Foodborne Toxoplasmosis

Jeffrey L. Jones[1] and J. P. Dubey[2]

[1]Parasitic Diseases Branch, Division of Parasitic Diseases and Malaria, Center for Global Health, Centers for Disease Control and Prevention, Atlanta, Georgia; and [2]Animal Parasitic Diseases Laboratory, Agricultural Research Institute, Animal and Natural Resources Institute, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland

Toxoplasmosis can be due to congenital infection or acquired infection after birth and is one of the leading illnesses associated with foodborne hospitalizations and deaths. Undercooked meat, especially pork, lamb, and wild game meat, and soil contaminated with cat feces on raw fruits and vegetables are the major sources of foodborne transmission for humans. The new trend in the production of free-range organically raised meat could increase the risk of *Toxoplasma gondii* contamination of meat. Foodborne transmission can be prevented by production practices that reduce *T. gondii* in meat, adequate cooking of meat, washing of raw fruits and vegetables, prevention of cross contamination in the kitchen, and measures that decrease spread of viable oocysts into the environment.

Toxoplasma gondii infections are widely prevalent in human beings and food animals [1]. Felids are the key species in the life cycle [2] of this parasite because they are the only hosts that excrete the environmentally resistant stage, the oocyst, in their feces. Humans become infected postnatally by ingesting tissue cysts from undercooked meat, consuming food contaminated with oocysts, or by accidentally ingesting oocysts from the environment. Clinical toxoplasmosis in humans has been linked epidemiologically to ingestion of *T. gondii* in food [1], and foodborne transmission is one of the major sources of *T. gondii* infection.

Clinical disease resulting from *T. gondii* infection occurs as acquired infection in immunocompetent persons (usually mild), disease in immunosuppressed persons (usually due to reactivation of chronic infection), congenital disease, and ocular disease (congenital or acquired) [3–5]. In a recent assessment of foodborne illnesses in the United States, toxoplasmosis was identified as the second leading cause of foodborne illness–related deaths and fourth leading cause of foodborne illness–related hospitalizations (an estimated 327 deaths, and 4428 hospitalizations annually) [6]. In addition, over 4800 infections are estimated to result in acute ocular disease annually [7], and 400–4000 congenital cases are estimated to occur in the United States annually [8]. Internationally, toxoplasmosis has also had a significant effect on health; for example, in Greece, researchers recently found toxoplasmosis to be in the top 5 contributors of foodborne illnesses to years of life lost, years lived with disability, and disability-adjusted life years per million persons (9.7, 14, and 23 years, respectively) [9].

TRANSMISSION OF *T. gondii*

Infected Meat

In the United States, poultry, pork, and beef are the main meats consumed. In a recent nationwide study of the prevalence of *T. gondii* in retail meat, viable organisms were isolated from only 7 of 2094 pork samples and none of 2094 beef or 2094 chicken samples [10]. However, parasitological surveys based on retail meat samples do not provide a true assessment of risk from fresh meat because nearly half of the pork and a substantial amount of chicken are injected with salts and water, which can kill *T. gondii* tissue cysts [11]; the product is labeled with the

Received 27 December 2011; accepted 15 May 2012; electronically published 22 May 2012.

Correspondence: Jeffrey L. Jones, MD, MPH, Parasitic Diseases Branch, Division of Parasitic Diseases and Malaria, Center for Global Health, Centers for Disease Control and Prevention, Bld. 24, 1600 Clifton Rd, NE, Mailstop A-06, Atlanta, GA 30333 (jlj1@cdc.gov).

Clinical Infectious Diseases 2012;55(6):845–51
Published by Oxford University Press on behalf of the Infectious Diseases Society of America 2012.
DOI: 10.1093/cid/cis508

ingredients of the treatments and the term "enhanced" meat may also be used [10]. Further, most retail chicken sold in the United States is frozen, which also kills *T. gondii* cysts. In this section, we will discuss meat sources of *T. gondii* for humans.

Pigs

Currently, there is no national identification system for individual pigs destined for human consumption, and pigs are not tested for *T. gondii* infection at slaughter. Meat from breeder pigs is generally processed for sausages and it is highly unlikely that *T. gondii* survives most commercial processing procedures (cooking, freezing, salting), suggesting that breeder pigs are not an important source of *T. gondii* transmission to humans; however, market pigs are a more likely source. The prevalence of *T. gondii* is declining, but even a 1% infection rate in market pigs would amount to 1 million infected pigs going to market for human consumption; a 50 kg market pig produces over 300 servings of meat.

An upsurge in consumer demand for "organically raised" and "free range"' pork products has resulted in increasing numbers of pigs being raised in nonconfinement systems. Swine producers have been recruited to produce animals for the organic market to fulfill a consumer demand for organically raised free-range pork that has increased an average of 20% per year in sales since 1990. Organic program standards [12] require that all animals raised must have access to the outdoors, including access to pasture for ruminants. Access to grass, soil, feed, or water contaminated with cat feces, or to rodents and wildlife infected with *T. gondii,* during outdoor pasturage substantially increases the risk of exposure of pigs to *T. gondii*. Kijlstra et al. [13] found that 0 of 621 conventionally raised pigs were seropositive for *T. gondii*, while 38 of 1295 (2.9%) pigs raised in "animal friendly" management systems were seropositive for *T. gondii*. Recently, viable *T. gondii* was isolated from 17 of 33 organically raised pigs from Michigan [14] (Table 1).

Cattle

The ingestion of beef or dairy products has not been considered important in the epidemiology of *T. gondii* because cattle are not a good host for this parasite [15]. However, we cannot be sure that beef does not play a role in *T. gondii* transmission as only relatively small amounts of beef have been tested for viable *T. gondii* parasites with negative results, and epidemiologic studies have linked undercooked beef to *T. gondii* infection (although the link was not definitive, see the "Risk Factors and Attributable Risk" section).

Chickens

In the United States, the per capita yearly consumption of poultry is estimated to be 37.2 kg and approximately 8.5 billion chickens are killed annually for human consumption. As noted above, in a recent survey, *T. gondii* was not isolated by bioassay from any of the 2094 chicken meat samples obtained from retail stores in the United States [10]. However, because many of the chicken meat samples were injected with enhancing

Table 1. Isolation of *Toxoplasma gondii* From Various Food Animals in the United States

Species	Source	No. Bioassayed and Tissue	% Positive	Reference[a]
Pigs	Abattoir, Maryland	50 diaphragms	24	Jacobs et al. 1960 [59]
	Abattoir, Iowa	1000 sow hearts	17	Dubey et al. 1995 [60]
	Massachusetts, 1 herd	55 market hogs hearts and tongues	92.7	Dubey et al. 2002 [61]
	Retail meat, nationwide	2094 pork	0.3	Dubey et al. 2005 [10]
	Maryland, herd	38 pigs	36.8	Dubey et al. 2008 [62]
	Michigan	33 organic pigs	51.5	Dubey et al. 2012 [14]
Sheep	Abattoir, Maryland	86	9.2	Jacobs et al. 1960 [59]
	Retail meat	50 lamb chops	4	Remington, 1968 [63]
	Abattoir, Maryland	68 lamb hearts	77.9	Dubey et al. 2008 [64]
Goats	Retail meat	112 seropositive hearts	25.8	Dubey et al. 2011 [18]
Cattle	Abattoir, Maryland	60 diaphragms	0	Jacobs et al. 1960 [59]
	Abattoir, Ohio	350 mixed tissues	0	Dubey and Streitel, 1976 [65]
	Retail meat, nationwide	2094 beef	0	Dubey et al. 2005 [10]
Chickens	Retail meat, nationwide	2094 breast meat	0	Dubey et al. 2005 [10]
Deer	Alabama	19	21	Lindsay et al. 1991 [66]
	Mississippi	73	28.7	Dubey et al. 2004 [67]
	Iowa, Minnesota	88	17	Dubey et al. 2008 [68]
Black bear	Pennsylvania	28	35.7	Dubey et al. 1995 [69]
	Pennsylvania	10	70	Dubey et al. 2004 [67]

[a] Modified from Dubey and Jones 2008 [70].

solutions or may have been "hard chilled" or frozen, the results of this study do not negate the possibility that infected chickens may be a source of *T. gondii* for humans. In contrast to the bioassay results, antibodies to *T. gondii* were found in 1.3% of the juice extracted from the breast meat from the 2094 chicken samples obtained at retail stores using an enzyme-linked immunosorbent assay, which suggests that *T. gondii* can occur in commercially marketed chickens in the United States, although the bioassay results from the retail meat survey [10] indicate that the risk from commercial chicken is small. The recent trend of consumers demanding meat from organically grown free range poultry will probably increase the prevalence of *T. gondii* in chickens consumed by humans. Unlike indoor raised chickens, the prevalence of *T. gondii* in free-range chickens is very high (United States, 17%–100%) [16]. The prevalence of *T. gondii* in chicken eggs is extremely low and the ingestion of uncooked eggs is not considered an important risk for toxoplasmosis [16].

Sheep

Approximately 3.5 million lambs are slaughtered for food in the United States each year, and the per capita consumption of lamb meat is about 0.3 kg per year [17]. Results of a recent study and previous surveys indicate the prevalence of *T. gondii* in lambs can be high (Table 1).

Goats

Although pasteurization will kill *T. gondii* in goat's milk, unpasteurized raw milk is sold by small goat farmers and goat cheeses made from raw milk could be a source of *T. gondii* infection. Little is known of the excretion of *T. gondii* in goat's milk [1]. Goat meat is also very popular with many ethnic groups in the United States. In a recent study, the seroprevalence of *T. gondii* antibodies in goat meat destined for human consumption in the United States was found to be 53.4% [18].

Horses

In France, severe human toxoplasmosis has been epidemiologically linked to ingestion of horse meat imported from Canada and Brazil [19] and viable *T. gondii* has been isolated from US horses slaughtered for export [20]. However, horse meat is not frequently used for human consumption in the United States.

Venison and Other Game

Deer are popular and abundant game animals in the United States. Antibodies to *T. gondii* are highly prevalent (30%–60%) in white-tailed deer; viable *T. gondii* was isolated from 17%–29% (Table 1). Other game animals frequently hunted in the United States each year, including bear, elk, moose, and wild pig, may also be sources of *T. gondii* infection; for example, the prevalence of *T. gondii* in black bears is very high (Table 1).

Role of Oocysts

Environmentally resistant oocysts are essential in the life cycle of *T. gondii* [2]. Only cats are known to excrete *T. gondii* oocysts in their feces, which contaminate soil and can be ingested by animals, or by humans on uncooked fruits and vegetables. Although cats can be reinfected with *T. gondii*, most shed oocysts in their feces only for several weeks once during their life. Approximately one-third of households in the United States own a cat; there are approximately 78 million domestic cats and 73 million feral cats (reviewed by Conrad et al., 2005) [21]. Oocysts can survive in the environment for long periods—for example, oocysts survived outdoors in Texas (6°C–36°C) in native cat feces, uncovered, for 46 days, for 334 days when covered, and outdoors in soil buried at the depth of 3–9 cm in Kansas for 18 months. The oocyst stage of *T. gondii* is highly resistant to disinfectants and freezing—for example, oocysts survived at −21°C for 28 days [22] but are killed by temperatures above 60°C [23]. Ultraviolet rays also will inactivate oocysts, depending on the dose [24, 25].

HUMAN SEROPREVALENCE IN THE UNITED STATES

Toxoplasma gondii seroreactivity is generally thought to remain for life [1]. Historical serological surveys have been summarized by Dubey [1]. An analysis of the US National Health and Examination Nutrition Study (NHANES) serum samples from 1988 to 1994 in persons over 12 years old showed an overall age-adjusted seroprevalence of 22.5% [26]. A more recent NHANES found a decrease in the age-adjusted *T. gondii* prevalence in US-born persons 12–49 years old from 14.1% in 1988–1994 to 9% in 1999–2004, and a seroprevalence of 7.7% in US-born and 28.1% in foreign-born women 15–44 years old in 1999–2004 [27] (Table 2). Prior studies have also shown a decrease in *T. gondii* seroprevalence in the United States over time. For example, in 1962 and 1989, *T. gondii* seroprevalence was examined among military recruits, showing rates of 14.4% and 9.5%, respectively [28, 29]. Worldwide, foci of high prevalence exist in Latin America, parts of Eastern/Central Europe, the Middle East, and parts of south-east Asia and Africa. A trend toward lower seroprevalence in the last several decades has been observed in many European countries as well as the United States [30].

RISK FACTORS AND ATTRIBUTABLE RISK

Sources of *T. gondii* infection include the ingestion of cysts in undercooked meat and oocysts in soil contaminated with cat feces, which may be present on uncooked fruits and vegetables. The proportion of infections caused by each of these sources is not known for the general population. However,

Table 2. Selected US Human *Toxoplasma gondii* Prevalence Studies

Year Sampled	Age Group	Source of Serum Sample	No. Tested	% Positive	Reference[a]
1962	US young adult	Military recruits	2680	14	Feldman 1965 [28]
1987	≥18 y-old	Maryland community	251	31	Roghmann et al. 1999 [43]
1989	US young adult	Military recruits	2862	9.5	Smith et al. 1996 [29]
1992–1993	≥18 y-old	Illinois swine farm workers	174	31	Weigel et al. 1999 [71]
1988–1994	US age-adjusted ≥12 y-old	NHANES	17 658	22.5	Jones et al. 2001 [26]
1999–2000	US age-adjusted 12–49 y, women 12–49 y	NHANES	4234	15.8, 14.9	Jones et al. 2003 [72]
1999–2004	US age-adjusted 6–49 y, women 15–44 y	NHANES	15 960	10.8, 11.0	Jones et al. 2007 [27]

Abbreviation: NHANES, National Health and Nutrition Examination Study.

[a] Adapted from Dubey and Jones 2008 [70].

recently, a sporozoite-specific antigen (indicator of oocyst infection) was identified and convenience samples of sera were assayed to determine exposure to oocysts [31]. Of 163 persons in the acute stage of *T. gondii* infection, 103 (63.2%) were found to have sporozoite-specific antibodies in their serum samples [31, 32].

A case-control study conducted in 2002–2007 in the United States found numerous foodborne risk factors for recent *T. gondii* infection, including eating raw ground beef; eating rare lamb; eating locally produced cured, dried, or smoked meat sold only in the local city or area; working with meat; drinking unpasteurized goat's milk; and eating raw oysters, clams, or mussels [33]. In this study, the highest absolute risk was found for ingestion of rare lamb (adjusted odds ratio [AOR], 8.4; attributable risk [AR], 20%) and the highest attributable risk was found for ingestion of locally produced cured, dried, or smoked meat (AOR, 2.0; AR, 22%). A survey of pregnant women in the United States has documented that many do not perceive the risk of *T. gondii* infection from undercooked meat [34].

Numerous epidemiological studies have found undercooked meat to be a risk for *T. gondii* infection, including a multicenter European study of pregnant women where ingestion of inadequately cooked meat (lamb, beef, or game) was identified as the main risk factor [35]. Rare lamb has been circumstantially linked to symptomatic toxoplasmosis in a family in New York City [36]. Rare hamburger and beef have been linked to outbreaks of toxoplasmosis in the United States [37, 38], but other meats could have been responsible for these outbreaks (pork [37], lamb [38]). In Korea, eating contaminated pork was linked to clinical toxoplasmosis and ocular disease [39]. Horse meat originating from Canada and Brazil was implicated in clinical toxoplasmosis from infection with atypical strains in immunocompetent persons [19]. In the southern United States, ingestion of undercooked venison was found to be a *T. gondii* infection risk factor for hunters [40] and was linked to clinical toxoplasmosis with ocular disease [41]. In a retrospective study of 131 mothers who had given birth to children infected with *T. gondii*, 50% recalled having eaten uncooked meat [42]. In addition, a study that compared *T. gondii* prevalence in a community of Seventh Day Adventists, a religious group that follows a diet containing no meat, with a control community that was not Seventh Day Adventist, found that the prevalence of *T. gondii* infection in the Seventh Day Adventist community was approximately half of that in the control community (24% vs 50%) [43]. However, because *T. gondii* is transmitted from multiple sources, and recall of risk factors is often incomplete, it is difficult to establish the specific exposure responsible for infection in an individual.

In addition to being a risk factor in a US case-control study [33], unpasteurized goat's milk was found to be a *T. gondii* infection risk in family clusters in the United States [44] and the United Kingdom [45], and was linked to toxoplasmosis in an infant [46]. Feeding goat milk whey to pigs was also identified as a source of *T. gondii* infection in pigs [47].

The study by Jones et al. [33] also found a *T. gondii* infection risk from eating raw oysters, mussels, and clams. *T. gondii* has been identified in wild mollusks [48] and is thought to be carried to the oceans through river systems [21] where it is filtered by mollusks. Since *T. gondii* oocysts are environmentally resistant, they are not generally killed by sewage treatment, water, or salt water [49].

PREVENTION OF FOODBORNE INFECTIONS

With regard to vaccines, a live sheep vaccine that produces protective immunity for 18 months is available to reduce loss of lambs [50]. In addition, an oral live vaccine can prevent cats from shedding oocysts [51]. However, commercial production of the cat vaccine was discontinued because of its high cost, the need to keep the vaccine frozen, its short shelf

life, and lack of interest among cat owners [1]. Vaccination with killed *T. gondii* has been unsuccessful and leads to only marginal protection at best. For humans, vaccination with live mutant or avirulent strains cannot be recommended because these strains may pose a risk to the fetus and there is no guarantee that the strains would not revert and lead to disease, particularly in immunosuppressed persons.

Reducing *T. gondii* contamination of meat by the meat industry is critical to prevent foodborne transmission of *T. gondii* to humans. Cats should be kept out of barns and food animal productions areas so that they do not contaminate the soil and animal food storage areas with cat feces that contain *T. gondii* oocysts, which could be ingested and infect food animals. In addition, appropriate rodent control is essential so that food animals do not become infected with *T. gondii* by ingestion of rodents that die in their production areas.

The US Department of Agriculture recommends cooking whole cuts of pork, lamb, veal, or beef to 145°F (62.8°C) or higher as measured with a food thermometer placed in the thickest part of the meat, with a 3-minute rest time after cooking [52]. However, in one instance under experimental conditions, *T. gondii* remained viable at 147.2° F (64°C) with a 3-minute rest [53], so in the opinion of the authors, it is best to cook whole cuts of pork, lamb, veal, or beef to a least 150°F (65.6°C) with a 3-minute rest. Ground meat and wild game meat should be cooked to 160° F (71.1°C) or higher, and poultry should be cooked to 165° F (73.9°C) or higher as measured with a food thermometer. Ground meats, wild game meats, and poultry do not require a rest time. Microwave cooking is unreliable for killing *T. gondii*.

Irradiation at 0.4–0.7 kGy or high-pressure processing at 300–400 MPa can inactivate *T. gondii* tissue cysts in meat. However, the effects of irradiation on color and of high-pressure treatment on color and texture have limited consumer acceptance [1, 54]. Freezing meat to an internal temperature of −12°C kills *T. gondii* tissue cysts [55]. Salting, curing, smoking, and the addition of solutions to meat to enhance color and taste can reduce the viability of *T. gondii* in meat; however, there is too much variability in these procedures to make a safety recommendation [56–58]. It is also important to prevent cross contamination from raw meat to other foods, especially those eaten raw. Practicing good hygienic measures and cooking meat adequately remain important preventive measures to take to reduce foodborne transmission (Table 3).

In conclusion, *T. gondii* is a common parasite that can lead to significant illness, particularly in pregnant women and immunosuppressed persons. *T. gondii* infection can be prevented

Table 3. Prevention of *Toxoplasma gondii* Exposure From Food and the Environment

• To prevent toxoplasmosis, meat should be adequately cooked (160°F [71.1°C] for ground meat and wild game meat; 150°F [65.6°C] for other whole cuts of meat with a 3-minute rest; and 165°F [73.9°C] for poultry); (see text). A food thermometer should be inserted in the thickest part of the meat to ensure that it is cooked through.
• Fruits and vegetables should be peeled or thoroughly washed before eaten.
• Cutting boards, dishes, counters, utensils, and hands should be washed with hot soapy water after they have been in contact with raw meat, poultry, seafood, or unwashed fruits and vegetables.
• Raw oysters, mussels, and clams should not be eaten.
• Unpasteurized goat's milk should not be ingested.
• Viscera of hunted animals should be buried to prevent scavenging by animals, especially cats, leading to their infection and the spread of *T. gondii* in the ecosystem.
• Pregnant women and immunosuppressed persons should wear gloves when they are gardening or handling soil or sand because of the possible presence of cat feces. Afterwards, they should wash their hands thoroughly.
• Sandboxes should be covered when not in use.
• Untreated water should not be ingested (especially in developing countries).
• If possible, pregnant women and immunosuppressed persons should avoid changing cat litter pans. If no one else is available to change the cat litter, they should wear gloves for this task and then wash their hands thoroughly. The litter box should be changed daily because *T. gondii* oocysts require more than 1 day to become infectious. They should be encouraged to keep their cats inside and not to adopt or handle stray cats. Cats should be fed only canned or dried commercial cat food or well-cooked table food; cats should not be given raw or undercooked meat.
• Health education for women of childbearing age and immunosuppressed persons should include information about preventing *T. gondii* transmission. At the first prenatal visit, healthcare providers should educate pregnant women about food hygiene and avoiding exposure to cat feces.
• The meat industry should continue efforts to reduce the presence of *T. gondii* in meat by employing good production practices such as keeping cats and rodents out of food animal production areas and using clean or treated water sources for the animals.
• The government should help encourage and disseminate information about animal production practices to reduce *T. gondii* contamination of meat and should encourage research in methods that reduce *T. gondii* contamination of meat, methods to prevent human infection from contaminated meat (including education about hygiene and cooking, and development of optimal cooking temperatures, irradiation, high-pressure treatment, and other methods to inactivate *T. gondii* cysts in meat), and methods to reduce contamination of the environment with *T. gondii* oocysts.

by production measures to reduce *T. gondii* in meat; adequate cooking, freezing, or physical/chemical treatment of meat; prevention of cross contamination from raw meat; and measures that prevent *T. gondii* infection of cats, and therefore, decrease spreading of oocysts into the environment (eg, keeping cats indoors and not feeding raw meat to cats). The trend in production of organically raised meat is likely to increase the risk of acquiring *T. gondii* from undercooked meat. Areas for future research that could help reduce foodborne transmission of *T. gondii* include: vaccine development; improved methods to deactivate *T. gondii* cysts in meat and oocysts in cat feces, soil, and water, and on fruits and vegetables; improved methods to prevent *T. gondii* infection in food animals, including those in field-raised production systems; improved methods to reduce environmental contamination by outdoor cat populations; and improved methods to educate the public and health professionals about prevention of foodborne toxoplasmosis.

Notes

Disclaimer. The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the Department of Health and Human Services, the Centers for Disease Control and Prevention, or the United States Department of Agriculture.

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

References

1. Dubey JP. Toxoplasmosis of animals and humans. 2nd ed. Boca Raton, Florida: CRC Press, **2010**:1–313.
2. Centers for Disease Control and Prevention. Parasites, toxoplasmosis, biology. Available at: http://www.cdc.gov/parasites/toxoplasmosis/biology.html. Accessed 4 May 2012.
3. Remington JS, McLeod R, Wilson CB, Desmonts G. Toxoplasmosis. In: Remington JS, Klein JO, Wilson CB, Nizet V, Maldonado YA, eds. Infectious diseases of the fetus and newborn infant. 7th ed. Philadelphia: Elsevier Saunders, **2011**:918–1041.
4. Montoya JG, Liesenfeld O. Toxoplasmosis. Lancet **2004**; 363:1965–76.
5. Holland GN. Ocular toxoplasmosis: a global reassessment. Part II: Disease manifestations and management. Am J Ophthalmol **2004**; 137:1–17.
6. Scallan E, Hoekstra RM, Angulo FJ, et al. Foodborne illness acquired in the United States—major pathogens. Emerg Infect Dis **2011**; 17:7–15.
7. Jones JL, Holland GN. Annual burden of ocular toxoplasmosis in the US. Am J Trop Med Hyg **2010**; 82:464–5.
8. Lopez A, Dietz VJ, Wilson M, Navin TR, Jones JL. Preventing congenital toxoplasmosis. MMWR Recomm Rep **2000**; 49(RR-2):59–68.
9. Gkogka E, Reij MW, Havelaar AH, Zwietering MH, Gorris GM. Risk-based estimate of effect of foodborne diseases on public health, Greece. Emerg Infect Dis **2011**; 17:1581–9.
10. Dubey JP, Hill DE, Jones JL, et al. Prevalence of viable *Toxoplasma gondii* in beef, chicken, and pork from retail meat stores in the United States: risk assessment to consumers. J Parasitol **2005**; 91:1082–93.
11. Hill DE, Sreekumar C, Gamble HR, Dubey JP. Effect of commonly used enhancement solutions on the viability of *Toxoplasma gondii* tissue cysts in pork loin. J Food Protect **2004**; 67:2230–3.
12. National Organic Program. USDA Agricultural Marketing Service. Available at: http://www.ams.usda.gov/AMSv1.0/nop. Accessed 14 December 2011.
13. Kijlstra A, Eissen OA, Cornelissen J, Munniksma K, Eijck I, Kortbeek T. *Toxoplasma gondii* infection in animal-friendly pig production systems. Invest Ophthalmol Vis Sci **2004**; 45:3165–9.
14. Dubey JP, Hill DE, Rozenboom D, et al. High prevalence and genotypes of *Toxoplasma gondii* isolated from organic pigs in northern USA. Vet Parasitol **2012**; doi:10.1016/j.vetpar.2012.03.008.
15. Dubey JP, Beattie CP. Toxoplasmosis of Animals and Man. Boca Raton: CRC Press **1988**; 1988:1–220 (toxoplasmosis in cattle 107–115).
16. Dubey JP. *Toxoplasma gondii* infections in chickens (*Gallus domesticus*): prevalence, clinical disease, diagnosis, and public health significance. Zoonoses Public Health **2010**; 57:60–73.
17. National Agriculture Statistic Service, USDA. Agricultural Statistics, 2010. Chapter XIII. Consumption and family living. Available at: http://www.nass.usda.gov/Publications/Ag_Statistics/2010/Chapter13.pdf. Accessed 14 December 2011.
18. Dubey JP, Rajendran C, Ferreira LR, et al. High prevalence and genotypes of *Toxoplasma gondii* isolated from goats, from a retail meat store, destined for human consumption in the USA. Int J Parasitol **2011**; 41:827–33.
19. Pomares C, Ajzenberg D, Bornard L, et al. Toxoplasmosis and horse meat, France. Emerg Infect Dis **2011**; 17:1327–8.
20. Al-Khalidi NW, Dubey JP. Prevalence of *Toxoplasma gondii* infection in horses. J Parasitol **1979**; 65:331–4.
21. Conrad PA, Miller MA, Kreuder C, et al. Transmission of *Toxoplasma*: clues from the study of sea otters as sentinels of *Toxoplasma gondii* flow into the marine environment. Int J Parasitol **2005**; 35:1155–68.
22. Frenkel JK, Dubey JP. Effects of freezing on the viability of *Toxoplasma* oocysts. J Parasitol **1973**; 59:587–8.
23. Wainwright KE, Miller MA, Barr BC, et al. Chemical inactivation of *Toxoplasma gondii* oocysts in water. J Parasitol **2007**; 93:925–31.
24. Wainwright KE, Lagunas-Solar M, Miller MA, et al. Physical inactivation of *Toxoplasma gondii* oocysts in water. Appl Environ Microbiol **2007**; 73:5663–6.
25. Dumètre A, Le Bras C, Baffet M, et al. Effects of ozone and ultraviolet radiation treatments on the infectivity of *Toxoplasma gondii* oocysts. Vet Parasitol **2008**; 153:209–13.
26. Jones JL, Kruszon-Moran D, Wilson M, McQuillan G, Navin T, McAuley JB. *Toxoplasma gondii* infection in the United States: seroprevalence and risk factors. Am J Epidemiol **2001**; 154:357–65.
27. Jones JL, Kruszon-Moran D, Sanders-Lewis K, Wilson M. *Toxoplasma gondii* infection in the United States, 1999–2004, decline from the prior decade. Am J Trop Med Hyg **2007**; 77:405–10.
28. Feldman HA. A nationwide serum survey of United States military recruits, 1962. VI. *Toxoplasma* antibodies. Am J Epidemiol **1965**; 81:385–91.
29. Smith KL, Wilson M, Hightower AW, et al. Prevalence of *Toxoplasma gondii* antibodies in US military recruits in 1989: comparison with data published in 1965. Clin Infect Dis **1996**; 23:1182–3.
30. Pappas G, Roussos N, Falagas ME. Toxoplasmosis snapshots: global status of *Toxoplasma gondii* seroprevalence and implications for pregnancy and congenital toxoplasmosis. Int J Parasitol **2009**; 39:1385–94.
31. Hill D, Coss C, Dubey JP, et al. Identification of a sporozoite-specific antigen from *Toxoplasma gondii*. J Parasitol **2011**; 97:328–37.
32. Boyer K, Hill D, Mui E, et al. Unrecognized Ingestion of *Toxoplasma gondii* oocysts leads to congenital toxoplasmosis and causes epidemics in North America. Clin Infect Dis **2011**; 53:1081–9.
33. Jones JL, Dargelas V, Roberts J, Press C, Remington JS, Montoya JG. Risk factors for *Toxoplasma gondii* infection in the United States. Clin Infect Dis **2009**; 49:878–84.
34. Jones JL, Ogunmodede F, Scheftel J, et al. Toxoplasmosis-related knowledge and practices among pregnant women in the United States. Infect Dis Obstet Gynecol **2003**; 11:139–45.

35. Cook AJ, Gilbert RE, Buffolano W. Sources of toxoplasma infection in pregnant women: European multicentre case-control study. European Research Network on Congenital Toxoplasmosis. BMJ **2000**; 321:142–7.
36. Masur H, Jones TC, Lempert JA, Cherubini TD. Outbreak of toxoplasmosis in a family and documentation of acquired retinochoroiditis. Am J Med **1978**; 64:396–402.
37. Kean BH, Kimball AC, Christenson WN. An epidemic of acute toxoplasmosis. JAMA **1969**; 208:1002–4.
38. Lord WG, Boni F, Bodek A, et al. Toxoplasmosis—Pennsylvania. MMWR **1975**; 24:285–6.
39. Choi WY, Nam HW, Kwak NH, et al. Foodborne outbreaks of human toxoplasmosis. J Infect Dis **1997**; 175:1280–2.
40. Sacks JJ, Delgado DG, Lobel HO, Parker RL. Toxoplasmosis infection associated with eating undercooked venison. Am J Epidemiol **1983**; 118:832–8.
41. Ross RD, Stec LA, Werner JC, Blumenkranz MS, Glazer L, Williams GA. Presumed acquired ocular toxoplasmosis in deer hunters. Retina **2001**; 21:226–9.
42. Boyer KM, Holfels E, Roizen N, et al. Risk factors for *Toxoplasma gondii* infection in mothers of infants with congenital toxoplasmosis: implications for prenatal management and screening. Am J Obstet Gynecol **2005**; 192:564–71.
43. Roghmann MC, Faulkner CT, Lefkowitz A, Patton S, Zimmerman J, Morris JG Jr. Decreased seroprevalence for *Toxoplasma gondii* in Seventh Day Adventists in Maryland. Am J Trop Med Hyg **1999**; 60:790–2.
44. Sacks JJ, Roberto RR, Brooks NF. Toxoplasmosis infection associated with raw goat's milk. JAMA. **1982**; 248:1728–32.
45. Skinner LJ, Timperley AC, Wightman D, Chatterton JM, Ho-Yen DO. Simultaneous diagnosis of toxoplasmosis in goats and goatowner's family. Scand J Infect Dis **1990**; 22:359–61.
46. Riemann HP, Meyer ME, Theis JH, Kelso G, Behymer DE. Toxoplasmosis in an infant fed unpasteurized goat milk. J Pediatr **1975**; 87:573–6.
47. Meersburg BG, van Riel JW, Cornelissen JB, Kijlstra A, Mul MF. Cats and goat whey associated with *Toxoplasma gondii* infection in pigs. Vector-Borne Zoon. Dis **2006**; 6:266–74.
48. Lindsay DS, Collins MV, Mitchell SM, et al. Survival of *Toxoplasma gondii* oocysts in eastern oysters (*Crassostrea virginica*). J Parasitol **2004**; 90:1054–7.
49. Jones JL, Dubey JP. Waterborne toxoplasmosis—recent developments. Exp Parasitol **2010**; 124:10–25.
50. Buxton D, Innes EA. A commercial vaccine for ovine toxoplasmosis. Parasitology **1995**; 110:S11–S16.
51. Frenkel JK, Pfefferkorn ER, Smith DD, Fishback JL. Prospective vaccine prepared from a new mutant of *Toxoplasma gondii* for use in cats. Am J Vet Res **1991**; 52:759–763.
52. Food Safety and Inspection Service, USDA. News and Events. USDA revises recommended cooking temperature for all whole cuts of meat, including pork, to 145°F. May 24, 2011. Available at: http://www.fsis.usda.gov/News_&_Events/NR_052411_01/index.asp. Accessed 14 December 2011.
53. Dubey JP, Kotula AW, Sharar A, Andrews CD, Lindsay DS. Effect of high temperature on infectivity of *Toxoplasma gondii* tissue cysts in pork. J Parasitol **1990**; 76:201–4.
54. Kijlstra A, Jongert E. Control of the risk of human toxoplasmosis transmitted by meat. Int J Parasitol **2008**; 38:1359–1370.
55. Kotula AW, Dubey JP, Sharar AK, Andrews CD, Shen SK, Lindsay DS. Effect of freezing on infectivity of *Toxoplasma gondii* tissue cysts in pork. J Food Protect **1991**; 54:687–90.
56. Dubey JP. Survival of *Toxoplasma gondii* tissue cysts in 0.85–6% NaCl solutions at 4–20°C. J Parasitol **1997**; 83:946–9.
57. Hill DE, Benedetto SMC, Coss C, McCrary JL, Fournet VM, Dubey JP. Effect of time and temperature on the viability of *Toxoplasma gondii* tissue cysts in enhanced pork loin. J Food Protect **2006**; 69:1961–5.
58. Mie T, Pointon AM, Hamilton DR, Kiermeier A. A qualitative assessment of *Toxoplasma gondii* risk in ready-to-eat smallgoods processing. J Food Protection **2008**; 71:1442–52.
59. Jacobs L, Remington JS, Melton ML. A survey of meat samples from swine, cattle, and sheep for the presence of encysted *Toxoplasma*. J Parasitol **1960**; 46:23–8.
60. Dubey JP, Thulliez P, Powell EC. *Toxoplasma gondii* in Iowa sows: comparison of antibody titers to isolation of *T. gondii* by bioassays in mice and cats. J Parasitol **1995**; 81:48–53.
61. Dubey JP, Gamble HR, Hill D, Sreekumar C, Romand S, Thulliez P. High prevalence of viable *Toxoplasma gondii* infection in market weight pigs from a farm in Massachusetts. J Parasitol **2002**; 88:1234–8.
62. Dubey JP, Hill DE, Sundar N, et al. Endemic toxoplasmosis in pigs on a farm in Maryland: isolation and genetic characterization of *Toxoplasma gondii*. J Parasitol **2008**; 94:36–41.
63. Remington JS. Toxoplasmosis and congenital infection. Birth Defects **1968**; 4:49–56.
64. Dubey JP, Sundar N, Hill D, et al. High prevalence and abundant atypical genotypes of *Toxoplasma gondii* isolated from lambs destined for human consumption in the USA. Int J Parasitol **2008**; 38:999–1006.
65. Dubey JP, Streitel RH. Shedding of *Sarcocystis* in feces of dogs and cats fed muscles of naturally infected food animals in the Midwestern United States. J Parasitol **1976**; 62:828–30.
66. Lindsay DS, Blagburn BL, Dubey JP, Mason WH. Prevalence and isolation of *Toxoplasma gondii* from white-tailed deer in Alabama. J Parasitol **1991**; 77:62–4.
67. Dubey JP, Graham DH, de Young RW, et al. Molecular and biologic characteristics of *Toxoplasma gondii* isolates from wildlife in the United States. J Parasitol **2004**; 90:67–71.
68. Dubey JP, Velmurugan GV, Ulrich V, et al. Transplacental toxoplasmosis in naturally-infected white-tailed deer: isolation and genetic characterisation of *Toxoplasma gondii* from foetuses of different gestational ages. Int J Parasitol **2008**; 38:1057–63.
69. Dubey JP, Humphreys JG, Thulliez P. Prevalence of viable *Toxoplasma gondii* tissue cysts and antibodies to *T. gondii* by various serologic tests in black bears (*Ursus americanus*) from Pennsylvania. J Parasitol **1995**; 81:109–12.
70. Jones JL, Dubey JP. *Toxoplasma gondii* infection in humans and animals in the United States. Int J Parasitol **2008**; 38:1257–78.
71. Weigel RM, Dubey JP, Dyer D, Siegel AM. Risk Factors for infection with *Toxoplasma gondii* for residents and workers on swine farms in Illinois, USA. Am J Trop Med Hyg **1999**; 60:793–8.
72. Jones JL, Kruszon-Moran D, Wilson M. *Toxoplasma gondii* infection in the United States, 1999–2000. Emerg Infect Dis **2003**; 9:1371–4.