

# Review on Toxoplasmosis and Its Zoonotic Importance

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## Review Article

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### ABSTRACT

The genus toxoplasma has a single species, *Toxoplasma gondii*, which is an intestinal coccidian of cats. The life cycle includes a facultative systemic phase, which is an important cause of abortion in sheep and may also cause a zoonosis. Natural infection has been described in almost every mammalian order, including over 19 species of captive and free-ranging ungulates. All felids, but the domestic cat is the most important final host for the parasite and any other mammals including man, or birds are the intermediate hosts. Fortunately, the pathological changes are relatively uncommon and most toxoplasma infections in animals and man are light and consequently asymptomatic. Human infections are particularly serious if they occur during pregnancy and may result in abortion or congenitally acquired disorders, which primarily affect the central nervous system. In Ethiopia, the prevalence of immunoglobulin G antibodies to *Toxoplasma gondii* has been determined by enzyme linked immunosorbent assay and the highest antibody titers were found in children, young and adults. Human immunodeficiency virus-1 frequently leads to resurgence of toxoplasmosis. The most important role of toxoplasmosis in ruminants is its association with abortion and perinatal mortality. In cats and dogs the most frequent signs of toxoplasmosis are associated with infection of central nervous system and the visual, respiratory and gastrointestinal systems.

## INTRODUCTION

Toxoplasmosis is the disease, which results from infection with the protozoan, *Toxoplasma gondii*. Infection has been observed in a wide range of avian and mammalian species and the disease has been recorded in most areas of the world. The organism is considered to be an extremely efficient parasite, as it rarely causes damage to its host <sup>[1]</sup>. Toxoplasmosis was first observed as an intracellular parasite of the spleen and other organs of the African rodent (*Chenodactylus gundi*), since that time increasing interest has been aroused owing to its identification as a pathogen in a large number of hosts, including man. Toxoplasma appears to be ubiquitous in distribution and to be a parasite without specificity in warm blooded-animals. Its predilection site is reticulo-endothelial and central nervous systems. Division is by longitudinal fission and a series of divisions may result in a mass of parasites within a single host cell. Then the aggregation of parasites becomes enclosed by a tough protective membrane called pseudocyst <sup>[2]</sup>. Members of the cat family are the only known definitive hosts for *Toxoplasma gondii* and, therefore act as the main reservoirs of infection. The infective stages of *Toxoplasma gondii* are tachyzoites, bradyzoites and sporozoites. Infection of toxoplasmosis is acquired by carnivorous, ingestion of feces containing oocysts or congenitally. Both domestic and wild cats can develop systemic and patent intestinal infection but all other animals and humans serve as the intermediate hosts in which the parasite may cause systemic infection only, which typically results in the formation of tissue cysts. Human infections are particularly serious if they occur during pregnancy and may result in abortion or congenitally acquired disorders, which primarily affect the central nervous system. People who handle raw meat (such as abattoir workers) are commonly exposed to the parasite. The importance of these various risk factors likely varies considerably between ethnic groups due to differences in cultural habits regarding exposure to undercooked meat, soil and cats. *Toxoplasma gondii* is a major abortifacient in sheep and goats but only rarely causes abortion in cattle and horses <sup>[3]</sup>. The objectives of this paper are to highlight the overall aspects of toxoplasmosis and to review the public health significance of toxoplasmosis.

**History of Toxoplasmosis**

The protozoan was first discovered by Nicollae and Manceaux, who in 1908 isolated it from the African rodent *Chenodactylus gundi*, then in 1909 differentiated the disease from leishmania and named it *Toxoplasma gondii*. The first recorded congenital case was not until 1923 and the first adult case not until 1940. In 1948, a serological dye test was created by Sabin and Feldman, which is now the standard basis for diagnostic tests <sup>[4]</sup>.

**Etiology**

The causative agent, *Toxoplasma gondii*, is a systemic coccidian, a universal parasite, a sporozoan and a member of the suborder Eimeriina. It is a specific parasite of the definitive host (members of the felidae family), but has a wide range of intermediate hosts <sup>[5]</sup>.

*T. gondii* has three infective stages:

- a) Tachyzoites – the rapidly multiplying form of the parasite present during the acute stage of infection in the intermediate hosts.
- b) Bradyzoites – present in the tissue cysts.
- c) Oocysts (containing sporozoites) – present only in the cat feces <sup>[6]</sup>.

*Toxoplasma gondii* has an enteroepithelial cycle, occurring only in domestic cats and certain other members of felidae. When immunity develops, shedding of oocysts in the feces ceases. *T. gondii* also has an extra-intestinal cycle that occurs in many mammals including cats and birds <sup>[7]</sup>.

The fecal oocysts develop to sporocysts. Systemic infection results in formation of rapidly multiplying forms known as tachyzoites. As immunity develops, the rate of replication of the organism declines and the organism may be found in tissue cysts; in this form they are referred to as bradyzoites <sup>[5]</sup>.

**Host range susceptibility**

The cat plays a central role in the epidemiology of toxoplasmosis and the disease is virtually absent from areas where cats do not occur. Epidemiological investigations in the USA indicate that 60% of cats are serologically positive to toxoplasma antigen, the majority acquiring infection by predation. As might be expected infections are more prevalent in stray cats. Congenital infection is rare. Toxoplasmosis occurs in domesticated and wild animals and birds in most parts of the world although difference in prevalence exists. A recent national survey in swine in the United States found a seroprevalence of 20%. **Table 1** shows the rate of seropositivity is higher in breeding swine than in feeders <sup>[3]</sup>.

**Table 1.** The seropositive prevalence of toxoplasmosis on worldwide basis on different animals and human being.

Animals	Prevalence in %
Swine	22
Sheep	21
Goats	25
Horse	15
Human	>25

The true seroprevalence of toxoplasmosis in cattle is not known due to the inaccuracy of the standard serological tests for cattle, hence low in cattle, reflective of relative unimportance of toxoplasmosis in cattle <sup>[8]</sup>. In pigs an epizootic condition has been described in the USA, the symptoms, lesions and organisms were seen in the lung, liver, kidneys and lymph nodes of the piglets and toxoplasma was recovered after mouse inoculation with material from the brain of the piglets’ mother. It is difficult to explain the widespread prevalence of toxoplasmosis in ruminants, particularly sheep, in view of the relatively low number of oocysts shed into the environment. Pregnant ewes are most commonly infected during periods of concentrate feeding prior to lambing, the stored food having been contaminated with cat feces in which millions of oocysts are present <sup>[3]</sup>. Tachyzoites have been demonstrated in urine, feces, milk, saliva and semen but are an unlikely source of infection. Tachyzoites cannot survive long in the environment and more susceptible to gastric digestion than bradyzoites. Oocysts may persist in the indoor and outdoor environment for more than a year <sup>[4]</sup>. Oocysts are extremely resistant to environmental influences and remain infective for a year in warm, moist climates and longer under cooler conditions. Recently flies, cockroaches, snails and earthworms have been implicated as transport hosts for toxoplasma oocysts. There is a high rate of infection among veterinary surgeons and people who keep kennels for dogs. Uterine infection in woman may lead to the death of the child. Up to 1/3 of the world’s human population is estimated to carry a toxoplasma infection <sup>[9]</sup>.

The Centers for Disease Control and Prevention (CDCP) notes that overall seroprevalence in the United States as determined with specimens collected by The National Health and Nutrition Examination Surveys (NHANES) between 1999 and 2004 was found to be 10.8%, with seroprevalence among women of child bearing age (15-44 years) of 11%. It is estimated that between 30-65% of world population is infected with toxoplasmosis <sup>[7]</sup>.

### Sources of infection

- a) Cat feces – the sole source of infection for sheep, cattle and horses.
- b) Tissues of intermediate hosts – cats become infected as a result of ingesting tissues of Intermediate Hosts (IH) infected with the parasite, commonly these are rodents and small birds but all animals can be IH for *Toxoplasma gondii*. Rodents serve as reservoirs of infection for a long time.
- c) Feeds of animals contaminated with cat feces due to nests of cats.
- d) Actions of earthworms and other soil inhabitants - brings superficially buried feral cat feces to surfaces, which contaminates pastures.
- e) Ingestion of meat, dead rodents, cannibalized piglets and blood while tail or ear biting by pigs <sup>[2]</sup>.

### Risk factors

- a) Pathogen risk factors – oocysts are extremely resistant to external influences and can survive in the environment for at least one year. Oocysts are destroyed by exposure to temperatures between 90°C or 194°F for 30 s and 50°C for 2.5 min.
- b) Environmental and management risk factors.
- c) A high rate of infection has been shown in sheep due to high rainfall, which allow a longer survival of oocysts on pasture. Sheep raised in high rainfall area without cat have almost no toxoplasmosis, whereas sheep raised in high rainfall with cats can have an infection rate as high as 32%. Direct sheep to sheep transmission might occur by close contact with grossly infected placenta <sup>[10]</sup>.

Two risk factors for contracting toxoplasmosis in human being are; infants born to mothers who become infected with toxoplasma for the first time during or just before pregnancy and persons with severely weakened immune systems, such as those with HIV-AIDS. Illness may result from an acute toxoplasma infection or reactivation of an infection that occurred earlier in life <sup>[11]</sup>.

### Transmission in animals

*Toxoplasma gondii* is a tissue cyst forming protozoan microorganism that is vertically transmitted to puppies if a bitch is infected during pregnancy. Infected bitches 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. Further spread of oocysts may occur via coprophagous insects which can contaminate vegetables, meat and animal fodder. Venereal transmission can occur in sheep. The prevalence is higher in veterinarians, abattoir workers and those who handle cats <sup>[3]</sup>. Other animals such as sheep, cattle and horses are infected due to the oocysts passed in the feces of the cat family. Oocysts are also the major source of infection for swine although it is possible for swine to be infected by the ingestion of tachyzoites or bradyzoites present in meat <sup>[12]</sup>.

The prevalence of infection is highest in young cats hunting for the first time. For the infection of cat 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 environment tend to nest and defecate in hay, straw mows, grain stores or other loose piles of commodity of the livestock feeds with manure and bedding from buildings that contain cat feces. Oocysts can survive in winter in cold climates but are less viable in arid environments. Infection of farm animals occurs as the result of infection 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 <sup>[2]</sup>.

### Transmission to human

The major modes of transmission of toxoplasmosis to human being include:

- 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.

In adults the incubation period for *Toxoplasma 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 <sup>[13]</sup>.

### Life cycle

**Figure 1** shows the life cycle of toxoplasmosis, unsporulated oocysts of toxoplasma are passed in the feces of acutely infected cats. The oocysts usually sporulate in 1 to 5 days forming two sporocysts, each containing 4 infective sporozoites. When the oocysts are ingested by any warm-blooded host, the sporozoites excyst, invade intestinal cells and begin to divide asexually to produce tachyzoites. These then migrate throughout the body, invading tissue cells and multiplying until the cells rupture. Eventually, tachyzoites excyst, becoming bradyzoites within the cells of the central nervous system, muscles and sometimes other

organs. The cysts typically persist until the death of the host without causing clinical signs. If the host is eaten by other animals, the bradyzoites excyst in the intestine and the process is repeated, forming new tissue cysts.

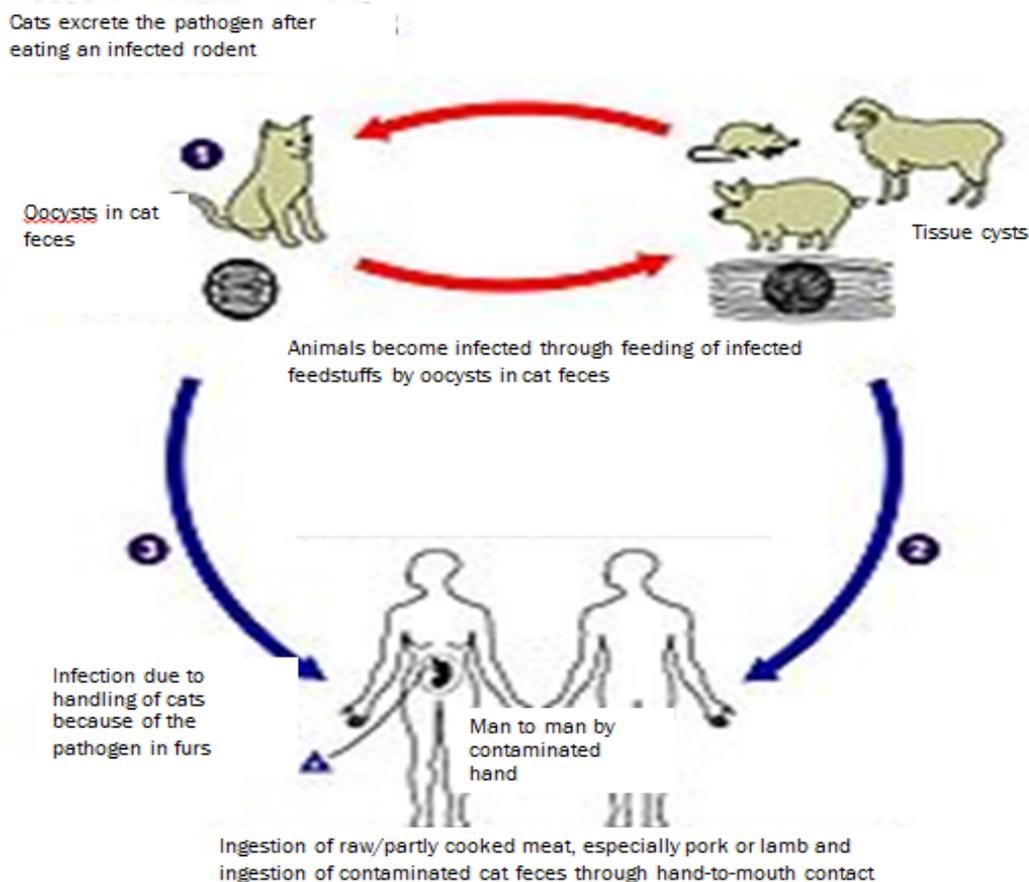


Figure 1. Life cycle of toxoplasmosis [14].

When fed tissue cysts, 97% of cats infected for the first time will produce oocysts, usually within 3-10 days. They may shed for up to 20 days but the majority of oocysts will be shed in just 1-2 days. Only 20% of cats fed oocysts will develop a patent infection, and the prepatent period may be 18 days or more. Contrary to previous beliefs, studies have shown that oocysts can be shed in low numbers by previously infected cats that are challenged again with the parasite or that become immunosuppressed.

Table 2. Major features of the life cycle of *Toxoplasma gondii*.

No.	Features	Interpretation
1	Life cycle	Indirect or direct. Also between IH
2	Infective stage for FH	Bradyzoite cysts, tachyzoites and small oocysts
3	Infective stage for IH	Bradyzoites, tachyzoites and oocysts
4	Asexual phase	In many hosts
5	Sexual phase	In cats

**Pathogenesis**

Infection from *Toxoplasma gondii* is acquired by carnivorousism, ingestion of feces containing oocysts or congenitally [15]. In 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 intestinal epithelium by undergoing sexual replication, culminating in the release of oocysts in the feces. Oocysts first seen in the feces at three days after infection and released for up to 20 days after infection. After exposure to air for 24 h 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 life time (Table 2) [11].

In all warm-blooded animals after ingestion of uncooked meat containing tissue cysts or feed contaminated with cat feces containing oocytes, *T. gondii* initiates extra intestinal replication. Bradizooites and sporozooites, respectively are released and infect intestinal epithelium. After several rounds of epithelial replication, tachyzoites emerge and disseminate via the blood stream and lymph. Tachyzoites infect tissues throughout the body and replicate intracellular until the cells burst, causing the tissue necrosis. Young and immunocompromised animals may succumb to generalized toxoplasmosis at this stage. Older animals

mount a powerful cell-mediated immune response to the tachyzoites. Tissue cysts are usually seen in the neurons and in cardiac and skeletal muscles. Tissue cysts remain viable in the host for many years [16].

Placental infection in large animals about 14 days after ingestion of oocysts. Infection acquired before 50 days of gestation may result in embryonic death and resorption. Infection b/n 60 and 100 days gestation usually causes fetal death or birth of weak lambs. Infection during the last month of gestation has no apparent effect on the fetus. In natural infections most abortions occur one month before parturition. In experimental infection of ewes 6 and 14 weeks of pregnancy, abortions occurred 1–2 months after inoculation [15].

### **Clinical signs and symptoms**

There are 4 forms of toxoplasmosis – subclinical, sub-acute, acute and chronic (latent). Most cases of exposure result in subclinical infection with no clinical signs. The sub-acute infections result in sudden death with few or no overt clinical signs. The acute form results from infection of tissues by tachyzoites and resultant tissue reactions. The organism affects various tissues and the clinical signs are referable to the tissue involved. The most commonly affected tissues are lungs, liver, brain, heart, placenta, eyes, spleen, lymph nodes and adrenal glands. The latent form of toxoplasmosis refers to the quiescent tissue phase. Bradyzoites within cysts remain inactive and cause no tissue reaction. Recrudescence of these cysts to the acute stage may result from stress or other cause of immunosuppression [17].

Most infections are acquired via the gastrointestinal tract. In cats, the lesions that result from the intraepithelial cycle are usually not serious and do not produce clinical signs. In other animals tachyzoites causes damage to many organs [18]. The clinical syndrome and the course of toxoplasmosis vary a great deal between species and age groups. The disease usually runs an acute course in cattle. Fever, dyspnea, nervous signs are seen earlier, followed by extreme lethargy and stillborn but not play significant role in causing bovine abortion.

Pigs are highly susceptible and all ages can be affected. The principal manifestations in sheep are fetal resorption, abortion, mummified lambs and neonatal death. The clinical disease in horse is rare. Natural outbreak in fowls has been reported and the parasite was transmitted to mice. In human beings, toxoplasma infections are associated with clinical signs such as mild fever and lymphadenopathy, and may appear similar to mononucleosis or Hodgkin disease. Signs may persist for 1–12 weeks; more severe disease is very rare in immunocompetent individuals. Ocular toxoplasmosis (retinitis), encephalitis, hepatitis, myositis, and pneumonia develop. Worldwide, toxoplasma encephalitis develops at some time in approximately 40% of individuals with AIDS. Approximately 10% of congenital toxoplasma infections result in abortion or neonatal death. Signs in this time include hydrocephalus, chorioretinitis, hepatosplenomegaly, microcephaly and small size fetus [10].

Recently studies shows that there is a cutaneous form of toxoplasmosis, where rare skin lesions may occur in the acquired form of the disease, including roseola and erythema multiform-like eruptions, prurigo-like nodules, urticaria and maculopapular lesions [6].

### **Diagnosis in animals**

*T. gondii* is an intestinal coccidian of cats. Its oocysts are usually diagnosed using a standard fecal floatation. Oocysts are unsporulated in fresh feces and measure 10 to 12 µm. The prepatent period is highly variable, ranging from 5 to 24 days, and depends on the route of infection. Toxoplasmosis should be added to the list of tentative diagnosis if major organ systems (lung, liver and CNS) are affected, and domestic cats are present in the area. The definitive diagnosis, however, requires the demonstration of a rising serum antibody titer or identification of the organism in biopsy or necropsy samples [2].

Diagnosis of toxoplasmosis on clinical grounds alone is generally not possible because of the wide variety of clinical signs that can occur. Immunosuppression from drug administration or other means (e.g. canine distemper) may allow rapid multiplication and dissemination of toxoplasma organisms with production of clinical disease [7].

Diagnosis may also be confirmed by inoculation of suspicious material into mice and subsequent examination of exudates or tissues for evidence of infection. Often a definitive diagnosis of toxoplasmosis is made only at autopsy. The possibility of immunosuppression resulting from the effects of canine distemper virus on thymus and other lymphatic tissues should be considered as a possible mechanism for activation of latent toxoplasma infection. The most convincing diagnosis is obtained by inoculating toxoplasma free mice by the intraperitoneal or intracerebral route with test material and the subsequent demonstration of tachyzoites or bradyzoites in smears of organs or serous cavities. A compliment fixation test and haemagglutination tests are also used to diagnose toxoplasmosis [3].

### **Diagnosis of toxoplasmosis in human being**

There are several different serological tests to diagnose toxoplasmosis in humans that are intended to differentiate between latent and acute infections.

- Serum IgM titers – indicate recent infection.
- Serum IgG titers – indicate previous infection.

- The modified latex agglutination test (MAT) detects IgG – helps to differentiate acute and chronic infections based on reactivity with acetone versus formalin- fixed antigen.
- Diagnosis of in utero infection is most commonly accomplished by detecting toxoplasma DNA in amniotic fluid using PCR.
- Ultrasonography to look for physical congenital defects of the fetus [7].

### **Differential diagnosis**

Toxoplasmosis should be considered in Differential Diagnosis (DDX) of multifocal CNS diseases, particularly in dogs that have been adequately vaccinated against distemper. Toxoplasmosis is rarely considered in a primary diagnostic list other than with problems of abortion and associated neonatal mortality. The differential diagnosis of abortion in cattle and sheep is related with brucellosis and in pig with leptospirosis. The cause of encephalitis in animals is related with viral infections (e.g. rabies), bacteria (e.g. *Listeria monocytogens*, sarcocystosis & *Haemophilus somnus*) and verminous encephalomyelitis related with migration of larva of parasitic species having somatic migration (e.g. *Micronema deletrix*, *Paraelaphostrongylus tenuis*) [12].

Samples for confirmation of diagnosis:

- a) Parasitology – fresh or chilled brain, lung, placenta
- b) Serology – fetal thoracic fluid
- c) Histology – placental cotyledons, lung, liver, brain, spinal cord, kidney and heart.

### **Necropsy and laboratory findings**

In the early stages of infection in cats, oocysts can be demonstrated in feces by using conventional parasitological flotation techniques to concentrate and identify the organism. Examination of cerebrospinal fluid from animals with neurologic disease as a result of toxoplasma infection may reveal an increase in protein (greater than 35 mg/dl) and an increase in cell number, particularly neutrophils, reflecting the necrosis and hemorrhage present at sites throughout the nervous systems. The most characteristic gross lesion of toxoplasmosis is the presence of white chalky foci of necrosis and calcification up to 2 mm in diameter in cotyledons [19].

### **Treatment in animals**

Specific proven treatment for toxoplasmosis in exotic ruminants is lacking. Anti-inflammatory drugs may be beneficial in conjunction with antibiotic therapy for the treatment of series ocular inflammation. Treating with a combination of sulphamethazine and pyrimethamine has proved to be effective in mitigating the effects of experimentally induced toxoplasmosis in pregnant ewes. Treatment is administered over 3 days for 3 periods with an interval of 5 days. Chemotherapy with sulphadiazine (60 mg/kg/day) for every 4-6 h and pyrimethamine (0.5-1 mg/kg/day) as a single dose, limits the spread of infection until host immunity is acquired. These two drugs are synergistic and inhibitors of folate metabolism. Folinic acid (1 mg/kg/day) may be administered to prevent their toxic side effects [12].

Auromycin has been shown to check the disease if treatment starts at the beginning of infection but has little effect if given during the invasive phase. The disease responds markedly to treatment with sulphonamides and pyrimethamine in synergic combination. Anticoccidials, such as monensin and toltrazuril decreases oocysts shedding in cats if the animal is exposed or becomes immunosuppressed.

### **Treatment in human**

In humans traditional therapy for clinical toxoplasmosis consists of a combination of pyrimethamine and sulphonamides but not recommended in pregnant women due to potential side effects on the fetus (due to inhibition of folate synthesis). Spiramycin, (a macrolide), is one of the current drugs of choice for pregnant women having toxoplasmosis. In order to prevent reactivation of disease in sero positive patients who develop AIDS, treatment with a trimethoprim –sulfamethoxazole, dapsone-pyrimetamine or fansider has been used [7].

### **Prevention and control**

There are two concerns in the control of toxoplasmosis in agricultural animals. The first one is to reduce the economic effects of infection in agricultural animals and the second one is to reduce the risk of human disease associated with consumption of infected meat especially meat of domestic animals [20].

On farms, control of toxoplasmosis is more difficult, but where possible animal feed should be covered to exclude access by cats and insects. Monensin and decoquinate have been administered to ewes in mid-pregnancy in attempts to control abortion due to toxoplasmosis. Fortunately a live vaccine consisting of tachyzoites attenuated by a repeated passage in mice is now available for sheep. The strain used has lost the capacity to form oocysts in cats. It is usually recommended to vaccinate the whole flock initially and thereafter only annual vaccination of replacements. The vaccine consists of 10000-1000000 tachyzoites and it is given as a single dose IM at least 3 weeks prior to tupping or lambing [3].

If a cat is found to be shedding oocysts, it should be removed from the premises temporarily and treated to eliminate shedding. Because cats are usually meticulous groomers, it is unlikely that oocysts will be found on their fur, so regular handling is not a significant risk. Microwave cooking, salting and smoking do not consistently kill all infective toxoplasma organisms. Freezing meat to a -12°C for at least 24 h will kill most toxoplasma tissue cysts, but sporulated oocysts can survive at -20°C for up to 28 days. Washing kitchen, utensils and surfaces that have come in contact with raw meat with soap and scalding hot water will kill any bradyzoites or tachyzoites. Individuals should always wash their hands thoroughly after contact with cat stool, litter or litter box. Cats should be kept out of sandboxes and other areas where children play as cats may be inclined to defecate in them <sup>[7]</sup>.

Infected ewes or does seldom abort from toxoplasmosis in subsequent pregnancies. The prevalence of abortion can be reduced by avoiding contamination of feedstuffs with feline feces. Cats should not be allowed to eat placentas or carcasses that may contain tachyzoites or tissue cysts. In endemic areas, exposing replacement ewes to aborting ewes may provide immunity before breeding age. Education of individuals with high risk (i.e., pregnant women and immunocompromised) decreases the transmission of toxoplasmosis <sup>[2]</sup>.

The following measures are particularly important for these individuals, but also apply to the prevention of toxoplasmosis in general:

- Cooking all meat to a minimum internal temperature of 67°C or 153°F.
- Peeling or thoroughly washing fruit and vegetables prior to consumption.
- Cleaning all surfaces and objects that come in contact with raw meat or unwashed fruit and vegetables.
- Avoiding contact with cat litter and garden soil, otherwise wearing gloves and washing hands thoroughly after.
- Avoiding feeding raw meat to cats.
- Keeping cats indoors so they do not become infected by eating small prey by predation <sup>[15]</sup>.

### **Zoonotic importance**

While seroprevalence studies indicate 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. A major importance of toxoplasmosis in farm animals is its zoonotic potential <sup>[21]</sup>.

Humans are the intermediate hosts for *T. gondii* and approximately one half of the population of the United States is infected. Infection of man is acquired or congenital. Acquired infections occur in two ways: either from the ingestion of oocysts shed in the feces of cats by direct hand contamination or indirectly from ingestion of contaminated food and ingestion of undercooked meat containing toxoplasma cysts. Most infections probably arise from the ingestion of oocysts from cat feces that contaminate food or that are inadvertently ingested because of poor hygienic practices. However, human infection can also result from ingestion of bradyzoites and tachyzoites in meat or tissues that are eaten or handled. The risk is with raw or undercooked meats. Beef is a minor source of infection, with pig, sheep and horse meat having greater risk <sup>[8]</sup>.

Tachyzoites are secreted in the milk of goats challenged with oocysts and raw goat's milk has a public health risk for toxoplasmosis, although the risk is minimal <sup>[17]</sup>.

There is usually no clinical disease in humans infected with *T. gondii* or the disease is mild and self-limiting. Significant disease can occur in humans suffering from acquired immune deficiency syndrome (AIDS), malignancy, in those treated with cytotoxic or immunosuppressive drugs, and in the very young and very old. There is also a risk for abortion or congenital infection of the fetus such as hydrocephalus, intracranial calcification and retinochoroiditis. Toxoplasmosis poses an occupational risk for veterinarians, farmers and slaughterhouse workers who handle infected material. The risk is particularly high with contact with lambing ewes in infected flocks, veterinarians and farm workers, especially if pregnant, should take precautions to avoid infection when handling infected material <sup>[8]</sup>.

### **The disease situation in Ethiopia**

Toxoplasma seroprevalence is variable, higher prevalence being observed in warm and moist areas than in cold or hot dry areas. Apart from this, variation may also be related to the age of the animals and husbandry practices. The overall prevalence recorded in sheep in Ethiopia and other African countries is 54.7% <sup>[22]</sup>.

The overall seroprevalence of 26.7% recorded in goats from Ethiopia. The prevalence rates ranging from 11.5% to 39% have been recorded in various African countries including Ethiopia <sup>[23]</sup>.

In Ethiopia, the prevalence of IgG antibodies to *T. gondii* has been determined by ELISA. One thousand and sixteen sera collected in different geographical regions were analyzed. Antibody titers >15 lu/ml were detected in 74.4% of the specimens, out of which titers exceeding 200 lu/ml in 1/3 were ELISA-positive sera. The highest antibody titers were found in children and 75% of young adults had sero-converted. As infection with the human immunodeficiency virus 1 (HIV-1) frequently leads to a resurgence of

toxoplasmosis, the study conducted by and showed the occurrence of high anti toxoplasma antibodies in both HIV infected (74.2%) and non HIV infected (83.3%) persons [24]. Similar results were also reported in the study carried out [25]. However, higher *T. gondii* antibody titers were recorded in persons infected with HIV when compared with HIV negative individuals in another study [26].

## CONCLUSION AND RECOMMENDATIONS

Toxoplasmosis is important parasitic zoonoses of wide range of animals including man and birds, caused by protozoa known as *Toxoplasma gondii*. Even though the disease has high rates of infection in farm animals; it has virtually no importance as a cause of clinical disease in farm animals except its association with abortion and neonatal disease in sheep. The major importance of toxoplasmosis is its zoonotic potential. Among the signs hydrocephalus, retinochoroiditis, convulsion and intracerebral calcifications in fetus and lymphadenitis and encephalitis in immunocompromised groups are the major findings of toxoplasmosis.

In general, toxoplasmosis is highly important in its high-risk groups and; in order to reduce its means of transmission, the following recommendations are forwarded:

- The adequate research should be carried out to estimate the public health importance of toxoplasmosis.
- Education of women of age 15-44 years, especially pregnant women, about the transmission, prevention and control of toxoplasmosis is required. Domestic and barn cats should be prevented from nesting and defecating in hay, straw mows, grain stores or other loose piles of commodity of livestock feeds present in the farms.
- Veterinarians, slaughterhouse and abattoir workers should take care when faced the sources of *T. gondii* in order to minimize its transmission.
- Individuals should always wash their hands thoroughly after contact with cat stool, litter or litter box.

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