

Ovine and Caprine Toxoplasmosis (*Toxoplasma gondii*)

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Abstract

Toxoplasmosis, caused by *Toxoplasma gondii* (*T. gondii*), is an economically important disease of livestock, especially sheep and goats, where it can cause early embryonic death and resorption, fetal death and mummification, abortion, stillbirth, and neonatal death.

Cats are the main reservoir for the toxoplasmosis which can contaminate the environments of other animal and human beings by their faeces that contain sporulated oocysts. Toxoplasmosis is diagnosed mainly by direct smear, Immunohistochemistry, serology testing and PCR. Preventive measures include education of the farmers, reduce environmental contamination by oocysts, reducing the number of cats capable of shedding oocysts, limiting the breeding of cats to maintain healthy adults, control of future breeding and adequate continuous control programs of stray cats. Feeding cats with commercial diets or with food processed either by cooking or freezing can reduce the risk of disease transmission. A live vaccine (Toxovax®) is commercially marketed in some countries for reducing losses to the sheep industry from congenital toxoplasmosis.

History, Life cycle, Clinical signs, Diagnosis, Mode of transmission, Epidemiology, Treatment, Control, Prevention and Vaccination against *T. gondii* infection in small animals have been reviewed in this article.

Keywords: *Toxoplasma gondii*, sheep, goat, abortion, diagnosis, control

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Introduction

Toxoplasmosis is an important zoonotic disease caused by the intracellular protozoan parasite *Toxoplasma gondii* (*T. gondii*). Mammals are intermediate hosts while wild and domestic felids are the definitive hosts. The main clinical sign of the disease in sheep, goats and humans is abortion (Dubey, 2009). The life cycle of the parasite consists of asexual reproductive stage in mammalian intermediate hosts and a sexual reproductive stage in the intestinal mucosa of feline definitive hosts (Garcia, 2001). Infection with *T. gondii* is widely prevalent in humans and animals worldwide and can occur pre- or post-natally (Dubey and Beattie, 1988).

It has been found that nearly one-third of the human population worldwide is infected (Dubey and Beattie, 1988). Toxoplasmosis causes heavy economic losses to sheep and goat industry and is considered as one of the main causes of infectious ovine and caprine abortion (Buxton *et al.*, 2007). Seroprevalence of *T. gondii* in sheep varied from 92% in France (Cabannes *et al.*, 1997) to 3 % in Pakistan (Zaki, 1995), while in goats varied from 69% in Austria (Edelhofer and Aspöck, 1996) to 4% in Senegal (Deconinck *et al.*, 1996).

Historical background

T. gondii was found for the first time by Nicholls and Bando in 1908 in the liver of the African rodent *Ctenodactylus gondii* (Dubey and Beattie, 1988; Dubey, 2008). Later, it became an important pathogen in livestock species, as reports from New Zealand described the existence of *T. gondii* in placental tissue from aborting sheep and within aborted ovine fetuses (Hartley and Marshall, 1957), but the route of transmission was not clear on that time. After it was found that this organism can infect sheep, as a herbivore animal species, trials were conducted to explore the routes of transmission other than eating grasses. The discovery in the late 1960's that cats can shed

a new environmentally stable form of the parasite in their faeces (Hutchison, 1965), led to the recognition of the cats as the definitive hosts of the parasite (Frenkel *et al.*, 1970) and the oocysts as major source of infection for animals and humans (Dubey 2004). The discovery of *T. gondii* oocysts helped to explain transmission of infection to herbivores and therefore, how the disease spread within and between flocks of grazing animals (Dubey and Beattie, 1988).

Etiology and life cycle

T. gondii belongs to Apicomplexa phylum, Sprozoa class, Eucoccida order, Emmerinae suborder and Sarcocystidae family (James, 1992). *Toxoplasma* has several strains; more than 95% of them are grouped into three genetic types (I, II and III). Type I is highly virulent in mice, type II is the most common type in persistently infected animals (sheep and goats) and type III is defined as non virulent strain. Clinical human infections are more often associated with type II strains (Sibley, 2003).

T. gondii life cycle includes definitive and intermediate hosts. The sexual and asexual cycle of the parasite can take place in the intestinal epithelial cell of the cat (definitive host), but in the intermediate host only asexual cycle takes place (Dubey 2008; Frenkel *et al.*, 1970; Dubey, 2004). In the cat, following a primary infection, oocysts are produced and shed in the feces. Oocysts require 1-5 days in adequate temperature and moisture to sporulate, before they become infective to birds and mammals (Gajadhar *et al.*, 2004; Dubey *et al.*, 1998a). When an intermediate host (sheep) gets infected by ingestion of contaminated feed or grazes on land with sporulated oocysts, the parasite (oocysts) will release and become able to actively invade and multiply within the gut cells. The tachyzoite stage of the parasite multiplies asexually by a process of endodyogeny within a parasitophorous vacuole and then the parasites eventually release from the ruptured

cell and invade further cells (Lingelbach and Joiner, 1998). By day four following infection, tachyzoites may be found in the mesenteric lymph nodes (Dubey, 1984; Dubey, 2004) and the parasites are also found in the circulation where they can spread throughout the host (Wastling *et al.*, 1993).

In pregnant animals, the tachyzoites invade and multiply within the caruncular septa in the placentome and then go on and invade the adjacent foetal trophoblast cells where they can spread to the rest of the fetus (Buxton and Finlayson, 1986). Tissue cysts may develop in visceral organs, including lungs, liver, and kidneys. They are more prevalent in muscular and neural tissues, including the brain, eye, skeletal, and cardiac muscle. Intact tissue cysts are probably harmless and can persist for the whole life of the host (Dubey *et al.*, 1998a).

When cats consume infected meat, the wall of the cyst is digested by the proteolytic enzymes in the stomach and small intestine of cats and bradyzoites are released in the gastrointestinal tract. Some of the bradyzoites penetrate the lamina propria of the intestine and multiply. Within a few hours, *T. gondii* may disseminate to extra-intestinal tissues. Other bradyzoites penetrate epithelial cells of the small intestine and initiate development of numerous generations asexually (Dubey and Frenkel, 1972). Oocysts of *T. gondii* are formed only in cats, including both domestic and wild felids. Cats shed oocysts after ingesting tachyzoites, bradyzoites, or sporozoites (Dubey, 2004). About three to ten days after infection, infected cats start to shed oocysts for two to three weeks (Dubey and Beattie, 1988).

Each infected cat may shed millions of oocysts to the environment (Dubey and Beattie, 1988), and as few as 200 sporulated oocysts can cause congenital disease in naive sheep (McColgan *et al.*, 1988). Under laboratory conditions, cats can shed as many as 500 million oocysts after ingesting one *T. gondii*-infected mouse (Dubey and Frenkel, 1972). Millions of oocysts were shed by cats fed even a few bradyzoites (Dubey, 2001). Up

to 13 million *T. gondii* oocysts were present per gram of cat feces (Schaes *et al.*, 2008). It has been reported that at any given time, approximately 1% of cats are expected to shed oocysts, based on the observation that most cats shed oocysts for about 1 week in their life (Dubey, 1995; Dubey, 2004). Cats shed millions of oocysts in their faeces that can survive for 12-18 months in the environment, depending on climactic conditions, and are an important source of infection for grazing animals (Tenter *et al.*, 2000, Innes 2009, Innes *et al.*, 2009). Shedding of oocysts tend to be more extensive amongst younger (neonatal cats) rather than older cats (Jackson and Hutchison, 1989; Buxton and Rodger, 2008).

Toxoplasmosis in cats

Cats are the definitive hosts of *T. gondii*; in fact they are the only animals that pass oocysts in their feces (Dubey and Beattie, 1988) for only 2 to 3 weeks following primary infection. Mature cats are less likely to shed *Toxoplasma* if they have been previously infected (Dubey and Beattie, 1988). The two main transmission mechanisms of *T. gondii* infection are through ingestion of either oocysts shed into the environment from the faeces of cats or viable tissue cysts found in raw or undercooked meat of intermediate hosts (Dubey and Jones, 2008; Innes *et al.*, 2009). Cats can suffer from clinical toxoplasmosis (Dubey and Jones, 2008). Most infected cats are asymptomatic, but early symptoms include lethargy, persistent fever and anorexia. Dyspnea and other signs of pneumonia are seen in many cats. Infections with severe respiratory signs are often fatal (Neufled and Brandt, 1974).

Toxoplasmosis in sheep

Sheep are important in many countries economically, as their products are a source of food or other benefits for humans. Sheep are commonly infected with *T. gondii*. Clinical symptoms in sheep include early embryonic death and resorption, mummification, stillbirths, neonatal death or birth of alive but

weak lambs (Buxton and Rodger, 2008). Up to 50% of sheep may develop fever, tremor, dyspnea and abortion in the last 4 weeks of pregnancy. Within 3-4 days after birth, death may occur with neonatal nervous sings (Waldeland, 1976). Malformations in the fetus have also been reported (Woods and Anderson, 1992).

Severity of infection is associated with the stage of pregnancy at which the ewe becomes infected. Infection during the early stage of gestation can result in fetal death, resorption and abortion, while infection in the latter stage of gestation (fetal immunity is relatively well developed), may have no clinical effect and lambs are usually born normal but infected and immune (Dubey and Beattie, 1988; Buxton *et al.*, 2007). The main pathological changes in infected animals are small white foci and necrosis on cotyledons and focal necrotic lesions in fetal brain, liver, and lungs (Buxton and Finlayson, 1986). The meat of infected sheep is a source of *T. gondii* infection for humans and carnivore animals (Dubey, 2009). Infection in sheep can occur after consumption of contaminated feedstuffs or grazing land with sporulated oocysts (Innes *et al.*, 2009) and transplacentally (Dubey, 1994; Esteban-Redondo *et al.*, 1995). Most infections in sheep occur following birth (Waldeland, 1977; Lunden *et al.*, 1994; Innes *et al.*, 2009). Some recent data suggest that in some circumstances persistently infected sheep may transmit the parasite to the fetus in subsequent pregnancies and abortions may occur (Morley *et al.*, 2005). Transmission of *T. gondii* tachyzoites in unpasteurised sheep or goat milk (Tenter *et al.*, 2001) also may occur via tachyzoites contained in blood products, tissue transplants, but are not probably important epidemiologically in animals (Tenter *et al.*, 2001).

Ovine toxoplasmosis occurs in temperate sheep rearing countries worldwide where the climatic conditions favour oocyst survival (Buxton and Rodger, 2008). Infection occurring at mid-gestation typically results in stillborn or weak lambs accompanied by a

small mummified foetus (Buxton and Rodger, 2008). Although the basic aspect was that toxoplasmosis can cause abortion only once in infected animals, a series of recently published papers from a group of researchers in England (Buxton *et al.*, 2007; Morley *et al.*, 2005; Morley *et al.*, 2008) have reported that repeated transplacental transmission of *T. gondii* in sheep may be more common than previously believed. All evidences presented were based on the detection of *T. gondii* DNA by Polymerase Chain Reaction (PCR) techniques. However, studies with different results also exist (Rodger *et al.*, 2006).

Toxoplasmosis in goat

Goats infected by *T. gondii* represent important sources of human infection due to consumption of meat and milk from infected animals (Dubey, 2004). Such fact is extremely important concerning the disease control and mainly for public health; since the consumption of goat milk is increased (Chiari and Neves, 1984; Chiari *et al.*, 1987) *T. gondii* tachyzoites have been also isolated from vaginal mucosa, saliva, nasal secretion and urine of experimentally infected goats (Dubey, 1980). The excretion of tachyzoites in the milk of naturally infected goats has also been reported (Chiari and Neves, 1984). A statistically significant correlation between positive serology for *T. gondii* in humans and consumption of goat milk has been found (Chiari *et al.*, 1987).

T. gondii can cause early embryonic death, resorption, fetal death, mummification, abortion, stillbirth and neonatal death depending on the stage of gestation (Dubey, 1991; John, 1999). The signs are more severe if the infection occurs in the first half, compared to the second half of gestation (Dubey, 1991).

Toxoplasmosis in cattle

Natural infection in cattle was first diagnosed in 1953 (Sanger *et al.*, 1953). Further observations showed that

toxoplasmosis is uncommon in cattle and does not appear to cause abortion (Dubey, 1986). Calves are more susceptible than adults (Nematollahi and Moghddam, 2008). Clinical signs of orally affected calves include diarrhea, anorexia, poor weight gain, depression, weakness, dyspnea and fever. In some cases just a modest lymphadenopathy may occur. Congenitally infected calves show fever, dyspnoea, cough, sneezing and neurological signs, while also stillbirths and neonatal deaths can be observed. If the disease occurs in adults, symptoms may include fever, dyspnoea, and nervous signs, followed by lethargy (Dubey, 1986).

Epidemiology of toxoplasmosis

Role of cats: *T. gondii* oocysts are shed by domestic cats and other felids resulting in widespread contamination of the environment (Dubey and Beattie, 1988). Domestic cats are the major source of contamination as they are common reservoir of infection and excrete large numbers of oocysts (Dubey and Frenkel, 1972; Dubey, 2001), while only a few cats may shed *T. gondii* oocysts at any given time. Latently infected cats can shed oocysts after being challenged by infection (Dubey, 1995), while congenitally infected kittens can also excrete oocysts (Dubey and Carpenter, 1993b).

Infection rates in cats are largely determined by the rate of infection in the local avian and rodent populations, which serve as a food source (Ruiz and Frenkel, 1980a). For example, *T. gondii* oocysts were found in 23.2% of cats in Costa Rica where infection in local rodents and birds was much higher (Ruiz and Frenkel, 1980a). For epidemiologic surveys seroprevalence data for cats are more useful than results of fecal examination because cats with antibodies have probably already shed oocysts and are indicators of environmental contamination (Dubey and Frenkel, 1972).

Under laboratory conditions, cats can shed as many as 500 million oocysts after ingesting one *T. gondii* infected mouse (Dubey and Frenkel, 1972). Cats fed even a few

bradyzoites can shed millions of oocysts (Dubey, 2001).

Environmental resistance of oocysts: Sporulated oocysts can survive for long periods under moderate environmental conditions. For example, they can survive in shaded and moist soil for months to years (Dubey and Beattie, 1988; Frenkel *et al.*, 1975). *T. gondii* oocysts are highly resistant to disinfectants, but are killed at temperatures above 60 °C (Dubey, 2004; Wainwright *et al.*, 2007a). Under laboratory conditions, oocysts remained infective from 30 days (in uncovered dishes at 37 °C) to 410 days or more, in covered and uncovered dishes at 4°C. Outdoors, infectivity varies from 46 days (uncovered, exposed to direct sunlight, mean air temperature is 20 °C) to 410 days or more (covered in shade and air temperature is 19.5 °C). *T. gondii* oocysts may remain infective for a year in warm climates and even longer in cool climates or in air-conditioned buildings (Yilmaz and Hopkins, 1972). Inactivation of *T. gondii* oocysts occurred with exposure to pulsed and continuous UV radiation at doses of ≥ 500 mJ/cm² (Wainwright *et al.*, 2007b).

Mode of transmission: Ingestion of contaminated water, food or unpasteurized milk with fecal oocysts shed by cat or oocysts from contaminated hands, utensils or surface (indirect transmission) is the most common mode of transmission (Dubey and Beattie, 1988; Dubey, 2008). Most sheep acquire *T. gondii* infection after birth. Although exact data are not available, it is thought that < 2% of sheep become congenitally-infected with *T. gondii*, and less than 4% of persistently infected sheep transmit it to the next generation (Buxton *et al.*, 2007; Dubey, 2009; Higa *et al.*, 2010). However, transplacental transmission from mother to fetus through infected placenta has been reported (Dubey and Sharma, 1980; Moura *et al.*, 2007; Dubey, 2008; Dubey and Jones, 2008; Lopes *et al.*, 2009; Scarpelli *et al.*, 2009). Also, *T. gondii* has been isolated from the semen of experimentally infected rams (Lopes *et al.*, 2009), bucks (Dubey and Sharma, 1980),

swine (Moura *et al.*, 2007), bulls and male dogs (Scarpelli *et al.*, 2009; Arantes *et al.*, 2009).

The main source for human infection is ingestion of uncooked meat containing viable tissue cysts or by ingesting food or water contaminated with oocysts from the feces of infected cats (Dubey, 2004), as well as unpasteurized milk (Higa *et al.*, 2010).

Water-borne transmission of *T. gondii* was considered uncommon but a large human outbreak linked to contamination of a municipal water reservoir in Canada by wild felids and the widespread infection by marine mammals has been detected (Dubey, 2004; Dubey, 2008). Furthermore, oocysts can be spread mechanically in the environment by flies, cockroaches, dung beetles and earthworms (Kniel *et al.*, 2002; Dubey, 2004).

Diagnosis

Histopathology: In abortion cases, multifocal necrosis and calcification might be seen in the placenta. The placental cotyledons can be bright to dark red (Dubey and Beattie, 1988; Buxton, 1998). Parasites can be detected in the placenta and in the fetal heart, brain, lung or liver (Dubey, 2008). Microscopically, necroses might be found in the white matter of the fetal cerebellum and cerebrum. Focal lymphoid-cell proliferations and micro necroses might be presented in fetal kidneys, adrenals, lymph nodes or brain (Buxton, 1998; Dubey, 2008; Dubey and Jones, 2008).

Immunohistochemistry: Immunohistochemical techniques allow visualization of both intact *T. gondii* and antigenic debris in tissue sections of aborted materials; they are convenient, sensitive methods and have the advantage, when compared with attempts at isolation, of detecting toxoplasma antigen even in decomposed tissues (Buxton, 1998; Dubey and Jones, 2008).

Direct smears: Direct smear from affected tissue proved rapid and easy diagnostic method (Terpsidis *et al.*, 2009).

Serological test: Serological test is used as

common method for diagnosis of toxoplasmosis which includes sabin Feldman dye test, indirect hemagglutination test (IHT), indirect fluorescent antibody test (IFAT), complement fixation test (CFT) and intradermal test (IDT) (Jacobs *et al.*, 1960; Dubey, 2008). Sabin–Feldman dye test was developed in 1948 by Albert Sabin and Harry Feldman (Dubey, 2008). The dye test is highly sensitive and specific with no evidence for false results in humans. The ability to identify *T. gondii* infections based on a simple serological test opened the field for extensive epidemiological studies on the incidence of infection (Dubey, 2008; Dubey, 2009), however it is very expensive, time consuming and not without hazard as it requires alive tachyzoites as antigen (Buxton, 1998).

The IHT is a simple, fast and inexpensive test using nonliving antigen; it's very practical and useful in veterinary and small diagnostic laboratories. This test measures antibodies that appear after two weeks or more after primary infection which mean no value for the test immediate infection but have less sensitivity than sabin Feldman dye test or IFAT (Jacobs *et al.*, 1960). IFAT requires intact tachyzoites and is more sensitive and specific compared to IHA and Enzyme-Linked Immunosorbent Assay (ELISA) that is used in the diagnosis of ovine toxoplasmosis (Jacobs *et al.*, 1960; Piergili, 2004).

The ELISA for *T. gondii* antibodies has been adapted for use in most domestic animals including sheep and goat (Dubey, 2008; Dubey, 2009). There is specific ELISA assays for both IgM and IgG subtypes. These ELISA assays are ideally suited to screen large numbers of samples and looking at the IgM/IgG ratio. The IgM/IgG ratio can be used to distinguish between the acute and chronic infections (Denmark and Chessum, 1978). Prenatal diagnosis of congenital toxoplasmosis may be made by detecting specific anti-toxoplasma IgM antibodies in fetal blood (Markell *et al.*, 1992), but congenital infections may be difficult to diagnose serologically, as maternal IgG crosses the

placental barrier and will appear and persist for several months in the circulation of the newborn. Since IgM antibodies do not cross the placenta, demonstration of anti-toxoplasma IgM at birth or up to several months of age is presumptive evidence of congenital toxoplasmosis (Brown and Neva, 1987).

The presence of specific antibodies in serum or tissue fluid from stillborn lambs or kids or in precolostral serum from live offspring indicates uterine infection (Buxton, 1998). Serological analysis using IFAT and ELISA has been widely employed in order to detect herds contaminated by toxoplasma, including swine and sheep flocks (Van der Puije *et al.*, 2000).

Molecular diagnosis: Burg *et al.* (1989) detected *T. gondii* DNA from a single tachyzoite using the B1 gene by PCR method for the first time. Several subsequent PCR tests have been developed using different gene targets. In general, this technique has been proven as a useful method in diagnosis of clinical toxoplasmosis (Dubey, 2008).

The B1 gene referred to as B1 repeat, is a 2214 base pair (bp) sequence with unknown function that is repeated 35 times in the genome of *T. gondii* (Jalal *et al.*, 2004; Edvinsson *et al.*, 2006). The PCR assay targeting the B1 gene has been used extensively (Jalal *et al.*, 2004). Recently, B1-PCR has been shown as the most sensitive protocol to detect *T. gondii* (Mason *et al.*, 2010).

Although some previous studies have reported the higher sensitivity of PCR targeting AF146527 over that of B1 gene which is usually used for diagnosis of toxoplasmosis, some recent studies suggest that the AF146527 element was absent in 4.8% of human *T. gondii*-positive samples, which may prove the B1 PCR technique as the choice one (Wahab *et al.*, 2010; Menotti *et al.*, 2010).

More recently, a 200-300-fold repeated (that exists in 200-300 copies/genome) 529 bp element of unknown function has been described in the genome of *T. gondii* (Edvinsson *et al.*, 2006; Kasper *et al.*, 2009). The higher sensitivity and accuracy of the 529-

bp PCR assay even in a faster protocol compared to B1 gene was reported (Edvinsson *et al.*, 2006; Kasper *et al.*, 2009). It has been postulated that an increased analytical sensitivity is achieved when a repeated DNA element is amplified, although some studies suggested no difference in analytical performance depending on the number of repeats (Wastling *et al.*, 1993; Edvinsson *et al.*, 2006).

Risk factors

Age: It has been reported that age can be associated with the seroprevalence of toxoplasmosis, as older sheep and goats had a higher prevalence of toxoplasmosis infection compared to younger sheep (Cavalcante *et al.*, 2008; Ramzan *et al.*, 2009; Kamani *et al.*, 2009).

Gender: It has been shown that female sheep and goats are more susceptible than males to toxoplasma infections (Ramzan *et al.*, 2009). Although there are other reports that did not show significant correlation between toxoplasma infection and the gender of the animals (Caballero-Ortega *et al.*, 2008; Cavalcante *et al.*, 2008).

Animal presence: The high seroprevalence of *T. gondii* antibodies in sheep may be associated with the presence of cats in almost every farm sampled. Newborn kittens are more dangerous than old cats (Dubey, 1994; Buxton and Rodger, 2008). Infected cats excrete toxoplasma oocysts which, after sporulation, become infectious to man and animals and remain infectious for a long period of time (Dubey and Jones, 2008). Also, multivariate analysis showed that the probability of infection was higher in herds where more than 10 cats were present. This might be related to greater environmental contamination by oocysts defecated in cat feces (Cavalcante *et al.*, 2008).

Climate: Higher prevalence rates of toxoplasmosis in warm and moist areas compared to those which are cold and dry is attributed to the longer viability of *T. gondii* oocysts in moist or humid environments (Van

der Puije *et al.*, 2000). A new study conducted in Mexico (Caballero-Ortega *et al.*, 2008) revealed that altitude and farm size, affects infection rate, as prevalence was higher at low altitudes and on large farms.

Management system: In extensive management systems, cats can be attracted to pen where animals are herded. It will also happen in free roaming pastures during the day. This may increase the chance of environment, food and water contamination (Cavalcante *et al.*, 2008).

Seroprevalence in intensively managed sheep was lower than in semi-intensively managed (Ragozo *et al.*, 2008). A recent study (Neto *et al.*, 2008) showed that both extensive/semi-intensive management systems were identified as risk factors associated with toxoplasmosis in goats. Use of wooden feeding troughs was also associated with goat toxoplasmosis. This might be due to fact that oocysts survive longer in moisture. The lack of feeding troughs also increased the probability of infection from pasture or water contaminated with sporulated oocysts (Cavalcante *et al.*, 2008).

Pharmaceuticals

To reduce economic losses due to toxoplasmosis, chemotherapeutic treatment of infected animals is essential in unvaccinated sheep flocks. Several drugs were used with good results such as decoquinatate (Buxton *et al.*, 1996), combination of pyrimethamine and sulfadimidine, vaquiloopruim and sulfadimidine or trimethoprim and sulfadimidine (Buxton *et al.*, 1993b). Injecting sulfadimidine in dose 33 mg/Kg/48 h, 4 injections in total, seems to be very effective in controlling toxoplasmic abortions in sheep flocks (Giadinis *et al.*, 2009). Moreover, monensin, given in the food during pregnancy, significantly reduced toxoplasma infection in sheep (Buxton *et al.*, 1988). Furthermore, clindamycin, spiramycin, atavaquone, arithromycin, clarithromycin and dapsona have been used with various results in non-ruminant species and humans (Giadinis *et al.*, 2009).

Prevention and control

Cats are born free of toxoplasma infection and start to excrete oocysts following a primary infection (Dubey and Jones, 2008). Cat faeces can create a large, potent, long-lasting source of infection for sheep. Oocyst contamination of farm foods and bedding, as well as pasture, is a threat to susceptible, pregnant sheep and goats, related to the number and distribution of cats (Dubey, 2008; Dubey and Jones, 2008) in the environment.

It is estimated that at any time given, about 1% of cats shed oocysts (Dubey and Beattie, 1988). Persistently infected mice, voles, shrews, rats, rabbits and small birds are the most important sources of cat infections (Jackson and Hutchison, 1989). Cats are considered as the main source of infection for sheep and goats (Dubey and Beattie 1988; Dubey and Jones, 2008). Ingestion of contaminated food and pasture is the most common source of small animal's infection (Dubey, 2004; Dubey and Jones, 2008). Water can be a real threat not only to animals but also to humans (Bowie *et al.*, 1997). Fields treated with manure and bedding from farm buildings where cats live can transmit oocysts and cause infection (Faull *et al.*, 1986). Cats defecating in farm feeds, such as hay and stored grain, will pose a risk for animals (Plant *et al.*, 1974). A single defecation may contain millions of oocysts (Lopes *et al.*, 2008). Further processing of the food disperse these oocysts evenly throughout the grain which can infect many sheep in flocks (McColgan *et al.*, 1988). During pregnancy in which the majority of herds are seronegative to *T. gondii*, all food and water should be kept away from cat's faeces and contaminated environment (Dubey, 1991; Hye-Youn Kim *et al.*, 2009).

Other measures to reduce environmental contamination by oocysts should be aimed to minimize the number of cats capable of shedding oocysts (Dubey and Jones, 2008). These would include limiting the breeding of cats, maintaining healthy adults and attempts to control future breeding, adequate and continuous control programs of stray cats to

reduce the risk of transmission of *T. gondii* and not allowing animals to live or stay outdoors, which will prevent them from hunting. Feeding cats with commercial diets or with food processed either by cooking or freezing can reduce the risk of disease transmission. Maintenance of a small healthy population of mature cats will reduce oocysts excretion, besides controlling the rodents population (Buxton and Rodge, 2008; Lopes *et al.*, 2008; Hye-Youn Kim *et al.*, 2009).

In the case of ovine toxoplasmosis, educating farmers to the principle infection root which is contamination of the environment with *Toxoplasma* oocysts via cat faeces and also measures that reduce the incidence of clinical disease, including good management of food and water, as well as vaccination with the live vaccine (Toxovax; Intervet B.V.) will reduce the disease occurrence (Buxton *et al.*, 2007). But further studies are needed to explore whether some sheep breeds have a particular genetic susceptibility to *T. gondii* (Buxton *et al.*, 2007).

Vaccination: Natural infection with *T. gondii* stimulates protective immunity in both sheep and goats (McColgan *et al.*, 1988) but inactivated toxoplasma tachyzoites, either alone (Beverley *et al.*, 1971) or in Freund's incomplete adjuvant (Wilkins *et al.*, 1987) do not protect pregnant sheep against experimental challenge with the parasite. The failure of these killed preparations in sheep may be partly because, in natural infections, persistence of the parasite in tissues continually stimulates immunity, as suggested in human toxoplasmosis (McHugh *et al.*, 1997). However experiments in which mice and hamsters were infected with a live temperature-sensitive mutant of *T. gondii*, which does not persist in the host, showed that it cannot form bradyzoites and cannot therefore form tissue cysts (Buxton, 1998).

A live vaccine (Toxovax) is commercially marketed in the UK, France and New Zealand for reducing losses to the sheep industry from congenital toxoplasmosis (Buxton and Innes,

1995). This vaccine was initially developed in New Zealand (Wilkins *et al.*, 1988).

The vaccine consists of a modified strain (S48) of *T. gondii*, which were originally isolated by mouse injection from a case of ovine abortion in New Zealand. After around

3000 passes twice weekly in laboratory mice, it was shown to lose its ability to develop bradyzoites in tissue cysts. The commercial vaccine consists of live cell culture-grown tachyzoites that have a shelf life of 10 days. It is recommended to be given 3 weeks before mating. One subcutaneous injection of this 2 ml suspension induces protective immunity for at least 18 months (Buxton and Innes, 1995). Abortions were reduced and lambing percentages significantly improved, compared to unvaccinated sheep in the same flocks (Spence *et al.*, 1992). After subcutaneous inoculation, S48 tachyzoites multiply locally, producing parasitemia and fever. Tachyzoites are controlled by the host immune response as soon as 10 days post infection and are not detectable by bioassays at 6 month post infection (Buxton *et al.*, 1993b).

Vaccinated sheep develop humoral and cellular immunity involving CD4, CD8 T cells, and IFN- γ (Wastling *et al.*, 1993; Wastling *et al.*, 1994). The mechanism of this persistent immunity in the absence of detectable live *T. gondii* is most intriguing and needs further research. It must be handled with care strictly according to the manufacturer's recommendations. As with sheep, the majority of goats previously exposed to infection with *T. gondii* develop a protective immunity to the parasite so that they are protected against subsequent challenge during pregnancy (Obendorf *et al.*, 1990).

The search for a non-infectious vaccine should continue because of the existing shortcomings of the live vaccine, its short shelf life and safety margins (Stanley *et al.*, 2004).

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توکسوپلاسموز در گوسفند و بز

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چکیده

بیماری توکسوپلاسموز که بوسیله تک یاخته توکسوپلازما گوندی (*T. gondii*) ایجاد می‌شود، بدلیل ایجاد مرگ زودرس جنین، مومیایی شدن، سقط، مرده زایی و مرگ و میر نوزادان، از اهمیت اقتصادی زیادی در دامهای اهلی و بویژه در گوسفند و بز برخوردار است.

گره‌ها اصلی ترین مخازن توکسوپلاسموز بوده، می‌توانند محیط سایر جانوران و انسان‌ها را از طریق مدفوع حاوی اووسیست‌های اسپوروله آلوده کنند.

توکسوپلاسموز بیشتر بوسیله مشاهده گسترش مستقیم، ایمونوهیستوشیمی، آزمایش‌های سرولوژی و PCR تشخیص داده می‌شود. اقدامات پیشگیرانه مشتمل هستند بر: آموزش دامداران، کاهش آلودگی محیط بوسیله اووسیست‌ها، کاهش جمعیت گره‌هایی که قادر به پخش اووسیست هستند، کاهش تولید مثل گره‌ها به منظر باقی گذاشتن گره‌های بالغ غیر آلوده، کنترل تولید مثل‌های آینده و برنامه‌های کنترلی منظم و پیوسته عقیم سازی گره‌ها. تغذیه گره‌ها با غذاهای تجاری آماده و یا با غذاهایی که بوسیله پختن یا انجاماد فرآوری شده‌اند می‌تواند نقش بسزایی در کاهش احتمال انتقال آلودگی داشته باشد. امروزه واکسن زنده (Toxovax®) در بازار برخی کشورها به منظور کاهش خسارات به صنعت پرورش گوسفند بوسیله توکسوپلاسموز مادرزادی ارایه شده است. در مطالعه حاضر، تاریخچه، چرخه زندگی، علائم بالینی، تشخیص، روشهای انتقال، اپیدمیولوژی، درمان، کنترل، پیشگیری و واکسیناسیون بر علیه آلودگی توکسوپلازما گوندی در دامهای کوچک بررسی می‌شود.

واژگان کلیدی: توکسوپلازما گوندی، گوسفند، بز، سقط، تشخیص، کنترل