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TRYPANOSOMIASIS AND ITS DIAGNOSTIC TECHNIQUES IN CAMEL: A COMPREHENSIVE REVIEW

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ABSTRACT

Trypanosomiasis, a parasitic disease caused by protozoan parasites of the genus Trypanosoma, poses a significant threat to camel populations worldwide. This study designed for the diagnostic techniques used for detecting Trypanosoma infections specifically in camels. The disease, commonly known as camel trypanosomiasis or surra, adversely impacts the health and productivity of camels, thereby affecting livelihoods dependent on these animals. Various diagnostic methods are employed for the detection of Trypanosoma infection in camels, including microscopic examination of blood smears, serological tests like ELISA (Enzyme-Linked Immunosorbent Assay), and molecular techniques such as PCR (Polymerase Chain Reaction). Each method has its strengths and limitations in terms of sensitivity, specificity, and practicality in field settings. Efforts to control and manage camel trypanosomiasis rely heavily on accurate and timely diagnosis. Early detection facilitates prompt treatment and prevents the spread of the disease within camel herds. Additionally, the development and optimization of diagnostic tools tailored specifically for camels are crucial for effective disease surveillance and control. This study underscores the importance of ongoing research and innovation in diagnostic methodologies to combat trypanosomiasis in camel populations efficiently.

Keywords: Trypanosomiasis, diagnostic techniques, camel, parasitic disease, protozoan.

INTRODUCTION

A serious health and financial risk to animals, especially camels, is posed by the protozoan parasite Trypanosoma (Desquesnes et al., 2013). Trypanosomes in camels induce the Surra sickness, which manifests as fever, anemia, weight loss, and neurological symptoms (Tarİq et al., 2022). For illness management and control to be effective, accurate diagnosis is crucial. Trypanosomes belong to the family Trypanosomatidae. They are single-celled protozoa with flagella. This genus contains a number of species that cause trypanosome infections, which can harm humans and domestic and wild animals alike. Trypanosoma brucei, Trypanosoma equiperdum, and Trypanosoma evansi, members of the Trypanozoon sub-genus, are the causative agents of livestock trypanosomoses, which have a substantial negative impact on the global animal productivity and socioeconomic conditions. Trypanosoma evansi is the pathogenic mammalian trypanosome that initially appeared in the blood of Indian horses and dromedaries in 1880. It developed from Trypanosoma brucei and adapted to mechanical transmission. Due to this adaptability, the species was able to spread outside of Africa's tsetse belt, which resulted in the development of a crippling disease that affects animals and is known as "surra" in Asia and Africa and "mal de cadeiras" in Brazil (Tarİq et al., 2021).
From a clinical standpoint, however, diseased camels may show a number of signs, including decreased food and water consumption. As the illness progresses, the impacted camels’ hump recedes, and their hair coat gets rough and drab with hair loss at the tail. Edema beneath the abdomen is one observable symptom that is especially apparent in the morning. Abortions are possible in pregnant camel, and calves delivered to infected dams usually die of the disease. Pallor is seen on the mucous membranes of the eyes, and camels that are afflicted may exhibit temperature fluctuations, with first peaks as high as 41ºC. Furthermore, as reported by Kohler-Rollefson et al., in 2001 and the Food and Agriculture Organization in 2000, the urine frequently has a distinguishable odor. Despite this, camels do not exhibit clear symptoms of the illness, and a clinical examination is not always helpful in making a definitive diagnosis. However, the Food and Agriculture Organization found in 2000 that parasites become detectable in the bloodstream 13 to 16 days after a blood meal by a mechanical vector.

Diagnosing trypanosomosis requires several main categories, including clinical, parasitological, molecular, chemical, and serological techniques. The employment of precise and sensitive diagnostic techniques is crucial in the context of research, especially in epidemiology, and field applicability must be taken into account. According to FAO (2000), the specific objectives of the tests and the availability of tools influence the diagnostic methods that are selected.

The parasitological techniques follow to diagnose T. evansi in camels are thought to be practical, quick, and affordable. However, their effectiveness is restricted, especially when it comes to recognizing animals that have trypanosome infections, especially when their parasitemia is low, and when the condition is chronic (Ahmed, 2008). The card agglutination test (CATT), ELISA, and LAT are examples of serological assays used to identify circulating antibodies in the serum of infected camels. Because these tests can be used in both field and laboratory settings, they offer a beneficial alternative (Songa, 2003).

Molecular diagnostic techniques have led to the development of multiple diagnostic assays that focus on the use of PCR to detect trypanosomal DNA. DNA-based techniques, such as polymerase chain reactions (PCR) and DNA probes, are particularly beneficial in these situations for assessing the effectiveness of treatment (Eyob and Matios, 2013). In the extracellular fluid of their mammalian hosts, trypanosomes proliferate and multiply, particularly in the circulation, where they encounter immune system defenses from both the innate and adaptive varieties. Because of selective pressure during evolution, many parasites have developed sophisticated escape strategies. Apart from their direct pathogenic effects, trypanosomes are capable of suppressing the immune system (Pathak and Singh 2005).

**Camel Trypanosomosis (Surra)**

i. **Etiology**

T. evansi is categorized under the subgenus Trypanozoon of the Brucei group and is recognized as the first pathogenic trypanosome, having been discovered in India in 1880. In contrast to other members of this group, T. evansi is not able to develop cyclically in tsetse Glossina species. Morphologically, T. brucei and T. evansi appear to be identical in blood smears. There is a difference in the molecular structure of Trypanosoma evansi’s kinetoplast DNA (Radostits et al., 2007).

ii. **Epidemiology**

Trypanosomiasis is one of the most important diseases affecting livestock, equines, and dogs within the Sub-Saharan region (Geiger et al., 2018; Anderson et al., 2011). Cross transmission of parasites between livestock and wildlife hosts has been
reported, especially in areas in close proximity to game parks and wildlife reserves. Wildlife species can survive within the tsetse belts across the Sub-Saharan region, despite being reservoir hosts for multiple species of trypanosome. The high prevalence of trypanosomiasis within protected areas traditionally has rendered these areas unattractive for livestock keeping and agricultural production (Bengis et al., 2002).

iii. **Distribution**

*T. evansi* is found throughout Africa, Asia, the Middle East, Central America, and South America. The predominant host species of the parasite varies based on the geographic region, indicating a broad host range. The main host of *T. evansi* is camels in sections of Africa that lie outside the northernmost limits of the tsetse fly belt and in some east African regions. In contrast, the primary species impacted in Central and South America are horses (OIE, 2013).

iv. **Transmission**

There are several ways that *T. evansi* can spread, such as by vampire bats and insects that feed on blood or lymph. Furthermore, there are four further transmission routes: iatrogenic, peroral, vertical, and horizontal. Each of these has unique epidemiological implications. Season, geography, and host species are only a few examples of the variables that affect the significance of various transmission pathways (Marc, 2013).

v. **Host factor**

Equine, donkey, mules, camel, canine, and feline species are the most severely afflicted by *T. evansi*. Of these, sheep and goats are less susceptible than camels, horses, dogs, and Asian elephants. It is less likely to harm cows and pigs. In particular, when it comes to identifying subclinical (non-patent) illnesses, rats and mice are highly susceptible a fact that (Reid et al., 2001).

vi. **Pathogenesis, Pathology and Clinical Findings**

Pathogenic conditions can be induced by *T. evansi* is unique to their host species by infecting a wide range of hosts. The illness in camels first appears as a rise in body temperature, which is a sign of parasitemia. Camels with infection show signs of growing anemia, severe depression, lethargy, and loss of condition. They also frequently die quickly, anemia was shown to be a noteworthy clinical feature in cases with camel trypanosomosis in Morocco (Rami et al., 2003).

Anemia plays a major role in the pathogenesis of African trypanosomiasis in general and surra in particular. Although the anemia in camels infected with *T. evansi* is characterized as macrocytic and hypochromic, it is hemolytic and hemophagocytic in the early stages of the illness. The mechanisms underlying the increased erythrophagocytic activity in this situation are still not entirely known (Enwezor and Sackey, 2005).

vii. **Treatment**

Quinapyramine sulfate and suramin are the two drugs available to treat Trypanosoma evansi infections in camels. The recommended dosage of suramin, which is given intravenously for both preventive and therapeutic purposes, is 12 mg/kg body weight. When used therapeutically, quinapyramine is administered subcutaneously as methyl sulfate; the usual dosage range for this is 3 to 5 mg/kg body weight. On the other hand, it is used as a
pro-salt chloride/methyl sulfate mixture, with a suggested dosage of 5 to 8.3 mg/kg body weight, for both therapeutic and preventive purposes. Still, some drugs, like homidium bromide, are not deemed therapeutic, and some drugs, such diminazene aceturate, are thought to be too toxic for camels (Evans et al., 1995).

viii. **Control and Prevention**

The prudent application of the few trypanocides that are now accessible is the main strategy for trypanosomosis prevention and control. However, decrease the emergence of drug resistance this entails the deliberate use of curative medications. Furthermore, the continuous deployment of environmentally friendly vector control programs is essential to these efforts (FAO, 2000) and (Anene et al., 2001). Three primary strategies have been used to control trypanosomosis in endemic countries, frequently in conjunction with altered management practices: managing vector populations, administering chemoprophylaxis, and using animals that exhibit tolerance to Trypanosoma infection (Anele et al., 2001).

**Diagnostic Approaches of Surra**

Since Surra does not show pathognomonic symptoms, an infection must be confirmed through laboratory testing. This calls for the use of molecular, serological, and parasitological techniques. Direct microscopic analysis of blood or buffy coats is the primary method for diagnosing parasites. Sometimes rodents like mice or rats receive subcutaneous injections of camel blood. The limited sensitivity of this testing method which often falls below 50 % must be noted (Monzon et al., 2002).

Underdiagnosis of *T. evansi* is a significant effect of the low sensitivity of parasitological diagnostic procedures, often leading to a reported infection level that is lower than the true prevalence. However, when it comes to distinguishing between infections that have been healed, serological tests such as the Card Agglutination Test for Trypanosomosis (CATT), Enzyme Linked Immunosorbent Assay (ELISA), and Immunofluorescent Antibody Test (IFAT) have limits despite their sensitivity. Because of this, it could be challenging to estimate the true prevalence of *T. evansi* infections in affected areas (Enwezor and Sackey, 2005).

**Parasitological Examination**

*T. evansi* infections can be diagnosed parasitologically using methods such as animal inoculation, parasite concentration techniques, and microscopic blood analysis. The direct microscopic analysis of blood is the simplest and most widely used method among these. Wet blood film technique or stained thick and thin smears can be used to accomplish this (FAO 2000). In the wet film smear, trypanosomes can be seen either directly between blood cells or indirectly by the movement that they cause within the blood cells. Furthermore, using a light microscope, thin smears of lymph and recently generated lymph can be used to identify various trypanosomes morphologically (Zhao, 2013).

The assessment of different techniques under experimental settings has shed light on their detection limits when it comes to the existence of distinct trypanosome species in a blood sample. The following order of sensitivity degradation was shown by the results: HCT (hematocrit centrifugation technique) > DG (differential count of trypanosomes per field) > thick film > thin film > wet film. There are a number of ways to perform this evaluation, including using light microscopy to identify parasites based on their physical traits and directly observing blood under a microscope (McOdimba, 2008). An alternative approach makes use of the wet film method to identify motile trypanosomes in dyed thick and thin
streaks. Wet blood film analysis is a rapid and effective method of testing for a large number of animals, although it is not very sensitive and may miss up to 50% of the affected animals. Improved diagnostic sensitivity has been achieved by improving the fundamental approach of evaluating fresh or stained blood films by centrifuging the blood and using a hematocrit procedure. Additionally, wet film technique is used to detect mobile trypanosomes, and light microscopy is used to identify parasites based on their appearance using stained thick and thin smears (Shahzad et al., 2012).

**Buffy Coat Technique**

Gather 70 µl of blood, then divide it evenly between two 75 × 1.5 mm heparinized capillary tubes. Place plasticine over the wet ends of the tubes and centrifugex them in a hematocrit centrifuge for five minutes at 3000 g, typically at 12,000 rpm. Throughout this process, the blood must remain concentrated in order to increase diagnostic sensitivity.

Following Review the capillary tube and focus on either the dark ground buffy coat technique (DG) or the hematocrit centrifuge technique (HCT) to optimize the accuracy of the diagnosis (Gutierrez, 2012).

**Animal Inoculation**

A dependable technique for identifying sub-patent evansi infections in camels is to inject laboratory rodents with tainted camel blood. This methodology supports the most effective direct diagnostic procedure, which is the inoculation of laboratory mice with camel blood (Zweygarth et al., 2000).

Minimize animal inoculation unless it is absolutely necessary, as the use of animals in biological testing is becoming a matter of great concern. One justified use for laboratory animals is the detection of subclinical diseases in domesticated animals; rats and mice are commonly used in this capacity because of the wide range of infectivity of *T. evansi* in small rodents (Monzon et al., 2002). Although there are certain sensitivity issues with rodent inoculation, buffy coat material can increase the efficiency of the procedure (Monzon et al., 2002).

*T. evansi* may be identified in blood at concentrations as low as 1.25 percent. For very sensitive detection, rats (1-2 ml) or mice (0.25–0.5 ml) are intraperitoneally injected with heparinized blood (Reid et al., 2001). Subsequently, animals are bled from the tail every 48 hours to identify parasitemia. Various parameters, such as the trypanosome strain, inoculum concentration, and the laboratory animal strain employed, determine the incubation period for the growth and toxicity of parasites. Typically, this period is short (5 ± 2 days), while it can occasionally extend up to 2 weeks (Reid et al., 2001).

**Chemical Examination**

It can be difficult to diagnose *T. evansi* infection at any stage based just on clinical investigation or microscopic blood analysis. Because of this restriction, *T. evansi* infection has been diagnosed using tests such Jon's nitric acid test, the formol gel test, the mercuric chloride test, and the stilbamidine test (El-Sawalhy, 1999).

**Mercuric Chloride Test**

As an indirect mass screening technique for dromedary herds, this methodology offers a dependable way to identify camel surra (Barnett, 1997). This approach involves adding one or two drops of the suspect serum to a test tube containing one milliliter of mercuric
chloride solution. After a 15-minute incubation period and a gradual mixing of the solution, a white precipitate is evident (Dargantes, 2005).

**Stilbamidine Test**

A test tube is filled with an aqueous solution of stilbamidine, usually 0.5–200 ml of a 10 % solution, for the Stilbamidine test. The solution is then covered with one or two drops of the suspected serum. In the event that coagulation occurs and it sinks and dissolves in five to ten minutes, the test is deemed successful. It's important to remember that stilbamidine is helpful in identifying surra and may also be able to distinguish between different infection phases (Barnett, 1997).

**Formol Gel Test**

A range of indirect tests are used in the mass screening of dromedary herds to detect the presence of the parasite. Adding two drops of 40 % formaldehyde to one milliliter of suspicious serum is one such technique. If the test is positive, a gel will form in about an hour (Knowler, 2003).

**Serological Examination**

Trypanosome infection in camels has been diagnosed using a variety of specialized serological tests, including capillary agglutination tests (Brown and Torres, 2008), immune fluorescent antibody tests, passive haemagglutination tests (Alan, 2013), and enzyme-linked immunosorbent assays (ELISA) (Gebreyohans and Gangwar, 2010). Using a straightforward ELISA method, a commonly available protein A peroxidase conjugate has been utilized in the serodiagnosis of camel trypanosomosis. Salwa and Shams (2012) have successfully adapted the "Testryp CATT" card agglutination test for the serodiagnosis of evansi infection in camels. Smith & Kline originally developed this test to identify Gambian sleeping sickness.

**Antibody Detection**

A specific type of serological test that searches for specific antibodies—blood proteins that are part of the immunoglobulins is necessary in order to assess the host's immune response to an infection. In horses, *T. evansi* and *T. equiperdum* have been identified using the complement fixation test (CFT), one of the antibody techniques (Antoin, 2004). Additionally, trypanosome herd identification has been accomplished by the use of enzyme-linked immunosorbent assays (ELISA) and indirect fluorescent antibody tests (IFAT) (Stephen, 2003). Additionally, the card agglutination test (CATT), which is thought to be the easiest test for *T. evansi*, has been used (Arknwa, 2007).

**Antigen Detection**

Trypanosome antigens have been shown to have a strong correlation with both patent and sub-patent camel illnesses, and their detection in blood or serum is thought to be more accurate (Olaho et al., 1996). Enzyme immunoassays have been developed in the field of diagnostics to specifically detect antigens as opposed to antibodies (Galal et al., 2014). The purpose of these tests is to detect the presence of *T. congolense*, *T. vivax*, and *T. brucei* circulating antigens in the blood of afflicted animals. Furthermore, *T. evansi* has been particularly tested using the latex agglutination test (LAT) (Desquesnes et al., 2007). Trypanosome antigen detection is seen to be as good as a parasitological diagnosis and is a
good sign of active infection, especially if the animal hasn't had treatment for the illness recently (Njiru et al., 2004).

The internal or somatic unsecreted antigen, which is only accessible during trypanosome lysis, is the target of the monoclonal antibody used in antigen ELISA. The test may produce negative findings in the early stages of infection, before the initial parasitaemic peak, since antigens are either absent or present in low concentration in the blood. Consequently, it is essential to combine parasitological methods with antigen detection ELISA for a precise trypanosomosis diagnosis (Imadeline and Majid, 2006). The use of two methods improves the diagnostic process's sensitivity and reliability, which results in a more thorough evaluation of the infection state.

Molecular Examination

Molecular techniques have become the main instruments in modern research because they are so successful in identifying parasites in both insect vectors and mammalian hosts (FAO, 2000). The basic idea underlying molecular tests is the identification of certain nucleotides that are representative of a species, subgenus, or even strain of trypanosomes. The two main methods used in molecular detection are DNA probes and polymerase chain reaction (PCR) (FAO, 2000). These techniques are useful because of their great sensitivity and specificity for the exact and precise identification of trypanosomes.

DNA Probes (Nucleic Acid Probes)

The material being examined is heated during DNA probe techniques in order to split its two strands. These strands are then attached to a membrane in a manner that inhibits recombination upon cooling. Next, a probe is added. This probe is a linear sequence of nucleotides that is intended to complement a particular sequence found in the parasite sample. The particular region of the parasite DNA is then bound by the probe, causing it to hybridize. The use of the probes in methods like ELISA is made easier by the ability to identify this interaction by tagging them with radioactive isotopes or enzymes (Jamie and Brisse, 2005). DNA sequences of the target parasite can be located and identified in the sample using this technique.

Polymerase Chain Reaction (PCR)

The DNA polymerase enzyme is used in the Polymerase Chain Reaction (PCR) method to duplicate DNA sequences and produce a sufficient amount of detectable DNA material. Two primers, one for each strand of DNA, are used in this procedure to denature the parasite's DNA using heat. The primers are short nucleotide sequences. The exact sites on the appropriate single strands of parasite DNA are intended to be complemented by these primers. Once DNA polymerase binds to these complementary sites, it starts to replicate the remaining sequences that extend from each primer. Small DNA segments are amplified by the polymerase with each repetition cycle. Certain temperature conditions and required materials such as primers that target a specific DNA region, DNA polymerase that can synthesise a copy of the targeted DNA segment, and deoxynucleotide triphosphates (dNTPs) are used to amplify a particular and limited amount of DNA (Holland et al., 2002).

To identify trypanosomes and reduce the possibility of false positive results, PCR and DNA probe technology can be used in tandem (Desquesnes et al., 2001). In this procedure, primers that target the particular section of the DNA, deoxynucleotide triphosphates (dNTPs) that aid in the production of the new DNA copy, and a DNA polymerase that can synthesise a
copy of the DNA segment are used. This method requires amplifying a limited amount or a particular region of DNA at a precise temperature (Holland et al., 2002).

**Immunological Significances**

Major immune responses are linked to trypanosomosis in camels. Research suggests that camels infected with *T. evansi*, both acute and chronic, have higher gamma-globulin (IgM) levels (Dary et al., 2011). However, since a sizable fraction of the produced antibodies are autoantibodies, this heightened immune response is not protective. Furthermore, increased leucocytosis, neutrophilia, and eosinophilia are suggestive of *T. evansi* infections in camels, according to data made by Holland and Hong (2012).

A considerable collection of mononuclear phagocytic cells is observed in the wounded tissue during the acute stage of the illness, with a notable reactivity in the lymph nodes and spleen. During this phase, the prevalence of plasma cells increases, which could account for the extensive hyperplasia of lymphoid tissues linked to *T. evansi* infections. But when the illness advances to its latter phases, the immune system's lymphoid cell count decreases (Verloo, 2008).

**Immune Response**

The host animal is impacted by trypanosomosis-induced immune system reactions. The immune system's main job is to protect the body against infections, but sometimes it can also overreact, react incorrectly, or cause immunological-mediated illnesses that show symptoms in the body (Stijlemans et al., 2007). Trypanosomes are rare in the general population, maybe as a result of the immune system's dual defense against self-antigens and parasites. By releasing tissue proteins and encouraging the synthesis of self-antigens, parasites can induce inflammation and tissue damage through molecular mimicry (Soares and Santos, 1999).

*T. evansi* interacts with the host's immune system through a variety of mechanisms, including innate and adaptive immunity, as an extracellular parasite. Trypanosomal DNA and the GPL anchor of the VSG are two molecules produced by dead trypanosomes that can activate macrophages and cause the release of pro-inflammatory substances such as TNF, IL-6, IL-1, IL-10, and NO. Controlling the early peak of parasitemia depends on this immune system response (Stijlemans et al., 2007).

Naessens (2006) revealed that increased levels of gammaglobulin (IgM) were observed in camels that had both acute and chronic infections with *T. evansi*. But since autoantibodies account for the majority of the produced antibodies, this increased IgM response is not protective. During the acute stage of the disease, significant immunological responses are detected in the spleen and lymph nodes, which may help to explain the particular extensive hyperplasia of lymphoid tissue reported in *T. evansi* infections. Throughout the late phases of the illness, the immune system loses lymphoid cells (Debson, 2009).

**Immune Evasion and Antigenic Variation**

African trypanosomes are mostly composed of roughly $5 \times 10^6$ dimers of variable surface glycoproteins (VSG), which entirely encase the trypanosomes in their bloodstream form. This VSG, which ranges in size from 12 to 15 nm, produces a thick coating that covers the trypanosome's whole surface. Surprisingly, $15–20\%$ of the parasite's total protein composition is found in this VSG in its bloodstream form. This surface coat's attachment to the trypanosomes' outer membrane is mostly dependent on glycosylphosphatidylinositol
(GPL) anchors (Field and Carrington, 2009). These GPL anchors may have a role in inducing the host's immune system in response to trypanosome infection by making the changing surface antigen water insoluble (Filex, 2010).

*Trypanosoma evansi* has a restricted repertoire of Variable Surface Glycoproteins (VSG), in contrast to trypanosomes that use a tsetse fly as an intermediary host. The rearrangement of the VSG repertoire within this vector and the interchange of genetic information are the causes of this divergence (Engstler et al., 2007). When parasitemia rises, most parasites are homotypes—that is, they share the same antigenic type. This particular homotype is recognised by the immune system of the host, which produces antibodies to combat it. As the dominating Variable Antigenic Type (VAT) parasites are eliminated, the parasitemia goes through a falling phase. Parallel to this, parasites that express heterotypes or minor VATs multiply, with one version out competing the others. Then, this dominant variation emerges as the new homotype, starting a new wave of parasitemia and helping to create a chronic and long-lasting infection. In this process, antigenic variation is crucially dependent on the production of Variable Surface Glycoproteins (VSG). In the end, this tactic helps the parasite by avoiding the host's defences and creating a long-lasting infection (Field et al., 2009).

### Immune Suppression

Both humoral antibody responses and T-cell-mediated immunity are impacted by the extensive immunosuppression that pathogenic trypanosomes cause. The host's immune system gradually deteriorates as a result of the immunopathology that results; either secondary infections or a high parasite burden finally cause the host to die. The production of suppressor T cells and macrophages, polyclonal stimulation of B cells, and modifications to antigen handling and presentation are among the mechanisms linked to trypanosome-mediated immunosuppression. Several investigations have confirmed these findings (Salwa and Shams, 2012).

The main agents responsible for immunosuppression are macrophages. They release a range of proteins and cytokines upon activation, which can have a number of effects, including as B-cell stimulation and T-cell repression. Normally activated macrophages emit a substance called tumour necrosis factor (TNF), which has two functions in the context of trypanosome infections: it controls parasitemia and plays a part in infection-associated pathophysiology, which includes fever, organ disorders, and anaemia. Trypanosome-induced immunosuppression is also linked to trypanosome enzymes. Membrane fluidity and cellular damage have been associated with enzymes including phospholipase, neuraminidases, and proteases (Fung et al., 2007).

### CONCLUSION

The critical impact of trypanosomiasis on camel populations worldwide, highlighting the significant economic and health implications for communities reliant on these animals. The study focused on diagnostic techniques crucial for early detection and management of Trypanosoma infections in camels, emphasizing the diverse array of methods available such as microscopy, serology, and molecular assays. Each method presents unique advantages and challenges, influencing their suitability for use in varied settings. Continued research and innovation in diagnostic tools tailored specifically for camels are essential for enhancing disease surveillance and implementing effective control measures. By improving diagnostic accuracy and timeliness, these efforts aim to mitigate the burden of camel trypanosomiasis and safeguard both animal welfare and human livelihoods dependent on camel husbandry.
AUTHORS CONTRIBUTION

Conceived and designed: MM Rahimmon, AH Mirani & JK Sahito, designed the title: MA, MM Rahimoon, PA Khoso & RA Leghari, Analyzed the data: MA & NP Junejo Contributed materials MA, wrote the paper: MA.

CONFLICT OF INTEREST

The authors declared that there is no conflict of interest.

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