Pakistan Veterinary Journal

ISSN: 0253-8318 (PRINT), 2074-7764 (ONLINE)
DOI: 10.29261/pakvetj/2024.221

REVIEW ARTICLE

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

Iqra Ayub[1], Sayydah Suryya Sani[1], Ahmad Zaman[1], Muhammad Razzaq Shahid[1], Waqas Bashir[1], Misbah Ijaz[2], Saba Zafar[1] and Azhar Rafique[1]*

[1]Department of Zoology, Government College University Faisalabad, Pakistan
[2]Department of Clinical Medicine and Surgery, FVS, University of Agriculture, Faisalabad, Pakistan
*Corresponding author: azharrafique96@gmail.com

ARTICLE HISTORY (24-083)

Received: February 12, 2024
Revised: June 14, 2024
Accepted: June 21, 2024
Published online: August 08, 2024

Key words:
AIDS
Toxoplasmosis
Life cycle
Transmission

ABSTRACT

Toxoplasma gondii (*T. gondii*) is an obligate intracellular protozoan parasite and causes infection both in humans and animals. *T. gondii*, unlike most species, is easily manipulated genetically in the laboratory. Recent developments in the field highlight genetic research. By establishing the relationships between genotype and clinical manifestations, the researcher can uncover associations with a species, virulence and resistance to drugs. Genetic knowledge is imperative for improving diagnostic accuracy and developing more effective treatments. The tachyzoite, bradyzoite, and sporozoite are the 3 infective phases of *T. gondii*. In a complicated life cycle, several stages are entangled. This review highlights the current understanding about the history, prevalence, and consequences of *T. gondii* infection in livestock and humans, 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, on-farm efficient biosecurity procedures may be adopted. It is necessary to have knowledge about possible risk factors involved in disease causation and spread. 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.

To Cite This Article: Ayub I, Sani SS, Zaman A, Shahid MR, Bashir W, Ijaz M, Zafar S and Rafique A, 2025. Prevalence of *Toxoplasma gondii* in humans and animals and its economic impacts. Pak Vet J, 45(1): 1-9. http://dx.doi.org/10.29261/pakvetj/2024.221

INTRODUCTION

Toxoplasma gondii (*T. gondii*) is a protozoan parasitic infection that is common in animals and humans all over the world. It has become the most common serious disease among acquired immunodeficiency syndrome (AIDS) patients. Toxoplasmosis is a recurrent 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* 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. The clinical consequences of toxoplasmosis were initially described in children with retino-chorditis, hydrocephalus and encephalitis in the early 1920s. *T. gondii* has become a common and serious disease in AIDS patients since 1980s and has immunosuppressive effects (Lim and Othman, 2014). In the same way, Remington (2006) described the scientific consequence of the infections in a variety of immunologically suppressed patients, going 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. Proper health care guiding congenital and parental toxoplasmosis was extremely helpful in preventing complications in early diagnosis of toxoplasmosis. Most of the afflicted neonates and vision impairment were found to be asymptomatic at delivery, were also documented after some years.

After an initial *T. gondii* infection, it can induce chronic or recurring infections (Remington, 2006; Chen *et al.*, 2022). Cats play an important role in spreading toxoplasmosis as definitive hosts that produce oocysts in

their feces, contaminating soil, water and food. Both males and females of all ages are infested with *T. gondii*. 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 (Dubey and Jones, 2014). Several researchers found that pet cats do not cause *T. gondii* infections in people (Paul *et al.,* 2021; Almuzaini, 2023; Munir *et al*., 2023); nonetheless, rare investigations found that such cats provide a significant risk of parasite infection (Jones *et al.,* 2009). In most of the developing and developed nations, the seroprevalence of *T. gondii* caused 14 to 77% 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. gondii* (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 µm, and has a conical frontal end and rounded later end.

Tachyzoite, as a highly aggressive parasitic organism, 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 10µm in size however 70µm has been documented in early stages. Thousands of closely packed bradyzoites make up the older cysts. On the cyst wall, several invaginations and coarse 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. At this stage the parasite may live for almost a year because of its multilayer structure (Mai *et al.,* 2009). The transmission of infection also differs amongst the animal's group. *T. gondii* reproduces through two life cycles: the sexual (definitive host) and the asexual (intermediate host). The disease is spread in intermediate hosts by sexually (carnivory), the sexual cycle (definitive hosts) and intermediate hosts themselves (Fig. 1). Sexual and asexual cycle as well as infection transmission patterns are 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 also spread the disease (Belluco *et al.,* 2018). Most of the 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 entero-epithelial 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 has not been fully cooked (Hill *et al*., 2010). In the host's stomach, different enzymes are activated that breakdown the eaten cyst and release parasites 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, fecal contents were estimated to have more than 10 million per mL cysts in cats (Kalogeropoulos *et al.,* 2022). The cysts discharged along feces into the environment are sporulated within five days.

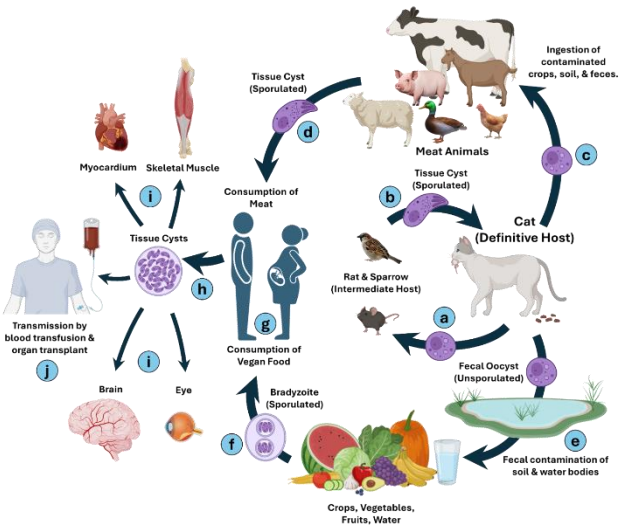

Fig. 1: Life cycle of *Toxoplasma gondii* (created with BioRender.com) **a.** Unsporulated oocysts in cat feces. **b.** Sporulated oocysts. **c.** Ingestion of Unsporulated oocysts by intermediate hosts (e.g. cattle, sheep, poultry and swine). **d.** Ingestion of tissue cysts via uncooked meat. **e.** 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

Effect of *T. gondii* infection on humans: 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 summarizes *T. gondii*-related human disorders in this review. These include 1) heart disease, 2) Encephalitis in AIDS, 3) Prostate cancer, 4) Asthma, 5) Auto-immune disease**.**

***Encephalitis in AIDS*:** Toxoplasmic Encephalitis is a lethal disease that interrupts the nervous system of immunocompromised people, such as acquired immunodeficiency syndrome (AIDS) patient and who have in transplantation process (receiver) (Lee *et al.,* 2014). Above 95% of toxoplasmic encephalitis 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 toxoplasmic encephalitis (Rahman and Rahman, 2020; Pawelczyk *et al*., 2022). According to a research in the United States of America, 10-40% of adult AIDS patients have a latent *T. gondii* infection, and around 33% of these people acquire toxoplasmic encephalitis (Rahman and Rahman, 2022). Latin America, Europe and Haiti have 3 to 4 percent higher infection than United States. Austia shows 47% toxoplasmic encephalitis in patients having positive AIDS (Zangerle *et al*., 1994: Karayigit *et al.,* 2020) Haiti, Africa, America and in Europe have 3 to 4 times the frequency of toxoplasmosis than the United States, owing to a significantly higher rate of latent infection (Zangerle *et al.,* 1994).

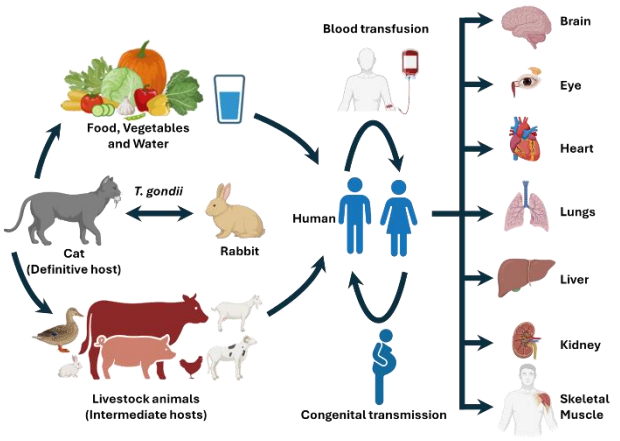

Fig. 2: Spread of *Toxoplasma gondii* into various human body organs (created with BioRender.com)

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 controlled participants (n=50) was investigated in 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. As a result, *T. gondii* infection is linked to the development of CHF. Furthermore, it was found that *T. gondii* infections are linked to cardiac diseases (Alvarado-Esquivel *et al.,* 2016; Panazzolo *et al.,* 2023).

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; Layton *et al*., 2023). Inflammation of the prostate gland and seminal vesicles has been observed in histopathologic examinations of the male sheep 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 exasperated prostates, signifying that *T. gondii* might be hazardous for prostate tumor.

Asthma: Toxoplasmosis is linked to an increased risk of asthma. It is 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 (Fenoy *et al.,* 2015; Soto *et al*., 2017; Elsadek *et al.,* 2022).

Auto-immune disease: Auto-immune illness occurs 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).

T. gondii infection in livestock production and prevalence

Prevalence in pigs: Epidemiological investigations revealed that *T. gondii* is found in pigs throughout the world, with prevalence ranging by age, pig category, region, and management method (Dubey *et al*., 2020). Global data on *T. gondii* infection in pigs was examined multiple periods in the past (Dubey, 2009a) and this issue had been examined in the United States (Hill and Dubey, 2013). A combined *T. gondii* occurrence of 12.3% 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 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 of 26% in pigs (Tonouhewa *et al.,* 2017). Pigs are thought to be the farming animal most infected with *T. gondii* in China (Table 1).

Infection due to *T. gondii* in pigs: Although mostly the *T. gondii* infections 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). 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, oocysts), 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 protozoan is a main reason for reproductive sufferers across the world. While most of the reports and research have been done on sheep and goats (Dubey, 2009b), it concluded that the toxoplasmosis is an abortive illness in goats (Dubey, 2010; Rafique *et al*., 2022).

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.

Prevalence in sheep and goats: In sheep and goats from all around the world, *T. gondii* antibodies have been reported (Belluco *et al.,* 2018). 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 major livestock species were published in the field of epidemiology of small ruminants (Belluco *et al.,* 2018). 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 meta-analysis reported that the total estimated incidence for sheep was 26.1% (17.0-37.0%) and for goats was 22.9% (12.4–36.0%) (Tonouhewa *et al.,* 2017). Goats had a higher antibody rate (62%) than sheep (4.1 to 26%) 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% of sheep in Ethiopia. *T. gondii* seroprevalences in flocks (59.7%) and single animals (31.8%) were linked to abortion in multiple sites in another study (Gebremedhin *et al*., 2014).

The seroprevalence of infection in goats (30.7%) was greater in lambs or sheep (22.0%) (Guo *et al*., 2016). Other

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	Immunohistochemistry 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	529bp repetitive and B1	0 (0)	--

studies have documented the presence of *T. gondii* in goats and sheep from the Caribbean islands of Dominica (67%, 58%), Grenada (48%, 57%), Montserrat (89%, 80%) and St. Kitts and Nevis (57%, 42%) (Hamilton *et al*., 2014). An additional survey indicates that 44.1% of sheep and 42.8% of goats in Grenada and Carriacou (modified Agglutination Test (MAT) titre 1:25) have antibodies against *T. gondii* (Taylor *et al*., 2021; Naeem *et al*., 2023).

T. gondii seroprevalence in sheep in China was assessed to be 11.8% (2306/19,567), whereas

seroprevalence in goats was estimated to be 17.6% (3263/18,566) (Dong *et al.,* 2018). In Myanmar, seroprevalence of 11.4% has been found in Goat (Bawm *et al.,* 2016). In other states of Asia, occurrence was found at 21.1% in sheep and 25.4% in goats, correspondingly (Khan *et al.,* 2017). In Pakistan, a greater seroprevalence of *T. gondii* in goats (42.8%) than sheep (26.2%) is reported (Ahmed *et al.,* 2016). *T. gondii* antibodies were identified in 36.4% (325/891) of sheep and 35.3% (198/555) of goats in Saudi Arabia (Alanazi, 2013).

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

Infection due to *T. gondii* in sheep and goats: Toxoplasmosis is thought to be responsible for 10-23% of sheep abortions in the US and Europe (Dubey, 2009b). *T. gondii* infection has been connected to 4 to 55% of ovine abortions in various parts of the world, including South and East America, according to recent findings.

The only obvious clinical symptom related with this parasite (horizontal transmission) is a short-term fever and depression lasting between 4 and 10 days, starting 3-6 days after infection (Castaño *et al.,* 2016; Feitosa *et al*., 2023).

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, several investigations using these methods reported unusually high percentage (10 or 20%) of *T. gondi* genes positive samples in the tissues of cow (Azizi *et al*., 2014; Amdouni *et al*., 2017). 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 are solicited to address these problems incurred by toxoplasmosis. 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 globally (Dubey, 2010) concentrating on specific parts of the world such as China (Deng *et al.,* 2018; Zahoor *et al*., 2023), South Asia (Khan *et al*., 2017; Arafa *et al*., 2023) and Africa (Tonouhewa *et al.,* 2017). Generally, the current review demonstrates a wide range of percentage of positive results, with 9.6% for cattle in China (Deng *et al.,* 2018), 27.8% in South Asia (Khan *et al.,* 2017) and 12.1% in Africa (Tonouhewa *et al.,* 2017) (Table 3).

Economic effect of toxoplasmosis on livestock: High rates of *T. gondii* in small and large ruminants lead to severe economic losses and serious zoonotic health hazards to susceptible populations. It is crucial to note that *T. gondii* contaminations in animals used for foodstuff manufacture can have an impact on human health and cost for treatment obviously (Dubey *et al*., 2020).

The economic impact of illnesses generally consists of a few factors that need to be considered. The direct costs of a sickness include not just losses but also costs associated with treating animals and preventing diseases. According to Bennett *et al*. (1999), the first factor is "the value of the disease's loss in projected production and/or resource wastage. Among a herd of sheep, the main products are lamb's meat, milk, and wool. Sheep infected with protozoan parasite may cause early embryonic death, resorption and mummification (Vilela *et al*., 2024) In an event of a primary *T. gondii* infection in sheep, there is a high risk of miscarriage (Dubey, 2009b; Mehnaz *et al*., 2023). 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.

The other part of an economic analysis is the cost of treating afflicted animals (Bennett, 2003; Arala *et al*., 2023). In toxoplasmosis case, cure expenditures may include medications for inflammation to lower fever or other veterinarian facilities.

Conclusions: The review of toxoplasmosis examines the frequency, transmission, and epidemiology of toxoplasmosis in different populations. The main ways that toxoplasma infection spreads are through humans, cattle and roaming animals. In this review we summarize the impact of *T. gondii* in various human diseases *i.e*., heart disease, encephalitis in AIDS patients, cancer etc. 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 several 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 unknot completely known yet. More research is needed to develop preventive measures to control this important parasite at national and international levels. Overall, to have a better knowledge and level of confidence in the pathways of infection with *T. gondii* in cattle, more epidemiological research is required, and the quality of these studies must 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 must be made aware of is toxoplasmosis. As a result, spreading awareness of toxoplasmosis caused by *T. gondii* needs to be prioritized more.

Conflict of interest: The Authors declare that there is no conflict of interest

Authors contribution: IA, SSS AH and MRS, conceived and designed the review, WB, MI and SZ corrected and edited the final manuscript AR conceptualized, critically revised the manuscript and critically revised the manuscript for important intellectual contents and approved the final version.

REFERENCES

Afonso E, Thulliez P and Gilot-Fromont E, 2006. Transmission of *Toxoplasma gondii* in an urban population of domestic cats (*Felis catus*). Int J Parasitol 36:1373-82.

Agerholm JS, Aalbaek B, Fog-Larsen AM, *et al*., 2006. Veterinary and medical aspects of abortion in Danish sheep. APMIS 114: 146-52.

Ahmed H, Malik A, Arshad M, *et al*., 2016. Seroprevalence and spatial distribution of toxoplasmosis in sheep and goats in North-Eastern Region of Pakistan. Korean J Parasitol 54:439-46.

Ahmed YF, Sokkar SM, Desouky HM, *et al*., 2008. Abortion due to toxoplasmosis in small ruminants. Glob Vet 2:337-42.

Alanazi AD, 2013. Determination of seropositivity for *Toxoplasma gondii* in sheep, goats and camels slaughtered for food and human consumptions in Riyadh municipal abattoirs, Saudi Arabia. J Egypt Soc Parasitol 43:569-76.

Al-hajj M and Kekillioğlu A, 2023. A review on *Toxoplasma gondii* (Nicolle & Manceaux, 1908) (Apicomplexa: Sarcocystidae). Int J Adv Nat Sci Eng Res 15:336-44.

Al-Kappany YM, Abbas IE, Devleesschauwer B, *et al*., 2018. Seroprevalence of anti-*Toxoplasma gondii* antibodies in Egyptian sheep and goats. BMC Vet Res 14:1-5.

Almuzaini AM, 2023. Flow of zoonotic toxoplasmosis in food chain. Pak Vet J 43(1):1-16.

Al-Malki ES, 2021. Toxoplasmosis: stages of the protozoan life cycle and risk assessment in humans and animals for an enhanced awareness and an improved socio-economic status. Saudi J Biol Sci *28*:962-969.

Almería S, Cabezón O, Paniagua J, *et al*., 2018. *Toxoplasma gondii* in sympatric domestic and wild ungulates in the Mediterranean ecosystem. Parasitol Res 117:665-71.

Alvarado-Esquivel C, Salcedo-Jaquez M, Sanchez-Anguiano LF, *et al*., 2016. Association between *Toxoplasma gondii* exposure and heart disease: A case-control study. J Clin Med Res 8:402-09.

Amdouni Y, Rjeibi MR, Rouatbi M, *et al*., 2017. Molecular detection of *Toxoplasma gondii* infection in slaughtered ruminants (sheep, goats and cattle) in Northwest Tunisia. Meat Sci 133:180-84.

Arafa F, Said H, Osman D, *et al*., 2023. Nanoformulation-based1,2,3-triazole sulfonamides for anti-Toxoplasma in vitro study. Trop Med Infect Dis 8:401.

Azizi H, Shiran B, Boroujeni AB, *et al*., 2014. Molecular survey of *Toxoplasma gondii* in sheep, cattle and meat products in Chaharmahal va Bakhtiari Province, Southwest of Iran. Iran J Parasitol 9:429-34.

Bawm S, Maung WY, Win MY, *et al*., 2016. Serological survey and factors associated with *Toxoplasma gondii* infection in domestic goats in Myanmar. Scientifica 2016

Belluco S, Simonato G, Mancin M, *et al*., 2018. *Toxoplasma gondii* infection and food consumption: a systematic review and meta-analysis of case-controlled studies. Crit Rev Food Sci Nutr 58:3085-96.

Bennett R, 2003. The 'direct costs' of livestock disease: the development of a system of models for the analysis of 30 endemic livestock diseases in Great Britain. J Agric Econ 54:55-71.

Bennett R, Christiansen K and Clifton-Hadley R, 1999. Preliminary estimates of the direct costs associated with endemic diseases of livestock in Great Britain. Prev Vet Med 39:155-71.

Bertranpetit E, Jombart T, Paradis E, *et al*., 2017. Phylogeography of *Toxoplasma gondii* points to a South American origin. Infect Genet Evol 48:150-55.

Cabral AD, Camargo CN, Galleti NTC, *et al*., 2013. Screening for *Toxoplasma gondii* in aborted bovine fetuses in Brazil. Arq Inst Biol 80:103-105.

Castaño P, Fuertes M, Regidor-Cerrillo J, *et al*., 2016. Experimental ovine toxoplasmosis: influence of the gestational stage on the clinical course, lesion development and parasite distribution. Vet Res 47:1-14.

Chaichan P, Mercier A, Galal L, *et al*., 2017. Geographical distribution of *Toxoplasma gondii* genotypes in Asia: A link with neighboring continents. Infect Genet Evol 53:227-38.

Chen R, Peng JJ, Mohsin M, *et al*., 2022. Construction and evaluation of the *Toxoplasma gondii* DNA vaccine targeting DEC-205. Pak Vet J 42(2): 256-260.

Chessa G, Chisu V, Porcu R, *et al*., 2014. Molecular characterization of *Toxoplasma gondii* Type II in sheep abortion in Sardinia, Italy. Parasite 21:6.

Danehchin L, Razmi G and Naghibi A, 2016. Isolation and genotyping of *Toxoplasma gondii* strains in ovine aborted fetuses in Khorasan Razavi Province, Iran. Korean J Parasitol 54:15-20.

De Berardinis A, Paludi D, Pennisi L, *et al*., 2017. *Toxoplasma gondii*, a foodborne pathogen in the swine production chain from a European perspective. Foodborne Pathog Dis 14:637-48.

de Moraes ÉPB, da Costa MM, Dantas AFM, *et al*., 2011. *Toxoplasma gondii* diagnosis in ovine aborted fetuses and stillborns in the State of Pernambuco, Brazil. Vet Parasitol 183:152-55.

Deng H, Devleesschauwer B, Liu M, *et al*., 2018. Seroprevalence of *Toxoplasma gondii* in pregnant women and livestock in the mainland of China: a systematic review and hierarchical meta-analysis. Sci Rep 8:6218-28.

Díaz P, Cabanelas E, Díaz JM, *et al*., 2016. Seroprevalence of *Toxoplasma gondii* and Neospora caninum in goats from north-western Spain. Ann Agric Environ Med 23:587-90.

Díaz-Cao JM, Lago AP, Lorenzo GL, *et al*., 2018. Broadening the diagnosis panel of reproductive pathogens associated with abortion in ruminants. Span J Agric Res 16, 17.

Dong H, Su R, Lu Y, *et al*., 2018. Prevalence, risk factors, and genotypes of *Toxoplasma gondii* in food animals and humans (2000–2017) from China. Front Microbiol 9:2108. https://doi.org/10.3389/fmicb.2018.02108

Dubey JP and Jones J, 2014. Comments on" Detection of *Toxoplasma gondii* in raw caprine, ovine, bovine, and camel milk using cell cultivation, cat bioassay, capture ELISA, and PCR methods in Iran". Foodborne Pathog Dis 11:500-501.

Dubey JP and Kirkbride CA, 1989. Enzootic toxoplasmosis in sheep in north-central United States. J Parasitol 75:673-76.

Dubey JP, 1992. Isolation of *Toxoplasma gondii* from a naturally infected beef cow. J Parasitol 78:151-53.

Dubey JP, 2009a. Toxoplasmosis in pigs-the last 20 years. Vet Parasitol 164:89-103.

Dubey JP, 2009b. Toxoplasmosis in sheep-the last 20 years. Vet Parasitol 163:1-14.

Dubey JP, 2010. *Toxoplasma gondii* infections in chickens (*Gallus domesticus*): prevalence, clinical disease, diagnosis and public health significance. Zoon Pub Health 57:60-73.

Dubey JP, Murata FHA, Cerqueira-Cézar *et al*., 2020. Public Health Significance of Toxoplasma gondii Infections in Cattle: 2009–2020. J Parasitol 106, 772–788

Elsadek A, Eraky M, ElBishry M, *et al*., 2022. Seropostivity of *Toxoplasma gondii* in children with bronchial asthma at Benha University Hospital, Egypt. Zag Uni Med J 1164-1168.

Feitosa TF, Vilela VLR, Batista SP, *et al*., 2023. Genetic diversity of *Toxoplasma gondii* in goats and sheep from the Northeast Region of Brazil destined for human consumption. Curr Res Parasitol Vector Borne Dis 5:100163.

García-Bocanegra I, Cabezón O, Hernández E, *et al*., 2013. *Toxoplasma gondii* in ruminant species (cattle, sheep, and goats) from southern Spain. J Parasitol 99:438-40.

Gazzonis AL, Veronesi F, Di Cerbo AR, *et al*., 2015. *Toxoplasma gondii* in small ruminants in Northern Italy–prevalence and risk factors. Ann Agric Environ Med 22:62-68.

Gebremedhin EZ, Abdurahaman M, Tessema TS, *et al*., 2014. Isolation and genotyping of viable *Toxoplasma gondii* from sheep and goats in Ethiopia destined for human consumption. Parasites Vectors 7:1-8.

Guevara RB, Fox BA and Bzik DJ, 2020. Toxoplasma gondii parasitophorous vacuole membrane-associated dense granule proteins regulate maturation of the cyst wall. MSphere 5:e00851-19.

Guo M, Mishra A, Buchanan RL, *et al*., 2016. A systematic meta-analysis of *Toxoplasma gondii* prevalence in food animals in the United States. Foodborne Pathog Dis 13:109-18.

Habibi GR, Imani AR, Gholami MR, *et al*., 2012. Detection and identification of *Toxoplasma gondii* type one infection in sheep aborted fetuses in Qazvin Province of Iran. Iran J Parasitol 7:64-72.

Hamilton CM, Katzer F, Innes EA, *et al*., 2014. Seroprevalence of *Toxoplasma gondii* in small ruminants from four Caribbean islands. Parasites Vectors 7:1-4.

Hill DE, Haley CHA, Wagner B, *et al*., 2010. Seroprevalence of and risk factors for *Toxoplasma gondii* in the US swine herd using sera collected during the National Animal Health Monitoring Survey (Swine 2006). Zoon Pub Health 57:53-59.

Hill DE and Dubey JP, 2013. *Toxoplasma gondii* prevalence in farm animals in the United States. Int J Parasitol 43:107-13.

Hurtado A, Aduriz G, Moreno B, *et al*., 2001. Single tube nested PCR for the detection of *Toxoplasma gondii* in fetal tissues from naturally aborted ewes. Vet Parasitol 102:17-27.

Jones JL, Dargelas V, Roberts J, *et al*., 2009. Risk factors for *Toxoplasma gondii* infection in the United States. Clin Infect Dis 49:878-84.

Kalogeropoulos D, Sakkas H, Mohammed B, *et al*., 2022. Ocular toxoplasmosis: a review of the current diagnostic and therapeutic approaches. Int Ophthalmol 42: 295-321.

Kamani J, Egwu GO, Mani AU, *et al*., 2010. Survey of *Toxoplasma gondii* DNA in aborted Ovine and Caprine fetuses by nested PCR in Borno state, Nigeria. Vet World 3:360-63.

Kantzoura V, Diakou A, Kouam MK, *et al*., 2013. Seroprevalence and risk factors associated with zoonotic parasitic infections in small ruminants in the Greek temperate environment. Parasitol Int 62:554-60.

Karayigit MO and Dincel GC, 2020. Role of ADAMTS-13 and nNOS expression in neuropathogenesis of listeric encephalitis of small ruminants. Biotechnic Histochem 95:584-596

Khan MU, Rashid I, Akbar H, *et al*., 2017. Seroprevalence of *Toxoplasma gondii* in South Asian countries. Rev Sci Tech 36:981-96.

Kirkbride CA, 1993. Diagnoses in 1,784 ovine abortions and stillbirths. J Vet Diagn 5:398-402.

Lahmar I, Lachkhem A, Slama D, *et al*., 2015. Prevalence of toxoplasmosis in sheep, goats and cattle in Southern Tunisia. J Bacteriol Parasitol 6:1-4.

Layton J, Theiopoulou DC, Rutenberg D, *et al.* 2023. Clinical spectrum, radiological findings, and outcomes of severe toxoplasmosis in immunocompetent hosts: A systematic review. Pathogens 12(4):543.

Lee SH, Lee SE, Seo MG, *et al*., 2014. Evidence of *Toxoplasma gondii* exposure among horses in Korea. J Vet Med Sci 76:1663-65.

Lim A, Kumar V, Hari Dass SA *et al*., 2013. *Toxoplasma gondii* infection enhances testicular steroidogenesis in rats. Mol Ecol 22:102-110.

Lim SSY and Othman RY, 2014. Recent advances in *Toxoplasma gondii* immunotherapeutics. Korean J Parasitol 52:581-93.

Lopes AP, Dubey JP, Neto F, *et al*., 2013. Seroprevalence of *Toxoplasma gondii* infection in cattle, sheep, goats and pigs from the North of Portugal for human consumption. Vet Parasitol 193:266-69.

Lopes WD, Santos TR, Luvizotto MCR, *et al*., 2011. Histopathology of the reproductive system of male sheep experimentally infected with *Toxoplasma gondii*. Parasitol Res 109:405-09.

Mai K, Sharman PA, Walker RA, *et al*., 2009. Oocyst wall formation and composition in coccidian parasites. Mem Inst Oswaldo Cruz 104:281-89.

Masala G, Porcu R, Daga C, *et al*., 2007. Detection of pathogens in ovine and caprine abortion samples from Sardinia, Italy, by PCR. J Vet Diagn Invest 19:96-98.

Masala G, Porcu R, Madau L, *et al*., 2003. Survey of ovine and caprine toxoplasmosis by IFAT and PCR assays in Sardinia, Italy. Vet Parasitol 117:15-21.

Mehnaz S, Sajid MS, Aslam MA, *et al*., 2023. Culicoides: A neglected parasite of prime importance in selected areas of Pakistan. Continental Vet J 3(2):86-93.

Montoya JG and Liesenfeld O, 2004. Toxoplasmosis. Lancet 363:1965-76.

Moore DP, Regidor-Cerrillo J, Morrell E, *et al*., 2008. The role of *Neospora caninum* and *Toxoplasma gondii* in spontaneous bovine abortion in Argentina. Vet Parasitol 156:163-67.

Moreno B, Collantes-Fernández E, Villa A, *et al*., 2012. Occurrence of *Neospora caninum* and *Toxoplasma gondii* infections in ovine and caprine abortions. Vet Parasitol 187:312-18.

Morley EK, Williams RH, Hughes JM, *et al*., 2005. Significant familial differences in the frequency of abortion and *Toxoplasma gondii* infection within a flock of Charollais sheep. Parasitology 131:181-85.

Morley EK, Williams RH, Hughes JM, *et al*., 2008. Evidence that primary infection of Charollais sheep with *Toxoplasma gondii* may not prevent foetal infection and abortion in subsequent lambings. Parasitology 135:169-73.

Munir F, Sindhu ZUD, Tayyeb, M, *et al*., 2023. Rationale to develop mRNA-based vaccines for *Trypanosoma brucei* (a review). Continental Vet J 3(1):26-35.

Naeem MI, Younus M. Nisa Q, *et al*., 2023. Zoonotic infertility due to toxoplasma gondii. In: Khan A, Abbas RZ, Hassan MF, Aguilar-Marcelino L, Saeed NM and Mohsin M (eds), Zoonosis, Unique Scientific Publishers, Faisalabad, Pakistan, 2:412-422.

Owen MR and Trees AJ, 1999. Genotyping of *Toxoplasma gondii* associated with abortion in sheep. J Parasitol 85:382-84.

Ozkaraca M, Irehan B, Parmaksiz A, *et al*., 2017. Determination of *Neospora caninum* and *Toxoplasma gondii* in aborted bovine foetuses by duplex PCR, immunohistochemistry and immunofluorescence methods. Med Weter 73:346-51.

Panazzolo G, Kmetiuk L, Domingues O, *et al*., 2023. One health approach in serosurvey of Toxoplasma gondii in former black slave (Quilombola) communities in Southern Brazil and among their dogs. Trop Med Infect Dis 8:377.

Partoandazanpoor A, Sadeghi-Dehkordi Z, Ekradi L, *et al*., 2020. Molecular diagnosis and pathological study of *Toxoplasma gondii* in aborted caprine and ovine fetuses in borderline of Iran–Iraq. Acta Parasitol 65:187-92.

Paul N, Rahman A and Rahman T, 2021. Serologic detection of *Toxoplasma gondii* in cat owners residing at Dhaka Metropolitan Area of Bangladesh. Adv Microbiol 11:257-65.

Pereira-Bueno J, Quintanilla-Gozalo A, Pérez-Pérez V, *et al*., 2004. Evaluation of ovine abortion associated with *Toxoplasma gondii* in Spain by different diagnostic techniques. Vet Parasitol 121: 33-43.

Petrikova J, Agmon-Levin N, Shapira Y, *et al*., 2010. Prevalence of Toxoplasma antibodies among patients with various autoimmune diseases. Ann Rheum Dis 69:8-11.

Rahman T and Rahman A, 2022. Impact of *Toxoplasma gondii* infection on human health. Bioresearch Commun 8:1093-1099.

Rafique A, Nasir S, Ashraf A, Nawaz Z, Zahid FM, Abbas A and Masood S, 2022. Serosurveillance and risk factors analysis of caprine toxoplasmosis in Faisalabad Punjab, Pakistan. Pak Vet J 42(1): 102106.

Rassouli M, Razmi GR, Bassami MR, *et al*., 2011. Study on ovine abortion associated with *Toxoplasma gondii* in affected herds of Khorasan Razavi Province, Iran based on PCR detection of fetal brains and maternal serology. Parasitology 138:691-97.

Razmi GR, Ghezi K, Mahooti A, *et al*., 2010. A serological study and subsequent isolation of *Toxoplasma gondii* from aborted ovine fetuses in Mashhad area, Iran. J Parasitol 96:812-14.

Reitt K, Hilbe M, Voegtlin A, *et al*., 2007. Aetiology of bovine abortion in Switzerland from 1986 to 1995–a retrospective study with emphasis on detection of *Neospora caninum* and *Toxoplasma gondii* by PCR. J Vet Med A 54:15-22.

Remington JP, 2006. *Remington: the science and practice of pharmacy* (Vol. 1). Lippincott Williams & Wilkins.

Sah RP, Dey AR, Rahman AA, *et al*., 2019. Molecular detection of *Toxoplasma gondii* from aborted fetuses of sheep, goats and cattle in Bangladesh. Vet Parasitol Reg Stud Reports 18:100347.

Scherrer P, Ryser-Degiorgis MP, Marti IA, *et al*., 2023. Exploring the epidemiological role of the Eurasian lynx (Lynx lynx) in the life cycle of Toxoplasma gondii. Int J Parasitol Parasites Wildl 21:1-10.

Schnydrig P, Vidal S, Brodard I, *et al*., 2017. Bacterial, fungal, parasitological and pathological analyses of abortions in small ruminants from 2012–2016. Schweiz Arch fur Tierheilkd 159:647-56.

Seefeldt SL, Kirkbride CA and Dubey JP, 1989. Comparison of enzyme-linked immunosorbent assay, indirect fluorescent antibody test, and direct agglutination test for detecting *Toxoplasma gondii* antibodies in naturally aborted ovine fetuses. J Vet Diagn Invest 1:124-27.

Senderowski C, Bojar Z, Formanek B, *et al*., 2010. Tribological behaviour of Fe-Al intermetallic coatings obtained by HVOF method. Inżynieria Materiałowa 31:390-93.

Shahbazi G, Rad NH, Madani R, *et al*., 2019. *Toxoplasma gondii* in aborted fetuses of sheep in Ardebil Area, North-West of Iran. Iran J Parasitol 14:430-35.

Soto AS, Fenoy IM, Sanchez VR, *et al*. 2017. *Toxoplasma gondii* serine protease inhibitor -1: Anew adjuvant candidate for asthma therapy. PLoS ONE 12(10):e0187002.

Steuber S, Niu A, Bauer C, *et al*., 1995. The detection of *Toxoplasma gondii* in abortion tissues of sheep using the polymerase chain reaction. DTW. Deutsche tierarztliche Wochenschrift 102:91-93.

Szeredi L, Jánosi S, Tenk M, *et al*., 2006. Epidemiological and pathological study on the causes of abortion in sheep and goats in Hungary (1998-2005). Acta Vet Hung 54:503-15.

Taylor C, Brodbelt DC, Dobson B, *et al*., 2021. Spatio temporal distribution and agroecological factors associated with canine leptospirosis in Great Britain. Prev Vet Med 193:105407.

Tonouhewa ABN, Akpo Y, Sessou P, *et al*., 2017. *Toxoplasma gondii* infection in meat animals from Africa: systematic review and meta-analysis of sero-epidemiological studies. Vet World 10:194-208.

Tzanidakis N, Maksimov P, Conraths FJ, *et al*., 2012. Toxoplasma gondii in sheep and goats: seroprevalence and potentia risk factors under dairy husbandry practices. Vet Parasitol 190:340-48.

Vilela VLR, Feitosa TF, Simões SVD *et al*., 2023. An abortion storm in a goat farm in the Northeast Region of Brazil was caused by the atypical *Toxoplasma gondii* genotype #13. Curr Res Parasitol Vector Borne Dis 21:100157.

Vilela VLR and Feitosa TF 2024. Recent advances in *Toxoplasma gondii* infection and Toxoplasmosis. Trop Med Infec Dis 9(7):160.

Wheeler R, Wilmore H, Savva D, *et al*., 1990. Diagnosis of ovine toxoplasmosis using PCR. Vet Rec 126:249.

Williams RH, Morley EK, Hughes JM, *et al*., 2005. High levels of congenital transmission of *Toxoplasma gondii* in longitudinal and cross-sectional studies on sheep farms provides evidence of vertical transmission in ovine hosts. Parasitology 130:301-07.

Yang N, Cui X, Qian W, *et al*., 2012a. Survey of nine abortifacient infectious agents in aborted bovine fetuses from dairy farms in Beijing, China, by PCR. Acta Vet Hung 60:83-92.

Yang N, Mu MY, Li HK, *et al*., 2012b. Seroprevalence of *Toxoplasma gondii* infection in slaughtered chickens, ducks, and geese in Shenyang, northeastern China. Parasite Vectors 5:1-4

Yazar S, Gur M, Ozdogru I, *et al*., 2006. Anti-*Toxoplasma gondii* antibodies in patients with chronic heart failure. J Med Microbiol 55:89-92.

Zahoor J, Kashif M, Nasir A, *et al*., 2023. Sero-epidemiology, therapeutic study and the risk factors associated with Trypanosomiasis (Surra) in camels in district Jhang, Punjab, Pakistan. Continental Vet J 3(1):42-48.

Zangerle R and Allerberger F, 1994. High risk of developing toxoplasmic encephalitis in AIDS patients seropositive to *Toxoplasma gondii*. J Acquir Immune Defic Syndr 7:207-208.