MERIT RESEARCH JOURNALS
www.meritresearchjournals.org

Merit Research Journal of Medicine and Medical Sciences (ISSN: 2354-3238) Vol. 2(4) pp. xxx-xxx, April, 2014
Available online http://www.meritresearchjournals.org/mms/index.htm

Original Research Article

Toxoplasmosis: Epidemiology with the emphasis of its public health importance

Teshager Dubie*[1], Getachew Terefe[1], Mebratu Asaye[1] and Tesfaye Sisay[2]

[1]College of Veterinary Medicine and Agriculture, Addis Ababa University, P.O. Box 34, Debre Zeit, Ethiopia.

[2]Institute of Biotechnology, College of Natural and Computational Sciences, Addis Ababa University, P.O.Box 1176, Addis Ababa, Ethiopia

***Corresponding Author's Email: teshager.dubie@yahoo.com**

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, brith 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 Toxoplama 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 immuno-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 immuno-compromised patients. *T. gondii* has been recognized in recent years as a significant cause of morbidity and mortality in children infected in uterus 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, Besnotia, Frenkella, Cryptosporidium, Isospora, Eimeria and Sarcocystis*. Different species were assigned to *Toxoplsama* 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 intermidate hosts. *T. gondii* has three infective stages;Tachyzoites-the rapidly multiplying form of parasite present during the acute stages of infection in the intermidate 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 cuntain tachyzoites or bradyzoites, although direct transmission of oocysts between cats can also occur. 'The ingestion

Table 1. Taxonomic classification of *Toxplasma gondii*

Kingdom	Protista
Sub kingdom	Protozoa
Phylum	Apicomplexa
Class	Conoidasida
Order	Eucoccidiorida
Sub order	Eimeriorina
Family	Sarcocystidae
Sub family	Toxoplasmatinae
Genus	Toxoplasma
Species	*Toxoplasma gondii*

Sources: (Tenter et al., 2000)

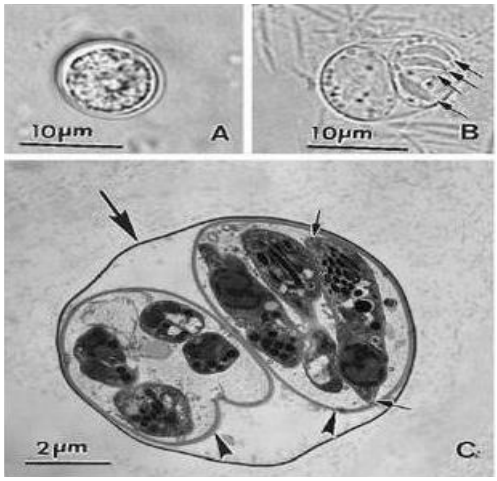


Figure 1. Oocysts of *T. gondii*: A = unsporulated oocyts, B and C = sporulated oocysts (arrows) (Dubey *et al*., 1998).

of mature bradyzoites is the most important route and results in the shedding of higher numhers of oocysts than when infection is acquired from other stages. Following infection, the cyst wall is digested in the cat's stomach, and in the intestinal epithelium the liberated bradyzoites initiate a cycle of schizogonous and gametogonous development culminating in the production of oocysts in 3-10 days. Oocysts are shed for only 1-2 weeks. During this cycle in the intestinal mucosa, the organisms may invade the extra intestinal organs where the development of tachyzoites and bradyzoites proceeds as in intermediate hosts (Urquhart *et al*.1987),

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

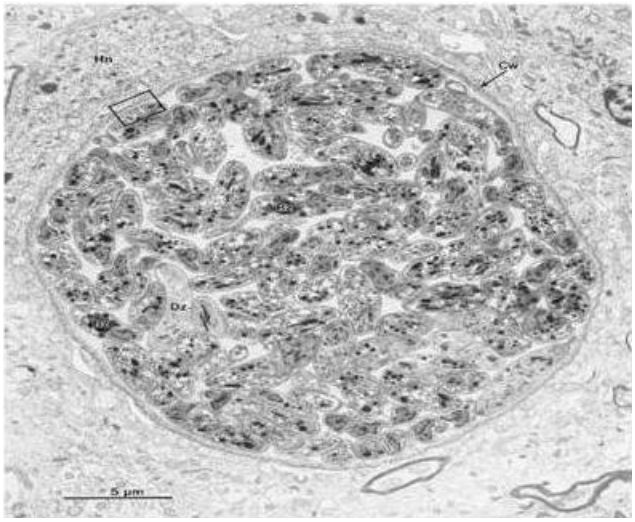


Figure 2. Electronmicograph of tissue cyst of *T. gondii* containing bradyzoites (Dubey *et al*., 2010)

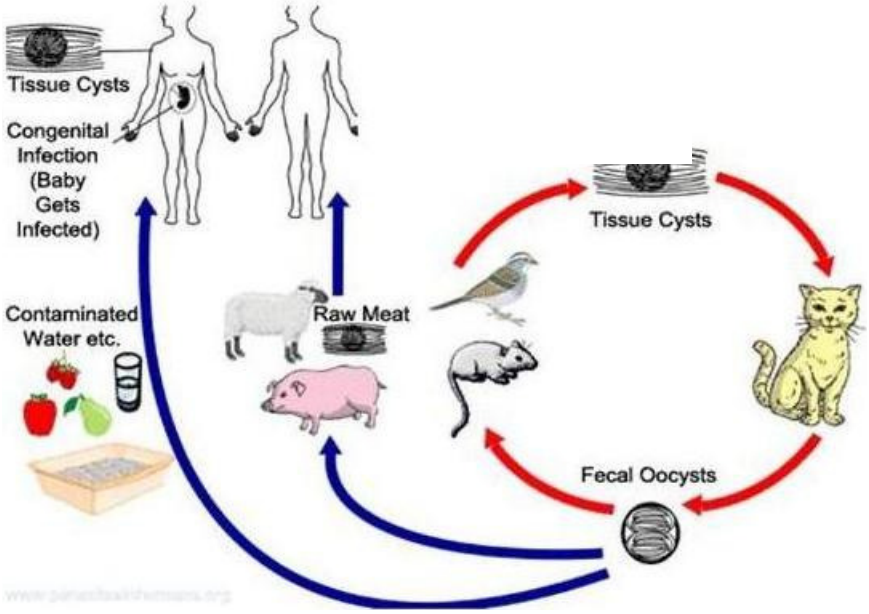
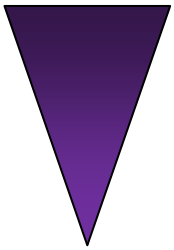


Figure 3. Life cycles of *Toxoplasma gondii (*www.parasitesinhumans.org)

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 intermidate 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 fetus 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 toxoplasmos): 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

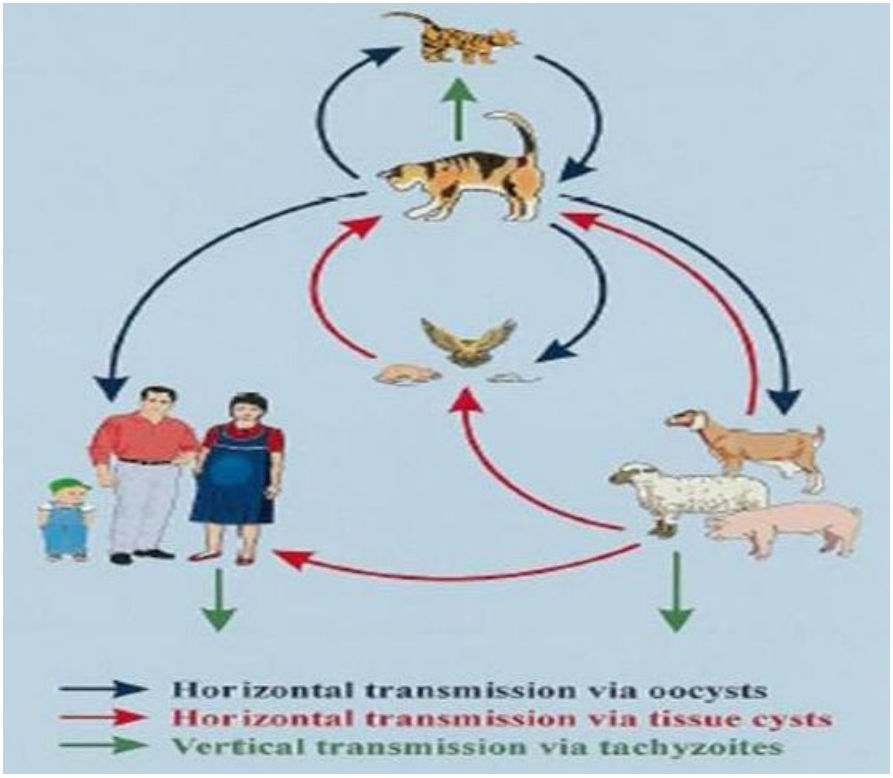


Figure 5. Transmission of *T. gondii* between definitive and intermediate hosts

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 hyiegenic 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 concurrent 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 toxoplasmosis in sheep and goats was considered to be 21% and 25% respectively (Smith, 1991). However, recent reports indicated that higher prevalence rates (92% and 81.6% respectively) have been recorded in sheep and goats kept on pasture with increased pressure of infection due to contamination of the environment with oocysts (Tenter *et al.,* 2000). In area where goat's milk is utilized, unpasteurized milk from acutely diseased goats is also an important source of infection, especially for children. Although tachyzoite in the milk are likely to be destroyed by gastric juice, penetration through sores in the oral mucosa might occur (Frenkel, 1990; Smith and Sherman, 1994).

The prevalence of *T. gondii* 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 edible 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 Yopougon, (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 greater 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.

Pathogenic significance of Toxoplasmosis

In cats

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).

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

Species of host	Prevalence	Reference	Remark
	1-84%	Lukesova *et al*., 1997	
cat	88.5%	Dubey *et al*., 2004	
		Riley *et al*., 2004	Wild felids
	22.9%	Bekele and Kasali, 1989	Ethiopia
sheep	21%	Smith, 1991	
	92%	Tenter *et al.,* 2000	
	11.6%	Bekele and Kasali, 1989	Ethiopia
goats	25%	Smith, 1991	
	81.6%	Tender *et al.,* 2000	
	0% - 50%	Tenter *et al.,* 2000	Domestic Poultry
poultry	71%	Tenter *et al.,* 2000	Wild Turkey
	2% - 100%	Dubey, 2002	Free ranging chicken
swine	0-100%	Da Silva *et al*., 2005	
	15 -85%	Dubey and Beatie, 1988	Worldwide
Human	80.0	Woldemichael *et al*., 1998	Ethiopia
	60%	Negash *et al*., 2008	Ethiopia
	85.5%	Gelaye *et al*., 2012	Pregnant women

In other animals

Sever death cases can be caused by *T. gondii* in many sheep and goats species. These include embryonic death and resorpition, 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 60^{th} to 70^{th} 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).

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, congential 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: chorioretinitis, hydrocephalus and intracelebral calcification. Besides, they display mental retardation, loss of hearing, cholangitis and death may occur (Morten and Eskild, 1995; Hokelk 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 pers-on. 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, sever 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 intraperitonial 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 immune-flourescent antibody test (IFAT), Indirect haemag-glutination 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 relay 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 cupture 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 *Toxoplasmi*c 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 immunocom-promised 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 recrudescent 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 parentrally 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 bradyzoites from the mutant strain (T-263) of *T. gondii*. After oral innoculation 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 (Mc culloch and Remingon, 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℃ and –20℃), 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).

REFERENCES

Adou-Bryn KD, Ouhon J, Nemer J, Yapo CG, Assoumou A (2004). Serological survey of acquired toxoplasmosis in women of childbearing age in Yopougon (Abidjan, Cote d`Ivoire). *Bull. Soc. Pathol. Exot*., 97: 345-348.

Alexander J, Roberts CW, Wolker W, Reichmann G, Hanter CA (2000). The immunology of *Toxoplasma gondii* infection in the immune-competent host. Pp 69 – 70.

Arantes TP, Lopes WD, Ferreira RM (2009). *Toxoplasma gondii* evidence for the tran-smission by semen in dogs. *Exp. Parasitol.,* 123:190–194).

Bastien P (2002). Molecular diagnosis of toxoplasmosis. *Trans. R. Soc. Trop. Med. Hyg*., 96: 205-15.

Bekele T, Kasali OB (1989). Toxoplasmosis in sheep, goats and cattle in central Ethiopia. *Vet. Res. Comm*., 13: 371-375.

Boothroyd JC (2009). *Toxoplasma gondii*: 25 years and 25 major advances for the field. *I. J. Parasitol.,* 39: 935–46. Doi:10.1016/ j.ijpara.2009.02.003. PMC 2895946. PMID 19630140.

Bouratbine A, Siala E, Chahed MK, Aoun K, Ben-Ismail R (2001). Seroepidemiologic profile of toxoplasmosis in Northern Tunisia. *Vet. Parasitol*., 8: 61-66.

Cohen O, Weissman D, Fauci AS (1999). The immunopathogenesis of HIV infection. In: Fundamental Immunology, Paul, W.E. (ed). Philadelphia,Lippincott-Raven, Pp 1455-1509.

Da Silva AV, Pezelico SB, De Lima VY, D'arc ML, Pin Hiro JP, Tanaka EM, Rebeiro MJ, Lanjoni H (2005). Genotyping *T.gondii* strains isolated dogs with neuro-logical signs. *Vet. Parasitol*., 127:23-27.

Derouin F, Garin YJ (1991). *Toxoplasma gondii*: blood and tissue kinetics during acute and chronic infections in mice. *Exp. Parasitol*., 73:460-468.

Dubey JP (1994). Toxoplasmosis: Zonosis up date, *J. Am. Vet. Med. Assoc*., 205: 1594-1595.

Dubey JP (2002b). A review of toxoplasmosis in wild birds. *Vet. Parasitol.,* 106:121-153.

Dubey JP (2009). Toxoplasmosis of animals and humans. CRC Press, Boca Raton, Florida. In press.

Dubey JP (2010). Toxoplamosis of humans and animals. 2nd edition, CRC Press, Beltsville, Maryland, U.S.A, pp 102-142.

Dubey JP (2004). Toxoplasmosis; water born zoonosis. *J. Vet. Parasitol*, 126: 57-72.

Dubey JP, Beattie CP (1988). Toxoplasmosis of animals and man, 1st ed. Re. press, Bocaroton, FL. Pp 341-347.

Dubey JP, Carpenter JL (1983). Histologically confirmed clinical toxoplasmosis in cats. *J. Am. Vet. Med. Assoc.,* 203: 1556-1566.

Dubey JP, Emond JP, Desmonts G, Anderson WR (1987). Serodiagnosis of postnatally and prenatally induced toxoplasmosis in sheep. *Am. J. Vet. Res*., 48: 1239-294.

Dubey JP, Emond JP, Desmonts G, Anderson WR (1987). erodiagnosis of postnatally and prenatally induced toxoplasmosis in sheep. *Am. J. Vet. Res*., 48: 1239-294.

Dubey JP, Frenkel JK (1972). Cyst-induced toxoplasmosis in cats. *J. Protozool*, 19:155-177.

Dubey JP, Morales ES, Lehmann T (2004b). Isolation and genotyping of *Toxoplasma gondii* from free-ranging chickens from Mexico. *J. Parasitol*., 90:411–413.

Dubey JP, Salant H, Sreekumar C, Dahl E, Vianna MC, Shen BSK, Kwok OCH, Spira D, Hamburger J, Lehmann T (2004c). High prevalence of *Toxoplasma gondii* in a commercial flock of chickens in Israel, and public health implications of free-range farming. *Vet. Parasitol*., 121:317–322.

Dubey JP, Sonn RJ, Hedstron O, Snhyder JP, Lassen EO (1990). Serologic and histologic diagnosis of toxoplasmic abortion in sheep in Oregon. *J. V. M. A*, 196: 291-294.

Dubey JP, (1995). Duration of immunity to shedding of *Toxoplasma gondii* oocysts by cats. *J. Parasitol.,* 81:410-415.

Dubey JP, Carpenter JL, Topper MJ, Uggla A (1989). Fatal Toxo plasmosis in dog *J.Am. Anim.Hosp. Assoc*., 25:659-664.

Dubey JP, Saville WJA, Stanek JP, Reed SM (2002). Prev-alence of *Toxoplasma gondii* antibodies in domestic cats from rural Ohio. *J. Parasitol*., 88: 802-803.

Dubey JP, Scandrett WB, Kwok OCH, Gajadhar AA (2000). Prevalence antibodies to *Toxoplasma gondii* in ostriches (Strthio camelus). *J. Parasitl.,* 86: 623-624.

Dubey JP, Sonn RJ, Hedstron O, Snhyder JP, Lassen ED (1990). Serologic and Histologic Diagnosis of toxoplasmic abortion in sheep in Oregon. *J. V. M. A,* 196:291-294.

Dubey JP, Lindsay DS, Speer CA (1998). Structures of *Toxoplasma gondii* tachyzoite, bradyzoites, and sporo zoites and biology and development of tissue cysts. *Clin. Microbiol. Rev.,* 11: 267-299.

Elmore AS, Jones LJ, Conrad AP, Patton S, Lindsay SD, Dubey JP (2010). *Toxoplasma gondii*: epidemiology, feline clinical aspects, and prevention. *Trends in Parasitol*, doi: 10.1016/j.pt.2010.01.009.

Elnahas A, Gerais AS, Elbashir MI, Eldien ES, Adam I (2003). Toxoplasmosis in pregnant Sudanese women. *Saudi Med. J.,* 24: 868-870.

Frenkel JK, Smith DD (1982). Inhibitory effects of Monensin on shedding toxoplasma oocysts by cats. *J. Parasitol*., 68: 851-855.

Frenkel. JK (1990). Toxoplasmosis in human beings. *J. A. V. M. A*., 196: 233-239.

Gelaye W, Kebede T, Hailu A (2012). Seroprevlence and risk factor of *Toxoplasma gondii* infection among pregnant women attending antenatal care at Tikur Anbesa General Specialized and Gandhi Memorial Hospitals, Addis Ababa, Ethiopia. 13th world congress on public health 23-27 April, 2012, Addis Ababa Ethiopia.

Hokelek M, Safdar A (2004). Toxoplasmosis. *Adv.Vet. Sci*., 8:335-341.

Lappin MR (1992). Protozoal disease. In: Hand book of small animal practice. 2nd ed. Morgan, R.V. (ed). Churchill Living Stone, UK.P. Pp 1236-1239.

Lukesova D, Literak I (1998). Shedding of *Toxoplasma gondii* oocysts by felidae zoos in Czech Republic. *Vet. Parasitol.* 4: 1-7.

MacLaren A, Attias M, de Souza W (2004). Aspects of the early moments of interaction between tachyzoites of *Toxoplasma gondii* with neutrophils. *Vet. Parasitol., 10*: 301-312.

Mc. Culloch WF, Remington JS (1995). Disease transmitted from animals to Man, 6th ed. Hubbert C Thomas Publisher, U.S.A. Pp 805-817.

Meireles RA, Galisteo Jr. AJ, Andrade Jr. HFD (2003). Serological survey of antibodies to *Toxoplasma gondii* in food animals from Sao paul state, Brazil. Braz.*J.vet.Ref.Animal.soci*., 40: 267-271.

Montoya JG, Liesenfeld O (2004). Toxoplasmosis. *Lanc*., 363:1965-1976.

Montoya JG, Remington JS (2000). *Toxoplasma gondii*. In: Principles and Practice of Infectious Diseases. Mandell, G.L, Bennett, J.E. and Dolin, R, (eds). Philadelphia, Churchill Living stone, Pp 2858-2888.

Morten L, Eskild P (1995). Detection by enzyme immunosorbent assay of *Toxoplasma gondii* igG anti bodies in dried blood spots on PKU-filter paper from new borns. *Scand J. Infect. Dis*., 27: 259-263.

Negash T, Tilahun G, Medhin G (2008). Seroprevalence of *Toxoplasma gondii* in Nazaret town, Ethiopia. *Eas. Afr. J. Pub. Hlth*., 5: 211-214

Onadeko MO, Joynson DH, Payne RA, Francis J (1996). The prevalence of *Toxoplasma* antibodies in pregnant Nigerian women and the occurrence of stillbirth and congenital malformation. *Afr. J. Med. Sci*., 25: 331-334.

Peleoux H, Burn E, Vrnet J (1998). Determination of anti *Toxoplasma gondii* immune globulin G avidity. *Microbiol. Infect. Dis*., 32: 69-72.

Pinon JM, Dumon H, Chemla C (2001). Strategy for the diagnosis of congenital toxoplasmosis. Evaluation of methods comparing mothers and new borns and stadered methods for post-natal detection of immunoglobulins G, M and A antibodies. *J. Clin. Microbiol.,* 39: 2267-2271.

Pipano E, Shkap V, Marcus S, Rapaport E (1992). Toxoplasmosis. *Isr. J. Vet. Med*., 47:1-13.

Radostitis, O.M., Blood, D.C and Gray, C.C.(2006): A text book of the disease of cattle, sheep, pigs and horses, 8th ed. W.B. saunders, London. Pp 1201-1206.

Radotits OM, Blood DC, Gay CC (1994). Veterinary Medicine. A text book of disease of cattle, sheep, pigs, goats and horses, 8th ed. W.B. Saunder, London. Pp 1201-1206.

Rilley SPD, Ofley J, Chomel B (2004). Exposure to feline and canine pathogens in bob cats and gray foxes in urban and rural zones of a nation park in California. *J. Wil. lif. Dis*., 40:11-22.

Sabin AB, Olitsky PK (1937). "*Toxoplasma* and Obligate Intracellular Parasitism". *Sci.,* 85: 336–338.

Simpore, J., Savadogo, A., Ilboudo, D., Nadambega, M.C. and Esposito, M. (2006): *Toxoplasma gondii*, HCV and HBV seroprevalence and co-infection among HIV-positive and -negative pregnant women in Burkina Faso. *J. Med. Virol*., 78: 730-733.

Singh S (2003). Mother to child transmission and diagnosis of *Toxoplasma gondii* infection during pregnancy. *Ind. J. Med. Microbiol*., 21: 69-76.

Smith BP (1996). Large Animal internal Medicine Mosby – year book ine., Pp 1564-1576.

Smith JL (1991). Food bore toxoplasmosis. *J. Food Saf.,* 12: 17-57.

Smith MC, Sherman DM (1994). Goat Medicine. Williams and Wilkins, London. Pp 425-426.

Soulsby EJL (1982). Helmiths, arthropods and protozoa of domesticated animals, 7th ed. Lea and Febiger, Philadelohia. Pp 670-680.

Stanford M, Gilbert R (2000). Epidemiology of ocular *toxoplasma*. Pp 251-259.

Tenter AM, Hecleeroth AR, Weiss LM (2000). *Toxoplasma gondii*: from animals to humans. *Int. J. parasitol*., 30: 1217-1258.

Ukthana Y (2006). Toxoplasmosis; beyond animals to humans. *Trends Parasitol.,* 22 :137-139.

Urquhart GM, Armour J, Duncan JL, Dunn AM, Tennings FW (1987). Veterinary parasitology, 2nd ed. Blak well Science Ltd.,UK. Pp 217-231.

Urquhrt GM, Armous J, Dunn AM, Jennits FW (1996). Veterinary Parasitology. 2nd ed, The Faculty of Veterinary Medicine, The University of Glasgaro, Scotland. Pp 238-241.

Wilkinson GT (1984). Disease of the cat and their management. 2nd ed. Blacks well, London. Pp 475-479.

Woldemichael T, Arnaud L, Fontanetz AL, Sahlu T, Henk GH (1998). Evaluation of the Eiken latex agglutination test for anti-toxoplasma antibodies and seroprevalence of toxoplasma infection among factory workers in Addis Ababa, Ethiopia. *Trans. Roy. Soci. Trop. Med. Hyg*., 92: 401-403.