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Insight into Trypanosomiasis in Animals: Various Approaches for its Diagnosis, Treatment and Control: A Review

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ABSTRACT

Trypanosomiasis is a haemoprotozoan disease entity caused by various members of *Trypanosoma* spp. affecting different species of domestic and wild animals like horses, mule, donkey, camel, cattle, buffaloes, sheep, goat, dogs, pig, elephant, deer, foxes, tiger and jackals with chief clinical signs of high intermittent fever, anaemia, loss of weight, edema of dependent parts, nervous symptoms, abortion and is responsible for major production losses. It can be transmitted through the biting flies wherein, the parasite may undergo biological or mechanical transmission. Various diagnostic methods from traditional to molecular are available for its diagnosis viz., microscopic examination, DNA detection by PCR, Card Agglutination Test and ELISA. The present review addresses important insights into trypanosomiasis, the etiological agent, advances and trends in its diagnosis, treatment aspects, prevention and control measures; which would help in limiting its prevalence in animals.

Key words: Trypanosomes, host range, clinical signs, treatment, diagnosis, prevention and control

INTRODUCTION

By 2050, there will be requirement of approximately 50% increase in the production of food for the consumption of human beings (Mahima *et al*., 2012). This will not be possible without the animal food. At present, animals are used to be affected by bacteria, viruses, fungi, parasitic infections etc. Among these blood protozoans play a significant effect in reducing the production by the animals. Trypanosomiasis is a haemoprotozoan disease entity caused by various members of *Trypanosoma* spp. and is transmitted through the biting flies wherein the parasite may undergo biological or mechanical transmission. It infects various species of domestic and wild animals like horses, mule, donkey, camel, cattle, buffaloes, sheep, goat, dogs, pig, elephant, deer, foxes, tiger and jackals with chief clinical signs of high intermittent fever, anaemia, loss of weight, edema of dependent parts, nervous symptoms, abortion and is responsible for major production losses. Animal trypanosomiasis is now a days considered as a permanent constraint for livestock productivity in Africa, Asia and Latin America with their geographical distribution still evolving (Desquesnes *et al*., 2013a). *Trypanosoma evansi* (Trypanozoon) was the first pathogenic mammalian trypanosome discovered by Griffith Evans in 1980, from the blood of Indian camel and later from the blood of Indian equines (Hoare, 1972). *T. evansi* is thought to be derived from

T. brucei (a cyclically transmitted trypanosome by tsetse flies), but parasite is no longer able to undergo its biological cycle in Glossina fly because of the loss of the maxicircles of kinetoplastic mitochondrial DNA (Borst *et al*., 1987; Lun and Desser, 1995; Lai *et al*., 2008). In the Indian sub-continent, the disease is mainly endemic and most of the epizootics have occurred particularly in bovines with a high mortality rate ranging from 20-90% (Gill, 1991). The literature on trypanosomiasis in Indian livestock has been extensively described in a classic monograph published by the ICAR (Gill, 1991). However, after two decades since its publication, a large volume of literature has come up on various aspects of trypanosomiasis.

The present review addresses important insights into trypanosomiasis, the etiological agent, advances and trends in its diagnosis, treatment aspects, prevention and control measures; which would help in limiting its prevalence in animals.

Etiology: In fresh blood smears, *T. evansi* is peculiar with slender shape having thin posterior extremity and free flagellum. Parasite has well developed and highly visible undulating membrane. Parasite shows active movement with limited displacements in microscope field in fresh wet mount smears. In stained thin blood smears, the parasite is always described as a monomorphic thin trypomastigote form of 15-33 μm in size, with a long free flagellum and thin posterior extremity and a small subterminal kinetoplast. Sometimes, intermediate forms of parasite with shorter free flagellum and almost terminal kinetoplast are also observed. In some cases small stumpy forms of parasite are also reported but with an inconsistent feature (Hoare, 1972).

Antigenic variation and characterization of *T. evansi*: Trypanosomes have ability to evade host immune response by altering the antigenic composition of its surface glycoprotein coat. Studies on fractionation chromatography revealed that the parasite has 7 distinct fractions in camel with molecular weights ranging between 14-65 kDa (Pathak *et al.,* 1994). Characterization of 7 stocks of *T. evansi* sourced from buffaloes, equids and camel from different localities of north India was carried out (Singh *et al*., 1994). The polypeptide profiling of Whole Cell Lysate (WCL) by SDS-PAGE (Singh *et al*., 1994) and of Cell Membrane (CM) and flagellar preparations by SDS-PAGE and Western blotting (Singh *et al*., 1995b) did not indicate antigenic variability of high degrees. DNA polymorphism in *T. evansi* isolates was studied by Polymerase Chain Reaction (PCR) by Omanwar *et al*. (2001) and hypothesized the adaptability of the organism to different hosts and geographical locations as the reason for polymorphism. Existence of genetic heterogeneity within *T. evansi* isolates derived from buffalo, dog, horse and camel revealed by polymerase chain reaction seems to confirm DNA-polymorphism hypothesis (Kundu *et al*., 2010).

Epidemiology: *T. evansi* is very widespread and affects all African countries, Arabian Peninsula, Iran, Kasakhstan, Afghanistan, Pakistan, India, China, Mongolia, Russia, Bhutan, Nepal, Myanmar, Laos, Vietnam, Cambodia, Thailand, Malaysia, Philippines and Indonesia (Luckins, 1988; Reid, 2002). Parasite is also distributed in Western countries Latin America, Central America to Mexico and Argentina to Panama Spain (Wells, 1972; Gutierrez *et al*., 2006). The distribution of parasite is suspected in Papua New Guinea (Reid *et al*., 1999). Surra in India is very old with records dating back from VIII centuries B.C. (Hoare, 1972) with prevalence in almost all over the country, where environment for the breeding of the fly vectors is most suitable (Bhatia *et al*., 2006). Incidences of trypanosomiasis outbreaks have been reported to be increased in camels after the advent of Indira Gandhi Canal and irrigation of vast tracts of arid

land in Western Rajasthan (Pathak and Khanna, 1995). Again the incidence of trypanosomiasis in bovines was found to be directly proportional to onset of monsoon to post monsoon in Punjab (Soodan *et al*., 1995), Andhra Pradesh (Prasad *et al*., 1997), West Bengal (Ray *et al*., 1992), Bihar (Sinha *et al*., 2006), Chhattisgarh (Agrawal *et al*., 2003) and Jammu (Raina *et al*., 2000). The disease is mostly asymptomatic but factors like flooding, intercurrent disease (Gupta *et al*., 2009), vaccination (Singla *et al*., 2010), transport (Kalra *et al*., 1994) and malnutrition (Malik *et al*., 2000) often changes an unapparent infection into clinical disease.

Host range: Parasite mainly affects member of camelidae and equidae but have widest host range amongst the salivarian trypanosomes and covers a variety of domestic and wild animals like horses, mule, donkey, camel, cattle, buffaloes, sheep, goat, dogs, pig, elephant, deer, foxes, tiger and jackals (Pathak and Singh, 2005). Outbreaks of acute trypanosomiasis in cattle and buffaloes have been reported from Haryana and Punjab (Batra *et al*., 1994; Gupta *et al*., 2003; Jindal *et al*., 2005). The incidence of bovine trypanosomiasis was found to be fairly high in cattle (58.86%) than buffaloes (41.14%) in Bihar (Sinha *et al*., 2006). Low level of prevalence in cattle (1.42%) and buffaloes (2.71%) was reported in Guntur district (Das *et al*., 1998) and in East Godavari (7.28%) districts of Andhra Pradesh (Bhaskara and Hafeez, 2005). On the basis of a performa developed and sent to field veterinarians, the prevalence was reported to be 42.12% in bovines, 39.78% in buffaloes and 2.15% in goats in Karnataka (Krishnappa *et al*., 2002). Among equines, a field outbreak with 100% morbidity and 66.6% mortality was reported from Mathura (Kumar *et al*., 1994). Again, outbreak in ponies was reported from Jammu (Raina *et al*., 2000). Surra in a Kathiawari mare has been reported from Udgir, Maharashtra (Bharkad *et al*., 2005). Cameline trypanosomiasis was found to be endemic in 18 districts of Rajasthan (Raisinghani and Lodha, 1989) with prevalence of (7.5%) by the wet-blood/Giemsa stain smears and 76 (31.66%) positive for antigen using double antibody sandwich ELISA (Pathak *et al*., 1993) from Western Rajasthan. On the bases of clinical observations in camels alone, a prevalence rate of 20.37% has been reported from Bikaner district of Rajasthan (Singh *et al*., 1997). Amongst dogs, 4.68% prevalence was found in Ludhiana (Singh *et al*., 1993). Incidence among dogs in and around Kolkata city was found somewhat lesser (Chowdhury *et al*., 2005). Exotic breeds are found to be more susceptible and usually experience acute fatal disease (Dakshinkar and Bhojne, 2001). Cases of trypanosomiasis have also been report from native dog breeds (Krishnamoorthy and Manohar, 2005). In sheep (Rao *et al*., 1987) and goats, reports of trypanosomiasis are scare (Rao and Hafeez, 1999; Jana and Jana, 2005) with uncommon apparent infections (Gill, 1991). Among zoo animals, incidence in Chitals in Bhilai zoo and hyena in Delhi zoo were reported (Arora, 1994). An outbreak of 'surra' in tigers at Ranthambore National Park with a fatal case report in a male tiger (Ramachandraiah *et al*., 1995) and an outbreak in circus tigers in Andhra Pradesh (Rao *et al*., 1995) have also been described. Case reports on circus tigress was reported from Chittoor, Andhra Pradesh (Devasena and Shobhamani, 2006) and on jungle cat (Felis chaus) from Nagpur zoo (Dakshinkar *et al*., 2002). The high incidence of trypanosomiasis in dogs and wild carnivores due to feeding of infected carcass is suggested to be an important mode of transmission (Bhatia *et al*., 2006). Trypanosomiasis in mithun (Bos frontalis) has been reported in Assam (Rajkhowa *et al*., 2003).

Clinical signs: The classical clinical signs of surra include intermittent fever, anemia mainly due to haemolysis of red blood cells as well as erythrophagocytosis (Bhatia *et al*., 2006), loss of appetite and weight, loss of condition, production losses, nervous symptoms, cachexia and death with or

without peculiar signs related to the host species (Gardiner and Mahmoud, 1990). Sometimes abortion may also be recorded. These clinical signs are observed in affected animal with variable intensity related to host species from unapparent to strong. The typical clinical signs and pathogenic effect of surra are observed in camel and equines although some different pathogenic effect and clinical signs are also observed in different host species.

In cattle and buffaloes, trypanosomiasis is an asymptomatic carrier state with occasional acute or peracute infection with death or pronounced symptoms. The observed clinical signs in cattle and buffaloes are mild and the disease is chronic in Africa and Latin America. Although in Asia acute form of disease is noticed with intermittent fever, anemia, loss of weight and production losses (Payne *et al*., 1993). A slower course of disease shows, dullness with recumbency or staggering gait, labored breathing, lachrymation, bellowing, profuse salivation, icterus, twitching of muscles often terminating in convulsions and death (Palanivel *et al*., 2008). Lumbar paralysis was reported in subclinical cases of trypanosomiasis in buffaloes from Bihar (Kumar *et al*., 2009b). In chronic cases, dullness, lachrymation, intermittent fever, anaemia, oedema in dependent part of body and progressive emaciation may occur (Muraleedharan and Srinivas, 1985; Rajguru *et al*., 2000). Postmortem lesions include hepatomegaly, splenomegaly, congestion of lungs, liquefaction of sub-epicardial fat, serious fluid in the plural and pericardial cavities, hemorrhages and petechiae (Palanivel *et al*., 2008). Transplacental transmissions of trypanosomiasis are also reported in cows (Kalra *et al*., 1994; Rajguru *et al*., 2000) and buffaloes (Rao *et al*., 2001). Chronic trypanosomiasis in crossbred cattle is also reported from an organized dairy farm in Punjab (Bharadwaj and Randhawa, 2010).

Infection in sheep and goats is generally considered as mild or asymptomatic in sheep. Unilateral superficial corneal ulceration and retinochoroiditis but without apparent loss of vision were observed in artificially infected goats (Morales *et al*., 2006).

Both acute and chronic forms of surra noticed in camel. Acute cases diagnosed with high fever, anaemia, weakness, emaciation and death. Chronic cases are more often and debilitating course of disease lasts for three years (tibrsa) with clinical signs of intermittent fever, dullness, progressive weakness, loss of appetite, loss of weight, oedema in ventral parts, anaemia and petechial hemorrhages on mucous membranes. Young animals are more affected, although disease found in all age groups. A special feature to camels may be the enlargement and suppuration of lymph glands of the inguinal region. Abortion and high neonatal mortality was also associated with *Trypanosoma evansi* infection in dromedary camels in the Canary Islands (Gutierrez *et al*., 2005). The affected camel urine gives a specific odour which is helpful for diagnosis of surra (Stephen, 1986) but not specific.

Equines are another species which shows classical clinical signs. Among equines horse is highly susceptible although mule, donkey and ass show lower susceptibility. Animal show intermittent fever, weakness, lethargy, anemia, severe weight loss, local or general cutaneous eruption, petechial hemorrhages on nictitating membrane, vulvar and vaginal mucosa, into the anterior chamber of the eye, abortion, locomotive disturbance, nervous signs and edema of lower part of body mainly reproductive organs , testicles, legs and lower abdomen. In chronic cases there is weight loss, anemia and jaundice with dark yellow colored urine (Jani and Jani, 1993; Varshney and Gupta, 1996; Laha *et al*., 2004). Hepatic insult (Varshney and Gupta, 1996) and endocrine dysfunctions (Varshney *et al*., 1999) were also reported in clinical cases of trypanosomiasis in horses. Generalized, nonsuppurative meningoencephalitis in white and gray matter of the brain may be observed (Seiler *et al*., 1981). Transplacental transmission in a donkey mare was reported (Pathak and Kapoor, 1999).

Clinical signs in pigs are symptomless or very mild with fever, anorexia, emaciation, abortion (Arunasalam *et al*., 1995) and low fertility in Thailand (Songa *et al*., 1987).

Dogs are highly susceptible to *T. evansi*, show clinical signs of intermittent fever, oedema of the head and larynx, abdominal wall and legs, anaemia, lymphadenopathy, tachycardia, weakness, muscular spasms, emaciation, paralysis of the hindquarters and myocarditis. Hepatic insult (Varshney *et al*., 1998) and jaundice (Baby *et al*., 2000) were also noted. Sometimes, ocular signs with conjunctivitis, lachrymation and keratitis may be seen. Corneal opacity is a cardinal sign and often bilateral can be seen. Haemato-biochemical changes included hypoglycemia, hypoalbuminemia, hyperglobinaemia and hyperkalemia (Sarvanan *et al*., 2005). Haemato-biochemical changes include hypoglycemia, hypoalbuminemia, hyperglobinaemia and hyperkalemia (Sarvanan *et al*., 2005). Concurrent infections of *T. evansi* with ehrlichiosis (Balachandran *et al*., 2007) and *T. evansi* with *Hepatozoon canis* (Pazhanivel *et al*., 2008) were also reported. Immunosupression is another phenomenon in trypanosomiasis, which is responsible for the development of intercurrent diseases (Mansfield and Wallace, 1974). Beside dogs, other carnivores are equally affected by *T. evansi*.

Cat shows general clinical signs similar to those in dogs except that the edema developed in the head and corneal opacity was accompanied by bilateral epiphora and photophobia (Thirunavukkarasu *et al*., 2000). Pathology of *T. evansi* in tiger was elaborated by Manohar *et al*. (2003) in similar manner. Concurrent infection of trypanosomiasis with tuberculosis in black bucks was reported from a deer park in Punjab (Gupta *et al*., 2009).

Diagnosis: Although clinical signs of surra are indicative, but conformed diagnosis is required for treatment, management and further studies. Various diagnostic methods from traditional to molecular are available for the diagnosis of surra. Diagnosis techniques for surra are based on microscopic examination (Tewari *et al*., 2013), DNA detection by PCR (Holland *et al*., 2001; Desquesnes and Davila, 2002; Parashar *et al*., 2015; Sudan *et al*., 2014), Card Agglutination Test and ELISA (Sudan *et al*., 2015). Every method has its own advantages and limitations. Mainly blood but sometimes other biological materials such as cerebrospinal fluid (in case on nervous signs), synovial fluid or lymph node material can also be used for collection and examination of parasites. Microscopic examination observation of fresh blood can be easily carried out by wet mount of blood, thin blood smear, thick blood smear but have low sensitivity with detects parasites above 10^5 trypanosomes per milliliter of blood. More than 50-80% of the infections are cryptic and undetectable by direct microscopy; therefore these methods are not sufficient to know the epidemiology and magnitude of the surra in country. Although, these methods are not sensitive but easy to conduct in field and less instruments required. Concentration methods like Hematocrit Centrifuge Technique (Woo, 1969) or dark ground Buffy Coat Method (Murray *et al*., 1977) increase the sensitivity of the test down to 100-200 trypanosomes per milliliter. The concentration method is low cost alternative of direct microscopy. Furthermore, the sensitivity of parasite detection can be enhanced by 10 folds by using buffy coat instead of whole blood. Mouse innoculation test used for cryptic trypanosomes with high sensitivity with 20-50 parasites per milliliter and considered the most efficient parasitological test for the diagnosis of scanty trypanosomes (Chaudhri *et al*., 1996; Singh *et al*., 2003). Jain *et al*. (2000) reported that mouse inoculation test have highest efficacy but only 86.23%.

Molecular diagnosis of *T. evansi* can be done by using Polymerase Chain Reaction (PCR) with a number of primers specific for the subgenus Trypanozoon, or to species levels (Desquesnes and

Davila, 2002). This PCR based detection of *T. evensi* is very sensitive but not validated in field (Claes *et al*., 2004). Comparative studies recommended that TBR primers is the most sensitive primers for detecting *T. evansi* (Pruvot *et al*., 2010) and the Phenol-Chloroform method is the most sensitive method for DNA isolation (Pruvot *et al*., 2013) and this can detect as less as 5-10 trypanosomes per milliliter of blood. In India, PCR was first employed (Basagoudanvar *et al*., 1998) for detection of *T. evansi* in infected camel blood samples. The process of DNA isolation (Jithendran *et al*., 1998) and application for detection of *T. evansi* was further standardized (Omanwar *et al*., 1999). This technique was reported as more sensitive after an experiment conducted on 217 camels in Rajasthan (Singh *et al*., 2004) and in captive and wild animals (Shailaja *et al*., 2005). Shahardar *et al*. (2009) targeted ribosomal DNA for detection of *T. evansi* in Indian dromedary.

Card Agglutination test and ELISA are based on antigen antibody reactions and helpful for the diagnosis of *T. evensi*. Card Agglutation test used to detect immunoglobulin M therefore, early infections can be diagnosed by this test, whereas, ELISA is generally used to detect immunoglobulin G, so used for established infections. This test provides the same range of sensitivity and 90-95% specificity in the various host species. ELISA and its antigen-detection variant were found more sensitive and specific for trypanosomes (Singh *et al*., 1995a; Jithendran *et al*., 1997; Jain *et al*., 2000; Singh and Choudhri, 2002). Sandwich-ELISA used for detection of circulating antigens in cattle and buffaloes with efficacy of 37.8% (Jeyabal *et al*., 2003). Dot-ELISA (Shahardar *et al*., 2002) and competition-inhibition ELISA (Shahardar *et al*., 2003) used with 46.66-63.0%, positivity, respectively to detect antibody in camel sera from an endemic area in Rajasthan. Detergent-solubilized antigen further increase the sensitivity to 68.8% (Shahardar *et al*., 2004). Card Agglutation test is highly sensitive in camels and horses, but has only 12% sensitivity in cattle (Desquesnes *et al*., 2011). Latex agglutination test was developed as rapid diagnosis of *T. evansi* antigens in field in different animals in Haryana (Rayulu *et al*., 2009). In India, efficacy of some other immunological tests viz. capillary tube agglutination and gel diffusion (Soodan *et al*., 1995), IFAT (Ray *et al*., 1992), counter immuno-electrophoresis (Ghorui and Samanta, 1998) could be determined with variable results.

Treatment: Various chemical compounds are being used for the treatment of trypanosomiasis. Among different compounds, the most widely used trypanocide compound is diminazene aceturate. Besides diminazene aceturate, other chemical compounds like isometamidium chloride, suramin, quinapyramine sulphate (curative), quinapyramine chloride (prophylactic) and cymelarsan (only for camel) are also available. The recommended drug for trypanosomiasis in buffalo, cattle, sheep and goats is diminazene aceturate with dose rate of 7 mg kg^{-1} deep intramuscularly. Resistance against diminazene aceturate in trypanosomes has been reported in different parts of the world (Desquesnes, 2004; Peregrine and Mamman, 1993). If animal not respond then, isometamidium chloride or melarsomine hydrochloride may be used at dose rate of 0.5 mg kg^{-1} b.wt. deep intramuscularly (Desquesnes, 2004). Alternate use of diminazene aceturate and isometamidium chloride was recommended, as these make a sanative pair, means that once resistance develops to one of the drugs, the other drug used to control the infection. Horses, dogs, and cats can be treated with diminazene aceturate or isometamidium chloride but adequate water should be provided to animal to avoid a toxic effect on the kidneys. Melarsomine dihydrochloride is the best drug for camel at the dose rate of 0.25-0.5 mg kg^{-1} b.wt. although other trypanocide

drugs could also be used (Desquesnes *et al*., 2013b). Melarsomine dihydrochloride is not recommended in buffaloes if treated with 0.75 mg kg^{-1} body weight because chances of development of nervous signs.

The drugs used for trypanosomiasis in India are diminazene and quinapyramine and most trial reports are based on only these two drugs while, suramin and cymelarsan are not commercialized. Comparative evaluation of diminazene, suramin, quinapyramine and isometamidium in buffaloes naturally infected with *T. evansi* was conducted and it was found that quinapyramine (4.4 mg kg^{-1} b.wt.) and isometamidium (0.5 mg kg^{-1} b.wt.) have good therapeutic activity while, quinapyramine was found better than isometamidium for prophylaxis. A study conducted on buffaloes infected clinically with *T. evansi* proved prophylactic efficacy of quinapyramine and suramin was similar (66.6%), while isometamidium and diminazene had 33.3 and 0%, respectively, although curative efficacy of these drugs was similar (Joshi and Singh, 2000). Early treatment with diminazene aceturate in bovines showed encouraging results (Batra *et al*., 1994). Quinapyramine prosalt was found as an effective chemoprophylaxis for crossbred cow calves (Chaudhri *et al*., 1996) and buffalo calves (Singh and Choudhri, 2002). Folic acid and cyanobalamin can fasten the recovery in *T. evansi* infected animals (Sangwan *et al*., 1993). Clinical disease in buffaloes was successfully treated with quinapyramine prosalt, methyl sulphatechloride combination (Raina *et al*., 2000) but with two doses at 72 h interval were found more effective (Chand *et al*., 2008), subclinical cases also responded well to this therapy (Kumar *et al*., 2009b). Quinapyramine pro-salt was effective therapeutic and prophylactic agent in camels (Pathak *et al*., 1997), horse (De and Mukherjee, 2006), dogs (Chowdhury *et al*., 2006; Sharma and Juyal, 2007), goat (Rao and Hafeez, 1999), black bucks (Gupta *et al*., 2009) and jungle cat (Sahoo *et al*., 2009) naturally infected with *T. evansi*. Recently, isometamidium hydrochloride is used for treatment of *T. evansi* in India (Kumar *et al*., 2009a).

Alternative approaches in treatment: Currently, in vivo and in vitro trypanocidal activity of free and nanoencapsulated curcumin against Trypanosoma evansi is evaluated and found significant positive result to reduce parasitemia on adult male wistar rats (Gressler *et al*., 2015). Treatment with essential oil of achyrocline satureioides in rats infected with *Trypanosoma evansi* revealed that essential oil did not eliminate the parasites from the bloodstream, but it reduced the number of trypanosomes, mainly by its nanoencapsulated form so, association of this natural product with a trypanocidal drug may enhance its curative effect (Baldissera *et al*., 2014a). Study on pre-clinical mouse model indicated that Heat shock protein 90 (Hsp90) from protozoan parasites may as a potential drug target of trypanosomiasis (Rochani *et al*., 2014). Alchornedine a new guanidine alkaloid from the leaves of *Alchornea glandulosa* was found effective against trypanosomiasis (Barrosa *et al*., 2014). *In vitro* trypanocidal activity of macela (*Achyrocline satureioides*) extracts against *Trypanosoma evansi* was also evaluated (Baldissera *et al*., 2014b).

Prevention and control: Not a single promising experimental result to develop a vaccine could be obtained till date because of the capacity of parasite to modulate its own antigen termed as antigenic variation that means ability of parasite to regularly switch its surface coat glycoprotein. Another reason is that the parasite undermining the hosts capacity to mount an efficient immune response and to maintain its immunological memory termed as immunodeficiency to the host (Pays *et al*., 2004). Although a solitary report, wherein formalin inactivated *T. evansi* (2×10^6 count)

were administered in mice and found protective against homologous challenge (Tewari *et al*., 2009). Immunization with *T. evansi* recombinant beta-tubulin, induced some protection against *T. evansi*, *T. equiperdum* and *T. brucei* infection in mice (Li *et al*., 2007).

Control of *T. evansi* includes use of antitrypanosomal drugs for therapeutic and prophylaxis, vector control and use of trypanotolarent breed (applicable for cattle) (Tewari *et al*., 2012). Various chemical compounds may be used for treatment and some have prophylactic property (e.g. quinapyramine chloride). Control of vector also reduces the prevalence of trypanosomiasis. *Glossina* spp. can be easily controlled by using insecticide impregnated screens and insect sterilization techniques in livestock breeding areas (Rodtian *et al*., 2012). Control of tabanid flies is difficult because of its high mobility and prolificacy and the larval stages of fly are generally spread over a wide area (Foil and Hogsette, 1994). Although control of tabanid flies is found efficient by insecticide sprays in small closed deforest areas. Stomoxys is another fly responsible for transmission of trypanosomiasis. This fly develop within the livestock area or the farm and are closely related to the farming systems and it can be controlled by trap systems and insecticide sprays on animals (Foil *et al*., 1991; Leprince *et al*., 1991) or use of fly proof system with mosquito net.

CONCLUSION

There is limited information on the impact trypanosomiasis among livestock in endemic countries, particularly its impact on host population dynamics and demographics, the economic losses due to the disease and social impact on animal owners. Few studies have quantified the economic losses of disease by expenditure on diagnosis, treatment and replacement of lost animals for limited animal species and for limited locations. The financial losses due to surra can be avoided by adopting an effective control strategy which includes treatment of infected animals and control of vector flies. This brief review emphasizes the fact that this parasite has an unlimited geographical distribution and unlimited host range. Parasite have unlimited range of potential reservoirs and its unlimited, nonspecific and ubiquitous range of potential vectors. It may suggested that DNA material has made *T. evansi* a better parasite, as it is less specific in terms of vector by losing parasite dependency only on tsetse flies, which are restricted to a specific area in Africa and parasite geographical distribution. It is still unclear that *T. evansi* is considered to be nonpathogenic to cattle in Africa and Latin America, but as a major parasite in Asia. This may be due to the genetic make-up of the parasites or the cattle or both. Inspite of above these, there is an obvious lack of in-depth studies on several aspects of the infection. Despite technological advancements, a simple, inexpensive and reliable test for *T. evansi* detection is still under study. New technologies should be developed which can implement in field. Vaccine development has limited scope and overcome of drug-resistance, new formulations and effective therapy would need to be evolved on priority. Vector control and change in animal husbandry practices to minimize exposure to biting flies merits attention in an integrated strategy.

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