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Public Health and Economic Significance of Toxoplasmosis

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Abstract
Toxoplasmosis is a zoonotic disease widely distributed throughout the world and is caused by the intracellular parasite *Toxoplasma gondii.* It is definitive host is *felidae family*, but has a wide range of intermediate hosts. There are three infective stages of *Toxoplasma gondii*: Tachyzoites, Bradyzoites and Oocysts. Major routes of transmission in human and animal populations are consuming an infected tissue source (such as raw or undercooked meat), ingestion of oocyst-contaminated food or water and infection of a fetus during pregnancy. After ingestion of tissue cysts, the released bradyzoites from tissue cyst invade the intestinal epithelium transforms into tachyzoite. After invasion of a cell, parasite has ability to actively penetrate hosts cells results in formation of a parasitophorous vacuole where parasite protects itself from host immune cell and replicates intracellularly, form tissue cyst. All extracellular forms of the parasite are directly affected by antibody normal immune response. It can result in a number of syndromes ranging from chorioretinitis to hydrocephalus and neurological deficits. In immunocompromised patient several health problems can be occur: like abortion, mental retardation, seizures, blindness, hydrocephalus, cerebral calcification, chorioretinitis, and ultimately death. The economic significance of *Toxoplasmosis* is mainly due to reproductive failure in animals, condemnation of meat and wastage of milk, treatment cost in humans and vaccination cost in cats. Diagnosis of toxoplasmosis is performed by isolation of the parasite from patients and more commonly by serological tests. Currently, sulfonamide drugs and pyrimethamine used in combination are the gold-standard medicines for treating toxoplasmosis. The best method of control and prevention are Cleaning and drying of the environment, the use of clean utensils for feed and water, bathing to remove adhering faeces or cysts and proper disposal of faeces cat are pre-requisites to avoid animal to- animal transmission. Pregnant woman should avoid cleaning litter boxes; avoid contact with contaminated garden soil, sandboxes, and eating raw meat whenever possible.
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1. Introduction

Toxoplasmosis is a zoonotic disease widely distributed throughout the world and is caused by the intracellular parasite *Toxoplasma gondii* (Dubey, 2007). *T.gondii*has been found in almost every country of the world in many species of carnivores, insectivores, rodents, pigs, herbivores, primates and other mammals as well as in birds (De Sousa, 2009). It was discovered in 1908 (Nicolle and Manceaux, 1908; Splendore, 1908) in the tissues of a common gundi (Ctenodactylus gundi), a rodent used for Leishmania research at the Pasteur Institute, Tunis, and in a rabbit in Brazil. The name Toxoplasma (toxon = arc, plasma = form) is derived from the crescent shape of the tachyzoite stage (Hill and Dubey , 2016). The definitive host of the parasite is the domestic cat and the entire cat family, in which the parasite reproduces sexually. *T. gondii*can also infect a wide range of intermediate hosts, affecting most warm-blooded animals (Muñiz – Hernandez and Mondragon -Flores, 2009).

Major routes of transmission in human and animal populations are consuming raw or undercooked meat containing *T. gondii*tissue cysts (Dema*et al.,* 2007), drink contaminated water and milk, eating unwashed raw vegetables or fruits, or anything contaminated with oocysts shed in the feces of an infected animal (Tenter *et al*.,2000).

*T. gondii*normally causes a subclinical infection in most animal species; however, a primary infection during pregnancy can cause fetal pathology, as well as abortion in humans and some animal species (Innes *et al.,* 2009; Carello*set al.,* 2013). Toxoplasmosis ranks high on the list of diseases that lead to the death of patients with AIDS (Dubey, 2010). However, following the widespread usage of highly active anti-retroviral therapy (HAART) opportunistic infections including *T. gondii*in AIDS patients have been declining significantly (Huruy*et al.,* 2010). When the human fetusis infected in pregnant women, it may present with hydrocephalus, chorioretinitis, deafness, impaired mental development (Jones *et al*., 2001) and intracerebral calcifications in live-born children (Weiss and Dubey, 2009).

The real economic impact of toxoplasmosis is difficult to estimate, because in most immunocompetent individuals, the infection goes unnoticed or has claimed clinical presentation to other diseases (Restrepo, 2007; Dubey, 2010). Toxoplasmosis can also cause significant economic livestock losses, particularly to the sheep industry result in early embryonic death and resorption, foetal death and mummification, abortion, stillbirth, and neonatal death (Dubey, 2009).

Diagnosis of toxoplasmosis is performed by isolation of the parasite from patients and more commonly by

serological tests (Gamble *et al*., 2005; Dubey, 2010). Currently, sulphonamide drugs and pyrimethamine used in combination are the gold-standard medicines for treating toxoplasmosis (McLeod *et al*., 2014).

The objective of this paper is to review on the issues and fundamental information about public healh importance of *Toxoplasmosis* and its economic significance.

2. Literature Review

2.1. Etiology and Risk Factors

2.1.1. Aetiology

The causative agent of toxoplasmosis is *T. gondii*. It is a specific parasite of definitive host *felidae family*, but has a wide range of intermediate hosts. There are three infective stage of *T. gondii*:

Tachyzoites (in-group)-the rapidly multiplying form of parasite present during the acute stages of infection in the intermediate host. They are able to invade and multiply rapidly in virtually all vertebrate cell types including brain, heart, lung, eye, muscles, placenta etc (Dubey *et al*., 1998).

Bradyzoites (in tissue cyst)- result from the conversion of tachyzoites into a slow-dividing stage that forms tissue cysts. Bradyzoites have a latent metabolism, well adapted to longterm survival. The resistance of bradyzoites to digestion (1 to 2 hour survival in acid pepsin) allows their transmission through ingestion (Dubey *et al*., 1998 ; Weiss and Kim, 2011).

Oocysts- which containing sporozoites present in the cat faeces. The oocyst wall is an extremely robust multilayer structure protecting the parasite from mechanical and chemical damage. It enables the parasite to survive for long periods, more than a year, in a moist environment. Oocysts are excreted in faeces by felids. They mature and become infectious after sporulation which occurs a few days after excretion (Hill and Dubey , 2016).

It has also, three strainshave been defined, (type I, type II and type III), of which type I is extremely virulent for mice; Type I virulent strains are found rarely in animals and in humans ; in many literature they have been associated with acquired ocular toxoplasmosis, congenital infection and cerebral toxoplasmosis in immunocompromised patients (Khan *et al*. 2005; Hunter and Sibley 2012).while type II strains are the most prevalent cause of human toxoplasmosis both in congenital infection and in AIDS patients in North America and Europe (Boothroyd and Grigg, 2002; Klaren and Kijlstra, 2002; McLeod *et al.,* 2012). Type III are considered completely avirulent in mice and are characterized by low tissue cysts burdens and limited ability to cause infection and human disease (Sibley and Ajioka, 2008; Hunter and Sibley, 2012). It is also present in animals and has been detected in AIDS patients, but does not seem to be associated with ocular Toxoplasmosis (Boothroyd and Grigg, 2002).

2.1.2. Major Risk Factors

The variation in climate has a marked influence on the habitat of *T. gondii*; for example, an elevation in ambient temperature and precipitation can modify the soil humidity, so that the sporulated oocysts persist for a long time viable in the moist environment cause risk factors for both humans and livestock contamination (Meerburg and Kijlstra, 2009). The main risk factor for humans is the consumption of raw or undercooked meat from animals that have been previously infected with the parasite (Cook *et al*., 2000) and ingestion of fruit, vegetables, soil or water contaminated with sporulated oocysts shed into the environment by cats during a few weeks following infection (Tenter, 2000). Cat fecal matter is particularly dangerous: Just one cyst consumed by a cat can result in thousands of oocysts. These oocysts are resilient to harsh environmental conditions and can survive over a year in contaminated soil. This is why physicians recommend pregnant or ill persons do not clean the cat's litter box at home (Robert *et al.,* 2012; Mai *et al.,* 2009). During the production of various meat products, meat of many animals is mixed, which also amplifies the risk in cases where only a few animals would be infected (Aspinall*et al*., 2002).

Surface water is increasingly being investigated as a risk factor and has been demonstrated to be an important source of infection in tropical and subtropical countries, where it may be contaminate of drinking water with oocysts used for human consumption without any purification (Petersen *et al*., 2010).

Risk factors can also be associated to host factors (age, sex), and farm management (Pinheirob*et al.,* 2009). Small flock size and access of cats to drinking water are potential risk factors for *T. gondii*infection in sheep. Prevalence in small ruminants is generally very high due to the continuous contamination of pastures by *T. gondii*oocysts (Cenci-Goga*et al.,* 2013). Free-living animals such as stray cats or domestic cat 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*etal*., 2003).

Persons at high risk are pregnant women, cat owners, veterinarians, abattoir workers, children, cooks, butchers and immunocompromised such as HIV-infected patients and foetuses with toxoplasmosis transmitted from their mothers via placenta (Sonar and Brahmbhatt, 2010).

2.2. Life Cycle and Mode of Transmission of Toxoplasmosis

*Toxoplasma gondii*is transmitted in diverse ways and its life cycle is often described as 'complex life cycle', thatin definitive host (extraintestinal cycle can occur simultaneously with enteroepithelial multiplies sexually) and in intermediate hosts (extraintestinal cycle multiplies asexually in tissues cells) (Jokelainen, 2013).When intermediate host ingests oocysts or tissue cysts with food or water, sporozoites or bradyzoitesare released into the gut lumen and pass through the gut epithelium to enter cells in the lamina propria. In cells it multiplies intracellularly by a form of fission (endodyogeny) to produce 8 – 16 parasites. It differentiate into the rapidly growing tachyzoite form and disseminate throughout the body. Both sporozoites and bradyzoites transform into tachyzoites that enter a host cell where they divide rapidly until the cell bursts (Opsteegh, 2011; Weiss and Kim, 2011). The tachyzoites can rapidly divided until death of the host cell, they will invade adjacent cells or traveling through the blood stream to attach cells elsewhere in the body. Then tachyzoite stage can transform into a slowly dividing bradyzoite (Klaren and Kijlstra, 2002) because as immunity develops, replication of tachyzoites decreases and tissue cysts develop which do not normally produce a host reaction (Saavedra, 2003)

Cats and wild felids are the only definitive hosts whereby the sexual and asexual part of the life cycle takes place in these animals (Bayarri*et al.,* 2012). They acquire the infection in one of two main ways: by consumption of cysts present in the organs / tissues of a chronically infected intermediate host prey (such as mice) or by ingestion of oocysts within their food or water (Conrad, 2010).

After ingestion of tissue cysts the cyst wall is digested by gastric acid, bile and lytic enzymes of the upper digestive tract, which results in releasing bradyzoites and the released bradyzoites, invade the intestinal epithelium (Dabritz and Conrad, 2010).The released organisms penetrate the enterocytes of cats and transform intracellularly into multinucleated schizonts after 3 to 7 days post-infection. The subsequent schizogony of sexual development (replication) occurs within the epithelial cells of the small intestine of felids, where schizonts mature and develop into microgametes (male) and macrogametes (female) after several generations (Dubey and Frenkel, 1972 ; Speer and Dubey, 2005). A diploid zygote that will develop into an oocyst forms after a female gamete is fertilized by a male gamete (Ferguson *et al*., 1975). The shed of millions of oocysts are finally voided in the faeces (Torgerson and Mastroiacovo, 2013) into the environment during a period of a few weeks (Dubey, 2001). Unsporulated oocysts are expelled into the environment with the feces in high numbers and become sporulated and infectious; under favourable conditions, an oocyst yields two sporocysts containing four haploid sporozoites (Dubey *et al*., 1970).

The routes of *T. gondii* transmission to humans have been clear since the discovery of sexual development in cat intestines in the late 1960s and early 1970s (Ferguson, 2009). Infection in humans and other warm-blooded animals can occur: by consuming raw or undercooked meat containing *T. gondii* tissue cysts (Cook *et al*., 2000; Tenter*et al*., 2000; Sakikawa*et al*., 2012) and drinking unpasteurized infected animal milk (Jone*et al.,* 2009); by eating unwashed raw vegetables or fruits, or anything contaminated with oocysts shed in the feces of an infected animal.Transmission from mother to fetustransplacentally occur, particularly when *T. gondii*is contracted during Pregnancy (Tenter*et al.,* 2000) and Transfusion or organ transplantation from an infected person can also transmit the organism (Dubey and Jones, 2008).

2.3. Public Health Significance of Toxoplasmosis

Toxoplasmosis is one of the more common parasitic zoonosis world-wide. Disease in humans caused by *T. gondii*was first recognised in the late 1930. In 1939, first proved that Toxoplasma isolates from humans and those previously obtained from animals belonged to the same species (Sabin, 1939). There are various Form of Toxoplasmosis**:**

Congenital toxoplasmosis(OT), It is an important vertical transmit infection to the foetus of a mother exposed to *Toxoplasma* for the first time during pregnancy. It can result in a number of syndromes ranging from chorioretinitis to hydrocephalus and neurological deficits (Jones *et al.*, 2001). In immunocompetent women that contract *T. gondii*during pregnancy can vertical transmit the infection to the foetus. The most likely mechanism of the vertical transmission is that tachyzoites may invade placenta during temporary parasitemia of the mother, replicate within cells and some of these may cross the placenta and enter the foetal circulation or foetal tissues (Remington and Desmonts, 1990). While the risk of intrauterine infection of the foetus increases during pregnancy, the effects on the foetus are stillbirth, abortion, and serious mental and physical developmental problems (Thiebaut*et al*., 2007).

Ocular toxoplasmosis (OT), Itcause retinochoroiditis, which may be the result of the immune system reaction to parasite antigens. Chorioretinitis is the most common clinical manifestation of ocular toxoplasmosis: the classic signs of infection are retinal scars and white-appearing lesions often associated with vitritis (Pfaff *et al*., 2013). Ocular toxoplasmosis is reported to be more common in South and Central America, the Caribbean and parts of tropical Africa compared with Europe and Northern America. Ocular disease in South America is more severe than in other continents with lesions that are larger, more numerous, more recurrent and more likely to damage vision due to the presence of extremely virulent atypical genotypes of the parasite. In South America

leading cause of blindness in the population, both in children and in adults (Ajzenberg, 2011; Petersen *et al.,* 2012).

In immunocompromised women with systemic lupus erythematous (SLE) or acquired immunodeficiency syndrome (AIDS) where previously infected, seropositive individuals have transmitted *T. gondii* congenitally to their foetus (Wechsler *et al*., 1986).

In immunocompetent humans a primary infection is followed by a lifelong immunity causing the parasite to remain in its encysted stage during lifetime, preventing transfer of infection to the fetus in women who have encountered the infection prior to pregnancy (Kijlstra*et al*., 2004). Most cases of *T. gondii* infections in immunocompetent humans are asymptomatic. Severe manifestations, such as encephalitis, sepsis syndrome/shock, myocarditis, or hepatitis may occur, but are very rare in immunocompetent humans (Ho-Yen, 1992).

The majority of *Toxoplasma* infection in immunocompromised hosts can be life threatening and happens almost always as reactivations of previous infections, such as individuals receiving corticosteroids or cytoxic drugs, patients with haematological malignancies, transplants or AIDS, or reactivation of tissue cysts in transplanted organs and in bone marrow transplants; primary infection is thought to be a rare event (Mele*et al.,* 2002).

2.4. Economic Impacts of Toxoplasmosis

*Toxoplasma gondii*is one of the most widely prevalent cyst forming Apicomplexan parasites and have been recorded worldwide. The parasite has significant impact on animal production particularly in sheep, goats and pigs (Sudan *et al.*, 2013). The real economic impact of toxoplasmosis is difficult to estimate, because in most immunocompetent individuals, the infection goes unnoticed or has claimed clinical presentation to other diseases (Restrepo, 2007; Dubey, 2010). However, it is estimated that the economic impact should be very high due to the loss of one or more days of work in mild cases, treatment and care needs, sick children, especially those with mental retardation and blindness, loss of quality lifestyle and the costs of hospitalization in severe cases and the cost of monitoring pregnant women and treatment during pregnancy who has *T. gondii*positive. This encompassed direct costs (screening, cost of illness, maternal and pediatric treatment), indirect costs (changed job situation of parents, human-capital of dead individuals, blindness and special schools) (Hill and Dubey, 2002; Kijlstra and Jongert, 2008; Jones *et al*., 2014).

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. It cause of economic losses due to abortion of *T. gondii*in the sheep and goat industry because of the high rates of infection (Teshager*et al*., 2014). Currently, the economic losses caused by *T. gondii*infection in sheep are difficult to estimate because the disease occurs sporadically . Moreover, only a small number of the lambs aborted are subjected to diagnosis. In addition, the material sent for diagnosis, besides being potentially inadequate, might also be examined erroneously and finally, serological testing lacks specificity (Dubey, 2009).

The cost of toxoplasma abortion in the UK flock was estimated to be £12 million. These estimates included loss of production, cost of treatment, control and monitoring but did not include the costs associated with human health (Bennett and Ijpelaar, 2003).

2.5. Diagnostic Approach of Toxoplasmosis in Animal and Human Populations

2.5.1. Clinical Signs

Toxoplasmosis in humans: The clinical spectrum of *T. gondii*infection varies from an asymptomatic state to severe illness. The parasite can affect the host's lymph nodes, eyes, central nervous system, liver, and heart (Alvarado-Esquivel *et al*., 2011).In most cases, the infected humans is usually asymptomatic or develops mild symptoms that go unnoticed in 80–90% of cases; however, when infection occurs in pregnant women and immunocompromised patient several health problems can occur. The transmission of *T. gondii*to the fetus might result in abortion, mental retardation, seizures, blindness, hydrocephalus, cerebral calcification, chorioretinitis, pneumonia, swelling of the brain, heart damage, swelling of the liver and spleen and ultimately death (Flatt and Shetty, 2013). The clinical spectrum of the disease varies widely and depends primarily on the immune status of the host and *Toxoplasma* lineage to which a person was exposed. In humans, toxoplasmosis may manifest basically in five ways: asymptomatic, acute infection, congenital, ocular and the immunocompromised patient (Ribera-Pascuet, 2013).

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*(Dubey*et al.,* 2000; Singh, 2003; Hokelek and Safdar, 2004).

Toxoplasmosis in food animals: Natural infection in non-pregnant animals usually elapses without symptoms, but primary infection during pregnancy can cause embryonic death, abortion, birth weak or clinically normal but infected animals. Globally, *T. gondii*is the cause abortions occur in sheep and goats (Buxton *et al.,*

2007).

Pig: The clinical signs reported from domestic pigs during *T. gondii*outbreaks include high fever, anorexia, dyspnea, vomiting, weakness, recumbency, abortions, and death (Kim *et al*., 2009) , with a 50% mortality rate, still-births, premature births and deaths soon after birth (Dubey and Jones, 2008).

Cattle:Although cattle are considered a poor host for *T. gondii* can be successfully infected with *T. gondii* oocysts but due to innate resistance the parasite is eliminated or reduced to undetectable levels within a few weeks (Bayarri*et al*., 2012). There is no confirmed report of clinical toxoplasmosis in cattle but there is an assumption as it causes abortion in cattle (Cenci-Goga*et al*., 2011). Calves are more susceptible than adults cattle. clinical signs of orally affected calves include diarrhea, anorexia, poor weight gain, depression, weakness, dyspnea and fever (Nematollahi and Moghddam, 2008).

Poultry: Toxoplasmic chickens show clinical signs like encephalitis, chorioretinitis, peripheral neuritis, torticollis an inability to stand and lateral recumbancy (Dubey, 2010).

Sheep and goat:*Toxoplasma gondii* causes abortion and neonatal mortality in sheep and goat, congenitally-infected lambs that survive the first week after birth usually grow asymptomatic and can be a source of infection for humans while adult goats can develop clinical toxoplasmosis involving liver, kidneys and brain (Mukarim, 2014). In sheep and goats a primary infection established during pregnancy may result in early embryonic death and resorption, mummification, weak lambs or kids, stillbirths, neonatal death, infertility according to the stage of pregnancy at which infection was initiated. In a typical case of abortion, a ewe or doe infected in mid-gestation produces a stillborn lamb/kid a few days earlier than the predicted end of pregnancy. Either a weak sibling or a 'mummified' fetus (Buxton, 2000) often accompanies the aborted fetus.

Camel:Acute toxoplasmosis is observed in camel with dysponea, many tachyzoites can be found in lungs and dysponea,plueral exudates and T.*gondii* can be isolated from camel meat using cat biopsy (Sadrebazzaz*et al*., 2006).

Toxoplasmosis in cats even as definitive hosts usually enrolled asymptomatic infection, even during removal of oocyst. Sometimes clinical signs are present, primarily associated with respiratory type interstitial pneumonia, dyspnoea, lethargy and anorexia, ocular signs (uveitis, retinochoroiditis) or neuromuscular signs (Hartmann *et al.,* 2013).

2.5.2. Pathogenesis

It is probably not by chance that the three main immune- privileged areas of the body, the placenta, the brain and the eye, are major targets of pathology in humans (Uwe *et al*., 2014). When the organism is ingested, bradtzoites are released from cysts or sporozoites are released from oocysts, and the organisms enter gastrointestinal cells. Following primary infection of intestinal epithelial cells, the parasite transforms into a stage called the tachyzoite. Tachyzoites candisseminates via the bloodstream throughout the host and has the ability to cross-vascular barriers, e.g. the blood-brain barrier, and to form local cysts (Lachenmaier*et al*., 2011; Feustel*et al*., 2012).

Invasion is an active process relying on parasite motility and the sequential secretion of proteins from secretory organelles, the micronemes, rhoptries, and the dense granules (Flegr*et al*., 2014). Host cell receptors consisting of laminin and lectin are involved in *T. gondii*tachyzoite attachment and penetration (Sonar and Brahmbhatt, 2010). It is the current understanding that dendritic cells and macrophages serve as 'Trojan horses' to guide the parasite throughout the body into the target organs (Courret*et al*., 2006; Lachenmaier*et al*., 2011). Parasite may spread first to mesenteric lymph nodes and then to distant organs by invasion of lymphatics, and blood and can multiply in virtually any cell in the body. After invasion of a cell, the ability of parasite to actively penetrate hosts cells results in formation of a parasitophorous vacuole. In the host cell, the parasite protects itself from toxic host molecules in a parasitephorous vacuole and replicates intracellularly within it (Uwe *et al*., 2014). The vacuole is formed primarily by invagination of host cell plasma membrane, which is pulled over the parasite through the concerted action of the actin-myosin cytoskeleton of the parasite (Hokelek, 2009). 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 tachyzoitesare also found to induce depression of non-phagocytic host cell surfaces as they push the plasma membranes to penetrate those (MacLaren*et al.*, 2004).

All extracellular forms of the parasite are directly affected by antibody normal immune response, but intracellular forms are not. The intracellular forms can transform into a slowly dividing bradyzoite. The stage differentiation from tachyzoite to bradyzoiteis thought to be triggered by certain cytokines of the cellular host immune response (Klaren and Kijlstra, 2002). This stage of the parasite is able to form a cyst wall around a large family of dividing parasites, thereby protecting the parasite against the mounting immune response of the host (Hokelek, 2009). In immunodeficient individuals and in some apparently immunologically healthy patients, the acute infection progresses, resulting in potentially lethal consequences such as pneumonitis, myocarditis, and necrotizing encephalitis (Hokelek, 2009).

2.5.3. Laboratory Approach

Diagnosis of toxoplasmosis is performed by isolation of the parasite from patients and more commonly by

serological tests (Gamble *et al*., 2005; Dubey, 2010).

Isolation of *T. gondii*: Direct isolation of live parasites is still considered the "gold standard" method for diagnosis of toxoplasmosis. Attempts of isolation of *T. gondii*are usually performed from aborted ovine, caprine fetuses and fetal membranes by inoculation of laboratory mice. Tissues samples are homogenized and then intraperitoneally inoculated in mice; in case of very virulent strains, tachyzoitescan be detected in peritoneal fluids 3-4 day, whereas the detection of cysts in mice brain by histological examination requires 40 days (Piergili-Fioretti, 2004).

Carpological tests: for the detection of oocysts in feces from naturally infected cats are of little significance because of the short patency (15 days). However, fecal samples from experimentally infected cats, e.g. following bioassay, can be processed by common flotation methods and by microscope examination of the oocysts (Mancianti*et al.,* 2010).

Tissue section:Tachyzoites and tissue cysts can be found in tissue sections and organs fixed and stained with hematoxylin and eosin. In cases of abortion and stillbirth in sheep and goats, affected placental cotyledons typically contain large foci of coagulative necrosis that may have become mineralised with time (Dubey, 2010).

Serologic Tests: The detection of Toxoplasma-specific antibodies is the primary diagnostic method to determine infection with Toxoplasma (Singh, 2003). The Sabin-Feldman dye test is the so-called "gold standard" serological test for detection of anti- *T. gondii*antibodies in humans (EFSA, 2007).

Polymerase Chain Reaction (PCR): Is used to detect *T. gondii*organisims in tissue samples. Parasite DNA can be extracted and purified from several tissues, including tissues from aborted fetuses, placenta, the central nervous system, blood and fluids. The specificity of this test is almost 100%, but the difficulty to extract DNA and concentrate large sample quantities results in limited sensitivity (Alfonso *et al*., 2009).

Histologic Diagnosis:Histological examinations of biological samples show a lack of reliability since the parasite is not evenly distributed in tissues and animals can be infected with few parasites (PiergiliFioretti, 2004; Cenci-Goga*et al*., 2011).

Enzyme-Linked Immunosorbent Assay (ELISA): are available for the detection of IgG and IgM and also for testing the avidity (functional affinity) of IgG antibodies in pregnant women in order to discriminate between recently acquired infection and those obtained in the more distant past (Hedman*et al*., 1989).

2.6. Management of Toxoplasmosis

2.6.1. Control and Preventionsof Toxoplasmosis

Cleaning and drying of the environment, the use of clean utensils for feed and water, bathing to remove adhering faeces or cysts and proper disposal of faeces cat are pre-requisites to avoid animal to- animal transmission. Measures to prevent infection from food animals**,** includekeeping the animals indoors; keeping cats away fromfarms, feed, and bedding production and storage; providingclean drinking water and blocking access to surface water; implementingstrict rodent control; and refraining from feedingoffal and raw goat whey. Controlled indoor husbandry has drasticallyreduced the prevalence of *T. gondii* infection in pigs andis considered an important factor in the decrease of seroprevalencesobserved in human populations (Edelhofer*et al*., 2010). Reducing the environmental oocyst burden is considered the most effective way to reduce *T. gondii* infections in farm animals (Kijlstra and Jongert, 2008).

Reduction of environmental contamination, The amount of infectious oocysts in the environment thus depends on feline population size, incidence of *T. gondii* infection in felines, amount of oocysts shed by an infected feline, fraction of oocysts that end up in the environment and sporulate, and survival of infectious oocysts (Dabritz and Conrad, 2010).To regulate the cat population, cat owners need to have their cats spayed or neutered in a timely manner. In addition, control strategies for stray cat populations must be implemented. To reduce the number of prey animals caught, the cat can be keept indoors, especially at night (Woods *et al*., 2003). Cat owners should be made aware that their free-roaming cats can acquire infection mainly through hunting or by eating raw meat and faecally shed the protozoan parasite, Toxoplasma; therefore, Cat owners should be encouraged to keep their pets indoors and collect cat faeces in litter boxes destined for disposal in sanitary landfills (Dabritz and Conrad, 2010).

To minimize Toxoplasmosis from food,*T.gondii*tissue cysts in meat are inactivated in different ways; cooking, irradiation and heating meat throughout to reach a temperature in excess of 66°C is sufficient to kill cysts in meat (Goldsmid*et al.,* 2003). Whereas the curing of meat with salt does not seem to affect the parasite immediately (Dubey, 2000). Overnight freezing causes cysts in meat tissue to lose infectivity. Various reports have described this effect at temperatures between –6°C and –40°C (Kotula*et al*., 1991). Fruits and vegetables should be peeled or thoroughly washed before they are eaten and raw milk should not be drunk (Narladkar*et al*., 2006).

Persons who work with soil or garden regularly should wear gloves to protect themselves from pathogens in soil, such as *Toxoplasma gondii,* that are spread by owned and feral free-roaming cats to food (Dabritz and Conrad, 2010).

Mass education**:** Education of pet owner's consumers of meat and milk about possible risk of toxoplasma infection and care to be taken for protection (Sonar and Brahmbhatt, 2010).Much of congenital toxoplasmosis can be prevented by educating women of childbearing age and pregnant women to avoid eating raw or undercooked meat, to avoid cross-contamination of other foods with raw or undercooked meat, and to use proper cat-litter and soil-related hygiene (Jeffrey *et al*., 2001).

Vaccination is another way to reduce infection but there is no approved human vaccine exists against *Toxoplasma gondii*. The research on human vaccines is ongoing(Verma and Khanna, 2013). Toxovax, the vaccine consists of tissue culture grown live attenuated S48 tachyzoites after around 3000 passages twice weekly it was shown to have lost its ability to develop tissue cyst and has been used to control toxoplasmosis in ewes in New Zealand only one injection 3 weeks before mating is recommended (Reddy, 2006).

2.6.2. Treatment of Toxoplasmosis

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 (Teshager*et al*., 2014). The aims of treatment are to control active infection and to prevent its transplacental transmission (Moncada and Montoya, 2012). Currently, sulphonamide drugs and pyrimethamine used in combination are the gold-standard medicines for treating toxoplasmosis (McLeod *et al*., 2014). Pyrimethamine is tolerated by most people, but it has some side effects like nausea, vomiting and diarrhea in the first few days of treatment while Sulfadiazine also causes skin rashes, itching and sensitivity to light, joint pain, fever and chills (Eghianruwa, 2014). For people who cannot tolerate pyrimethamine, sulfadiazine or clindamycin, the combination of: pyrimethamine with azithromycin, atovaquone with pyrimethamine and atovaquone with sulfadiazine can be used (Jayawardena, 2008).

3. Conclusion and Recommendations

Toxoplasmosis is a zoonotic disease widely distributed throughout the world and is caused by the intracellular parasite *Toxoplasma gondii.* It is transmitted to humans by consuming of raw or undercooked meat containing *T. gondii* tissue cysts, eating unwashed raw vegetables or fruits, or anything contaminated with oocysts shed in the feces of an infected animal and transplacentally transmitted from mother to fetus. The majority of *Toxoplasma* infection occur in immunocompromised hosts, immunocompetent host and pregnant women can be life threatening and whom symptoms are more severe.

Recommendations: Based on this conclusion the following recommendations are forwarded.

- Creating public awareness about toxoplasmosis should be promoted.
- Feeding cats with cooked animal byproductsshould be practiced.
- Strategies to prevent exposure of food animal to *T. gondii*should focus on improvement of management of farm, proper farm sanitation, feed hygiene, rodent control and prevention of access of cats to farms.
- Empty cat litter trays daily, dispose of litter carefully and disinfect tray with boiling water. If this is done every day, even ifa cat is excreting oocysts, they will not have sporulated and therefore will not be infectious by the time the litter is changed
- It is very important that doctors, veterinarians and people working in the health and environmental industries work together for the development of control strategies, new therapies and effective vaccines for the control of *T. gondii*infection in animals, peoples and, especially in cats.

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