma gondii* is a protozoan (single-celled) parasite found in muscle and other tissues of many warm-blooded animals including pigs and humans. 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, in their feces, the environmentally resistant and infectious stage called the oocyst.

Infection with *Toxoplasma* occurs when pigs, and other animals, accidentally ingest oocysts in soil or water, or eat tissues of infected rodents, wildlife, or meat containing tissue cysts, a second infectious stage of the organism. 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 to 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.

**Figure 1. Life cycle of Toxoplasma gondii.**

**Toxoplasmosis in Humans**

Human infection rates with *Toxoplasma gondii* are relative high as compared with many other diseases. Serological surveys report infection rates well above 50% of the population in some countries. Prevalence rates in France, for example, have been reported to range from 42-84%. 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, 1999-2004) involving over 15,000 people, 10.8% were positive for *T. gondii* antibodies, indicating infection with the parasite. This contrasts with a prevalence of 14.1% for the period of 1988-1994. Prevalence in women of child bearing age was 11% in the 1999-2004 survey. Prevalence of infection was lower in persons 6-11 years of age (3.6%) and increased with age, up to 15.7% in persons 40-49 years of age.

Exposure of healthy adults to *Toxoplasma gondii* generally results in either an asymptomatic infection or a mild “flu-like” illness. Acute disease occurs occasionally and includes lymphadenopathy, chorioretinitis, respiratory involvement, or central nervous system infection.

Major health problems can result when *T. gondii* is transmitted from a pregnant woman to her unborn baby. 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. At risk are women who acquire a primary infection either during, or shortly before, pregnancy. 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.

Following initial infection, *Toxoplasma* cysts can remain viable in the body for a long period of time and these latent infections can be reactivated in newly immunosuppressed people such as those being treated for malignancies or after organ transplants. In these cases, the results can be severe illness and death. Toxoplastic encephalitis, a disease caused by parasites multiplying in the brain, results from either acute infection or reactivation of a latent infection. It is the most common clinical presentation of toxoplasmosis in people with AIDS.

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 toxoplastic encephalitis in AIDS cases. The Centers for Disease Control estimates that *T. gondii* is responsible for approximately 20% of all deaths attributed to foodborne pathogens.

**Sources of Human Infection**

Humans may become infected with *T. gondii* in one of three ways: 1) by accidental ingestion of oocysts contaminating the environment; 2) by ingestion of tissue cysts when consuming or improperly handling undercooked or raw meat from infected animals, or 3) by congenital infection, when previously unexposed women become infected around or during pregnancy and transmit the infection to the fetus. Congenital transmission of infection can be documented if early testing is performed. While other sources of infection are more difficult to document, serological tests are available which can determine whether the source of infection was by ingestion of oocysts or by consumption of tissue cysts in meat.

The oocyst stage, excreted by cats, is very resistant to environmental fluctuations and oocysts can survive for years in soil, even in adverse conditions. Humans can be exposed to oocysts through gardening activities, by eating unwashed fruits and vegetables contaminated with oocysts, by drinking water contaminated with oocysts, or by cleaning a cat litter box or from other contact with cat feces (e.g., in sandboxes).

Meat, infected with *Toxoplasma* cysts (bradyzoites) can also be a source of human infection if it is not properly cooked by the consumer or otherwise treated to kill the parasite (e.g., treated by commercial cooking, freezing or curing methods that have been validated for inactivating *Toxoplasma*). The importance of meat as a source of human infection is not clear and most evidence for the role of meat is indirect. For example, 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.

A survey of major commodity meats in the retail meat cases of a representative sample of major U.S. metropolitan areas found that viable *Toxoplasma* was present in 0.38% of pork samples. No viable *Toxoplasma* was found in beef or chicken, but up to 1.3% of chickens were found to have been exposed to the parasite. It is likely that freezing of chicken is responsible for inactivating the parasite before it reaches the consumer. Given that consumers of fresh meat are provided with guidance on cooking, coupled with the low prevalence of viable *Toxoplasma* in retail pork, the risk to consumers from eating pork can be estimated to be quite low.

A recent risk assessment comparing *Toxoplasma* positive and negative adults found that the greatest associated risk in infected individuals was cat ownership. Other associated factors included eating rare lamb, eating raw ground beef, drinking unpasteurized goat milk, working with meat, eating raw oysters, clams and mussels and eating locally produced, cured, dried, or smoked meats.

**Toxoplasma in Pigs**

Most species of livestock, including sheep, goats, and pigs, are susceptible to infection with *Toxoplasma gondii*, although animals exposed to this parasite 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.

Sheep and goats are important hosts of *Toxoplasma* in some countries and pose a major risk for human exposure, but are a minor species in the U.S. Similarly, free-range chickens are known to be infected, often at high rates, but have not been indicated as a source of human infection in the U.S.

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 national 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 USDA’s National Animal Health Monitoring System (NAHMS) swine survey. In the 1995 NAHMS swine survey, sow prevalence had fallen to 15.0% and finisher pigs had a seroprevalence of 3.2%; in 2000 these same populations had prevalence rates of 6.0% and 0.9%, respectively. In the 2006 NAHMS swine survey, seroprevalence in market hogs was 2.6%.

These national survey results suggest that the frequency of *T. gondii* infection in pigs has declined, and the likely reason for this decline in prevalence is the increased use of confinement housing systems in the U.S. pork industry. However, there is a clear difference in prevalence rates between breeding animals (sows) and market hogs as well as between production systems. Pigs raised outdoors or in limited confinement are always at risk for exposure to *T. gondii*. For example, data collected in 1997-1998 from various U.S. states found infection rates as high as 50% where pigs were raised in outdoor systems.

**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.

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 moist climates for several years. 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. 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 *Toxoplasma*. It is not possible to detect the microscopic tissue cysts by visual inspection. Methods for testing pigs include bioassay and serology. Bioassay is the most definitive method for the detection of *T. gondii* infection, but requires inoculation of portions of tissue into mice or cats. This procedure requires several weeks to determine the presence of parasites and is therefore not suitable for testing slaughtered animals.

Sero logicals assays include various forms of agglutination tests and ELISA. The most sensitive and specific test method is the ELISA. The recent development and commercialization of an ELISA that is both sensitive and specific allows for wider use of serologic testing and is particularly useful for herd surveillance.

**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) (U.S. Code of Federal Regulations, 9CFR318.10) are also effective for the inactivation of *T. gondii*. For that reason, processed pork products should be safe for human consumption without further treatment. 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 in closely controlled scientific studies. From these data, we know that *T. gondii* is killed in 336 seconds at 49°C (120°F), in 44 seconds at 55°C (131°F), and in 6 seconds at 61°C (142°F). 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 National Pork Board’s *Trichinella* Fact Sheet). Thus methods for the destruction of *Trichinella* 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 National Pork Board’s *Trichinella* Fact Sheet).

- **Freezing** - Thermal death curves have also been developed to establish the effect of cold treatment on the viability of *T. gondii* in pork. Although tissue cysts remained viable at temperatures slightly below freezing (11.2 days at -6.7°C (20°F) and 22.4 days at -3.9°C and -1.0°C (25 and 30°F)) parasites were inactivated almost instantaneously at temperatures of -9.4°C (15°F) and lower. Based on these data, the predicted times required to kill *T. gondii* are shorter than those required to kill *Trichinella* (see National Pork Board’s *Trichinella* Fact Sheet); thus, processing times for pork prescribed by the USDA’s Code of Federal Regulations to kill *Trichinella* 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. Pum ping of pork products with salt solutions containing 2% NaCl or ≥1.4% potassium or sodium lactate has been shown
to inactivate *T. gondii* tissue cysts in pork.

- **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.

**Consumer handling of pork products**

The USDA advises consumers to cook pork to an internal temperature of 63.8°C (145°F) followed by a 3 minute rest, which is effective for killing *T. gondii*.

**Prevention of infection**

Despite the widespread distribution of *T. gondii* in wildlife and the opportunity for cats to contaminate the environment with resistant oocyst stage, it is possible to raise pigs free from *T. gondii* infection, through good production practices to reduce risk of exposure of pigs, including the introduction of barn-only boots, resulted in a reduction in *Toxoplasma* prevalence on infected farms after three production cycles. These results demonstrate that good production practices can be implemented to greatly reduce the risk of exposure to *Toxoplasma* in confinement-raised pigs.

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

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<th>Year</th>
<th>No. tested</th>
<th>% positive</th>
<th>Pig type</th>
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<td>Market hogs</td>
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<td>Sows</td>
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<td>6,238</td>
<td>2.6</td>
<td>Market hogs</td>
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**Selected References and Additional Reading**


**Web Resources**

CDC Toxoplasma Web Page  

Food Safety News Article  