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Review

Review on Zoonotic Importance, Diagnostic Methods and Control Strategies of Toxoplasmosis in Human and Animals

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Abstract

The obligatory intracellular protozoan parasite Toxoplasma gondii is the cause of toxoplasmosis, a zoonotic disease that is common in both veterinary and human medicine. Mostwarm-blooded animals are susceptible to infection by this parasite. Although the disease is subclinical and poorly understood, timely and precise diagnosis is crucial for the use of control and preventative strategies. This review was aimed to elucidate the zoonotic importance, diagnostic methods and control strategies of toxoplasmosis in human and animals. Humans are reservoir hosts for T. gondii, while cattle, sheep, goats, poultry, pigs, and camels are the definitive hosts, along with domestic cats and other felids. According to this, the parasite can only finish its sexual life cycle in these specific host species; its environmentally resistant oocysts can only be expelled along with the infected Felids' excrement. Oocysts play a crucial role in T. gondii's life cycle. If consumed, they can infect a wide range of warm-blooded intermediate hosts after one to several days of development (sporulation) in the environment. In tissue cysts, tachyzoites and bradyzoites are second-order entities. The preferred locations for tissue cyst formation include the brain, skeletal, and cardiac muscles, as well as the retina of infected intermediate hosts. It is acknowledged as a primary factor in both human and animal abortion. Consuming raw meat, food, or water tainted with oocysts and vertically (congenital infection) is the primary way that the disease is contracted. Since felidae play a major role in the spread of T. gondii by shedding oocysts, prevention and control measures for animals should concentrate on preventing hunting and feeding of the species. Humans can maintain control by properly cleaning raw vegetables and meats, using gardening gloves, and washing their hands with soap and water. As a One Health illness, toxoplasmosis poses a serious threat to human health as well as that of domestic animals, wildlife, and ecosystems. People who depend on animal resources also view it as a threat. Public education and awareness creation are crucial. Furthermore, the control of the diseases depends on a precise diagnosis of toxoplasmosis made using molecular and microscopic methods.

Keywords: Animals, Control, Diagnosis, Humans, Toxoplasmosis

INTRODUCTION

Toxoplasma gondii, an intracellular protozoan parasite, is the cause of the neglected zoonotic parasitic illness toxoplasmosis (Al-Malki, 2021).While gondii was initially obtained from the rodent Ctenodactylus gingundi in Tunis in 1908 (Lahmar *et al*., 2020) and a rabbit from South America the same year (Smith *et al*., 2021), the term T. gondiis is derived from the Greek word (toxon: bow and plasma: shape) (Mose *et al*., 2020). Most warm-blooded mammals are likely susceptible to infection by the parasite (Schlüter *et al*., 2014; Hide, 2016). According to Ferreira *et al*. (2019), domestic cats and other felids are T. gondii's confirmed hosts. According to Stelzer *et al*. (2019), this suggests that the parasite can only finish its sexual life cycle in these species, and that the only way

to release environmentally resistant oocysts is through the excrement of infected Felids.

One of the biggest obstacles to the growth of the livestock business is protozoan parasites. Among these, Toxoplasma gondii, an obligatory intracellular parasite, is the cause of the global zoonotic illness toxoplasmosis (Hill and Dubey, 2002). The three main morphological forms of T. gondii are tissue cyst, trophozoite, and oocyst, and they are more likely to survive and proliferate in warm, humid environments (Kenneth and George, 2004). All vertebrate animals, including humans, are susceptible to toxoplasma gondii's wide host range (Dubey, 2010). Humans and other animals serve as intermediate hosts for oocysts released into the environment by cats and wild felids, who are the sole definitive hosts (Bayarri *et al*., 2012). Cats can become infected by ingesting oocysts with food or water or by consuming cysts found in the tissues of infected intermediate hosts (Tenter *et al*., 2001). However, intake of live cysts in undercooked meat, unpasteurized milk, food and water contaminated with oocysts, and congenitally during pregnancy are the ways in which intermediate hosts harbor T. gondii (Blood *et al*., 2007).

According to Edwards and Dubey (2013), toxoplasmosis is a disease that is economically significant to the livestock industry and causes reproductive issues in farm animals, including stillbirths, abortions, postnatal mortality, and fetal abnormalities. A major risk to public health arises from zoonotic transmissions of Toxoplasma gondii (Tenter *et al*., 2001). The Centers for Disease Control and Prevention (CDC) has designated five parasitic diseases as the focus of public health initiatives, with toxoplasmosis being the primary cause of food-borne illness-related deaths in humans (Scallan *et al*., 2011). According to Edwards and Dubey (2013), T. gondii is extremely common in farm animals and around one-third of the human population has a chronic infection. Immunocompromised people and expectant mothers experience significant issues with it, including encephalitis, epilepsy, schizophrenia, lymphadenopathy, and ophthalmic problems (Kijlstra and Jongert, 2009).

Antitoxoplasma antibodies in serum or the agent's isolation from infected patients' tissues are the two most popular serological methods used to diagnose toxoplasmosis (Gamble *et al*., 2005). Although there isn't a suitable medication or vaccine to prevent T. gondii at this time, cats can get cooked meat, avoid eating raw meat, and dispose of their waste appropriately. These are important preventative and control measures (Elmore *et al*., 2010).

Ethiopia is the country with the most publications on toxoplasmosis among Eastern African nations (Teweldemedhin *et al*., 2019; Tarekegn *et al*., 2020). Gelaye *et al*. (2015) found that toxoplasmosis was highly prevalent in Ethiopian humans and animals, but accurate data on the disease's prevalence is scarce (Tadesse *et al*., 2014). Regular research on humans and animals will be required to accurately determine prevalence. The two primary ways that people contract toxoplasmosis are by eating tissue cysts from undercooked meat that contain bradyzoites or by ingesting oocysts from food products like vegetables and fruit or water tainted by sporozoites found in the excrement of cats (MirzaAlizadeh *et al*., 2018). Because they are the only hosts capable of excreting the environmentally resistant oocytes, felids play a crucial role in the epidemiology of T. gondii (Ahmad, 2018). In small ruminants, age, sex, height, and water supply all significantly predict T. gondii seropositivity (Tegegne *et al*., 2016; Jilo *et al*., 2021).The reservoir hosts help spread the illness from one another to the ultimate host (Rahman *et al*., 2018; Khalil, 2013).

Due to their high susceptibility to infection, sheep and goats are particularly vulnerable to this parasite, which is thought to be a major cause of reproductive failure in small ruminants. This parasite also increases the risk of abortive disease in these animals and results in large financial losses. Controlling T. gondii infections in livestock is crucial because farm animals can both serve as a potential reservoir for the parasite and a direct source of infection for humans. Toxoplasmosis is extremely difficult to prevent and control due to its complicated life cycle, necessitating a multidisciplinary and comprehensive approach (Pepe *et al*., 2021). This makes a precise diagnosis of toxoplasmosis using microscopic and molecular techniques essential. Therefore, the objectives of this paper are to review the main diagnostic methods of toxoplasmosis infection in animal and human and to describe the means of the parasite infection, prevention and control in domestic animals, and wildlife.

Toxoplasmosis

Charles Nicolle and Louis Manceaux of the Institute of Pasteur in Tunis made the initial discovery of Toxoplasma in the desert rodent Ctenodactylusgundii in 1908. Around the same period, Toxoplasma was independently found in a rabbit at Sao Paulo by Alfonso Splendore (Dubey, 2010). The name Toxoplasma was given to it because of its crescent shape (Greek: toxon = arc, plasma = form). The identification of T. gondii as a common parasite of warm-blooded hosts with a global distribution occurred in 1948 with the development of a T. gondii specific antibody test, the Sabin-Feldman dye test.

Etiology

Toxoplasma gondii is an obligatory intracellular protozoan parasite belonging to the phylum Apicomplexa and order Coccidia that causes toxoplasmosis. Although it has a wide range of intermediate hosts, it is a particular

parasite belonging to the definitive host felidae family (Jones and Dubey, 2012). There are three infectious phases in Toxoplasma gondii: a) Tachyzoites: The parasite's quickly proliferating form, which is present in intermediate hosts during the acute stage of infection. b) Oocysts, which are exclusively seen in cat feces and include sporozoites (Klaus, 2003). c) Tissue cysts: walled formations that are frequently discovered in the central nervous system and muscles, and which are home to latent Toxoplasma gondii bradyzoites (Alvarado-Esquivel, 2003).

Host Range and Susceptibility

The epidemiology of toxoplasmosis is largely dependent on cats, and the disease is almost nonexistent in regions without cat populations. 60% of cats in the USA are serologically positive for the toxoplasma antigen, with most of them becoming infected through predation, according to epidemiological studies. As might be predicted, infections are more common in humans, agricultural animals, and stray cats (Urquhart *et al*., 1996). Due to the usual serological tests for cattle being inaccurate, the true seroprevalence of toxoplasmosis in cattle is unknown; therefore, the low seroprevalence in cattle is indicative of the relative unimportance of toxoplasmosis in cattle (Montoya and Liesenfeld, 2004). An epizootic condition in pigs has been reported in the USA; toxoplasma was retrieved following mice inoculation with material from the mother piglets' brain to explain the symptoms, lesions, and organisms observed in the piglets' lung, liver, kidneys, and lymph nodes. Given that oocysts released into the environment are relatively few in ruminants, especially sheep, there is a distinct frequency of toxoplasmosis in these animals. The majority of the time, pregnant ewes became infected during concentrated feeding sessions before lambing because the stored food was tainted with millions of oocysts from cat excrement (Urquhart *et al*., 1996).

Although they have been found in urine, feces, milk, saliva, and semen, tachyzoites are not expected to cause illness. Bradyzoites are more resistant to stomach digestion than tachyzoites, and they cannot endure in the environment for very long. According to Stover *et al*. (1990), oocysts can survive for over a year in both indoor and outdoor environments. Oocytes are incredibly resilient to environmental changes; in warm, humid areas, they can spread infection for up to a year, and longer in colder regions. In recent times, toxoplasma oocytes have been linked to earthworms, flies, cockroaches, and snails as transport hosts. A woman's uterine infection may cause the infant to die. It is estimated that up to one-third of people on the planet are infected with toxoplasma (Montoya and Liesenfeld 2004). According to estimates, toxoplasmosis affects between 30 and 65 percent of people on Earth (Tenter *et al*., 2000).

Sources of Infection

The most common ways to contract Toxoplasma gondii infection are through eating or drinking food and water tainted with the parasite's resistant stage (oocystes), which is excreted in the feces of infected cats, or by consuming the parasite's encysted stage (tissue cysts), which is present in contaminated meat. The following are typical sources of T. gondii infection: The only source of infection for sheep, cattle, and horses is cat excrement. Tissues of intermediate hosts generally rodents and small birds, although any species can be an intermediate host (IH) for Toxoplasma gondii cats contract the parasite by consuming the tissues of these hosts. For a very long time, rodents act as infection reservoirs. Animal meals tainted by cat excrement from cat nests. Feral cat excrement that has been surface-buried is brought to the surface by earthworms and other soil dwellers, contaminating pastures. Pig biting their tails or ears while consuming meat, dead rodents, cannibalized piglets, and blood (Radostitis, 1994).

Epidemiology of Toxoplasmosis in human and animals

Toxoplasma gondii is found all over the world. Though highly varied, seroprevalence rates are generally 10–30% in North America, Northern Europe, and Southeast Asia; 30–50% in Central and Southern Europe; and higher in tropical regions of Africa and Latin America. Periodically, there are small epidemics, which are typically linked to tainted food or water. It is believed that immunity to the majority of strains is lifelong. Although large numbers of humans and animals have been exposed to this organism even in extremely cold places like the Arctic, it is more common in warm, humid settings (Radostitis, 1994).

Human seroprevalence rates of Toxoplasma gondii have varied between different groups of people, ranging from 74.4% to 96.7% (Woldemichael *et al*., 1998; Yimer *et al*., 2005; Shimelis *et al*., 2009; Gebremedihin *et al*., 2013). The two main ways that humans contract T. gondii infection are through eating raw meat that contains live tissue cysts and drinking water or food tainted with oocysts. Animals with the infection typically exhibit T. gondii cysts in various bodily tissues and humans can become infected by consuming such raw or undercooked tissues. Eating undercooked or cured pork products is thought to be the cause of 63 percent of human toxoplasmosis infections in Europe (Cook *et al*., 2000; Dubey, 2004, 2008).

Pregnant women may be at risk for congental trans-

mission if they consume meat from sheep and cattle that has been infected with T. gondii. Public health may be at risk from freshly consumed homemade cheeses created on small, family-run farms using tainted milk that hasn't been pasteurized. Vertical transmission in humans has been linked to varied morbidity, stillbirths, and miscarriages (Tenter *et al*., 2000). Water-borne transmission of T. gondii was thought to be rare until recently, when a significant human outbreak was connected to feral cats contaminating a municipal water reservoir in Canada (Dubey, 2010). In soil, earthworms, dung beetles, flies, and cockroaches can all manually disperse oocysts. It is known that they can endure for extended periods of time on fruits and vegetables (Kniel *et al*., 2002).

Nearly all homeothermic animals, including humans, are susceptible to infection by Toxoplasma gondii. The incidence of the disease varies among species based on socio-cultural practices, geographic location, and climate. The presence of cats that deposit oocysts, which become infectious to humans and animals following sporulation, may also be linked to the prevalence rate. Domestic cats and other felines excrete Toxoplasma gondii oocysts, which lead to widespread environmental contamination. The sporulated oocysts can live in damp soil for months or years (Dubey, 2010).

Risk Factors

Infection with Toxoplasma gondii is linked to the following risk factors: Oocysts have a year-long survival rate in the environment and exhibit high resistance to external stimuli, making them a pathogen risk factor. Temperatures of 90°C or 194°F for 30 seconds and 50°C for 2.5 minutes are known to kill oocysts. The prevalence of clinical illness is largely reliant on T. gondii strains (Bowie *et al*., 1997).

Toxoplasma gondii infection is linked to environmental and management risk factors, such as feeding farm animals on grass polluted with cat excrement and creating an environment that is conducive to the survival of infectious oocystes. Because of the prolonged survival of oocysts on pasture caused by excessive rainfall, a significant rate of infection has been seen in sheep. Compared to intensive farming systems, extensive animal husbandry carries greater risk. Host risk factors: While sheep raised in high rainfall areas with cats can have an infection rate as high as 32%, sheep raised in high rainfall areas without cats nearly never get toxoplasmosis. By intimate contact with a severely infected placenta, direct transfer from sheep to sheep may take place (Bowie *et al*., 1997). Infants born to mothers who first contract toxoplasma during or shortly before pregnancy, as well as individuals with significantly compromised immune systems, such as those living with HIV/AIDS, are two risk factors for developing toxoplasmosis in humans (less than 5 and older than 65). Acute toxoplasma infection or the recurrence of a prior infection can both cause illness (Conrad *et al*., 2005).

Transmission

The most prevalent routes of toxoplasmosis transmission include by the transplacental route, raw meat or tissue cysts, unpasteurized milk, eating food or drink contaminated by oocysts spread by cats and other felines, and cleaning the cat's litter box (Mohamed, 2020). Toxoplasma gondii oocysts can live for a very long time in the soil. A person who comes into close contact with dirt or kids playing in the ground can become infected through contaminated or dirty hands (Hussain *et al*., 2017). First of all, it can be sent from intermediate hosts to definitive hosts and vice versa. Second, the parasite can spread from one permanent host to another. Thirdly, Figure 1 below shows the relationship between intermediate hosts and this mode of transmission.

Life Cycle of Toxoplasma gondii

The two phases of the life cycle of T. gondii are the sexual phase, which occurs in the definitive host, and the asexual phase, which occurs in any warm-blooded animal, including humans and birds. According to Dubey (2009), the sexual phase of the life cycle of T. gondii takes place in the intestine of the cat, and the tachyzoites, which develop into tissue cysts in neural, eye, and muscle tissue, spread throughout the body. After a varied number of schizogonies, bradyzoites pierce the small intestine's epithelial cells and start a series of multiple generations that eventually result in the formation of microgamonts and macrogamonts. As per Rahman *et al*., (2018), the macrogamonts are fertilized by the microgamonts. Figure 2 below summarizes the life cycle of T. gondii in both humans and domestic animals.

Pathogenesis

Consumption of feces containing oocysts, congenital infection, or carnivorrism are the three ways that Toxoplasma gondii infections occur (Prusa *et al*., 2013). When raw meat harboring tissue cysts is consumed by an unexposed cat, T. gondii starts to replicate the entero epithelium. After undergoing sexual replication and being freed from tissue cysts by digestion in the stomach and small intestine, bradyzoites infiltrate intestinal epithelium, resulting in the discharge of oocysts in the stool. Oocysts are released for up to 20 days following infection, with the first appearance being in the stool three days after infection. Following a 24-hour exposure to the air oocysts sporulate, become infectious, and can live for up to a

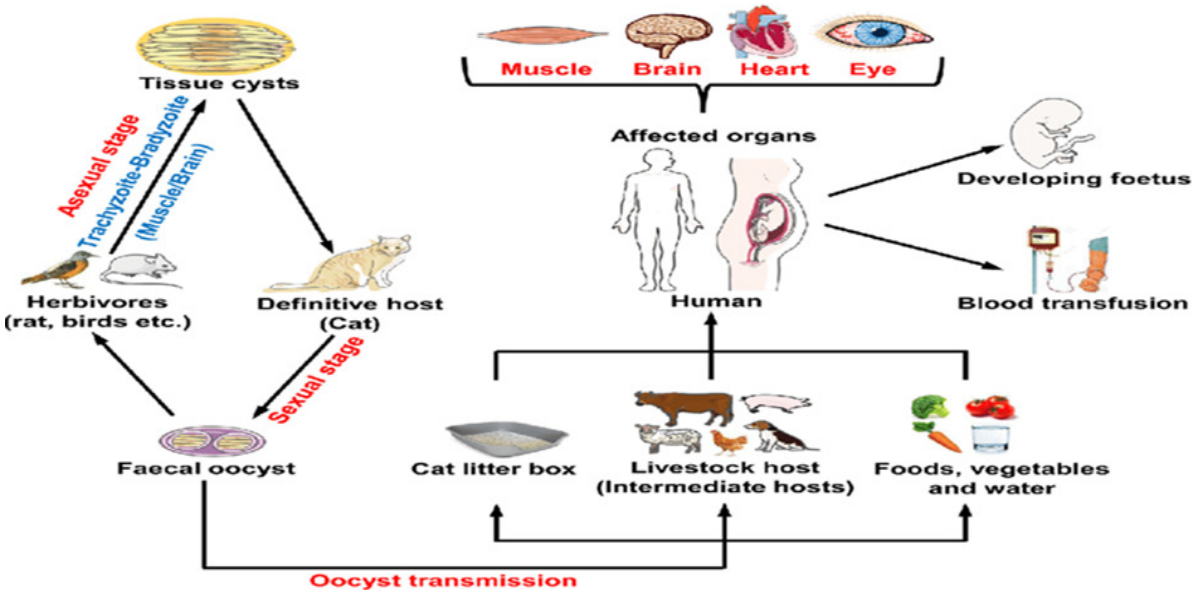

Figure 1. Transmission routes of toxoplasmosis

Source: (Rahman *et al.,* 2018)

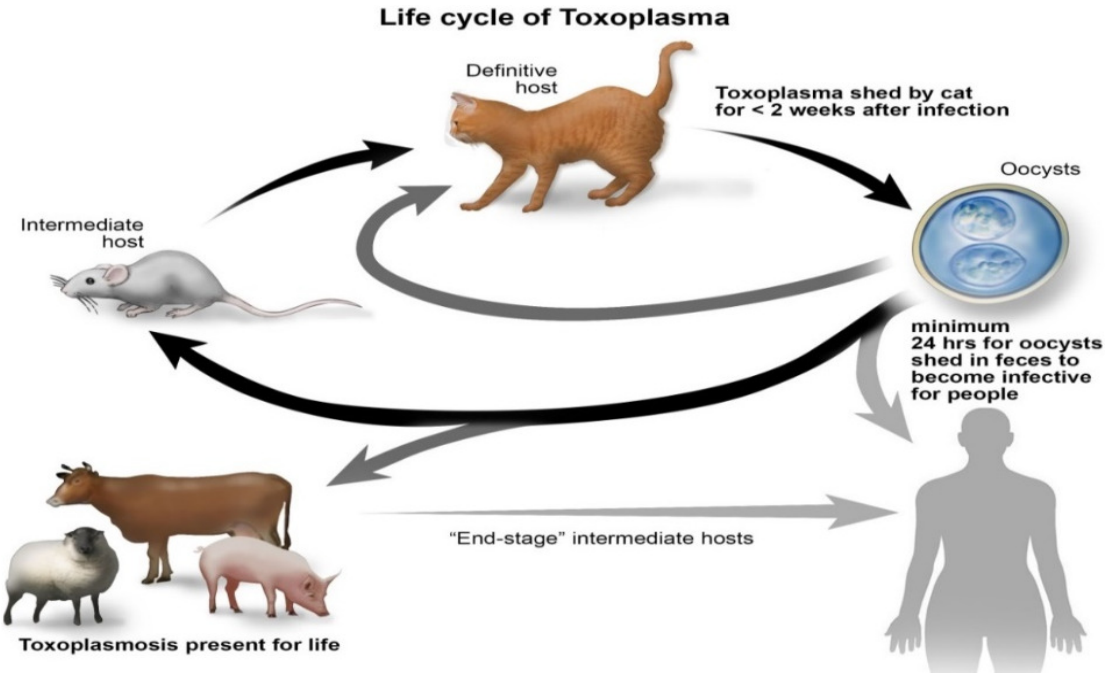

Figure 2: Life cycle of *T. gondii* in domestic animals and in humans

Source: (Rahman *et al*., 2018)

year in the environment. Cats only ever shed oocysts once in their lifetime since they often get immune to T. gondii after the initial infection (Conrad *et al*., 2005).

Toxoplasma gondii causes further intestinal replication in all warm-blooded animals upon consumption of raw meat containing tissue cysts or feed tainted with oocyte-bearing cat excrement. Intestinal epithelium becomes infected by discharged bradyzoites and sporozoites, respectively. Following multiple rounds of epithelial replication, tachyzoites appear and spread into lymphatic and vascular systems. All over the body, chyzoites attack tissues and multiply intracellularly until the cells rupture, resulting in tissue necrosis. At this point, widespread toxoplasmosis may kill young, immuno-compromised animals (Conrad *et al*., 2005). In reaction to the tachyzoites, older animals develop a robust cell-mediated

immune response. Typically, tissue cysts are observed in the heart and skeletal muscles, as well as in neurons. For many years, tissue cysts can still proliferate within their host (Susan, 1998).

Incubation Period

The length of the incubation period depends on the infection dose. Animal incubation times are most likely comparable to human incubation times, which range from five to twenty-three days. Adults can contract Toxoplasma gondii infection anywhere from 10 to 23 days after consuming undercooked meat and 5 to 20 days after consuming cat feces oocysts (Dubey, 1998). Five to six days after being vaccinated, kittens in the experimental group experienced diarrhea. Years after an animal was infected, reactivation can happen (Radostitis, 1994).

Clinical Signs of Toxoplasmosis in Domestic Animals and humans

Clinical toxoplasmosis is more severe in cats, who often experience indications of ascites, lethargy, and dyspnea along with hepatitis or cholangiohepatitis, pneumonia, and encephalitis. Hepatic failure, hyperplastic cholangitis, and hepatitis with abdominal involvement have not yet been reported cases (Cohen *et al.,* 2016). Furthermore reported conditions include thickening of the gastric wall as a result of eosinophilic fibrosing gastritis, extra-intestinal enteritis, inflammatory intestinal illness, and regional lymphadenopathy. In cats exhibiting severe respiratory or neurological indications, the condition may be swiftly lethal (De Tommasi *et al*., 2014).

Dogs may exhibit neurological illness, including paresis or paralysis in cases of encephalomyelitis, ataxia, seizures, and cranial nerve impairments (Gerhold *et al*., 2014). Other canine cases of Toxoplasmosis that have been documented include: ocular disease characterized as necrotizing conjunctivitis, anterior uveitis, endo-phthalmitis, and chorioretinitis; and noise sensitivity myositis, which first manifested as an aberrant gait, muscular wasting, and stiffness (Migliore *et al*., 2017). The most common clinical manifestation of acute toxoplasmosis in humans is enlarged lymph nodes, which can also cause fever, exhaustion, headache, sore throat, and muscle soreness. On the other hand, eye illness is a common and dangerous side effect of T. gondii infection (Dubey, 2021).

Congenital Toxoplasmosis

After receiving antibiotic therapy, most newborns with congenital toxoplasmosis go on to develop normally. Serious difficulties can, however, arise within the first few years of life in as many as 4% of instances. These consist of demise, irreversible brain injury, and permanent visual impairment (loss of vision in one or both eyes, partial or total) (Bowie, 1997).

Diagnostic Methods of Toxoplasmosis

Microscoic Diagnostic Methods

The generic and inconsistent nature of T. gondii infection's clinical signs makes them difficult to diagnose (Tenter *et al*., 2000; Boothroyd, 2002). T. gondii has only been found under a microscope in tissue, water, and fecal samples. On the other hand, identification just using microscopy is less accurate and sensitive. The tissue cysts can be dyed, which aids in separating the parasites from the host cells, and the oocysts in feces can be enriched from huge quantities of samples by centrifugation for analysis. According to Silva *et al*. (2010), staining with Giemsa, Hematoxylin and Eosin, and Periodic Acid Schiff is an easy and affordable method that is frequently utilized to stain the amylopection granules in Bradyzoites. These techniques take a while to complete and demand a high level of expertise to produce accurate detection outcomes. Although it is difficult to use for normal use, electron microscopes are also used to detect tissue cysts in mouse brains and oocysts in the small intestines of sick cats (Liu *et al*., 2015).

Bioassay

Bioassays are one of the most sensitive methods for finding cysts in animal tissues, and they are widely regarded as the gold standard for identifying T. gondii infection. Possible specimens utilized for the isolation include excretions, secretions, bodily fluids, lymph nodes, and tissues from the muscles and brain. Cats and mice are frequently employed in T. gondii bioassays. INF-gamma knockout mice are recommended because of their greater susceptibility to T. gondii infection, which can lead to a better success rate in T. gondii isolation. All things considered, the bioassay is costly and time-consuming (typically taking six weeks). Consequently, it is unsuitable for widespread screening (Dubey *et al*., 2013).

Serological Assays

Most people with toxoplasma gondii infection have vague clinical symptoms; serological testing is the primary method used to diagnose the infection. Different antibody classes or antigens can be detected using a range of serological assays, including dye test, MAT, ELISA, IFAT,

Table 1. Summary of serological methods for detection of T. gondii infection

Serological methods	Antigens or antibodies used	Antibody/antigen type tested
DT	Live tachyzoite	IgG, IgM, IgA
MAT	Formalin-fixed tachyzoite	IgG
IFAT	Killed whole tachyzoite	IgG, IgM
IHA	Tanned red blood cells sensitized with soluble antigens	IgG
ELISA	Tachyzoite lysate antigen, recombinant antigens, specific antibodies	IgG, IgM, IgA, antigens
ISAGA	anti-human IgM antibodies	IgM
LAT	Soluble antigen coated latex particles	IgG, IgM
PIA	Antigen coated gold nanoparticles	IgG
WB	Tachyzoite lysate antigen, recombinant antigens	IgG, IgM
ICT	Antigens or antibodies labeled with colloidal gold	IgG, ESA
Avidity Test	tachyzoite lysate antigen, recombinant antigens	IgG, IgA, IgE

Source: (Liu *et al.,* 2015).

ISAGA, and IHA. These techniques are compiled in (Table 1). The animal species used determines the sensitivity and specificity of the procedures, and the lack of reference sera from experimentally infected animals makes it challenging to determine cutoff levels. The test that appears to be most suited to a wide range of species right now is the MAT. Nonetheless, particular ELISA have been created for a few household animal species. Although these serological assays were initially designed for serum analysis, they have been modified to assess the risk of Toxoplasma in meat by analyzing meat juice (Dubey *et a*l., 2016).

In the absence of sera, the sole method for detecting Toxoplasma antibodies is by the analysis of meat juice, albeit less sensitively (retail outlets). However, because of the uneven distribution of tissue cysts and the small quantity of the tissue sample used (about 50 mg for PCR tests, against 50 to 500 g for bioassays), these techniques are actually less sensitive than bioassays. A technique based on sequence-specific magnetic capture of T. gondii DNA followed by DNA amplification has been developed in an effort to improve the sensitivity of PCR detection. With an anticipated detection limit of roughly one cyst per 100 g, it permitted the examination of homogenates of 100 g tissue samples (Opsteegh *et al*., 2010).

Although very few laboratories still use the dye test, which is based on parasite lysis by serum antibodies in the presence of complement, it has long been the gold standard in terms of sensitivity and specificity (Robert and Marie, 2012). Numerous techniques have been developed, including hemagglutination, enzyme-linked immunosorbent assays, capture ELISAs that enable the detection of particular isotypes of IgM, IgA, or IgE, and immunosorbent agglutination assays that are also appropriate for detecting IgM, IgA, or IgE. These days, the majority of clinical laboratories screen for particular IgG and IgM on a regular basis using an ELISA, while reference laboratories primarily employ other methods (Robert and Marie, 2012).

Modified agglutination test

Formalin-fixed T. gondii tachyzoites are added to U-shaped microtiter plates for the Modified Agglutination Test, followed by the addition of diluted test sera. Negative serum samples result in a compact pellet of precipitated tachyzoites at the bottom of the well, whereas positive serum samples will generate a thin mat of agglutination. Due to normal IgM binding to the parasite's surface, Fulton and Turk originally reported this test having low specificity and sensitivity. This was later enhanced by preparing the antigen in a buffer containing 2-mercaptoethanol, which eliminated non-specific IgM. IgG antibodies are detected by this test, regardless of the host species; however, false negative results may arise in the early phases of acute infection. In most species, the specificity and sensitivity of MAT are similar to those of DT; nevertheless, in dogs, it may result in a significant rate of false negative results (Zhu *et al*., 2012).

Depending on the preservative employed to produce the antigen, different MAT findings are obtained. When acetone is substituted for formalin in MAT, IgG antibodies in acute infection can be found, which is highly helpful in the diagnosis of acute glandular toxoplasmosis and toxoplasmosis in AIDS patients. Furthermore, MAT has a greater sensitivity than other serological tests when it comes to identifying cardiac secretions for the assessment of T. gondii infection in sheep that have been killed for human consumption (Villena *et al*., 2012). Because MAT is so straightforward and precise, it can be used for both epidemiological surveys and laboratory diagnosis.

Indirect fluorescent antibody test

A common method for identifying T. gondii antibodies in both humans and animals is the indirect fluorescent antibody test, which is a straightforward procedure that can identify both IgG and IgM antibodies (Sucilathangam

et al., 2010; Rodrigues *et al*., 2009). After adding the fluorescent antispecies antibodies and incubating the killed T. gondii tachyzoites with test serum, the outcome is examined under a fluorescence microscope. Test results indicate 80.4–100% sensitivities and 91.4–95.8% specificities (Santos *et al*., 2010; Shaapan *et al*., 2008). Commercially available fluorescently labeled antibodies covering a wide range of species, and the procedure is not too expensive. Individual variation may arise because the test requires a fluorescent microscope and the results are interpreted by eye. Certain species-specific conjugates might be hard to locate, and there's a chance that anti-nuclear and rheumatoid factor antibodies could react with one another (Liu *et al*., 2015).

Enzyme-linked immunosbsorbent assay

The solid phase antigen or antibody, enzyme-labeled antigen or antibody, and the substrate of the enzyme reaction which can be adjusted to test both antigens and antibodies are often included in an ELISA system. A huge number of samples can be evaluated at once using automated ELISA. To identify T.gondii antibodies or antigens, several ELISA techniques were developed. Indirect ELISA and sandwich ELISA are two examples, as shown in Figure 3 below. In the indirect ELISA, the sample containing antibodies is added after the antigen has been coated onto the solid phase. The addition of a secondary enzyme-linked antibody enhances the antigen antibody reaction, and the reaction can be measured by measuring the color that appears (Wang *et al*., 2014).

Depending on the kind of enzyme-linked antibody, nearly all of the tests are performed to find anti-T. gondii IgG, IgM, and IgA antibodies rather than antigens. In people and animals, the traditional indirect ELISAs that use the coated antigen of tachyzoite lysate have a good degree of agreement with DT, MAT, or IFAT for detecting IgG or IgM antibodies. Even with good results, TLA-based ELISA might differ greatly between labs or between batches, making it challenging to standardize and evaluating the test findings. Recombinant proteins have the advantages of a precise antigen and simple standardization, making them a viable option. Over the course of the last two decades, a multitude of recombinant antigens have been expressed in Escherichia coli or yeast, such as the granule antigens GRA1, GRA2, GRA4, GRA6, GRA7, and GRA8, the rhoptry proteins ROP1 and ROP2, matrix protein MAG1, microneme proteins MIC2, MIC3, MIC4, and MIC5, and the surface antigens SAG1 and SAG2. Their potential diagnostic utility was assessed in humans or animals through ELISA, in order to identify particular IgG and IgM antibodies (Kotresha, 2010; Lau *et al*., 2012).

It has been demonstrated that employing combinations of recombinant antigens is more sensitive and specific than using a single antigen. Combinations of SAG2A, GRA2, GRA4, ROP2, GRA8, and GRA7, for instance, may be helpful in identifying IgG antibodies in people who have just contracted an illness. According to Aubert *et al*. (2000), ROP1, SAG1, GRA7, GRA8, and GRA6 show promise in identifying certain IgM antibodies, whereas GRA7 and GRA8 are employed to identify particular IgA antibodies (Kotresha, 2010). discovered a sporozoite-specific embryogenesis-related protein that can be used to distinguish between tissue cyst-induced infection and oocyst-induced infection by reacting with oocyst-specific antibodies.

The sample containing the antibodies or antigens is added to the solid phase of the sandwich ELISA after the antigens or antibodies have been coated on it. The antibody antigen complex is adhered to the solid phase following washing and incubation. By adding particular antigens or antibodies that have been enzame-labeled, the trapped antibodies or antigens can be found. The sandwich ELISSA was created to find antigens and antibodies against T. gondii. It is feasible to identify circulating antigen and T. gondii-specific IgM, IgG, and IgA antibodies using an enhanced ELISA format (Gae et al., 2016). The specific antigen coated onto the solid phase, the enzyme-conjugated secondary antibody, and the substrates are the main components of the indirect ELISA technology, which is nearly exclusively employed for the detection of T. gondii antibodies rather than antigens. The particular antibody coated onto the solid phase, the enzyme-conjugated antibody, and the substrate are the components of the sandwich ELISA technique used to detect T. gondii antigens (Liu *et al*., 2015).

Molecular Methods

Molecular techniques are attractive for the detection of T. gondii infection because of their high sensitivity and specificity (Switaj *et al*., 2005). Furthermore, PCR-based genetic techniques have been used to identify numerous T. gondii genotypes from a variety of animals and birds (Dubey *et al*., 2013).

Conventional PCR

PCR can be employed in addition to serology to diagnose T. gondii infection because of the inherent limitations of standard diagnostic techniques. With the use of PCR, an effective in vitro enzymatic amplification technique, targeted DNA amplification from minuscule amounts of starting material can be completed quickly. When detecting T. gondii in biological samples, many multicopy targeting genes are typically employed to attain high sensitivity. These genes include the B1 gene, the 529 bp repeat element, and the internal transcribed spacer (ITS-1) or 18S rDNA sequences (Table 2). It is rare to find a

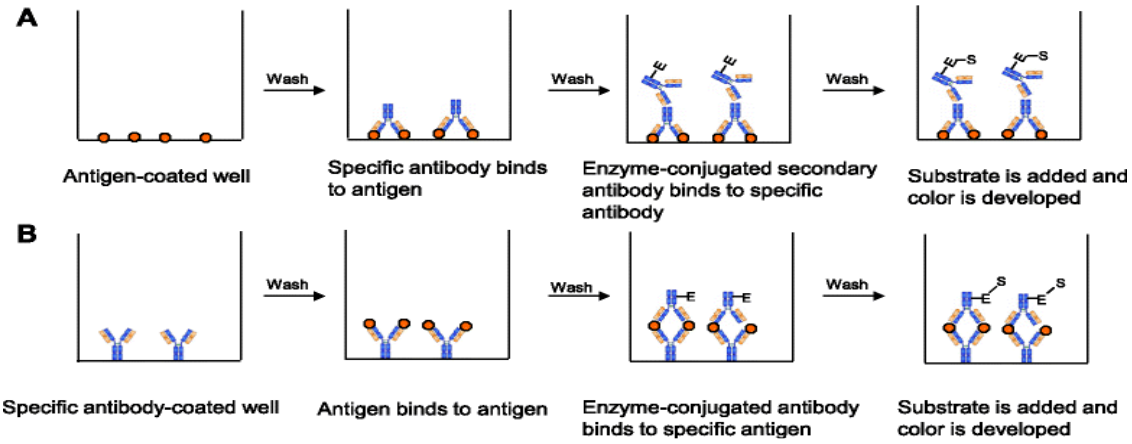

Figure 3. Schematic diagram of ELISA.
Source: (Liu *et al.,* 2015).

Table 2. Summary of the molecular approaches used for detection of *T. gondii*

Molecular methods	Main purposes	DNA target regions	Ref
Conventional PCR	Species detection	B1 gene, 529 bp repeat element, 18S rDNA gene, SAG1, SAG2, and GRA1	Cao *et al*., 2014
Real-time PCR	Species detection	B1 gene, 529 bp repeat element, 18S rDNA gene, SAG1	Huan *et al.*,2013
LAMP	Species detection	529-bp repetitive element, B1, SAG1, SAG2, GRA1, oocyst wall protein genes	Wang *et al.,*2014
Microsatellite analysis	Genotyping	TUB2, W35, TgM-A, B18, B17; M33, IV.1, XI.1, M48, M102, N60, N82, AA, N61, and N83	
Multilocus sequence typing	Genotyping	BTUB, SAG2,, GRA6, and SAG3	Liu *et al.,* 2015
PCR-RFLP	Genotyping	SAG1, SAG2, SAG3, BTUB, GRA6, c22-8, c29-2, L358, PK1 and Apico	Cao *et al*.,2014
RAPD-PCR	Genotyping	Genomic DNA	
High-resolution melting(HRM) analysis	Genotyping	B1 gene	Costa *et al*.,2011

Source: (Liu *et al.,* 2015)

parasitaemia present. Because of this, the negative predictive value of blood PCR is minimal. Some labs have also employed a number of other single-copy genes, including SAG1, SAG2, and GRA1, as PCR targets (Cao *et al*., 2014). In 1989, the first PCR technique for detecting T. gondii was developed, focusing on the B1 gene. In patients with impaired immune systems, this technique is frequently utilized for the prenatal diagnosis of congenital toxoplasmosis and T. gondii infection. It has been claimed that PCR using the 529 bp repeat region is 10 to 100 times more sensitive than the B1 gene. A few studies have also used the multicopy ITS-1 and 18S rDNA as their targets, demonstrating a similar sensitivity of the B1 gene (Calderaro *et al*., 2006).

Real-time PCR

Low amounts of target DNA can be found and the initial copies of a certain template DNA can be measured using real-time PCR. Using probes or intercalating dyes, the amplification product is monitored at each cycle and can be quantified using a standard with a known concentration. Human blood, cerebral fluid, aqueous humor, amniotic fluid, and other samples have all been effectively treated with real-time PCR to detect T. gondii DNA (Nogui *et al*., 2009). Because real-time PCR can measure the level of T. gondii infection, it is also used to assess the course of toxoplasmosis and the effectiveness of treatment (Menotti *et al*., 2003).

When compared to nested-PCR and conventional PCR, the real-time PCR assay utilizing the B1 gene is thought to be the most effective method for diagnosing congenital toxoplasmosis (Teixeira *et al*., 2013). Realtime PCR, being a quick closed-tube method, removes the potential risk of contamination and yields quantitative findings that are repeatable. It can therefore be standardized (Bretagne *et al*., 2006). As reviewed by Opsteegh *et al*. (2016), who also described a sequence-

specific magnetic capture method that can overcome the limited sample size and heterogeneous distribution of T. gondii tissue cysts in order to isolate T. gondii DNA from large tissue samples. These methods, when coupled with real-time PCR, can be used to meat samples and offer a substitute for bioassays in assessing the amount of T. gondii present in different animal tissues that are contaminated with food (Jurankova *et al*., 2014).

Diagnosis of Toxoplasmosis in Animals

Because there are so many different clinical symptoms that might appear, it is typically impossible to diagnose toxoplasmosis solely based on clinical criteria. Immunosuppression brought on by medication or other treatments (such as canine distemper) may facilitate the fast growth and spread of toxoplasma organisms, leading to the development of clinical illness (Tenter, 2009).

Fecal Examination

Cats can contract the intestinal coccidian Toxoplasma gondii. Typically, a typical fecal flotation is used to diagnose its oocysts. Oocysts are unsporulated, fresh excrement, and the infectious stages are crescent-shaped cells that have rounded posterior ends and pointed apical ends, measuring around 5 micrometers in length and 2 micrometers in width. They are surrounded by a complex membrane known as a pellicle, which promotes motility and structural integrity (Robert-Gangneux and Dardé, 2012).

Diagnosis of Toxoplasmosis in Human Being

Physical Examination

The most typical physical examination finding of immunocompetent patients with toxoplasmosis is painless lymphadenopathy. Fever, malaise, myalgia, and an amaculopapular skin rash that does not affect the palms or soles are among the other findings. Examination of the posterior pole in cases of retinochoroiditis reveals several yellow-white cotton-like patches with hazy borders that are grouped in small areas. Fever and tachypnea are considered vital symptoms (Urquhart *et al*., 1996).

Serological Tests

Various serological assays frequently assess distinct antibodies with distinct rising and falling trends over time following infection. To determine whether a person has recently contracted the virus or if they were more likely to have been infected in the distant past, a battery of serological testing is usually needed. A patient's history of infection can be ascertained with an IgM test, which helps establish how long ago the infection occurred. Confirmatory testing ought to be done because there is a considerable chance that a positive IgM test result could be misinterpreted. Commercial test kits for measuring IgM antibodies are widely available, however their specificity is typically inadequate, and the reported results are commonly misconstrued. Furthermore, IgM antibodies might last anywhere from several months to a year or longer (Radostitis, 1994).

The Skin Test

Toxoplasma collected from mouse inoculation is immediately frozen and thawed to create the antigen. For a dye test to yield a positive result there must be at least 7 mm of in duration or erythema. It is the standard reference test for toxoplasmosis and measures IgG; however, most laboratories do not have live T. gondii, hence it is not available (Dubey *et al*., 1995).

Differential Diagnosis

When making a differential diagnosis for several CNS illnesses, toxoplasmosis should be taken into account, especially in cases where dogs have received the recommended dose of the distemper vaccine. Other than in relation to issues with abortion and related infant mortality, toxoplasmosis is rarely taken into account in a primary diagnostic list. Abortion in sheep, cattle, and pigs is differentially diagnosed with leptospirosis and brucellosis, respectively. Animal encephalitis is caused by bacteria (Listeria monocytogenes), viruses (like rabies), and verminous encephalomyelitis, which is linked to the somatic migration of parasitic species' larvae (Micronema deletrix, Paraelaphostrongylus tenuis). The following samples are used to confirm the diagnosis: a) Parasitology: fresh or cooled lung, brain, placenta, and feces b) Fetal thoracic fluid serology c) Histology: lung, liver, brain, spinal cord, kidney, heart, and placental cotyledons (Torda, 2001).

Morbidity and Mortality

The consequences of infection are more severe in immunocompromised individuals and pregnant women. Congenital toxoplasmosis rates vary, with estimates ranging from one case per 3,000 births to one case per 10,000 births. Most pregnant women only transmit T. gondii to the developing fetus if they are initially exposed to the virus. Nonetheless, some women with immunological suppression appear to have passed on

reactivated organisms from past infections (Alvarado-Esquivel *et al*., 2013).

A congenitally infected fetus may have been born to a seropositive, immunocompetent mother in rare instances; this may have happened as a result of a recrudescent infection or after she contracted the infection from another strain of the organism. During the first trimester, there is an estimated 25% chance of transfer from an infected mother; most of these fetuses will have severe clinical symptoms. Right now, there is a significant rate of fetal mortality. On the other hand, 70–90% of newborns are asymptomatic and 50–65% of infants are thought to contract the infection during the third trimester; nonetheless, some will subsequently exhibit clinical indications if treatment is not received (Alvarado-Esquivel *et al*., 2013).

Treatment of Toxoplasmosis

There are several medications that can be used to treat toxoplasmosis. Pregnancy and the postpartum period should be properly examined for both the mother and the unborn child if one is infected. Individuals with weakened immune systems, such as those suffering from AIDS, may require medicine for the duration of their life or as long as they remain immunosuppressed (Torda, 2001).

There is currently no specific, effective treatment for toxoplasmosis in exotic ruminants. When treating series ocular inflammation, anti-inflammatory medications may be helpful in addition to antibiotic therapy. Pregnant ewes afflicted with toxoplasmosis produced by experimentation have shown improvement when treated with a pyrimethamine and sulphamethazine combination. Three days of treatment are given over three intervals, separated by five days. Chemotherapy that uses pyrimethamine (0.5–1 mg/kg/day) as a single dose and sulphadiazine (60 mg/kg/day) every 4-6 hours stops the spread of infection until host immunity is developed. These two medications work well together as folate metabolism inhibitors. To avoid their harmful side effects, folinic acid (1 mg/kg/day) may be given (Torda, 2001).

Traditional human treatment for clinical toxoplasmosis involves pyrimethamine and sulphonamides; however, pregnant women should not take this medication due to possible fetal side effects (due to inhibition of folate synthesis). Nowadays, one of the recommended medications for pregnant patients with toxoplasmosis is spiramycin, a macrolide. Patients who test positive for AIDS can avoid reactivation of their disease by receiving treatment with fansider, dapsone-pyrimetamine, or trimethoprim-sulfamethoxazole. Antibiotics might not be able to eliminate parasites that are actively dividing as well as tissue cysts. Treatment with spiramycin (Rovamycine) can be started if a pregnant woman is found to have acute T. gondii infection in an attempt to stop the infection from spreading to the fetus (Tenter, 2009).

Prevention and Control strategies

Regarding the management of toxoplasmosis in farm animals, there are two issues. Reducing the economic impact of infection in agricultural animals is the first goal, and lowering the danger of human illness from eating contaminated meat, particularly from domestic animals, is the second (Kar and Misra, 2004). Although it is more difficult to control toxoplasmosis on farms, animal feed should be covered whenever possible to keep cats and insects out. Toxoplasmosis-related abortions have been attempted to be controlled by giving ewes in mid-pregnancy decoquinate and monensin.

Thankfully, a live vaccine for sheep that contains tachyzoites weakened by repeated passage in mice is now accessible. The vaccination is administered intramuscularly as a single dose at least three weeks before lambing, and it contains 10000-1000000 tachyzoites (Urquhart *et al*., 1996). Cooking in the microwave, adding salt, or smoking do not always eliminate all pathogenic Toxoplasma organisms. Most toxoplasma tissue cysts can be killed by freezing meat at -12°C for at least 24 hours; however, sporulated oocysts can endure up to 28 days at -20°C. All bradyzoites and tachyzoites can be eliminated by washing kitchenware, dishes, and surfaces that have come into contact with raw meat in hot, soapy water. Sand boxes and other places where kids play should be off-limits to cats since they might urinate there (Tenter, 2000).

In general, the following actions are crucial for preventing toxoplasmosis: frying all meat for ten minutes at a minimum temperature of 67°C, or 153°F; removing the seeds or thoroughly cleaning fruits and vegetables before eating them; washing any items and surfaces that come into contact with raw meat or unwashed produce; avoiding coming into contact with garden soil and cat litter; if not, using gloves and carefully cleaning your hands after gardening; Don't give your cats raw meat; keeping cats inside to prevent infection from small prey being eaten by predators (Prusa *et al*., 2013).

One health approaches for the control of Toxoplasmosis

One Health is an international strategy that acknowledges the interdependence of human, animal, plant, and environmental health from local to global levels. It takes a holistic approach that promotes and expands transdisciplinary collaborations, integrative research, clinical practice, capacity building, policy, and communication among numerous stakeholders (Aguirre *et al*., 2019). Because toxoplasmosis poses a threat to

people who depend on animal resources and has a substantial negative impact on the health of domestic animals, wildlife, humans, and ecosystems, it is considered a One Health illness (Jenkins *et al*., 2015). The consequences of toxoplasmosis are becoming more well-documented, which highlights the need for comprehensive, transdisciplinary measures to restrict transmission through the use of the One Health concept, as well as increased institutional understanding of the infection routes. To date, this kind of collaboration has proven difficult to achieve, maybe due in part to ignorance of the biology of T. gondii or its grave health consequences (NGO *et al*., 2017).

In order to develop a comprehensive strategy and the capacity building necessary for effective research efforts, transdisciplinarity refers to a research methodology that crosses numerous disciplinary boundaries. An approach to One Health is necessary in the ecology of infectious diseases. Since T. gondi is known to be impacted by environmental factors, efforts to reduce exposure may have an impact on the health of the ecosystem. It is unclear how long tissue cysts from an infected animal that dies in the field would survive or remain infectious, or how resistant the parasite will be in its oocyst form. Effective, truly transdisciplinary collaborations including scientists from a wide range of disciplines, including but not limited to earth, environmental, biological, ecological, social, and health sciences, will be necessary to close these scientific and knowledge gaps (Aguirre et al., 2019).

Awareness Creation

Increased awareness of the dangers and routes of T. gondii exposure is essential for the management of toxoplasmosis. Health professionals working with the public, including those in human and veterinary medicine should endeavor to communicate current understandings of the life cycle, routes of transmission, and optimal precautions to avoid exposure more effectively. For instance, the risks of exposure to oocysts should not be minimized, even while the risks of infection from consuming tissue cysts are acknowledged. Due to concerns about food biosecurity, outdoor cats should not be allowed near community gardens and should not be allowed anywhere that food is grown. In addition to limiting domestic cats' access to the outdoors and taking steps to lower the number of free-roaming domestic cats and the corresponding quantity of T. gondii oocysts, children should avoid areas where cat excrement may be present. To reduce the effects of toxoplasmosis on society and the environment, widespread involvement is essential, particularly among medical professionals who treat humans and animals (Loss et al., 2015).

Chemotherapy

There are few available chemotherapy treatments for toxoplasmosis at this time (Dunay *et al*., 2018). Notwithstanding a concentrated effort over the past ten years to use high throughput screening techniques to find novel compounds, including natural items, that have anti-Toxoplasma action or to modify or enhance already-approved medications. The first line of treatment for toxoplasmosis consists of pyrimethamine plus sulfa-diazine, which work together to target two different stages of the metabolism of folic acid. Additional treatments involve the combination of trimethoprim and sulfame-thoxazole, azithromycin or atovaquone, or pyrimethamine and clindamycin. On the other hand, toxicity and unfavorable side effects are frequently linked to all treatments (Deng *et al*., 2019).

Vaccination for the control of toxoplasmosis

Globally, vaccination has been widely utilized to combat infectious diseases. The difficulty in developing a vaccine to control toxoplasmosis stems from T. gondii's complex life cycle and its ability to infect any warm-blooded animal. This includes deciding which host(s) to target based on the parasite's life cycle stage, which parasite antigens to target, and the best delivery method. Whether a single vaccine might meet each of these needs is up for debate. In order to minimize the spread of the parasite to humans through consumption of undercooked meat, a vaccine targeting T. gondii in livestock would not only need to prevent the formation of bradyzoite cysts (Tenter *et al*., 2000); it would also need to lessen the financial loss incurred by animal abortion, particularly in sheep and goats (Stelzer *et al*., 2019), in order to promote widespread use.

A vaccination directed against felids, the sole known host of T. gondii, would be able to prevent the release of highly contagious oocysts into the environment and, as a result, lower the risk of infection through ingestion in humans and cattle.although one possible barrier to success may be the way the vaccine is delivered in order to attain adequate coverage. On the other hand, it has shown to be very successful in immunizing cats against oocyst shedding when live, attenuated strains of T. gondii are used for the infection. The first documented live attenuated vaccine studied was the T-263 strain of T. gondii, a chemically-induced mutant that matures into micro- and macrogametes but is incapable of producing oocysts. Two doses of T-263 tissue cysts or bradyzoites digested by pepsin administered orally elicited antibody responses and inhibited oocyst shedding in 84% of cases (Dubey, 2016).

Zoonotic Importance

While seroprevalence studies show relatively high rates of infection in farm animals, the infection is subclinical, and Toxoplasma gondii has virtually no importance as a cause of clinical disease in farm animals with the exception of that associated with abortion and neonatal disease in sheep (KaŠková *et al*., 2007). Toxoplasmosis is important in farm animals because of its potential to spread zoonotic diseases. T. gondii uses humans as its intermediate host, and about 50% of Americans are infected with the virus. Man is either born infected or acquired. There are two ways that infections can be acquired: either directly by hand contamination from cat feces carrying oocysts, or indirectly through eating contaminated food and undercooked meat that contains toxoplasma cysts. The majority of illnesses most likely result from eating oocysts from cat feces that contaminate food or accidentally enter the mouth due to inadequate hygiene habits. On the other hand, handling or eating meat that contains bradyzoites and tachyzoites can also infect humans. Meat from pigs, sheep, and horses carries a higher risk of infection than beef (Smith, 1991). Goats challenged with toxoplasmosis secrete chyzoites in their milk, albeit there is very little risk (Dubey, 1990).

When humans are infected with T. gondii, the illness is typically mild and self-limiting or not present at all. Acute immune deficiency syndrome (AIDS), cancer, anyone receiving cytotoxic or immunosuppressive medication, and the very young and very old can all experience significant disease. Additionally, there is a chance of miscarriage or congenital infections such retino-choroiditis, hydrocephalus, and cerebral calcification. Those who handle infected material at slaughterhouses, farms, and veterinary clinics run the danger of contracting toxoplasmosis at work. Veterinarians and agricultural workers, particularly those who are pregnant, should take steps to prevent infection when handling infected material because there is a higher risk of infection when they come into contact with lambing ewes in infected flocks (Smith, 1991).

The Disease Situation in Ethiopia

The seroprevalence of toxoplasma varies, with warmer, moister climates showing a higher prevalence than colder, drier climates. In addition, the age of the animals and the methods used in their husbandry may have an impact on variation. In the East Hararghe Zone, 302 (22.2%) of the 1360 domestic ruminant sera examined showed the presence of anti-T. gondii IgG antibodies. In the research area, cattle had the lowest seroprevalence of infection at 10.7% and sheep had the highest at 33.7% (Tilahun *et al*., 2018). ELISA has been used to detect the prevalence of IgG antibodies to T. gondii in persons in Ethiopia. Children showed the highest antibody titers, and 75% of young adults were sero-converted. Since toxoplasmosis often reemerges after infection with the human immune deficiency virus type 1 (HIV-1), persons infected with HIV had greater T. gondii antibody titers than HIV-negative people (Negash *et al*., 2004).

CONCLUSION AND RECOMMENDATIONS

A common food-borne zoonose that is prevalent throughout the world, toxoplasmosis is caused by T. gondii. This condition is a dangerous parasite illness that can be fatal and is a result of Sub-Saharan Africa's extreme poverty. The disease may become more contagious if one lives with domestic cats and is exposed to their droppings. The most common food animals to have T. gondii cysts in their meat are sheep and goats, who also pose a significant risk to human health. Because the oocytes thrive better in hot, humid conditions and lower altitudes, infection is frequently higher in these regions of the world. Since most T. gondii infections are asymptomatic and unreliable for diagnosis, outbreaks are rarely seen and recorded. Additionally, there are currently few effective chemotherapy treatments for toxoplasmosis. Moreover, the development of a vaccine to prevent toxoplasmosis is difficult because of the intricacy of T. gondii's life cycle and its capacity to infect any warm-blooded mammal. However, toxoplasmosis control is crucial for reasons related to health, the economy, and the environment.

Therefore,

- It is imperative to raise public education and knowledge on the consequences of toxoplasmosis in order to lower the disease's risk. It's crucial for immunocompromised individuals and pregnant women to understand the condition.
- Community involvement is crucial for illness control and prevention in addition to diagnosis.
- Avoid consuming raw meat and untreated water. When gardening or coming into contact with soil, wear personal protective equipment.
- Serological and molecular based study is needed in order to know the current status of the toxoplasmosis in Ethiopia
- The greatest strategies for preventing exposure are creating a livestock vaccination, raising awareness, and working together on One Health.

REFERENCES

Aguirre A, Longcore T, Barbieri M, Dabritz H, Hill D, Klein N, Lepczyk C, Lilly L, McLeod R, Milcarsky J, Murphy E (2019). The one health approach to toxoplasmosis: epidemiology, control, and prevention strategies. Eco Health, 16(2), 378-390.

Ahmad S (2018). Water related ocular diseases. Saudi J of Ophthalmology, 32(3), 227-233.

Al-Malki, E. (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(1), 962.

Alvarado-Esquivel C, Gayosso-Dominguez EA, Villena I, Dubey JP (2013). Seroprevalence of Toxoplasma gondii infection in captive mammals in three zoos in Mexico City, Mexico. J. Zoo and Wildlife Med. 803-806.

Aubert D, Maine G, Villena I, Hunt J, Howard L, Sheu M (2000). Recombinant antigens to detect Toxoplasma gondii-specific immunoglobulin G and immunoglobulin M in human sera by enzyme immunoassay. J ClinMicrobiol.38 :1144–50.

Bayarri S, Gracia MJ, L´azaro R, P´erez-Arquillu´e C, Herrera A (2012).Toxoplasma Gondii in Meat and Food Safety Implications–A Review, J. Zoonosis Lorenzo-Morales, Ed.In Tech Open, London, UK, 229–254.

Blood DC, Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007). Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats, Saunders Ltd, London, UK.

Boothroyd J, Grigg M (2002). Population biology of Toxoplasma gondii and its relevance to human infection: do different strains cause different disease?.Current opinion in microbiology,5(4), pp.438-442.

Bowie WR, King AS, Werker DH, Isaac-renton JL, Bell A, Eng SBV, Marion SA (1997). Outbreak of toxoplasmosis associated with municipal drinking water. The BC Toxoplasma investigation team.Lancet., 350, 173-177.

Bretagne, S., Costa, J. (2006). Towards a nucleic acid-based diagnosis in clinical parasitology and mycology.ClChimica Acta,363(1-2), pp.221-228.

Calderaro, A., Piccolo, G., Gorrini, C., Peruzzi, S., Zerbini, L., Bommezzadri, S., Dettori, G. Chezzi, C.(2006). Comparison between two real-time PCR assays and a nested-PCR for the detection of Toxoplasma gondii.Acta bio-medica: Atenei Parmensis,77(2), pp.75-80.

Cao, L., Cheng, R., Yao, L., Yuan, S., Yao, X. (2013).Establishment and application of a loop-mediated isothermal amplification method for simple, specific, sensitive, and rapid detection of toxoplasma gondii.J of Vet Medical Sci, pp.13-0275.

Cohen T, Blois, S., Vince, A. (2016). Fatal extraintestinal toxoplasmosis in a young male cat with enlarged mesenteric lymph nodes.The Canadian VeterJ,57(5), 483.

Conrad P, Melo CC, Costa VM. (2005). Transmission of toxoplasma: Clues from the study of sea otters as sentinels of Toxoplasma gondii flow into the marine environment. Int. J. Parasitol., 35,1155-1168.

Cook A.J.C., Gilbert R.E., Buffolano W., Zufferey J., Petersen E., Jenum P.A., Foulon W., Semprini A.E. and Dunn D.T. (2000). Sources of Toxoplasma infection in pregnant women: European multicentre case control study. Br. Med. J., 321: 142–147.

Costa J, Cabaret O, Moukoury S, Bretagne S (2011). Genotyping of the protozoan pathogen Toxoplasma gondii using high-resolution melting analysis of the repeated B1 gene. J. Microbio. methods, 86 (3), pp.357-363.

da Silva P, Shiraishi C, da Silva A, Gonçalves G, Sant'Ana D, de Almeida Araújo E (2010). Toxoplasma gondii: a morphometric analysis of the wall and epithelial cells of pigs intestine.Exp'tparasitol, 125 (4), pp.380-383.

De Tommasi A, Morini M, Turba M, Otranto D, Bettini G (2014).Hyperplastic cholangitis in a naturally Toxoplasma gondii-infected cat.Veter Quarterly, 34(4), 229-231.

Deng Y, Wu T, Zhai S, Li C (2019). Recent progress on anti-Toxoplasma drugs discovery: Design, synthesis and screening. European jl of medichem, 183, 111711.

dos Santos, T., Nunes, C., Luvizotto, M., de Moura, A., Lopes, W., da Costa, A., Bresciani, K.(2010). Detection of Toxoplasma gondiioocysts in environmental samples from public schools. Veterinary parasitology, 171(1-2), pp.53-57.

Dubey J (2009). History of the discovery of the life cycle of Toxoplasma gondii. International j for parasito, 39(8), 877-882.

Dubey J (2016). Transmission of Toxoplasma gondiiFrom land to sea, a personal perspective.A Century of Parasitology, 148-164.

Dubey J.P. (2010). Toxoplasmosis of animals and humans. 2nd edition. Boca Raton Florida,

Dubey JP (2001). Oocyst shedding by cats fed isolated bradyzoites and comparison of infectivity of bradyzoites of the VEG strain Toxoplasma gondii to cats and mice. J. Parasitol., 87: 215–219.

Dubey JP (2010). Toxoplasmosis of Animals and Humans, CRC Press, Boca Raton, FL, USA, 2nd edition.

Dubey JP, Jones JL (2008). Toxoplasma gondii infection in humans and animals in the United States. Int. J. Parasitol. 38, 1257-1278.

Dubey, J. (2021). Outbreaks of clinical toxoplasmosis in humans: five decades of personal experience, perspectives and lessons learned.Parasites& Vectors, 14(1), 1-12.

Dubey, J., Darrington, C., Tiao, N., Ferreira, L., Choudhary, S., Molla, B., Saville, W., Tilahun, G., Kwok, O., Gebreyes, W. (2013).Isolation of viable Toxoplasma gondii from tissues and feces of cats from Addis Ababa, Ethiopia.The J l of parasit, 99(1), pp.56-58.

Dubey, J., Darrington, C., Tiao, N., Ferreira, L., Choudhary, S., Molla, B., Saville, W., Tilahun, G., Kwok, O., Gebreyes, W. (2013).Isolation of viable Toxoplasma gondii from tissues and feces of cats from Addis Ababa, Ethiopia.TheJl of parasit, 99(1), pp.56-58.

Dubey, J.P. (1990). Status of toxoplasmosis in cattle inUnited States.J. Am. Vet. Med. Assoc., 196, 257.

Dubey, J.P. (1998). Toxoplasmosis of animals and man. Vet. Med. Assoc. Baca Raton Florida, 205, 1593-1598.

Dubey, J.P., Lappin M.R. and ThulliezP. (1995). Diagnosis of induced toxoplasmosis in neonatalcats. Journal of the American Veterinary Medical Association, 207(2), 179-185.

Dunay, I., Gajurel, K., Dhakal, R., Liesenfeld, O. and Montoya, J.G. (2018). Treatment of toxoplasmosis: historical perspective, animal models, and current clinical practice. Clinical microbiology reviews, 31(4), 57-17.

Edwards J. F. and Dubey J. P. (2013). Toxoplasma gondii abortion storm in sheep on a Texas farm and isolation of mouse virulent atypical genotype T. gondiifrom an aborted lamb from a chronically infected Ewe, Veterinary Parasitology, 192(1–3), 129–136.

Elmore SA, Jones JL, Conrad PA, Patton S, Lindsay DS, Dubey JP (2010). Toxoplasma gondii: epidemiology, feline clinical aspects, and prevention, Trends in Parasitology, 26(4), 190–196.

Ferreira, S., Torelli, F., Klein, S., Fyumagwa, R., Karesh, W., Hofer, H., Seeber, F., East, M. (2019). Evidence of high exposure to Toxoplasma gondii in free-ranging and captive African carnivores Inter J for Parasitology: Parasites and Wildlife,8, 111-117.

Gamble HR, Dubey JP, Lambillotte DN (2005). Comparison of a commercial ELISA with the modified agglutination test for detection of Toxoplasma infection in the domestic pig,Veterinary Parasitology, 128(3-4),177–181.

Gao X, Wang H, Wang H, Qin H, Xiao J.(2016). Land use and soil contamination with Toxoplasma gondiioocysts in urban areas. Sci. of the Tot.En't. 568 : 1086–1091.

Gebremedhin EZ, Anteneh HA, Tesfaye ST, Kassu DT, Girmay M, Maria V, Vincenzo Di M. Eric C, Pierre D (2013). Seroepidemiology of Toxoplasma gondii infection in women of

childbearing age in central Ethiopia. BMC Infectious Diseases, 13: 101.

Gelaye W, Kebede T, HailuA. (2015).High prevalence of anti-toxoplasma antibodies and absence of Toxoplasma gondii infection risk factors among pregnant women attending routine antenatal care in two Hospitals of Addis Ababa, Ethiopia. Inter J of Infectious Diseases, 34, 41-45.

Gerhold R, Newman S, Grunenwald C, Crews A, Hodshon A, Su C (2014). Acute onset of encephalomyelitis with atypical lesions associated with dual infection of Sarcocystisneurona and Toxoplasma gondii in a dog.Vetparasitol, 205(3-4), 697-701.

Hide G (2016). Role of vertical transmission of Toxoplasma gondii in prevalence of infection. Expert review of anti-infective therapy, 14(3), 335-344.

Hill D, Dubey JP (2002). Toxoplasma gondii: transmission, diagnosis and prevention, Clinical Microbiology and Infection, 8(10), 634–640.

Hussain M, Stitt V, Szabo EA, Nelan B (2017). Toxoplasma gondii in the Food Supply.Pathogens, 6(2), 21.

Jenkins E, Simon A, Bachand N, Stephen C (2015). Wildlife parasites in a One Health world. Trends in Parasitology, 31(5), 174-180.

Jilo K, Tegegne D, Kasim S, Dabasa G, Zewdei W (2021). Seroprevalence and Public Health Significance of Toxoplasmosis in Small Ruminants of Pastoral Community in Yabello District, Borana Zone, Southern Ethiopia. Vet Medicine International.

Jones JL, Dubey JP (2012). Foodborne toxoplasmosis. Clinical Infectious Diseases, 55(6): 845-851.

Jurankova J, Basso W, Neumayerová H, Baláž V, Jánová E, Sidler X, Deplazes P, Koudela B (2014). Brain is the predilection site of Toxoplasma gondii in experimentally inoculated pigs as revealed by magnetic capture and real-time PCR. Food microbiology, 38, pp.167-170.

Kaòková Š, Šulc J, Nouzová K, Fajfrlík K, Frynta D, Flegr J (2007). Women infected with parasite Toxoplasma have more sons. Naturwissenschaften, 94(2): 122-127.

Kar N, Misra B (2004). Toxoplasma seropositivity, and depression: a case report. BMC Psychiatry 4(1): 1.

Kenneth J. and George R. (2004).Toxoplasma Gondii, An Introduction to Infectious Diseases, McGraw-Hill Companies, New York, NY, USA, 4th edition.

Khalil M, Ahmed A, Elrayah I (2013). Prevalence and Risk factors for Toxoplasma gondii infection in Humans from Khartoum State, Sudan.Int j of public health and epidemiology, 2(3), 60-66.

Kijlstra A.and Jongert E. (2009). Toxoplasma-safe meat: close to reality.Trends in arasitology, 25(1), 18–22, 2009.

Klaus SN (2003). Leishmaniasis and other protozoan infections.Fitzpatrick's Dermatology in GeneralMedicine.

Kniel KE, Lindsay DS, Summer SS, Hackney CR, Pierson MD, Dubey JP (2002). Examination of attachment and survival of Toxoplasma gondii oocysts on raspberries and blueberries. J. Parasitol., 88: 790–793.

Kotresha,D.,Noordin,R.(2010).Recombinant proteins in the diagnosis of toxoplasmosis. Apmis, 118(8), pp.529-542.

Lahmar I, Lachkhem A, Babba O, Slama D, Trabelsi A, Passebosc-Faure K, Dardé M, Babba H (2020).First isolation and molecular characterization of Toxoplasma gondii strains from human congenital toxoplasmosis cases in Monastir, Tunisia. Scientific reports, 10(1), 1-7.

Liu, Q., Wang, Z., Huang, S., Zhu, X.(2015). Diagnosis of toxoplasmosis and typing of Toxoplasma gondii. Parasites & vectors, 8(1), pp.1-14.

Loss, S., Will, T., Marra, P. (2015). Direct mortality of birds from anthropogenic causes. Annual Review of Ecology, Evolution, and Systematics, 46, 99-120.

Menotti, J., Vilela, G., Romand, S., Garin, Y., Ades, L., Gluckman, E., Derouin, F., Ribaud, P. (2003).Comparison of PCR-enzyme-linked immunosorbent assay and real-time PCR assay for diagnosis of an unusual case of cerebral toxoplasmosis in a stem cell transplant recipient. J of clinical microbiology, 41(11), pp.5313-5316.

Migliore, S., La Marca, S., Stabile, C., Presti, V., Vitale, M. (2017). A rare case of acute toxoplasmosis in a stray dog due to infection of T. gondii clonal type I: public health concern in urban settings with stray animals?. BMC veterinary research, 13(1), 1-4.

MirzaAlizadeh, A., Jazaeri, S., Shemshadi, B., Hashempour-Baltork, F., Sarlak, Z., Pilevar, Z., Hosseini, H. (2018). A review on inactivation methods of Toxoplasma gondii in foods.Pathogens and global health, 112(6), 306-319.

Mohamed, K. (2020). Toxoplasmosis in humans and animals in Saudi Arabia: A systematic review. The J of Infection in Developing Countries, 14(08), 800-811.

Montoya, J. and Liesenfeld O. (2004).Toxoplasmosis. Lancet., 363: 1965-1976.

Mose, J., Kagira, J., Kamau, D., Maina, N., Ngotho, M., Karanja, S. (2020). A review on the present advances on studies of toxoplasmosis in eastern Africa. Bio Med Research International.

Negash T, Tilahun G, Patton S, Prevot F, Dorchies PH (2004). Serological survey ontoxoplasmosis in sheep and goats in Nazareth, Ethiopia. Revue De MedecineVeterinaire, 155, 486-488.

NGO H, Zhou Y, Lorenzi H, Wang K, Kim T, Zhou Y, El Bissati K, Mui E, Fraczek L, Rajagopala, S.V., Roberts, C. (2017). Toxoplasma modulates signature pathways of human epilepsy, neuro degeneration and cancer. Scientific reports, 7(1),1-32.

Nogui F, Mattas S, TurcatoJúnior G, Lewi D (2009). Neurotoxoplasmosis diagnosis for HIV-1 patients by real-time PCR of cerebrospinal fluid. Brazilian J. Infectious Diseases, 13(1), pp.18-23.

Opsteegh, M., Schares, G., Blaga, R., van der Giessen, J. (2016). Experimental studies on Toxoplasma gondii in the main livestock species (GP/EFSA/BIOHAZ/2013/01) Final report. EFSA Supporting Publications, 13(2), p.995E.

Pepe P, Bosco A, Capuano, F\, Baldi, L., Giordano, A., Mancusi, A., Buonanno, M., Morena, L., Pinto, R., Sarnelli, P., Cringoli G (2021).Towards an Integrated Approach for Monitoring Toxoplasmosis in Southern Italy. Animals, 11(7), 1949

Prusa AR, Kasper DC.,OlischarM., HussleinP., PollakA.andHaydeM. (2013). Evaluation of serological prenatal screening to detect Toxoplasma gondii infections in Austria. Neonatology, 103(1): 27-34

Radostitis Blood, D.C. (1994). Veterinary Medicine: A text book of the disease of cattle, sheep, pigs,goats and horses, 8th edn, ELBS, BaillierTindasll, 68.

Rahman, T., Rahman, A., Chakraborty, S. (2018). Infection of Toxoplasma gondii in humans and livestock animals: an emerging silent threat for Bangladesh. Open J of Medical Microbiology, 8(04), 109-117.

Robert-Gangneux, F. and Dardé M.L. (2012). Epidemiology and diagnostic strategies forToxoplasmosis. Clin. Microbiol. Rev., 25(2): 264.

Rodrigues I, Castro A, Gomes M, Amaral W, Avelino M (2009). Congenital toxoplasmosis: evaluation of serological methods for the detection of anti-ToxoplasmagondiiIgM and IgA antibodies. Memorias do Instituto. Oswaldo Cruz, 104, pp.434-440.

Scallan E., Hoekstra R. M., Angulo F. J. et al. (2011). Foodborne illness acquired in the United States-major pathogens, Emerging Infectious Diseases, 17(1), 7–15.

Schlüter D, Däubener W, Schares G, Groß U, Pleyer U, Lüder C (2014). Animals are key to human toxoplasmosis. International Journal of Medical Microbiology, 304(7), 917-929.

Shaapan R., El-Nawawi F., Tawfik M.(2008). Sensitivity and specificity of various serological tests for the detection of Toxoplasma gondii infection in naturally infected sheep. Vet Parasitol.153 : 359–62.

Shimelis T. et al., (2009). Seroprevalence of latent Toxoplasma gondii infection among HIV-infected and HIV-uninfected people in Addis Ababa, Ethiopia: a comparative cross-sectional study. BMC Research Notes, 2: 213.

Smith, J., Ashander, L., Arruda, S., Cordeiro, C., Lie, S.,Rochet, E., Belfort Jr, R., Furtado, J. (2021). Pathogenesis of ocular toxoplasmosis. Progress in Retinal and Eye Research, 81, 100882.

Smith, J.L. (1991). Food born toxoplasmosis. J. Food Safety, 12, 17-58.

Stelzer, S., Basso, W., Silván, J., Ortega-Mora, L., Maksimov, P., Gethmann, J., Conraths, F., Schares, G. (2019).Toxoplasma gondii infection and toxoplasmosis in farm animals: Risk factors and economic impact. Food and Water borne Parasitology, 15, 37.

Stover J, Kenneth HE, Schwartzman JD, Kasper LH (1990).Toxoplasma gondiiin a collection of non-domestic ruminants. J. Zoo. Wild. Med., 21, 295.

Sucilathangam, G., Palaniappan, N., Sreekumar, C., Anna, T.(2010).IgG– Indirect fluorescent antibody technique to detect seroprevalence of Toxoplasma gondii in immunocompetent and immunodeficient patients in southern districts of Tamil Nadu. Ind. J. Med. Microbiol. 28(4), pp.354-357.

Susan (1998).The Merck Veterinary Manual. 8th ed, 488-489

Switaj K, Master A, Skrzypczak M, Zaborowski, P.(2005). Recent trends in molecular diagnostics for Toxoplasma gondii infections. Clinical Microbiology and Infection, 11(3), pp.170-176.

Tadesse, L., Tafesse, F., Hamamy, H. (2014).Communities and community genetics in Ethiopia. The Pan African Med.l J. 18.

Tarekegn Z, Dejene H, Addisu A, Dagnachew S (2020). Potential risk factors associated with seropositivity for Toxoplasma gondii among pregnant women and HIV infected individuals in Ethiopia: A systematic review and meta-analysis. PLoS neglected tropical diseases, 14(12), 8944.

Tegegne D, Abdurahaman M, Yohannes M (2016). Seroepi-demiology and associated risk factors of Toxoplasma gondii in sheep and goats in Southwestern Ethiopia. BMC veterinary research, 12(1), 1-6.

Teixeira L, Kanunfre K, Shimokawa P, Targa L, Rodrigues J, Domingues W, Yamamoto L, Okay T (2013). The performance of four molecular methods for the laboratory diag-nosis of congenital toxoplasmosis in amniotic fluid samples. Revista da Sociedade-Brasileira de Medicina Tropical, 46, pp.584-588.

Tenter AM, Heckeroth AR, Weiss LM, Louis M (2001). Erratum to "Toxoplasma gondii: from animals to humans. Int. J. Parasitol. 31(2), 217–220.

Tenter AM, Heckeroth AR, Weiss LM (2000). Toxoplasma gondii from animals to humans. Int. J. Parasitol., 30: 1217–1258.

Tenter AM (2009). Toxoplasma gondii in animals used for human consumption. Memórias do InstitutoOswaldo Cruz, 104 (2), 364-369.

Tenter A, Heckeroth A, Weiss L (2000). Toxoplasma gondii: from animals to humans. Int. J. Parasitol. 30(12-13), pp.1217-1258.

Teweldemedhin M, Gebremichael A, Geberkirstos G, Hadush H, Gebrewahid T, Asgedom S, Gidey B, Asres N, Gebreyesus H (2019). Seroprevalence and risk factors of Toxoplasma gondii among pregnant women in Adwa district, northern Ethiopia. BMC infectious diseases, 19(1),1-9.

Tilahun B, Hailu Y, Tilahun G, Ashenafi F, Shimelis S (2018). Seroprevalence and risk factors ofToxoplasmagondii infection among Domestic Ruminants in East Hararghe Zone of Oromia Region, Ethiopia. Vet. Med. Int., 3-4.

Torda A (2001). Toxoplasmosis. Are cats really the source? Aust Fam Physician, 30, 743-747.

U.S.A: CRC Press, Pp 1-313

Urquhart GM, Armour J, Duncan JL, Dunn AM, Jennings FW (1996). Veterinary helminthology.Veterinary Parasitology, 2, 35-38.

Villena I, Durand B, Aubert D, Blaga R, Geers R, Thomas M, Perret C, Alliot A, Escotte-Binet S, Thébault A, Boireau P (2012). New strategy for the survey of Toxoplasma gondii in meat for human consumption. Veterinary Parasitology, 183(3-4), pp.203-208.

Wang Z, Ge W, Huang S, Li J, Zhu X, Liu Q (2014). Evaluation of recombinant granule antigens GRA1 and GRA7 for serodiagnosis of Toxoplasma gondii infection in dogs.BMC veterinary research, 10(1), pp.1-6.

Woldemichael T., Fontanet A.L., Sahlu T., Gili S.H., Messele T., Rinke de Wit T.F., Yeneneh H., Coutinho R.A. and Gool TV (1998). Evaluation of the Eiken Latex agglutination test for anti-toxoplasma antibodies and seroprevalence of Toxoplasma infection among factory workers in Addis Ababa, Ethiopia. Trans. Roy. Trop. Med. Hyg., 92: 401–403.

Yimer E, Abebe P, Kasahun J, Woldemichal T, Bekele A, Zewudie B, Beyene M (2005). Seroprevalence of human toxoplasmosis in Addis Ababa, Ethiopia. Ethiop. Vet. J., 9: 109–122.

Zhu C, Cui L, Zhang L (2012). Comparison of a commercial ELISA with the modified agglutination test for detection of Toxoplasma gondii antibodies in sera of naturally infected dogs and cats. Iranian J. Parasitol. 7(3), p.89.