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Invited Review

Toxoplasma gondii prevalence in farm animals in the United States

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ABSTRACT

Toxoplasmosis, caused by *Toxoplasma gondii*, is one of the most common parasitic infections of humans and other warmblooded animals. It has been found worldwide and nearly one-third of humans have been exposed to the parasite. Congenital infection occurs when a woman becomes infected during pregnancy and transmits the parasite to the foetus. Besides congenital infection, humans become infected by ingesting food or water contaminated with sporulated oocysts from infected cat faeces or through ingestion of tissue cysts in undercooked or uncooked meat. Food animals (pigs, chickens, lambs and goats) become infected by the same routes, resulting in meat products containing tissue cysts, which can then infect consumers. *Toxoplasma* infection is common in food animals in the United States. Implementation of management factors such as biosecure confinement housing are important in reducing the levels of infection in animals destined for human consumption.

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1. Introduction

Toxoplasmosis continues to be a significant public health problem in the United States (US). It is estimated that 1,075,242 persons are infected with *Toxoplasma gondii* and approximately 2,839 persons develop symptomatic ocular disease annually (Jones and Holland, 2010). In the US, various surveys have found that 10–50% of the adult population has been exposed to *T. gondii* (Dubey and Beattie, 1988; Jones et al., 2001, 2003, 2007; Dubey and Jones, 2008). The cost of illness in the US caused by *Toxoplasma* has been estimated to be nearly US $3 billion and an annual loss of 11,000 quality-adjusted life year (QALY) (Batz et al., 2012; Hoffmann et al., 2012). Recent publications have linked suicide and schizophrenia to *Toxoplasma* infection (Pedersen et al., 2012; Torrey et al., 2012).

The relative contribution of foodborne (meat) sources of *Toxoplasma* infection versus oocyst transmission of *Toxoplasma* to human infection is unknown, and various studies have suggested widely disparate estimates of foodborne transmission. Mead et al. (1999) suggested that *T. gondii* is one of three pathogens (together with *Salmonella* and *Listeria*) which account for >75% of all deaths due to foodborne disease in the US Roghmann et al. (1999) suggested that 50% of *Toxoplasma* infections in the US could be ascribed to foodborne transmission. Scallan et al. (2011) estimated that *Toxoplasma* caused 8% of hospitalizations and 24% of deaths resulting from foodborne illnesses. In contrast, Dubey et al. (2005), in a nationwide survey of retail meats (beef, chicken and pork) found no viable *Toxoplasma* in any beef or chicken samples, and seven positive pork samples out of 2,094 samples assayed from each type of meat, concluding that there was not enough viable *Toxoplasma* present in retail meats to account for the level of *Toxoplasma* infection found in the US population. Recent studies (Boyer et al., 2011; Hill et al., 2011) have suggested that oocyst exposure is the predominate route of *Toxoplasma* transmission in the US.

Despite the uncertainty of human infection sources, *Toxoplasma* is recognised as a foodborne risk and infection is common in many domesticated animals used for food in the US, including pigs, chickens, lambs and goats. Animal infections with *Toxoplasma*, especially infections in non-meat eating ruminants, birds and pigs raised in confinement, likely result from environmental exposure to *T. gondii* oocysts. Oocyst contamination of the environment is widespread as a result of faecal contamination of soil and groundwater by the estimated 140 million domestic and feral cats in the US, each of which can deposit hundreds of millions of oocysts in faeces during infection (www.avma.org; Levy and Crawford, 2004; Dubey, 2010a). Oocyst contaminated runoff surface water entering the marine environment has resulted in devastating disease in endangered sea otters off the west coast of the US (Miller et al., 2002; Conrad et al., 2005), and even wild herbivores have been shown to have very high seroprevalence as a result of exposure to the environmentally resistant oocysts (Hill et al., 2005). Most *T. gondii* isolates from human and animal sources in North

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America have been grouped into one of three clonal lineages including Types I, II and III (Dardé et al., 1992; Howe and Sibley, 1995; Ajzenberg et al., 2002a,b), and are biologically and genetically different from isolates from Brazil and Columbia, but similar to isolates from Europe (Dubey et al., 2002a, 2007a; Lehmann et al., 2006; Dubey and Su, 2009). Recent genotyping studies of isolates from pigs, lambs and goats demonstrate that the Type II lineage predominates in food animals in the US, followed by Type III isolates and atypical genotypes; Type I isolates have rarely been found in farm animals (Dubey et al., 2008a,b, 2011; Velmurugan et al., 2009).

2. Surveillance in foods

Transmission of *Toxoplasma* from consumption of infected meat products is difficult to quantify, since meat from infected animals may undergo post-harvest treatments such as heating, freezing, salting or pumping (injection of water and salt-based solutions to retard microbial growth) that can render the parasite non-viable (Hill et al., 2004, 2006), and few comprehensive assessments have been completed in meat available for retail purchase. Complicating matters is the fact that the number of *T. gondii* organisms in meat from naturally infected food animals is very low, making the parasite difficult and expensive to detect by direct methods. It is estimated that as few as one tissue cyst may be present in 100 g of meat (Dubey, 2010a). In addition, there is no predilection site for *Toxoplasma* in meat animals; virtually all edible portions of an animal can harbour viable *T. gondii* tissue cysts (Dubey et al., 1986), and tissue cysts can remain viable in food animals for years.

Beef, chicken and pork are the main meat types consumed in the US In a case control study of 148 recently (<6 months) infected individuals, Jones et al. (2009) identified elevated risks of infection associated with eating raw ground beef, rare lamb, locally produced cured, dried or smoked meat, raw oysters, clams or mussels, working with meat and drinking unpasteurized goat's milk.

The relative risk to US consumers of acquiring *T. gondii* infection from undercooked meat was recently determined in a nationwide survey of retail chicken, beef and pork. The survey of 698 retail outlets in 28 metropolitan statistical areas (MSAs as defined by the US Census Bureau) covered 80% of the US population. Only pork was found to harbour viable *T. gondii* tissue cysts, which were isolated from 0.38% of samples (7/2,094) by cat bioassay, and 0.57% of pork samples were suspected to be infected based on positive ELISA results (Dubey et al., 2005). No beef samples were positive by bioassay or by ELISA, while 1.4% of chickens were positive by ELISA only. The northeastern United States had a higher number of positive pork samples than other regions of the country, reflecting the higher risk of pig infection due to regional management practises (outdoor versus confinement rearing; Dubey et al., 2005). Thus, while the extent of human infection resulting from meat sources remains undetermined, the lack of viable organisms in beef and chicken and the low prevalence of *T. gondii* infection in market pigs found in this comprehensive study would not account for the estimated incidence and measured seroprevalence in humans in the US.

3. Pigs

Serological surveys for *Toxoplasma* prevalence have been conducted frequently in the US over the last 30 years. Of the major meat animal species investigated thus far, pigs (and chickens) are the only species shown to frequently harbour the parasite (Dubey et al., 1991; (http://www.pork.org/FileLibrary/ResearchDocuments/00-130%20-PATTONUofTenn.pdf); Dubey and Jones, 2008; Hill et al., 2010). Dubey et al. (1991), in a nationwide survey conducted in 1984, found 23% of market pigs and 42% of sows were seropositive for *Toxoplasma*. Viable *T. gondii* tissue cysts were isolated from 17% of 1,000 adult pigs (sows) from a slaughter plant in Iowa, US (Dubey et al., 1995a), and from 51 of 55 (92.7%) market weight pigs from the New England region of the US (Dubey et al., 2002b). Serological surveys of pigs from Illinois (US) pig farms revealed an infection rate of 3% in market weight animals, but higher in breeding pigs (20%), suggesting that age is a factor for pigs acquiring *Toxoplasma* infection (Weigel et al., 1995a,b). Serological surveys on New England farms of pigs raised predominantly in outdoor conditions revealed an overall infection rate of 47% (Gamble et al., 1999). The United States Department of Agriculture (USDA) initiated the National Animal Health Monitoring System (NAHMS) in 1983 to collect, analyse and disseminate data on animal health, management and productivity in US domestic livestock populations, including swine (http://nahms.aphis.usda.gov; Losinger et al., 1998). The program includes an on-farm serological sampling component which can be used to monitor seroprevalence of various pathogens including, since 1990, *T. gondii*. Sera and data on management practises for this study are collected from breeding/grower/finisher swine production sites located in 17 states accounting for >90% of US swine production (Arkansas, Colorado, Iowa, Illinois, Indiana, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Carolina, Ohio, Pennsylvania, South Dakota, Texas, Oklahoma and Wisconsin). Only sows were sampled in 1990. Grower/finisher and sow/breeder populations were surveyed concurrently in 1995 and 2000. In 2006 the swine biological sampling targeted grower/finisher swine, which is the source of most fresh pork consumed in the US The initial 1990 survey documented nearly 20% seroprevalence of *Toxoplasma* in the US sow population. Subsequent NAHMS surveys have shown a decline in *Toxoplasma* seroprevalence in sows from 20% in 1990 to 15% in 1995, to 6% in 2000 (Patton et al., 1996, 2002 (see URL above)). In contrast, seroprevalence in grower/finisher swine has remained somewhat stable over that period. The measured seroprevalence in 1995 and 2000 was 3.2% and 0.9%, respectively (http://www.pork.org/FileLibrary/ResearchDocuments/00-130%20-PATTONUofTenn.pdf), and 2.6% in 2006 (herd prevalence of 21.6%; Hill et al., 2010). Data from the 2006 NAHMS for swine indicated that 145 of 185 farms had no *Toxoplasma*-positive samples. A sampling of grower/finisher populations in Tennessee, North Carolina and Illinois during this same period revealed a seroprevalence of 1–3% (Assadi-Rad et al., 1995; Dubey et al., 1995b; Patton et al., 1996). Risk factors associated with *Toxoplasma* infection identified during these surveys included swine raised outdoors and exposed to organic material potentially contaminated with cat faeces and oocysts, hogs raised on small farms, and cats and other sylvatic reservoirs on the premises. The steep decline in seroprevalence in grower/finisher hogs from 23% in 1984 (Dubey et al., 1991), to 3.2% in 1995 (NAHMS Swine 1995), to 0.9% in 2000 (NAHMS Swine 2000; (http://www.pork.org/FileLibrary/ResearchDocuments/00-130%20-PATTONUofTenn.pdf), to 2.6% in 2006 (NAHMS Swine 2006; Hill et al., 2010) likely reflects the increased use of confinement housing systems in the US pork industry; these studies indicated that pigs raised indoors in biosecure environments have a lower level of infection (Gamble et al., 1999; Dubey et al., 2002b). NAHMS 2006 data indicated that the odds of a farm being positive for *Toxoplasma* were 7.7 times higher when grower/finisher pigs were not housed in total confinement (Hill et al., 2010). While the reduced seroprevalence in sow populations probably resulted from the large-scale movement of the swine industry towards total confinement rearing (~80% of sows are kept in confinement) and an emphasis on facility biosecurity, the stable seroprevalence of ~2.0% in grower/finisher pigs may reflect gaps in adherence to good production practises known to prevent exposure to *Toxoplasma* in confinement-reared pigs. Boot hygiene has been shown to be essential