CHAPTER – 2

REVIEW OF LITERATURE
2. REVIEW OF LITERATURE

2.1. Trypanosoma evansi

2.1.1. Origin and History of Trypanosoma evansi

*Trypanosoma evansi* was discovered by Griffith Evans in 1880 and is the first pathogenic flagellar protozoan parasite identified in the blood of Indian equines and dromedaries (Hoare 1972). The disease was named as ‘surra’ by local Indians meaning emaciated (Al-Rawashdeh *et al*., 2000). Its principal host was camels and horses, which were originated from Africa and was mechanically transmitted by the bites of biting insects (Aradiab and Majid, 2006). *T. evansi* were thought to be derived from *T. brucei brucei* and were transmitted cyclically by tsetse flies but due to the loss of the maxicircles of kinetoplastic mitochondrial DNA were no longer able to undergo its cyclic transmission in *Glossina* (Lun and Desser, 1995; Lai *et al*., 2008). Further, little informations were available regarding the existence of strains of different pathogenicity of *T. evansi* (Queiroz *et al*., 2000). However, some strains are considered as highly pathogenic due to the result of the host vector factors related to susceptibility of host- species and insect densities (Hoare 1972). Moreover, surra or trypanosomosis is considered as the neglected disease due to healthy carriers and were easily found to be spreaded into Asia, France (Desquesnes *et al*., 2008), Latin America and more recently to Spain (Gutierrez *et al*., 2005). *T. evansi* infection has the potentiality to be considered as an emerging disease (Davison *et al*., 1999) in cattle with diverse pathogenicity among southeast Asian countries, from Myanmar to Indonesia (Kaewthamasorn and Wongsamee, 2006) including water buffaloes, horses, pigs, and causes nervous symptoms, loss of weight, fever and abortion (Lohr *et al*., 1986). Abortions related to *T. evansi* was observed in buffaloes (Lohr *et al*., 1986), cattle (Kashiwazaki *et al*., 1998), pigs (Arunasalam *et al*., 1995), ewes (Elhassan *et al*., 1995), mares (Silva *et al*., 1995a) and camels (Yagil 1982). In trypanosomosis, the exact mechanism responsible for the reproductive disturbances related to abortion were not fully understood but was attributed due to intrauterine infection (Losos and Ikede, 1972) or due to stress of infection (Lohr *et al*., 1986). Moreover, zoonotic potentiality of *T. evansi* was reported in humans in India (Joshi *et al*., 2005; Powar *et al*., 2006) due to significant contact between parasite and humans demonstrated by a serological survey.
2.1.2. Parasite Taxonomy and Morphology

*Trypanosoma evansi* is unicellular flagellar protozoa belonging to,

**Phylum:** Sarcomastigophora

**Order:** Kinetoplastidae

**Family:** Trypanosomatidae

**Genus:** Trypanosoma

**Sub-genus:** Trypanozoon (Salivarian group)

![Morphology of *T. evansi* (100 X) in cattle blood smear](image)

*Figure 1: Morphology of *T. evansi* (100 X) in cattle blood smear*

Further, the ‘trypanozoons’ includes the pathogenic species such as *T. evansi*, *T. brucei* (*T. brucei brucei* and *T. brucei rhodesiense*) and *T. equiperdum* (FAO 2000). *T. brucei brucei* causes the complex disease known as nagana in livestock involving various number of *Trypanosoma* species including *T. brucei brucei*, *T. vivax* and *T. congolense* which have a great significance on the breeding of cattle in Africa. Moreover, for this disease, wild animals often act as a reservoir. *T. brucei rhodesiense* and *T. brucei gambiense* causes Human African Trypanosomosis (HAT) or sleeping sickness exposing 60 million people in 36 sub-Saharan African countries and infecting 70,000 persons (Rodgers 2009). This
disease has proved to be fatal in the absence of proper treatment. *Trypanosoma equiperdum* causes disease called dourine and are transmitted sexually in equidae.

Surra means ‘rotten’ (Vittoz 1955) in Hindi and were mainly evolved in camels. Surra and *Trypanosoma evansi* were found under various names, especially parasite with more than 30 names (Stephen 1986). In Asia, surra is employed presently for *T.evansi* infection but were also known by various names such as ‘purana’ (chronic or old), ‘dubla’ (emaciated) (Luckins 1988), ‘tibersa’ (three- year disease) or ‘makhi ki bimari’ (Horse-fly disease) (Gill 1977). Even though, *T. evansi* is the name commonly used for the parasite, but in some areas, names such as *T. annamense* and *T. kirdanii* were also used (Hoare 1972).

*Trypanosoma evansi* is morphologically identical to the other member of the sub genus ‘Trypanozoon’ and is described as monomorphic in stained thin blood smear with the length of 15 to 34 µm leaf like slender and in some strain, it may be pleomorphic. It has been observed that *T. evansi* is smaller in size when compared to *Trypanosoma theileri* and is larger compared to *T. congoense*. In fresh blood smear, *T. evansi* consists of slender free flagellum for active movements comprising of one half of its length which is narrow and extended to posterior end (Queiroz et al., 2000) along with undulating membrane for trapping of light. The parasite shows active movement under microscope in fresh wet mount smear with limited displacement. Intermediate forms of parasite with almost terminal kinetoplast and shorter free flagellum are also observed and in some cases stumpy forms of parasite with an inconsistent features are also reported (Hoare 1972). The past and the present observations of *T. evansi* concluded that the shape and the size is directly related to the host immune response and the growing condition of the parasites than its genetic characteristics (Tejero et al., 2008).

2.1.3. The trypanosome flagellum

The trypanosomes are flagellated protozoa with single, large mitochondrion known as kinetoplast and are known for its tropical diseases. They consist of single long flagellum with multifunctional aspects. Kinetoplast consists of dense circular DNA which is visible under light microscope. Trypanosomes belong to kinetoplastida order and are divided into two families; trypanosomatidae (monoflagellated) and bononidae (biflagellated). Species belonging to Trypanosomatidae family are mainly parasites with large range of hosts such as mammals, reptiles, birds, fishes, frogs, snails, insects, and plants (Vickerman 1976). As many of the parasites were significant to human and animals, they were extensively
considered as the source of investigation. Human African Trypanosomes (HAT) found in central Africa such as *Trypanosoma brucei rhodesiense* and *T. b. gambiense* cause sleeping sickness. While, American trypanosomes such as, *Trypanosoma cruzi* causes Chagas disease.

2.1.3.1. Study of flagellum using trypanosomes as model

Trypanosomes are considered as the excellent model to study the biology of flagellum (Bastin *et al*., 1999a; Godsel and Engman, 1999; Kohl *et al*., 2003; McKean *et al*., 2003). Trypanosomes can be easily propagated in culture in the presence of flagella throughout its life cycle retaining the old flagellum and thus providing the opportunity to compare the new flagellum assembled in the cell with the matured flagellum (Bastin *et al*., 1999a). Moreover, the genome of trypanosomes were fully sequenced (El- Sayed *et al*., 2000; Hall *et al*., 2003) ([http://www.genedb.org/genedb/tryp/index.jsp](http://www.genedb.org/genedb/tryp/index.jsp)) and the absence of introns helps in easy gene identification and cloning along with the availability of potent and flexible tools for reverse genetics (Clayton 1999; Motyka and Englund, 2004).

2.1.3.2. Composition of flagellum of trypanosomes

Analysis of trypanosome flagellum by electron microscopy showed the presence of axoneme, the para flagellar rod (PFR), the flagellum attachment zone (FAZ), electron dense particles such as intra flagellar transport (IFT) particles, the flagellum membrane and the flagellum connector (FC) (Vickerman 1962; Angelopoulos 1970; Sherwin and Gull, 1989; Bastin *et al*., 2000; Moreira- Leite *et al*., 2001). The molecular composition of flagellum was obtained after screening of expression libraries or purification of proteins along with specific antibodies (Schlaeppi *et al*., 1989; Birkett *et al*., 1992). Further, genome sequence analysis using homology search tools of *T. brucei*, *T. cruzi* and *L. major* were useful for the identification of flagellar genes (Kohl *et al*., 2004).

2.1.3.3. Functions of flagellum

2.1.3.3.1. Cell motility

It was reported that flagellum plays a very important role in trypanosome mobility (Hill 2003). In all kinetoplastids, flagellum helps in swimming through generation of waves from the distal tip to the base (Walker 1961; Alexander and Burns, 1983). Further, the axoneme portion of flagellum including protein associated with the components of radial spoke (RSP3), central pair (PF 16, PF 20) or of dynein arms (light, intermediate or many
chains) were reported to play a very important role in trypanosome and its mobility (Kohl et al., 2001).

2.1.3.3.2. Sensitivity

It is very important to study the environment and differentiation programmes prior to the adhesion of flagellum to the host surface as very little factors were known to trigger differentiation processes. *In vitro*, it was observed that the addition of cis-aconitate or the temperature drop causes the transformation to the procyclic stage from the bloodstream (Czichos et al., 1986; Matthews et al., 1995), but the mechanism is still obscure (Saas et al., 2000). In different organisms, flagellum was known for their sensory functions (Pazour and Witman, 2003) as trypanosomes were motile due to the presence of flagellum.

2.1.3.3.3. Adhesion to the host surface and host parasite interaction

The parasites are attached to the host surfaces by flagellum (Bastin et al., 2000). The flagellum portion of the trypanosomes were used physically to anchor trypanosomes to the brush border of the epithelium of salivary gland and were reported to penetrate deeply to the host micro villi by expanding its membrane (Tetley and Vickerman, 1985). Even though, only one portion of flagellum is attached to the epithelial cells, the posterior end being still mobile helps in contributing sufficient accessibility of nutrients to the flagellar pocket; which is the main site for endocytosis (Tetley and Vickerman, 1985). Moreover, identification of optimal substrate was required for adhesion of flagellum prior to its attachment to the host tissue. Several surface proteins were involved in mediating and facilitating the adhesion. Sometimes, *in vitro* incubation with plastics also causes adhesion of parasites leading to differentiation of flagellum (Brooker 1970; Beattie and Gull, 1997). Several calcium-binding proteins present in the flagellum of trypanosomes (Ruben and Patton, 1987; Bastin et al., 1999b; Godsel and Engman, 1999; Ridgley et al., 2000) were well known for signaling and control of mobility. For the possible immunological intervention, the flagellar pocket regions were considered as the privileged site (Radwanska et al., 2000) and its associated proteins have been identified to contribute to trafficking and virulence (Field and Carrington, 2009). Moreover, it has been reported that paraflagellar rod protein are the critical organelle involved in mediating attachment to the vector cell surface (Gadelha et al., 2005) and binding of macro molecule with the host for eliciting host antibody response. Further, paraflagellar rod protein gene 1 (PFR 1) and paraflagellar rod protein gene 2 (PFR 2) have been cloned and expressed in prokaryotic
system (Maharana et al., 2011a; 2011b). Moreover, flagellar pocket antigens and paraflagellar rod protein has been explored (M kunza et al., 1995; Obishakin et al., 2014) in the development of vaccine and diagnostic test.

2.1.4. Geographical Distribution of Surra/ trypanosomosis

*T. evansi* is widely spread in all countries where camels are present including all African countries, Arabian Peninsula, Iran, Pakistan, Kazakhstan, Mongolia, India, China, Nepal, Bhutan, Russia, Laos, Vietnam, Myanmar, Thailand, Malaysia, Cambodia, Philippines and Indonesia (Luckins 1988; Reid 2002). The distribution of parasites is also suspected in western countries like Latin America, Argentina to Panama Spain, Central America to Mexico (Wells 1972; Gutierrez et al., 2006) and in Papua New Guinea (Reid et al., 1999). It was reported that camels introduced parasites into Australia, South-West Africa and Northern America and recently in the Canary Islands (Gutierrez et al., 1998; Gutierrez et al., 2006).

India

In India, surra was considered as very old disease, dating back to VIII centuries B.C (Hoare 1972). Surra is prevalent almost all the parts of the country, as the environment is suitable for the breeding of the fly vectors (Bhatia et al., 2006). Prevalence of trypanosomosis is reported to be more in camels due to irrigation facilities and the advent of Indira Gandhi canal in western Rajasthan (Pathak and Khanna, 1995). The incidence of trypanosomosis in bovine is directly proportional to the onset of monsoon to the post monsoon in Punjab (Soodan et al., 1995), West Bengal (Ray et al., 1992), Andhra Pradesh (Prasad et al., 1997), Chattisgarh (Agrawal et al., 2003), Bihar (Sinha et al., 2006) and Jammu (Raina et al., 2000). The factors like transport (Kalra et al., 1994), flooding, vaccination (Singla et al., 2010), intercurrent disease (Gupta et al., 2009) and malnutrition (Malik et al., 2000) often plays a very important role in the conversion of inapparent infection into clinical disease. Moreover, introduction of parasite into the new area is governed by high infection prevalence with mortality of 30% to 100% (Elamin et al., 1999).

2.1.5. Various host range of *T. evansi*

Amongst salivarian trypanosomes, *T. evansi* has the widest host range and is especially pathogenic in camelids and equids including large range of wild and domestic hosts.
worldwide. It was found that the large host range of \textit{T. evansi} was due to the loss of maxicircle kinetoplast DNA (Lun and Desser, 1995), but the same was not observed in \textit{T. equiperdum} (Brun et al., 1998) and thus had lesser host range compared to \textit{T. evansi}. The host-range of \textit{T. evansi} is highly variable from one geographical area to another.

2.1.5.1. Middle East and Africa countries

\textit{T. evansi} is the main parasite of camels (\textit{Camelus dromedaries}) which acts as the main host and reservoir including other cameldae, such as the Bractrian camel (\textit{Camelus bactrianus}). \textit{T. evansi} was also present in equidae, especially in horses (\textit{Equus caballus}), asses and donkeys (\textit{Equus asinus}) and mules with most often chronic infection.

In Africa, \textit{T. evansi} was found to be pathogenic to cattle (\textit{Bos taurus}) (Dia and Desquesnes, 2007), pigs (\textit{Sus scrofa}), goats (\textit{Capra hircus}) and domestic sheep (\textit{Ovis aries}) but were considered as non-pathogenic in the African buffalo (\textit{Syncerus caffer}) (Reduth et al., 1994). \textit{T. evansi} is occasionally observed in domestic cats (\textit{Felis domesticus}) (Tarello 2005) and dogs (\textit{Canis familiaris}) as they are infected from the infected animals by eating fresh raw meat.

2.1.5.2. Asian Countries

In Asia, trypanosomosis were mainly observed in water buffaloes and are considered as the major reservoir for \textit{T. evansi} infection. In Philippines, the disease was found not only in horses and buffaloes but also in pigs, goats and cattle (Dargantes et al., 2009). \textit{T. evansi} has been observed in sick elephants (Hin-On et al., 2004) in Thailand with the detection of few seropositive animals (Tuntasuvan and Luckins 1998). Various other animals like the sambar deer (\textit{Cervus unicolor}), antelope (\textit{Saiga tatarica}), Rusa deer (\textit{C. timorensis}) (Indrakamhang 1998), barking deer (\textit{Muntiacus muntjak}), hog dear (\textit{Axis porcinus}) (Tuntasuvan et al., 2000), Capreolus spp. (Stephen 1986), chital dear or spotted dear (\textit{Axis axis}) (Losos 1980), wild pigs, wild sheep (\textit{Ovis ammon}), rabbits, pikas (\textit{Ochotona pallasi}), tapirs (\textit{Tapirus indicus}) and rodents such as \textit{R. tanezumi}, \textit{Niviventer}, \textit{Rattus sp.}, \textit{Leopoldamys} and \textit{Bandicota sp.} (Jittapalapong et al., 2008; Milocco et al., 2012) and hamsters (\textit{Cricetus cricetus}) (Stephen 1986) was also observed with \textit{T. evansi} infection. In central Asia, hamsters and Pikas were spontaneously infected in enzootic areas (Hoare 1972). The ‘Black nap hare’ (\textit{Lepus nigricollis}) or the Indian hare, Mungos (\textit{Herpes tesjavanicus}), wolves, the orangutan (\textit{Pongo pygmaeus}), jackals (\textit{Canis aureus}), foxes (\textit{Vulpes sp.}), civet cats (\textit{Paradoxurus}), woodcats (\textit{Felis bengalensis javanensis}), hyenas
and badgers (*Helictis pierri* and *H. personatus*) can be infected with *T. evansi* naturally or experimentally including chicks in experimental conditions (Gill 1977). *T. evansi* was recently found in the Himalayan black bear (*Selenarctos hibetanus*) (Muhammad et al., 2007) and in Asian rhinoceros (*Dicerorhinus sumatrensis sumatrensis*) in Malaysia (Khan et al., 2004). Moreover, it was also observed that the experimental infection of *T. evansi* in chicks was identified long back (Gill 1977) while, in young pigeons were identified recently (Mandal 2008).

India has widest host range for *T. evansi* affecting mainly the members of *camelidae* and *equidae* and covers a wide variety of domestic and wild animals like camel, horses, cattle, buffaloes, donkey, mule, sheep, dogs, goat, pig, deer, foxes, and jackals (Pathak and Singh, 2005). Moreover, *T. evansi* infection has also been observed in Leopards (*Panthera pardus*), tigers (*Panthera tigris*) and jaguars (*Panthera onca*) (Bhaskararao et al., 1995; Sinha et al., 1971) including elephants (*Elephas maximus indicus*) (Stephen 1986). Punjab and Haryana showed the outbreaks of acute trypanosomosis in cattle and buffaloes (Batra et al., 1994; Jindal et al., 2005). Bihar showed high incidence of trypanosomosis in cattle (58.86%) compared to buffaloes (41.14%) (Sinha et al., 2006). Guntur district was reported with comparatively less *T. evansi* infection in cattle (1.42%) and buffaloes (2.71%) (Das et al., 1998) than in East Godavari (7.28%) districts of Andhra Pradesh (Bhaskara and Hafeez, 2005). The prevalence of disease in Karnataka was reported to be 39.78% in buffaloes, 42.12% in bovines and 2.15% in goats (Krishnappa et al., 2002). Mathura was reported with 100% morbidity and 66.6% mortality amongst equines (Kumar et al., 1994). Further, Jammu also reported the outbreaks in ponies (Raina et al., 2000). Udgir, Maharashtra also reported the incidence of the *T. evansi* infection in a Kathiawari mare (Bharkad et al., 2005). Eighteen districts of Rajasthan showed the outbreak of cameline trypanosomosis (Raisinghani and Lodha, 1989) and Western Rajasthan showed the prevalence of 31.66% positive for *T. evansi* antigen using double antibody sandwich ELISA and 7.5% by Giemsa stain smears/ wet blood test. Further, Bikaner district of Rajasthan showed 20.37% prevalence rate in camels (Singh et al., 1997). Dogs showed the prevalence of 4.68% in Ludhiana (Singh et al., 1993) and showed lesser incidence of trypanosomosis and around Kolkata (Chowdhury et al., 2005). Exotic breeds were also found to be more susceptible for acute fatal disease (Dakshinkar and Bhojne, 2001). Incidence of trypanosomosis has been reported from native dog breeds (Krishnamoorthy and Manohar, 2005). Cases of *T. evansi* infections were less in sheep (Rao et al., 1987) and goats (Rao and Hafeez, 1999; Jana and Jana, 2005) with unusual infections (Gill
Outbreaks of ‘Surra’ were reported among zoo animals like hyena in Delhi zoo and Chitals in Bhilai zoo (Arora 1994). Incidence of trypanosomosis at Ranthambore national park was reported in male tiger (Ramachandraiah et al., 1995) and in circus tigers in Andhra Pradesh (Rao et al., 1995). Further, outbreaks in circus tigress were also reported from Chittoor district of Andhra Pradesh (Devesana and Shobhamani, 2006) including jungal cat (Felis chaus) from Nagpur zoo (Dakshinkar et al., 2002) and mithun (Bos frontalis) from Assam (Rajkhowa et al., 2003). Feeding of infected carcass by wild carnivores and dogs were considered as the important mode of transmission for surra (Bhatia et al., 2006).

2.1.5.3. Australia and European countries

Trypanosoma evansi was introduced in the early XXth century in Australia and Canada by infected horses but was eradicated due to good control measures (Hoare 1972). A huge threat for T. evansi infection was discovered in Australia as the later can affect horses, cattle, camels and several wild animals including wild pigs, Rusa dear and wallabies (Macropus agilis and Thylogale brunii) (Reid et al., 2001). Moreover, Japanese vole (Microtus montebelli) has proved to be highly susceptible to T. evansi was introduced into the Canary Islands by camels from Maurutania or Mali (Gutierrez 1998; 2000; 2005) and thereafter was introduced into France and Spain (Desquesnes et al., 2008; 2009a).

2.1.5.4. Other parts of the world

T. evansi was found in various domestic and wild host species like cattle, buffaloes, horses, sheep and goats. Brazil and Guyana showed 73% to 83% of prevalence of T. evansi in horses (Desquesnes 2004; Herrera et al., 2004; Silva et al., 1995a), 40% in water buffaloes (Herrera et al., 2004; Davila et al., 2003) and cattle at 10% prevalence in Brazil. T. evansi was commonly found in dogs. Dogs are also infected by T. cruzi and Leishmania (Savani et al., 2005), causing death with cardiac sign and ocular hemorrhages (Desquesnes 2004). T. evansi are also seen in Peru in Guinea pigs (Cavia porcellus) where they are reared for meat purpose. Vampire bat (Desmodus rotundus) in the Latin America can act as host, vector and reservoir simultaneously (Hoare 1965). Moreover, Capybara (Hydrochoerus hydrochaeris), which is the biggest rodent in the world also acts as a main reservoir of T. evansi (Franke et al., 1994, Morales et al., 1976). In Brazil, susceptibility of T. evansi in Capybaras was shown (Herrera et al., 2004). Further, 25% to 70% of the animals showed antibody carriers in venezuela (Toro et al., 1980; Reveron et al., 1992).
Amongst camelids, *Lama pacos* and *Lama glama* has the susceptibility for *T. evansi* along with *Lama guanicoe* under experimental condition (Kinne *et al*., 2001). Prevalence of *T. evansi* infection in South American Coatis (*Nasua nasua*) was observed at 16% (103), including red howler monkey (*Alouatta seniculus* and *A. ursina*), wild dogs (*Canus azarae*), wild pigs (*Collared peccary, Tayassutajacu*), white tail deer (*Odocoileus virginianus chiriquensis*), Ocelots (*Leopardus pardalis*), white-lipped peccary (*Tayassupecari*) (Desquesnes 2004) and armadillos (*Oasypus sp.*) (Herrera *et al*., 2005). Bats eating fruits and arthropods such as *Platyrhinus sp.*, *Myotis sp.*, *Carollia sp.*, have been infected along with marsupials such as the omnivorous *Didelphis* sp., and *Monodelphis* sp. (Herrera *et al*., 2004).

*T. evansi* infection has also been found in Latin America in Chiroptera, Edentates, marsupials, primates, carnivores, lagomorphs, artiodactyls and perissodactyls and however their epidemiological studies and its importance has to be determined for each species while some may not be a choice for mechanical vectors, because of its low parasitaemia rates (Desquesnes 2004; Wells 1984), but still these animals may act as a source of infection for various carnivores animals. Almost all mammals were susceptible to *T. evansi* infection including some birds and in humans reported recently in India (Joshi *et al*., 2005) which may be due to contamination caused by the physical contact of wound with the blood of infected animals (Powar *et al*., 2006).

### 2.1.6. Clinical Symptoms/Signs

The classical clinical signs of the pathogenic effects of *T. evansi* includes anaemia due to erythrophagocytosis and haemolysis of red blood cells (Bhatia *et al*., 2006), intermittent fever, loss of condition, loss of weight and appetite, nervous symptoms, production losses, abortion, cachexia and even death with or without significant sign related to the host species (Gardiner and Mahmoud, 1990). The clinical symptoms observed in the affected animals varies from one host species to another and with variant intensity from inapparent to strong. Moreover, different geographical areas and epidemiological conditions add to the intensity of these signs. The typical clinical expression of surra is commonly observed in camelids and equines but however different clinical signs and pathogenic effect are also observed in different host species.
2.1.6.1. Cattle and Buffaloes

In Asian countries, surra is still considered as an important disease in cattle and buffaloes particularly in the Philippines, Indonesia, Thailand and Vietnam (Reid 2002). Animals infected with surra results in the loss of milk and meat production, weight loss, anemia, losses in draught ability and death in acute condition and in chronic conditions, lacrymation, anaemia, dullness, oedema and emaciation may occur (Muraleedharan and Srinivas, 1985; Rajguru et al., 2000). In India, trypanosomosis were observed as early as 1891, showing sometimes high mortality rates (> 90%) (Gill 1977), with lumbar paralysis in buffaloes (Kumar et al., 2009b). Punjab also showed the significant prevalence of chronic trypanosomosis in cross bred cattle in an organized dairy farm (Bharadwaj and Randhawa, 2010). High mortality rate due to surra was also recorded in Mauritius. Moreover, trypanosomosis in Indonesian heifers showed anemia, fever, reduction in body weight and abortion (Payne et al., 1993) while in cattle showed induced haematocrit drops, loss of weight, hyperthermia (Payne et al., 1993; Payne et al., 1991; Payne et al., 1992), nervous disorders (Tuntasuvan et al., 1997) and even death (Losos 1980). Further, Buffaloes in Thailand showed the signs such as stiffness, emaciation, fever, conjunctivitis, oedema (swelling of legs), anaemia, inappetence, diarrhea, dyspnea, abortion, recumbancy, nervous signs (Sudarto et al., 1990) and death (Indrakamhang 1998). Studies in Thailand showed 15-54% seropositives in cattle and buffaloes (Lang et al., 2001). Similarly, hundreds of outbreaks due to imports of buffaloes from Cambodia and Thailand have led to 10% mortality and 10-40% prevalence of T. evansi in buffaloes.

In Latin America and Africa, the T. evansi causing trypanosomosis has been considered as a mild asymptomatic or chronic disease in Bovine with sometimes showing difficulties to infect animals experimentally (Kageruka and Mortelmans, 1971). Moreover, Eventhough some clinical signs have been recorded in Venezuela, no economic impact has been demonstrated (Garcia et al., 2006). In Philippines, two main clinical signs has been observed in buffaloes: an acute disease causing death within hours or wasting sickness leading to recumbency and death (Reid 2002; Dargantes et al., 2009). Abortions of 47% have been recorded due to trypanosomosis. A very high mortality is also observed in the area newly infected with surra in beef cattle (Chobjit et al., 2006). Moreover, dairy cattle with surra showed symptoms such as fever, abortion and decreased milk yield and even death if untreated (Pholpark et al., 1999; Kashiwazaki et al., 1998). Transplacental transmissions of surra were found in cows (Kalra et al., 1994; Rajguru et al., 2000) and
buffaloes (Rao et al., 2001). Moreover, postmortem lesions includes splenomegaly, liquefaction of sub-epicardial fat, hepatomegaly, congestion of lungs, fluid in the pericardial, hemorrhages and plural cavities (Palanivel et al., 2008).

2.1.6.2. Camels and Horses

Surra in camels (Camelus dromedaria and C. bactrianus) showed both acute and chronic forms. Acute cases showed anaemia, high fever, emaciation, weakness and death. Chronic cases may last for 2-3 years (Tibersa) (Parsani et al., 2008) with symptoms like dullness, intermittent fever, loss of appetite, progressive weakness, oedema, loss of weight, petechial hemorrhages and anaemia. Young animals are more susceptible to disease although all age- groups may be infected. Nervous disorders, enlargement and suppuration of lymph glands are also observed. High neonatal mortality and abortion were observed in dromedary camels in Canary Islands (Gutierrez et al., 2005). The urine of affected camel exhibits a specific odour which is significant for the diagnosis of trypanosomosis (Stephen 1986).

Equines also shows a specific classical symptoms for trypanosomosis such as weakness, anaemia, fever, local or general cutaneous eruption, lethargy, severe weight loss, vulvar and vaginal mucosa, petechial hemorrhages, abortion, nervous signs, locomotive disorders, oedema mainly in reproductive organs, legs, lower abdomen and testicles (Stephen 1986). Chronic cases of trypanosomosis shows anaemia, weight loss and jaundice with dark yellow colored urine (Jani and Jani, 1993; Laha et al., 2004; Varshney and Gupta, 1996). Moreover, clinical cases of trypanosomosis in horses show endocrine dysfunctions (Varshney and Gupta, 1996). T. evansi in horses causes frequent relapses of infection as it showed regular changes of variable surface glycoprotein (VSG) present in both intra- and extra-vascular fluids (Sudarto et al., 1990). Further, persistant erection of the penis due to intravascular coagulation (Stephen 1986) along with non suppurative meningo encephalitis in white and gray matter of brain was observed in horses (Seiler et al., 1981). Transplacental transmission was also reported in donkey mare (Pathak and Kapoor, 1999).

2.1.6.3. Goats and Sheep

Goats showed less susceptibility (Jacquiet et al., 1993) for surra. In Philippines, symptoms like progressive emaciation, fluctuating fever, coughing, anemia, diarrhea, testicular enlargement were observed in most of species of goats (Dargantes et al., 2005) along with moderate (Ngeranwa et al., 1993) infections showing signs like fever, salivation,
lacrymation, nervous symptoms, loss of appetite, hypothermia, ocular lesions (Morales et al., 2006) and death (Youssif et al., 2008).

Trypanosomosis in sheep is generally mild or asymptomatic (Boehringer and Prosen, 1961). Clinical symptoms such as fever, anaemia, and loss of appetite are observed (Desquesnes 1997). Yankasa sheep with T. evansi Nigerian isolate showed symptoms like pale mucous membrane, fever, loss of appetite, apiphora, dullness, emaciation and rough haired coat (Audu et al., 1999).

2.1.6.4. Pigs

In pigs, symptoms such as anorexia, fever, emaciation and absorption were observed in Mayalasia (Arunasalam et al., 1995) and decreased fertility in Thailand (Songa et al., 1987). Moreover 85% of T. evansi prevalence was reported in the province of chachoengsao. Similarly, outbreaks in phitsanulok province reported symptoms such as anaemia, fever, nervous symptoms and urticarial plaques on udders or scrotum on ventral parts of the body around teats (Teeraprasert 1984) and finally, in the province of Nakhon-Pathom, clinical signs such as fever and abortion (Thepsumethanone et al., 1984) were observed in pigs.

2.1.6.5. Carnivores

Dogs are highly prone to T. evansi infection and sometimes causes death in acute cases (Gill 1977) and has showed clinical signs even after treatment (Singh et al., 1993) such as oedema of head, legs and abdominal larynx, fever, abdominal wall, weakness, anaemia, emaciation, myocarditis, paresis of the hind-quarters (Desquesnes 2004), sexual excitement and different ocular signs like lacrymation, corneal opacity, conjunctivitis, keratitis along with haemorrhagic signs such as deposition of fibrin in the anterior region of the eye, presence of parasites in the ocular aqueous fluid has been observed which may sometimes recede even after treatment (Savani et al., 2005; Silva et al., 1995b; Sonika et al., 2007). Dogs residing near to the slaughter houses or hunting dogs are more susceptible to T. evansi infection. Moreover, transmission of infection by Stomoxys (dog fly) in dogs was observed by coming in close contact with the infected animals. Seasonal conditions also play a vital role in the transmission of infections in dogs (Singh et al., 1993)

Cats have showed comparatively less susceptibility, however experimental infection have showed mild symptoms such as fever, hyporexia, apathy, vomiting (Da Silva et al., 2009), hyperproteinaemia, muscular pain, hypoalbuminaemia and hyperglobulinaemia (Da Silva
Various other carnivores such as tigers (Bhaskararao et al., 1995), ocelots (felis pardalis), leopards (Veer et al., 2002) and hyenas have also been found to be susceptible to *T. evansi* infection.

### 2.1.6.6. Wild Hosts

Trypanosomosis was reported in wild hosts such as capybaras, vampire bats and coatis. Experiment infection in coatis showed the existence of clinical signs such as myocarditis, anaemia and meningoencephalitis (Herrera et al., 2002). Existence of *T. evansi* infection was also reported in other wild hosts such as deer, wild pigs and rodents and was also considered to be healthy carriers. Experimental infections showed that number of other species are also susceptible and receptive to *T. evansi* infection such as Rusa dear, feral pigs, wallaby found in Papua New Guinea (PNG) and Australia (Reid et al., 1999) along with dusky pademelons (Thyl ogalebrunii) and Agile wallabies (Macropus agilis) with clinical signs such as weakness, anorexia, anaemia, ataxia, splenomegaly, ulcerative gastritis (Reid et al., 2001). Trypanosomosis observed in Himalayan charming bears in Pakistan, reported symptoms such as accelerated pulse, pyrexia, depression, tachypnea, ataxia and anaemic mucous membranes (Muhammad et al., 2007). Further, Malaysian Sumatran rhinoceroses (*Dicerorhinus sumatrensis sumatrensis*) exhibited signs like incoordination, depression, muscle tremor, anorexia, nasal haemorrhage, labored breathing, recumbency and finally death (Khan et al., 2004). *T. evansi* infection was also found in Kuwiat in dorcas gazelles (*Gazella dorcas saudiya*), sand gazelle (*gazella subgutturosa marica*) and in Arabian dorcas gazelles (*Gazella dorcas saudiya*) showing clinical symptoms such as paresis of hindquarters and immediate death (http://priory.com/vet/Trypanosoma gazelles. html).

### 2.1.6.7. Other Domesticated Species

Asian elephants in India and Myanmar (Burma) reported symptoms such as oedema of the face, neck, trunk, brisket, limbs and lower abdomen, fever, anorexia, anaemia, sluggish movement, dry and hard skin, sleepy moods, restlessness, ecchymoses, reluctance to work, conjunctiva, and even sudden death for trypanosomosis (Stephen 1986). Fatal and moderate cases of *T. evansi* infection have been reported in Thailand (Arjkumpa et al., 2012; Hin-On et al., 2004) especially in *Cervus porcinus* (hog dear) showing symptoms such as excitation, paresis, convulsion, lateral recumbency and high death rate (Onah et al., 1999; Tuntasuvan et al., 1998). In Mauritius, outbreak of *T. evansi* in cervus unicolor,
showed clinical symptoms such as rapid loss of condition, fever, anaemia, emaciation and death (Gill 1977). Mortality rate of 20% were found in deer farm in South China (Chen et al., 1983) and further in Malaysia (Perak), outbreak of *T. evansi* infection in Java deer (*cerveus timorensis*) reported clinical signs such as inappetence, anaemia, recumbency, lethal evolution and respiratory distress (Nurulaini et al., 2007).

2.1.7. Transmission

2.1.7.1. Mechanical transmission

*T. evansi* has a complex and multiple means of transmission such as via blood sucking insects, biting insects and vampire bats depending on the host and geographical area. It was reported that eventhough all blood sucking flies are capable of transmitting the disease, tse-tse flies were most common for transmission and were mainly seen in Africa (Womack 2001). Further, transmission may be vertical, horizontal, pre-oral and iatrogenic depending on location, season and host species and with various epidemiological characteristics.

Mechanical transmission by the bites of biting insects was considered as the most significant mode of transmission in livestock, camels and other large animals. The process of mechanical transmission is a non-specific process with the movement of flies after feeding on infected blood from the infected host to the non-specific host. A small amount of blood is retained in the mouthparts of the insect approximately 1-12nL in tabanid fly and 0.03 nL in stomoxys (Foil et al., 1987) and are inoculated into another host during the next attempt to bite (Foil et al., 1987; Desquesnes et al., 2009b; Krinsky 1976; Desquesnes et al., 2005). Further, it’s been reported that when parasitemia rises above $10^6$ trypanosomes/ mL, the chances of transmission are significant (Desquesnes et al., 2009b). Thus, tabanid and *Stomoxys* were responsible in camels exhibiting high parasitaemia ($>10^8$ *T. evansi*/ mL). Moreover, trypanosomes do not survive for longer period of time in the biting insects. Research has found that shorter time lapse of less than 30 minutes between two blood meals showed efficient transmission of infection (Sumba et al., 1998; Mihok et al., 1995). Further, *T. evansi* transmission were not only directly linked to the parasitaemia but were also related to the number of biting insects residing near to the host regions (Desquesnes et al., 2009b) including the morphology, size and its density. Thus, *stomoxys* with large number is equally efficient than tabanid with low number.
Immediate mechanical transmission affects a group of animals and the chances of infection for *T. evansi* are high in a given herd. Further, the transmission may be observed within the same species (camels) or within different species (camels and goats). Transmission is also prevalent between domestic and wild herbivores such as cattle, buffalo or horses when they gaze with capybaras or deer mainly seen in Brazil during extensive breeding conditions (Franke *et al*., 1994).

Eventhough, tabanids and *Stomoxys* were the main vectors for the mechanical transmission of *T. evansi*, however, Hippoboscids were also suspected mainly in horses and camels (*Hippobosca equina* and *H. camelina*) (Gill 1977). In the local conditions, various other insects such as ceratopogonidae, culicidae also plays an important role in transmission of *T. evansi*. Further, transmission in experimental condition was successful with *Anopheles fuliginosus, A. aegypti* and *A. argenteus* but its epidemiological importance were yet to be identified (Gill 1977). Moreover experimental transmission has been successfully identified in almost 29 *Tabanus sp.*, including *Tabanus ditaeniatus, T. rufiventris, T. rubidus, T. immanis, T. optatus, T. malayensis, T. partitus, T. ceylonicus, T. tenens* and *T. striatus* along with *Haematopota sp.*, *H. truncate, H. pungens, H. irrorata, H. cingulata, Chrysops flaviventris, C. dispar, C. fasciata* and *Lyperosia minuta* (Gill 1977). While in various *Stomoxys sp.*, such as *Stomoxys calcitrans, S. taeniatus, S. varipes, S. pallidus* and *Hippobosca squalid* (Sumba *et al*., 1998; Mihok *et al*., 1995), experimental transmission were demonstrated.

Further, the important role of reduviid bugs for mechanical transmission experimentally were also demonstrated (Manz 1985). As these bugs are not able to transmit infection quickly from one host to another, they are alternatively ingested by the host species and thus can transmit the parasite by pre-oral route of infection such as in sheep ingested with *T. melophagium* parasite after chewing the cyclical vector (*Melophagus ovinus*) (Hoare 1972). Transmission may also occur due to sucking flies via simple wound contamination, thus resulting in increased risk of mechanical transmission.

### 2.1.7.2. Alternate means of transmission

The disease can also be transmitted by iatrogenic transmission using non-sterile needles or other surgical instruments during mass treatment and vaccination campaign (Davila and Silva, 2000).
2.1.7.3. Vampire Bats: Biological vectors

Vampire bats are considered as a new biological system for the transmission of *T. evansi* in Latin America (Hoare 1972) and are infected by the oral route during leaking of blood from the infected prey such as cattle and horses and acts as a true host reservoir and permanent vectors by contaminating its host. Thus in the absence of main host such as horse, *T. evansi* can easily be sustained in a vampire colony making them suitable for the reservoir of parasites. *Desmodus rotundus*, a vampire bat can act as a reservoir, biological vectors and a host of parasite simultaneously.

2.1.8. Diagnosis

Eventhough clinical symptoms of trypanosomosis are significant, diagnosis of surra by various diagnostic techniques are required for its treatment, studies and for its control regimes. Different diagnostic methods were used for the detection of surra such as parasitological, molecular and serological tests.

1.1.1.1. Parasitological techniques (Identification of agents)

This is the direct classical parasitological methods for the detection of trypanosomosis such as examination of blood or lymph node samples by direct microscopy method. Traditional identification method by microscopy for *T. evansi* is difficult if the region also has the presence of other *Trypanozoon* spp., along with *T. evansi*. Moreover, as most of the infections are cryptic, thus are not detectable by direct microscopy method.

Direct microscopic method includes wet blood film, thin stained blood smear, thick stained blood smear. As these methods have low sensitivity ($10^5$ trypanosomes per milliliter of blood), they are usually not opted as the appropriate diagnostic method for the detection of surra. As *T. evansi* induces mild clinical or subclinical carrier state low parasitemia infections, in most of the host species, it becomes very difficult to confirm the parasites. Thus, in this situation, concentration methods are used for the diagnosis. Various concentration methods such as haematocrit centrifugation method (Woo 1969) or buffy coat technique (Murray et al., 1977) has showed increased sensitivity of the test with 100-200 trypanosomes per milliliter. Further, the sensitivity of the test can be increased by 10 folds by using buffy coat instead of whole blood. Thus concentration method can be considered as a low cost alternative method of direct microscopy. Mouse inoculation test has also proved to have high efficacy of 86.23% (Jain et al., 2000) with high sensitivity (20-50 trypanosomes per milliliter). This test is mainly used for cryptic trypanosomes and
is considered to be very effective parasitological test for the detection of scanty trypanosomes (Chaudhri et al., 1996; Singh et al., 2003).

2.1.8.2. Molecular methods

Specific DNA probes (Masiga and Gibson, 1990; Reid and Copeman, 2003) can be used for the detection of trypanosomes species. Further, species-specific polymerase chain reaction (PCR) has also been developed (Sengupta et al., 2010).

2.1.8.2.1. DNA probes

For the detection of trypanosomal DNA in the infected blood or tissues, a specific DNA probes were used. As this test requires further evaluation, it was not preferred for routine analysis (Basagoudanavar et al., 2001). Techniques based on polymerase chain reaction were preferred compared to DNA probes.

2.1.8.2.2. Polymerase chain reaction (PCR)

PCR uses number of species specific primers or specific for the subgenus Trypanozoon (Desquesnes et al., 2001). PCR method is based on DNA amplification process, and the sensitivity of PCR is directly proportional to the parasitaemia. Comparative analysis has showed that TBR primers are the most sensitive primers for the detection of *T. evansi* infection (Pruvot et al., 2010). PCR was found to be more sensitive in high susceptible host such as camels, horses, dogs, etc than in mild susceptible hosts such as cattle, buffalo, pig, etc In India, PCR was first used in camel blood samples for the detection of *T. evansi* infection (Basagoudanvar et al., 1998). PCR can detect as low as 0.15 trypanosomes/ ml and has the sensitivity of 0.015 pg/ ml (Sengupta et al., 2010). Thus, the application of PCR is more favored compared to parasitological methods for the identification of acute and/ or chronic stage of trypanosomosis in domestic and wild animals.

2.1.8.3. Serological tests

Various standard serological methods were used for the detection of trypanosomal antibody/ antigen such as enzyme linked immunosorbant assay (ELISA) (Davison et al., 1999; Franke et al., 1994; IAEA 1993; Reid and Copeman, 2002; Reid and Copeman, 2003), latex agglutination test (LAT) (Holland et al., 2002), Indirect immune fluorescent antibody test (IFAT), card agglutination test (CATT) (Baijana Songa and Hamers, 1988; Njiru et al., 2004; Reid and Copeman, 2003) and trypanalysis test. The usage of filter
paper blood spots in ELISA and serum instead of whole blood in CATT were the alternative options for the latter use (Holland et al., 2002). Moreover, ELISA and CATT were extensively evaluated in buffaloes in Vietnam and Indonesia (Davison et al., 1999; Holland et al., 2002; Verloo et al., 2000).

2.1.8.3.1. **Enzyme linked immunosorbant assay (ELISA)**

Serological tests such as ELISA are having more advantage for the detection of carrier status of animals as the animals once infected remains infected for life (Atarhouch et al., 2003) and are applicable for mass screening in the field for effective control of the disease with better production (Lejon et al., 2003). ELISA qualifies as a universal test for correctly identifying healthy animals as it is not stain specific (OIE 2012).

Different types of ELISA were mainly employed for the detection of antibody/antigen of *T. evansi* such as Indirect ELISA (I-ELISA) (Sengupta et al., 2014), competitive inhibition ELISA (CI-ELISA) (Shahardar et al., 2003; Ligi et al., 2016b) and double antibody sandwich ELISA (Ag-ELISA) (Jeyabal et al., 2003; Rayulu et al., 2007). I-ELISA and CI-ELISA were used for the detection of antibody of *T. evansi* while Ag-ELISA was used for the detection of antigens of *T. evansi*. CI-ELISA and Ag-ELISA uses monoclonal antibody (MAbs) produced against specific antigens of *T. evansi* and the research have proved the superiority of monoclonal antibody over polyclonal antibody (Dial et al., 1992). Different antigens of *T. evansi* such as whole cell lysate (WCL), native flagellar antigen (FLA) (Ligi et al., 2015, 2016a, 2016b), variable surface glycoprotein (VSG) (Sengupta et al., 2014), invariable surface glycoprotein (ISG) (Rudramurthy et al., 2015) were reported to have different efficacies. *T. evansi* whole cell lysate antigen based ELISA were used earlier for sero-diagnosis of surra (Luckins 1977) and showed diagnostic sensitivity for the detection of chronic *T. evansi* infection (Sudan et al., 2015). Further, monoclonal antibody based CI-ELISA has been accepted as a reliable and sensitive method for the detection of antibody in animals and humans (Knowles et al., 1991; Gurtler 1996). Moreover, earlier studies using native glycoprotein based ELISA has been reported for the detection of antibody in cattle from different parts of India (Kundu et al., 2013).

2.1.8.3.2. **Latex agglutination tests**

Latex agglutination test was used as the rapid test for the diagnosis of *T. evansi* antigens in different animals in field in Haryana (Rayulu et al., 2009).
2.1.8.3.3. Card agglutination tests (CATT)

Card agglutination test kit developed at the laboratory of serology, Institute of Tropical Medicine, Antwerp (Verloo et al., 2001) was used for the diagnosis of *T. evansi*. This test showed high sensitivity in horses and camels but showed comparatively less sensitivity in cattle (Desquesnes et al., 2011).

2.1.9. Treatment

For the treatment of trypanosomosis, various chemical compounds were used as drugs such as diminazene aceturate, isometamidium chloride, melarsomine hydrochloride etc. Among all the drugs, diminazeneaceturate is the widely used trypanocidal compound and the resistance against this drug was reported in many parts of world (Desquesnes 2004, Peregrine and Mamman, 1993). Diminazene aceturate is highly recommended drug for cattle, buffalo, goats and sheep with the dose rate of 7 mg kg$^{-1}$ intramuscularly, followed by melarsomine hydrochloride or isometamidium chloride with the dose rate of 0.5 mg kg$^{-1}$ (Desquesnes 2004). For camels, melarsomine dihydrochloride is the best drug with the dose rate of 0.25-0.5 mg kg$^{-1}$ body weight, but the same drug were not recommended in buffaloes with the dose of 0.75 mg kg$^{-1}$ body weight which may cause nervous symptoms (Desquesnes et al., 2013).

In India, drugs used are diminazene and quinapyramine for surra while cymelarsan and suramin are not commercialized. In naturally infected buffaloes, isometamedium (0.5 mg kg$^{-1}$ body weight) and quinapyramine (4.4 mg kg$^{-1}$ body weight) showed good therapeutic activity with quinapyramine proving better for prophylaxis than isometamedium. Similarly in clinically infected buffaloes, suramine and quinapyramine showed good prophylactive efficacy of 66.6% thendiminazene and isometamidium with 33.3% and 0% respectively, eventhough all these drugs showed similar curative efficacy (Joshi and Singh, 2000). Further, diminazene aceturate in bovines (Batra et al., 1994), quinapyramine prosalt in buffalo calves (Singh and Chaudhri, 2002) and crossbred cow calves (Chaudhri et al., 1996) has showed good effect. Moreover the combination of drugs such as methyl sulphate chloride and quinapyramine prosalt were successfully used for the clinical disease in buffaloes (Raina et al., 2000) along with subclinical cases (Kumar et al., 2009b). Quinapyramine prosalt were active and effective prophylactive and therapeutic agents in horse (De and Mukherjee, 2006), camels (Pathak et al., 1997), goat (Rao and Hafeez, 1999), dogs (Chowdhury et al., 2006), jungle cat (Sahoo et al., 2009) and black bucks.
(Gupta et al., 2009) with the natural infection. It was also found that two doses of methyl sulphate chloride and quinapyramine prosalt at 72hr interval were more effective instead of single dose (Chand et al., 2008). Moreover, in India, Cyanobalamin and folic acid showed faster recovery of T. evansi infected animals (Sangwan et al., 1993) along with isometamidium hydrochloride (Kumar et al., 2009a).

2.1.9.1. Alternative methods of treatment

Recently essential oil treatment of achyrocline satureioides in T. evansi infected rats showed reduced number of trypanosomes, eventhough it did not remove parasites completely from the bloodstream, the interaction of the trypanocidal drug along with this herbal product were reported to increase the curative efficiency (Baldissera et al., 2014a). Moreover, significant good result were observed in adult wistar male rats with reduced parasitaemia due to the effect of in vitro and in vivo trypanocidal activity of nonencapsulated and free curcumin against T. evansi. Further, studies on mouse model has revealed that heat shock protein 90 (Hsp 90) isolated from protozoan parasites, acts as an efficient drug target of surra (Rochani et al., 2014) along with new guanidine alkaloid such as, Alchornedine extracted from the leaves of Alchornea glandulosa has also proved to be more effective against surra (Barrosa et al., 2014). The effect of macela (Achyrocline satureioides) against T. evansi was also studied (Baldissera et al., 2014b).

2.1.9.2. Prevention and Control regime

It was reported that due to the ability of T. evansi to change its surface glycoprotein coat, termed as antigenic variation and further involved in host immunodeficiency, no vaccines were developed till date (Pays et al., 2004). Further, recombinant β-tubulin protein of T. evansi when immunized was found to induce some protection against T. evansi, T. brucei and T. equiperdum in mice (Li et al., 2007). T. evansi inactivated with formalin also helped in inducing protection against T. evansi (Tewari et al., 2009).

For T. evansi control regime, use of antitypanosomal drugs for its prophylaxis and therapeutic quality, trypanotolarent breed (mainly in cattle), and vector control were applicable (Tewari et al., 2012). Further, various chemical compounds having prophylactic property were also used for the treatment (eg. Quinapyramine chloride). Vector control also plays an important role in reducing the occurrence of trypanosomosis such as Glossina spp. which were found to be easily controlled by the usage of insect sterilization techniques and insecticide impregnated screens in the breeding areas of livestocks.
Further, the control of tabanid flies were comparatively difficult as its larvae were found to be distributed over a wide areas along with its high prolificacy and mobility (Foil and Hogsette, 1994). Insecticidal sprays were efficient to a large extend to control tabanid flies. Stomoxys flies, which are usually visible in the farm or within the livestock were controlled by insecticidal sprays, trap systems on animals or by the usage of fly proof mosquito net (Foil et al., 1991; Leprince et al., 1991).