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An Overview of the Various Methods for Diagnosis, Treatment, and Controlling of Trypanosomiasis in Domestic, Pet, and Wild Animals

Neeraj Kumar[1], M.K. Verma[2*], Javid Ur Rahman[3], Anand Kumar Singh[4], Sachin Patidar[5] and Jayshree Jakhar[6]

[1]Research Scholar, Department of Veterinary Pathology,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.
[2]Ph.D. Scholar, Department of Veterinary Pharmacology and Toxicology,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.
[3]Ph.D. Scholar, Department of Animal Genetics & Breeding,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.
[4]Ph.D. Scholar, Department of Veterinary Medicine,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.
[5]Research Scholar, Department of Veterinary Parasitology,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.
[6]Research Scholar, Department of Veterinary Pathology,
GB Pant University of Agriculture and Technology, Pantnagar, (Uttarakhand), India.

(Corresponding author: M.K. Verma)*
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ABSTRACT: Trypanosomiasis is an infectious haemoprotozoan disease that affects various domestic, pet, and wild animals with the most common clinical manifestations being intermittent fever, wasting, anaemia, oedema of dependent parts, nervous symptoms, and occasionally abortion. The disease is transmitted (biologically or mechanically) through the biting of flies. Microscopic examination, Micro-haematocrit centrifugation technique, animal inoculation, DNA detection by PCR, CFT, and ELISA are some of the diagnostic procedures available, ranging from traditional to molecular. In this review, we will cover the impact of trypanosomiasis on domestic, pet and wild animals, as well as transmission, pathogenesis, control, and prevention. In addition, the paper provides an update on the impact of trypanosomosis on erythrocyte homeostasis and infection-associated anemia.

Keywords: Trypanosomiasis, *T. evensi*, etiology, host range and susceptibility, prevalence, transmission, pathogenesis, clinical signs, diagnosis, treatment, and control.

INTRODUCTION

Trypanosomiasis is an infectious protozoan disease caused by several species of Trypanosoma. One of four major types of trypanosomes causes most pathogenic disease globally, including *T. evansi*, *T. vivax*, *T. brucei*, *T. equiperdum*, and *T. congolense* (Radwanska *et al*., 2008). Trypanosomiasis is endemic worldwide, and it has an adverse influence on livestock productivity due to high morbidity and mortality, low performance, reduce production, infertility, and high treatment costs. Trypanosomosis is of critical importance because of its destructive effects on animal health, which result in significant financial losses for the dairy industry due to loss of condition, milk yield reduction, and other factors. Total economic losses due to Trypanosomiasis recorded INR 9,872.33 per lactation in buffaloes (Singh *et al.*, 2014). Trypanosomosis directly affects the reproductivity of cattle by reducing birth rates, increasing mortality rate, and sometimes, a chronic case leads to abortion. *Trypanosoma evansi* has the most extensive geographical range of pathogenic trypanosome species, with foci of infection linked to flooded areas in numerous countries in South America, Africa, and Asia. It is transmitted mechanically by biting flies. Animal trypanosomiasis has particular problems in diagnosis due to non-specific clinical signs. Traditional diagnostic methods have low sensitivity, which hinders the diagnosis of trypanosomiasis in animals with low parasitemia. Many attempts have been made to develop an effective and safe vaccine against trypanosomasis but due to the surface antigen variants, no successful experimental results to develop a vaccine have been produced to date. As a result, trypanosomiasis is primarily prevented using prophylactic drugs, vector control, reducing the proximity of livestock to reservoir hosts, and development of trypanotolerant livestock.

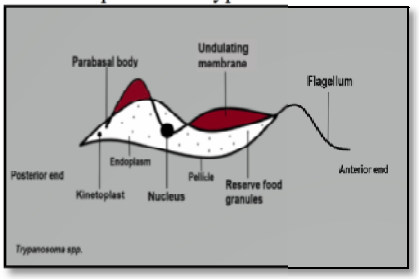

Fig. 1. Structure of Trypanosoma parasite.

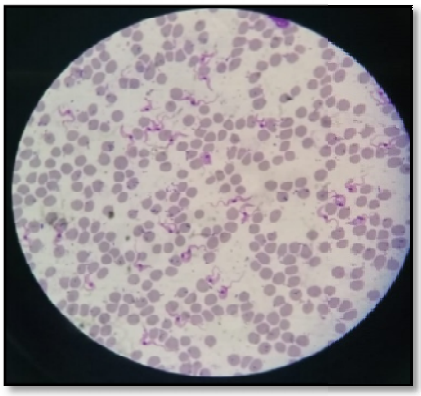

Fig. 2. *Trypanosoma* spp. in blood sample of buffalo.

History: In 1841, Gabriel Valentin saw flagellates in the blood of trout (fish) that today are included in Trypanosoma (Leadbeater & McCready, 2000). Trypanosoma was first discovered, described, and identified by the scientist Griffith Evans in 1880 when he was working in the Dera Islamic Khan District of Punjab (Pakistan). The species of Trypanosoma that was isolated by Evans is *T. evansi*. Camel was the first animal in which he found this protozoon, and the second animal was equine (Hoare, 1972). Thirty-three different names were credited to these trypanosomes, and out of these four names were attribute in 'Hindi' language, used by the livestock owners, according to clinical signs of disease, e.g., "Purana" means old, "Tibersa" means chronic infection which takes three years, "Dubla" means emaciated, "Makkhi ki bimri" (House fly disease) and "Surra" (In Hindi and Marathi word that means "rotten" and "heavy breathing from the nose," respectively). Some Gujrati names based on Gujarat state (India) have also been coined as "Dance makhi rog" means (Tabanus biting fly diseases) and "Chakri rog" means (circling movement due to acute Trypanosomiasis). The disease is additionally known by different nearby terms all through the world, including "Mard-el-debab"which means "sickness of the Gadflies" used by the Algerian peoples (North African). "Mal das cadeiras" (*T. equinum*) in Brazil; "Murrina" (*T. hippicum*) in Panama (Rodrigues *et al*., 2009); "Su-auru" (*T. ninaekohlyakimovi*) in Russia (Hoare, 1972) and "bayawak" in the northern Philippines and "yuleye" in Kenya (Dirie and Abdurahman, 2003).

Etiology. Trypanosomiasis is caused by the unicellular protozoa belonging to the genus Trypanosoma. There are several species comes under genus Trypanosoma but some important species which cause Trypanosomiasis in animals are *T. evensi*, *T. cruzi*, *T. vivex*, *T. brucei brucei*, *T. congolense*, *T. equiperdum*, and *T. equinum* (Desquesnes *et al*., 2013). Trypanosomes are found in extracellularly or outside the blood cells. The protozoa is always described as a monomorphic thin trypomastigote form ranging in size from 15-33 µm, with a long free flagellum and thin posterior extremity, as well as a short subterminal kinetoplast, in stained thin blood smears (Jaiswal *et al*., 2015). The size and shape of parasites vary from species to species, but some basic morphological features are common in all species, like a flagellum that arises from the parabasal body at the posterior end of the body and runs to the anterior end, where it is connected to the anterior pellicle along its length to form an undulating membrane (Heddergott *et al*., 2012). It may be continued beyond the anterior end of the body as a whip-like free flagellum. A key element of trypanosome cell biology and the life cycle is the flagellum, a vital and multifunctional organelle involved in cell propulsion, morphogenesis, and cytokinesis. The flagellar membrane is a specialised subdomain of the cell surface that houses several parasite virulence proteins that play a role in signaling and parasite-host interactions (Langousis and Hill, 2014). Another common feature is Kinetoplast. It is a distinct, well-defined body seen near the posterior end of the trypanosome, and it may differ in size and position according to the species.

Host range and susceptibility: Today, trypanosomiasis is considered chiefly veterinary significance disease, infecting a wide scope of financially significant warm blooded animals like ponies, dairy cattle, goats, buffaloes, canines, and camels. The parasite is also found in wild animals like antelope, wild boar, elephants, foxes, hare, monkeys, jackals, capybaras and tigers (Desquesnes *et al*., 2013). Trypansoma has also been recorded in mice, guinea pigs, deer, birds (Zídková *et al*., 2012) and fish (Pathak and Singh, 2005). In Africa, *T. evansi* is primarily a parasite of dromedaries (*Camelus dromedarius*). *T. evansi* is exceptionally pathogenic in the equidae family, particularly in ponies (*Equuscaballus*), asses (*Equus asinus*) and their crossbreeds (donkeys). In nature, diseases (Dorine) with *T. equiperdum* are only found in horses. The disease can also affect donkeys and mules without showing any symptoms (Buscher *et al*., 2019). Most of the cases of acute trypanosomiasis in cattle and buffaloes are chronic and asymptomatic (Bharadwaj and Randhawa, 2010). *T. evensi* infection was found to be more common in cattle, buffalo, and horses in northern India than in southern India, with surra infection being more common in horses than in other species (Lahaand Sasmal, 2009). Trypanosoma infection is more common in sheep than in goats. However, in sheep and goats, the disease is usually mild. Capybara (*Hydrochoerus hydrochaeris*), the biggest rodent in the world, is potentially a major reservoir of *T. evansi* infection in Brazil. The first case of *T. evansi* in a nestling pigeon has been reported in India, indicating that pigeons may be a natural host (Mandal *et al*., 2008). A pig usually shows mild signs of disease (Hamill *et al*., 2013). Trypanosomosis can infact either indigenous or cross breeds of dogs (Akpa *et al*., 2008). However, Exotic dogs are more prone to trypanosomiasis and usually experience acute fatal disease. However, some cases of trypanosomiasis have been recorded in native dog breeds (Krishnamoorthy and Manohar 2005). *T. evansi* has also been reported in domestic cats and llamas (Magri *et al*., 2021). *T. evensi* has also been observed in Asian elephants (*Elephas maximus*). Trypanosoma has also been reported in

African sharptooth catfish (*Clarias gariepinus*) several times (Osman *et al*., 2020). Many cases of Trypanosoma in fish have also been reported from India (Gupta *et al*., 2009). *T. evansi* has also been reported in leopards (*Panthera pardus*), jaguars (*Panthera onca*), and tigers (*Panthera tigris*) in India (Upadhye & Dhoot 2000). Incidence of surrain the chitals (*Axis axis*) and hyena were also reported in India (Arora, 1994). An outbreak in the Java deer was also reported from Malaysia (Nurulaini *et al*., 2007). In 2000, a deer and twelve tigers mortality were reported due to trypanosomiasis at Nandankanan zoological garden, Orissa (Khan*et al*., 2015).

Table 1: List of Trypanosoma species and their vector with mode of transmission.

Trypanosoma Spp.	Animals Effected	Vector Involve	Mode of Transmission
T. evensi (*salivarian section)*	cattle, buffalo, sheep, goat, horses, mule, donkey, camel, pig, dog, elephant, rabbit, guinea pig, deer, monkey, foxes, tiger, and jackals	Tabanid flies, *Stomoxy* spp, tsetse flies (Glossina), *Haematopota* spp., *Chrysops* spp. *Aedes*, Musca *crassirostris* and *Lyperosia* spp. and vampire bats	Mechanical or acyclic
T. brucei (*salivarian section*)	Cattle, sheep, goats, camels, wild animals, and rats (white albino rats), cause acute and severe disease in most severe in dogs, horses, cats.	tsetse flies (Glossina), Stomoxys, *Musca sorbens*, midgut, and proboscis both	Cyclical or biologically
T. congolense (*salivarian section*)	Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals	Stomoxys, tsetse flies (Glossina), midgut, and proboscis both	Cyclical or biologically
T.vivex (*salivarian section),*	Bovine (mainly), sheep, goats, camels, horses, various wild animals	Tabanus, tsetse flies (Glossina), and Stomoxys, only in proboscis	Mechanical and Cyclical or biologically
T. borreli and *Trypanosoma mukasai*	Fishes (common carp, *Carassius auratus*, goldfish, tench *Tincatinca*, *Cyprinus carpio and Clariasgarie pinus*).	leech (*Hirudinaria manillensis*)	Cyclical or biologically
T. theilerie (*Stercoraria section)*	Cattle, domestic buffalo	*Haematopota pluvialis*, Haernatopota *italica*, *Hybomitra micans and Tabanus bromius*, *Stomoxys*	Mechanical or cyclic
T.equiperdum (Salivarian section)	Horses, mules, donkeys	Venereal transmission by coitus	Mechanical and Venereal transmission (by coitus) or direct contact
T. simiae and *T. godfreyi* (*salivarian section)*	Domestic pigs and wild warthogs	*Glossina* spp. and Tabanus, stomoxys flies	Mechanical or cyclic
T. cruzi (Stercoraria section)	Dogs	Reduviid blood sucking bugs	Cyclical or biologically

Prevalence: Trypanosomiasis outbreaks are most common during the rainy and post-monsoon seasons, when the vector density is at its peak, although sporadic cases are recorded throughout the year (Sindato *et al*., 2007). Harish *et al*. (2006) found that cattle have a higher prevalence (8.93%) than buffaloes (4.18%). Muraleedharan (2015) reported that the prevalence of surra more occurs in cross-bred cattle than native cattle breeds. Gangwar, (2019), reported prevalence of surra is higher in female animals. The higher prevalence of surra in females may be attributed to stress, lactation, and pregnancy. Chowdhury *et al*. (2005) reported that surra affects both male and female dogs equally. Young dogs below two months are more susceptible to infection (Prasad *et al*., 2015). Acute infection is common in young dogs between 5-6 months of age (Eloy and Lucheis, 2009).

Epidemiology: It has been suggested that *T. evansi* and *T. equiperdum* had an ancestor and descended from human pathogenic *T. brucei* species in Africa (Lai *et al*., 2008). Previously they were known as *T. b. evansi. T. bruceia* and *T. b. equiperdum* had limited geographical distribution and cause slipping sickness in human beings in the most northern boundaries of Africa, which is transmitted by tsi tsi fly, but due to partial dyskinetoplastidy (Dk) or total akinetoplastidy (Ak) loss of maxicircles of mitochondrial or kinetoplastic (kDNA) evolution occurred and *T. evensi* adapted a non-cyclical mechanism of transmission involving loss of pleomorphism (Aregawi *et al*., 2019). This "evolution" is mainly related to the new modes of transmission of *T. evansi* (mechanical transmission) since it is no longer capable of infecting the invertebrate vector and forming subsequent life-stages (Carnes *et al*., 2015). Kinetoplastic mitochondrial DNA is required for oxidative phosphorylation, which is required for the tse tse's (Glossina) developmental processes in the midgut (Paris *et al*., 2011). *T. evansi* is not limited to tse tse fly transmission and has a wide range of vector specificity, including *Tabanus, Stomoxys, Haematopota, Chrysops, and Lyperosia.* This changed kDNA feature has been utilised to distinguish *T. evansi* from African *T. brucei* subspecies for a long time (Richardson *et al*., 2017). However, genetic analysis of both *T. evansi* and *T. brucei* has recently revealed that the issue is more complex and that many *T. evansi* are closely related to *T. brucei*, even closer than the relationship between *T. evansi* from different geographic locations (Carnes *et al*., 2015). *T. evansi* is thought to have spread from Africa

to Asia by diseased host animals, primarily dromedary camels, horses, and mules through (Aregawi, 2019) export and trade of these animals from Africa by caravans and wars (military campaigns) (Hoare, 1972).

Transmission: Surra can be transmitted in several ways in animals. It also includes a variety of complex transmission mechanisms that change in relative importance based on the geographical area and the hosts. The parasite is spread mechanically by biting insects and can be found in both blood stream and tissues. The most important vectors are belongs to the family Tabanidae (Tabanus, Haematopota, Chrysops, and Atylotus) and Muscidae (Stomoxys) (Baldacchino *et al*., 2014). Other flies including, *Hippoboscas* spp., and mosquitoes, which have been documented to transmit the disease locally (Banerjee *et al*., 2015). Tse tse flies (*Glossina* spp.) in Africa, may also serve as a mechanical vector for *T. evansi*. In India, the transmission of surra mainly occur by biting of tabanus files (Weber *et al*., 2019). South and Central America, have adopted vampire bats that serve as both vectors and reservoir hosts, are capable of transmitting *T. evensi* by wound contamination. In bats, trypanosomes duplicate primarily as blood forms and do not go through a cyclic development (Nwoha, 2013). Besides mechanical transmission, *T. evansi* was also documented directly transmitted through milk or during coitus in equine and sheep (Campigotto *et al*., 2015). Similarly, leeches can transmit trypanosomes, and their ability to transmit *T. evansi* should be investigated, particularly in Asia with the buffalo leech (*Hirudinaria manillensis*) (Hamilton *et al*., 2005). Tabanus is widely distributed during the monsoon season (Mulandane *et al*., 2020). Transmission by Tabanid flies is often accomplished through an interrupted feeding habit. Since the male tabanid's mouth portion is not compatible with sucking blood from the host, only female tabanids play a role in transmission. Mechanical transmission occurs when a biting fly begins a blood meal on an infected host, starts feeding on contaminated blood, is interrupted (because of the bite's pain, which causes the host to defend itself), flies away from the infected host, and land on another animal to continue its blood meal, allowing blood pathogens to be transferred from its mouth parts to a vulnerable species (Baldacchino *et al*., 2013). When an insect feeds on blood, some blood is left in its mouth parts due to capillary action, which is estimated to be 1–12 nano litre (0.001-0.0012 microliter) in tabanids and 0.03 nano litre (0.00003 microliter) in Stomoxys. When the insect inoculates a small portion of saliva with anticoagulant properties, before sucking the blood of the second host, and that some of the remaining blood may be partially inoculated into another animal during the early stages of its next attempt to bite (Desquesnes *et al.,* 2005). As a result, the transmission of *T. evansi* was not successful after a lengthy delay; however, transmission is only possible when the animals are in a group or herd. In the early stages of the blood feeding period, blood from the insect's gut or crop is regurgitated, providing another route for the transmission of trypanosomes to reach the host immediately. This will increase the likelihood of trypanosomes surviving inside the insects and allow for delayed transmission because the parasite can live for up to 5–7 hours in the insect's gut, which is particularly beneficial in *Trypanosoma vivax* in tabanids. Since there are no digestive secretions in the crop of *stomoxys* spp., Trypanosoma can live for a long time (Coronado *et al*., 2004). Cyclic transmission is another method of transmission in which development occurs inside the vector. Insects, particularly bugs, become infected with the trypomastigote stage after ingesting contaminated blood. Trypomastigotes develop into epimastigotes and subsequently metacyclic trypomastigotes in the midgut after a week. The trypomastigotes stage develops in the posterior gut, and the bug discharges faecal material on or around the body of the host during or immediately after feeding. After penetrating or contaminating the wound, the organism may reach into the blood. The high prevalence of trypanosoma infection in dogs and wild carnivores is thought to be due to the feeding of infected carcasses (Bhatia *et al*., 2006). Surra transmission has also been confirmed through the placenta to the foetus of a camel during an acute stage of infection. Iatrogenic transmission may also be another path of transmission. *T. equiperdum* is found in tissue that is passed from one horse to another during sexual intercourse. The detection of *T. equiperdum* infection in foals may also indicate that the parasite can be transmitted directly to foals through udder lesions or milk (Ahmed *et al*., 2018).

Pathogenisity: Trypanosoma pathogenicity is overly complex and depends on age, breed, species, immunity, health status, nutritional status, vector density, host preference, and the environment (Vanden & Delespaux, 2011). Infection begins when a Trypanosome-infected fly feeds on the susceptible host and injects infective metacyclic trypanosomes into the skin, where they differentiate into blood stream forms and spread to the vascular system. The draining lymph node is the primary route of dissemination, and trypanosomes containing metacyclic antigens are found in the afferent lymph, but not in the efferent lymph, where only blood stream forms are found. The earliest clinical sign of infection with *T. evansi* in the mammalian hostsis the development of chancre: a local skin reaction (Nwoha *et al*., 2013). The number of metacyclic parasite types inoculated into the skin can be linked to the chancre's onset, scale, and length. CD4+ T lymphocytes are essential in the onset of inflammation and the development of a chancre (Mwangi *et al*., 1990). Tsetse flies inject a greater number of metacyclics of *Trypanosoma brucei* strains than *Trypanosoma congolense* and *Trypanosoma vivax* strains, which is consistent with previous findings. *T. brucei* infections are associated with the most severe chancres (Naessens *et al*., 2003). The severity of chancre is determined by the amount of metacyclic trypanosomes that trypanosomes inject into the skin. Trypanosomes, particularly *T. evansi* and *T. brucei brucei*, are distributed throughout the body via the blood stream, whereas *T. congolense*, remain within the blood vessels (Abubakar *et al*., 2005). Pathogenesis begins when

metacyclic trypanosomes enter the blood stream and cause pyrexia. The temperature is normally fluctuating. Trypanosomes contain many genes that code for antigenically distinct glycosylphosphatidylinositol (GPI)-anchored variable surface glycoprotein (VSGs), which cause the host to produce specific antibodies against these proteins (Matthews, 2005). After a few days, these antibodies kill almost all the trypanosomes in the blood, and the body temperature drops. However, a few parasites survive because they can express a particular variant of the surface glycoprotein gene, which antibodies cannot recognise. These surviving trypanosomes multiply again, causing the temperature to rise again. Trypanosomes that express these distinct VSGs will multiply indefinitely before the host immune system develops specific antibodies against them again. The infection becomes chronic because of the parasite's constant change of surface glycoproteins against which the host has yet to develop specific antibodies. Anaemia is a major feature of the pathology of surra. Horses and dogs have more severe anaemia than the other animals (Jaimes-Dueñez *et al*., 2017). Three phases of anaemia have been reported to occur in surra including, phase I (acute crises phage), phase II (chronic phage) and phase III (recovery phage). Acute crises phage or phage 1 begins with the appearance of trypanosomes in the blood circulation (Abenga and Anosa, 2006). During this phase, anaemia is classified as macrocytic and normochromic type (Stijlemans *et al*., 2018). At this stage, death commonly outcome due to severe pancytopenia. Some sub-acute cases have been produced experimentally in rats infected with *T. brucei* and *T. congolense* (Mbaya *et al*., 2011). Surviving animals enter phase 2, which lasts several months and is characterised by low phage parasitaemia that is normally undetectable. Owing to the chronic existence of this phage, death is uncommon, and anaemia is usually normochromic and normocytic (Stijlemans *et al*., 2010). Parasitaemia is mild to non-existent in phase 3. Blood and tissues are free of parasites. During this phase, there is a low level of parasitemia and total erythrocyte values begin to return normal, and other pathological changes undergo resolution leading to self-recovery as commonly encountered in trypanotolerant wildlife (Mbaya *et al*., 2009). The loss of a significant number of red blood cells leads to anemia. Animal become dull, anorexic and loss of body condition. In the later stages, anaemia continues to play a prominent role, with possibly other causes like tissue anoxia, which results in a fall in tissue pH and vascular damage (Connor, 1994). The central nervous system is susceptible to anoxia with concomitant development of cerebral anoxia leads to death (FAO, 2000). Erythrophagocytosis is thought to be caused by several reasons, including Immunological mechanisms, macrophage hyperactivation, erythrocyte injuries via haemolysins, proteases (Umar *et al*., 2007), phospholipases and neuraminidases (Guegan *et al*., 2013). The hypothesis that trypanosomalsialidase (SA)-mediated desialylation of erythrocytes leads to their phagocytosis has recently been validated (Coustou *et al*., 2012).

Clinical signs and symptoms: The typical clinical sign and symptoms of surra are usually observed in equines and camels, but the pathogenic effects of surra are observed in a variety of domestic and wild hosts, depending on the species. Species specific clinical findings are provided below.

Cattle and buffaloes: Surra in cattle and buffaloes is normally asymptomatic, and they are thought to be carrying latent subclinical infections that can last for months. The acute type of disease is characterised by high fever, increased respiration and pulse rate, anaemia, weight loss, output losses, fatigue, and the appearance of the animal is dull that dashes against the wall and dies within 2-3 hours (Gangwar *et al*., 2019). The animal strikes its head against a fixed target and moves in a circle. With recumbency, certain animals exhibit nervous signs. The chronic course of the disease is characterised by intermittent fever, anaemia, lachrymation, dullness with recumbency or stumbling gait, oedema in lower body parts, laboured breathing, loss of body condition, and production loss (Rajguru *et al*., 2000). Young calves also show high intermittent fever (105°F), and nervous signs and death within 24–48 hours. Abortion in cattle and buffalo was detected in several cases (Kumar *et al*., 2012).

Goat and sheep: Goats and sheep are usually low susceptible to surra. Parasitemia in goats is normally mild but persistent, making them an effective reservoir for most trypanosomes found in other species. In an experiment, inoculated goats showed a subclinical course (fever and arthritis) of the disease, with just a few outbreaks (Gutierrez *et al*., 2004). In artificially contaminated goats, other studies display retinochoroiditis and unilateral superficial corneal ulceration, but no apparent loss of vision (Morales *et al*., 2006).

Equines: Clinical signs of surra in equines vary with severity of the disease, variable intra- and inter-species susceptibility of the host and infecting strain (Buscher *et al*., 2019). Typical clinical signs include high fever, weakness, anaemia, lethargy, weight loss, grinding of teeth, abdominal pain, lacrimation, hyperasthesia, local or general cutaneous eruption, petechial hemorrhages on (nictitating membrane, vulvar and vaginal mucosa, anterior chamber of the eye) and generalised oedema of dependent parts, especially the lower part of hind legs, throat, abdomen and testicle and sheath or udder. Abortion is occurs occasionally. In the neurological phase paralysis of the hind quarter and lips usually leads to death which is probably called ("*Mal de Caderas*", Murrina) (Rodrigues *et al*., 2015).

Camel: Growing camels are highly susceptible to infection shortly after weaning. The acute form of the disease can last up to 3 months and is characterised by intermittent fever, anorexia with the hump disappearing as the disease progresses, urticarial plaques on the flank and neck, recurrent keratoconjunctivitis, oedema under the belly, dullness and depression (Eyob and Matios, 2013) as well as severe anemia, and distinct odour of urine. The most prevalent type of case is chronic in nature, with symptoms including, repeated fever episodes, oedema in lower regions of the body, urticaria

plaques, and occasionally petechial haemorrhages in the serous membranes. If left untreated, death is finally ensured; nevertheless, some individuals may harbour trypanosomes for up to 2 years (Parsani *et al*., 2008).

Dogs: *T. evansi* is highly contagious in dogs, and infection is typically fatal. In acute infections, which can be symptomatic or asymptomatic, death can occur within a week and most commonly within a month (Baby *et al*., 2000). The acute form characterised by intermittent fever, anorexia, anaemia, weakness, emaciation, generalized lymphadenopathy, oedema of the head, abdomen and legs, nervous signs include paralysis of the hindquarters, difficulty in swallowing, hoarse voice, and staggering gait leads to death (Antoine-Moussiaux, *et al*., 2008). Occular signs are mainly associated with conjunctivitis, keratitis, corneal opacity, and unilateral anterior uveitis. Haemorrhages in the eyes can often be seen (Defontis, 2012)

Cat: Clinical symptoms in cats are similar to dogs, with the exception that edoema of head and corneal opacity was associated with photophobia and bilateral epiphora. Pyrexia, convulsions, and hind limb paralysis are all possible symptoms in cats (Kirchhoff, 2011).

Pigs: Surra symptoms in pigs are are usually mild, include mild fever, anemia, anorexia, fatigue, inappetence, dehydration, reduced fertility, enlargement of lymph nodes, petechial haemorrhages leading to plaques or lesions, urticaria, and abortion. Sometimes rapid emaciation, frequent coughing, and diarrhea are seen in piglets (Premaalatha *et al*., 2014).

Elephants: Elephants are affected the same as a camel. Affected elephants show pyrexia, anaemia, loss of apetite, oedema of the face, neck, brisket, trunk, limbs and lower abdomen, dry and hard skin, restlessness, reluctance to work, dullness, listness and sleepy appearance. Urine is scanty and progressively becomes viscid and sometimes marked greenish in colour. Swelling between the jaws and the lower part of the abdomen is often seen (Desquesnes *et al*., 2013).

Monkeys: *T. brucie* infection in monkeys resulted in either persistent parasitaemia or a discontinuous course characterised by relapse lasting 2 to 5 days. Infected monkeys developed acute symptoms including fever, dullness, anemia, enlarged superficial lymph nodes, stiffness of joints, and peri-orbital oedema (Waema *et al*., 2014).

Deer: Symptoms reported from Indian Sambar deer (*Cervus unicolor*) outbreaks in Mauritius, including high fever, anaemia, rapid loss of condition, emaciation, and death (Nurulaini *et al*., 2007).

Bears: *T. evensi* was reported natural infections in four Himalayan charming bears in Pakistan; the animals exhibited high fever, anaemia, high pulse and respiration rate, tachypnea, ataxia and depression (Muhammad *et al*., 2007).

Tigers: Clinical signs in affected tiger are intermittent temperature (102-105°F), anorexia, lacrimation and pale mucous membrane, enlargement of prescapular lymph node, nervous signs like seizures, uncontrolled movements in the body and sometimes a transitory change in consciousness (Khan *et al*., 2015).

Diagnosis: Diagnosis of trypanosomiasis might be difficult due to no precise clinical and pathological signs. Thus laboratory diagnoses are necessary to facilitate a fast, accurate diagnosis. The diagnosis of trypanosomosis is based on various parasitological techniques, such as direct microscopy, concentration techniques (micro-haematocrit centrifugation technique), animal inoculation, and indirect methods (serological and biochemical) (Calistri *et al*., 2013). The most used approach for diagnosing trypanosomiasis is blood examination by light microscopy (Sivajothi *et al*., 2018). It is, however, most reliable in cases of acute or early infection. In chronic phage infection, however, diagnosis by direct microscopy may be difficult because trypanosomes can not appear until later stages (González *et al*., 2006). Direct blood examination has been shown to be 48.3, 45.4, and 53.8 % sensitive in naturally infected buffalo, camels, and horses, respectively, which is similar to the sensitivity of examining Giemsa-stained smears (45.6%) in natural infection (Singh *et al*., 2004). A wet film can be used to detect motile trypanosomes, and for determination of parasite morphology. Stained thick and thin smears can be used. The detection limit for trypanosomes in the blood by direct microscopy is normally about 10^4 trypanosomes per millilitre of blood. This method, on the other hand, is ideal for screening large numbers of animals (François *et al*., 2005). Instead of blood, and lymph aspirate, cerebrospinal fluid, milk, and vaginal or preputial discharge may be used (Suganuma *et al*., 2016). Since only a small amount of blood is used in the thin smear, the sensitivity of this procedure is extremely poor. The sensitivity of this technique can be enhanced significantly by lysis of the erythrocytes (Hagos *et al*., 2010). The micro-haematocrit centrifugation technique (MHCT) is another method based on the principle that centrifugation of anti-coagulated blood separates trypanosomes from blood cells (Holland *et al*. 2001). Another method is the animal inoculation test. Mice are usually used because they are small, cheap, easily handable and readily available. After an inoculation, blood is drawn from the tail tip and examined under a microscope for the presence of trypanosomes for a period of 14 to 60 days (Njiru *et al*., 2005). This test is regarded as the gold standard for detecting *T. evansi* when the buffy coat is used in place of whole blood (Singh *et al*., 2003). The indirect approach, which includes biochemical and serological tests, is the second choice. These tests are focused on the detection of immunoglobulins or antibodies in the serum of infected animals as a result of trypanosome infection. Different biochemical tests have been developed namely, stilbamidine test, formol get test, mercuric chloride precipitation test, flocculation test and thymol turbidity test. The precipitation of immunoglobulins by mercuric or formalin chloride is the basis for the reactions observed when these chemicals are applied to serum. These tests are used to screen a large number of animals in a herd. However, the results of these biochemical tests can produce false negatives or ambiguous results, which can be misleading and have a weak association

with patent parasitaemia. Indirect immunofluorescent antibody test (IFAT), complement fixation test, enzyme associated immunosorbent assay (ELISA), capillary agglutination test (CAT), double immunodiffusion (DID), counter immunoelectrophoresis (CIEP), and card agglutination test are some of the basic serodiagnostic studies. These tests are focused on the identification of circulatory antibodies in the serum produced by the host in response to infection. Since antibodies will survive for weeks, even months, depending on the immune state of the host, and all trypanosomes have vanished from the host either by drug therapy or self-cure, a positive result is not evidence of active infection. The only OIE-recommended assays for *T. equiperdum* infection are CFT and IFAT (Jacobson, 2004). For *T. evansi*, OIE recommends ELISA, IFAT, CATT and immune trypanolysis (IT) (Ngaira *et al*., 2005). For *T. vivax, T. brucei*, and *T. congolense* infection, OIE recommends using ELISA and IFAT (Büscher *et al*., 2019). IFAT and ELISA have been shown to have greater sensitivity and diagnostic value. In experimentally infected dogs, IFAT shows 100 % sensitivity (Aquino *et al*., 2002). In horses and camels, the CAT is extremely sensitive, but in cattle, it is less sensitive (Desquesnes *et al*., 2011). The most used molecular method of diagnosis is polymerase chain reaction (PCR). Quantitative PCR (qPCR) with sequencing is useful for the detection of low parasitaemia and for species determination (Suganuma *et al*., 2016).

Control: All the major trypanosomes evade host immune defences by switching the dense coat of variant surface glycoprotein (VSG) on the cell surface on a regular basis, which is a major immune-evading strategies that effectively precludes the production of traditional vaccines (Hutchinson *et al*., 2016). As a result, animal trypanosomiasis is generally controlled using traps or insecticides to eliminate the vector and trypanocides drugs to reduce the parasite (Holmes, 2013). In most poor rural endemic areas, trypanocidal medications are the primary intervention option because vector control is expensive when implemented on a large scale and is not always sustainable or effective. The use of chemotherapeutic and chemoprophylactic medicines to control parasites has the dual impact of minimising infection-related losses while also eliminating the transmissible trypanosome reservoir (Welburn *et al*., 2015). Trypanocidal drugs are mostly used for treatment and less for prevention due to the emergence of drug resistance and the high costs involved in dealing with large herds of animals (Grace *et al*., 2009). Diminazene aceturate is a commonly used trypanocidal drug due to its potency against both *T. vivax* and *T. congolense* and its comparatively minimal toxic side effects. Diminazene is now the most widely used trypanocidal drug in ruminants. Diminazene aceturate was found to be effective in treating trypanosomiasis in dogs when given at doses of 3.5 mg/kg for *T. congolense* infection and 7 mg/kg for *T. evansi* and *T. b. brucei* (Ogbu, 2017). This compound, however, causes toxicity in camels, so it is not recommended for camels (Maina *et al*., 2003). It is primarily used as a curative at lower dose rates (0.25–0.5 mg/kg b.wt) and as a prophylactic at higher dose rates (1.0 to 2.0 mg/kg b.wt) in sheep, buffalo, and camels. Melarsomine dihydrochloride is the best trypanocidal for camel, with a dosage rate of 0.25-0.5 mg/kg body weight intramuscularly (Desquesnes *et al.*, 2013). However, it is not recommended for buffalo because it can trigger nervous signs (Desquesnes *et al.*, 2011). Quinapyramine has been used in Asia and Africa for decades and is effective against *T. evansi*, *T. vivax*, *T. brucei* and *T. congolense* (Giordani *et al*., 2016). In India, quinapyramine chloride is the medicine of choice for surra (Macaraeg *et al*., 2013). It is commonly marketed as quinapyramine chloride alone, which is primarily therapeutic or curative, or in combination with quinapyramine sulphate, which has preventative effects. Quinapyramine pro-salt was also found to be an effective curative and prophylactic agent against *T. evansi* infection in camels, goats, horses, dogs and other wild animals (black bucks, and jungle cat) (Sahoo *et al*., 2009).

Prevention: There are currently no vaccine available to prevent infection with trypanosomes. Reduced exposure of vectors to sensitive livestock is the goal of preventive strategies. The use of fly-proof pens, fly repellents, fogging, and fly traps, as well as limiting animal movement from endemic to non-endemic areas, are all examples of these strategies. Before the start of the rainy season, a chemoprophylactic drugs should be injected into susceptible animals in the endemic region, and the drug should be repeated as required during the rainy season. Controlling mechanical vectors in the field, especially tabanus, is difficult due to their high mobility and diversity in each area, and larval stages are typically dispersed over a large area (Lehane *et al*., 2016). To minimise the fly population, fly repellents, selective and separate grazing, confinement, use of baits and traps appear to be viable options. The Nzi and Vavoua traps are the most effective traps for mechanical vectors (tabanus, stomoxys) (Mihok, 2002). Insecticide sprays have been shown to be effective in controlling tabanus in small closed deforested areas (Bouyer *et al*., 2013). In surra-endemic locations, separating infected animals from vulnerable animals by more than 50 metres may also be beneficial in limiting *T. evansi* transmission (Barros and Foil, 2007). The use of cow's dry dung cake or hay to produced smoke by a slow fire is one of the traditional methods for controlling biting flies and other insects in Asia (Ntonifor *et al*., 2006). However, since it only covers a small area, and smoke can also affect the health of animals. Mosquito nets are commonly used for horses in India. Carnivores may be contaminated by consuming infected carcass blood, bone, or meat, so infected dead animals should be disposed of carefully. Some indigenous African cattle breeds (N'Dama, Muturu, and Dahomey) are more resistant to trypanosomiasis than imported breeds. This phenomenon is known as 'trypano tolerance,' which is defined as the ability to survive and work after being infected with trypanosomes. Continued study into host-parasite interactions is also needed in the absence of a

vaccination method to limit the spread of these illnesses.

CONCLUSION

Surra is present all over the world. Due to the suitable environment, it enhances mobility and prolificacy of vector population. It is still unclear how *T. evansi* expressed a surprising and spectacular ability to develop in an extensive range of hosts leading to a no less spectacular, potentially unlimited, geographical distribution. Pathogenicity of trypanosome is still unclear what is the exact mechanism of anaemia due to trypanosomiasis, its need further research. In India, surra is a major disease of camel, buffalo, horse and dogs. On cattle population, more attention is necessary to surra as it may cause severe economic loss. The incidence of surra in cattle and buffaloes in India has been underestimated because the infection is usually subclinical in them usually and buffaloes may act as reservoirs. Vector control is also a challenging task in India. Vector files can be controlled by an effective and economic insecticide. This field also requires some research to find an alternate way to control vectors as well as more research is also necessary to find an alternate treatment for this disease.

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