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REVIEW ARTICLE

# Prevalence of *Toxoplasma gondii* in Human and Animals and its Economic Impacts: A Review

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**A B S T R A C T**

*Toxoplasma gondii* (*T. gondii*) is a human and veterinary infection that is an obligate intracellular protozoan parasite. Plasmodium species and other members of the phylum Apicomplexa are also major pathogens. *T. gondii*, unlike most of these species, is easily manipulated genetically in the lab. Because of the great efficacy of transient and stable transfection, the availability of numerous cell markers and the relative simplicity with which the parasite may be investigated using modern microscopic methods, cell biology research is more easily undertaken in *T. gondii*. The tachyzoite, bradyzoite, and sporozoite are the 3 infective phases of *T. gondii*. In a complicated life cycle, several stages are intertwined. This review will highlight current understanding about the history, prevalence, and consequences of *T. gondii* infection in livestock and human, and the impacts of toxoplasmosis on cattle. It gives an indication of potential risk factors for *T. gondii* infection in cattle. To avoid *T. gondii* infection, adopt efficient biosecurity procedures on farms it is necessary to have knowledge about possible risk factors. Many studies have found cat-related risk factors, as well as those linked to possible infected feed, water, and contact to a potentially polluted location. There is a scarcity of published data on the expenses of *T. gondii* infections in animal productivity. Furthermore, the fact that this infection in animals can have an impact on human health should be addressed.

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## INTRODUCTION

*Toxoplasma gondii* (*T. gondii*) is a protozaon parasitic infection that is common in animals and humans all over the world. It has become most common serious disease among Acquired immunodeficiency syndrome (AIDS) patients. Toxoplasmosis is because of the recurrence of latent infection in AIDS patients. However, the mechanisms of reactivation remain unclear. The biology and anatomy of *T. gondii* phases (tachyzoite, bradyzoite, and tissue cyst) in intermediate host (human) as well as the resilient (oocyst) stage external the host are the subjects of this review.

**History of toxoplasmosis disease:** *T. gondii (T. gondii)* was first discovered in the African's rodent *Ctenodactylus gundi* (common gundi) in rabbit in Brazil (Al-hajj and Kekillioğlu 2023), and is now generally known as a widespread infection in warm-blooded species such as rodents and mammals. Toxoplasmosis' clinical consequences were initially described in children with retinochorditis, hydrocephalus and encephalitis in the early 1920s. *T. gondii is become common serious disease among AIDS patients* in the 1980s with immunological suppression (Lim and Othman, 2014). In the same way, (Remington 2006) described the scientific consequence of the infections in a variety of immunologically suppressed patient, with those go through cancer treatment or organ transplantation. In the immunocompetent group, toxoplasmosis is often unrecognized; nevertheless, nonspecific flu-like symptoms, lymphadenopathy and certain consequences linked with primary infection have been documented. The gestational age largely impacted the likelihood and harshness and inborn infection. Proper health care guiding of inborn and parental toxoplasmosis was extremely helpful in preventing complications in early diagnosis of toxoplasmosis. Most afflicted neonates and vision impairment were found to be asymptomatic at delivery, was also documented after some years.

After an initial *T. gondii* infection, it can induce chronic or recurring infections (Remington 2006). The cat plays a crucial part in parasite transmission reportedly since it is parasite's host and spreads oocysts fastly via its faeces. *T. gondii* infested both males and females, and infected people of all ages. In humid and warm settings, this parasite infection is very common (Senderowski *et al.*, 2010). The frequency of this parasite illness, on the other hand, was largely determined by the sanitation water supplies available (Dubey and Jones, 2014). Several researchers found that pet cats do not cause *T. gondii* infections in people (Paul *et al.*, 2021); nonetheless, rare investigations found that such cats provide a significant risk of parasite infection (Jones *et al.*, 2009). In most developing and developed nations, the seroprevalence of *T. gondii* caused 14 percent to 77 percent of pregnant women to suffer (Montoya and Liesenfeld, 2004). Toxoplasmosis is the major issue in cattle, and the disease's ecological effect is well-known. The result and hazards of this infection are mostly determined by *T. gondii* genotypes, and three of its clonal ancestries have been identified (Kalogeropoulos *et al.*, 2022). Only strain III was obtained from animals, whereas strains I and II were identified from HIV positive people.

**Basic life cycle:** The tachyzoite (in groups or clones), bradyzoite (in tissue cysts) and sporozoite are the 3 infective phases of *T. gondi* (in oocysts). In a complicated life cycle, several stages are intertwined (Fig. 1). The word "tachyzoite" has replaced the "trophozoite" in use (trophicos 5 feeding in Greek). Endodyozoites and endozoites are other names for tachyzoites. Clones, terminal colonies, or groups are assemblages of many tachyzoites. Tachyzoite typically has a crescent shape, averages from 2 to 6 mm, and has a conical frontal end and rounded later end.

A highly aggressive parasitic organism, tachyzoites can infect almost any kind of vertebrate cell. A stage of tachyzoites called bradyzoites causes cysts in different kinds of tissues. Muscle cells have extended cysts, while brain cells have fewer spheroid cysts. The size of younger and older cysts differed significantly. Most juvenile cysts are only 10 m in size; however, 100 m size has been documented in early stages. Thousands of closely packed bradyzoites make up the older cysts. On the cyst wall, several invaginations and granular granules have been identified (Guevara *et al*., 2020). Bradyzoites have evolved a style of life that allows them to persist for long periods of time, and cysts might thrive in the intracellular zone. The multilayer structure of the oocyst wall protects them from mechanical and chemical disturbances. The parasite may live for almost a year because to its multilayer structure (Mai *et al.*, 2009). The transmission of infection also differed amongst the animal's group. The disease is spread in intermediate host by sexually (carnivory), the sexual cycle (definitive hosts) and intermediate hosts themselves (Fig. 1). Sexual and asexual cycle as well as infection transmission pattern is determined by the physical characteristics and structure of intermediate and definitive host population in a survival habitat (Afonso *et al.*, 2006).

**Way of transmission:** Reportedly the disease is spread commonly by contaminated food, water, inherited transfer, consumption of oocyst or tissue cysts (Al-Malki, 2021). On the other hand, transmission of diseases varied greatly among communities, owing to differences in eating culture and habits. Unpasteurized milk and tachyzoites infecting blood products can spread the disease (Belluco *et al.*, 2018). Most intermediate hosts have tissue cysts in their brains and muscles. Cats have been documented to get toxoplasmosis via diseased prey *i.e*., rodents or birds, and then serve as hosts for sexual reproduction to continue the enteroepithelial cycle, and the creation of oocysts (Scherrer *et al.*, 2023). Skunks, raccoons, foxes, and bears in the wild can contract toxoplasmosis by ingesting cysts from tissues. Humans get toxoplasmosis by eating infected meat that hasn't been fully cooked meat of mutton or pig (Hill *et al*., 2010). In the host's stomach different enzymes are activated that breakdown the eaten cyst, releasing parasite in bradyzoites form. Parasites have been found to be extremely resilient to protease and to persist in the small intestine of the host organism. Within two weeks of ingestion, faecal contents were estimated to have removed more than 10 million cysts in cats, according to a report. In five days of being discharged into the surroundings, the oocyst sporulates.

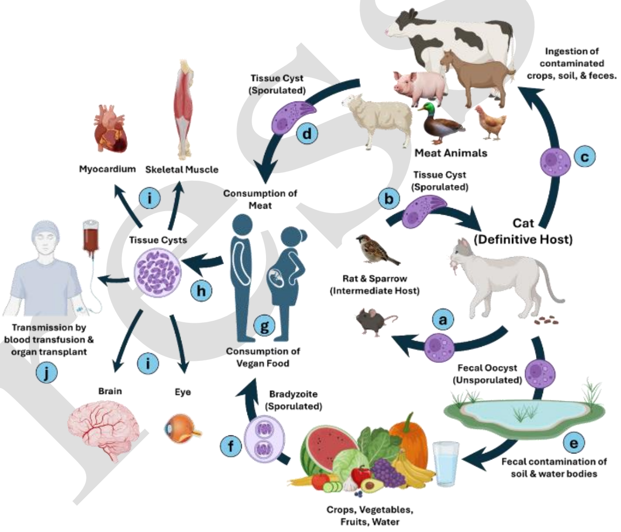


**Fig. 1:** Life cycle of *Toxoplasma gondii* (created with BioRender.com) **a.** Unsporulated oocysts in cat feces. **b.** Sporulated oocysts. **c.** Ingestion of Unsporulated oocysts by intermediate hosts (e.g. cattle, sheep, poultry and swine). **d.** Ingestion of tissue cysts via uncooked meat. **e.** Contamination of vegetables, fruits, crops or drinking water **f.** Ingestion of bradyzoite via vegan food **g.** Intermediate hosts (humans). **h.** Tachyzoites transmitted through the placenta to the fetus. **i.** Tissue cyst. **j.** Transmission by blood transfusion and organ transplant (Naeem *et al.*, 2023).

**Effect of *T. Gondii* infection on human:** Although *T. gondii* has been connected to a number of illnesses and serious clinical outcomes, more investigation is required. *T. gondii* invaded several organs in the human body (Fig. 2). We have summarized *T. gondii*-related human disorders in the review. These include 1) Heart Disease, 2) Encephalitis in AIDS, 3) Prostate cancer, 4) Asthma, 5) Auto-immune disease.

***Encephalitis in AIDS*:** Toxoplasmic Encephalitis (TE) is a lethal disease that disturbs the nervous system of immunocompromised people, such as Acquired immunodeficiency syndrome patient and who have in transplantation process (receiver) (Lee *et al.*, 2014). Above 95% of TE in AIDS patients is caused by the recurrence of long-lasting *T. gondii* infection, which is due to the increasing loss of cellular immune system. In AIDS, a decrease in CD4+ cell count below 100/mm3 causes TE

(Rahman and Rahman, 2020). According to a research done by United States, 10-40% of adult Acquired immunodeficiency syndrome patients have a latent *T. gondii* infection, and around 33% of these people acquire TE (Rahman and Rahman, 2022). Haiti, Africa, America and in Europe have 3 to 4 times the frequency of TE than the United States, owing to a significantly higher rate of latent infection. *T. gondii* specific antibody positive acquired immunodeficiency patients acquire TE in 47 percent of cases, according to an Austrian research (Zangerle *et al.*, 1994).

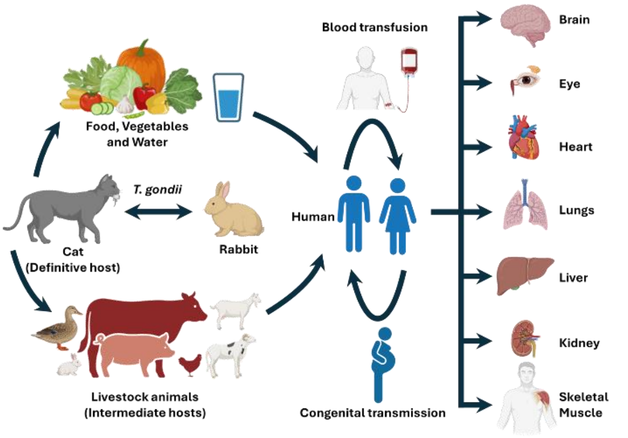


**Fig. 2:** Spread of *Toxoplasma gondii* into various human body organs (created with BioRender.com)

**Heart disease:** The circulatory, neuroendocrine, and immunological systems all interact in chronic heart failure (CHF). The positivity rate for anti-Toxoplasma antibodies (IgG and IgM) in patients with chronic heart failure (n=97) and controller participants (n=50) was investigated in a research using ELISA (Yazar *et al.*, 2006). *T. gondii* IgG antibodies were detected in 66 of 97 (68 percent) patients and 18 of 50 (36 percent) healthy people, according to the research. As a result, *T. gondii* infection is linked to the development of CHF. Furthermore, a further investigation found that *T. gondii* infections are linked to cardiac diseases (Alvarado-Esquivel *et al.*, 2016).

**Prostate cancer:** Testosterone levels in male rats infested with the infection are higher. It has been linked to an increased risk of prostrate tumor and muscular hypertrophy in humans (Lim *et al.*, 2013). Inflammation of the prostate gland and seminal vesicles has been seen in histopathologic examinations of the male sheep's who is infested with *T. gondii* (Lopes *et al.*, 2011). This infection caused a long-lasting inflammatory response in the prostate in a mouse model, resulting in micro-glandular hyperplasia in the irritated prostates, signifying that *T. gondii* might be a hazardous for prostate tumor.

**Asthma:** Toxoplasmosis is linked to an increased risk of asthma. It's plausible since histamine has an anti-inflammatory impact. Asthmatic patients produce a lot of histamine, which influences the cytokine TH1/TH2 balance and has anti-inflammatory properties (Rahman and Rahman, 2022). *T. gondii* infection, on the other hand, predominantly induces Th1 response, which might explain a possible negative link between *T. gondii* infection and incidence of asthma.

**Auto-immune disease:** A class of diseases known as auto-immune illnesses occur when the immune system targets self-antigens. *T. gondii*-specific antibodies were significantly more common in patients with systemic sclerosis (p=0.0001), thyroid autoimmune diseases (p=0.0001), pemphigus vulgaris (p=0.0001) and vasculitis than in control persons (Petrikova *et al.*, 2010). A class of diseases known as auto-immune illnesses occur when the immune system targets self-antigens. T. gondii-specific antibodies were significantly more common in patients with systemic sclerosis (p=0.0001), thyroid autoimmune diseases (p=0.0001), pemphigus vulgaris (p=0.0001) and vasculitis than in control persons.

### *T. gondii* infection in livestock –production and prevalence

**Prevalence in pigs:** Epidemiological investigations revealed that *T. gondii* is found in pigs all throughout the world, with prevalence ranging by age, pig category, region, and management method.

Up to 2009, global data *on T. gondii* infection in pig was examined multiple periods in the past (Dubey, 2009a). More recently, this issue in the United States had examined (Hill and Dubey, 2013). A combined *T. gondii* occurrence of 12.3 percent was found based on reports of direct *T. gondii* identification in pigs, with a 95 % estimate range extending from 0 to 55% (Belluco *et al.*, 2018). In Europe, reports of the prevalence of T. gondii antibodies in particular in breeding sows and piglets ranged from 0% to 64% (De Berardinis *et al.*, 2017). A comprehensive analysis and meta-data inspection of sero-epidemiological study undertaken in Africa revealed that seroprevalences ranged from 9.3% to 40.6%, with a *T. gondii* occurrence in pigs of 26%. (Tonouhewa *et al.*, 2017). Pigs are thought to be the farming animal most commonly infected with *T. gondii* in China (Table 1).

**Infection due to *T. gondii* in pigs:** Although most *T. gondii* infection in pigs are asymptomatic, few incidences of clinical illness following spontaneous infection have been reported across the world (Dubey, 2009a). Anorexia, lethargy, temperature, cyanosis, dyspnea, diarrhea, limb weakness, ocular and nasal discharge neurological indications, and mortality are all common symptoms in clinically infected pigs (Dubey, 2009a). No one of these symptoms are diagnostic of toxoplasmosis. The infection can also be linked to abnormality in pig female reproductive system, which includes abortion, shrinkage of fetus, fetus death, and newborn death (Dubey, 2009a). The medical manifestation of infection in pigs is assumed to be influenced by the age of the host and immunological status, contagion with other pathogens, the dependent stage of *T. gondii* (e.g. tissue cysts, oocyst), infection dosage, and the genetic background of *T. gondii*.

**Sheep and goats:** *T. gondii* infection is common in goats and sheep, and in small ruminants this protozoa organism is a main reason of reproductive sufferers across the world. While most reports and research has been done on sheep (Dubey, 2009b), toxoplasmosis is just as important, if not more so, in goats as an abortive illness (Dubey, 2010).

**Table 1:** Global, regional and national pooled seroprevalence of *T. gondii* in pigs (results from studies performed in 47 countries)

| WHO regions/ country | Number studies | Positive samples/total samples | Pooled prevalence (95% CI) | Weight | Heterogeneity | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | $X^2$ | Df | $I^2$ (%) | *P* value |
| Global | 150 | 23,696/148,092 | 19% (17-22%) | 100 | 20975.69 | 149 | 99.29% | < 0.001 |
| Europe | 51 | 6431/58354 | 13% (10-15%) | 34.32 | 4293.7 | 50 | 98.84% | < 0.001 |
| Netherlands | 4 | 475/7560 | 5% (1-12%) | 2.74 | 390.67 | 3 | 99.23% | < 0.001 |
| Poland | 4 | 548/3119 | 18% (13-24%) | 2.69 | 37.42 | 3 | 91.98% | < 0.001 |
| Finland | 2 | 90/3200 | 3% (2-3%) | 1.37 | NE | 1 | NE | NE |
| Switzerland | 1 | 63/270 | 23% (18-29%) | 1.37 | NE | 0 | NE | NE |
| Sweden | 2 | 97/1779 | 5% (4-7%) | 1.37 | NE | 1 | NE | NE |
| Italy | 8 | 864/6225 | 23% (13-36%) | 5.25 | 624.35 | 7 | 98.88% | < 0.001 |
| Serbia | 4 | 263/1293 | 25% (12-42%) | 2.52 | 90.55 | 3 | 96.69% | < 0.001 |
| Portugal | 3 | 104/968 | 11% (6-16%) | 2.02 | 38.1 | 2 | 92.8% | < 0.001 |
| Spain | 5 | 1435/8573 | 19% (13-26%) | 3.42 | 238.49 | 4 | 98.32% | < 0.001 |
| Austria | 2 | 434/6065 | 7% (7-8%) | 1.37 | NE | 1 | NE | NE |
| Romania | 2 | 1016/4029 | 25% (24-26%) | 0.67 | NE | 0 | NE | NE |
| Czech Republic | 4 | 377/4152 | 10% (1-26%) | 2.71 | 435.86 | 3 | 99.31% | < 0.001 |
| Poland | 4 | 548/3119 | 18% (13-24%) | 2.69 | 37.42 | 3 | 91.98% | < 0.001 |
| Denmark | 1 | 38/254 | 15% (11-20%) | 0.67 | NE | 0 | NE | NE |
| Ireland | 1 | 15/317 | 5% (3-8%) | 0.67 | NE | 0 | NE | NE |
| Greek | 1 | 26/609 | 4% (3-6%) | 0.67 | NE | 0 | NE | NE |
| Latvia | 1 | 34/803 | 4% (3-6%) | 0.67 | NE | 0 | NE | NE |
| UK | 2 | 121/2691 | 4% (4-5%) | 1.37 | NE | 1 | NE | NE |
| Germany | 1 | 140/1500 | 9% (8-11%) | 0.68 | NE | 0 | NE | NE |
| France | 1 | 248/3595 | 7% (6-8%) | 0.67 | NE | 0 | NE | NE |
| Slovakia | 1 | 21/970 | 2% (1-3%) | 0.67 | NE | 0 | NE | NE |
| Estonia | 1 | 22/382 | 6% (4-9%) | 0.67 | NE | 0 | NE | NE |
| S. America | 34 | 2136/9883 | 23% (17-30%) | 22.33 | 1936.35 | 33 | 98.30% | < 0.001 |
| Hawaii | 1 | 247/509 | 49% (44-53%) | 2.01 | NE | 0 | NE | NE |
| Argentina | 3 | 404/827 | 48% (39-58%) | 0.68 | NE | 2 | NE | NE |
| Chile | 1 | 30/340 | 9% (6-12%) | 0.67 | NE | 0 | NE | NE |
| Brazil | 27 | 1386/7974 | 20% (14-27%) | 17.66 | 1235.89 | 26 | 97.90% | < 0.001 |
| Peru | 2 | 99/573 | 30% (24-36%) | 1.3 | NE | 1 | NE | NE |
| N. America | 27 | 7447/50325 | 25% (19-33%) | 18.04 | 8199.72 | 26 | 99.68% | < 0.001 |
| USA | 12 | 5930/35054 | 25% (15-36%) | 8 | 1936.35 | 33 | 98.30% | < 0.001 |
| Canada | 3 | 421/10291 | 5% (0-15%) | 2.06 | NE | 2 | NE | NE |
| Panama | 1 | 93/290 | 32% (27-38%) | 0.67 | NE | 0 | NE | NE |
| Mexico | 7 | 841/4149 | 32% (18-48%) | 4.65 | 5197.4 | 11 | 99.79% | < 0.001 |
| Costa Rica | 1 | 216/496 | 44% (39-48%) | 0.68 | NE | 2 | NE | NE |
| West Indies | 3 | 162/541 | 33% (17-52%) | 1.98 | 524.17 | 6 | 98.86% | < 0.001 |
| African Region | 9 | 875/3050 | 25% (17-34%) | 6.02 | 229.64 | 8 | 96.52% | < 0.001 |
| Ghana | 1 | 260/641 | 41% (37-44%) | 0.68 | NE | 0 | NE | NE |
| Ethiopia | 1 | 129/402 | 32% (28-37%) | 0.68 | NE | 0 | NE | NE |
| Egypt | 1 | 40/100 | 40% (30-50%) | 0.64 | NE | 0 | NE | NE |
| Burkina Faso | 2 | 209/723 | 29% (26-32%) | 1.35 | NE | 1 | NE | NE |
| Nigeria | 1 | 88/302 | 29% (24-35%) | 0.67 | NE | 0 | NE | NE |
| Zimbabwe | 3 | 149/882 | 12% (1-31%) | 1.99 | NE | 2 | NE | NE |
| Oceania | 1 | 1/49 | 2% (1-4%) | 0.60 | NE | 0 | NE | NE |
| Asia | 28 | 6806/26431 | 21% (16-26%) | 18.69 | 2647.92 | 27 | 98.98% | < 0.001 |
| Vietnam | 1 | 87/742 | 27% (24-31%) | 0.68 | NE | 0 | NE | NE |
| Nepal | 1 | 160/587 | 12% (9-14%) | 0.68 | NE | 0 | NE | NE |
| Indonesia | 3 | 50/723 | 7% (1-15%) | 2.01 | NE | 2 | NE | NE |
| Malaysia | 2 | 19/222 | 6% (3-9%) | 1.30 | NE | 1 | NE | NE |
| China | 17 | 4612/19997 | 24% (19-30%) | 11.56 | 1232.95 | 16 | 98.70% | < 0.001 |
| Thailand | 1 | 10/14 | 71% (42-92%) | 47.00 | NE | 1 | NE | NE |
| Taiwan | 2 | 1860/3991 | 47% (45-48%) | 1.34 | NE | 1 | NE | NE |
| Japan | 1 | 8/155 | 5% (2-10%) | 0.66 | NE | 0 | NE | NE |

NE: Not exist UK: United Kingdom, USA: United States; df: degrees of freedom

Moreover, diseases that affect humans also affect animals, and infections in small livestock may be a major factor in the transfer of those diseases to humans (Belluco *et al.,* 2018).

**Prevalence in sheep and goats:** In sheep and goats from all around the world, *T. gondii* antibodies have been found. Europe, Brazil, Middle East and the North America, were the countries with the most seroprevalence reports at the time. Between 2010 and 2018, more papers from regions with sparse data and those where sheep and goats are significant livestock species were published in the field of epidemiology of small ruminants. These studies are from diverse Mediterranean countries, Sub-Saharan African countries and regions of Asia (including Pakistan, China and Southeast Asia) (Tzanidakis *et al.,* 2012; García-Bocanegra *et al.,* 2013; Kantzoura *et al.,* 2013; Gazzonis *et al.,* 2015; Ahmed *et al.,* 2016; Khan *et al.,* 2017; Dong *et al.,* 2018).

In Africa from 1969 to 2016 a recent meta-analysis of data found that the total estimated incidence for sheep was 26.1 percent (17.0–37.0 percent) and for goats was 22.9 percent (12.4–36.0 percent) (Tonouhewa *et al.,* 2017). Goats had a higher antibody rate (62%) than sheep (4.1 to 26 percent) in Egypt (Al-Kappany *et al.,* 2018). *T. gondii* antibodies were detected in 34.52% of goats and 40.2% of sheep in Tunisia (Lahmar *et al.,* 2015). According to Gebremedhin *et al.* (2014), the infection was discovered in

27.6% of goats and 33.7 percent of sheep in Ethiopia. Significant *T. gondii* seroprevalences in flocks (59.7%) and single animals (31.8%) were linked to abortion in multiple sites in another study conducted in the same country (Gebremedhin *et al*., 2014).

A rigorous meta-analysis conducted in the US yielded estimates of *T. gondii* contamination in animal feed prepared in country, as well as small ruminants. The seroprevalence of infection in goats (30.7%) was greater than in lambs or sheep (22.0%) (Guo *et al.,* 2016). Other studies have documented the presence of *T. gondii* in goats and sheep from the Caribbean islands of Dominica (67 percent, 58 percent), Grenada (48 percent, 57 percent), Montserrat (89 percent, 80 percent) and St. Kitts and Nevis (57 percent, 42 percent) (Hamilton *et al*., 2014). An additional survey indicates that 44.1 percent of sheep and 42.8 percent of goats in Grenada and Carriacou (modified Agglutination Test (MAT) titre 1:25) have antibodies against *T. gondii*. 930 sheep had blood samples taken, and after analysis in two different Brazilian climate zones, the overall predictable occurrence was 22.1% (Taylor *et al*., 2021).

In terms of Asia, China's *T. gondii* infection condition was freshly examined. *T. gondii* seroprevalence in sheep was assessed to be 11.8 percent (2306/19,567), whereas seroprevalence in goats was estimated to be 17.6 percent (3263/18,566) (Dong *et al.,* 2018). In Myanmar seroprevalence of 11.4 percent have found in Goat (Bawm *et al.,* 2016). In other states of Asia, occurrence was found at 21.1 percent in sheep and 25.4 percent in goats, correspondingly (Khan *et al.,* 2017). In Pakistan, goats (42.8%) had a greater seroprevalence of *T. gondii* than sheep (26.2%), according to the findings (Ahmed *et al.*, 2016). *T. gondii* antibodies were identified in 36.4 percent (325/891) of sheep and 35.3 percent (198/555) of goats in Saudi Arabia (Alanazi, 2013).

The great occurrences have been found in both goats and sheep. In one research from Greece, sheep shows greater seroprevalence for *T. gondii* infection (48.6% [729/1501]) than goats (30.7 percent [167/542]) (Tzanidakis *et al*., 2012). The 540 goat and sheep serum samples were tested in Thessaly, with a seroprevalence of 24.6 percent (Kantzoura *et al*., 2013). In one more study, specific antibody in contrast to *T. gondii* was found in 53.72 percent of sheep and 61.4 percent of goat from assorted groups, respectively (Diaz *et al.,* 2016). Antibodies were detected in 96.7 percent of goat farmhouse and 87.6 percent of sheep farms in Northern Italy; 41.7 percent of goats and 59.4 percent of sheep tested positive. Sheep had a much greater seroprevalence than goats (Gazzonis *et al.,* 2015). A modified agglutination test (MAT) revealed that 33.7 percent of sheep and 18.6 percent of goat in Portugal were seropositive (Lopes *et al.,* 2013). Seropositivity was found in 248 (49.3 percent) of 503 sheep and 124 (25.1 percent) of 494 goats in Southern Spain. Sheep and goat herd seroprevalence rates were 84.7 percent (61/72) and 72.2 percent (52/72) correspondingly (García-Bocanegra *et al.,* 2013). In one more research conducted in the similar zone, the seroprevalence in sheep was 41.3 percent and 5.7 percent in goat (Almería *et al.,* 2018). Single (48%) and in herd (74%) *T. gondii* prevalence level in goat were rise in northeastern portion of Spain; within-herd occurrence was 53 percent (Díaz *et al*., 2016) (Table 2).

**Infection due to *T. gondii* in sheep and goats:** Toxoplasmosis is thought to be responsible for ten to twenty-three percent of sheep abortions in the US and Europe (Dubey, 2009b). *T. gondii* infection has been connected to 4 to 55 percent of ovine abortions in various parts of the world, including the South and East America, according to recent findings.

The only obvious clinical symptom related with this parasite (horizontal transmission) a short-term fever and depression lasting between 4 and 10 days, starting 3-6 days after infection (Dubey, 1981; Castaño *et al.,* 2016).

**Cattle prevalence:** Estimates of the seropositivity of *T. gondii* in cattle can be determined using highly precise assays, which may be helpful in tracking cow exposure. With the introduction of novel technologies, such as PCR for gene detection, a number of investigations using these methods reported unusually high percentage (10 or 20 percent) of *T. gondi* genes positive samples in the tissue of cow (Amdouni *et al*., 2017; Azizi *et al*., 2014). According to a recent investigation, the percentage of cattle positive for *T. gondii* varies geographically. Compared to North America, Central America has a considerably higher percentage of positive cattle (Belluco *et al*., 2018). This could imply that cow susceptibility to *T. gondii* is influenced by the parasite's worldwide variation in genotype (Bertranpetit *et al*., 2017; Chaichan *et al*., 2017). Future studies will need to address these problems. The prevalence of *T. gondii* precise antibodies in cattle has been extensively studied during the past few decades. Many of these articles have already been evaluated, either globally (Dubey, 2010) or freshly, via concentrating on specific parts of the world such as China (Deng *et al.,* 2018), S. Asia (Khan *et al*., 2017) and Africa (Tonouhewa *et al.,* 2017). Generally, these reviews demonstrate a wide range in described percentage of positive results, with 9.6 percent for cattle in China (Deng *et al.,* 2018), 27.8 percent in South Asia (Khan *et al.,* 2017) and 12.1 percent in Africa (Tonouhewa *et al.,* 2017) (Table 3).

**Economic effect of toxoplasmosis on livestock:** We concentrated on studies that analyzed the expenses of *T. gondii* in farm animal in our evaluation. It is crucial to note, but, that *T. gondii* contaminations in animals used for foodstuff manufacture can have an impact on human health and cost money. These characteristics are hard to evaluate and are away from the scope of this article (Nicholls and Culpepper, 2021).

The economic impact of illnesses generally consists of a number of factors that need to be taken into account. The direct costs of a sickness (C) include not just losses (L), but also costs associated with treating animals (T) and preventing diseases (P). According to Bennett *et al*. (1999), the first factor is "the value of the disease's loss in projected production and/or resource wastage (L)". Among a herd of sheep, the main products are lamb's meat, milk, and wool. For example, in the event of a primary *T. gondii* infection in sheep, there is a high risk of miscarriage (Dubey, 2009b), and the damage is consequently minimum the worth of the new-born lamb. Furthermore, in dairy flocks, fever following acute illness, but especially the incidence of miscarriage, is associated with production of milk loss which is the major cause of earnings for these farms.

**Table 2:** Characteristics of the included cross-sectional studies for prevalence of *T. gondii* in the aborted fetuses and stillbirths of sheep

| First author (Publication year) | Place of study | Sample | Methods | Sample size (n) | Serological results n (%) | Gene | Molecular results n (%) | Histopathology results n (%) |
|---|---|---|---|---|---|---|---|---|
| Seefeldt *et al.* (1989) | USA | Serum | ELISA | 377 | ELISA = 58 (15.38), | -- | -- | -- |
| Dubey and Kirkbride (1989) | USA | Serum | MAT | 30 | 11 (36.66) | -- | -- | -- |
| Wheeler *et al.* (1990) | UK | Placental and fetal tissues | PCR | 5 | -- | P30 | 5(100) | -- |
| Kirkbride (1993) | USA | Serum or body cavity fluids | MAT | 1,784 | 190 (10.7) | -- | -- | 274 (15.35) |
| Steuber *et al.* (1995) | Germany | Placental and fetal tissues | Immunohistochemy and PCR | 47 | -- | B1 | 5 (10.63) | 5 (10.63) |
| Owen and Trees (1999) | UK | Placenta | PCR | 13 | -- | B1 | 13 (100) | -- |
| Hurtado *et al.* (2001) | Spain | Brain, spleen, | Histopathology, IFA, | 53 | 9/41 (21.95) | ITS1 | 10 (18.86) | 5 (9.43) |
| Masala *et al.* (2003) | Italy | Placenta | Nested-PCR | 133 | -- | ITS1 | 42 (31.5) | -- |
| Pereira-Bueno *et al.* (2004) | Spain | Brain, heart, liver, | Histopathology, IFA, ELISA, and nested-PCR | 173 | 30/106 (28.3) | B1 | 12/173 (6.9) | 15/173 (8.7) |
| Williams *et al.* (2005) | UK | Brain, heart, and tongue | Nested-PCR | 70 | -- | SAG1 | 63 (90) | -- |
| Morley *et al.* (2005) | UK | Brain, and cord tissues | Nested-PCR | 155 | -- | SAG1 | 78 (50.32) | -- |
| Agerholm *et al.* (2006) | Denmark | myocardium, liver, skeletal muscle, brain | Histopathology, immunohistochemistry, and EIA | 45 | -- | -- | 3 (6.66) | 3 (6.66) |
| Szeredi *et al.* (2006) | Hungary | Fetal membrane | Immunohistochemistry | 246 | -- | -- | -- | 2 (0.8) |
| Masala *et al.* (2007) | Italy | Fetus and placenta | PCR | 368 | -- | ITS1 | 63 (17.11) | -- |
| Ahmed *et al.* (2008) | Egypt | Internal organs | Nested-PCR | 8 | -- | B1 | 8 (100) | 8 (100) |
| Morley *et al.* (2008) | United Kingdom | Brain, heart, and tongue | Nested-PCR | 22 | -- | SAG1 | 22 (100) | -- |
| Razmi *et al.* (2010) | Irans | Thoracic and abdominal fluids | IFA | 325 | 17 (5.2) | -- | -- | |
| Kamani *et al.* (2010) | Nigeria | Brain, and placenta | Nested-PCR | 31 | -- | ITS1 | 0 (0) | |
| de Moraes *et al.* (2011) | Brazil | Brain, cerebellum, , liver, and placenta | Histopathology and nested-PCR | 35 | -- | B1 | 5 (14.3) | 5 (14.3) |
| Rassouli *et al.* (2011) | Iran | Brain | Histopathology, nested-PCR, and IFA | 200 | -- | B1 | 27 (13·5%) | 0 (0) |
| Moreno *et al.* (2012) | Spain | Brain, lung, heart, liver, spleen, and kidney | Histopathology and nested-PCR | 74 | -- | ITS1 | 4 (5.4) | -- |
| Habibi *et al.* (2012) | Iran | Brain | Histopathology, nested-PCR, and bioassay | 18 | -- | B1 | 12 (66.66) | 0 (0) |
| Chessa *et al.* (2014) | Italy | Placenta, brain, and liver | Nested-PCR | 161 | -- | ITS1 | 21 (13.04) | -- |
| Danehchin *et al.* (2016) | Iran | Brain | Semi-nested-PCR | 37 | -- | B1 | 20 (54) | -- |
| Schnydrig *et al.* (2017) | Switzerland | Placenta, liver, and lung | Histopathology and real-time PCR | 17 | -- | B1 | 3 (17.64) | -- |
| Díaz-Cao *et al.* (2018) | Spain | Brain | Real-time PCR | 11 | -- | -- | 0 (0) | -- |
| Shahbazi *et al.* (2019) | Iran | Brain | Histopathology, IFA, and nested-PCR | 75 | 21 (28) | GRA6 | 48 (64) | -- |
| Partoandazanpoor *et al.* (2019) | Iran | Brain | Histopathology and nested-PCR | 111 | -- | B1 | 9 (8.1) | -- |
| Sah *et al.* (2019) | Bangladesh | Brain, liver, and placenta | PCR | 5 | -- | 529 bp repetitive and B1 | 4 (80) | -- |

**Table 3:** Characteristics of the included cross-sectional studies for prevalence of *T. gondii* in the aborted fetuses and stillbirths of cattle

| First author (Publication year) | Place of study | Sample | Methods | Sample size (n) | Gene | Molecular results n (%) | Histopathology results n (%) |
|---|---|---|---|---|---|---|---|
| Reitt *et al.* (2007) | Switzerland | Fetus and placenta | Immunohistochemistry and real-time PCR | 223 | B1 | 0/76 (0) | 8 (3.59) |
| Moore *et al.* (2008) | Argentina | Brain, and placenta | Nested-PCR | 70 | ITS1 | 0 (0) | -- |
| Yang *et al.* (2012a, b) | China | Brain | Nested-PCR | 80 | SAG1 | Fetus: 1 (1.3) and dam blood: 4 (5) | -- |
| Cabral *et al.* (2013) | Brazil | Brain, thymus, and placenta | Histopathology and PCR | 105 | 529 bp repetitive | 0 (0) | 75 (71.43) |
| Ozkaraca *et al.* (2017) | Turkey | Brain, and thymus | Duplex PCR, immunohistochemistry, and immunofluorescence | 102 | ITS1 | 0 (0) | 0 (0) |
| Díaz-Cao *et al.* (2018) | Spain | Brain | Real-time PCR | 25 | -- | 0 (0) | -- |
| Sah *et al.* (2019) | Bangladesh | Brain and placenta | PCR | 2 | 529 bp repetitive and B1 | 0 (0) | -- |

The other part of an economic analysis is the cost of treating afflicted animals (T) (Bennett, 2003). In toxoplasmosis case, cure expenditures may include medications for inflammation to lower fever or other veterinarian facilities.

**Conclusion:** The review of toxoplasmosis examines the frequency, transmission, and epidemiology of the illness in different populations. The main ways that toxoplasma infection spreads are through humans, cattle, and roaming animals. Eating uncooked or partially cooked meals carrying the parasite can spread *T. gondii*. Socioeconomic status has been found to be one of the decisive factors in sickness epidemiology. Despite the numerous studies on *T. gondii* infections in cattle that have been compiled in these publications, there are still some important gaps in our knowledge. In herbivores, the pathways of infections appear to be obvious, with oocysts being the most likely source. However, there remains uncertainty over a number of matters, such as the impact of pollution from pastures and water, as well as the level of cultivation. The significance of oocyst infection or the presence or absorption of sick intermediate species, such as rats, is unknown, though. Overall, to have a better knowledge and level of confidence in the epidemiologically significant T pathways infection with *T. gondii* in cattle, more epidemiological research is required, and the quality of these studies has to be improved. More research on the commercial costs of *T. gondii* infection and toxoplasmosis in cattle is clearly needed. This study has implications for academics, politicians, healthcare providers, and veterinary professionals. One of the lesser-known diseases that the public has to be made aware of is toxoplasmosis. As a result, spreading awareness of toxoplasmosis caused by *T. gondii* needs to be prioritized more.

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