Toxoplasmosis: Epidemiology with the emphasis of its public health importance

Teshager Dubie*1, Getachew Terefe1, Mebratu Asaye1 and Tesfaye Sisay2

Abstract

Toxoplasmosis is a protozoal disease, which is capable of infecting any warm-blooded animals, including humans. Wild and domestic cats are the only known definitive hosts of Toxoplasma; they can develop both systemic and patent intestinal infection. All other animals and humans serve as intermediate hosts in which the parasite may cause systemic infection, which typically results in the formation of tissue cysts. In all species, Toxoplasma infection is usually subclinical, although it may occasionally cause mild, non-specific signs. Infection may have much more serious consequences in immunocompromised or pregnant animals and people and HIV AIDS patients. The major modes of transmission include consumption of undercooked meat containing Toxoplasma cysts, fecal-oral transfer of Toxoplasma oocysts from cat feces (either directly or in contaminated food, water or soil), and vertical transmission from mother to fetus if primary infection occurs during pregnancy. The major public health significance is the risk of having cats in the same hold with pregnant women, children and immunocompromised patients which are highly susceptible to the disease. When women are exposed during pregnancy, birth defects such as abortion, still birth, blindness and hydrocephalus are the most commonly encountered congenital defects. The economic significance of T.gondii is mainly due to reproductive failure in animals, condemnation of meat and wastage of milk, treatment cost in humans and vaccination cost in cats.

Keywords: Toxoplasma gondii, Toxoplasmosis, Public health, Definitive host, Intermediate host.

INTRODUCTION

Both domestic and wild animals are affected by diversity of diseases of different origin. They may also carry disease causing pathogenic agents that can seriously affect the wellbeing of other animals and man. Such infections of economic and public health significance may range from the intracellular viruses and protozoans to the multicellular helminthes and arthropods. Among protozoan parasitic diseases of economic and public health importance is toxoplasmosis caused by various species of Toxoplasma of which T. gondii is the most important. Toxoplasma gondii is an obligate intracellular protozoan that infects humans and a wide range of mammalian and birds (Smith and Reduck, 2000).

It is one of the most known zoonotic diseases among physicians, veterinarians and the public. This parasite is known to cause congenital diseases and abortion both in
humans and animals (Dubey and Beatie, 1988). Wild and domestic cats are the only definitive hosts of *T. gondii*; they develop both systemic and patent intestinal infection. All other animals and humans serve as intermediate hosts in which the parasite may cause systemic infection, which typically results in the formation of tissue cysts. Wild and domestic cats, therefore, serve as the main reservoir of infection for other animals and man. There are three infectious stages of *T. gondii*: tachyzoites (rapidly multiplying form), bradyzoites (tissue cyst form), and sporozoites (in oocysts) (Urquhart, 1996).

In all species, *Toxoplasma* infection is usually subclinical, although it may occasionally cause mild, non-specific signs. Infection may have much more serious consequences in immune-compromised or pregnant animals and humans. However, this agent is responsible for visual losses in at least 1% of the infected individuals, with deaths and great morbidity in fetuses (Culloch et al., 1995) and immune-compromised patients. *T. gondii* has been recognized in recent years as a significant cause of morbidity and mortality in children infected in utero and in immunocompromised patients, notably bone-marrow, heart transplant and AIDS patients. Acute primary infection poses greatest risk, particularly for children infected in uterus and for immunocompromised patients. However, a significant number of patients suffer from the sequelae of reactivation of a latent infection. Particularly, the increasing numbers of AIDS patients with latent *T. gondii* infection are at risk for developing central nervous system disease due to *T. gondii* infection of the brain (Tenter et al., 2000).

Free-living animals such as stray cats and dogs could be used as sentinels of environmental spreading with *T. gondii* in densely built urban areas, since they are exposed without any protection to all the infective forms of the parasite. Living in the same environment, dogs and humans are similarly exposed to *T. gondii* contamination and despite their different hygienic behaviors; canine toxoplasmosis might be an important epidemiological indicator of the risk of toxoplasmosis to man (Meireles et al., 2004).

The objectives of this review are therefore, to high-light:
- The available knowledge on animal and human toxoplasmosis
- The possible management approaches to combat the problem.

**General overview of toxoplasmosis**

**Historical background**

The organism was first discovered in 1908 in Tunis by Charles Nicolle and Louis Manceaux from North African rodent, *Ctenodactylus gundi*. In the same year it was also described in Brazil by Alfonso Splendore in rabbits. Then in 1909, the disease was differentiated from Leishmania and named as *Toxoplasma gondii* (Ukthana, 2006).

Between 1908 and 1937, there were a number of reports identifying *Toxoplasma*-like organisms in a number of animal species, including humans. However, the first detailed scientific study was undertaken using techniques previously employed in studies of viruses. They showed that *Toxoplasma* was an obligate intracellular parasite that could be passed in laboratory animals by intracranial, subcutaneous and intraperitoneal inoculation of brain homogenates. Interestingly, they also noted that mice, fed on recently dead, infected animals, became infected. They suggested as early as 1937 that “one method of natural dissemination may be by means of eating of *Toxoplasma*-contaminated tissue” (Sabin and Olitsky, 1937).

**Taxonomic classification of Toxoplasmosis**

It belongs to the phylum Apicomplexa, which consists of protozoan featured with polarized cell structures and complex cytoskeleton and organelle arrangement at their apical end together with *Hammondia, Neospora, Besnoitia, Frenkella, Cryptosporidium, Isospora, Eimeria and Sarcocystis*. Different species were assigned to *Toxoplasma* isolates based on the species of the host from which they were isolated. However, no biological and serological differences exist among the various isolates. Hence *Toxoplasma gondii* is the unique species of *Toxoplasma* organisms known to date. The taxonomic classification of *T. gondii* presented as below (Table 1)

**Etiology of Toxoplasmosis**

The causative agent of toxoplasmosis is *T. gondii*, which is a coccidian, universal parasite. It is a specific parasite of definitive host *felidae family*, but has a wide range of intermediate hosts. *T. gondii* has three infective stages; Tachyzoites-the rapidly multiplying form of parasite present during the acute stages of infection in the intermediate host; Bradyzoites-slowly multiplying form of the parasite present in the tissue cysts; Oocysts-which containing sporozoites present in the cat faeces (Radostitis et al., 2006).

**Life cycle of Toxoplasma gondii**

**Sexual phase (enteroepithelial life cycle)**

Most cats become infected by ingesting *Toxoplasma* infected animals, usually rudcnls, whose tissues contain tachyzoites or bradyzoites, although direct transmission of oocysts between cats can also occur. 'The ingestion
Table 1. Taxonomic classification of *Toxoplasma gondii*

<table>
<thead>
<tr>
<th>Kingdom</th>
<th>Protista</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subkingdom</td>
<td>Protozoa</td>
</tr>
<tr>
<td>Phylum</td>
<td>Apicomplexa</td>
</tr>
<tr>
<td>Class</td>
<td>Conoidasida</td>
</tr>
<tr>
<td>Order</td>
<td>Eucoccidiorida</td>
</tr>
<tr>
<td>Suborder</td>
<td>Eimeriorina</td>
</tr>
<tr>
<td>Family</td>
<td>Sarcocystida</td>
</tr>
<tr>
<td>Subfamily</td>
<td>Toxoplasmatinae</td>
</tr>
<tr>
<td>Genus</td>
<td>Toxoplasma</td>
</tr>
<tr>
<td>Species</td>
<td><em>Toxoplasma gondii</em></td>
</tr>
</tbody>
</table>

Sources: (Tenter et al., 2000)

Figure 1. Oocysts of *T. gondii*: A = unsporulated oocysts, B and C = sporulated oocysts (arrows) (Dubey et al., 1998).

Asexual phase (extraintestinal life cycle)

The *Toxoplasma gondii* oocysts present in the excreta of cats are ingested by other prospective hosts as shown in Figure 1. Humans acquire this parasite after eating uncooked meat and contaminated foods that contain oocysts or through eating with unwashed hands. In the host's body, sporozoites are released that invade the macrophages of the intestine. The sporozoites are differentiated into motile tachyzoites, which are then distributed to other parts of the body via blood circulation. The tachyzoites enter the bradyzoite stage, resulting in tissue cysts (Figure 2). These tissue cysts, upon ingestion by cats, will initiate the sexual life cycle again. Healthy cats can get infected this way too (Boothroyd, 2000).

Epidemiology

Host range and susceptibility

Toxoplasmosis is a true zoonosis occurring naturally in
man, domestic and wild animals and birds (Radostitis et al., 1994). As shown in Figure 4 among farm animals, pigs, sheep and goats are more susceptible to *T. gondii* infection than others. The cat plays a central role in the epidemiology of toxoplasmosis and the disease is virtually absent from areas where cats do not occur (Urquhat et al., 1996). The incidence of infection in humans and animals may vary in different parts of a country or regions depending on the presence of felines and a suitable ambient condition; such as temperature, humidity and aeration needed for development of the infective stages, the type of test used and the species of hosts examined. Environmental conditions, cultural habits, and animal species are among factors that may determine the degree of natural spread of *Toxoplasma gondii* (Radostits et al., 2006).

Infection rates in cats are largely determined by the rates of infection in the local avian and rodents' population, which serve as food sources. For instance, *T. gondii* oocysts were found in 23.2% of 237 cats in Costa Rica where infection in the local rodents and birds was high. The prevalence of *T. gondii* infection in feral cats is very high, when compared to owned cats, as they are more engaged at predating rodents (Dubey et al., 2002b.)
- Pigs, sheep, goats
- Free ranging poultry, pigeon’s farm deer, game animals (including hares, and birds), domestic rabbits and dog.
- Horses, commercially raised poultry.
- Buffaloes, cattle.

**Figure 4. Relative susceptibility of meat-producing and game animals to T.gondii infection.**

**Sources and reservoirs of Toxoplasma infection**

*T. gondii* is a communicable pathogen that enters its host via ingestion of one of its three forms, the oocysts, the trachyzoite, or tissue cysts (bradyzoites) from contaminated water, soil, or infected meat (Dubey, 2009). *Toxoplasma gondii* can perpetuate itself in all three of the major types of disease reservoirs. These are: Domestic and wild cats; Non-living reservoirs of *T. gondii* include soil and water contaminated with feces; *T. gondii* can be found as bradyzoites in tissue cysts of intermediate hosts (animal reservoirs), but the domesticated cat is currently considered to be the only reservoir in which the sexual stages of *T. gondii* can be carried out. Chickens are also considered one of the most important hosts in the epidemiology of *Toxoplasma gondii* infection because they are an efficient source of infection for cats that excrete the environmentally resistant oocysts. Many other intermediate hosts including sheep, goats, rodents, cattle, swine, chicken and birds may carry an infective stage of *T. gondii* encysted in their tissues (Dubey, 2009).

Congenital toxoplasmosis is a group of symptoms that occur when the fetus is infected with the parasite *Toxoplasma gondii* through the placenta. It is generally considered as a serious health problem in pregnant women, who can pass the infection to the fetus or newborn and cause severe consequences in the infant (e.g., mental retardation, blindness, and epilepsy) and in immuno-compromised people (Singh, 2003; Hokelek and Safdar, 2004; Dubey et al., 2000). The greatest risk of congenital toxoplasmosis occur during the first trimester in fetuses than during the second or third trimester in fetus, but the highest risk of transmission occur during the third trimester. This high chance of transmission had been thought to be relating to the larger size of the uterus (Singh, 2003).

**Tissue cysts in undercooked meat**

Major public health concern is the risk of having cats in the same hold with pregnant women (Dubey, 2004). Approximately one-third of the human population has been exposed *T. gondii* (Singh, 2003). Half billion humans have antibodies to *T. gondii* (Dubey et al., 2000).

However, the sources of infection for humans, worldwide, vary greatly with culture, ethnic, geographical location and eating habits differences (Tenter et al., 2000). Carcass or fresh meat of a wide range of intermediate hosts containing viable parasites in tissue cysts is a possible source of infection for flesh eating animals and humans. Moreover, contaminated water and vegetables can also be important source of *T. gondii*. The product of ovine or caprine conception, when abortion or stillbirth has been caused by toxoplasmosis can be a source of infection. The placenta alone may contain many infectious dose of the parasite (Frenkel, 1990; Radostits et al., 1994).

**Mode of transmission of Toxoplasma infection**

According to the Center for Diseases Control (CDC), there are three primary principal routes by which *T. gondii* is transmitted (Figure 5). These include:

First, Ingestion of oocysts that pass in cat feces: This is the most well known and common modes of transmission to other animals in which tissue cysts develop through exposure to cat litter or soil, water. Oocysts are only shed by cats. Unsporulated oocysts in fresh feces are not infective; they need appropriate oxygen, humidity and temperature to sporulate. Sporulated oocysts are the most environmentally resistant life stage of the parasite. Ingestion of as few as ten oocysts may infect an intermediate host, while ingestion of 100 or more oocysts can cause a patent infection in a cat, which may shed tens to hundreds of millions of oocysts. Second, Food born transmission: this is possible through consumption of *T. gondii* tissue cysts in raw or undercooked meats, unpasteurized milk and consumption of oocysts in foods infected by contaminated fomites (CDC, 2008). Tachyzoites are potentially infective, and may be found in the tissues of acutely infected animals, as well as the milk of sheep, goats, cows, and sometimes chicken eggs. However, tachyzoites are killed relatively easily by pasteurization. Third, Transplacental transmission (congenital toxoplasmosis): This is the transmission of *T. gondii* from a mother to her offspring in uterus through the blood during pregnancy. Acute infections in pregnant women can be
transmitted to the fetus and cause severe illness (e.g., mental retardation, blindness, and epilepsy) (Montoya and Remington, 2000).

Additionally, sexual Transmission of *T. gondii*; A recent study in dogs demonstrated that *T. gondii* can be transmitted sexually in canine species. Male dogs were infected by *T. gondii*; it was then found in their semen. The infected semen was then used to artificially inseminate four uninfected female dogs. Seven days after insemination, all four dogs had antibodies to *T. gondii*. Two of the pregnant dogs had miscarriages; the other two delivered four puppies, none of whom lived longer than three weeks and all of which had cysts containing *T. gondii* in their brains (Arantes et al., 2009).

**Toxoplasmosis in different species of animals**

**Cats**

In domestic cats the global prevalence of infection with *T. gondii* varies from 1% (Lukesova et al., 1997) to 84.4% (Dubey et al., 2004). In wild felids prevalence as high as 88.5% was recorded in bobcats (Riley et al., 2004). The magnitude of prevalence has been shown to be variable based on the method of investigation, the geographical area, feeding habit and age of cats. Usually lower prevalence were observed with coproscopy. The prevalence of oocyst excretion ranges from 0.4% to 41.3% (Tenter et al., 2000) in domestic cats and higher value (52.9%) was reported in wild felines. Antibody titers cannot be used as indicator of oocyst excretion because antibodies are developed after oocyst excretion has ceased. It is used rather as indicator of environmental contamination (Dubey, 2004).

Factors, such as management and hygienic standards in breeding, density of cats and environmental conditions are effective on the acquisition of *T. gondii* oocysts by animals (Tenter et al., 2000). Humidity and temperature favor the oocysts survival. Although cats of any age can die due to toxoplasmosis, kittens and those with depressed immunity are the most likely. However, the greater proportions of cats with healthy immunity infected with *T. gondii* remains asymptomatic (Dubey and Carpenter, 1993).

**Other domestic animals**

*Toxoplasma* infection has been demonstrated in dogs in most areas of the world (Tenter et al., 2000). The prevalence of *T. gondii* infection varies depending on the feeding habit, age, sex, geographical location, and the type of tests employed to detect the infection. It was found to be higher in stray and hunting dogs than in domesticated dogs. The dogs can acquire this parasite...
either from infected soil or ingesting cat feces. The dogs that eat raw or incompletely cooked meat are also at a
great risk of developing this disease. The severity of the
diseases depends upon the number of the parasites ingested. Canine toxoplasmosis might be an important
epidemiological indicator of the risk of toxoplasmosis to
man. Among companion animals, fatal toxoplasmosis
may occur in dogs that are immune-suppressed following
coincident infection with distemper virus (Dubey and
Carpenter, 1989).

Among farm animals small ruminants are more
susceptible to toxoplasmosis and the ones to be affected
commonly. On world basis, the seroprevalence of
infection was found to range from 0% to 50% in domestic poultry in many parts of the world while as high as 71% was observed in wild turkeys (Tenter et al., 2000). In free ranging chicken the prevalence has currently been determined to range from 2% to 100% (Dubey, 2002).

Higher prevalence of toxoplasmosis has also been
recorded in swine. Globally, the prevalence is variable
and range from 0-100% on individual animal basis
depending on the age, husbandry practice, sex and other
risk factors. Viable T.gondii has been demonstrated from
eatable tissues of pigs and commercial pork preparations
indicating its serious public health implications. Outbreaks of toxoplasmosis in pigs have caused higher
mortalities in younger pigs than adult pigs (Tenter et al.,
2000; Da Silva et al., 2005).

Humans

Toxoplasmosis is zoonotic protozoal diseases among
physicians, veterinarians and the public. The parasite is
known to cause congenital diseases and abortion both in
humans and animals (Dubey and Beatie, 1988). It is
endemic worldwide and depending on the geographic
location, 15 to 85% of the human population is
asymptomatically infected. The T. gondii seroprevalence
estimated for human population varies greatly among
different countries, among different geographical areas
within the same country, and among different ethnic
groups living in the same area. In sub-Saharan Africa the
prevalence of T. gondii, increased at the same time as
HIV. Toxoplasmosis prevalence; 75.4% in Nigeria
(Onadeko et al., 1996); 60% from AIDS patients in Coˆte
d’Ivoire, at Yopougou, (Adou-Bryn et al., 2004); 58.4% in
Tunisia (Bouratbine et al., 2001) and 34.1% from
pregnant women in Sudan (ElNahas et al., 2003). A study
undertaken in Burkina Faso from 2004 to 2005 in 336
pregnant women (aged between 18 and 45) using ELISA
for serum antibodies against T. gondii and HIV showed that
the prevalence of T. gondii was 25.3% and the HIV
sero-status (61.6%) seems to be associated with
higher prevalence rates of both T. gondii (Simpore et al.,
2006).

The mechanisms by which HIV induces susceptibility
to opportunistic infections such as toxoplasmosis are
likely multiple. These include depletion of CD4 T cells;
impaired production of IL-2, IL-12, and IFN-gamma; and
impaired cytotoxic T-lymphocyte activity. Cells from
HIV infected patients exhibit decreased in vitro
production of IL-12 and IFN-gamma, and decreased
expression of CD154 in response to T. gondii (Cohen et
al., 1999).

Generally the prevalence of toxoplasmosis in different
animals can be summarized in Table 2 below.

<table>
<thead>
<tr>
<th>Pathogen signicance of Toxoplasmosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>In cats</strong></td>
</tr>
<tr>
<td>Once ingested T. gondii actively invades the intestinal epithelial cells or engulfed by them. Penetration of host cells by the parasites is mediated by parasite motility through actin and myosin and exocytosis of parasite organelles that aid parasite invasion. This invasion process has been found to be supported by the host cells cytoskeletal structures (MacLaren et al., 2004). It replicates intracellularly within the parasitophorous vacuole and progress to infect the lamina propria cells (Montoya and Liesenfeld, 2004). After invasion of a cell, the parasite multiplies and eventually fills and destroys the cells. Located in the lamina propria and the Payer’s patches, dendritic cells (DCs), neutrophils and macrophages become infected by free merozoites crossing the epithelium or by ingestion of apoptotic and infected enterocytes. The tachyzoites are also found to induce depression of non-phagocytic host cell surfaces as they push the plasma membranes to penetrate them (MacLaren et al., 2004). Depending on the strain of T. gondii and the genetic makeup of the host excess production of IFN-γ rather to control extensive multiplication of the merozoites causes acute inflammation of intestine leading to infiltration of villi with inflammatory cells, haemorrhages and loss of epithelial barrier that culminate in death. This can be exacerbated by commensal intestinal bacteria crossing the epithelium (Montoya and Liesenfeld, 2004).</td>
</tr>
</tbody>
</table>
In other animals

Sever death cases can be caused by *T. gondii* in many sheep and goats species. These include embryonic death and resorption, fetal death and mummification, abortion, stillbirth and neonatal death in goats and sheep (Tenter et al., 2000). Although syndrome of fever, dyspnoea, and generalized tremor are seen, the principal manifestations of toxoplasmosis in pregnant ewes are abortion, stillbirth, birth of mummified fetuses, neonatal death, retention of the fetus and birth of full term lambs that show locomotory and suckling disorder (Urguhart et al., 1996; Radostitis et al., 1994; Smith, 1996). Age of the fetus at the time of *T. gondii* infection in the ewe is one of the known causes for this variability in clinical responses. *Toxoplasma gondii* infection acquired before 50 days gestation may result in early embryonic death and resorption, probably because of the ovine fetus is not immunologically mature until 60th to 70th day of gestation. Infection of the ewe between 60 and 100 days of gestation generally results in death and retention of fetus and birth of weak lambs. Infection in the last 30 days of gestation may result in subclinical infection (Dubey et al., 1987).

Among domestic food animals, *Toxoplasma gondii* is pathogenic for goats. Unlike in sheep, *Toxoplasma gondii* can cause encephalitis, nephritis, abomasitis, enteritis and cystitis in adult goats. Abortion, still birth or birth of weak and non-viable kids is the manifestation of clinical toxoplasmosis in pregnant does (Dubey et al., 1990).

### Table 2. Reported prevalence of toxoplasmosis in different species of animals.

<table>
<thead>
<tr>
<th>Species of host</th>
<th>Prevalence</th>
<th>Reference</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>cat</td>
<td>1-84%</td>
<td>Lukesova et al., 1997</td>
<td></td>
</tr>
<tr>
<td></td>
<td>88.5%</td>
<td>Dubey et al., 2004</td>
<td></td>
</tr>
<tr>
<td></td>
<td>22.9%</td>
<td>Bekele and Kasali, 1989</td>
<td>Ethiopia</td>
</tr>
<tr>
<td>sheep</td>
<td>21%</td>
<td>Smith, 1991</td>
<td></td>
</tr>
<tr>
<td></td>
<td>92%</td>
<td>Tenter et al., 2000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>11.6%</td>
<td>Bekele and Kasali, 1989</td>
<td>Ethiopia</td>
</tr>
<tr>
<td>goats</td>
<td>25%</td>
<td>Smith, 1991</td>
<td></td>
</tr>
<tr>
<td></td>
<td>81.6%</td>
<td>Tenter et al., 2000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0% - 50%</td>
<td>Tenter et al., 2000</td>
<td>Domestic Poultry</td>
</tr>
<tr>
<td>poultry</td>
<td>71%</td>
<td>Tenter et al., 2000</td>
<td>Wild Turkey</td>
</tr>
<tr>
<td>swine</td>
<td>0-100%</td>
<td>Da Silva et al., 2005</td>
<td></td>
</tr>
<tr>
<td>Human</td>
<td>15 -85%</td>
<td>Dubey and Beatle, 1988</td>
<td>Worldwide</td>
</tr>
<tr>
<td></td>
<td>80.0</td>
<td>Woldemichael et al., 1998</td>
<td>Ethiopia</td>
</tr>
<tr>
<td></td>
<td>60%</td>
<td>Negash et al., 2008</td>
<td>Ethiopia</td>
</tr>
<tr>
<td></td>
<td>85.5%</td>
<td>Gelaye et al., 2012</td>
<td>Pregnant women</td>
</tr>
</tbody>
</table>

In human

It causes several clinical syndromes including encephalitis, chorio-retinitis, mental retardation and loss of vision in congenitally-infected children. Generally, prenatally acquired toxoplasmosis is more severe than postnatal acquired infection. The severity and likelihood of infection is dependent on the trimester of pregnancy during primary infection with *T. gondii* (Singh, 2003; Hokelek and Safdar, 2004; Dubey et al., 2000).

*T. gondii* can easily be transmitted from acutely infected pregnant women to their offspring. This form of congenital infection can result in abortion, fetal resorption and still birth (if infection occur during the first trimester), or in the birth of severely hand capped child when infection of the mother occurs during second trimester. However, when infections of pregnant women occur during the third trimester of pregnancy, congenital toxoplasmosis most often runs subclinical infections for the first 20-30 years of life. Certain severely diseased children may exhibit a classic tetrad of signs: chorioretnitis, hydrocephalus and intracelebral calcification. Besides, they display mental retardation, loss of hearing, cholangitis and death may occur (Morten and Eskild, 1995; Hokelek and Safdar, 2004; Dubey et al., 2000). If the mother acquires the infection in the first trimester and the infection is not treated, maternal to fetal transmission of parasite is less (17% of the fetuses are infected) and disease in infants is usually sever. If the mother acquires infection in the third trimester and the infection in not treated maternal to fetal transmission of the parasite is commonly greater (65% of fetuses are infected) and involvement is mild or in apparent at birth (Hokelek and Safdar, 2004). These differences in rates of transmission are most likely related to placental blood flow, the virulence and amount of *T.gondii* acquired and the immunologic ability of the mother to restrict parasitemia. The severity of *Toxoplasma* infections are correlated with the immune status of the infected person. Toxoplasmosis in immunocompetent adolescents or
adults is generally mild or unapparent. Mild infections can result in lymphadenopathy, fever, fatigue, and malaise, all of which usually resolve within weeks to months without specific treatment (Hokelek and Safdar, 2004).

The rapid onset of immunity limits rapidly dividing tachyzoites and facilitates their conversion in to the dormant cyst stage (Alexander et al., 2000). Currently with the advent of the PCR technology which can be applied directly on tissues of suspected individuals, it has been evident that severe ocular toxoplasmosis is caused by mixed infections with various T. gondii genotypes. Moreover, severe ocular toxoplasmosis has occurred in immunocompetent individuals infected postnatally (Stanford and Gilbert, 2000).

**Diagnosis, treatment and control of toxoplasmosis**

**Diagnosis**

Definitive diagnosis of toxoplasmosis cannot be achieved through clinico-pathological examination since the clinical manifestations are non-specific to the diseases and often the diseases run asymptomatic or subclinical courses. Therefore, the diagnosis of toxoplasmosis can be aided by integration of various diagnostic techniques (Urquhart et al., 1987).

**Coprology**

Oocysts of T. gondii can be recovered from cat feces by salt floatation method or by sucrose solution, however, it must be noted that the cat only pass oocysts for a period of 10-20 days following initial infection, and re-infection cannot usually accompanied by oocyst shedding. Therefore, the absence of oocyst in the fecal smears may not necessarily imply that the cat is free of toxoplasmosis (Pipano et al., 1992; Wilkinson, 1984; Lappin, 1992).

**Isolation of the organism (Bioassay)**

Toxoplasmosis can be diagnosed by isolation of T. gondii from cultures of body fluids (blood, CSF, bronchoalveolar lavage fluid) or tissue biopsy specimen in the appropriate clinical setting. This is the most convincing diagnostic methods and is obtained by inoculation of suspected materials into toxoplasma free mice by the intraperitoneal or intracerebral route and subsequent demonstration of tachyzoites or bradyzoites in smears of organs or serous cavities (Urquhart et al. 1987; Soulsby, 1982). A highly virulent strain produces acute and generalized fatal infection one-fourteen days after the intra peritoneal route of infection and few days earlier if the intracerebral route has been used. Isolation of toxoplasma organisms from acute infection is complicated; therefore, failure to identify the parasite does not necessarily reflect lack of causality. Unfortunately, isolation studies may not be helpful for a rapid diagnosis of toxoplasmosis since up to six weeks of culture may be required (Urquhart et al., 1996).

**Serology**

Serological examination is used to indicate the presence of infection by detecting toxoplasma specific antibodies or parasitic antigens in body fluid of infected individuals (Dubey et al., 1990). Serologic examination of ewes is also helpful in excluding toxoplasmosis as a cause of ovine abortion. If specific antibodies are not found abortion is not a result of toxoplasmosis, because antibodies would have peaked before abortion (Dubey et al., 1987). Commonly used serological tests include modified agglutination test (MAT), Enzyme linked immunosorbent assay (ELISA), Indirect immunofluorescent antibody test (IFAT), Indirect haemagglutination test (IHAT), Latex agglutination test (LAT), Sabin-feldman dye test (SFDT) and Complement fixation test (CFT) (Dubey et al., 1990).

Diagnosis in pregnant women can be achieved by detection of Toxoplasma specific antibodies to determine infection with Toxoplasma (Singh, 2003). Animal studies indicate that parasitemia is present for only a very limited time following T. gondii acute infection. Therefore, since the parasite is predominantly localized with in the brain and muscles, diagnosis of primary infection during pregnancy has mainly to rely on serological methods to detect Toxoplasma specific antibodies (Derovin and Garin, 1991).

Diagnosis of congenital toxoplasmosis in newborn children presents many difficulties because of the transfer of maternal IgG antibodies to fetus, low sensitivity of serologic tests and lack of availability and cost of T. gondii specific IgA detection kits. Newborn infants suspected of congenital toxoplasmosis should be tested by both an IgG and IgA capture ELISA. Detection of Toxoplasma specific IgA antibodies is more sensitive than IgM detection in congenitally infected babies. Mean antibody survival times are 23 days for IgG, five days for IgM and six days for IgA. Therefore, the detection of anti Toxoplasma IgM and IgA in the infants’ blood from 15days to 3 months after birth is proof of congenital toxoplasmosis (pinon et al., 2001; peleoux et al., 1998).

**PCR test**

In the past decade, the use of the polymerase chain reaction (PCR) has made a significant improvement in both the prenatal diagnosis of congenital toxoplasmosis and the detection of acute disease in the immunocompromised patient. PCR has been successfully used to...
diagnose toxoplasmosis in congenital, ocular and immunocompromised patients. For this purpose PCR with amniotic fluid, placental and brain tissues, whole blood, cerebrospinal fluid, urine, vitreous fluid, aqueous humor, bronchoalveolar lavage fluid, and pleural and peritoneal fluids has proved of value. Diagnosis is particularly useful in immune compromised patients or patients with AIDS in whom antibody synthesis may be delayed and low, or where it cannot be made by finding *T. gondii* in host tissue removed by biopsy or at necropsy (Bastien, 2002).

Immunohistochemical staining can also be used to identify *T. gondii* tissue cysts or tachyzoites in tissues. Electron micrographic examination, computed tomography techniques, and inoculation of biopsy into mice/or cell cultures can help diagnosis (Dubey *et al.*, 2000).

**Treatment**

Most individuals with healthy immune systems will not require treatment to *T. gondii* because the healthy immune can control the disease. The exception would be healthy mothers who acquire *T. gondii* for the first time after becoming pregnant as the fetus is in danger of acquiring the parasite. There is no approved treatment for clinical toxoplasmosis in cats. Sulphonamides, trimethoprim, pyrimethamine, and clindamycin, either alone or in combination, have been used to treat cats with clinical toxoplasmosis, with varying results. Ponazuril, an approved treatment for equine protozoal myelo-encephalitis caused by Sarcocystis neurona in horses, is excellent in treating acute toxoplasmosis in mice and in preventing recrudescence encephalitis in mice, and should be evaluated in domestic cats. The recommended treatment in cases of human cerebral toxoplasmosis is pyrimethamine and sulfadiazine (plus folinic acid) (Elmore *et al.*, 2010).

**Prevention and Control**

**Cats**

As domestic cats play a key role in the epidemiology of toxoplasmosis through shedding of oocysts, control and prevention measures should target them. Domestic cats should be provided with adequately cooked meat and should also be prevented from hunting birds and rodents; however, it is not practical especially in developing countries (Wilkinson, 1984; Urquhart *et al.*, 1987).

Prevention of oocyst shedding by cats is the key to controlling the spread of *T. gondii*. Cats are thought to become infected with *T. gondii* mainly by ingesting tissue cysts from musculature of other animals. Cats shed oocysts for only one - two weeks after primary infection and they usually become immune to re-shedding of oocysts (Dubey and Frenkel, 1972). However, later studies indicated that this immunity is not life-long as expected and cats can re-shed oocysts (Dubey, 1995). The ingestion of live bradizoites is necessary to acquire immunity to oocyst shedding because parenterally administered *T. gondii* (of any stage) do not induce protective immunity to oocyst shedding in cats (Frenkel and Smith, 1982). A new vaccine for cats contains live bradizoites from the mutant strain (T-263) of *T. gondii*. After oral inoculation with T-263 bradyzoites, the coccidian cycle is arrested at the sexual stage because only one gamont develops; thus oocyst are not produced. Chemoprophylaxis with sulfadiazine-pyrimethamine or with monensin is also useful as they prevent the shedding of oocysts (Frenkel and Smith, 1982).

**Sheep and Goats**

The control of toxoplasmosis in sheep and goats can be approached in several ways. The first is to prevent susceptible goats and sheep to the oocysts in cats’ feces during pregnancy. This could be achieved by keeping feed in a closed container and by proper disposal of cats’ feces. The second method, which could enable us to prevent reproductive losses due to toxoplasmosis is through encouraging exposure of ewes and does to infection before breeding to develop protective immunity. Chemoprophylaxis by adding the anti-coccidial drug, Monensin, to the feed is effective in reducing lambs losses (McCulloch and Remington, 1975; Smith and Sherma, 1994; Smith, 1996).

The good immunity obtained as a result of prior infection indicates that vaccination is realistic strategy to prevent clinical toxoplasmosis. Vaccination trial with incomplete strain S48 showed a significance reduction in reproductive wastage. However, fears that a live vaccine could result in the presence of tissue cysts in meat used for human consumption were shown to be unwarranted (Wilkins and O’Connel, 1992).

**Human**

As toxoplasmosis is clinically important in pregnant women and in immune deficient patient, measures for prevention of infection involve; the provision of adequately cooked and frozen meat (cysts are usually killed at 60°C and –20°C), avoidance of contact with cat feces, newborn lambs and kids and fetal membranes and to shun unpasteurized goat's milk. Additionally precautions such as washing of hands prior to eating and after disposing cat feces and gardening, and the wearing of gloves when handling aborted fetus and placenta should be routinely practiced (Urquhart *et al.*, 1987).