The Zoologist Vol. 1 (4) 100-111 (2006)

TOXOPLASMOSIS A RE-EMERGING ANCIENT DISEASE.

Neils, J.S.[1]* and Lawal, I.A.[2]

[1]*Department of Animal Production and Aquaculture, Adamawa State University, Mubi*

[2]*Department of Veterinary Parasitology and Entomology, Faculty of Veterinary Medicine, ABU, Zaria.*

Abstract

Toxoplasma gondii, the protozoan causative agent of toxoplasmosis, was first discovered in 1908 by Nicolle and Manceaux in an African rodent, *Ctenodactylus gondi*. The parasite occurs as a single species, divided into three types on account of its virulence. The parasite has the *Felidae* as the definitive, while other warm-blooded animals including man are paratenic hosts. The disease toxoplasmosis has a worldwide distribution. Serological surveys carried out in different parts of the world showed that, free range chickens had positive titers ranging from 17.9 to 82.6%; sheep, 9 to 23.1%; goats, 0-100%; cattle, 3 to 20.9%; swine, 6.7 to 28.8%; in dog, 6 - 86%; wild animals, 1 to 33.9%; horses and donkeys, though said to be resistant to *Toxoplasma gondii* are also affected with prevalence ranging from 0- 6%. The infection in sea mammals ranges from 7.6 to 22.8%. Human infections are wide spread with seroprevalence of 0- 90%. Human associated with domestic cats as pet are at high-risk for toxoplasmosis. Of recent it was found to cause abortion in sheep, human and even death in laboratory animals. *Toxoplasma gondii* has been found to kill a marmot (a hibernating animal), but in heterothermic (poikilothermic) animals, it does not multiply.

Key words: Toxoplasmosis, paratenic hosts, tissue cysts, abortion and prevalence.

Introduction

Toxoplasmosis is a disease caused by *Toxoplasma gondii*; in intestinal coccidia which belong to the Phylum Apicomplexa. It is an ancient group of protozoan that consists of an estimated 5000 species (Dubey and Beattie, 1988). The felids are the definitive hosts while it has an unusually wide range of paratenic hosts, (PH), high prevalence, and a benign coexistence with most hosts, making it as one of the most successful parasite on earth (Zhang *et al.*, 2004). Infections by this parasite are common in many warm-blooded animals, including man (Vikoren *et al.*, 2004; Kikuchi *et al.*, 2004). About 300 species of mammals and 30 of birds have been identified as PH (Smyth, 1996; Silbey, 2003).

The parasite was first discovered in 1908 in a North African rodent, *Ctenodactylus gondi*, then reported in a laboratory rabbit in Sao Paulo, Brazil and Darling, almost at the same time, but independent reports (Dubey and Beattie, 1988).

The parasite is intracellular and infects any type of nucleated cell in the tissue or body of an organism except the immature red blood cell (Daryani *et al.*, 2003). All classes of livestock, wild grazing animals, birds and sea mammals are potential PH to *T. gondii*. However, animals like monkeys, rats, donkeys and horses are known to be highly resistant (Smyth, 1996). A host may die of acute toxoplasmosis, but more often recovers with acquisition of strong immunity. Infected animals do not show clinical signs, but there may be death depending upon the organ the parasite infects. Organs may be seriously damaged due to the presence of bradyzoites and this could lead to death. (Dubey *et al.*, 2003). The parasite could cause abortion; stillbirth and even mummifications may occur in sheep (Kimbita *et al.*, 2001).

Infection in humans occurs but varies between ethnic groups and type of work. Thus higher prevalence were found in rural than urban settlements, in people who have much contact with soil, and due to sanitary and cooking habits (Fan *et al.*, 2003). Also prevalence was high in children who play with cats, in soil and or ate soil contaminated by cat faeces, and in abattoir

Corespondence Address

workers (Durbey and Beattie, 1988).

Infected pregnant woman may transmit the parasite transplacentally to her foetus and for this to occur, the parasite must have been acquired during pregnancy but transmission does not occur in subsequent pregnancies (Dubey and Beattie, 1988; Ali *et al*, 2003).

In Nigeria, few works on the seroprevalence of disease have been reported; Aganga *et al*, (1981), reported the presence of antibodies to the parasite in food animals (cattle, goats, sheep and swine) in some parts of Northern Nigeria. Aganga *et al.*, (1986) and Abubakar *et al.*, (1995), reported the prevalence of antibodies to *T. gondii* in camels. Okewole and Akpan (2002) reported the case of clinical feline toxoplasmosis in young and adult cats. Recently, Joshua *et al.*, (2003) found Toxoplasma antibodies in cattle slaughtered for consumption at township abattoir in Ibadan.

This review is an attempt to elucidate the disease toxoplasmosis, its prevalence in different species of animals, possible pathologic effects in both animals and humans. Prevention and control measures have also been discussed.

Epidemiology

Toxoplasmosis occurs in domesticated and wild animals and birds, and man. The *Felidae* family is the definitive host while other animals including man are PH. Some of the definitive host includes the domestic cat (*Felis catus*); jaguarondi (*F. yagouaroundi*); ocelot (*F.paradalis*); mountain lion (*F. concolor*); leopard cat (*F. bengalensis*) and bobcat (*Lynx rufus*). For PH, there is no specificity and almost any warm-blooded animal is capable of becoming infected, including man, (Jakubek *et al.*, 2001; Gennari *et al.*, 2004). The disease has a worldwide distribution, though prevalence varies according to climatic conditions, (Dubey *et al.*, 2003; Pereira-Bueno *et al.*, 2004; Zhang *et al.*, 2004).

Domestic animals

Free-range chickens

Work has been extensively done on free-range chickens where the parasites were isolated from the body tissues like: brain, leg muscle, breast and thighs, ovary, and shelled eggs, heart, gizzard and liver. Not much is known of the prevalence of T.gondii in commercially raised birds, however, Dubey *et al.*, (2004) found 46.6% of antibodies in a commercial flock of chickens in Israel. Dubey *et al.*, (2003) reported 16 (40%) of 40 free-range local chickens from Parana, Brazil had antibodies to the parasite. When ducks and chicks were screened in Egypt, 40.4% prevalence rate was found in 2-3 weeks old and about 50% of these birds died of the disease (Dubey *et al.*, 2003). In India, Sreekumar *et al.*, (2003) reported 17.5% seropositive titres in 133 birds sampled; 55% of 118 were confirmed seropositive in free-range birds in USA, (Dubey *et al* ., 2003). Some free-range birds in Argentina were also positive for *T.gondii* antibodies, (Dubey *et al.*, 2003). The authors concluded that, the prevalence of *T. gondii* in free-range chickens is a good indicator of the prevalence of the parasite oocysts in the environment since birds feed from the ground.

Food animals

Hartley and Marshall in 1951 isolated T.gondii from aborted material and claimed to be one of the main causes of abortions in sheep. Thereafter, *T.gondii* was recognized as one of the main causes of infective ovine abortion in New Zealand, Australia, England, Norway, and U.S.A. and may be in France (Dubey and Beattie, 1988). *T. gondii*-induced abortion can occur in ewes of all ages, although maiden ewes are most affected. Abortion occurs in ewes that acquire infection during pregnancy and less than 1% of ewes abort to toxoplasmosis in subsequent infection during pregnancies (Radostits *et al.*, 2000).

Hurtado *et al.*, (2001) conducted a survey of the presence of *T. gondii* in naturally aborted fetuses of ewes and found

that brains of the aborted fetuses gave a good diagnosis with PCR. Dubey and Beattie, (1988) had earlier stated that toxoplasmosis is a problem in the sheep as it is related to abortion. Pereira-Bueno *et al.*, (2004) serologically evaluated 173 ovine abortions associated with T.gondii, using different diagnostic techniques and reported that 40 (23.1%) of 173 abortions were due to the parasite.

In Nigeria, Aganga *et al.*, (1981), surveyed food animals serologically and found 18 (9.0%) of 200 sheep seropositive to *T. gondii* antibodies.

Cases of toxoplasmosis in goats have been reported in different parts of the world, the disease was found to cause abortion similar to that in sheep. An infection rate of 0-100% could be found in goat farm. Infection is always higher in older animals (Dubey and Beattie, 1988) and when sporulated oocysts are given to pregnant animal, it aborted, (Radostits *et al.*, 2000). Aganga *et al.*, (1981) found 9 (4.5%) of 200 goats were positive.

Cattle are thought to be relatively resistant to toxoplasmosis; however, an isolation of the parasite was reported in Czechoslovakia, (Dubey and Beattie, 1988). Aganga *et al.*, (1981) reported 3.0% seropositivity in 200 cattle tested and Joshua *et al.*, (2003) found 99 (16.9%) of 586 surveyed to have antibodies to *T. gondii*. Similarly, camels were screened, Abubakar *et al.*, (1995) reported 14.4% antibodies in Sokoto, while earlier, Aganga *et al.*, (1986) reported 9.4 % seroprevalence in Kano.

Swine seem to be more susceptible than cattle as T.gondii has been isolated from various tissues of pigs in the United States and Taiwan, (Dubey and Beattie, 1988; Fan *et al.*, 2004). Although there was no isolation of the parasite reported in Nigeria, Aganga *et al.*, (1981), found seropositivity of 11 (6.79%) of 162 pigs to *T. gondii* antigen.

Domestic and Feral cats

Cats are pivotal in the epidemiology of toxoplasmosis because they are the only definitive hosts known to excrete environmentally resistant oocysts in faeces. A worldwide sero-survey revealed that *T.gondii* infection varied from 2.1-74.4%, although the infection is common in cats but clinical toxoplasmosis is usually considered rare (Samad *et al.*, 1997; Gauss *et al.*, 2003).

The prevalence of T.gondii infection varies according to the life style of cats. It is higher in feral cats that hunt for their food than those kept and fed. Feral cats therefore, play important role in the spread of the parasite because of their lifestyle (Labbelle *et al.*, 2001; Ali *et al.*, 2003).

Wild animals

Little is known of toxoplasmosis in buffaloes, however a prevalence rate of 24% was found in 837 wild animals and 8 of 121 buffaloes were positive to skin test for *T.gondii*. Viable *T. gondii* parasites were demonstrated in edible tissues of red deer in New Zealand, roe deer in West Germany, and proghorn, moose, and mule deer in the US (Dubey and Beattie, 1988; Vikoren *et al.*, 2004).

Other wild animals found to be positive for antibodies against *T.gondii*, are Black bear (*Ursus americanus*), Feral pigs (*Sus scrofa*), Black tailed deer (*Odocoileus hemionus columbianus*), White tailed deer (*Odocoileus virgianus*), Fallow deer (*Dama dama*) and Axis deer (*Axis axis*), (Dubey and Beattie, 1988).

Dog

Generally, excretion of oocysts does not occur in normal dogs. However, age and concurrent infections are the two main factors that determine the clinical outcome of canine toxoplasmosis. Dogs and people can acquire infection from the same source if they leave in the same house or in neighborhood. Cases of 9-86% seroprevalence were found in dogs in the U.S.S.R. Prenatal infection have been reported in pregnant bitches that are infected, (Dubey and Beattie, 1988).

Horses and Donkeys

These animals are said to be resistant to *T. gondii* infection, however,

when five horses were experimentally infected with millions of tachyzoites, four recovered and one that was inoculated orally died 17 days post infection (DPI). A survey of donkeys in India showed that, donkeys had high titers of 64 with IHA in only 6% of 67 animals (Dubey and Beattie, 1988).

Sea mammals

T. gondii infection has been reported in sea lions (*Zalophus californianus*) which were housed in fresh water. A serological survey of 43 sea lions, one was found positive and died due the disease. Harbour seal (*Phoca vitulina richardii*) had toxoplasmosis concomitantly with staphylococcal infection and a northern fur seal (*Callorhrus ursinus*) proved positive to *T. gondii* antibodies (Dubey and Beattie, 1988). Lambourn *et al.*, (2001) found harbor seals with antibodies to *T .gondii* in 29 (7.6%) of 380 at Southern Puget Sound Washington. Cobezon *et al.*, (2004) also found 13.0% of 23 male and 22.8% of 35 female dolphins at the Spanish Mediterranean coast to be positive. Dubey *et al.*, (2003) found antibodies to *T. gondii* in marine mammals that were presented for either post mortem or clinical examination and recently antibodies were found in elephant seal (*Mirounga angustirostris)*. When grey seals were experimentally infected, Gajadhar *et al.*, (2004), found that these mammals are susceptible to *T. gondii*.

Man

Toxoplasma gondii infection is widespread among humans and its prevalence varies widely from place to place. It seems that human infection ranges from 0-90%, and infection rises with age. Infections are common in warm climates and mountainous regions. This probably is related to the conditions favorable for sporulation and survival of oocysts, (Fan *et al.*, 2003). The disease is zoonotic and prevalence of infection varies between ethnic groups, but may be due to sanitary and cooking habits rather then to genetic differences. Outbreaks of infection have been recorded among children who played with soil contaminated by cat feces. Higher prevalence rates are found in low altitudes and in wet areas than in mountainous and dry areas. T.gondii infection is also higher in abattoir workers (Dubey and Beattie 1988; Ali *et al.*, 2003).

Fan *et al.*, (2003) surveyed Chinese Aboriginal and Han people residing in mountainous areas of Northern Thailand. They found antibodies to *T.gondii* in 123 (20.6%) of 597, and attributed the high percentage to the habits of the aboriginal people who are known to eat raw meat during hunting.

Toxoplasmosis in humans has been found to cause serious problem in pregnant women, it is known to cause abortion but infection must be acquired during pregnancy. Abortion does not occur in subsequent pregnancies (Soulsby, 1986; Dubey and Beattie, 1988; Paul *et al.*, 2001).

Hibernating and Poikilothermic animals

An interesting phenomenon is the ability of *T.gondii* to remain dormant in hibernating animals and to resume activity when they waken. This is exemplified by infection in marmot, the animal remained well during the period of hibernation, but when it wakened, it developed acute toxoplasmosis and died. *T. gondii* can survive, but does not multiply in poikilothermic animals, (Dubey and Beattie, 1988).

Etiology

There is only one known species of *Toxoplasma* that was discovered by Nicolle and Manceaux in 1908, though Levine in 1977 attempted the inclusion of *Hammondia hammondi* as the second species (Soulsby, 1986). The causative agent *T. gondii* is a systemic coccidian, a universal parasite, sporozoan and a member of the suborder *Eimerina*. The parasite has the *Felidae* as definitive host, but has a wide range of intermediate hosts (Soulsby, 1986; Smyth, 1996).

Animals become infected either by taking the sporulated oocyst from the cat feces or

in carnivores and man through the intake of improperly cooked or raw meat infected with tachyzoites and/or bradyzoites (tissue cysts).

Toxoplasma gondii isolates have now been classified into 3 genetic types (I, II, III) based on restriction fragment length polymerization (RFLP), genotypes II and III are said to be more virulent (Fuentes *et al.*, 2001, Dubey *et al.*, 2004].

Transmission

Fundamental to the transmission of *T. gondii* is the cat and other *Felidae*. An infected cat may shed oocysts in its feces. These are not immediately infectious, but must first sporulate outside the body of the host; this process usually takes 1-5 days, depending upon temperature, moisture, and other environmental conditions. As a rule, the duration for excretion is from 1-3 weeks and is rarely repeated, although it may be re-stimulated by malnutrition, infection with *Isospora felis* or by administration of cortisone (Samad, 1997). The proportion of cats excreting oocysts at any one moment is not high, being usually not more than 2% in most countries. But a cat may shed millions of oocysts, and they are very hardy, capable of surviving in the soil for a year or more, therefore, the danger of infection is obvious. The milk of the cats is another source of infection to its kittens (Powell *et al.*, 2001).

Life cycle

In cat

Cat gets infected by ingesting oocysts, tachyzoites or bradyzoites from mice or rats that were previously infected. When these two latter forms are taken, there are two routes of invasion:

i). The intestinal development - the intestinal epithelia are invaded and followed by several series of asexual generations during which the parasite divide by an unusual process known as *endodyogeny*. This asexual multiplication is followed by macro-and micro-gametogenesis then fertilization takes place, the zygotes develop into a thick wall oocyst. The oocyst can only be infective when it sporulates in 2-3 days under favorable conditions. The definitive host is able to secrete oocysts for 1-3weeks after which no more secretion except when cat is stressed or administered corticosteroid (Samad *et al.*, 1997).

The prepatent periods after ingestion of oocysts are > 20 days, >19 days after ingestion of mouse infected with tachyzoites and 3-10 days of mouse with tissue cysts (Smyth, 1996).

ii). Extraintestinal development: this occurs after asexual multiplications. Some of the organisms by pass the intestine into other tissues and organs. The mesenteric lymph nodes are infected first, followed by the liver, lungs then other tissues like the muscles, and heart. Tissues cysts are formed and multiply only when eaten by either the definitive host or another PH.

Intermediate PH

Toxoplasma can only complete its sexual development in the cat. In the paratenic hosts, when oocysts are ingested the sporozoites hatch and cross the gut wall and invade the macrophages and almost any host cell types except red blood cells (RBC) (Anonymous[1], 2004). The endodyogeny in the infected cell is very rapid and before immunity develops, the parasite fill the entire cell. The accumulation of these organisms, bounded only by plasmalemma of the host cell, is known as pseudocyst. Within the pseudocyst , the zoites divide rapidly again and are known as tachyzoites. The host cell eventually burst to release the tachyzoites, which invade other host cells. This proliferative phase is usually slowed down by the host immunity and the zoites now called bradyzoites accumulate to form tissue cysts (Ferguson, 1999).

These tissue cysts are found in almost every part of the body especially the central nervous system (CNS), lungs and muscles. The cysts become latent except when there is immunosuppression in animals and/or when HIV/AIDS is acquired in humans (Samad *et al.*, 1997).

Pathogenesis and Clinical findings

Toxoplasma gondii is an intracellular parasite that attacks most organs, with affinity for the reticuloendothelial (RE) and CN systems. Sporozoites from oocysts or bradyzoites penetrate and multiply in the intestinal epithelium. After the invasion of the cell, the parasite multiply and eventually fills and destroys the cell. Liberated parasites reach other organs via the blood stream. The stage of parasitaemia commences approximately 5 days after initial infection and declines with the development of immunity 2-3 weeks at which stage the organism localizes as tissues cysts (Smyth, 1996; Radostits *et al.*, 2000).

The clinical characteristics of the disease vary with the tissue cells infected, which in its self varies depending on whether the disease is congenital or acquired and the genetic type of the infected animal, (Dubey *et al.*, 2004).

Naturally acquired infection

Generally, toxoplasmosis is a disease that when acquired may not show signs. The parasite has been isolated from healthy animals and man. However, this assertion depends upon the immunocompetency of an animal or individual (Hurtado *et al.*, 2001).

Sheep

Toxoplasma gondii-induced abortion can occur in ewes of all ages, although maiden ewes are most affected. Ewes that acquire the infection during pregnancy abort but usually less than 1% of ewes abort due to toxoplasmosis in subsequent pregnancies. Masala *et al.*, (2003), in Italy, found 31.5% of 2421 samples from sheep to be positive to *T.gondii*, this includes aborted fetuses.

Lesions in aborted material

The main changes in the placenta are focal inflammation, and necrosis of the fetal cotyledon but inter-cotyledonary areas are normal. Lesions may vary from micro- to macroscopic where the characteristic lesions consist of white flecks or multiple white, chalky nodules up to 2mm in diameter. These foci may be sparse or dense and may occur in any plane of the cotyledons and are viewed better when properly washed in saline solution, (Masala *et al.*, 2003).

Fetus - *Toxoplasma* induced gross lesions in fetus are generally non-specific (anasarca, excessive fluid, hydrocephalus, etc) and are possibly related to impaired nutrition and intrauterine death. Small-circumscribed discrete chalky nodules up to 1mm in diameter may be seen in the liver (Pereira-Bueno *et al.*, 2004).

Congenital infection

In sheep, congenital infection occurs and the principal manifestations are encephalitis and febrile exanthema with pneumonitis and enterocolitis (when heavy infection occurs).

In humans, when infection occurs during pregnancy, premature birth or intrauterine growth retardation or both may occur. Other conditions likely to be seen are hyperbilirunemia, hydrocephaly or microcephaly, intracranial calcification and retinochoroiditis. Retinochoroiditis do not show immediately but later in life and at times during adult hood (Dubey and Beattie, 1988).

Other animals

At times stillborn or weak calves may be observed with *T. gondii* infection. The disease does not play any important role in bovine abortion. However, calves with congenitally acquired infection show fever, dyspnea, coughing, sneezing, nasal discharge, colonic convulsions, grinding of teeth and tremor of the head and neck. Death occurs after a course of 2-6 days, (Dubey and Beattie, 1988).

Caprine toxoplasmosis is manifested by perinatal deaths, including abortions and stillbirths. Systemic disease with a high case fatality rate can occur especially in young goats, (Radostits *et al.*, 2000; Nishi *et al.*, 2001).

Pigs are highly susceptible; pigs of all ages

can be affected. Young pigs are often acutely ill with a high fever of 40-42oC, they develop diarrhea and die after a course of several weeks. Pigs of 2-4 weeks of age have additional signs including wasting, dyspnea, coughing, nervous signs especially ataxia, (Radostits *et al.*, 2000).

Diagnosis

Clinical signs of toxoplasmosis are nonspecific and cannot be depended upon for a definite diagnosis. Therefore, diagnosis is made by biologic, serologic or histological methods or by some combination of them (Dubey and Beattie, 1988).

Diagnosis can be carried out by the following methods:

1. Use of laboratory animals

T .gondii can be isolated from patients by inoculation of laboratory animals and tissue culture. Secretions, excretions, body fluids, and tissues taken by biopsy (such as lymph nodes or muscle tissue) specimens from which to attempt isolation. Cerebral fluid from a child with congenital infection and encephalitis or lymph node material from a person with lymphadenopathy is good sources of *T.gondii,* (Fan *et al.,* 2003; Sreekmar *et al.*, 2004).

2. Biopsy

Diagnosis can also be made by finding *T.gondii* in host tissue removed by biopsy or at necropsy. A rapid diagnosis can be made by microscopic examination of impression smears stained with Giemsa stain. Careful diagnosis should be made as the host cells at times look like the parasites when undergoing degeneration, only crescent-like parasites should be confirmed (Dubey *et al.*, 2004).

3. Serology

Serology is only an aid to diagnosis (Foudrinier *et al.*, 2003). Even with all the available serologic tests, the results of examining one positive serum sample only establishes that the host has been infected at some time in the past. However, sampling 2-4 weeks after the first and when the titer is 26 fold, it indicates acute acquired infection (Foudrinier *et al.*, 2003).

Some serological methods that are performed for the detection of *Toxoplasma* antibodies:

i. Detection of humoral antibodies- many serologic tests have been used for the detection of IgG and IgM to *T.gondii* antibodies. The methods are:

Sabin-Feldman dye test (DT). This test remains the definitive test for human toxoplamosis. The DT antibodies occur more in man than any type of antibodies. This test is a complement-mediated neutralizing type of antigen-antibody reaction. It uses live tachyzoites that are incubated with accessories at 370C for 1hr and a dye (methylene blue) is added. Tachyzoites that are unaffected by antibodies are stained (Dubey and Beattie, 1988; Aganga *et al.*, 1981; Wongkamchai *et al.*, 1997; Dando *et al.*, 2001).

ii. Detection of circulating antigens-the ELISA is used for the detection of antigens in an infected animal and man. This is also useful in the diagnosis of *Toxoplasma* in congenital infection and immunosuppressed patients (Daryani *et al.*, 2003; Nishi *et al.*, 2001).

Other methods employed are:

iii. Demonstration of *T.gondii* by Immunoperoxidase staining (Wongkamchai *et al.*, 1997).

iv. Antigen-specific Lymphocyte Transformation (Pereira-Bueno *et al.*, 2004).

v. PCR (Villard *et al.*, 2003; Pereira-Bueno *et al.*, 2004; Sreekumar *et al.*, 2004).

It is noteworthy that antibody titer has no correlation with the severity of signs or symptoms.

Treatment

Sulfonamides and Pyrimethamine (Daraprim) are two drugs widely used for therapy of toxoplasmosis. These two drugs act synergistically by blocking the metabolic pathway involving p-aminobenzoid acid (PABA) and the folic-folinic acid cycle, respectively. These two drugs are well tolerated, but sometimes thrombocytopenia and/or leucopoenia may

develop (Radostits *et al.*, 2000). These effects can be overcome by giving folinic acid and yeast without interfering with treatment because the vertebrate host can utilize presynthetized folinic acid while *Toxoplasma* parasite cannot (Youn *et al.*, 2003).

Other commonly used sulfonamides like, Sulfadiazine, Sulfamethazine and Sulfamerazine, are also effective against toxoplasmosis.

Generally, any Sulfonamide that diffuses across the host cell membrane is useful as antitoxoplasmosis, these drugs are very effective against the proliferative stage, but cannot eradicate infection (Dubey and Beattie, 1988). Sulfa compounds are excreted within few hours of administration; therefore, treatment has to be given on daily divided doses usually for several weeks or months, 2-10mg of folinic acid could be added.

Other drugs like Diaminodiphenylsulfone (SDDS), Spiramycin, Lasalocid and Monensin have been found to be very effective in experimentally induced *Toxoplasma* infection. The combination of Spiramycin and Sulfonamide has been tried in infected pregnant women, but there was uncertainty in the prevention of prenatal fetal infection (Wallon *et al.*, 1999; Sciammarella, 2002).

Youn *et al.*, (2003) tried herb extracts of *Torilis japonica* against *T. gondii*, the extract inhibited 99.3% proliferation of the parasite while *Sophora flavescens*, had 98.7% inhibition. This may be an opening to new antiprotozoal drugs.

Ponazuril (PFA) was used in vitro to determine its efficacy in mice, it was found to prevent tachyzoite multiplication and can be used as a prophylaxis (Mitchell *et al.*, 2004).

Prevention and Control

Cats

i. Never feed uncooked meat, viscera, or bones to cats.

ii. Keep cats indoors to prevent hunting and scavenging.

iii. Trash cans be covered to prevent scavenging.

iv. Queens can be spayed to reduce cat population

Sheep and Goats

i. Cull sheep that had aborted due to toxoplasmosis.

ii. Fetal membranes and dead fetuses should be incinerated to prevent infection of felids and other animals on the farm.

iii. Cats should not be allowed near pregnant sheep and goats.

iv. Grains should always be covered to prevent contamination by oocysts.

Zoo animals

i. Cats including other *Felidae* should be housed in a building separately from others especially marsupials.

ii. As a rule never feed cats with raw or under cooked meat, but if it is necessary then feed frozen meat which is least infective.

iii. Utensils used for cleaning cat cages should be autoclaved or heated at 70oC for at least 10 minutes

Man

i. Wash Hands thoroughly with soap and water after handling meat or cat

ii. Cutting boards, sink tops, knives, and other materials coming in contact with uncooked meat should be thoroughly washed.

iii. Cook meat to 70oC before human or animal consumption

iv. Pregnant women should avoid contact with cats, soil, and raw meat and should be aware of the dangers it could cause

v. Cat litter should be emptied every day and not by pregnant woman

vi. Vegetables should be properly and thoroughly washed before eating

vii. Expectant mothers should be aware of the dangers of toxoplasmosis (Sciammarella, 2002; Anonymous[2], 2004).

Vaccination

T.gondii is highly immunogenic but infection can occur even in hosts solidly immune to the parasite (Dubey and Beattie,

1988). Immunization of cats is desirable for the prevention of shedding of oocysts. Such a vaccine is not available. Infection of cats with less virulent strain of *T.gondii* does not prevent oocysts shedding on challenge. However, certain anticoccidials (Lasalocid, Monensin, Sulfadiazine, Bay V19142, Pyremethamine, 2-sulfomyl-4, 4-diaminodiphenyl sulfane) can prevent or minimize oocysts shedding (Kajerova *et al.*, 2003, Daryani *et al.*, 2003).

References

Abubakar, M.B., Adedeji, O.S. and MacChide, D.A. (1995). Seroprevalence of *T. gondii* Antibodies in Camels in Sokoto, Nigeria. Trop. Vet. 13(1&2), 1-4.

Aganga, A.O., Belino, E.D., Adegboye, D.S. and Ilemobade, A.A. (1981). A serological survey of Toxoplasmosis in Food Animals (cattle, sheep, goats and swine) in two Northern States of Nigeria. Int. J. Zoonosis 8, 57-62.

Aganga, A.D., Mijinyawa, S.M. and Aganga, A.A. (1986). Detection of *T. gondii* antibodies antibodies in slaughtered camels (*Camelus dramedraius)* in Nigeria. Nig. Vet. J. 15 (1&2): 91-93.

Ali, C.N., Harris, J.A., Watkins, J.D. and Adesiyun, A.A.(2003). Seroepidemiology of Toxoplasma gondii in dogs in Trinidad and Tobago.Vet. Parasit. 113, 179-187.

Anonymous[1], (2004). Toxoplasmosis. "http ://www.cdc.gov/ncidad/dpd/parasites/ toxoplasmosis/default.htm"

Anonymous[2], (2004). Toxoplasmosis. www.MedicineNet.com/script/main /art.asp

Cobezon, O., Resendes, A.R., Domingo, M., Raga, J.A., Agusti, C., Alegre, F., Mons, J.L., Dubey, J.P. and Alemeria,S. (2004). Seroprevalence of *Toxoplasma gondii* antibodies in wild dolphins from the Spanish Mediterranean coast. J. of Parasit. 90, 634-644.

Dando, C., Gabriel, K.E., Remington, J.SD. and Parmley, S.F. [2001]. Simple and Efficient method of measuring anti-*Toxoplasma* Immunoglobulin antibodies in Human sera using complement-mediated lysis of transgenic tachyzoites expressing B-galactosidase. J. Clin. Microbiol. 39 (6), 2122-2125.

Daryani, A., Hosseini, A.Z. and Dalimi, A. (2003). Immune responses against excreted/secreted antigens of *Toxoplasma gondii* tachyzoites in the murine model. Vet. Parasit. 113,123-134.

Dubey, J.P. and Beattie, C.P. (1988). Toxoplasmosis of Animals and Man. CRC Press, Inc. Boca Raton, Florida. 230pp.

Dubey, J.P., Navarro,I.T., Graham, D.H., Dahl, E.,Freire, R.L., Prudencio, L.B., Sreekumar,C., Vianna, M.C. and Lehmann,T. (2003). Characterization of *Toxoplasma gondii* isolates from free-range chickens from Parana, Brazil. Vet. Parasit. 117, 229-234.

Dubey,J.P., Graham, D.H., Dahl, E., Hilali, M., El-Ghaysh, A., Sreekumar, C. Kwok,O.C.H., Shen, S.K. and Lehmann,T. (2003). Isolation and Molecular Characterization of *Toxoplasma gondii* from chickens and ducks from Egypt. Vet. Parasit. 114, 89-95.

Dubey, J.P., Graham, D.H., Dahl, E., Sreekumar, C., lehamnn, T., Davis, M.F. and Morishita, T.Y. (2003). Toxoplasma gondii isolates from free-range chickens from the United States. The J. Parasit. 89 (5), 1060-1062.

Dubey, J.P., Venturini, M.C., Venturini, L., Piscopo, M., Graham, D.H., Dahl, E. Sreekumar, C., Vianna, M.C. and Lehmann, T. (2003) Isolation and Genotyping of *Toxoplasma gondii* from free-ranging chickens from Argentina. J. Parasit. 89 (5) 1063-1064.

Dubey, J.P., Salant, H., Sreekumar,C., Dahl, E., Vianna, M.C.B., Shen, S.K., Kwok, O.C.H., Spira D., Hambuger, J. and Lehmann, T.V. (2004). High prevalence of *Toxoplasma gondii* in a commercial flock of chickens in Israel, and public health implications of free-range farming. Vet. Parasit. 121, 317-322.

Dubey, J.P., Zarnke, R., Thomas, N.J., Wong, S.K., Van Bonn, W.,Briggs, M., Davis, J.W., Ewing, E., Mense, M., Kwok, O.C.H., Romand, S. and Thulliez, P. (2003). *Toxoplasma gondi, Neospora canunum, Sarcocystis neurona* and *Sarcocystis canis*-like infections in marine mammals. Vet. Parasit. 116, 275-296.

Dubey, J.P., Lipscomb, T.P. and Mense, M. (2004). Toxoplasmosis in an elephant seal (Mirounga angustirostris). J. Parasit. 90 (2), 410-411.

Dubey, J.P., Morales, E.S. and Lehmann, T. (2004). Isolation and genotyping of *Toxoplasma gondii* from free-range chickens from Mexico. J. Parasit. 90 (2), 411-413.

Dubey, J.P., Graham, D.H., De Young, R.W., Dahl, E., Eberhard, M.L., Nace, E.K., Won, K., Bishop, H., Punkosdy, G., Sreekmar, C., Vianna, M.C.B., Shen, S.K., Kwok, O.C.H., Summers, J.A., Demarais, S., Humpreys, J.G. and Lehmann, T. (2004). Molecular and Biological characteristics of *Toxoplasma gondii* isolates from wildwife in the United States. J. Parasit. 90 (1), 67-71.

Fan, C-K., Liao, C-W., Wu, M-S., Su, K-E. and Han, B-C. (2003). Seroepidemiology of Toxoplasma gondii infection among Chinese Aboriginal and Han people residing in mountains areas of Northern Thailand. The J. Parasit. 89 (6) 1239-1242.

Fan, C-K., Su, K-E. and Tsai, Y-J. (2004). Serological survey of *Toxoplasma gondii* infection among slaughtered pigs in Northern Taiwan. The J. Parasit 90 (3), 653-654.

Ferguson, D.J.P. (1999). Structural and Molecular differences in the host Parasite Relationship between the enteric (Coccidian) and exoenteric forms of *Toxoplasma gondii.* J. Protozool. Research 9, 141-151.

Foudrinier, F., Villena, I., Jaussaud,R., Aubert, D., Chemba, C., Martinot, F. and Pinon. J.M. (2003). Clinical value of specific immunoglobulin E detection by Enzyme-Linked Immunosorbent Assay in cases of acquired and congenital Toxoplasmosis. J. Clin. Microbiol.. 41 (4): 1681 – 1688.

Fuentes, I., Rubio, J.M., Ramerez, C. and Alvar, J. (2001). Genotypic characterization of *Toxoplasma gondii* strains associated with human Toxoplasmosis in Spain: Direct analysis from Clinical samples. J. Clin. Microbiol. 39 (4), 1566-1570.

Gajadhar, A.A., Measures, L., Forbes, L.B., Kapel, C. and Dubey, J.P. (2004). Experimental *Toxoplasma gondii* infection in grey seals (*Halichoerus grypus*). J. Parasit. 90 (2), 255-259.

Gauss, C.B.L., Almeria, S, Ortuno, A., Garcia, F and dubey, J.P. (2003). Seroprevalence of *Toxoplasma gondii* antibodies in domestic cats from Barcelona, Spain. J. Parasit. 89 (5), 1067-1068.

Gennari, S.M., Canon-Franco, W.A., Yai, L.E.O., De Souza, S.L.P., Santos, L.C., Farias N.A.R., Ruas, J., Rossi, F.W. and Gomes, A.A.B. (2004). Seroprevalence of *Toxoplasma gondii* antibodies from wild cats from Brazil. Vet. Parasit. 121, 337-340.

Hurtado,A., Aduriz, G., Moreno, B., Barandika, J and Garcia-Preez,A.L. (2001). Single tube nested PCR for the detection of *Toxoplasma gondii*

in fetal tissues from naturally aborted ewes. Vet. Parasit. 102, 17-27.

Jakubek, E-B., Brojer, C., Regnersen, C., Uggla, A., Schares, G. and Bjorkman, C. (2001). Seroprevalence of Toxoplasma gondii and Neospora cainnum in Swedish red foxes (*Vulpes vulpes*). Vet. Parasit. 102, 167-172.

Joshua, R.A. and Akinwumi, K.A. (2003). Prevalence of antibodies to *Toxoplasma gondii* in four breeds of cattle at Ibadan, Nigeria. Trop. Vet. 21 (3), 134-137.

Kimbita, E.N., Xuan, X., Huang, X., Miyazawa, M., Fukumoto, S., Mishima, M., Suzuki, H., Sugimoto, C., Nagasawa, H. Fujisaki, K., Suzuki, N., Mikami, T. and Igarashi, I. (2001). Serodiagosis of *Toxoplasma gondii* infection in cats by enzymes-linked immunosorbent assay using recombinant SAG1. Vet. Parasit. 102,35-44.

Kajerova, V., Literak, I. and Sedlak, K. (2003). Experimental infection of budgerigars *(Melopsittacus undulatus)* with a low virulent K21 strain of *Toxoplasma gondii*. Vet. Parasit. 116, 297-304.

Kikuchi, Y., Chomel, B.B., Kasten, R.W., Martenson, J.S., Swift, P.K. and O'Brien, S.J. (2004). Seroprevalence of *Toxoplasma gondii* in American free-range or captive pumas (*Felis concolor*) and bobcats (*Lynx rufus*). Vet. Parasit. 120, 1-9.

Labelle, P., Dubey,J.P., Mikaelian, I., Blanchette, N., Lafond, R., St-Onge, S. and Martineau, D. (2001). Seroprevalence of antibodies to *Toxoplasma gondii* in Lynx (*Lynx canadensis*) and Bobcats (*Lynx rufus*) from Quebec. J. Parasit. 87 (5), 1194-1196.

Lambourn, D.M., Jeffries, S.J. and Dubey, J.P. (2001). Seroprevalence of *Toxoplasma gondii* in harbor seals (*Phoca vitulina*) in Southern Puget Sound, Washington. J. Parasit. 87 (5), 1196-1197.

Masala, G., Porcu, R.,Madau, L.,Tanda, A.,Ibba, B., Satta, G. and Tola, S. (2003). Survey of ovine toxoplasmosis by IFAT and PCR assays in Sardinia, Italy. Vet. Parasit. 117, 15-21.

Mitchell, S.M., Zajac, A.M., Davies, W.L. and Lindsay, D.S.(2004). Efficacy of Ponazuril *in vitro* and in preventing and treating *Toxoplasma gondii* infections in mice. J. Parasit. 90 (3), 639-642.

Paul, M., Petersen, E. and Szczapa, J. (2001). Prevalence of congenital *Toxoplasma gondii* infection among newborns from the Poznan region of Poland: Validation of a new combined enzyme Immunassay for *Toxoplasma gondii*- specific ImmunoglobulinA and Immunglobulin M antibodies. J. Clin. Microbiol. 39 (5), 1912-1916.

Nishi, S.M., Kasai, N. and Gennari S.M. (2001). Antibody levels in goats fed Toxoplasma *gondii* oocysts. J. Parasit. 87 (2), 445-447.

Okewole,E.A. and Akpan, M.O. (2002). Clinical feline toxoplasmosis: parasitological, Haematological and serological findings in retroviral infected and uninfected cats. Vet. Arhiv 72 (2),67-79.

Pereira-Bueno, J., Quintanilla-Gozalo,A., Perez-Perez, V., Alvarez-Garcia, G., Collantes-Fernandez, E. and Ortega-Mora, L.M. (2004). Evaluation of ovine abortion associated with *Toxoplasma gondii* in Spain by different diagnostic techniques.Vet. Parasit. 121, 33-43.

Powell, C.C., Brewer, M. and Lappin, M.R. (2001). Detection of *Toxoplasma gondii* in the milk of experimentally infected lactating cats. Vet. Parasit. 102, 29-33.

Radostits, O.M., Gay, C.C., Blood, D.C. and Hinchcliff, K.W. (2000). Diseases caused by Protozoa. IN:

Veterinary Medicine. A textbook of Diseases of Cattle, Sheep, Pigs, Goats and Horses. 9th Edition. W.B. Saunders.pp1289-1321.

Samad, MD.A., Islam, MD. R., Dey, B. C. and Alam, MD. M. (1997). Effects of Corticosteroids in stray cats with natural antibodies to *Toxopasma gondii*. Journal of Protozoology Research 7, 1-8.

Sciammarella, J. (2002). Toxoplasmosis. HYPERLINK http://www. .emedicine.com/"

Sibley, L.D.(2003). Recent origins among ancient parasites. Vet. Parasit. 115,185-198.

Smyth, J.D. (1996). Tissue cyst-forming coccidian. In: Animal Parasitology. Cambridge University Press. Pp 99-104

Soulsby, E.J.L. (1986). Toxoplasma Nicolle and Manceaux 1908. In: Helminthes, Arthropods and Protozoa of domesticated animals. ELBS Ed. Bailliere Tindall, London.pp 670-682.

Sreekumar, C., Graham, D.H., Dahl, E., Lehmann, T., Raman, M., Bhaleao,D.P., Vianna,,M.C.B. and Dubey, J.P. (2003). Gentypes of Toxoplasma gondii isolates from chickens from India. Vet. Parasit. 118, 187-194.

Sreekmar, C., Rao, J.R., Mishira, A.K., Ray, D., Joshi, P. and Singh, R.K. (2004). Detection of toxoplasmosis in experimentally infected goats by PCR. Vet. Rec. 154,632-635.

Villard, O., Filiseti, D., Roch-Deries, F., Garweg, J., Flament, J. and Candolfi, E. (2003). Comparison of Enzyme-Linked Immunosorbent Assay, Immunoblotting and PCR for diagnosis of Toxoplasmic Chorioretinitis. J. Clin. Microbiol. (8), 3537-3541.

Vikoren, T., Tharaldsen, J., Fredriksen, B. and Handeland, K. (2004). Prevalence of *Toxoplasma gondii* antibodies in wild red deer, roe deer, moose, and reindeer from. Norway. Vet. Parasit.120, 159-169.

Wallon, M., Liou, C., Gavner,P. and Peyron, F. (1999). Congenital toxoplasmosis: Systematic review of evidence of efficacy of treatment in pregnancy. HYPERLINK "http:// bmj. Bmj journals.com/" http://bmj.bmjjournals.com

Wongkamchai, S., mahakittikun, V., Dechkum, N., Pasuralerkskkun, S. and Monkong, N. (1997). An immunocytochemical streptavidin-Biotin-Peroxidase- complex assay for the diagnosis of *Toxoplasma* infection. J. Protozool. Res. 7,114-121.

Youn, H.J., Lakritz, J., Kim, D.Y., Rottinghaus, G.E. and Marsh, A.E. [2003]. Anti-protozoal efficacy of medicinal herb extracts against *Toxoplasma gondii* and *Neospora caninum*. Vet. Parasit. 116, 7-14.

Zhang, S.Y., Jiang, S.F., He, Y.Y., Pan, C.E., Zhu, M. and Wei, M.X. [(004). Serologic prevalence of *Toxoplasma gondii* in field mice (*Microtus fortis)*from Yuanjiang, H uan Province, People's Republic of China. J. Parasit. 90 (20, 437-438.