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Prevalence of Camels Toxoplasmosis in Gedarif State Eastern Sudan

الانتشار المصلي لداء المقوسات في الإبل بولاية القضارف

شرق السودان

A thesis submitted to the College of Graduate Studies in the fulfillment of the requirements for the Master degree in Preventive Medicine

By:

Asjud Mohamed Ahmed Jomaa

BVSc, 2005, U of K

Supervisor:

Prof. Dr. Mohamed Abdelsalam Abdalla

Co-supervisor:

Prof. Dr. Salah Hassan Idris

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DEDICATION

To my Mother and Father with love and gratitudes,

To my husband Dr.yousif,
To my lovely kids,
Mohamed , Tasneem and Arwa

With love and respect

Acknowledgements

First of all, I would like to thank the merciful Allah for helping me in completing this work.

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Abstract

The current study was conducted to evaluate the seroprevalence of camel toxoplasmosis in four localities Gedarif state in the period from 2015-2016 . Serum samples were collected from 300 camels, 161(53.7%) of samples represents males and 139(46.3%) were collected from females. Latex Agglutination Test(LAT) was applied to screen all serum samples for Toxoplasmosis while ELISA was also used to confirm the positive result obtained by LAT . Using LAT, out of 300 serum samples 149 (49.7%) were positive to *Toxoplasma gondii*. Percentage of positive cases was more in females (52.3 %) than in males (47.7 %).The result of LAT test in camels show a seroreactivity correlated with significance between the surveyed locations (P<0.05). 149 samples were positive to toxoplasma gondii tested from different localities in Gedarif state was(AL-Shwak25(16.77%),AL-Butana48(32.21%),Wasat AL-Gedarif, 10(6.71%)and AL-Rahad ,66(44.29%)) with LAT test. The ELISA test showed that 44 (29.9 %) samples were positive for toxoplasmosis, the males were 21 samples (47.7 %)which was lower than the percentage in females 23 samples (52.3 %). The objectives were to estimate the seroprevalence of T. gondii infection and to assess risk factors from camels in the Gedarif State, Eastern Sudan, Also compare efficacy of latex agglutination test (LAT) and indirect enzyme linked immunosorbent assay (iELISA) in determination of *Toxoplasma gondii*seroprevalence.

الخلاصة

اجريت هذه الدراسة للتحري عن الاجسام المضادة للتوكسوبلازما في مصل الابل المتواجدة في اربعة محليات في ولاية القضارف من الفترة من 2015- 2016م .

جمعت 300 عينة منها 161 ذكور(53.7%)و139 من الاناث (46.3%) وباعمار مختلفة، لاجل التحرى عن الاجسام المضادة استخدم اختبار اللاتكسالتلازنى كاختبار مسحى لجميع العينات وايضا استخدم اختبار الاليزا الغير مباشر كفحص تاكيدى لفحص العينات الموجبة لاختبار اللاتكسالتلازنى وان 149(49.7%) من المجموع الكلى للعينات اظهرت اجساما مضادة موجبة لطفيل التوكسوبلازما باستعمال اختبار اللاتكسالتلازنى ،وكانت نسبة الاصابة في الاناث (52.3%)اكثر منها في الذكور (47.7%) نتيجة اختبار اللاتكس في الابل اوضحت ارتباط ين الموقع والنتيجة الموجبة .

عدد العينات الموجبة باختبار تراص اللاتكس التلازني 149 من محليات مختلفة في ولاية القضارف كانت النتيجة علي النحو التالي كل محلية على حدا هى الشواك25(16.77%)- البطانة48 (32.21%)- وسط القضارف 10(6.71%)- الرهد 66(44.29%).اما اختبار الاليزا غير المباشر الخاص بطفيل التوكسوبلازما والذى اجرى على العينات الموجبة لفحص اللاتكسالتلازنى فقد اظهرت تفاعلا موجبا مع 44(29.9%) عينة فقط وكانت نسبة الذكور 21 (47.7%) اقل من نسبة الاناث 23(52.3%).

الهدف من الدراسة هو تقييم الانتشار المصلى للاصابة بالتوكسوبلازما وقياس عوامل خطورة المرض في الإبل في ولاية القضارف شرق السودان وايضا لمعرفة كفاءة اختبار اللاتكس واختبار الاليزا الذي يستخدم لتاكيد النتائج الموجبة باختبار اللاتكس في تحديد الانتشار المصلى للتوكسوبلازماقوندياى.

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CHAPTER ONE

Introduction

Toxoplasmosis is one of the most significant animal zoonosis, *distributed worldwide and affecting almost all warm-blooded animal species , and especially humans (*Tender*et al 2000).The zoonotic* protozoan parasite, Toxoplasma gondii are widely prevalent in humans and animals(tender *et al* 2000 and Innes *et a*l 2011).. However, very few *data is available about the prevalence of human (*Siddig*et al 2010, Musa et al 2010 and Anon et al 2010) and animal toxoplasmosis in the Sudan including camel (*Elamin*et al 1992 and* Manal *2003). All* mammals, including humans, and birds are intermediate hosts, whereasFelidae (cats) are intermediate and definitive host, they are the only animalsthat pass oocyst in their feces. Sheep and goat meats are important infectionsources for toxoplasmosis (Sevgili *et al.*,2005).Humans become infected post-natally by ingesting tissue cysts from undercooked meat, consuming food or drinkcontaminated with oocysts or by accidentally ingesting oocysts from the environment. Felids are the most important host in the life cycle of *T. gondii* because they excrete environmentally resistant oocysts. Toxoplasmosis can rarely cause clinical disease in chickens (Dubey, 2010). The vast majority of natural *T, gondii* infections in domestic animals are subclinical. Clinical signs, when present, are generally vague and non-specific and may include a period of fever, anorexia, respiratory distress and sometimes diarrhoea. Central nervous system disorders are rarely reported. *T. gondii* infection, however, is the major cause of abortion and perinatal mortality in sheep and goats (Buxton and Brebner, 1998).Epidemiological information of protozoal agentscausing abortion and reproductive failure

in farm animalshas not been available in the Sudan. Yet these animals haveimportant role in local economy. Risk factors associated withthe prevalence of toxoplasmosis in Sudanese and theiranimals are unknown. Consumption of raw or undercookedmeat or/and milk, which are among the main risk factors foracquiring human infection, are popular tradition in the Sudan(*Seri et al*., 2003). Previous study in the Sudan by Elamin *et al* (1992) showed high prevalence rate (67%) in camel at Butana plain (mid- Eastern Sudan) at Gedarif, Subagh and Al-Showak area**.**

Objectives of the study:

The main goal of the present work:

- To study the prevalence of toxoplasmosis among camels using LAT and iELISA tests at different localities in Gedarif State .
- To increase the importance of scientific research for more epidemiological data on human toxoplasmosis in the Sudan.

Chapter one

Literature review

1-1. Sudanese camel:

In Sudan, remains of camel have been found in Marrowy, estimated closely to 15-25BC. (Adsion, 1934). According to very reserved estimates, camel population in the Sudan is about 4809000million, Ministry of Animal Resources And fisheries 2015and camels in(Fig 1) Gedarif State is estimated to be about 348172(Ministry of Animal Resources And fisheries 2015).

Camel in Sudan are raised mainly in a belt north of 12˚ N latitude, The Food and Agriculture Organization (FAO) of the United Nations estimates the world camel population to be 26,989,193 of which 89% are single-humped dromedary and 11 % are Bactrian (two-humped) (FAOSTAT 2015). Africa has 85% (estimated to be 24 million) of the world's camel population. More than 60% of the world's camelpopulation is found in the Horn of Africa region. In Sudan camels are classified into pack and riding (Babiker, 2000). They are concentrated in ButanaState of North –eastern Sudan, in Northern kordfan and Darfur states (in the Northern dry land of Sudan). According to Ageb (1995) Butana camel population is about 750,000 head representing 25% of Sudanese camel population in an area representing 4% of Sudan land, and the main camel owning tribes in east of Sudan are Kawhla, Rashida, Lahawiene, Shakria, Hedendwa, and BiniAmir. Sudan is home to some of the most well –Known camel nomads tribes, the Kababish, Shukria, Hadendowa and other tribal groups in Sudan breed distinctive types of camels (Mason and Mule, 1960) .

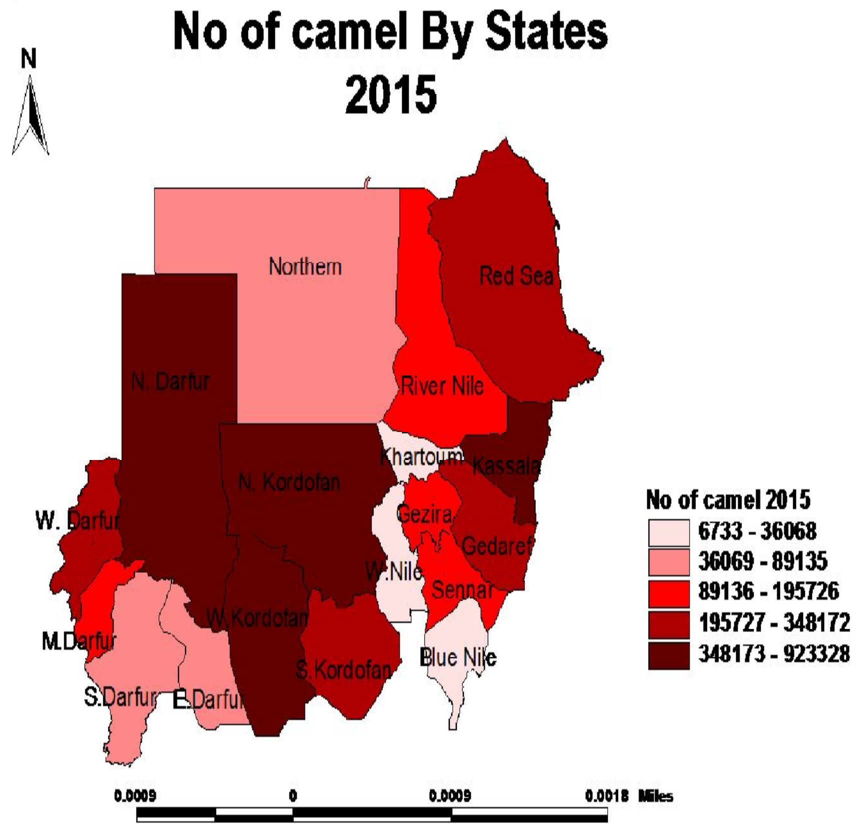

Figure.1-**Source:** Ministry of Animal Resources And fisheries (2015).

1-2: *Toxoplasma gondii*

Toxoplasma gondii is one of the most successful parasites worldwide, capable of infecting virtually all warm blooded animals. It is estimated that up to one third of the world's human population is also infected (Tenter*et al.,* 2000). *Toxoplasma gondii* is a world-wide, polyxenous, intracellular coccidian parasite, has a facultative heteroxenous life cycle and can probably infect all warm-blooded animals (mammals, birds and humans). All of these facts taken together indicate that toxoplasmosis is a neglected zoonosis in many ways and needs more attention.The parasiteis also play a major cause of abortion in sheep and goats, and thereby the cause of substantial economic losses (Innes *et al.,* 2009a). The first description of *Toxoplasma gondii*merozoites in the spleen, liver and blood of gondii, North African rodents, was given by Nicolle and Manceaux (1908). The causative,agent of toxoplasmosis is a coccidian parasite*, Toxoplasma gondii*so named because the organism was first identified in an African rodent called *a gondi*. The word toxoplasma was derived from the Greek word "*toxon*" or arc, reflecting the shape of the parasite by light microscopy (Nath and Sinai, 2003).*T. gondii*is the only known member of the genus *Toxoplasma*, but as a protozoan parasite it belongs to the phylum of Apicomplexa together with other coccidianspecies, piroplasms and plasmodia. *T. gondii*is present in all geographical regions of the world.

1-3: Classification of *Toxoplasma*

Members of the genus *Toxoplasma* belong to the kingdom *Protista,* sub-kingdom*Protozoa* and phylum *Sporozoa*or *Apicomplexa*(Levine *et al.,* 1980). They are further classified as belonging to the class *Sporozoea,* Subclass *Coccidea,* Superorder *Eucoccidia,*Order *Eucoccidida, Suborder Eimeriina*and Family *Eimeriidae*(Mehlhom and Walldorf, 1988).

*Toxoplasma gondii*and other species of the Phylum *Sporozoa*such as *Plasmodium* spp. are characterized by the occurrence of the name-giving sporocysts(and/or oocysts) which produce the infectious sporozoites. In Tablel is listed the various tissue-forming coccidia including *T. gondii.* The life cycle of each of the listed species comprises a regular alternation of different sexual and/ or asexual generations.

Morphologically, the Sporozoa are a relatively uniform group with each possessing a typical apical complex. Due to the presence of this fine-structural feature, several groups of parasites were added to or excluded from the sporozoa. This led to the systematic concept ofLevine *et al.* (1980) which was however not generally accepted and has since been modified (Mehlhom and Walldorf, 1988).Apart from only a few species, the coccidia are intracellular parasites which have a life cycle consisting of three phases: *Schizogony*(asexual multiplication), *Gamegony*(sexual phase, which proceeds in general as oogamy with macrogametes and microgametes) and *Sporogony*in which the zygote initiates another asexual reproduction leading to theproduction of numerous infectious sporozoites. In species that are transmitted by the fecal-oral route, the sporozoites always include resistant stages (oocystand/sporocysts).

1-4: Taxonomy:

Toxoplasma gondii, is classified according to Ferguson (2002) as follows:

Kingdom	Animalia
Sub Kingdom	Protozoa
Phylum	Apicomplexa
Class	Sporozoea
Subclass	Coccidia
Order	Eucoccidea
Suborder	Eimeriina
Family	Sarcocystidae
Genus	*Toxoplasma*
Species	*Gondii*

1-5: Toxoplasmosis or (*T. gondii*infection):

Toxoplasmosis is a worldwide zoonotic disease caused by *T.gondii*(Weiss and Kim, 2007; Tenter, 2009). Toxoplasmosis isone of the major causes of infectious reproductive failure(Freyre*et al.,* 1999; Weiss and Kim, 2007). It causeseconomic losses in livestock and serious public healthimplications, due to severe neurological and reproductivesymptoms (Tenter*et al.,* 2000). There is a wide spectrum ofthe diseases associated with *T. gondii*infection which isdependent on: the host species; the immune status of thehost and the virulence of the particular strain of the parasite(Innes, 2010). *T. gondii*infection is a common congenitaldisease in humans and domestic animals. The prevalence ofthe disease showed considerable geographical

variation(Tenter*et al.,* 2000; Weiss and Kim, 2007; Ortega-Mora *etal.,* 2007; Tenter, 2009).

1-6:*Toxoplasma* strains:

T.gondii has unusual structure dominated by three clonal lineages that predominate in North America and Europe. Molecular genotyping has shown that approximately 90% of *T.gondii* isolates that have been analyzed can be classified into three lineage (Type I, II, III) (Zhou *et al.* , 2009).Type I is highly virulent in murine, whereas type II is relatively avirulent and type III is of intermediate virulence in mice (Peyron *et al,* . 2006).

1-7: Morphology of *T. gondii*:

T. gondii parasite occurs inthree morphological stages, they are: trophozoite, tissue cyst and oocyst. The trophoziote and tissue cyst represent stages in asexual multiplication (Schizogony). While the oocyst is formed by sexual reproduction (gametogony or Sporogony)

1-8-1: Tachyzoites stage:

Measure approximately (4-8 μm) in length and(2-3 μm) in width and generally crescent shape, with apical complex at theblunt end of the parasite (Dubey, 2004).It requires an intracellular habitat to survive and multiply, despite having its own golgi apparatus, ribosomes and mitochondria (Montoya *et al*., 2005) .Tachyzoites have a crescent shape and are approximately 2x6μm. Their anterior (conoidal) ends are pointed and their posterior ends are round. They have a pellicle (outer covering), polar ring, conoid, ropthries, Micronemes, mithocondria, subpellicular microtubules, endoplasmic retriculum, Golgi apparatus, ribosomes, rough surface endoplasmic reticulum, micropore, and a well defined nucleus. The nucleus is usually situated toward the posterior end or in the central area of the cell. Chromatin is distributed in clumps

throughout the nucleus and the nucleolus is usually located centrally within the nucleus (Sheffield and Melton, 1968).Tachyzoites multiply rapidly to destroy the host cell within 48 hours, they replicate with a generation time of 6 to 9 h in vitro until exiting the cell to infect neighboring cells, usually after accumulating 64 to 128 parasites per cell (Coppens and Joiner, 2001).In the tachyzoite, the conoid defines the apical end of the parasite and is thoughtto be associated with the penetration of the host cell. Micronemes, rhoptries and dense granules are the three major secretory organelles, found predominantly at the apical end of the parasite. Microneme proteins are released very early in the invasion process, facilitating host-cell binding and gliding motility. Rhoptry proteins are also releasedduring invasion, and can be detected within the lumen and membrane of the newly generated parasitophorous vacuole (PV). Dense granule proteins are released during and after the formation of the PV, modifying the PV environment for intracellular survival and replication of the parasite (Ajioka *et al*, 2001) Tachyzoites are not resistant to gastric secretions and are thus much less infection via the oral route than either oocysts or bradyzoites (Weiss *et al*., 2000).
Tachyzoite are associated with the acute disease phase (Fig2) .

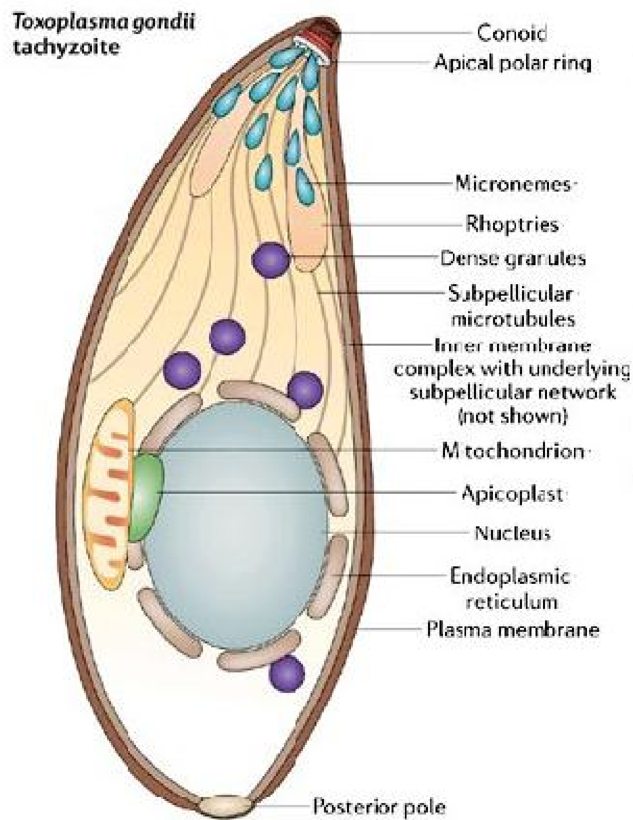

Figure 2:- Showing schematic Ultrastructure of *T . gondii* tachyzoite (James *et al.* , 2001) .

1-8-2: Bradyzoites stage:-

Bradyzoites are also called cystziotes.The quiescent bradyzoites or cystizoites that occupy cysts in infected tissue. Bradyzoite observed during disease chronic stage (Tobin *et al*., 2010). The spheroidal cyst, had very resistance membrane contain as few as 50 and up to as several thousand bradyzoites which has only come into contact with host cells, if the cyst exposure to the stress will give ruptures (Ferguson, 2002). Tissue cysts are formed most commonly in the brain, liver and muscles (Hakan *et al*., 2010).Comparing to tachyzoites, this is a slowly replicating life stage which forms cysts during a chronic phase. Like tachyzoites, bradyzoites remain intracellular and divide of by a unique binary fission termed endodyogeny (Weiss *et al*., 2000). Bradyzoites

differ only slightly structurally from tachyzoites. They are slender, smaller in size, and usually have a nucleus situated toward the posterior end, whereas the nucleus in tachyzoites is more centrally located. They contain several glycogen granules which stain red with periodic acid-schiff (PAS) reagent; these are either indiscrete particles or absent in tachyzoites. Biologically, bradyzoites are less susceptible todestruction by proteolytic enzymes than tachyzoites (Dubey and Beattie, 1988). The sizeoftissue cysts is variable, but on average a mature cyst is 50 to 70 µm and contains 1000-2000 crescent-shaped 7 by 1.5µm bradyzoites. Tissue cyst depends on cyst age, the host cell parasitized, the strain of *T. gondii* and the cytological method used formeasurement. Young and old cysts can be distinguished readily by their ultra structural 10 features. Degenerating cysts are often seen in the brains of mice with chronic toxoplasmosis (Weiss *et al*., 2000). Bradyzoites develop in cysts within host cells in a variety of tissues, but they are common in neural and muscular tissues such as brain, heart, skeletal muscle and retina. Cysts are not static structures; they regularly break down or rupture host cells and reinvade others (Weiss *et al*., 2000). When tissue cysts rupture, however, they elicit a strong inflammatory response resulting in the formation of glial nodules in the brains ofchronically infected hosts.

The tissue cyst wall is elastic, thin (<0.5µm), and argyrophylic and encloses hundreds of crescent-shaped slender bradyzoites. The bradyzoites are about 7x1.5µm(Mehlhorn and Frenkel, 1980). Initially, the tissue cyst develops in the host cell cytoplasm and its wall is intimately associated with the host cell endoplasmic reticulumand mithocondria and the cyst wall is partly of host origin. Some bradyzoites may degenerate in tissue cysts, especially in older cysts (Pavesio *et al*., 1992). The bradyzoite has a nucleus situated toward the posterior end. It

contains electron dense rhoptries, and several glycogen granules, which are either in discrete particles or absent in tachyzoites.The prepatent period in cats following infection by bradyzoites is shorter than that following infection with tachyzoites (Dubey and Fenkel, 1976). Tissue cysts are more numerous in animals in the chronic stage of infection after the host has acquiredimmunity than in animals in the acute stage of infection. However, tissue cysts have been observed in mice infected for only 3 days (Dubey and Frenkel, 1976) and in cells in culture systems devoid of known immune factors (Hoff *et al*., 1977). Dubey (1993)reported that it is possible that development of functional immunity and the formation of tissue cysts are coincidental (Fig 3).

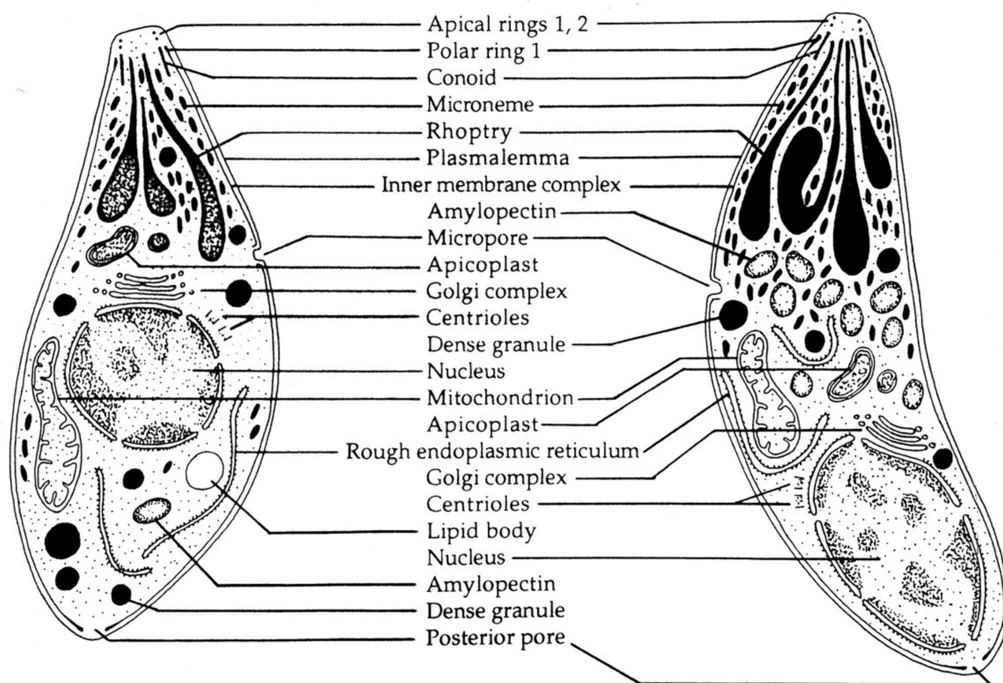

Figure 3: Schematic drawings of a tachyzoite (left) and a bradyzoite (right) of *T. gondii* . The drawings are composited of electron micrographs (Dubey *et al*., 1998).

1-8-3: Oocysts stage :

Oocystes develop only in definitive hosts – in the intestine of cats andother felines when cats get infected by ingestion of either tissue cysts orOocysts. The parasites develop in the intestinal epithelial cells, where bothSchizogony and gametogony take place. Male and female gametocytesdevelop and after Fertilization, the zygote gets surrounded by a thin, butextremely resistance wall.Oocysts are highly resistant to environmental conditions and can remain infectious for as long 18 months in water or warm moist soils (Johnson, 2009; Jones and Dubey, 2010).They do not survive well in a rid cool climates, they can remain infectious for weeks in body fluids at room temperature, and in meat for as long as the meat is edible and uncooked (CDC, 2008).The sporozoite is similar to the tachyzoite, except that there is an abundance of micronemes, rhoptries, and amylopectin granules in the former. Sporozoites are (2 by 6-8 μm) in size with a subterminal nucleus (Tenter *et al*., 2001)(Fig4).

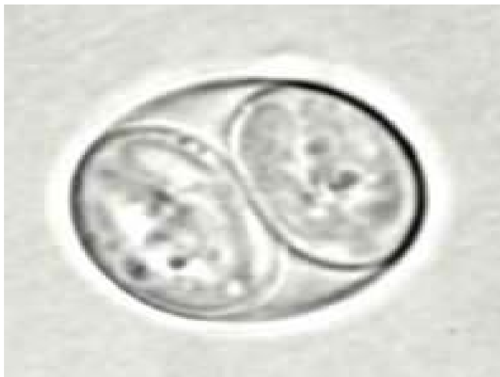

Figure 4: Sporulated oocyst of *T. gondii* (Pappas and Wordrop , 2004).

1-9: The life cycle and transmission of *T. gondii*

The life cycle of *T.gondii* includes both sexual and asexual multiplication. Sexual multiplication of *T. gondii* takes place in the gut of felines, making them the definitive hosts. Many feline species have been shown capable definitive hosts (Dubey, 2009a). If a cat ingests a *T. gondii* infected animal or meat, bradyzoites are released from the tissue cysts contained in their meal. In the previously uninfected cat, these bradyzoites invade epithelial cells of the cat's small intestine, where they start multiplying +asexually. After five asexual stages of multiplication gametogony begins. Female macrogamonts and male microgamonts are formed, and upon fertilization of the macrogamete by a microgamete, a zygote and an oocyst wall are formed. The nucleus divides twice and two sporoblasts (each with two nuclei) are formed. As the epithelial cells rupture, millions of oocysts containing sporoblasts are
discharged into the intestinal lumen of the cat and eventually shed into the environment or cat litter box. Depending on temperature and humidity these sporoblastssporulate within 1 to 5 days to become infectious sporozoites with a haploid DNA content (4 sporozoites per sporoblast). Sporulatedoocysts are infectious to cats (leading to another round of sexual multiplication) (Dubey, 1996a), but even more so to an unequalled range of intermediate hosts: Probably all warm-blooded animals can be infected. If an intermediate host ingests oocystssporozoites will be released into the gut lumen and pass through the gut epithelium to enter cells in the lamina propria. In case an intermediate host ingests tissue cysts the released bradyzoites behave similarly to these sporozoites: Both sporozoites and bradyzoites transform into tachyzoites that enter a host cell where they divide rapidly until the cell bursts. Next, they continue to infect neighbouring cells.

Tachyzoites disseminate through the body by the circulation mostly intracellularly in leucocytes (Unno*et al*., 2008), and finally enter various nucleated cells, but especially those in nervous and muscle tissue, where they transform into slowly dividing bradyzoites surrounded by a cyst wall. The fate of these tissue cysts is not entirely clear. Tissue cysts seem to remain present lifelong in most hosts, although individual cysts are thought to rupture occasionally. This occasional cyst rupture is considered responsible for the persistence of antibodies in the host, because the released bradyzoites could stimulate the immune response in the immune competent host. Released bradyzoites transforming back into rapidly-dividing tachyzoites could explain the reactivation resulting in clinical symptoms or even fatal toxoplasmosis in immune-compromised.individuals.Althoughintermediate hosts do not shed *T. gondii* they are infectious via carnivorism. Both felines and intermediate hosts are susceptible to infection via tissue cysts, which means that intermediate hosts are also infectious to each other. This ability to complete a cycle without the necessity to pass through the definitive host is quite unique in the world of parasites. Another interesting characteristic of *T. gondii* is the ability to change the behavior of rodents, causing them to specificallyloose their aversion for cats (*Vyaset al.,* 2007).

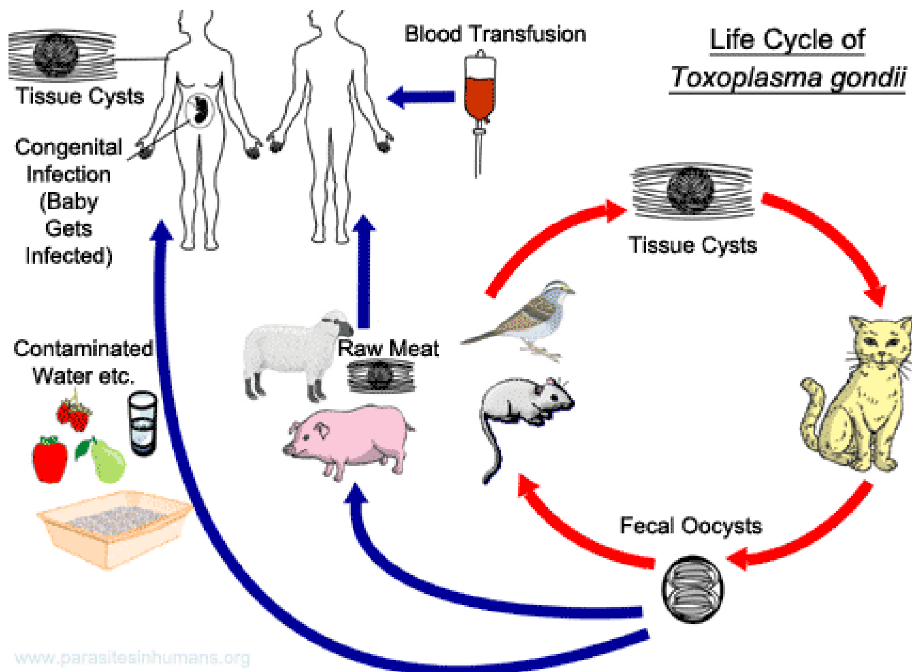

Figure.5: Source :visitwww.cdc.gov/parasites/toxoplasmosis.

T. gondii can be transmitted horizontally and vertically (Taylor *et al.,* 2007; Tenter, 2009). Transmission occurs following ingestion of food, feedstuff and water contaminated with Sporulatedoocyst or/and tachyzoites and bradyzoites (Tenter*et al*., 2000; Dubey, 2004; Weiss andKim, 2007; Taylor et al., 2007; Tenter, 2009).Transmission occurs following ingestion of sporulatedoocysts or bradyzoites within cysts present in the tissues of numerous food animals. The frequency of infection is extremely variable in the different regions of the world. Seroprevalence in the human population rangesd from 0% to 90%(Deuby*et al,*1988) and infection is more common in warm climates and in low-lying areas than in cold climates and mountainous regions, where conditionsfor sporulation and survival of oocysts are less favourable(Desmonts,1961) The prevalence of *T. gondii* infection also varies between ethnic groups, and it is thought that this is largely due to sanitary and cooking habits rather than genetic differences. A seroprevalence of 80% has been reported from Paris where undercooked

meat is often consumed(Desmonts,1961) . Lower seroprevalences (10–40%) have been reported in countries from Southeast Asia where meat is cooked thoroughly (Zuber and Jaquier,*et al*,1995).Transmission of *T. gondii* occurs byingestion of sporulatedoocystsorbradyzoites in tissues of food-producinganimals. It also occurs transplacentaly, by blood transfusion or aerosols (Dubey, 1994;Esteban-Redondo *et al*., 1999; Tenter*et al*.,2000).

1-10: Epidemiology of Toxoplasmosis:

T. gondii infection occurs all over the world and the mainreason of its widespread is the lack of host specificity (Tenter*et al*., 2000). It is the common parasitic zoonoses worldwide. The cat plays a central role in the epidemiology of toxoplasmosis. Epidemiological investigations in USA and elsewhere indicated that 60% of cats are serologically positive to Toxoplasma antigen. About three to ten daysfollowing infection, cats start to shed oocysts for 2-3 weeks with peak output of ten Millions of oocysts at 6-8 days p.i(Dubey and Frenkel, 1972; Dubey and Bettie, 1988). Santos et al., (2009) considered dog as a common source of T. gondii infection for both, humans and animals. Infection in animals is associated with feed or grazing range land contaminated with sporulatedoocysts (Innes *etal*., 2009) and transplacentally (Dubey, 1994). Additionally, seroprevalenceof toxoplasmosis in free-ranging chickens is a good indicator of general prevalence of *T. gondii*oocysts in the soil (Dubey*et al*., 2005). According to Alexander and Stimson (1988) and Van der Puije*et al*., (2000), female animals are generally more susceptible to protozoan infection than male. Oral application of tachyzoites might have cause an infection (Dubey, 1998c; Sacks *et al*., 1982).

1-11: Animal Toxoplasmosis:

Similarly to *T. gondii* infection in humans, the infection usually remains asymptomatic inmost other species.There are, however, some species in which *T. gondii* infection can have serious consequences(Innes, 1997). For Australian marsupials (Canfield *et al*., 1990) and New World monkeys(Epiphanio et al., 2003) primary infection with *T. gondii* is often fatal. Female Pallas catscan transmit *T. gondii* to their offspring when chronically infected, which often leadsto fatal toxoplasmosis in kittens, and is a common cause for failure of captive breedingprograms (Kenny *et al*., 2002). Some pigeon breeds or species are highly susceptible to clinical toxoplasmosis, and canaries show an unusually severe eye infection with symptoms varying from blindness to complete ocular atrophy (Dubey, 2002). The high susceptibility formarsupials, New World monkeys and Pallas cats is considered a result from their evolutionarydevelopment separated from cats and *T. gondii*:felines were first introduced in Australia bysettlers in the late 18th century, New World monkeys live high up in trees, and the exposure to*T. gondii* in wild Pallas cats and other hosts in Mongolia is very low (Brown et al., 2005).Camel and cattle are from the most useful domestic food animals important to the economy of many countries particularly in the Africa and Middle East Regions. Beside their social and economic status, they play a very important role in the national income, as they are an important source of meat, milk and hide and constitute a major item in the livestock foreign trade list (Schwartz *et al*., 1992; Schoonman*et al*., 2010). Similarly to *T. gondii* infection in humans, the infection usually remains asymptomatic in most other species. Congenital transmission resulting in abortion or offspring born with abnormalities is the most commonly observed problem. Especially sheep and goats are susceptible

to congenital toxoplasmosis and in these animals *T. gondii* is an important cause of abortion (Buxton *et al*., 2007; Dubey, 2009c). To prevent these abortions, an attenuated live vaccine, based on a strain (S48) that has lost its ability to develop tissue cysts by continuous passage in mice (Buxton, 1993; Wastling*et al.,* 1993), has been developed and is commercially available (Toxovax®). In addition, *T. gondii* ranked second on a list of prioritized emerging zoonoses in The Netherlands (Havelaar*et al*., 2010). The parasite is also a major cause of abortion in sheep and goats, and thereby the cause of substantial economic losses (Innes *et al.,* 2009a).

1-12: Transmission of *T.gondii*

Infection with *T. gondii* can occur through four routes of transmission, including :

(1) congenitally by transmission of tachyzoites during primary infection of the mother;

(2) by ingestion of food or water contaminated with oocysts;

(3) by ingestion of raw or undercooked meat containing the bradyzoite form in tissue cysts (Dubey, 1996), or

(4) by receiving blood or tissues with tachyzoites or bradyzoites.

camels acquire *T. gondii* infection through ingestion or inhalation of sporulatedoocysts that are shed by cats or wild felids in the environment(Elamin 1992). The prevalence of *T. gondii* infection in camels varies widely depending on the localities of the world(Shaapan *et al*, 2008) ,ranging from 3.12% in Iran(Dehkordi,*et al*,2013) to 90.90% in Turkey(Utuk , *et al*,2012).The human infective dose for *T. gondii* is not established but extrapolation fromanimal studies suggests a dose of less than 104 organisms (Remington *et al*., 1995).The relative importance of these different sources of infection is not defined andmay vary from one

region to another, depending on diet, culinary methods, prevalence of infected cats (Remington *et al*., 1995), farming techniques (Bustamante and Suarez. 2000; Remington *et al*., 1995), climatic conditions such as temperature, rainfall and humidity (Mensah *et al*., 2000).*Transmission by Oocysts.-* Cats and all felidaes are fundamental for thetransmission of *T. gondii*, because they are the only species capable of shedding oocysts in their feces. Felidae excrete *T. gondii* oocysts in feces 3 to 10 days after ingesting bradyzoites, ≥ 18 days after ingesting of sporulated oocysts, and ≥ 13 days after ingesting tachyzoites (Dubey, 1998a). Those must sporulate outside the body of the host. The sporulation process which usually takes from 1 to 5 days depends on temperature, moisture, and other environmental conditions. As a rule, the duration of excretion is from 1 to 3 weeks and is rarely repeated, and may be re-stimulated by malnutrition by *Isospora felis*, or by administration of cortisone (Dubey and Beattie, 1988). Transmission can occur by consumption of water, fruit, or vegetables contaminated with oocysts shed in the feces of infected cats.

1-13: Human Toxoplasmosis:

Toxoplasmosis is one of the most common infections in humans worldwide (Tenter*et al.,* 2000; Tenter, 2009).About 3-80% of healthy adults have been exposed to the parasite (Weiss and Kim, 2007). Other serological studies worldwide (Ira *et al.,*2009) showed that over one third of the human population had antibody against *T. gondii.* This lends to support the importance of the zoonotic view of toxoplasmosis,particularly in pregnant women and immune compromised patients (Tenter*et al.,* 2000). The disease is one of the most prevalent zoonotic parasitic infections. About two billion people throughout the world are infected, with considerable geographical

variation. Human toxoplasmosis infection attributed to man use of animals as pets or for food (Tenter*et al.,* 2000; Tenter, 2009). Its transmission to humans is usually attributed to ingestion of undercooked or raw meat or primary offal (viscera) from infected livestock (Tenter, 2009; Ciamak-Ghazaoi, 2005; Tenter*et al.,* 2000; El Hassan *et al.,* 1991). The fore author stated that, the infection rate in livestock is an important predictor of human toxoplasmosis risk. Since contaminated meat is a significant source of infection in humans, it is particularly important to ensure continuous surveillance of*T.gondii*prevalence in animal species destined for human consumption (Ciamak-Ghazaoi, 2005; Tenter*et al.,* 2000). Unlike neosporosis, toxoplasmosis is a zoonotic disease and infection in people may result in severe disease in the developing foetus and in immune-compromised individual as well as eye disease in immuno-competent individuals following infection with *T. gondii*(Glanser*et al.,* 1992). Applying methods to estimate disease impact such as Disability Adjusted Life Years (DALYs) has shown toxoplasmosis to be one of the most significant food borne pathogens across the world (Kortbeek*et al.,* 2009).is the main source of human toxoplasmosis. Professional groups, such as abattoir workers, butchers and hunters may be infected during evisceration and handling of infected meat (Buzby and Roberts, 1997; Swai and Schoonman 2009). Blood transfusion or organ-transplantation from infected donor can also act as a source of infection (Schaffner, 2001).In immune-competent individuals the acute phase of the infection usually passesasymptomatically or signs are limited to a transient lymphadenopathy and mild fever-likesymptoms. Consequences such as encephalitis, pneumonitis, myocarditis or disseminatedinfections are highly unlikely. These consequences are, however, more common and maylead to fatal

toxoplasmosis in immune-compromised individuals, such as those receivingcorticosteroids or cytotoxic drugs, patients with hematological malignancies, transplantsor AIDS. Especially in AIDS patients *T. gondii*was an important cause of death, usually byencephalitis. However, since the introduction of highly active antiretroviral therapy (HAART)this is under control in the developed world. In immune-compromised individuals includinghaematopoietic stem cell transplant patients, toxoplasmosis is not necessarily caused bprimary infection. Recrudescence of a latent infection is a more common cause (Martino *etal*., 2000). However for patients receiving a solid organ, and especially a heart (muscle tissueis a predilection site for *T. gondii*), the risk of toxoplasmosis is highest in case the donor ispositive and the recipient is negative (Derouin and Pelloux, 2008). Toxoplasmosis in transplantpatients can be prevented by serological screening of donor and recipient and, if necessary, prophylactic treatment with co-trimoxazole (often already administered as prophylaxis forpneumocystosis) or pyrimethamine-sulphadioxide (Derouin and Pelloux, 2008).

1-14: Diagnosis of Toxoplasmosis:

Diagnosis of toxoplasmosis on clinical ground is usually difficult, and recourse must be made to the demonstration of either the organism or antibodies against it. The most convincing diagnosis is the isolation of the parasite by inoculation of suspect material into mice (Solusby, 1982). It has the disadvantage that unless the strain of toxoplasma is highly virulent, it requires three weeks before examination of the mice will yield recognizable *Toxoplasma* cysts (Uroquhart*et al*., 1996). In the main, diagnosis is based on a correlation of clinical and serological findings (Manal, 2003). The most useful and widely studied methods for

serodiagnosis are: dye test (Sabin and Feldman, 1948), indirect immunofluorescence antibody test (Remington *et al*., 1968), direct and indirect haemagglutination test (Jacobs and Lunde, 1957). More recently, ELISA test has been developed which is capable ofdetecting a recent infection by the estimation of IgM, as compared to IgG, antibody (Uroquhart*et al*., \996). Zhang and Wei (2001) reported that Modified Agglutination Test (MAT) and Latex Agglutination Test (LA T) could alternatively be used for the diagnosis of toxoplasmosis. Zhang *et al.* (1999) suggested that Immunosorbent Agglutination Assay (IgM, (SAGA) is a sensitive, specific, easy to perform, and is useful for mass screening and diagnosing recent toxoplasmosis infection or reactivation. Polymerase chain reaction (PCR)-based testing has become the preferred method for diagnosis, occasionally replacing tissue biopsy (Lewis *et al.,* 2002). Because of the lack of specific clinical manifestations during acute infection, *T gondii* is mainly a laboratory diagnosis. Diagnosis of *T.gondii* in animal and human is very difficult and recourse must be made demonstrate either the organism or the antibodies against it (Taylor *et al.,* 2007). The most convincing diagnostic tools include: serological tests, Bioassay, Histopathology, Immunohistochemistry and molecular techniques as well as tissue impression smear. The diagnosis of congenital toxoplasmosis can be performed by identifying the agent using histological slides and the polymerase chain reaction (PCR) with aborted fetuses and placentas (Pereira-Bueno *et al*., 2004).

1-15: Control of Toxoplasmosis in the Sudan:

In order to build control strategy, data on seroprevalence of *T. gondii* infection is considered as indicator of environment contamination with the parasite. However, there are very few works in animal and human toxoplasmosis in the Sudan. Though few, most of the available data was

on camel toxoplasmosis (Seri *et al*., 2003). The other available data onanimal toxoplasmosis in the Sudan are collected from few slaughtered animals (ZeinEldin *et al*., 1985; Khalil and Elrayah, 2011; Abdel-Hafez, 2013). Consistent data on human toxoplasmosis in the Sudan have not been reached (Musa 2008; Anon 2010; Siddig, 2010**)**. The shedding of oocysts in cat's faeces is generally considered to be a primary factor in the dissemination of the disease (Dubey, 2004). In view of this attempts were made to establish the possible degree of contact with cats in the surveyed populations.

1-16: Treatment of Toxoplasmosis:

Although it is very difficult, treatment of infected animals can reduce the economic losses due to toxoplasmosis in unvaccinated flocks. There are several drugs with good results such as decoquinate, monesin, clindamycine and sulphadimidine (Buxton *et al.*, 1996; Weiss and Kim, 2007; Giadinis*et al*., 2009). The use of Combinations of pyrimethrine and sulphadimidine, vacuiloprium and sulphadimidine or trimethoprim and sulphadimidine (Buxton *et al*., 1993b) were also recommended.

1-17: Seroprevalence of *T. gondii* Antibodies in Camels from the Sudan:

Most of the work on animal toxoplasmosis in the Sudan was conducted in camels (Seri *et al*., 2003). Since ZeinEldin (1985), there were many reports in camel toxoplasmosis in the Sudan (Bornstein and Musa 1987; Elamin*et al*., 1992; Manal 2003). The last study was that of Husna*et al.,* (2012) who reported seroprevalence of 44% in camels from Tumbool Slaughterhouse using LAT. Khalil and Elrayah (2011) reported seroprevalence of 20% in camel from El-Kadaro area using the same technique. The later authors reported antibody titres ranging from 1:8

(17.1%), 1:16 (2.9%) and 1:32 (0.0%). ZeinEldin (1985) reported 54% seroprevalence in slaughtered camels from Kordofan and central region of the Sudan. Their results showed widespread of Toxoplasma among meat producing animals in the Sudan.More widespread seroprevalence (61.7%) and (67%) of *T. gondii* in camels were reported by Manal (2003) and Elamin*et al*. (1992) respectively. Elamin*et al*. (1992) reported seroprevalence of 67% prevalence rate in pastoral camels from Butana plains using LAT (Two fold dilutions ranging from 1:8 to 1:256). The prevalence rate increased significantly with age (74.2% in camels aged over 7 years). The prevalence rate of seropositivity decreased proportionally with the level of serum dilution. At dilution of 1:32 and above, the prevalence rate was 25.9%. There were no sex linked differences ($p>0.05$) in seropositivity. The overall prevalence among female was 22.7% and male 29.1% camels. Using LAT, Manal and Majid (2008) reported over all prevalence of 51.3% of anti-*T. gondii* antibodies from sera of calf-camels with diarrhoea from different parts of the Sudan.

1-18: Prevention Measures:

Food and water should be kept away from cat's faeces and any contaminative environment (Dubey, 1991). Other control measures include minimizing number of cats shedding oocysts (Dubey and Jones, 2008) by limiting the breeding of cats. In addition to adequate and continuous control programs of stray and feral cats can reduce the risk of *T.gondii*transmission, beside controlling the rodent's population (Buxton and Rodger, 2008). Education of farmers on the principles of the route of infection and measures that reduce the prevalence of clinical cases aswell as vaccination will reduce animal and human toxoplasmosis (Buxton *et al.,* 2007; Ogendi*et al.,* 2013). Prevention measures for *T.*

*gondii*infection in humans have not been reached (Bout *et al.,* 2002; Camossi*et al.,* 2013). Heating of meat to 67oC or higher is considered sufficient to immediately kill tissue cysts (Dubey 2000).

CHAPTER TWO

Materials and Methods

2-1: Study area and survey :

Gedarif state is situated between long: 33°-45 and 36°-45 East and lat 12°-45 and 16°-00 North and have borders with Sinar, AljezeraKassala, Khartoum and Nile state. With Ethiopia in frontiers .Gedarif state is an area of 71,621 km square.. The study also included part of Butana area which is one of the most important grazing areas and is situated in the north part of Gedarif state .

2-2:Topography of Study Area:

2-2-1: Soil:

The State is a flat plain, with almost no relief other than small, scattered hills and seasonally flowing watercourses. The principal soil type throughout the State is vertisols. Other soils, which occupy small fractions of the area, include a mixture of alluvial clays, silts, and sands of varying depths on the banks of the seasonal rivers, and rocks, stones and gravels in some sites.

2-2-2: Climate:

Gedarif state is a large state (area =71,621 square km) with varying land use and socio economic activities and with varying climatic conditions that range from semidesert in the north to wet monsoon type of climate in the extreme south. The climate in Gedarif state can be divided as follows:

(a) Arid to semi arid zone in the northern part of the state with mean maximum summer temperatures of 37c .and minimum temperature of 13 c in winter.

(b) The dry to wet monsoon zones with maximum summer temperatures of 35c., and minimum winter temperatures of 18 c. Most of the rain fall in Gedarif state occurs in summer. The range mean annual rainfall ranges from 200 mm in the north to 800 mm in the south parts of the state from July to the end of September.

2-2-3:Vegetation :

Gedarif State has savannah vegetation and the land is characterized by tall trees and open forest of Talih (Acacia Seyal) and gum Arabic (Acacia Senegal). Most of the trees at present are cleared and burnt to provide more land for durra (sorghum) cultivation and charcoal production. Since time immemorial the Butana in north part of Gedarif state has been know to have excellent pastures (Akhtar, 1994) and the best grazing land in Sudan. The grasses atribes from adjacent as well as far away States use it as grazing land during and after the rainy season. In addition a lot of varieties of grasses and other plants are available.re palatable with high nutritional value for animals. This is why many nomadic July to the end of September.

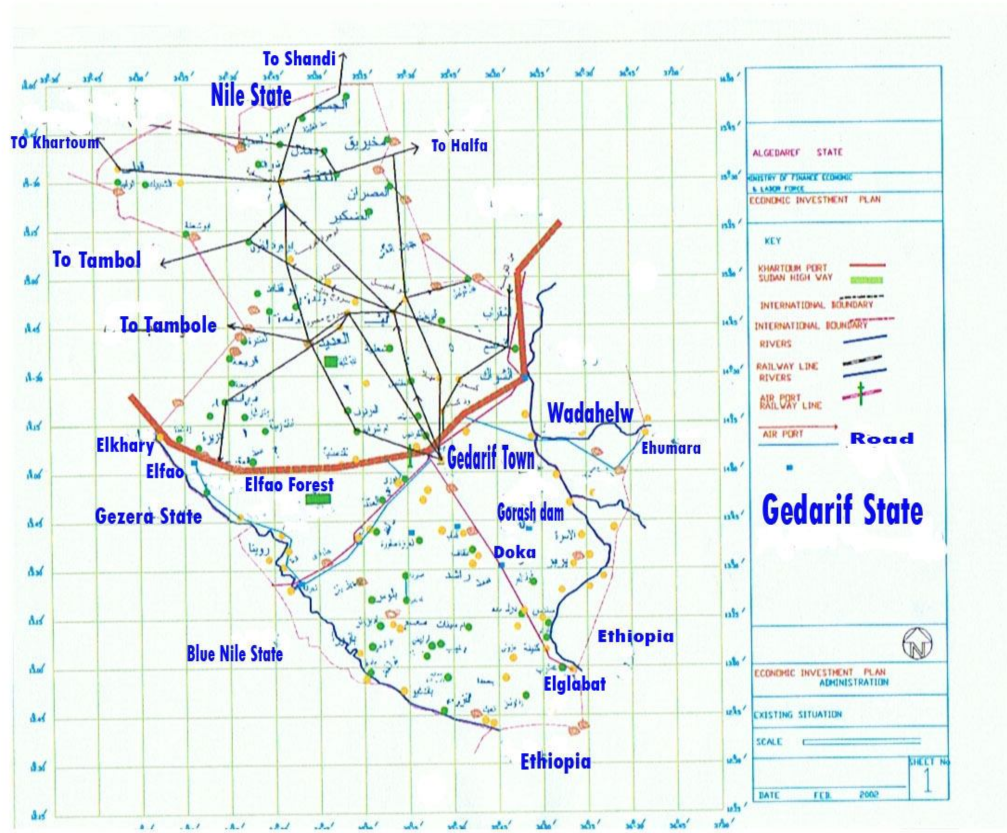

Figure 6-**Al Gedarif State Ministry of Urban Planning and Public Utilities (2016)**

2-4: Samples collection

Three hundred blood samples (represent 161 males and 139 females) were collected by jugular vein puncturein sterile tubes without anti-coagulant and labeledsamples were kept at−20∘C and stored for further analysis.Serumsamples collected from different localaties in Gedarif state (60samples Wasat AL- Gedarif-60 samples AL-shwak-100 samples AL- Rahad and80 samples AL-Butana)the samples were collected from different localaties ,during the period from 2015-2016 and were examined at the Gedarif veterinary research labolatery.

2-5: Laboratory kits:-

2-5-1: Latex agglutination test (LAT)

The serum samples and Toxoplasma antigen (Spinreact, S.A./S.A.U., Ctra. Santa Coloma, Spain) were kept one hour in room temperature before beginning of the test. A total of 50 µl of each serum to be tested was placed on a LAT plate. Then the vial of antigen was shacking gently and 25 µl of antigen was put beside each of the sera. The antigens and the serum were mixed on the plate with a stirrer and spread over the entire circle. Then the plate was rotated manually for 4 minutes and the reading was taken immediately. Any agglutination was considered as positive, whereas no reaction (negative) was indicated as the absence of Toxoplasma antibody in the sera.

2-5-2: Enzyme-linked immunosorbentassayELISA.

ELISA uses crude soluble antigens adsorbed onto the walls of microtiter plate wells and the antigen-antibody reaction is enhanced by the addition of a secondary enzyme-linked antibody, and the reaction can be assessed objectively by quantization of the colour that developed by an ELISA reader.ELISA Technique. Commercial iELISA kits (Ruminant SerumToxoplasmosis) for detection of anti-*T. gondii*antibodies were purchased from Lsivet (Nouzilly, France). Positive serum samples will present yellow colour; the colour visualized in each well is proportional to the titer of antibody specific to *T. gondii*present in the diluted sample (1/400). All samples which had antibody titer ≥30 were considered positive.

2-6: Statistical Analysis.

The serological results and other information gathered during this investigation such as location, sex, and age of the sampled animals were edited and analyzed statistically using statistical package(SPSS version 21). To identify the association of the risk factors with the chi-square($\chi 2$ test) and one-way ANOVA were used. The statistical significance level used was $p \leq 0.05$.

CHAPTER THREE

Result:

In total 300 camels sera were tested from different localities in Gedarif state(Wasat AL-Gedarif, AL-Shwak, AL-RahadandAL-Butana) using the latex agglutination test, the result of this test in camels 49.7%(149camels)and 52.3%and 47.7% of infection was detected in females and males respectively. Table 2.show a seroreactivity correlated with significancebetween the surveyed locations ($P<0.05$).

3-1: Seroprevelence of *Toxoplasma gondii*incamels by using Latex agglutination test 3-1: in localities (LAT) in Gedarif state

The positive sample with LAT test was149 and the percentage of infection in each locality wereAL-Shwak16.77% ,AL-Butana32.21%,Wasat AL-Gedarif6.71%and AL-Rahad44.29%. High infection in AL-Rahad locality 44.29%. There is significant differences between different localities.

Table (2) Seroprevalence of *Toxoplasma gondii* in camels by LAT test in localities in Gedarif State

			Sex		Total
			Female	Male	
Localities	AL-Shwak	Count	28	32	60
		% within Localities	46.7%	53.3%	100.0%
		% within Sex	20.1%	19.9%	20.0%
	AL-Butana	Count	34	46	80
		% within Localities	42.5%	57.5%	100.0%
		% within Sex	24.5%	28.6%	26.7%
	Wasat AL-Gadaref	Count	18	42	60
		% within Localities	30.0%	70.0%	100.0%
		% within Sex	12.9%	26.1%	20.0%
	AL-Rahad	Count	59	41	100
		% within Localities	59.0%	41.0%	100.0%
		% within Sex	42.4%	25.5%	33.3%
Total		Count	139	161	300
		% within Localities	46.3%	53.7%	100.0%
		% within Sex	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	13.365[a]	3	.004
Likelihood Ratio	13.591	3	.004
Linear-by-Linear Association	2.437	1	.118
N of Valid Cases	300		

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 27.80.

3-2: Seropositivity to *Toxoplasma gondii*using Latex agglutination test in camels at different Sex:

Positive sample were 78 in females(52.3%) and in males 71 (47.7%)

Results of this test showed that, percent of infection in female animals(52.3%) was more higher than that in male animals(47.7%). There was significant differences ($P<0.05$) between males and females infection percentage (Table3).

Table(3)Seropositivity to *Toxoplasma gondii*using Latex agglutination test in camels at different Sex in Gedarif state

			Sex		Total
			Female	Male	
LAT-TEST	Positive	Count	78	71	149
		% within LAT-TEST	52.3%	47.7%	100.0%
		% within Sex	56.1%	44.1%	49.7%
	Negative	Count	61	90	151
		% within LAT-TEST	40.4%	59.6%	100.0%
		% within Sex	43.9%	55.9%	50.3%
Total		Count	139	161	300
		% within LAT-TEST	46.3%	53.7%	100.0%
		% within Sex	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.308[a]	1	.038		
Continuity Correction[b]	3.841	1	.050		
Likelihood Ratio	4.318	1	.038		
Fisher's Exact Test				.049	.025
Linear-by-Linear Association	4.294	1	.038		
N of Valid Cases	300				

3-3: Seropositivity to *Toxoplasma gondii*using Latex agglutination test in camels at different Age:

As shown in table 4 positive sample from camels at different agewere73 animal (49%),36 animals(24.2%),24 animals(16.1%),13animals(8.7%),3animals(2%) respectively according to their ages.

Table (4)Seropositivity to Toxoplasma gondiiusing Latex agglutination test in camels at different Age in Gedarif state:

			LAT-TEST		Total
			Positive	Negative	
Age sets	1-2	Count	73	69	142
		% within Age sets	51.4%	48.6%	100.0%
		% within LAT-TEST	49.0%	45.7%	47.3%
	3-4	Count	36	42	78
		% within Age sets	46.2%	53.8%	100.0%
		% within LAT-TEST	24.2%	27.8%	26.0%
	5-6	Count	24	24	48
		% within Age sets	50.0%	50.0%	100.0%
		% within LAT-TEST	16.1%	15.9%	16.0%
	7-8	Count	13	10	23
		% within Age sets	56.5%	43.5%	100.0%
		% within LAT-TEST	8.7%	6.6%	7.7%
	9-10	Count	3	6	9
		% within Age sets	33.3%	66.7%	100.0%
		% within LAT-TEST	2.0%	4.0%	3.0%
Total		Count	149	151	300
		% within Age sets	49.7%	50.3%	100.0%
		% within LAT-TEST	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.952[a]	4	.745
Likelihood Ratio	1.973	4	.741
Linear-by-Linear Association	.141	1	.708
N of Valid Cases	300		

a. 2 cells (20.0%) have expected count less than 5. The minimum expected count is 4.47.

3-4: ELISA Test:

ELISA was used to confirm the positive reactors for LAT which detects nonspecific antibodies for *T.gondii*,the result of ELISA revealed 44 (29.9%) positive cases149 animals as shown**in table6**.

3-5: Seroprevelence of *Toxoplasma gondii*in camels by using ELISA test in localities in Gedarif state:

Forty four serum sample from different localaties were assayed withELISA test detected this results **Wasat AL-Gedarif** 5 samples (50%),**AL-Shwak** 12 samples (48%),**AL-Rahad**18samples(28.1%)**andAL-Butan**a 9 samples (18.8%).There were significant statistical differences in the sero-prevalences of the surveyed localities, (Table 6).

Table (5)Seroprevalence of *Toxoplasma gondii* in camels by using ELISAtest in localities

			ELISA-TEST		Total
			Positive	Negative	
L0calities	ShwakAL-	Count	12	13	25
		% within Localities	48.0%	52.0%	100.0%
	ButanaAL-	Count	9	39	48
		% within localities	18.8%	81.3%	100.0%
	Wasat AL	Count	5	5	10
		% within Localities	50.0%	50.0%	100.0%
	Rahad	Count	18	46	64
		% within Localities	28.1%	71.9%	100.0%
Total		Count	44	103	147
		% within localities	29.9%	70.1%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.773[a]	3	.032
Likelihood Ratio	8.568	3	.036
Linear-by-Linear Association	.540	1	.463
N of Valid Cases	147		

a. 1 cells (12.5%) have expected count less than 5. The minimum expected count is 2.99

3-6: Seropositivity to *Toxoplasma gondii*using ELISA test in camels at different Age:

Serum samples positive with LAT test from camels according to their age were 1-2years72 samples , 3-4years36 samples ,5-6years24 samples ,7-8years12 samples ,9-10 years3 samples .where as positive samples with ELISA test from camels at different age were 23 samples (31.9%) ,12 samples(33.3%) ,5samples (20.8%) ,2 samples (16.7%) ,2 samples (66.7%)respectively. No significant difference between the sero-prevalence in the different age groups in the total samples was established (P<0.05).

Table (6)**serpositivity to** ***toxoplasma .gondii*** **using by ELISA test in camels at different Age in Gedarif state**

			ELISA-TEST		Total
			Positive	Negative	
Age sets	1-2	Count	23	49	72
		% within Age sets	31.9%	68.1%	100.0%
	3-4	Count	12	24	36
		% within Age sets	33.3%	66.7%	100.0%
	5-6	Count	5	19	24
		% within Age sets	20.8%	79.2%	100.0%
	7-8	Count	2	10	12
		% within Age sets	16.7%	83.3%	100.0%
	9-10	Count	2	1	3
		% within Age sets	66.7%	33.3%	100.0%
Total		Count	44	103	147
		% within Age sets	29.9%	70.1%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.222[a]	4	.377
Likelihood Ratio	4.190	4	.381
Linear-by-Linear Association	.344	1	.557
N of Valid Cases	147		

a. 3 cells (30.0%) have expected count less than 5. The minimum .expected count is .90

3-7: Seropositivity to *Toxoplasma gondii*using ELISA test in camels at differentSex in Gedarif state:

Allpositiveserum samples with ELISA were 44 samples , The number of females were 23(52.3%)and males were21(47.7%).No Significant different between them ($P<0.05$).

Table (7)Seropositivity to *Toxoplasma gondii* using ELISA test in camels at different sex in Gedarif state

			ELISA-TEST		Total
			Positive	Negative	
Sex	Female	Count	23	54	77
		% within Sex	29.9%	70.1%	100.0%
		% within ELISA-TEST	52.3%	52.4%	52.4%
	Male	Count	21	49	70
		% within Sex	30.0%	70.0%	100.0%
		% within ELISA-TEST	47.7%	47.6%	47.6%
Total		Count	44	103	147
		% within Sex	29.9%	70.1%	100.0%
		% within ELISA-TEST	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.000[a]	1	.986		
Continuity Correction[b]	.000	1	1.000		
Likelihood Ratio	.000	1	.986		
Fisher's Exact Test				1.000	.564
Linear-by-Linear Association	.000	1	.986		
N of Valid Cases	147				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 20.95.

b. Computed only for a 2x2 table

3-8: Comparison between the latex agglutination test (LAT) and ELISA test

The 300serum samples were tested by both LAT test and ELISA kits were compared. Result by LATtest was obtained on 149positive samples (49.7%) . But 44 sera were positive only by ELISA test (29.9%)in .There was significant differencebetween the two test. infection in males and females by two tests was same, LAT test and ELISA test showed infection in females 52.3%and in males 47.7%.There were no statistically significant differences in the ser-prevalence among the age groups shown as in table 3 and table 6.The positive samples with LAT test was149 seraand 25 sera (16.8%) in AL-Shwak, 48 sera (32.2%) in AL-Butana,10 sera (6.7%) in Wasat AL-Gedarif and 66 sera (44.29%)in (AL-Rahad). 44 positive serum sample by ELISA from positive result in LAT test were detected in this result (Wasat AL-Gedarif 5 sera (50%),AL-Shwak 12 sera (48%),AL- Rahad18 sera (27.3%) and AL-Butana 9 sera (18.8%) .There were significant statistical differences between different localities (Table 5,8)

Chapter four

Discussion

Until now insufficient data are available on camel toxoplasmosis in the world, and there have been limited number of reports on camel toxoplasmosis from Sudan.Camels for this study were chosen according to their role in human life in Gedarif State, Sudan. Camels were the source of meat and un-boiled milk or raw liver are consume by nomads around Gedarif State.

This result runs parallel with the finding of Husna *et al*., (2012) who reported seroprevalence of 44% in camels from Tumbool Slaughterhouse using LAT,andsimilarly in Ethiopia (49.6%) using DAT and(40.5%) ELISA tests (Gebremedhin,2014,).in Iraq (48%) where used complement fixation test CFT (Saleem and Fatohi,1993). and slaughtered animals (44.1%) in Tanta abattoir (Ibrahim *et al.*, 1997). The prevalence of this study was lower than seroprevelence for toxoplasmosis in camel using the LAT in the Butana plains, mid-eastern of Sudan was 67%(Elamin *et al*., 1992.) and prevelence for *Toxoplasmagondii* seropositivity was detect infection rate in Sudan using the LAT(61.7%) reported by Manal (2003).The higher reported was 20% in camel from El-Kadaro area using the same technique(Khalil and Elrayah ,2011) . Also the higher than results in Abu Dhabi (30.9%) in racing camels (Afzal and Sakkir, 1994).In Egypt reported 30.7% prevalence rate in camel (Shaapan and Fathia, 2008) . Much higher seroprevalence has been reported from Turkey (90.9%) by Utuk *et al*.(2012). Lower seroprevalenc was recorded earlier from Iran 3.12% Dehkordi,*et al* (2013), Saudi Arabia (6.5%) Al-Anazi (2011) ,(16%) Hussein,*et al*(1988), (13.1%), Al-Anazi (2012) United Arab Emirates (22.4%) Abu-Zeid (2002) and Egypt (17.4

- 31.4%) Shaapan,*et al*(2008),Hilali,*et al*(1998), Abu-Zeid,(2006).The variation in seroprevalence between the present study and African and Arabian countries might be due to the difference in density of cats and wild felids, climatic conditions(Dubey JP:2010), farming and management practices(Dubey JP2008), sample size(Khalil MK, Elrayah IE:2011), cut-off values and sensitivity difference in the serological tests employed (Dubey JP:2010,Al-Anazi AD:2011).

The results showed an almost no agreement between the two tests in detecting Toxoplasma infection in camel in gedarif state. However, LAT detected more positive samples than ELISA(Table 6)

Conclusions

In conclusion, this study suggests widespread infection with *T. gondii*among the Camels and that people are at a higher risk of acquiring *T. gondii*infection. Therefore, prevention of toxoplasmosis ,through biosecurity measures and education of pastoralists about the identified risks. Moreover, the role of animals in the epidemiology of human toxoplasmosis need more care in the country .

In order to build control strategies for reducing *T. gondii*infection in Sudanese people and their livestock.

RECOMMENDATIONS

- Feeding and watering troughs for domestic animals should be treated regularly and be protected from being accessed by cats to avoid contamination by *T. gondii*oocystsin the Sudan are widely infected with *T. gondii.*
- Study using various serologicaltests is recommended for accurate assessment ofcamel toxoplasmosis in the Sudan.
- Health education on the zoonotic significance of toxoplasmosis, mode of transmission and maintenance of a high standard of personal hygiene should be introduced .
- The study recommends the need for further researches in the whole country using different serological tests and to determine the impact of these findings on the human population.

References

Abdel Hafez, A. A. (2013). Epidemiological study on Toxoplasma gondii infection in Beja sheep in The Red Sea State, Sudan. PhD thesis, Sudan academy of Science. Acad Sci., 147 : 763-6.

Abu-Zeid Y, Enan M, Ahmed A, Shaheen H, Ramadan G, Al Shamsi U, Al Tayyari W: Genotyping of Toxoplasma gondii isolated from camels from AbuDhabi. The 6th Annual U.A.E. University Research Conference, 04/2005. 2006:24–26.

Abu-Zeid YA: Protein G ELISA for detection of antibodies against *Toxoplasma* SAG1 in dromedaries. *J Egypt SocParasitol*2002, 32:247–257.

Adison.(1934) .A short guide to the museum of Antiquities , KhartoumNationalHistoryMuseum .Khartoum .Sudan.

Afzal, M. and Sakkir, M. (1994). Rev. Sci. Tech., 13(3):787-92.

Ageb. H .M 1995.Camel in eastern Sudan .Diseases and potential productivity .Camel Res. Lab . ShowakGedarifState .Sudan .

Ajioka, J.W; J.M. Fitzpatrick, and C.P. Reitter. 2001. *Toxoplasma gondii* genomics: shedding light on pathogenesis and chemotherapy. Expert Reviews in Molecular Medicine 6:1-19.

Akhtar, M. 1994. Geo-ecosystem and pastoral degradation in the Butana. AnimalResearch Development 39:17-26.

Al Gedarif State Ministry of Urban Planning And Public Utilities (2016).

Al-Anazi AD: Prevalence of Neosporacaninum and Toxoplasma gondii antibodies in sera from camels (Camelusdromedarius) in RiyadhProvince, Saudi Arabia. J Egypt SocParasitol 2011, 41:245–250.

Alexander J. and Stimson W.H. (1988). Sex hormons and the course of parasitic infection. Parasitol. Today. 4(7): 189-193.

Animal Interface (ICOPHAI): One health for sustainabledevelopment. August 14-17 (2013), SummervilleResorts Porto de Galinhas, Brazil, pp. 320.

Anon (2010). Annual report 2006 - 2010: Private Medicallaboratories. Port Sudan, Red Sea State, Sudan.

Arko-Mensah, J., K.M. Bosompem, E.A. Canacoo, J.M. Wastling, and B.D. Akanmori. 2000. The seroprevalence of toxoplasmosis in pigs in Ghana. Acta Tropica 76: 27-31.

B **Faye*, O M A Abdelhadi**, A I Ahmed*** and S A Bakheit** .Livestock Research for Rural Development 23 (10) 2011

B.; Elzubeir A.E.A. and Yassin T.T.M. (1987). Survey for certain zoonotic diseases in camels in Sudan.Rev.Elev. Med. Vet. Pays. Trop., 40(3): 531- 533.

Bornstein S. and Musa B.E. (1987). Prevalenc of antibodies to some viral pathogens, *Brucellaabortus*and*Toxoplasmagondii*in serum from camels (*Camelusdromedarius*) in Sudan. J. Vet. Med. B.,34: 364-370.

Bout, D.T., Mevelec, M.N., Velge-Roussel, F., Dimier-Poisson, I., and Lebrun M. (2002). Prospects forHuman Toxoplasma Vaccine. Current drug targets-immune,endocrine&metabolic disorders, Bentham SciencePublishers Ltd. Pp. 227-234.

Brown, M., Lappin, M.R., Brown, J.L., Munkhtsog, B., Swanson, W.F. 2005. Exploring the ecologic basis for extreme susceptibility of

Pallas′ cats (Otocolobus manul) to fatal toxoplasmosis. J Wildl Dis 41, 691-700.

Bustamante J., and F. Suárez. 2000. Bustamante J, Suárez F. 2000. Estudio comparativo de frecuencias de toxoplasmosis en porcinos procedentes de crianza tecnificada y no tecnificada. Rev Inv Vet Perú 11: 32-39. doi: 10.15381/rivep.v11i1.6782 .

Buxton D, Maley SW, Wright SE, Rodger S, Bartley P, Innes EA. Toxo-plasma gondii and ovine toxoplasmosis: new aspects of an old story. Vet Parasitol. 2007;149(1-2):25-8.

Buxton D. (1998). Protozoan infections (Toxoplasmagondii, Neosporacaninum and Sarcocystes spp.) in sheepand goats: Recent advance. Vet. Res, 29 (3-4): 289-310.

Buxton D. and Rodger S. (2008). Toxoplasmosis andneosporosis. In Diseases of sheep, 4th (aitken, ID ed),Wiley-Blackwell, Hobken, p 112-118.

Buxton D.; Brebner J.; Wright S.; Maley S.; Thomson K. and Milard K. (1996). Decoquinate and the control of experimental ovine toxoplasmosis. Vet. Rec., 138: 434-436.

Buxton, D. 1993. Toxoplasmosis: the first commercial vaccine. Parasitology Today 9, 335-337.

Buzbey J.C. and Roberts T. (1997). Economic costs andtrade impacts of microbial foodborne illness. World healthStatist. Quart, 50 (1-2): 57-66.

Buzby, J. C., and T. Roberts. 1997. Guillain-Barré Syndrome Increases Foodborne Disease Costs. FoodReview, Economic Research Service, U.S. Department of Agriculture, 20(3, September): 36–42.

Camossi, L., Fornazari, F., Richini-Pereira, V., da Silva,R., and Langoni, H. (2013). Toxoplasma gondii: Efficacyof an irradiated vaccine against experimental infectionchallenge in Wistar female rats. Proceedings of 2nd International Congress on Pathogens at the Human-

Canfield, P.J., Hartley, W.J., Dubey, J.P. 1990. Lesions of toxoplasmosis in **Australian marsupials. J Comp Pathol 103, 159-167.**

CDC. Toxoplasmosis [Net] in: CDC. DPDX:Laboratory Identification of parasite of public health concern . (2008) : Available from http// www. Cde. Gov / toxoplasmosis1Tchronically infected pregnant women: Predominance of type I in Europe and types II and III in Colombia (South America). Microbes Infect.; 8(9-10): 2333-2340.

Ciamak-Ghazaoi, D.V.M. (2005). Serological survey of antibodies to Toxoplasma gondii in Ardabil, Iran. J. App. Res, Vet. Med., 3(1): 44- 47.

Coppens, I., K.A. Joiner. 2001. Parasite-host cell interactions in toxoplasmosis: new avenues for intervention? www-ermm.cbcu.cam.ac.uk 1-24.

Dehkordi F. S., Borujeni M. R., Rahimi E., Abdizadeh R. (2013). Detection of *Toxoplasma gondii* in raw caprine, ovine, buffalo, bovine, and camel milk using cell cultivation, cat bioassay, capture ELISA, and PCR methods in Iran. Foodborne Pathog. Dis. 10, 120–125. 10.1089/fpd.2012.1311.

Derouin, F., Pelloux, H. 2008. Prevention of toxoplasmosis in transplant patients. ClinMicrobiol Infect 14, 1089-1101.

Desmonts, G. (1961). Annales de BiologieClinique, 19: 13–28.

Dubey J. P. (1991). Toxoplasmosis: An overview. J. Trop.Med. Pub. Heal., 22: 88-92.

Dubey J. P. (1994). Toxoplasmosis. J.A..Vet.Med. Assoc, 205: 153-159.

Dubey J. P. (2000). The scientific basis for prevention of Toxoplasma gondiiinfection: Studies on tissue cystsurvival, risk factors and hygiene measures. In: Ambroise-Thomas P., Peaterson E. editors. CongenitalToxoplasmosis: Scientific Background, Clinicalmanagement and control. Paris: Spinger-Verlag, France,271-275.

Dubey J. P. (2004). Toxoplasmosis - a water borne zoonosis. Vet. Parasitol,126(1-2): 57-72.

Dubey J. P. and Beattie C.P. (1988). Toxoplasmosis of animals and man. Pp 220. CRC Press, Boca Raton, USA.

Dubey J. P. and Frenkel J.K. (1972). Cyst-induced toxoplasmosis in cats. J. Protoz., 23(24): 537-546.

Dubey J. P., and hassanein R. (2005). Zoonotic toxoplasmosis in chickens. Egyp. Soci. Parasitol., 35(1): 341-350.

Dubey J. P., Romand S., Hilali M., Kwok O. C., Thulliez P. (1998). Seroprevalence of antibodies to Neosporacaninum and *Toxoplasma gondii*in water buffaloes190 (*Bubalusbubalis*) from Egypt. International journal ofParasitology. 28(3):527-529**.**

Dubey JP, Jones JL: Toxoplasma gondii infection in humans and animals in the United States. Int J Parasitol 2008, 38:1257–1278.

Dubey JP. Toxoplasma gondii infections in chickens (Gallus domesticus): prevalence, clinical disease, diagnosis and public health significance. Zoonoses Public Health. 2010;57(1):60–73. doi: 10.1111/j.1863- 2378.2009.01274.x. [PubMed: 19744305].

Dubey JP: Toxoplasmosis of Animals and Humans. 2nd edition. Boca Raton, Florida: CRC Press; 2010:1–239.

Dubey, J.P. (1986a). A review of toxoplasmosis in pigs. Veterinary Parasitology, 19: 181-223.

Dubey, J.P. 1993. Toxoplasma, Neospora, Sarcocystis, and other tissue cystforming coccidian of humans and animals. In: Kreier, J.P. 1993. Parasitic protozoa. 2nd ed. Vol. 6. Academic Press, Inc.

Dubey, J.P. 1996. Infectivity and pathogenicity of Toxoplasma gondiioocysts for cats. J Parasitol 82, 957-961.

Dubey, J.P. 1998b. Re-examination of resistance of Toxoplasma gondii tachyzoites and bradyzoites to pepsin and trypsin digestion. Parasitology 116:43- 50.

Dubey, J.P. 2002. A review of toxoplasmosis in wild birds. Vet Parasitol 106, 121 153.

Dubey, J.P. 2009 . History of the discovery of the life cycle of Toxoplasma gondii. International Journal for Parasitology 39, 877-882.

Dubey, J.P., and C.P. Beattie. 1988. Toxoplasmosis of animals and man. CRC Press. Boca Raton, Florida. Pp. 1-220.

Dubey, J.P., and J.K. Frenkel. 1976. Feline toxoplasmosis from acutely infected mice and the development of Toxoplasma cysts. Journal of Protozoology 23:537- 546.

Elamin,E.A.; Elias, S.; Daugschies, A. and Rommel, M. (1992). Prevalence of *Toxoplasma gondii*antibodiesinpastoral camels (*Camelusdromedarius*) in the Butanaplains, mid-Eastern Sudan *Veterinary Parasitology .43 (3-4):* 171-175.

Epiphanio, S., Sinhorini, I.L., Catao-Dias, J.L. 2003. Pathology of toxoplasmosis in captive new world primates. J Comp Pathol129, 196-204.

FAOSTAT, 2015. Food and Agriculture Organization Corporate Statistical Database, Gossner, C. et al., 2014. Human-Dromedary Camel Interactions and the Risk of Acquiring Zoonotic Middle East Respiratory Syndrome Coronavirus Infection, Zoonoses Public Health.

Faye B 2009 'L'élevage des grands camélidés : vers un changement de paradigme. Renc. Rech. Ruminants 16: 345-348.

Fayer, R. (1981). Fayer, R., 1981. Toxoplasmosis update and public health implications. Canadian Vet. J., 22: 344-352.

Feldman, H.A. (1974). Breaking the transmission chain of Toxoplasma: a programme forthe prevention of human toxoplasmosis. Bulletin o f New York Academy o f Medicine-,50: 110.

Ferguson T.R. (2002). Apicomplexa. J. Parsitol., 18: 355-357. fetus and newborn infant. 5th ed. Philadelphia: W.B. Saunders.

Frenkel, J.K. (1973). Toxoplasma in and around us. BioScience; 23: 343-352.

Freyre, A.; Bonino,J.; Falc´on, J.; Castells,D.;Correa,O.and Casaretto, A. (1999). The incidence andeconomic significance of ovine toxoplasmosis inUruguay. Veterinary Parasitology 81 (1): 85–88.

G. Desmonts, "S´erologie de la toxoplasmose," Annales de Biologie Clinique, vol. 19, pp. 13–28, 1961.

Gebremedhin, E.Z., A.Y. Hassen, T. Gebregergis, S.T. Tesfaye, D. Fufa, T. Getachew, D.M. Vincenzo and V. Maria, 2014. First report of Toxoplasma gondii in camels (Camelusdromedarius) in Ethiopia: bioassay and sero-epidemiological investigation. BMC Veterinary Research, 10: 222.

Giadinis N.; Terpsidis K.; Diakou A.; Siarkou V.; Karatzias H. and papazahariaadou M. (2009). Treatment of sheep toxoplasmosis with sulphadimidine. InProceedings of the world sheep Veterinary Congress,Stavanger, Norway. gondii in experimentally infected sheep. J Med Microbiol 38, 360-365.

Gibson, C.L, and Coleman, N. (1958). The prevalence of Toxoplasma antibodies in Guatamala and Costa Rica. American Journal o f Tropical Medicine and Hygiene', 7:334-338.

Glasner, P. D.; Silveira, C.; Kruszon-Moran, D.; Martins, M. C.; Burnier, J. Camargo, M. E.; Nussenblatt, R. B.; Kaslow, R. A. and Belfot, R. Jr. (1992). An unusually high prevalence of oculartoxoplasmosis in Southern Brazil. Am J Ophthalmol 114 : 136-144.

Hakan, L. ; H. Murate and B. Itzhak. (2010). Toxoplasmosis. Web MD. professional. industry spotlight (http://emedicine medscane. com/article/1000028-overview).

Havelaar, A.H., van Rosse, F., Bucura, C., Toetenel, M.A., Haagsma, J.A., Kurowicka, D., Heesterbeek, J.A.P., Speybroeck, N., Langelaar, M.F.M., van der Giessen, J.W.B., Cooke, R.M., Braks, M.A.B. 2010. Prioritizing emerging zoonoses in the Netherlands. PLoS ONE 5, e13965.

Hoff, R.L., J.P. Dubey, A.M. Behbehani, and J.K. frenkel. 1977. *Toxoplasma gondii* cysts in cell culture: new biologic evidence. Journal of Parasitology 63:1121-1124.

Husna M. E.; Siham E.; Abdel-Aziz B. E. (2012). Serosurveillance of Toxoplasma gondiiAntibodies in CamelsatTumbool Slaughterhouse, Central Sudan. The Sudan J.Vet. Res. (2012).27: 65-67.

Hussein MF, Bakkar MN, Basmaeil SM, Garelnabi AR: Prevalence of Toxoplasmosis in Saudi Arabian camels (Camelusdromedarius). Vet Parasitol1988, 28:175–178.

I. Esteban-Redondo, S. W. Maley, K. Thomson, S. Nicoll, S. Wright, D. **Buxton, and E. A. Innes**, “Detection of T. gondii in tissues of sheep

and cattle following oral infection," Veterinary Parasitology, vol. 86, no. 3, pp. 155–171, 1999 **.**

Ibrahim, B. B.; Salama, M. M.; Gawish, N. I. and Haridy, F. M. (1997). J. Egypt. Soc. Parasitol., 27(1):273-278.

Innes, E.A. (2010). Vaccination against Toxoplasma gondii:an increasing priority for collaborative research? ExpertRev Vaccines. 9:1117- 1119.

Innes, E.A. 1997. Toxoplasmosis: comparative species susceptibility and host immune response. Comp Immunol MicrobiolInfect Dis 20, 131-138.

Innes,E.A., Bartley, P.M., Maley, S., Katzer, F., Buxton, D. 2009b.Veterinary vaccines against Toxoplasma gondii. MemInstOswaldo Cruz 104, 246-251.

Ira, J. B.; Jeroen,P.andSaeij (2009). Communication between *Toxoplasma gondii*and its host: impact on parasite growth, development, immune evasion, and virulence. Parasitology.**117**: 458-476.

J. and Akanmori B. (2000). The prevalence of anti- Toxoplasma gondiiantibodies in Ghananian sheep and goats. Acta. Tropica, 76: 21-26.

Jacobs, L.; Lunde, M.N.,. (1957).Haemagglutination test for toxoplasmosis. Science. 125: 1035. Journal of American Medical association, 248:1728-1732.

James, W. A. ; M. Jennifer and P. Reitter. (2001). Expert Review in molecular medicin. Cambridg University (http:// Locke-citizendium. orgi 8080 /wiki /*Toxoplasma - gondii*).

Johnson, H. R. (2009). The substrate specificities and physiological function of the *Toxoplasma gondii* apicoplast phosphate translocator. M.Sc. Thesis. Collage of Science. University of Tromso Norway. pp 69.

Jones, J. L. and J. P. Dubey.(2010). Water borne toxoplasmosis-recent developments. Exp. Parasitol.,124:10-25.

Kenny, D.E., Lappin, M.R., Knightly, F., Baler, J., Brewer, M., Getzy, D.M. 2002. Toxoplasmosis in Pallas′ cats (Otocolobus felismanul) at the Denver Zoological Gardens. J Zoo Wildl Med 33, 131-138.

Khalil, M. K. and Elrayah, I. E. (2011). Seroprevalence of Toxoplasma gondii antibodies in farm animals (camels, cattle, and sheep) in Sudan. Journal of Medicine and Animal Health Vol. 3(3), pp. 36-39.

Kortbeek, L. M.; Hofhuis, A.; Nijhuis, C. D. M. and Havelaar, A. H.(2009). Congenital toxoplasmosis andDALYs in the Netherlands. Men InstOswaldo Cruz. 104,370-373.

Krahenbuhl, J.L and Remington, J.S. (1982). The immunology of Toxoplasma and toxoplasmosis. In ‘Immunology of Parasitic Infection’. 2nd Ed. S. Cohen and K.S. Warren. Blackwell Scientific Publications, Oxford. p356.

Levine, N.D., Corliss, J.O., Cox, F.E.G., Deroux, G., Grain, J., Hongberg, B.M., Leedale, G.F., Loeblich, A.R., Lom, S., Sprague, V., Vavra, J, and Wallace, F.G. (1980). Anewly revised classification of the protozoa. Journal o f Protozoology; 27: 37-57.

Lewis, J.S.Jr; Khoury, H.; Storch, G.A.; DiPersio, J. (2002). PCR for the diagnosis of toxoplasmosis after hematopoietic stem cell transplantation. Expert. Rev. Mol. Diagn. 2(6): 616-624.

Manal Y.I. (2003). Study on Toxoplasma and Sarcocystosis from camel *(Camelus dromedaries)* in the Sudan. PhD. Thesis. University of Khartoum, Sudan.

Manal Y.I. and Majid A.M. (2008). Association of Diarrhoeawith congenital toxoplasmosis in calf-camels (*Camelusdromedarius*). Int. J. Trop.Med., 3(1): 10-11.

MARF (Ministry of Animal Resources and Fisheries). 2015. Department of Animal Resources Economics Administration Records. Khartoum,

Martino, R., Maertens, J., Bretagne, S., Rovira, M., Deconinck, E., Ullmann, A.J., Held, T., Cordonnier, C. 2000. Toxoplasmosis after hematopoietic stem cell transplantation. Clin Infect Dis 31, 1188-1195.

Mason, I.L. and Maule, J.P. 1960. *The indigenous livestock of eastern and southern Africa.* Technical Communication 14, Farnham Royal (Bucks), CAB (Commonwealth Agricultural Bureaux), 248 pp.

Mehlhom, H. and Walldorf, V. (1988) Life Cycles. In 'Parasitology in Focus. Facts andTrends'. Mehlhom, H. (1988). Springler-verlag. Berlin Heidelberg, New York.

Mehlhorn, H., and J.K. Frenkel. 1980. Ultra structural comparison of cysts and zoites of *Toxoplasma gondii, Sarcocystis muris,* and *Hammondia hammondi* in skeletal muscle of mice. Journal of Parasitology 66:59-67.

Montoya JG, Kovacs JA, Remington JS. Toxoplasma gondii. In: Mandell GL, Bennett JE, Dolin R. Principles and Practice of Infectious Diseases, 6th edition, Elsevier, Churchill, Livingstone, 2005, p 3170-3198.

Montoya, J. G. ; J. A. Kovacs and J. S. Remington. (2005).Toxoplsma gondii. In: Principle and practice of infectious Diseases. 6th edition by G. L. Mandell, J. Bennett, and R. Dolin (eds). Elsevier Churchill Livingstone. 2: 3170-3198.

Musa H.O. (2008). Detection of *Toxoplasma gondii*antibodies in females in port Sudan City. B.Sc. thesis,University of Ahlia, PortSudan.

Nath, A.; Sinai, A.P., (2003). Cerebral toxoplasmosis. Curro Treat. Options NeuroL. 5(1): 3-12**.**

Nicolle C &Manceaux L. (1908). Sur une infection á corps de Leishman (ou organisms voisins) du gondi. C.R. HebdSeancesAcad Sci., 147 : 763-**766.**

P. Zuber and P. Jaquier, "Epid´emiologie de la toxoplasmose situation au niveaumondial," SchweizerischeMedizinischeWochenchrift, vol. 125, pp. 19S–22S, 1995.

Pappas, P. W. and S. M. Wordrop. (2004). Toxoplasma gondii WWW. Google . com .

Pavesio, C. E., M.L. Chiappino, P.Y. Setzer, and B.A. Nichols.1992. *Toxoplasma*gondii: differentiation and death of bradyzoites. Parasitology Research 78:1-9.

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

Peyron, F.; J.R. Lobry;K. Musset;B. Ferradiz; J. E. Gomez-Marin, ; E. Petersen;V. Meroni;B. Rausher; C. Mercier; S. Picot and M. F. Cesbron-Delauw.(2006). Serotyping of Toxoplasma gondii in chronically infected pregnant women: Predominance of type I in Europe and types II and III in Colombia (South America). Microbes Infect.; 8(9-10): 2333-2340.

Remington, J. S.; Miller, M.J.; Brownlee, 1., o (1968). Serological diagnosis of Toxoplasma gondiiinfection. Pediatrics. 41: 1082.

Remington, J.S., R. McLeod, P. Thulliez, and G. Desmonts. 1995 Toxoplasmosis.
Rev. Inv. Vet. Perú 11:32-39.

Rodriquez-Ponce, E., Molina, J.M. and Hemadez, S. (1995). Seroprevalence of gut toxoplasmosis on Grand Canary Island (Spain). Preventive Veterinary Medicine 24(4): 229-234.

Sabin, . A.B.; Feldman, H.A. (1948). Dyes as microchemical indicators of anew immunity phenomenon affecting a protozoan parasite (Toxoplasma). Science. 108:660-663.

Sacks J.J.; Roberto R.R. and Brooks N.F. (1982). Toxoplasmosis infection associated with raw goat′ s milk. Journal of American Medical association, 248:1728-1732.

Saleem AN, Fatohi FA (1993). Prevalence of *Toxoplasma*-linkeand*Brucella*antibodies in cattle with clinical and gynecological disturbaces in Mosul, Iraq. Iraq J. Vet. Sci., 6: 48-52.

Santos, T.R., Costa, A.J., Toniollo, G.H., Luvizoto, M.C.R., Benetti, A.H., Santos, R.R., Matta, D.H., Lopez, W.D.Z., Oliveira, J.A., and Oliveira, G.P. (2009). Prevalence of anti-*Toxoplasma gondii*214antibodies in dairy cattle, dogs and humans from theJauru micro-region, MatoGrosso State, Brazil. VeterinaryParasitology, 161, 324-326.

Schaffner A. (2001). Pretranslpant evaluation forinfections in donors and recipients of solid organs. Clin.Infect. Dis., 33(1): 9-14.

Schoonman, L.B., T. Wilsmore and E.S. Swai, (2010). Sero-epidemiological investigation of bovine toxoplasmosis in traditional and smallholder cattle production systems of Tanga Region, Tanzania. Trop Anim Health Prod., 42: 579-87.

Schwartz, H.J. and M. Dioli, 1992. The one humped camel (Camelus dromedaries) in Eastern Africa.A pictorial guide to disease, health care and Management .VerlagJosefMargraf Scientific Books. D-6992 Weikersheim FR Germany, pp: 282.

Seri I.H.; Abakar D.A. and Idris F.O. (2003). A note on camel toxoplasmosis in the Sudan. 7th Sci Cong. Egyptian Soc for cattle Diseases. 153-157.

Sevgili, M. C. B. ;S. Nalbantoglu and Z.Vatansever (2005). Determination of Seropositivity for Toxoplasma gondii in sheep in Sanliurfa Province. Turkey. J. Vet. Anim. Sci., 29:107-111.

Shaapan R. M. and Fathia Khalil A. M. (2008). Evaluation of Different *Toxoplasma gondii*isolatesasAntigens used in the Modified Agglutination Test for thedetection of Toxoplasmosis in Camels and Donkeys.American-Eurasian J. Agric& Environ. Sci., 3(6): 837- 840.

Sheffield, H.G., and M.L.Melton. 1968. The fine structure and reproduction of Toxoplasma gondii. Journal of Parasitology 54:209-2226.

Siddig. A. B. (2010). Study on Toxoplasmosis in Humans and Cats in the Red Sea State, Sudan M.Sc. Thesis SUST.

Solusby, E.J.L. () 982). Toxoplasma. In: Helminths, Arthropods and Protozoa of domestic animals. 7th edition. BailliereTindall. London. 670-682.

Sukthana Y. (2006). Toxoplasmosis: beyond animals to humans. Trends Parasitol. 22: 137-142.

Taylor M.A.; Coop R.L.; and Wall R.L. (ed.). (2007). Veterinary Parasitology. Third Edition, b- BlackwellPublishing, Pp 121-258**.**

Tenter, A.M. (2009). Toxoplasma gondii in animals used for human consumption. MemInstOswaldo Cruz, Rio de Janeiro, Vol. 104(2): 364-369.

Tenter, A.M.; Heckeroth, A.R.; Weiss, L.M., (2000). Toxoplasma gondii: from animals to humans. Int. J. Parasitol. 30 : 1217-1258.

Tobin, C. ;A. Pallard and L. Knoll. (2010).*Toxoplasma* cyst wail formation in activated bone marrow-derivesd macrophage and bradyzoite condition. J. Aug. 12(42): 2091-2093.

Unno, A., Suzuki, K., Xuan, X., Nishikawa, Y., Kitoh, K., Takashima, Y. 2008. Dissemination of extracellular and intracellular Toxoplasma gondiitachyzoites in the blood flow. ParasitolInt 57, 515-518.

Uroquhart, G.M; Armour, J.; Duncan, J.L.; Jennings, F.W. (1996). Toxoplasma. In: 2nd Veterinary Parasitology. edition. Blackwell Science Ltd. London. 234-238.

Utuk AE, Kirbas A, Babur C, Balkaya I: Detection of *Toxoplasma gondii* antibodies and some helminthic parasites in camels from Nevsehir province of Turkey. Isr J Vet Med. 2012, 2012 (67): 106-108.

Vyas, A., Kim, S.K., Giacomini, N., Boothroyd, J.C., Sapolsky, R.M. 2007. Behavioral changes induced by Toxoplasma infection of rodents are highly specific to aversion of cat odors. Proc Natl Acad Sci U S A 104, 6442-6447.

Wastling J.; Nicolle S. and Buxton D. (1993). Comparison of two genes amplification method for the detection of Toxoplasma gondiiin experimentally infected sheep. J. Med.Microbiol, 83: 360-365.

Weiss, L.M., and K. Kim. 2000. The development and biology of bradyzoites of *Toxoplasma gondii*. Frontiers in Bioscience 5: 391-405.

Weiss, L.M., and Kim, K. (2007). Toxoplasma gondii, The Model Apicomplexan-Perspective and Methods. Elsevier Ltd. Wochenchrift, vol. 125, pp. 19S–22S, 1995.

ZeinEldin, E. A.; Elkhawad, S. E. and Kheir, H. S. M. (1985). A serological Survey for Toxoplasma antibodies in cattle, sheep, goats and camels (Camelusdromedaries) in the Sudan. Rev. Elv. Med. Vet. Paystrop., 38(3): 247-249.

Zhang, S.; Wei, M.; Zhao, H.; Shi, G., (1999). Establishment of immunoglobulin M (IgM)immunosorbent agglutination assay (ISAGA) for diagnosis of toxoplasmosis. ZhongguJi Sheng Chong Xue Yu JiSheng Chong Bing ZaZhi. 17(4): 225-227.

Zhang, S.Y.; Wei, M.X., (2001). Quantitative and . qualitative comparison of three agglutination tests for detecting Toxoplasma gondiiantibodies. ZhongguJi Sheng Chong Xue Yu Ji Sheng Chong Bing ZaZhi. 18(1): 46-48.

Zhou, P.; H. Zhang; R. Q. Lin; D. L. Zhang; H. Q. Song ; C. Su and X. Q. Zhu. (2009). Genetic characterization of *Toxoplasma gondii* isolates from China .In.J.Parasitol.,58:193-195.