ABSTRACT
Toxoplasmosis is a zoonotic infection of animals caused by the protozoan parasite Toxoplasma gondii, which is an obligate intracellular protozoan parasite and cats have a major influence on the epidemiology of the disease. Accordingly, T. gondii is a coccidian parasite with cats as the definitive host, and all warm-blooded animals can be used as intermediate hosts. T. gondii readily infects human beings, and infection is relatively common depending on environmental conditions, age, and immune status. After birth, humans are usually infected with T. gondii by ingestion of oocysts in food or water that have been contaminated with cat feces or by ingestion of tissue cysts. However, before birth, human toxoplasmosis can result from a congenital or acquired infection. The life cycle of T. gondii involves two phases. The sexual phase takes place in the definitive host (Felidae family) and the asexual phase in any warm-blooded animal, including humans and birds. The diagnosis of toxoplasmosis was performed by the isolation of the parasite from patients and by serological tests such as indirect fluorescent antibody (IFA) and enzyme-linked immunosorbent assay (ELISA). The major importance of toxoplasmosis in farm animals is its zoonotic potential. Since cats are key to the transmission of T. gondii as they are the only definitive hosts that shed the oocysts, prevention and control methods should target the Felidae family. Generally, domestic and barn cats should be restricted in farm environments from nesting and defecating in hay, straw mows, grain stores, or other loose piles of livestock feed.

Keywords: Toxoplasma gondii, Toxoplasmosis, Zoonotic importance.

LIST OF ABBREVIATION
AC: Acetone; AIDS: Acquired Immune Deficiency Syndrome; DNA: Deoxyribonucleic Acid; ELISA: Enzyme-Linked Immunosorbent Assay; IFA: Indirect Fluorescent Antibody; MAT: Modified Agglutination Test; PCR: Polymerase Chain Reaction; OT: Ocular Toxoplasmosis.

INTRODUCTION
Toxoplasmosis is among the most important protozoan parasites of worldwide zoonotic disease caused by an obligate intracellular parasite Toxoplasma gondii that can infect almost all warm-blooded animals, including humans and is considered one of the most successful eukaryotic pathogens as reviewed by [1]. The parasite causes abortion in animals...
and humans [2]. During different periods of the parasite life cycle, individual parasites convert into various cellular stages. These stages include the sporozoites, bradyzoites, and tachyzoites. And also consists of two stages: a sexual stage that appears in cat (the definitive host) and an asexual stage that occur in all warm-blooded animals (intermediate host), including humans. It can affect both humans and animals, including camels, cattle, sheep, goats, poultry, and wild animals [3]. The preferred hosts are felids, although a recent study [4] developmental success in mice treated with specific enzyme inhibition and diet modification [5].

The parasite is a cosmopolitan protozoon with no host specificity in the asexual stage, whereas, in the sexual stage, it is specific to felids. Cats are the definitive host for Toxoplasmosis while cattle, sheep, goats, poultry, and camel are reservoir hosts and facilitate the transmission of this disease to humans [6,7]. Toxoplasma infection in livestock leads to significant economic losses because of reproductive failure, i.e., abortion, fetal resorption, and barrenness. Humans are not the primary host, so the zoonotic parasite will act differently in humans and it causes health problems that range from those that are minor to the very serious [8]. Of infected humans, very few have symptoms because a healthy individual immune system usually keeps the parasite from causing illness. However, pregnant women who have compromised immune systems should be cautious, as the disease is abortion fanciest [9].

*T. gondii* infection is substantially more likely to occur in pregnant and immunocompromised people than in immunocompetent people [7]. Intermediate hosts such as rodents become infected after ingesting soil, water, or plant material contaminated with oocysts [10]. Despite its complex yet simple transmission patterns, epidemiological research on Toxoplasmosis is lacking in sub-Saharan Africa [11]. Given the geographical distribution of Toxoplasmosis and the 30% seropositivity rate worldwide [12].

Oocysts, the environmentally robust stage of *T. gondii*, play an important role in the epidemiology of this zoonotic parasite. Attributes of oocysts’ biology may further explain the global distribution of *T. gondii* and how it has evolved to be one of the most prevalent infectious agents of animals and humans. While oocysts are exclusively deposited on land due to definitive hosts being solely terrestrial animals, the prevalent nature of infections observed in aquatic animals demonstrates a significant role in waterborne transmission. High prevalence of *T. gondii* exposure in marine species (up to 100% in some populations [13] further suggests that oocyst transport to, and accumulation in nearshore or open ocean habitats is possible and epidemiologically significant [14].

The diagnosis of toxoplasmosis can be established by serologic tests, amplification of specific nucleic acid sequences by PCR, histologic demonstration of the parasite and/or its antigens by immune peroxidase stain, or isolation of the organism [15]. Globally, caprine animals are susceptible to toxoplasmosis as a result of social contact [16]. It is known to result in reproductive miscarriage [17]. Due to abortion or the production of weak offspring in food animals, this may result in significant economic losses [18]. Due to inbreeding, the infection incidence varies greatly from herd to herd; an average rate of 30% has been observed. A significant factor in disease transmission from domesticated animals to people is the high prevalence of toxoplasmosis in cattle, sheep, and goats [19]. The socioeconomic impact of this disease on people suffering from it and the cost of care for sick children, especially those with mental retardation and blindness, are enormous [20]. In Ethiopia, the incidence of toxoplasmosis is increasing whereas the disease is neglected and infection in humans of children-bearing age is unknown. Therefore, the objectives of this paper are to review the available literature on the etiology, epidemiology, diagnosis, and control of Toxoplasmosis in animals and humans, and its zoonotic importance and status in Ethiopia.

**TOXOPLASMOSIS**

**History of Toxoplasmosis**

Nicolle and Manceaux isolated *Toxoplasma gondii* from the African rodent Chenodactylus gundi in 1908 and used that isolation to name the parasite. The condition was identified as *Toxoplasma gondii* in 1909 after being recognized as leishmania [15].

About the organism’s crescent-shaped morphology, the name of the genus is derived from the Greek word toxon, which means crescent. A serological dye test created in 1948 by Sabin and Feldman provided the foundation for particular diagnostic assays [21].

**Etiology**

*Toxoplasma gondii*, the causative agent, is a systemic coccidian, a universal parasite, a sporozoan, and a member of the suborder Eimeriina. It is a particular parasite of the final host (members of the felidae family), although it has a variety of intermediate hosts [21].

**Epidemiology**

**Geographical distribution**

*Toxoplasma gondii* is a widespread microbe that can be found throughout the world, although tropical regions have greater rates of infection because humidity and warmer temperatures promote the maturation of oocysts in the soil. Around the world, there are significant regional differences.
in the seroprevalence of T. gondii. The parasite is notably more common in Western European, South American, and African nations [22].

Cats play an important role in maintaining T. gondii in nature because they are the definitive hosts for this protozoan parasite and rarely develop clinical disease as a result of infection. Toxoplasmosis has been discovered to be absent from islands that are geographically remote and don’t contain cats [23]. This result can be explained by the group’s hunting techniques and diet, which include wild birds, rodents, and, occasionally, placertes infected with Toxoplasma and stillborn fetuses [24]. The global prevalence of T. gondii infection in humans and animals varies depending on the country’s geographic location. The causes of these variations are not yet known [25]. Infections with T. gondii in cats are usually asymptomatic, and vertical transmissions occur only infrequently. However, latent infections with T. gondii are common in domestic cats and wild felines throughout the world [26].

**Predisposing Factors**

Environmental variables may affect how naturally T. gondii infections spread. Warm, low-lying areas are more susceptible to infection than cold, mountainous areas, while humid regions are more susceptible to infection than dry ones. This is most likely connected to elements that support sporulation and oocyst survival [27].

**Human Health Risk Factors**

Humans are easily infected by T. gondii, and while clinical sickness is not common (roughly 30% of the population depending on age and environment), infection is rather common (about 30% of the population depending on age and environment). In pregnant women, the parasite can pose a serious threat to the fetus if the mother contracts it for the first time while carrying it, as well as immunosuppressed people, such as those undergoing tissue transplants, those with AIDS, certain types of cancer patients, and those receiving certain cancer therapies, are at a higher risk of developing clinical illness. If untreated, these people run the risk of getting an infection that might kill them quickly [28].

**Transmission of Toxoplasmosis**

Fecal-oral transmission is the most common pathway through which T. gondii is spread from the primary host to secondary hosts. Only felids can transmit oocysts (although oocysts can also be produced in the lab) [29].

Toxoplasmosis is mostly spread by cats. Epidemiologic statistics show that the majority of cats get an infection in the wild shortly after weaning, either by sharing food supplied by the dam or by consuming raw pet food. Consequently, wild cats are more likely than domestic cats to get infected with T. gondii. Humans are one of the intermediary host species when Toxoplasma gondii is transmitted vertically. Transplacental vertical transmission is facilitated by tachyzoites, usually after the primary and during the acute phases of infection [28]. T. gondii can also be transmitted via blood or leucocytes from infected donors [30]. T. gondii tissue cysts have been found in the edible tissues of most food animals [20].

Besides its vertical transmission through T. gondii tachyzoites passed from the placenta to the fetus, a horizontal transmission can occur by drinking water, meat, milk, fruits, or vegetables contaminated with bradyzoites or oocytes released from cats [31].

Toxoplasma gondii can also spread through semen in cases where the secondary host is a man and through congenital transmission in cases where the secondary host is a pregnant woman. The connection between these two routes is possible. T. gondii has been linked to sex-transmitted infections in rats [32], dogs [33], sheep [34], and even humans [35].

**Transmission in Animals**

Toxoplasma gondii is a tissue cyst-forming protozoan microorganism that is vertically transmitted to puppies if a female is infected during pregnancy. Infected dogs usually remain clinically normal, but the puppies are severely affected. Toxoplasmosis is known to cause fetal death and is a rare cause of abortion in dogs. Premature birth, stillbirth, and the birth of weak puppies are common in toxoplasmosis [21]. In the environment, oocysts are distributed through wind, rain, surface water, or harvested feeds. They may also be spread via earthworms, coprophagous invertebrates, or manure. Hay, straw, and grain that are contaminated with cat feces have been identified as sources of infection for livestock [35].

The prevalence of infection is highest in young cats hunting for the first time. For the infection of cats, the period of excretion of oocysts is short, approximately 2 weeks. The number of cats excreting oocysts in their feces at any point in time is likely to be quite small, but the contamination of the environment over time is significant. Domestic and barn cats in farm environments tend to nest and defecate in hay, straw mows, grain stores, or other loose piles of commodities that the livestock feeds with manure and bedding from buildings that contain cat feces. Oocysts can survive in the winter in cold climates but are less viable in arid environments [21].

Infection of farm animals occurs as the result of feed or water contaminated with cat feces, which contain infective oocysts, via the ingestion of contaminated stored feeds, contaminated pastures, or contaminated water supplies [36].
Transmission to human

The major modes of transmission of Toxoplasmosis to humans include the consumption of undercooked meat containing Toxoplasma cysts, Fecal-oral transfer of Toxoplasma cysts from cat feces directly or in contaminated food, water, or soil. Vertical transmission from mother to fetus if primary infection occurs during pregnancy [37].

In adults, the incubation period for T. gondii infection ranges from 10 to 23 days after the ingestion of undercooked meat and from 5 to 20 days after the ingestion of oocysts from cat feces [38].

Sources of Infection

Tissue cysts of T. gondii contained in the meat of livestock are an important source of infection for humans. In meat-producing animals, tissue cysts of T. gondii are most frequently observed in the tissues of infected pigs, sheep, and goats, and less frequently in infected poultry, rabbits, dogs, and horses. Tissue cysts are found only rarely in beef or buffalo meat, although antibodies in up to 92% of cattle and up to 20% of buffaloes are evidence of past exposure to the parasite [39].

Cat feces are the sole source of infection for sheep, cattle, and horses. Cats become infected as a result of ingesting tissues of intermediate hosts infected with the parasite; commonly, these are rodents and small birds, but all animals can be IH for T. gondii. Rodents have served as reservoirs of infection for a long time. Feeds of animals contaminated with cat feces due to nests of cats, Actions of earthworms, and other soil inhabitants bring superficially buried feral cat feces to surfaces, which contaminates pastures. Ingestion of meat, dead rodents, cannibalized piglets, and blood while tail- or ear-biting by pigs [21].

Life Cycle of T. gondii

Toxoplasma gondii is a parasite with a distinct life cycle that involves both asexual and sexual reproduction. The sexual cycle of the only species in the genus Toxoplasma, T. gondii, is exclusive to felines [40]. T. gondii can infect a wide variety of warm-blooded animals due to its asexual life cycle, including various species of mammals and birds. T. gondii replicates asexually in the intermediate host and develops into permanent tissue cysts that include the bradyzoite stage [41]. Felids ingest T. gondii by eating encysted bradyzoites on infected intermediate hosts. Bradyzoites are released from cysts and enter the small intestine’s epithelial cells under the action of digesting enzymes and acid. Although the parasite may spread throughout the body of the final host and induce clinical signs, this is not typical [42].

Feline felids that have consumed one of the three infectious forms—tachyzoites, bradyzoites, or oocysts—will shed unpolluted oocysts. The longevity and infectiousness of unpolluted oocysts, which are incapable of infecting hosts, are influenced by climatic factors, specifically temperature extremes and a drop in relative humidity [43]. The sporulated oocysts become infective and environmentally resistant for periods depending on local climatic conditions, up to 12–18 months, and remain viable when stored for at least 54 months at 4 ºC in water [44].

Tachyzoites and bradyzoites are two separate kinds of asexually or clonally reproducing forms that grow in warm-blooded intermediate hosts. Tachyzoites are a type of parasite that proliferates rapidly and is frequently linked to acute infection, reactivation, and vertical transmission. The bradyzoites, on the other hand, are a dormant, encysted form that is slow-growing and metabolically active. Bradyzoites are frequently linked to persistent toxoplasmosis, immunological evasion, and resistance to presently existing anti-toxoplasmosis pharmacotherapies [45].

In immunoprotein people, tissue cysts develop as a result of these protozoans spreading throughout the body via the blood or lymph, which results in a chronic stage of the asexual cycle. Cysts are primarily found in the hosts’ brains, skeletal muscles, and cardiac muscles. The cysts are broken down by proteolytic enzymes after they are consumed by intermediary hosts like dogs, pigs, rats, or humans. Then, bradyzoites are discharged and transformed back into tachyzoites as they infect the epithelium of the intestinal lumen and quickly spread within leukocytes, infecting their hosts’ bodies [27].

Evasion is mediated by cell type tropism and sequestration to immune-privileged sites such as the brain and skeletal muscle, as well as the process of encystation. Both tachyzoites and bradyzoites follow a cell division scheme known as endodyogeny, consisting of a single round of DNA replication by semi-closed nuclear mitosis. The internal assembly of two daughter cells occurs concomitantly with nuclear mitosis inside the mother cell. Tachyzoite cell division is rapid, generating two new cells per mother cell every 6–8 h. Bradyzoites instead divide slower but can assemble and sustain between 1000 and 2000 bradyzoites per cyst [46].
Pathogenesis of Toxoplasmosis

Infection with *Toxoplasma gondii* is acquired by carnivory, ingestion of feces containing oocytes, or congenitally [47]. In an unexposed cat after ingestion of uncooked meat containing tissue cysts, *T. gondii* initiates enteroepithelial replication. Bradyzoites are released from tissue cysts by digestion in the stomach and small intestine and invade the intestinal epithelium by undergoing sexual replication, culminating in the release of oocytes in the feces. Oocysts are first seen in the feces three days after infection and are released for up to 20 days after infection. After exposure to air for 24 hours, oocysts sporulate, become infective, and may persist in the environment for up to one year. Cats generally develop immunity to *T. gondii* after the initial infection and therefore only shed oocysts once in their lifetime [48].

**Clinical Manifestations**

The clinical manifestations of toxoplasmosis vary depending on parasite characteristics such as the virulence of the strain and inoculum size, as well as host factors such as genetic background and immune status [7]. There are at least three genetic types of *T. gondii*: types I, II, and III. They differ in virulence and epidemiological patterns of occurrence [50].

**In animals**

The extent of *T. gondii* infection in cats depends on the availability of infected birds and small mammals. Toxoplasmosis has been confirmed in some 200 species of vertebrates, including primates, ruminants, swine, equine, carnivores, rodents, marsupials, insectivores, and numerous avian species. *T. gondii* causes abortion and neonatal mortality in sheep worldwide. *T. gondii* has been recognized as one of the main causes of infective ovine abortion in New Zealand, Australia, the United Kingdom, Norway, and the Czech Republic all reported having suspected cases of clinical caprine toxoplasmosis. According to Masala et al. [51], *T. gondii* was found in 6.4% of 362 fetuses and 3.0% of 211 aborted tissues in Italy.

**In human**

The most prevalent illnesses were tuberculosis (8.7%), meningeal cryptococcosis (21%), and brain toxoplasmosis (32.2%). More research conducted in Mexico revealed that 47% of AIDS patients' neurological symptoms in the state of Yucatan are caused by toxoplasmosis [52]. The reactivation of a chronic or latent infection brought on by the loss of cellular immune surveillance accounts for more than 95% of toxoplasmic encephalopathy in AIDS patients. According to a study conducted in Thailand [53], among HIV-positive and *T. gondii*-infected individuals, clinical disease is common in immunocompromised individuals or results from an acute infection of the expectant mother. The severity of congenital infections depends on the stage of pregnancy when the acute infection occurs, spontaneous abortions, or neurological disorders [49].

![Figure 1. Toxoplasma gondii life cycle; Source: (Esch and Petersen, 2013).](image-url)
*T. gondii* antibody-positive groups, 43.2% displayed symptoms and signs of acute toxoplasmosis affecting the eye and/or the central nervous system.

Ocular toxoplasmosis used to be attributed to congenital infection, but recently, researchers compared prenatal and postnatal toxoplasmosis. They concluded that at least two-thirds of ocular toxoplasmosis is caused by postnatal infection, which has major public health implications [54].

**Diagnostic Technique**

Human toxoplasmosis can be diagnosed by serological, molecular, or histological methods. Sometimes, these techniques are combined to improve the effectiveness of diagnosis [55]. The diagnosis of *T. gondii* infection or toxoplasmosis may be made using serologic tests, polymerase chain reaction (PCR) amplification of specific nucleic acid sequences, immunoperoxidase staining of tissue, or isolation of the organism [56]. The main test that is advised for the diagnosis of *T. gondii* in both people and various animals is the MAT [27].

**Indirect fluorescent antibody test**

Patient serum is tested for action against dead organisms as part of the IFAT process. The patient’s antibody (IgG) recognizes fixed tachyzoites, and then a fluorescent-labeled anti-human antibody (IgG) is added to enable fluorescence microscopy identification of the antibody. No fluorescence is seen in the absence of an antibody against the *T. gondii* tachyzoite [57].

**Modified Agglutination Test**

The development of a simple direct agglutination test has greatly aided in the serological diagnosis of toxoplasmosis in humans and other animals. In this test, no special equipment or conjugates are needed. The MAT has been extensively used for the diagnosis of toxoplasmosis in animals. The sensitivity and specificity of MAT have been validated by comparing serologic data and the isolation of the parasite from naturally and experimentally infected pigs [23].

In the MAT, sera are treated with 2-mercaptoethanol to remove non-specific IgM or IgM-like substances. This test detects only IgG antibodies; therefore, it may give false-negative results during the early stages of acute infection. The MAT is commercially available (*Toxo-Screen DA*, bioMérieux, Charbonnieres-Beins, France). The Toxo-Screen DA is the same test as the MAT. The results obtained with MAT differ depending on the preservative used to prepare the antigen. Jones (2003) reported that using acetone (AC test) in place of formalin (HS test) can detect IgG present during acute infection. The AC test has been very useful in the diagnosis of toxoplasmosis in AIDS patients and acute glandular toxoplasmosis. Although serum is preferred, the test works with blood plasma and even whole blood; hemolysis does not interfere with the test [27].

**Enzyme-linked immunosorbent assay**

The ELISA system usually includes the solid phase antigen or antibody, enzyme-labeled antigen or antibody, and the substrate of the enzyme reaction, which can be modified to test both antibodies and antigens. ELISA can be automated so that a large number of samples can be simultaneously tested. There have been different types of ELISA developed to detect *T. gondii* antibodies or antigens, such as indirect ELISA, and sandwich ELISA. In the indirect ELISA, the antigen is coated onto the solid phase and the sample containing antibodies is added, the antigen-antibody reaction is enhanced by the addition of a secondary enzyme-linked antibody, and the reaction can be evaluated by quantification of the color that develops [1].

**Detection of *T. gondii* DNA**

Toxoplasma gondii DNA can be detected by PCR. There are many different markers, but the 35-copy B1 gene and the 300-copy 529-bp element are most frequently used. The disadvantage of using a single-copy gene target is that the sensitivity is compromised compared to highly repetitive sequences. However, this can be compensated for to some extent by nested PCR. The 529-bp repeat marker is 10–100 times more sensitive than the B1 marker. The advantage of the recently developed real-time PCR assay is that it also quantifies the parasite DNA. Overall, PCR is very sensitive (it can detect DNA from one tachyzoite), specific, and can provide a rapid diagnosis. It is most suitable for clinical specimens [39].

**Treatments of Toxoplasmosis**

To treat Toxoplasmosis, dihydropteroate synthetase, and dihydrofolate reductase inhibitors were suggested. One of the efficient dihydrofolate reductase inhibitors used to treat this condition is pyrimethamine. Additionally, medications including sulfamethoxazole and trimethoprim were utilized to treat this illness [58]. Depending on where you obtain medical care, the course of treatment for toxoplasmosis during pregnancy may change. Spiramycin may be prescribed if the infection occurs before the 16th week of pregnancy [59]. Congenital toxoplasmosis can cause neurological issues in newborns, although using this medication may lessen that risk [60].

**Prevention and Control**

Although preventive interventions can considerably lower the chance of contracting *T. gondii* infection, they cannot always ward off infection [61]. Accordingly, various agencies, including the World Health Organization, have recommended techniques for the surveillance and management of
Toxoplasmosis is a disease that is neglected in Ethiopia, and it is unknown whether it affects pregnant women. According to a few studies conducted in Ethiopia, the prevalence of toxoplasmosis in the general population ranged from 20.2% to 97.7% [74]. With approximately 82 million people and a high AIDS prevalence, Ethiopia is the second-most populated country in the Horn of Africa. Notably, Adem and Ame [75] found that *T. gondii* antibodies were present in HIV patients with a seroprevalence of 93.3%. The current study showed a strong association between the average monthly income of pregnant women and seropositivity for *T. gondii*. This finding agrees with the previous study conducted in Zambia [76].

In contrast, a study conducted in central Ethiopia showed the absence of a significant association between average monthly income and seropositivity for *T. gondii*. More seropositivity was found among pregnant women with low socio-economic status than high socioeconomic. This significant association could be due to pregnant women of low socio-economic status being more prone to live and work in highly dense areas with poor sanitary conditions and a lack of good education on good hygienic practices [77]. Studies conducted in different geographical locations of Ethiopia from 2007 to 2020 indicated a high seroprevalence of *T. gondii* infection in humans, sheep, goats, and pigs. Hydrocephalus, retinochoroiditis, convulsion, and intracerebral calcifications in fetuses, and lymphadenitis and encephalitis in immunocompromised groups are the major findings of toxoplasmosis [15].
Recently, research in southern Ethiopia found that sheep and goats had an overall seroprevalence of 26.09% for *Toxoplasma gondii* antibodies utilizing the indirect enzyme-linked immunosorbent assay (ELISA) as a diagnostic technique in people [82]. Although *toxoplasma gondii* seroprevalence in pigs in Ethiopia has rarely been reported, a modified agglutination test conducted by Kebeta [83] at pig farms in and around Addis Ababa found a general.

### Table 1. Status of Toxoplasmosis in human

<table>
<thead>
<tr>
<th>Study Area</th>
<th>Population surveyed</th>
<th>No.</th>
<th>Serological test</th>
<th>%</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adama, Hospital</td>
<td>People aged 15 days-65 years</td>
<td>65</td>
<td>MAT</td>
<td>60.0</td>
<td>[78]</td>
</tr>
<tr>
<td>Butajira, Addis Ababa</td>
<td>Patients aged 15–49 years</td>
<td>456</td>
<td>ELISA-VI</td>
<td>95.1</td>
<td>[74]</td>
</tr>
<tr>
<td>Mettu Karl Hospital, Ethiopia</td>
<td>HIV/AIDS Patients</td>
<td>120</td>
<td>Different model checking and model diagnostic test</td>
<td>60</td>
<td>[79]</td>
</tr>
<tr>
<td>Addis Ababa area</td>
<td>Hospitalized patients</td>
<td>330</td>
<td>ELISA-BC</td>
<td>93.3</td>
<td>[80]</td>
</tr>
<tr>
<td>Jimma town, Southwestern Ethiopia</td>
<td>Pregnant women</td>
<td>201</td>
<td>ELISA</td>
<td>83.6</td>
<td>[81]</td>
</tr>
</tbody>
</table>

### Table 2. Status of Toxoplasmosis in animal

<table>
<thead>
<tr>
<th>Host</th>
<th>Year</th>
<th>Population surveyed</th>
<th>No.</th>
<th>Method</th>
<th>%</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>2011</td>
<td>Central Ethiopia</td>
<td>36</td>
<td>MAT</td>
<td>91.67</td>
<td>[84]</td>
</tr>
<tr>
<td>Sheep</td>
<td>1999</td>
<td>Nazareth</td>
<td>116</td>
<td>MDAT</td>
<td>52.6</td>
<td>[85]</td>
</tr>
<tr>
<td>Goat</td>
<td>2010–2011</td>
<td>Central Ethiopia</td>
<td>927</td>
<td>ELISA</td>
<td>19.7</td>
<td>[86]</td>
</tr>
<tr>
<td>Sheep</td>
<td>2010–2011</td>
<td>Central Ethiopia</td>
<td>1130</td>
<td>ELISA</td>
<td>31.59</td>
<td>[82]</td>
</tr>
<tr>
<td>Chicken</td>
<td>2012–2013</td>
<td>Central Ethiopia</td>
<td>601</td>
<td>MAT</td>
<td>30.45</td>
<td>[87]</td>
</tr>
</tbody>
</table>

### CONCLUSIONS

Toxoplasmosis is a zoonotic parasitic disease caused by the protozoa *T. gondii*. It infects a wide range of animals including mammals and birds. While seroprevalence studies indicate relatively high rates of infection in animals subclinical *T. gondii* has virtually no importance as a cause of clinical disease in farm animals except that associated with abortion and neonatal disease in sheep. The major importance of Toxoplasmosis in farm animals is its zoonotic potential as it is the cause of hydrocephalus, retinochoroiditis, convulsions, and intracerebral calcifications in fetuses, lymphadenitis, and encephalitis in immunocompromised patients. For this reason, the epidemiology of Toxoplasmosis in pregnant women immunocompromised patients, and farm animals is important. Thus, health education for women of childbearing age should include information about preventing *T. gondii* transmission from food, soil, litter boxes, and contact with infected cats; Awareness creation should be given to pregnant women about food hygiene and avoiding exposure to cat feces; Domestic and barn cats should be restricted in farm environments not nesting and defecating in the hay, straw mows, grain stores, or other loose piles of commodity livestock feeds, and The government and meat industry should continue efforts to reduce the presence of *T. gondii* in meat.

### DECLARATION

**ETHICS APPROVAL AND CONSENT TO PARTICIPATE**

Not applicable.
CONSENT FOR PUBLICATION

Not applicable.

AVAILABILITY OF DATA AND MATERIALS

All the datasets generated or analyzed during this study are included in this manuscript.

COMPETING INTERESTS

All authors have nothing to disclose in this work.

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AUTHORS’ CONTRIBUTIONS

All authors contributed to data collection, study design, data interpretation, reference search, manuscript writing and editing, and all authors have approved the submission of the final manuscript.

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