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Distribution, Clinico-Pathologic Perspectives and Zoonotic Potentials of *Trypanosoma evansi*

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Abstract

Trypanosoma evansi is a protozoan parasite causing "Surra", a disease of livestock and wildlife across Africa, Asia, and Latin America. Transmitted by biting flies, it infects diverse animal species and causes substantial economic losses. Although human infections are rare, emerging cases highlight their zoonotic potential, particularly in genetically susceptible or immunocompromised individuals. Limited awareness and non-specific symptoms may lead to underdiagnosis, emphasizing the need for further research on its public health impact. Indiscriminate use of trypanocides, interference with vaccination programs, and preponderance of hematophagous arthropods (vectors) also exacerbate the onset of disease. Detection of trypanosomes in circulation utilizing conventional parasitological techniques is inadequate, and greater than 70 percent of infections are cryptic and undetectable by direct microscopy. The other most common tests employed are Enzyme Linked Immunosorbent Assay (ELISA), Card Agglutination Test for *T. evansi* (CATT/*T. evansi*) and Deoxyribonucleic Acid (DNA)-based techniques. Furthermore, evidence from studies has demonstrated that the need to adopt effective control measures requires an integrated approach through proper management, effective use of chemotherapeutics, chemoprophylaxis and vector control. This review was conducted through a comprehensive survey of the available literature on *Trypanosoma evansi* and Non-Tsetse Transmitted Animal Trypanosomosis (NTTAT). Relevant publications were identified using electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar. This review provides an overview of the economic importance, epidemiology, geographical distribution,

pathogenesis, clinical manifestation, emerging zoonosis, pathology, diagnosis and control of *Trypanosoma evansi*, while also emphasizing the existing challenges and outlining future research directions.

Keywords

Trypanosoma evansi, Surra, Zoonosis, Diagnosis

1. Introduction

Livestock production remains a cornerstone of Africa's agricultural economy, contributing significantly to income generation, employment, and household food security [1]. Beyond its nutritional value, livestock provides draught power, manure, hides, and serves as a primary source of livelihood for rural populations [2]. However, the sector faces persistent productivity challenges arising from parasitic and infectious diseases, inadequate veterinary infrastructure, and climatic stressors [3]-[5]. Among these, trypanosomiasis stands out as one of the most important constraints to sustainable livestock production, responsible for severe economic losses through reduced fertility, lowered milk and meat yields, abortion, mortality, and high treatment costs [6] [7].

African Animal Trypanosomiasis (AAT), transmitted mainly by tsetse and other biting flies, remains endemic across vast regions of Sub-Saharan Africa, where over 50 million cattle and 60 million people are at risk [8] [9]. The disease contributes to food insecurity and poverty by reducing draft power and limiting mixed farming systems, particularly among resource-poor rural communities [10]. In addition, *Trypanosoma evansi*, a non-tsetse-transmitted species, has extended this burden beyond the tsetse belt through mechanical transmission by Tabanus and Stomoxys flies, causing "Surra" in multiple domestic and wild hosts [11]. Its widespread occurrence, ability to cross species barriers, and occasional zoonotic spill-over highlight its global and One Health significance [12] [13].

This review therefore synthesizes current knowledge on the epidemiology, pathogenesis, clinical manifestations, pathology, and zoonotic potential of *T. evansi*, emphasizing diagnostic challenges, control measures, and emerging research gaps relevant to endemic and at-risk regions.

2. History and Geographical Distribution of *Trypanosoma evansi*

In 1880, Griffith Evans described for the first time pathogenic organisms in the blood of Equidae and Camelidae in India, associated with severe anemia, fever, lethargy and stertorous breathing sounds (Surra) and called these parasitic organisms Hematozoon(s) [14]. In 1885, Steel renamed the organism *Spirochaete evansi*. Many other researchers renamed this organism over the years but in 1896, the organism was properly classified as a parasite and named *Trypanosoma evansi*

by a French Veterinarian, Chauvrat [15].

Surra has been reported all over the regions of Africa, Asia (Eastern & South-eastern) and the Middle East. Fewer reports confirm the presence of the disease in non-endemic Europe and South America [16] [17]. Although a wide range of wild and domestic animals are affected by *T. evansi* [18], prevalence of the disease among domestic animals was higher in camels and cattle in Africa [19]-[21]; Cattle and Dogs in Asia [18] [22]. While in non-endemic Europe and South America, incidence of *T. evansi* in horses was ascribed to the importation of horses and camels from endemic zones [23]. The wildlife hosts (Water Buffaloes, Lions, Giraffe, Antelopes, Hippopotamus, Warthogs, Feral pigs, Leopards, Hyenas, Impala and Deers) of trypanosomes like *T. evansi* are considered reservoirs as the presence of these hemoparasites in their circulatory system produces no clinical disease. However, they are important in the transmission of disease, especially to humans and domestic animals [13].

Human-related pursuits for biodiversity (Wildlife parks), poverty alleviation (through increased farming), increasing human population, and climatic factors have led to the continuous encroachment into the wildlife habitat [13] [24]. Thereby, predisposing humans [12] [25] as well as domestic animals to the infestation/infection by *Trypanosoma evansi* and other species of trypanosomes.

A good understanding of the epidemiology of the disease is essential for the design and implementation of effective control strategies to curb the incidence and spread of this disease.

3. Economic Impact of *Trypanosoma evansi*

The growing human population, rising incomes, and accelerating urbanization have been proposed as drivers for an increased demand for livestock products [26] [27]. However, the livestock sector encounters multiple challenges that impede its ability to meet rising demand, thereby constraining economic growth within the industry [28].

African Animal Trypanosomosis (AAT) is a parasitic disease that causes serious economic losses in livestock due to anemia, loss of condition and emaciation [29] while many untreated cases are fatal [30]. There is limited information on the impact of Surra among livestock in endemic countries, particularly its impact on host population dynamics and demographics, the economic losses due to the disease, and finally, the social impact on animal owners. Surra is widely recognized as an economically significant disease, responsible for high mortality, reduced milk and meat yields, poor carcass quality, diminished reproductive performance, decreased draught power and manure output, as well as immunosuppression in livestock [31]. Few studies have quantified economic losses, including costs for diagnosis, treatment, and animal replacement. Little information is available on the financial benefits of treating/controlling Surra in infected animal populations. Globally, infections caused by *T. evansi* and other pathogenic trypanosome species collectively result in annual economic losses estimated at US$4 - 4.5 billion [28]. The scale of

this impact is evident in regional assessments: in Somaliland, camel herds lose an estimated US$404,630 annually from reduced milk production and sales, projecting up to US$223 million if extended nationwide [32] [33]. In the Brazilian Pantanal, a major outbreak caused losses exceeding US$160 million [34], while similar economic strain has been documented in Ethiopia due to cattle mortality, reduced traction power, and increased treatment expenditure [34]. The low calving performance among buffaloes in Mindanao (in Philippines) is intricately linked to abortion and infertility [35]. In Thailand, abortions and reproductive failure due to Surra have also been reported in buffaloes [36], cattle [37], camels [38], and horses [39]. The death of buffalo cows during their most productive phase reduces their life expectancy (by almost half) and has a major impact on farmers. Females at this age are highly valued for draught power and as breeding animals for replacement or sale (to provide additional income) or home consumption. Surra has been proven to cause mortality in buffalo after experimental [40]. Whilst mortalities in draught buffalo caused by Surra could be partly associated with stress due to overwork, other factors such as malnutrition, concurrent infections, and adverse climatic conditions may contribute to the animals' reduced resistance and higher susceptibility to the disease [41]. Surra is also lethal in other livestock species, such as horses [42], camels [38], guanaco [43], cattle [44], goats [35] and even pigs [31] [41].

T. evansi infection imposes a significant economic burden by reducing livestock productivity and hindering trade. Implementing control strategies, including routine treatment and enhanced management practices, is critical to minimizing these losses and supporting the livelihoods of communities reliant on livestock.

4. Constraints to Livestock Production in Africa

Livestock production in Africa is constrained by multiple, interrelated factors. These include the high prevalence of diseases, particularly parasitic and infectious infections [45] [46], poor management practices [46] [47], limited availability and adoption of improved forage resources [48] [49], and inefficiencies in marketing systems [46] [50]. Seasonal feed shortages [51] and the growing impacts of climate change on disease dynamics and resource availability further exacerbate these challenges [52]. Parasitic diseases remain a major constraint to livestock production in Africa, significantly limiting productivity and profitability [46] [53]-[57]. Vector-borne diseases, including trypanosomiasis, piroplasmosis, and East Coast Fever, restrict the use of extensive regions in Africa for livestock rearing by adversely affecting animal health and overall productivity [58]. Numerous infectious diseases severely impact the animal industry, causing losses of hundreds of millions of dollars annually in developing countries [59]. Environmental consequences further complicate the challenges faced by programs aiming to eradicate vectors such as tsetse flies [60]. Amongst the parasitic diseases, *Trypanosoma evansi* (Surra) is recognized by the World Organization for Animal Health (WOAH) as a multiple-species disease included in the list of WOAH-notifiable diseases [61]. This

multi-host of the disease's characteristic is attributed to the fact that the mechanical vectors, such as tabanids, do not have strict host preference [62]. In the non-tsetse belt of Africa, Surra is principally a disease of camels and horses but cattle and goats are also highly susceptible [63]. The infection with the pathogen leads to clinical signs that include pyrexia, progressive anaemia, loss of condition and lassitude. Episodes of fever and parasitaemia occur during the course of the disease. Subcutaneous oedema occurs, especially in the lower parts of the body such as abdomen, ventral neck, chin and genitalia. Urticarial plaques and petechial haemorrhages of the serous membranes are commonly seen. Abortion has been noted in buffaloes in Asia. It is possible that immunodeficiencies also result from infection with *T. evansi* [64].

5. Trypanosomosis in Africa

African Animal Trypanosomosis (AAT) continues to be recognized as one of the most significant cattle diseases affecting sustainable livestock production and mixed farming systems in Africa. The resurgence of the disease, driven by persistent challenges in vector control and the limited effectiveness of chemotherapy and chemoprophylaxis, remains a major obstacle to agricultural development across the continent [65] [66]. Tsetse flies remain widely distributed across Sub-Saharan Africa, covering an estimated 9 - 10 million square kilometers. Within these regions, more than 50 million cattle and approximately 60 million people are considered at risk of African trypanosomosis, highlighting its continued importance as both a veterinary and public health concern [8] [9].

Trypanosomosis due to *T. evansi* (Surra) is an important disease in camelids; horses are also highly susceptible to this infection. Infected camels and equines may die within three months [31]. Also, immunosuppression or vaccination failure predisposes cattle, buffalo, pigs, goats and sheep to Surra [63] [67] [68]. Beyond Africa, Surra has also been reported in Asia, Latin America, and sporadic cases due to animal importation in Europe [23]-[31].

Tsetse-transmitted African trypanosomosis is responsible for 55,000 human and 3 million livestock deaths annually [69] [70] and hinders mixed farming through reduced work efficiency of draft animals. The decrease in national and international funding for research and surveillance of trypanosomiasis has resulted in insufficient information on the current status of the disease [71].

African animal trypanosomosis remains a persistent barrier to livestock production and rural livelihoods. Continued investment in research, development of effective control strategies, and robust surveillance measures is critical to reducing the economic and public health impact.

6. Aetiology and Structural Biology

Trypanosoma evansi is a salivary flagellated protozoan belonging to the genus Trypanosoma (Order-Kinetoplastida: Family-Trypanosomatidae) [43]. It possesses a single, elongated nucleus and a large, disc-shaped mitochondrion containing a

kinetoplast, which is a dense network of mitochondrial DNA. However, in Glossina, this mitochondrial DNA is incomplete or absent, preventing *T. evansi* from completing its developmental cycle in the tsetse fly vector [31]. A prominent undulating membrane runs along the length of the body, supported by a single flagellum emerging from the basal body at the posterior end. This membrane and flagellum confer motility, facilitating movement through host blood and lymphatic systems [31] [72]. *Trypanosoma evansi* ranges between 14 - 33 μm long and 1.5 - 4 μm in width [73]. The morphological characteristics of these parasites are the same as those of other members of the subgenus Trypanozoon, and therefore classified as monomorphic, though certain strains may be pleomorphic [74]. Under phase-contrast microscopy of fresh blood, Trypanosoma exhibits a prominent undulating membrane, which produces characteristic light pockets during movement [75]. On Giemsa-stained thin blood smears, *Trypanosoma evansi* appears as a monomorphic, slender trypomastigote with an elongated body and a centrally located nucleus [31]. *Trypanosoma evansi* is also enveloped in Variant Surface Glycoproteins (VSGs), which are encoded by a large gene repertoire, each defining a distinct Variable Antigen Type [76]. Among these VSGs, RoTat 1.2 has become a widely used diagnostic target because it is present in nearly all Type A isolates of *T. evansi*. However, certain non-RoTat 1.2 Type A strains, as well as all Type B strains, lack the RoTat 1.2 gene and its associated VSG, highlighting the molecular variability within the species [77].

The principal pathogenic African trypanosomes are classified into three subgenera within the Salivaria section: Nannomonas (*T. congolense*), Duttonella (*T. vivax*), and Trypanozoon (*T. brucei* complex, including *T. evansi* and *T. equiperdum*). Tsetse-transmitted trypanosomosis ("nagana") caused by *T. congolense*, *T. vivax*, and *T. brucei* affects livestock across vast areas of Sub-Saharan Africa [31]. Non-tsetse-transmitted trypanosomoses, caused by *T. evansi*, *T. equiperdum*, and *T. vivax* can be transmitted either mechanically by biting flies or through sexual contact in horses (equines), leading to animal morbidity and economic losses [31] [77].

African trypanosomes, whether transmitted by tsetse flies or other vectors, continue to pose significant challenges to livestock health and productivity across affected regions, emphasizing the need for integrated control strategies and ongoing surveillance to reduce their economic and veterinary impact.

7. Epidemiology of *Trypanosoma evansi*

7.1. Species

Trypanosoma evansi can infect domestic and wild animals [16]. Horses, camels, buffaloes, cattle, goats, pigs, and dogs are among the susceptible domestic animals; however, the clinical severity varies [78]. In horses, camels, and dogs, the disease is often rapidly fatal, though it may also be fatal in water buffalo, cattle, goat, sheep, pigs, and llamas [43]. Research has also shown that common rodents are a significant reservoir host for *T. evansi* [79] and experimentally susceptible [80]. Various

wildlife, including wallabies [81], pigeons [82], mongoose, monkey, and bandicoot [83], have been experimentally infected. Few cases of human infection have also been reported [84].

7.2. Age

Trypanosoma evansi infects animals across all age groups. However, studies in camels report a higher prevalence of infection in adults (>4 years old), which may be attributed to heavy stress associated with their use for various purposes and poor management practices [85] [86].

7.3. Sex

Several epidemiological studies have reported variations in prevalence between sexes. Higher infection rates have been reported in males [19] and linked to the fact that bulls are typically subjected to strenuous activities, such as those used as drought animals in agriculture, transportation of goods, which could have contributed to stressors that could affect their immunity, and predispose them to infection [87]. Conversely, females may also exhibit more vulnerability to trypanosomes [88] during pregnancy, parturition, lactation, and malnutrition [87].

7.4. Management Practice and Seasonal Variation

There are little to no documented reports of *T. evansi* management practices solely. However, in pastoral and nomadic systems, where herds are moved in search of pasture and water, they are easily exposed to trypanosome vectors. In endemic areas, higher prevalence of trypanosomes is generally associated with rainy or wet seasons, largely due to increased abundance of *Tabanus* spp. and *Stomoxys* spp. [89]. In Ethiopia, camel herders consistently reported Surra outbreaks in all seasons, with the highest prevalence during the rainy season and the least in the dry season [90] [91]. Importantly, a study also found no significant seasonal differences in Trypansoma prevalence, suggesting that the disease may persist year-round [92].

7.5. Immune Status

An animal's vulnerability to *Trypanosoma evansi* infection is often determined by its immunological condition. The prevalence of *T. evansi* has been found to be considerably higher in camels older than four, pregnant, and in poor physical condition [85] [93].

8. *Trypanosoma evansi* as an Emerging Zoonotic Potential

Although human infections with *Trypanosoma evansi* are exceptionally uncommon, a few have been documented in Asia and Africa. While the cases from Africa remain unconfirmed suspected cases, those reported cases from Asia have primarily been linked to accidental iatrogenic inoculation or contamination of skin breaches with infected meat or blood [84] [94] [95]. Nonetheless, the possibility

of alternative transmission routes has been suggested, including peroral exposure [95] and mechanical transmission by hematophagous arthropods such as Tabanids and *Stomoxys* spp. [31]. In spite of the fact that the epidemiological significance of these pathways in humans remains insufficiently elucidated, it has been suggested that populations at increased risk of susceptibility include livestock farmers and veterinarians, owing to their frequent occupational exposure to infected animals [25] [31]. Moreover, individuals with underlying genetic deficiencies, such as mutations in Apolipoprotein L1, as well as those with liver dysfunction, exhibit heightened vulnerability to infection [94] [96]. Climate change is another increasingly recognized factor influencing the epidemiology of vector-borne diseases, including Surra. Rising temperatures, shifting rainfall patterns, and extreme weather events create ecological conditions that favor the survival and spread of the vectors (*Tabanus* spp., *Stomoxys* spp.) responsible for the mechanical transmission of *T. evansi* [52] [58]. Furthermore, climate-induced stress weakens the immunity of animals and enhances spill-over risk to humans [52] [58].

These factors underscore the need for further investigation to clarify potential risk factors and to better assess the zoonotic threat posed by *T. evansi* as the epidemiological relevance of these pathways in humans remains insufficiently elucidated. Also, the general lack of awareness of the zoonotic ability of *T. evansi* may lead to underdiagnosis of infections in humans. Bridging this diagnostic and awareness gap requires focused human surveillance. Although confirmed cases remain rare, evidence suggests that the public-health burden of *T. evansi* is underestimated [12]. Targeted seroprevalence studies among high-risk occupational groups could reveal silent infections and guide early detection within existing One Health frameworks.

9. Transmission

Trypanosoma evansi can be transmitted through several routes, with mechanical transmission by biting insects being the primary mode in camels and other large livestock [11]. Although *Trypanosoma evansi* does not undergo a full developmental cycle within its vector, it is effectively spread through mechanical transmission by blood-feeding (hematophagous) flies, for example, Tabanus, Chrysops, Haematopota, Hippobosca, Haematobia, and Stomoxys [97]. Other insects such as mosquitoes (Aedes, Anopheles), biting midges, lice (*Haematopinus tuberculatus*), and reduviid bugs have been shown experimentally to transmit *T. evansi*, but their epidemiological impact appears restricted [31]. In addition to vector transmission, iatrogenic spread of *T. evansi* can occur through contaminated instruments; this has been demonstrated in camels, where high infection rates and reproductive losses were associated with repeated reproductive procedures using non-sterile needles [78]. Other routes include vertical transmission across the placenta and exposure of oral or mucosal wounds to infected blood and tissues in carnivores and scavengers [31]. In South and Central America, the vampire bat (*Desmodus*

rotundus) serves as a host, reservoir, and biological vector, transmitting it to livestock through blood feeding [61].

10. Incubation Period

The incubation period of *Trypanosoma evansi* varies with host species, age, and immune status. In experimental conditions, it ranges from 2 - 3 days in rats, 5 - 10 days in mice, 5 - 7 days in hamsters and rabbits, 3 - 5 days in guinea pigs, and up to 4 months in sheep and goats, whereas chickens remain resistant even at high infective doses [98]. However, the WOAH Terrestrial Code standardizes the incubation period at 90 days for all susceptible species [9].

11. Pathogenesis

T. evansi infection is introduced into the host through contaminated mouthparts of hematophagous flies, vampire bat bites, or other routes of exposure to infected blood. The parasite gains direct entry into the host's bloodstream [99]. Once established, the parasite uses antigenic diversity to evade immune clearance. Its surface is covered by Variant Surface Glycoproteins (VSGs), which are periodically switched to generate new antigenic types [76]. This mechanism leads to waves of parasitemia, causing hemolysis and anemia [100]. It has also been hypothesized that erythrocytes may acquire trypanosomal antigen, resulting in immunological reactions and complement-mediated destruction of [31] [41] [75] [101]. Chronic infection with *T. evansi* leads to sustained antigenic stimulation, which progressively exhausts lymphocytes and induces apoptosis, thereby weakening adaptive immunity and predisposing the host to secondary infections [102]. Persistent immune activation in chronic infections also promotes the formation of circulating immune complexes that deposit in the kidneys, liver, and vascular endothelium [103]. These complexes trigger complement activation and inflammatory injury, contributing to glomerulonephritis, hepatic lesions, and vasculitis [104]. Prolonged hypergammaglobulinemia and polyclonal B-cell activation further exacerbate immune dysregulation, resulting in autoantibody production and tissue damage [102]. Consequently, chronic trypanosomiasis manifests as immune-mediated anemia, edema, and organ dysfunction driven by sustained inflammation and immune complex deposition [104].

Pathogenicity and Virulence

There are two genetically distinct types of *T. evansi*: Type A and Type B [105] [106]. *T. evansi* type A, which is the most abundant and found in Africa, Asia, and Latin America, is characterised by the presence of the gene for the Variant Surface Glycoprotein (VSG) RoTat 1.2 [106]. In contrast, *T. evansi* type B is rare and has only been reported in Africa: Kenya, Chad, Sudan, and Ethiopia [107]. Type A isolates are frequently associated with acute or subacute infections, while Type B strains are not identifiable by standard serological testing because they lack the RoTat 1.2 gene [108].

12. Clinical Signs of *Trypanosoma evansi*

The clinical signs vary depending on the host species, parasite load, and the stage of infection. Disease expression is typically categorized into acute, subacute, and chronic forms, these forms may overlap during the same outbreak or even in individual animals [78] [109]. Surra is basically a disease of camelids and equines, in which typical clinical expression is described, but various pathogenic effects are observed depending on the various domestic and wild hosts concerned [90] [110]. These signs by host categories will be described in the following:

12.1. Camels and Horses

The typical clinical expression of Surra can be described in camels and horses, while donkeys, asses, and mules are of lower susceptibility. Surra in camels (*Camelus dromedarius* and *C. bactrianus*) may be acute with high fever, anaemia, weakness, and death; it is also frequently fatal sometimes within a few months; however, it is more often chronic than in horses and can frequently last 2 - 3 years (also called Tibersa) [35]. More recent studies confirm these patterns, noting that in camels the disease may progress as either a fulminant acute infection with high mortality or a chronic debilitating condition lasting several years, often with reproductive losses and immunosuppression [90] [109]. Furthermore, Surra has been associated with impaired fertility in male camels as well as immune dysfunction, characterized by depletion of circulating B-cells [110].

12.2. Cattle and Buffalo

In cattle and buffaloes, *T. evansi* infection often manifests as a chronic condition characterized by intermittent fever, progressive anemia, weight loss, and general debility. Occasional neurologic signs, such as: circling, excitation, or incoordination—may appear in severe or long-standing cases [111]. Abortions and reproductive losses have also been documented in buffalo herds [31]. Because parasitemia may be low and clinical signs non-specific, many infected bovines may remain subclinical carriers, complicating diagnosis and control efforts [61] [78].

12.3. Sheep and Goats

In small ruminants such as sheep and goats, *Trypanosoma evansi* infection is often mild or subclinical, although clinical disease can occur under conditions of high parasite load or stress. Reported signs include intermittent fever, lethargy, progressive weight loss, anemia, and occasional edema of the limbs or face, especially in goats [35]. In sheep, infections are frequently less apparent but may still cause hematological alterations, such as decreased packed cell volume and leukopenia [112]. Because parasitemia tends to be low and clinical signs are non-specific, small ruminants may act as silent carriers, sustaining parasite transmission in endemic regions [31]. In an experimental infection of Yankasa sheep with a Nigerian isolate of *T. evansi*, acute and chronic evolutions were observed, with fever, pale mucous membrane, epiphora, loss of appetite, emaciation, dullness, and

rough-haired coat; in acute evolution, the animals died within 2 weeks; postmortem observation indicated enlargement of the spleen and lymph nodes [113]. Goats are also most often of low susceptibility [63]; thus in experimental infections with a camel isolate from the Canary Islands, they showed mild symptoms with a few episodes of fever in early infection and arthritis in the next 6 months; although low, parasitaemia remained persistent [38]. In Philippines, experimental infection led to the observation of fluctuating fever, progressive emaciation, anaemia, coughing, testicular enlargement, and diarrhoea but not in all animals [35].

12.4. Other Naturally Infected Domesticated Species

In a variety of domesticated species beyond camels and livestock, *Trypanosoma evansi* infection presents with a broad spectrum of clinical signs, often reflecting host susceptibility, parasite load, and disease chronicity. In dogs, Surra may manifest as acute, subacute, or chronic disease, with signs that include fever, profound anemia, lethargy, edema of limbs, ocular lesions (such as corneal opacities), neurologic deficits, and in severe cases, sudden death [114] [115]. Pigs experimentally infected with *T. evansi* generally develop mild disease, showing little overt clinical pathology or impact on growth under low parasite challenges [67]. In other species, non-specific signs such as fever, weight loss, weakness, and edema have been documented, though infections are often subclinical or masked by overlapping conditions [31] [61]. In deer, the clinical outcome of *T. evansi* infection varies widely across species. Gill [116] documented acute and fatal forms in *Antilope cervicapra* and *Axis* species, while more chronic infections with anemia, progressive weight loss, and abortion were noted in *Axis axis* and *Rusa timorensis*. In *Cervus unicolor* from Mauritius, outbreaks produced acute fever, rapid emaciation, anemia, and high mortality [116]. Neurological involvement has been reported in *Cervus porcinus* (hog deer) in Thailand, with paresis, recumbency, convulsions, and death; histopathology confirmed parasite presence in the Virchow-Robin spaces of the brain [44]. Similarly, outbreaks in Java deer (*Cervus timorensis*) in Malaysia were associated with anemia, inappetence, respiratory distress, recumbency, and death, with Surra accounting for a mortality rate of about 27%, often complicated by concurrent hemoparasitic infections [117] [118].

12.5. Wild Hosts

Surra is classically described in a number of favored wild hosts such as Vampire bats, Capybaras, and Coatis. In the latter, experimental infections revealed the existence of severe anaemia, myocarditis, and meningoencephalitis [68]. *Trypanosoma evansi* is also present in a large range of other wild animals, including wild pigs, deer, and rodents, which are mostly healthy carriers. Experimental infections have been carried out and have demonstrated that a number of other species are receptive and susceptible to the parasite [119]. Agile wallabies (*Macropus agilis*) and dusky pademelons (*Thylogale brunii*) both proved to be very susceptible to the infection; they developed high parasitaemia 6 days post-infection, persisting

until death, between 1 week and 2 months; clinical signs were anorexia, weakness, ataxia, and anaemia [120]. *Trypanosoma evansi* was observed in 4 natural infections in Himalayan charming bears, in Pakistan; the animals exhibited pyrexia, accelerated pulse, tachypnea, depression, anaemic mucous membranes, and ataxia [121].

Surra in captive Sumatran rhinoceroses (*Dicerorhinus sumatrensis sumatrensis*) in Malaysia presented with depression, anorexia, incoordination, muscle tremor, nasal haemorrhage, recumbency, and labored breathing followed by death [122]. *Trypanosoma evansi* was suspected in a herd of Arabian dorcas gazelles (*Gazella dorcas saudiya*) and in one Sand gazelle (*Gazella subgutturosa marica*) in Kuwait; it revealed clinical signs of paresis of hindquarters and sudden death.

13. Diagnostic Tools

Definitive diagnosis of *Trypanosoma evansi* infection relies on laboratory confirmation. Direct detection is possible using parasitological methods such as microscopy of stained blood smears or concentration techniques, while molecular tools, including Polymerase Chain Reaction (PCR), provide higher sensitivity and specificity by identifying parasite DNA [123] [124]. Alternatively, serological assays such as Enzyme-Linked Immunosorbent Assay (ELISA) and the card agglutination test for *T. evansi* (CATT/*T. evansi*) are commonly employed to demonstrate immune contact, particularly in chronic or low-parasitemia cases [31] [77]. Parasitological diagnosis of *Trypanosoma evansi* is commonly performed on blood samples, although other body fluids, such as cerebrospinal fluid in animals showing nervous symptoms, or lymph node and joint aspirates may also be examined [31]. Direct microscopic examination of fresh blood at magnifications of ×400 - 500 is easy to perform and inexpensive, but its diagnostic value is constrained by low sensitivity, as parasites are typically visible only when parasitemia exceeds approximately 10 trypanosomes/mL of blood [61] [123]. To improve diagnostic sensitivity, enrichment techniques are frequently employed, enabling detection at lower parasitemia levels of about 100 - 200 trypanosomes/mL of blood. Common examples include the Hematocrit Centrifugation Technique (HCT), which concentrates parasites at the buffy coat-plasma interface after centrifugation [31] [125]; the Buffy Coat Method (BCM) under dark-ground or phase-contrast microscopy, which not only enhances visualization of motile trypanosomes, but also aids in the assessment of anaemia [126]. For situations requiring higher sensitivity, experimental inoculation of laboratory rodents can be employed to detect *T. evansi*. This technique significantly lowers the threshold of detection, making it possible to identify infections at parasitemia levels as low as 20 - 50 trypanosomes/mL of blood [31] [125].

Molecular evidence of *T. evansi* DNA can be determined using Polymerase Chain Reaction (PCR) tests. Although relatively costly and technically demanding, PCR is widely used to enhance the sensitivity of *T. evansi* detection. Comparative studies have recommended the use of Trypanozoon Repetitive DNA (TBR)

primers, which target high-copy repetitive sequences and are considered among the most sensitive for detecting *T. evansi* [127] [128]. In parallel, the phenol-chloroform DNA extraction protocol has been identified as the most efficient preparation method for high-quality parasite DNA [129] [130]. When combined, these methods can achieve diagnostic sensitivity levels for samples with as low as 5 - 10 trypanosomes/mL of blood or other biological fluids.

In addition to the use of parasitological or molecular tools for detecting *T. evansi* infection, serological tests that prove the immune contact between the host and the parasite are quite useful. They can be applied to investigations at herd or population level (prevalence or incidence studies), follow-up (seasonal or interannual variations), or control method assessment (trypanocide treatment or vector control). The most common tools are the Card Agglutination Test for *T. evansi* (CATT/*T. evansi*) [131] and the ELISA *T. evansi* [132]. CATT can detect immunoglobulin M and, therefore, early infections, whereas ELISA is generally used to detect immunoglobulin G, that is, established infections. Consequently, these tests are complementary and work well together. ELISA *T. evansi* is quite robust, regardless of the host species. It provides the same range of sensitivity and specificity (90% - 95%) in the various host species investigated, for example, camels, cattle, buffalo, and horses. The sensitivity of CATT *T. evansi* varies from one host to another. CATT seems highly sensitive in camels and horses, although it has a very low sensitivity in cattle (12%), even under experimental conditions [41]. An integrated approach that combines parasitological, molecular, and serological techniques is often recommended to improve diagnostic accuracy.

14. Methods of Treatment

The therapeutic management of Surra relies largely on a small number of trypanocidal drugs, each with varying efficacy, host specificity, and limitations. While treatment may temporarily control disease, challenges such as drug resistance, toxicity, and limited availability complicate long-term management in endemic regions.

Suramin has been one of the earliest compounds used in the treatment of *T. evansi*. Although its efficacy in cattle and buffaloes is limited, Suramin is highly effective in camels and horses, but its efficacy is limited in cattle and buffaloes. Suramin is administered intravenously and has been widely used in areas where trypanomosis in camel prevalent. However, its high cost, restricted availability, and the occurrence of relapses have reduced its reliability as a first-line treatment [31].

Diminazene aceturate is also widely accepted as a trypanocidal drug for the treatment of *Trypanosoma evansi* infection in several susceptible animal hosts. It is typically administered intramuscularly and reduces parasitemia effectively in the short term. However, relapses are common because the drug has limited ability to eliminate tissue-residing parasites, and its efficacy varies across regions and host species. Also, concerns about toxicity at higher doses (particularly neurotox-

icity and nephrotoxicity) limit its long-term use [31]. Reports of resistance to Diminazene aceturate in endemic areas have further limited its application, although it remains in use due to its relative affordability and accessibility compared to newer compounds [133] [134].

When treatment with Diminazine aceturate is ineffective, the use of Isometamidium chloride (Samorin®) is employed for Trypanosoma species such as *T. congolense* and *T. vivax*, but it has also been attempted *T. evansi* infections. This drug possesses both prophylactic and therapeutic benefits, but its efficacy in Surra has proven inconsistent, and it is not considered a preferred treatment compared to Melarsomine or Quinapyramine [128]. Quinapyramine salts, including Quinapyramine sulfate and Quinapyramine dimethylsulfate/chloride, remain among the most widely used drugs in camels and equines. These compounds exist in both curative and prophylactic formulations, allowing for flexibility in field application. Nevertheless, resistance to quinapyramine has been reported in several regions, raising concerns about its long-term effectiveness [135].

Melarsomine dihydrochloride is an organo-arsenical trypanocide and is considered one of the most effective treatments against *T. evansi* in camels and other livestock. It has shown superior efficacy compared to Suramin and Quinapyramine, including activity against resistant strains. The main drawbacks of Melarsomine are its relatively high cost and limited distribution, which restrict widespread use in resource-poor settings [31] [136].

Despite the availability of these chemotherapeutic agents, significant limitations remain. Resistance to commonly used compounds such as Quinapyramine and suramin has been documented in endemic areas, reducing their effectiveness [31]. Moreover, the toxicity associated with arsenical compounds such as Melarsomine poses potential safety concerns, particularly in prolonged or repeated treatments [23]. Accessibility is another challenge; the high cost and limited supply of drugs, especially Cymelarsan®, restrict their application in many developing countries where Surra is endemic.

In addition to trypanocidal therapy, supportive treatment is often administered to improve clinical recovery. Nutritional supplementation, iron therapy, vitamins, and general parasite control (such as deworming) help restore productivity in infected animals and reduce mortality [135].

Emerging therapeutic alternatives are under investigation. Research into combination therapies, novel trypanocides, immunomodulatory approaches, and plant-derived compounds is ongoing; however, these remain experimental and have not yet been integrated into field practice [137].

Measures such as the provision of novel, safe and affordable therapeutic options, improved drug stewardship, and integrated approaches that combine chemotherapy with supportive care and vector management, such measures, will greatly impact the constraint on livestock productivity and a persistent threat to animal and, potentially, human health due to Surra. Finally, progress toward a practical vaccine, although historically elusive, remains a critical priority if long-term and sustaina-

ble control of *T. evansi* is to be achieved.

15. Control

A multifaceted approach is pertinent for the effective control of *Trypanosoma evansi*. This process should encompass chemotherapy, vector management, and improved husbandry practices. Vector control plays a critical role in reducing disease transmission and can be achieved through several means, including targeting biting flies (Tabanids and Stomoxys), the use of insecticides, traps, and environmental management [31]. To further limit the spread of disease, strengthened surveillance, movement control of infected animals, and improved awareness among farmers and veterinarians are also essential [61].

The unavailability of effective vaccines against *Trypanosoma evansi* is largely due to the parasite's ability to evade the immune system through extensive variation of its surface glycoproteins. Hence, disease control relies primarily on the use of Trypanocidal drugs and preventive management measures aimed at minimizing exposure to infection [71].

Integrated approaches that combine these strategies are considered the most effective for sustainable control of Surra.

16. Conclusions

Trypanosoma evansi is the most widely distributed mechanically transmitted haemoprotozoan parasite, which is pathogenic to both domestic and wild animals globally. In susceptible hosts, the disease (Surra) causes severe anemia, edema, immunosuppression, and neurological disturbances that can lead to death, inflicting considerable economic losses through reduced productivity, abortion, infertility, lowered milk yield, interference with vaccination programs and mortality, in addition to the cost of Trypanocidal treatments. The occurrence of Surra, although seasonal, with higher incidence amid the rainy season, coincides with increased populations of biting flies (Tabanus and Stomoxys). Conventional parasitological methods for diagnosis, which depend on detecting circulating trypanosomes, lack sensitivity, as more than 70% of infections remain cryptic and undetectable by direct microscopy. Serological tests such as ELISA and CATT/*T. evansi*, along with DNA-based molecular assays, are therefore preferred for improved detection. For Surra control measures to achieve optimal effectiveness, an integrated approach involving proper management, chemotherapy, chemoprophylaxis, and vector control should be employed. Future research could target the development, validation, and field implementation of diagnostic tools that are highly sensitive, affordable, simple, and specific, to accurately distinguish infected from non-infected animals and to better quantify the epidemiological impact of the disease.

Strengthening diagnostic capacity is vital for early detection and control of *T. evansi*. There is a pressing need for affordable, rapid Point-of-Care Tests (POCTs) suited to field use in resource-limited settings [9] [31]. Dot-blot assays employing specific or recombinant DNA (cDNA) or the use of immunodominant protein

antigens may show more promising sensitive detection methods to enhance routine screening, diagnosis and effective treatment, thereby boosting One Health surveillance.

17. Recommendations

Effective control of *Trypanosoma evansi* requires a comprehensive and sustainable approach that integrates diagnostics, therapeutics, vector management, and stakeholder participation. The following recommendations are proposed to strengthen disease control and research efforts:

1) Development of Improved Diagnostic Tools

There is an urgent need to design and validate highly sensitive, specific, and affordable diagnostic tools capable of distinguishing active infections from prior exposure. Field-deployable molecular and antigen-based assays should be prioritized to enhance early detection and epidemiological mapping.

2) Integrated Control Strategies

Disease control should combine chemotherapy, chemoprophylaxis, and vector management with improved husbandry practices. Regular monitoring of drug efficacy and responsible use of trypanocides are essential to minimize the emergence of resistance and treatment failure.

3) Enhanced Vector Control

Control of mechanical vectors such as Tabanus and Stomoxys species should focus on insecticide-treated traps, repellents, and environmental sanitation, especially during and after the rainy season when fly populations peak.

4) Capacity Building and Farmer Education

Awareness campaigns and training programs for livestock owners, animal health workers, and extension officers should be intensified to promote early recognition of Surra, proper drug use, and preventive management practices.

5) Surveillance and Epidemiological Mapping

Strengthening active surveillance through regular screening and reporting is vital for understanding disease distribution. Incorporating geospatial and molecular epidemiology tools would help identify high-risk zones and improve resource allocation.

6) Promotion of Research and Innovation

Continued investment in research is required to explore new trypanocidal compounds, combination therapies, and alternative control measures. Collaborative studies across endemic regions should focus on understanding resistance mechanisms and host-parasite interactions.

7) Adoption of a One Health Framework

Considering the zoonotic potential of *T. evansi*, disease control should align with the One Health approach, promoting collaboration between veterinary, medical, and environmental sectors to mitigate animal and human health risks.

Authors' Contributions

Conceptualization: G. O. Egwu and A. M. Bello. Methodology: G. Enid and S. I. Enem.

Literature Search & Data Collection: O. Z. Tenuche and G. Enid. Investigation: O. Z. Tenuche. Writing—Original Draft: O. Z. Tenuche and A. M. Bello. Writing—Review & Editing: G. O. Egwu and O. Z. Tenuche. Supervision: G. O. Egwu and S. I. Enem.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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