Vol 13 No 1, p 7-14

PARASITIC DISEASES OF CAMELS – AN UPDATE
1. PROTOZOAL DISEASES

M.B. Chhabra* and Arun K. Sangwan
Department of Veterinary Parasitology, College of Veterinary Sciences,
CCS Haryana Agricultural University, Hisar-125 004, Haryana, India

ABSTRACT

Protozoal diseases, particularly trypanosomosis caused by the flagellate *Trypanosoma evansi,* are a considerable constraint on the health and productivity of the dromedary camels throughout the tropics and subtropics. With the developments in the field of molecular biology and their application in parasitic infections, there have been great strides of progress in the diagnostic techniques. Apart from detection of parasitoses, these tests are useful aids to monitor the effectiveness of therapeutic interventions. As a consequence, more reliable epidemiological data on the distribution and incidence of these diseases has been generated. DNA-based technologies have enabled the characterisation of species, subspecies and stocks (strains) of camel parasitising trypanosomes. Chemotherapy and strategic control of trypanosomosis continues to be another fertile area which has witnessed a fair number of recent reports. Other protozoan parasites and their occasional association with disease are now being reported more frequently. These include the gut-dwelling coccidia, the tissue-cystic forms *(Sarcocystis* and *Toxoplasma), Balantidium, Cryptosporidium*, etc. Possible changes in camel husbandry practices may lead to increase in their prevalence and economic impact. As such, there is need to include these in the overall parasitic disease surveillance among camels.

Key Words: Camels, parasites, parasitoses, protozoal diseases, protozoan parasites.

Protozoal diseases of camels, in sharp contrast to those of cattle, have not been researched and understood very well apart from trypanosomosis. Several factors including the importance of camels as draught, food, sport and show animals, and consequent upsurge of interest in their health and well-being, have altered the situation. Additionally, the availability of improved methodologies for the detection of infections, have increasingly brought them into focus. In the recent past, there have been comprehensive published reviews (Gatt- Rutter, 1967; Boid *et al*, 1985), a conference paper (Luckins, 1988) and an elaborative chapter in a book (Wernery and Kaaden, 2002) on the subject. However, there is a substantial mass of new information since then, which has contributed to advancements in various aspects and is highly relevant. Accordingly, the objective of this review was to collect this material and present an update. Understandably, a major part of this relates to cameline trypanosomosis.

Trypanosomosis

Epidemiology: The disease caused by the flagellate protozoan *Trypanosoma evansi* and first reported in 1880 among camels (surra) in India, is ranked as probably the most serious infectious disease of camels. It is widespread throughout the Middle East, South Asia, Far East and outside the tsetse belt in Africa (Luckins, 1988; Wernery and Kaaden, 1995 and 2002). Although noted for a high rate of morbidity and even mortality, the prevalence and pathogenicity of surra varies considerably between different geographical regions (Zelleke *et al*, 1992; Reid, 2003). Reports during the last 15 years cover practically all the countries where *T. evansi* is endemic. As prevalence data are based on different tests, which differ widely in sensitivity, the figures should only be taken as rough estimates. Generally, the areas of highest trypanosomosis prevalence were also those with heavy infestations of tabanids or other haematophagous flies. In Somalia, 160 (5.33%) of 3000 blood samples from dromedary camels were found infected (Dirie *et al*, 1989) with *T. evansi*. Prevalence of 13.2% was detected in 310 camels in Saudi Arabia (Hussein *et al*, 1991). *Trypanosoma evansi* prevalence ranged from 1.7% in blood smears to 56.4% using micro-ELISA among 1039 camels in 33 herds in Somalia (Baumann and Zessin, 1992) where one of the world's largest dromedary populations (5.3 million) is kept. Trypanosomosis along with brucellosis was rated as an important limiting factor on production. Endemicity of cameline trypanosomosis in 18 districts of Rajasthan, India was reported by Raisinghani and Lodha (1989). Blood samples from 240 camels from Western Rajasthan, examined for *T. evansi*, revealed 18 (7.5%) infected by the wet blood/ Giemsa stain smears and 76 (31.66%) positive for antigen using

SEND REPRINT REQUEST TO M.B. CHHABRA **Present address:** 2518, Sector D-2, Vasant Kunj, New Delhi-110070, India.

double antibody sandwich ELISA (Pathak *et al*, 1993). In Mauritania, prevalence ranging between 1-4% on blood smear examination, and 16.5-24.3% using different serological tests was recorded (Dia *et al*, 1997). Prevalence and infection pattern of *T. evansi* in camels in mid-Eastern Sudan was reported by Elamin *et al* (1998). Surra prevalence among camels in Jordan using thick smear and sub-inoculation techniques was found in 36 (8.2%) out of 437 and 33% of 257 examined in another survey (Al-Rawashdeh *et al*, 2000). In Southern Iran, 32 camels were found to be infected with *T. evansi* by blood examination and rat inoculation of the 333 samples (Zarif-ferd and Hashemi Fesharki, 2000). Trypanosomosis due to *T. evansi* was found in 7.70% of 546 camels in eastern Ethiopia (Woldemeskel *et al*, 2001), while in another province of Ethiopia, 43 (10.9%) of 391 camels were found positive by clinical and laboratory examinations (Tekle and Abebe, 2001). In an investigation into the animal-level risk factors for *T. evansi* infection in eastern and central Kenya, Ngaira and coworkers (2002) concluded that camels managed under nomadic pastoralism had higher risk of exposure than those from ranching systems of management. In Morocco, an epidemiological survey of camel trypanosomosis for the first time in 1997-98, found overall seroprevalence of 14.1% by card agglutination test (CATT) and 18.2% by Ab-ELISA (Atarhouch *et al*, 2003). Current prevalence rates of camel trypanosomosis (*T. evansi*) in selected herds of northern Kenya (Oyieke, 2003) were found to be 19.2% by mouse inoculation (MI) and 11.5% by blood smear (BS) techniques. Two major foci of camel trypanosomosis in Saharan Morocco in 1997-98 had seroprevalence of 35.4 and 43.3% by Ab-ELISA (Rami *et al*, 2003). Moreover, these workers found more camels anaemic in summer than in spring. Several factors of husbandry including stress, excessive work, overcrowding and continual contact with animals coming from other infected regions, contributed to the persistence of the disease. A comparison of two camel rearing systems indicated that camels kept under ranch management have higher trypanosomosis than those kept in a traditional system (Njiru *et al*, 2002). In a recent survey among pastoralists in Turkana districts of Kenya (Mochabo *et al*, 2005) annual incidence of trypanosomosis was identified at 11.4% with a higher incidence and mortality rate at 15% and 9.9% in adult camels than 6.9% and 5.2% in young camels, respectively.

Characterisation of strains and antigens: Iso-enzyme pattern using thin-layer-starch-gel electrophoresis revealed two of the *T. evansi* stocks isolated in Kuwait to be identical to those from Nigeria, Sudan and Kenya (Al-Taqi, 1989). The application of DNA probes for identification and characterisation of *T. evansi* strains (Hamers *et al*, 1990) opened up the use of this technology for strain differences and antigenic variation. Electrophoretic karyotyping of *T. evansi* isolates (Waitumbi and Young, 1994) was suggested as a sensitive epidemiological tool for identification of drug-resistant parasites. Fractionation by chromatography revealed 7 distinct fractions in *T. evansi* (camel) antigen and analysis by SDS-polyacrylamide electrophoresis (PAGE) revealed protein bands with molecular weights ranging between 14-65 kDA (Pathak *et al*, 1994). Polypeptide profiles of 7 stocks of *T. evansi* whole-cell lysate by SDS-PAGE (Singh *et al*, 1994a) and of cell membrane and flagellar preparations by SDS-PAGE and western blotting (Singh *et al*, 1995) did not indicate antigenic variability of high degree. According to Masiga and Nyangao (2001), the development of specific DNA probes has greatly improved the identification of trypanosomes in areas where more than one morphologically indistinguishable species are prevalent. Omanwar *et al* (2001) studied DNA polymorphism in *T. evansi* stocks and hypothesised the adaptability of the organism to different hosts and geographical regions as the reason.

Clinical and haematological aspects: The belief that dromedary owners and herdsmen can diagnose surra from urine odour, was scientifically investigated by Hunter (1986). The urine of a slaughter dromedary which had clinical surra on examination was dark brown and with a strong pungent odour. This odour was not present in the other 22 camels which included one with low parasitaemia. Camels with *T. evansi* parasitaemia in peripheral blood had significantly lower packed cell volume (PCV) and haemoglobin (Hb) values as also eosinophil and neutrophil counts (Yagoub, 1989) Animals concurrently harbouring *Haemonchus longistipes* in high intensity showed bigger decreases in PCV and Hb. Those infected only with *H. longistipes* showed a significant increase in eosinophil counts compared to controls. It was concluded that *T. evansi* has a more pronounced effect on camel health than *H. longistipes*. Clinical signs of emaciation, pale mucous membranes, dry coat, intermittent fever, weakness and lachrymation were observed (Karram *et al*, 1991). Haematological changes included significant decrease in Hb and PCV alongwith normocytic hypochromic anaemia associated with leucocytosis, eosinophilia and monocytosis. Haematological profile in another study (Samy and Gadir, 1992) showed macrolytic normochromic anaemia. Lymphopenia was found during parasitaemia while lymphocytosis was found during the later stage. Histopathologically, there was erythrophagocytosis and destruction of lymphoid follicles during parasitaemia and hyperplastic

proliferation of lymphocytes toward the end. In the chronic form, along with long aparasitaemic periods, the animals show severe immunosuppression characterised by an increased susceptibility to secondary infection (Njiru, 1997). Although anaemia is the main pathological feature, particularly in chronic cases, it is not regarded as a significant feature in acute disease (Ouma *et al*, 1997). In the latter, the accompanying activation of haemolytic complement may be a major factor contributing to death (Njiru *et al*, 2000a). Experimental *T. evansi* infection of camels produced microlytic normochromic anaemia (Haroun *et al*, 2000). Neutrophilia and lymphocytosis were also observed. The activity of serum enzymes viz. AST, ALP, LDH and GGT increased after infection. There was concurrent increase in globulin concentration and a decrease in albumin and glucose concentration. The main histopathological observations were fatty changes focally distributed in the liver. Similar changes induced by natural trypanosomosis in racing dromedaries were also reported (Chaudhary and Iqbal, 2000). Clinical examination in camel trypanosomosis in Morocco revealed enlargement of lymph nodes was the most frequent sign in seropositive animals (Atarhouch *et al*, 2003). An outbreak of abortions and delivery of premature or weak calves leading to neonatal mortality attributable to *T. evansi* in camels was reported recently from Canary Islands (Gutierrez *et al*, 2005).

Diagnosis: The clinical signs of surra in camels are not pathognomonic enough for more than a provisional diagnosis. Demonstration and identification of trypanosomes in the blood remains the basis of a reliable diagnosis. Concentration techniques such as microhaematocrit centrifugation (MHCT) were devised to overcome the problem of scanty parasitaemia. A silicon centrifugation technique although simple and rapid (Nessiem, 1994) has not found much application for camel trypanosomosis. On the other hand, Reid *et al* (2001) suggested that the sensitivity of mouse inoculation (MI) and MHCT was improved 10-fold through the use of buffy coat in place of whole blood. Standard trypanosome detection methods (STDM) based on direct microscopy, concentration and animal inoculation methods are generally recommended (OIE, 2000). Wernery *et al* (2001) considered MHCT as the most sensitive diagnostic test. The limitations in terms of low sensitivity of parasitological diagnostic techniques (Rae and Luckins, 1992) have been a driving force for research into alternate techniques including immunodiagnostic, antigen-detection assays and DNA based methods. Enzyme-linked immunoassay (ELISA) for circulating antibodies (Rae *et al*, 1989) and its antigen-detection variant (Ag-ELISA) have been used extensively for more efficient and accurate detection of *T. evansi* in dromedary camels (Nantulya *et al*, 1989; Olaho-Mukani *et al*, 1992, Waitumbi and Nantulya, 1993; Singh *et al*, 1994b; Pathak *et al*, 1997; Jain *et al*, 2000). The latter test (Ag-ELISA) can also be used to assess the success of chemotherapy in the control of camel trypanosomosis as antigens disappear from circulation within 30 days of treatment (Olaho-Mukani *et al*, 1992). Other serological tests in use include card agglutination test (CATT), a simple and quick test for antibodies that could detect 89% of the samples positive for blood smears and also had good correlation with low PCV results (Diall *et al*, 1994; Pathak *et al*, 1997; Gutirrez *et al*, 2000; Atarhouch *et al*, 2003). Another major development was the introduction of card latex agglutination test which detects trypanosomal antigens in serum, plasma or whole blood with equal sensitivity and specificity as the ELISA-antigen detection. This test, available commercially under the brand name Suratex (Brentec Diagnostics, Nairobi) is not dependent on laboratory use and is thus a pen-side diagnostic test (Nantulya, 1994). Suratex and Ag-ELISA could diagnose the camels with sub-patent infections (Olaho-Mukani *et al*, 1996). These workers also observed that Suratex was 100% specific, showed sensitivity ranging from 93.2% to 94.6% and detected 124 (93%) of the 133 MI-positive camels as against 118 (89%) detected by Ag-ELISA. Further, Suratex was much more sensitive than HCT (Olaho-Mukani *et al*, 1997; Njiru *et al*, 2000b; Ngaira *et al*, 2002). A dissenting report of Delafosse and Doutoum (2000) did not recommend Suratex because of low sensitivity and specificity of the test in their hands. These workers found that mercuric chloride with PCV gave the highest sensitivity, specificity and predictive value at low cost. A team in India devised improvements in Ab-ELISA viz. a Dot-ELISA (Shahardar *et al*, 2002) and competition inhibition-ELISA (Shahardar *et al*, 2003) with 46.66% and 63% positivity, respectively in detecting antibody in camel sera from an endemic area in Rajasthan. Use of detergent solubilised antigen (Shahardar *et al*, 2004) further raised the positivity to 68.8%.

A new dimension has been added to the diagnostic approach with the application of DNA-based technologies, notably polymerase chain reaction (PCR). For the diagnosis of camel trypanosomosis, Ijaz *et al* (1998) suggested the use of PCR during both acute and chronic phases of infection, and for use in the evaluation of treatment. Basagoudanavar *et al* (1998) employed PCR in camel blood samples and concluded that PCR provided a useful diagnostic tool for detecting *T. evansi* infected camels. Higher sensitivity of this molecular technique compared to parasitological and

serological methods, based on 217 camels in Rajasthan was also reported by Singh *et al* (2004).

Treatment and Control: A number of drugs have been in use in the past. Among these, the naphthalene compound Suramin (Naganol) at a double I.V. dose of 50 ml was successful in controlling trypanosomosis (*T. evansi*) in camels in Egypt (Karram *et al*, 1991). Due to resistance problem, its production and use has now been given up. Of the currently available chemotherapeutic agents, Quinapyramine (mostly pro-salt) has been effectively used in curing trypanosomosis in camels (Singh *et al*, 1987; Raina *et al*, 1990; Olaho-Mukani *et al*, 1992; Haroun *et al*, 2003). An arsenical Cymelarsan S.C. injection was introduced (Zelleke *et al*, 1989) and has been evaluated extensively to be effective against *T. evansi* infection in camels (Gool *et al*, 1992; Otsyla *et al*, 1992; Nyangao *et al*, 1995; Njiru *et al*, 2000b; Rami *et al*, 2003; Gutierrez *et al*, 2005). The drug was indicated for all forms of the infection and at the dose range of 0.2 to 1.2 mg per kg b.wt., was well-tolerated. Camels appear to be more susceptible to the toxic action of some trypanocidal drugs than other species (Ali, 1988). This being the case with diminazene aceturate, a stable form of this drug namely Trypan was formulated but reportedly (Maina *et al*, 2003) may not be effective in curing camels with acute *T. evansi* infections. With regard to drug resistant parasites, Waitumbi and Young (1994) suggested that observation of Karyotype patterns recurring in isolates from herds kept under chemoprophylaxis could help in their identification. Drug resistance in trypanosomosis (Olaho-Mukani *et al*, 1995; El-Rayah *et al*, 1999) requires monitoring and remedial action. Traditional knowledge regarding prophylaxis and treatment provides some alternative approaches to control. Camel pastoralists are much aware of the disease-transmitting vectors and have developed strategies to avoid exposure to biting flies (Kohler-Rollefson, 1994).

Coccidiosis

Gut-dwelling coccidia (mainly *Eimeria* spp.) are distributed widely among camel population with high prevalence rates of infection involving several species (Luckins, 1992). Various workers have reported prevalence rates of infection in different camel-rearing parts of the world. In India, prevalence of *Eimeria* ranging from 14.18% in Gujarat (Pethkar and Momin, 1990) to 25.19% in Rajasthan (Partani *et al*, 1999) has been recorded. The latter report was based on 897 faecal samples. Higher prevalence (28.78%) in farm camels compared to field camels (21.6%) and higher prevalence in rainy season and also in camel calves below one year age was recorded. The species encountered were *E. dromedarii, E. cameli, E. pellerdyi, E. rajasthani and E. nolleri*. Raisinghani *et al* (1987) presented a case report of a 6-month old camel calf with diarrhoea and abdominal pain excreting *Isospora* oocysts at 2000 oocysts per g of fluid faeces. The calf responded well to Neftin (Furazolidone) and was completely cured. *Isospora* excretion in scouring camel calves was reported from Kenya (Younan *et al*, 2002). Five calves, 18-32 days old, showing signs of diarrhoea were found excreting Isospora oocysts. Diarrhoea lasted for approximately 10 days after which one severely dehydrated *Isospora*-secreting calf died. Histological examination revealed multiple ulcerative colitis. *Eimeria cameli* were sporadically observed in the faeces of older animals. In a study from Iraq (Al-Sadi, 1994), the basic histopathological lesion in camel coccidiosis was in the form of necrotic enteritis. Pathological studies on camel coccidiosis were also reported from UAE (Kinne and Wernery, 1998) and from Iran (Tafti *et al*, 2001). The latter study indicated that the most important and frequent pathologic lesion in the digestive tract resulted from *Eimeria* spp. infections (63% of 100 slaughtered camels). Diagnostic histopathological lesions included the presence of developmental stages in mucosal epithelium and glands in crypts of Lieberkuhn, together with eosinophilic enteritis, lymphoid hyperplasia, congestion and oedema of submucosa. There does not appear to be any specific therapy for camel coccidiosis but anticoccidials effective against coccidiosis in ruminants could be useful.

Tissue Cyst-forming coccidia: Sarcocystis cameli is the only species reported from camel, with tissue cysts occurring in various muscles of the body. Abattoir examination of 192 dromedaries in Afghanistan (Kirmse and Mohanbabu, 1986) led to detection of Sarcocystis by direct observation of oesophageal muscle sandwiched between glass plates (47.3%), histologically (61.5%) and by peptic digestion (66.3%). Feeding dogs with camel meat experimentally resulted in passage of sporocysts of *Sarcocystis* spp. (Warrag and Hussein, 1983; Hilali *et al*, 1992). In a later trial (Hilali *et al*, 1995), meat samples from oesophagus and tongue of camels slaughtered in Saudi Arabia, when fed to dogs resulted in excretion of *S. cameli* sporocysts. *Sarcocystis* in meat producing animals including camels was reported from Ethiopia (Woldemeskel and Gumi, 2001). In the latter study, haematoxylin eosin stained tissue samples from various muscles were found infected in 45.45% of 121 camels. The musculatures in the order of frequency of harbouring cysts were: oesophagus, shoulder, diaphragm, cardiac and masseter muscles. Histopathological observations of *S. cameli* in the heart of dromedary were made (Dadhich, 2000). The main economic impact of sarcocystosis appears to be the condemnation of heavily infected tissues at meat inspection.

Sporadic records regarding other tissue cyst-forming coccidia relate to *Hammondia heydorni* (Nassar *et al*, 1983; Warrag and Hussein, 1983; Hilali *et al*, 1992; Hilali *et al*, 1995), *Toxoplasma gondii* (Hilali *et al*, 1995) and *Besnoitia* spp. (Tafti *et al*, 2001). Serological prevalence of *T. gondii* (Hussein *et al*, 1998; Elamin *et al*, 1992; Hilali *et al*, 1998) and of *Neospora caninum* (Hilali *et al*, 1998) were also reported. A case of acute toxoplasmosis in a 6-year old female camel, characterised by anorexia dyspnoea, abortion and fatal termination, had been documented (Hagemoser *et al*, 1990).

Balantidiosis

The ciliate *Balantidium coli* is a common inhabitant of the alimentary tract of the camels, which is occasionally associated with severe diarrhoea (Boid *et al*, 1985). Mainly cysts were detected in 30.21% of 897 camel faecal samples in Rajasthan, India (Partani *et al*, 1998). Higher incidence was recorded in farm camels than field animals. Four of the field camels had severe diarrhoea and were passing trophozoites. Tafti *et al* (2001) regarded balantidiosis (19%) as the second most prevalent (after *Eimeria* spp. infections) pathologic lesion of the digestive system in 100 slaughtered camels in Iran. Caecum and colon affected with *Balantidium* spp. displayed necrosis of the epithelium and penetration of trophozoites of the protozoa into the mucosa. In Ethiopia, *B. coli* was observed in 11.92% of the 260 faecal samples examined (Tekle and Abebe, 2001).

Other protozoa

Theileria-like bodies variously designated as *Theileria camelensis* and *T. dromedarii* had been reported in the past. However, the identification is doubtful since it was based only on piroplasm stage being found in the blood smears. Since the camel tick *Hyalomma dromedarii* is also found parasitising cattle and in endemic areas, can act as a vector of bovine *Theileria annulata*, it is quite likely that the *Theileria* bodies seen in camel blood were of bovine origin briefly surviving in the abnormal host. This aspect needs further evidence and research.

Wernery (1991) reported isolation of *Trichomonas foetus* from the uterus of breeding camels (24/68 camels which were barren for 1-4 years). One preputial washing of a camel bull which had served the herd was also found positive for *T. foetus*. The samples were negative for bacterial and fungal agents of abortion. Detection of an *Eperythrozoon* spp. From camel was reported once (Hussein *et al*, 1991). Chronic cryptosporidiosis in a bactrian camel (*Camelus bactrianus*) was described (Fayer *et al*, 1991). Considering its broad host range, it is quite likely to be more prevalent as natural infection in dromedarian camels also, and should be looked for in obscure cases of diarrhoea.

Concluding remarks

In spite of an impressive volume of work resulting in a better understanding of the important protozoal diseases of camels, especially trypanosomosis, there is an obvious need for further work on many aspects. Notwithstanding the technological advancements, a simple inexpensive and reliable pen-side test for detection of trypanosomosis still eludes us. Due to the limitationof antigenic variations discounting the option of vaccine development and keeping an eye on the drug resistance factor, new formulations of effective therapy would also need to be evolved at least in the short term. A shift from the pastoral to high-tech and intensive farm-rearing with resultant confinement, overcrowding, stress, lack of exercise and absence of high-browsing is likely to create conditions conducive for increase of coccidiosis, balantidiosis and other presently-less-menacing protozoa. Nevertheless, the need to include these entities in the overall parasitic disease surveillance among camels is undeniable.

References

Ali BH (1988). A survey of some drugs commonly used in the camel. Veterinary Research Communications 12:67-75.

Al-Rawashdeh OF, Al- Ani FK, Sharrif LA, Al- Qudah KM, Al- Hami Y and Frank N (2000). A survey of camel (*Camelus dromedarius*) diseases in Jordan. Journal of Zoo and Wildlife Medicine 31:335-338

Al-Sadi HI (1994). Pathology of some diseases of the one humped camel (*Camelus dromedarius*). Iraqi Journal of Veterinary Sciences 7:1-10.

Al-Taqi MM (1989). Characterisation of *Trypanosoma* (Trypanozoon) *evansi* from dromedary camels in Kuwait by isoenzyme electrophoresis. Veterinary Parasitology 32: 247-253.

Atarhouch T, Rami M, Bendahman MN and Dakkak A (2003). Camel trypanosomosis in Morocco 1: results of a first epidemiological survey. Veterinary Parasitology 111: 277-286.

Basagoudanavar SH, Rao JR, Omanwar S, Singh RK and Butchaiah G (1998). A sensitive polymerase chain reaction for detection of *Trypanosoma evansi* in camels (*Camelus dromedarius*). Journal of Parasitic Diseases 22: 40-45.

Baumann MPO, and Zessin KH (1992). Productivity and health of camels (*Camelus dromedarius*) in Somalia:associations with trypanosomosis and brucellosis. Tropical Animal Health and Production 24:145-156.

Boid R, Jones TW and Luckins AC (1985). The camel in health and disease. 3. Protozoal diseases. British Veterinary Journal 141:87-105.

Chaudhary ZI and Iqbal J (2000). Incidence, biochemical and haematological alterations induced by natural trypanosomosis in racing dromedary camels. Acta Tropica 77:209-213.

Dadhich H (2000). Histopathological observations of *Sarcocystis cameli* in the heart of dromedary (*Camelus dromedarius*). Acta Parasitologica 45:243-244.

Delafosse A and Doutoum AA (2000). Comparison of three serological tests for field diagnosis of camel surra (*Trypanosoma evansi* infection) in Chad. Revue d'Elevage et de Medecine Veterinaire des Pays Tropicaux 53:249 256.

Dia ML, Diop C, Thiam A, Aminetou M, and Jacquiet P (1997). Importance of camel trypanosomosis and its vectors in Mauritania. Journal of Camel Practice and Research 4: 271-276.

Diall O, Songa EB, Magnus E, Kouyate B, Diallo B, Meirvenne N van, Hamers R, Van Meirvenne N, Bajyana Songa E (1994). Evaluation of a direct serological card agglutination test in the diagnosis of camel trypanosomiasis due to *Trypanosoma evansi*. Revue Scientifique et Technique Office International des Epizooties 13:793-800.

Dirie MF, Wallbanks KR, Aden AA, Bornstein S and Ibrahim MD (1989). Camel trypanosomosis and its vectors in Somalia. Veterinary Parasitology 32:285-291.

Elamin EA, El-Bashir MOA and Saeed EMA (1998). Prevalence and infection pattern of *Trypanosoma evansi* in camels in mid-eastern Sudan. Tropical Animal Health and Production 30:107-114.

Elamin EA, Slias S, Dougsches A and Ronmel M (1992). Prevalence of Toxoplasma gondii antibodies in pastoral camels (*Camelus dromedarius*) in the Butana plains, mid-eastern Sudan. Veterinary Parasitology 43:171-175.

El-Rayah IE, Kaminsky R, Schmid C and El-Malik KH (1999). Drug resistance in Sudanese *Trypanosoma evansi*. Veterinary Parasitology 80:281-287.

Fayer R, Phillips L, Anderson BC and Bush M (1991). Chronic cryptosporidiosis in a bactrian camel (*Camelus bactrianus*). Journal of Zoo and Wildlife Medicine. 22: 228-232.

Gatt-Rutter TGE (1967). Disease of camels. 2. Protozoan Diseases. Veterinary Bulletin 37:611-618.

Gool F van, Zelleke D, Musa M and Van Gool F (1992). Cymelarsan: a new treatment for *Trypanosoma evansi* infections in camels. In: Allen, W. R., Higgins, A. J., Mayhew, I. G., Snow, D. H., Wade, J. F. (Eds.). Proceedings of the First International Camel Conference, Dubai (UAE), February 2-6. pp 37-41.

Gutierrez C, Corbera JA, Juste MC, Doreste F and Morales I (2005). An outbreak of abortions and high neonatal mortality associated with *Trypanosoma evansi* infection in dromedary camels in the Canary Islands. Veterinary Parasitology 130:163-168.

Gutierrez C, Juste MC, Corbera JA, Magnus E, Verloo D and Montoya JA (2000). Camel trypanosomosis in the Canary Islands: assessment of seroprevalence and infection rates using the card agglutination test (CATT/*T. evansi*) and parasite detection tests. Veterinary Parasitology 90:155-159.

Hagemoser WA, Dubey JP and Thompson JR (1990). Acute toxoplasmosis in a camel. Journal of the American Veterinary Medical Association 196:347.

Hamers R, Viseshakul N and Songa EB (1990). Identification and characterisation of *T. evansi* strains with DNA probes. Camel Newsletter 7:74.

Haroun, E M, El Mitnawi, T, Omer, OH, Ali, B H and Mahmoud, OM (2003). A preliminary comparative study on the efficacy of quinapyramine sulphate/ chloride and melarsoprol in rats, experimentally infected with *Trypanosoma evansi*. Bulgarian Journal of Veterinary Medicine 6:215-221.

Haroun EM, Magzoub M, Mahmoud OM, Al Qarawi AA, Al Hawas AM and Omer OH (2000). Some clinico-pathological aspects of experimental *Trypanosoma evansi* infection in Najdi camels (*Camelus dromedarius*). Journal of Camel Practice and Research 7:101-106.

Hilali M, Fatani A and Al-Atiya S (1995). Isolation of tissue cysts of *Toxoplasma*, *Isospora*, *Hammondia* and *Sarcocystis* from camel (*Camelus dromedarius*) meat in Saudi Arabia. Veterinary Parasitology 58:353-356.

Hilali M, Nassar AM and El-Ghaysh A (1992).Camel (*Camelus dromedarius*) and sheep (*Ovis aries*) meat as a source of dog infection with some coccidian parasites. Veterinary Parasitology 43:37-43.

Hilali M, Romand S, Thulliez P, Kwak OCH and Dubey, J.P. (1998). Prevalence of *Neospora caninum* and *Toxoplasma gondii* antibodies in sera from camels from Egypt. Veterinary Parasitology 75:269-271.

Hunter AG (1986). Urine odour in a camel suffering from surra (*T. evansi* infection). Tropical Animal Health and Production 18:146-148.

Hussein HS, Al Asgah NA, Al Khalifa MS and Diab FM (1991). The blood parasites of indigenous livestock in Saudi Arabia. Arab Gulf Journal of Scientific Research 9:143-160

Hussein MF, Bakkar MN, Basmeil SM and Gar-el-Nabi AR (1998). Prevalence of toxoplasmosis in Saudi Arabian camels. Veterinary Parasitology 28:175-178

Ijaz MK, Nur e Kamal MSA, Mohamed AIA and Dar FK (1998). Comparative studies on the sensitivity of polymerase chain reaction and microscopic examination for the detection of *Trypanosoma evansi* in experimentally infected mice. Preventive Veterinary Medicine 38:289-293.

Jain S, Pareek R, Pathak KML and Kapoor M (2000). Comparison of six different tests for the detection of *Trypanosoma evansi* in dromedaries. Journal of Camel Practice and Research 7:215 217.

Karram MH, Ibrahim H, Ali TSA, Manaa AM and Abdel Ali TS (1991). Clinical and haematological changes in camel infested with *Trypanosoma evansi* and microfilaria. Assiut Veterinary Medical Journal 25:118-128

Kinne J and Wernery U (1998). Pathological studies on camel coccidiosis in the United Arab Emirates. Proc. 3rd Annual Meeting for Animal Production under Arid conditions. Al-Ain, UAE, May 2-3, 1998, pp 131-142.

Kirmse P and Mohanbabu B (1986). Sarcosystis sp. in the one-humped camel (*Camelus dromedarius*) from Afghanistan. British Veterinary Journal 142:73-74.

Kohler-Rollefson I (1994). Ethnoveterinary practices of camel pastoralists in northern Africa and India. Journal of Camel Practice and Research 1:75-82.

Luckins AG (1988). *Trypanosoma evansi* in Asia. Parasitology Today 4:137-142.

Luckins AG (1992). Protozoal diseases of camels. In: Allen W R, Higgins A J, Mayhew IG, Snow DH, Wade JF (Eds). Proceedings of the First International Camel Conference. pp 23-27.

Maina N, Ngotho JM, Njiru ZK, Karanja WM, Gem CO, Karanja SM, Kibugu JK and Ndung'u JM (2003).

Efficacy of Trypan(R) (diminazene diaceturate) in camels infected with *Trypanosoma evansi*. Journal of Camel Practice and Research 10:51-55.

Masiga RC and Nyang'ao JMN(2001). Identification of trypanosome species from camel using polymerase chain reaction and procyclic transformation test. Journal of Camel Practice and Research 8:17-22.

Mochabo KOM, Kitala PM, Gathura PB, Ogara WO, Catley A, Eregae EM and Kaitho TD (2005). Community perceptions of important camel diseases in Lapur Division of Turkana District, Kenya. Tropical animal Health and Production 37:187-204.

Nantulya VM (1994). Suratex: a simple latex agglutination antigen test for diagnosis of *Trypanosoma evansi* infections (Surra). Tropical Medicine and Parasitology 45:9-12.

Nantulya VM, Lindqvist KJ, Diall O and Olaho Mukani W (1989). Two simple antigen detection enzyme immunoassays for the diagnosis of *Trypanosoma evansi* infections in the dromedary camel (*Camelus dromedarius*). Tropical Medicine and Parasitology 40:415-418.

Nassar AM, Hilali M, and Rommel M (1983). *Hammondia heydorni* infection in camels (*Camelus dromedarius*) and water buffaloes (*Bubalus bubalis*) in Egypt. Zeitschrift fur Parasitenkunde 69:693-694.

Nessiem MG (1994). Evaluation of the silicon centrifugation technique in the detection of *Trypanosoma evansi* infection in camels and experimental animals. Tropical Animal Health and Production 26:227-229.

Ngaira JM, Bett B and Karanja SM (2002). Animal level risk factors for *Trypanosoma evansi* infection in camels in eastern and central parts of Kenya. Onderstepoort Journal of Veterinary Research 69:263-271.

Njiru ZK (1997). In vitro function of dromedary granulocytes during experimental infection with *Trypanosoma evansi*. Journal of Camel Practice and Research 4:283-286.

Njiru ZK, Olaho Mukani W, Khaemba BM, Ochieng RS and Ndung' u JM (2000a). Haematological and serological changes during acute *Trypanosoma evansi* infection in dromedary camels (*Camelus dromedarius*). Journal of Camel Practice and Research 7:113-116.

Njiru ZK, Ole Mapeny IM, Ouma JO, Ndung' u JM and Olaho Mukani W (2000b). Prevalence of trypanosomosis in camel calves: a pilot study in Laikipia District of Kenya. Revue d'Elevage et de Medecine Veterinaire des Pays Tropicaux 53:183-186.

Njiru ZK, Bett B, Ole Mapeny IM, Githiori JB and Ndung' u JM (2002).Trypanosomosis and helminthosis in camels: comparison of ranch and traditional camel management systems in Kenya. Journal of Camel Practice and Research 9:67-71.

Nyangao JM, Olaho-Mukani W, Maribei JM and Omuse JK (1995). Evaluation of the efficacy of melarsenoxyde cysteamine (Cymelarsan) in treatment of camels experimentally infected with *Trypanosoma evansi* using antigen trapping enzyme linked immunosorbent assay. Journal of Veterinary Pharmacology and Therapeutics 18:468-470.

OIE (2000). Surra (*Trypanosoma evansi*). In: Manual of Standards for Diagnostic Tests and Vaccines. Office International des Epizooties, Paris. pp 891-900.

Olaho Mukani W, Munyua WK, Njogu AR, Mutugi MW, Omuse JK and Sayer PD (1992). Application of an antigen enzyme linked immunosorbent assay for the diagnosis of trypanosomosis in camels in Kenya. In: Allen WR, Higgins AJ, Mayhew IG, Snow DH, Wade JF (Eds.). Proceedings of the First International Camel Conference. 2-6 February, Dubai (UAE). pp 33-36.

Olaho-Mukani W, Nyangao JM and Ouma JO (1995). Elimination of drug-resistant *Trypanosoma evansi* in a group of camels by sequential treatment with melarsomine. Journal of Camel Practice and Research 2:83-85.

Olaho Mukani W, Nyang'ao JMN and Ouma JO (1996). Comparison of Suratex ^{R}, parasite detection and antigen ELISA for the evaluation of treatment efficacy and diagnosis of surra in dromedary camels. Journal of Camel Practice and Research 3:1-5.

Olaho Mukani W, Mboloi MM, Muriuki SP, Ouma JO, Guya SO and Ndung'u JM (1997). Application of pen side diagnosis in the control of surra in dromedary camels in Kenya. Journal of Camel Practice and Research 4:281-282.

Omanwar S, Rao JR, Singh RK and Butchaiah G (2001). DNA polymorphism in *Trypanosoma evansi* isolates defined by randomly amplified polymorphic DNA PCR. Veterinary Record 148:244-246.

Otsyula M, Kamar K, Mutugi M and Njogu AR (1992). Preliminary efficacy trial of Cymelarsan, a novel trypanocide, in camels naturally infected with *Trypanosoma evansi* in Kenya. Acta Tropica 50:271-273.

Ouma JO, Olaho-Mukani W, Wisheteni BEL and Guya SO (1997). Changes in classical pathway complement activity in dromedary camels experimentally infected with *Trypanosoma evansi*. Veterinary Immunology and Immunopathology 57:135-140.

Oyieke FA (2003). Occurrence and transmission of camel trypanosomiasis in Northern Kenya. Journal of Camel Practice and Research 10:17-21.

Partani AK, Kumar D. and Bhan AK (1998). Prevalence of *Balantidium coli* infection in camels at Bikaner, Rajasthan. Journal of Veterinary Parasitology 12:118-121.

Partani AK, Kumar D and Monohar GS (1999). Prevalence of *Eimeria* infection in camels (*Camelus dromedarius*) at Bikaner (Rajasthan). Journal of Camel Practice and Research 6:69-71.

Pathak KML, Arora JK and Kapoor M (1993). Camel trypanosomosis in Rajasthan, India. Veterinary Parasitology 49:319-323.

Pathak KML, Arora JK, Sahal P and Kapoor M (1994). Fractionation and characterisation of somatic antigen of *Trypanosoma evansi*. Indian Veterinary Journal 71:218-221.

Pathak KML, Yadvendra Singh, Meirvenne NV, Kapoor M and Singh Y (1997). Evaluation of various diagnostic techniques for *Trypanosoma evansi* infections in naturally infected camels. Veterinary Parasitology 69:49-54.

Pethkar DK and Momin RR (1990). Studies on incidence of intestinal protozoan infections in camel. Abstr. First Asian Congress of Veterinary Parasitology, Patna, India. 26-28 November, pp 48.

Rae PF and Luckins AG (1992). Problems in the diagnosis of cameline trypanosomosis. In: Allen WR, Higgins AJ, Mayhew IG, Snow DH, Wade JF (Eds). Proceedings of the First International Camel Conference, Dubai. February 2-6, 1992, pp 29-31.

Rae PF, Thrusfield MV, Higgins A, Aitken CGG, Jones TW and Luckins AG (1989). Evaluation of enzyme immunoassay in the diagnosis of camel (*Camelus dromedarius*) trypanosomosis: a preliminary investigation. Epidemiology and Infection 102:297-307.

Raina AK, Sarup S, and Sharma RD (1990).Note on chemotherapeutic efficacy of Tevansi (quinapyramine prosalt) and quinapyramine sulphate in *Trypanosoma evansi* infection in rats. Livestock Adviser 15:12-14.

Raisinghani PM and Lodha KR (1989). Incidence of *Trypanosoma evansi* infection in camels of Rajasthan. Indian Journal of Animal Sciences 59:1390-1392.

Raisinghani PM, Manohar GS and Yadav JS (1987). *Isospora* infection in the Indian camel (*Camelus dromedarius*). Indian Journal of Parasitology 11:93-94.

Rami M, Atarhouch T, Bendahman MN, Azlaf R, Kechna R, and Dakkak A (2003). Camel trypanosomosis in Morocco: 2. A pilot disease control trial. Veterinary Parasitology 115:223-231.

Reid SA, Dargantes A, Hussein A and Constantine CC (2003). Geographic variation in the pathogenicity of *Trypanosoma evansi*. Minutes of the 24th Annual meeting of the OIE ad hoc group on non-tsetse transmitted animal trypanosomioses, Paris, France, 18th May 2003.

Reid SA, Hussein A and Copeman DB (2001). Evaluation and improvement of parasitological tests for *Trypanosoma evansi* infection. Veterinary Parasitology 102:291-297.

Samy M and Gadir FA (1991). Pathomorphological and clinicopathological studies on camel trypanosomiasis in United Arab Emirates. Egyptian Journal of Comparative Pathology and Clinical Pathology 4:47-56.

Shahardar RA, Mishra AK and Rao JR (2002). Dot ELISA for detection of antibodies against *Trypanosoma evansi* in dromedary camels. Journal of Veterinary Parasitology 16:163-164.

Shahardar RA, Rao JR and Mishra AK (2003). Detection of antibodies against *Trypanosoma evansi* in dromedary camels by competitive inhibition enzyme linked immunosorbent assay. Journal of Applied Animal Research 24:41-48.

Shahardar RA, Mishra AK and Rao JR (2004).Detection of antibodies against *Trypanosoma evansi* in dromedary camels by ELISA using solubilised antigens. Indian Journal of Animal Sciences 74:3-6

Singh N, Pathak KML and Kumar, R (2004). A comparative evaluation of parasitological, serological and DNA amplification methods for diagnosis of natural *Trypanosoma evansi* infection in camels. Veterinary Parasitology 126:365-373.

Singh V, Raisinghani PM, Lodha KR and Manohar GS (1987). Trypanocidal value of Tevansi in experimental *Trypanosoma evansi* infection in buffalo calves. Indian Veterinary Journal 64:467-470.

Singh V, Singh A and Chhabra MB (1994a). Polypeptide profiles of whole cell lysate of *Trypanosoma evansi* stocks from northern India. Indian Journal of Animal Sciences 64:14-17.

Singh V, Gahlot AK and Chhabra MB (1994b). Evaluation of some sero diagnostic tests for *Trypanosoma evansi* infection in camel. Journal of Camel Practice and Research 1:30-33.

Singh V, Singh A and Chhabra MB (1995). Polypeptide profiles and antigenic characterisation of cell membrane and flagellar preparations of different stocks of *Trypanosoma evansi*. Veterinary Parasitology 56:269-279.

Tafti AK, Maleki M and Oryan A (2001). Pathological study of intestines and mesenteric lymph nodes of camels (*Camelus dromedarius*) slaughtered in Iran. Journal of Camel Practice and Research 8:209-213.

Tekle T and Abebe G (2001). Trypanosomosis and helminthoses: major health problems of camels (*Camelus dromedarius*) in the Southern Rangelands of Borena, Ethiopia. Journal of Camel Practice and Research 8:39-42.

Waitumbi JN and Nantulya VM (1993). A comparison of the antigen detection ELISA and parasite detection for diagnosis of *Trypanosoma evansi* infections in camels. Veterinary Parasitology 49:159-178.

Waitumbi JN and Young JR (1994). Electrophoretic karyotyping is a sensitive epidemiological tool for studying *Trypanosoma evansi* infections. Veterinary Parasitology 52:47-56.

Warrag M and Hussein HS (1983). The camel (*Camelus dromedarius*) as intermediate host for *Hammondia heydorni*. Journal of Parasitology 69:926-929.

Wernery U (1991). The barren camel with endometritis - isolation of *Trichomonas foetus* and different bacteria. Journal of Veterinary Medicine. Series B 38:523-528.

Wernery U and Kaaden OR (1995). Infectious Diseases of Camelids. Blackwell Wissenschafts-Verlag, Berlin.

Wernery U and Kaaden OR (2002). Infectious Diseases of Camelids. 2nd Edn, Blackwell Science Berlin, Vienna. pp 272-303.

Wernery U, Zachariah R, Mumford JA and Luckins T (2001). Preliminary evaluation of diagnostic tests using horses experimentally infected with *Trypanosoma evansi*. Veterinary Journal 161:287-300.

Woldemeskel M and Gumi B (2001). Prevalence of sarcocysts in one humped camel (*Camelus dromedarius*) from Southern Ethiopia. Journal of Veterinary Medicine. Series B 48:223-226.

Woldemeskel M, Issa A, Mersie A and Potgieter LND (2001). Investigation of parasitic diseases of one-humped camel (*Camelus dromedarius*) in eastern Ethiopia. Journal of Camel Practice and Research 8:77-81.

Yagoub IA (1989). Haematological studies in dromedary camels with single or concurrent natural infections of *Trypanosoma evansi* and *Haemonchus longistipes*. Acta Veterinaria Beograd 39:109-119.

Younan M, McDonough SP, Herbert D, Saez J and Kibor A (2002). *Isospora* excretion in scouring camel calves (*Camelus dromedarius*). Veterinary Record 151:548-549.

Zarif-fard MR and Hashemi-Fesharki R (2000). Study on tissue and blood protozoa of camels in southern Iran. Journal of Camel Practice and Research 7:193-194.

Zelleke D, Kassa B and Abebe S (1989). Efficacy of RM110, a novel trypanocide, in the treatment of *Trypanosoma evansi* infections in camels. Tropical Animal Health and Production 21:223-226.

Zelleke D, Musa MM and Gool F van (1992). Cymelarsan-A new treatment for *Trypanosoma evansi* infections in camels : Results of field trials in Africa. Proceedings of the First International Camel Conference, Dubai. February 2-6, 1992, pp 37-41.