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Camel trypanosomosis - a review

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ABSTRACT

Camel trypanosomosis (surra), caused by *Trypanosoma evansi*, is the most important single cause of morbidity and mortality in camels. The disease, transmitted non-cyclically by other haematophagus flies (e.g. Tabanus) is endemic in Africa, Asia and South America, and in addition to camels other species of domesticated livestock are affected. Because of the wide geographic range of surra, its control has attracted international attention, with a focus on formulating and implementing effective strategies aimed at increasing productivity and achieving a decrease in mortality and morbidity. In this review, the clinico-pathological effects of surra are presented, as their understanding may help in the design of effective control. Anaemia appears to be a major component of the pathology of surra. Its development and persistence in the course of the disease induce anoxic conditions which manifest signs of dysfunction in various organs as a result of a fall in tissue pH and vascular damage. This is followed by the release of large quantities of cytoplasmic and mitochondrial enzymes, especially aspartate alanine transferase (AST) and alanine transferase (ALT), among others, into serum, causing further cellular and tissue damage. The net effect associated with the above changes is immunosuppression which later develops and predisposes the animals to other infections and death if untreated. Therefore, emphasis is placed on accurate diagnosis of surra, treatment with effective trypanocidal drugs such as trypan and the use of vector control methods in the control and management of this disease.

Key words: trypanosomosis, surra, *Trypanosoma evansi*, Tabanus, anoxia, immunosuppression

Introduction

The camel is the most efficient domesticated animal for converting fodder into work, transport, milk and meat.

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Camel trypanosomosis, also known as surra, is a disease of camels caused by *Trypanosoma evansi*. The disease is the most important single cause of economic losses in camel rearing areas, causing morbidity of up to 30.0% and mortality of around 3.0% (NGERENWA et al., 1993; EGBE-NWIYI and CHAUDRY, 1994; PACHOLEK et al., 2001; NJIRU et al., 2002).The causative agent, *Trypanosoma evansi*, was discovered by Griffith Evans in 1880 in infected camels and equids in the Dara Ismail Khan district of Punjab (EVANS, cited by INDRAKAMHANG, 1998). Since then, studies have shown that the parasite can infect all species of domesticated livestock, although the principle host varies geographically (INDRAKAMHANG, 1998; EL-SAWALHY and SEED, 1999; AL-RAWASHDEH et al., 2000).

In Africa, beyond the northernmost limits of the tsetse fly belt, and in parts of East Africa, camels are the most important host (DIA et al., 1997), whilst in Central and South America the horse is principally affected (MONZON et al., 1990, SILVA et al., 1995). In Asia, a much wider range of hosts is involved, including the Bactrian camel and dromedaries, cattle, buffalo, horses and pigs (PATHAK et al., 1993; PARTOUTOMO et al., 1994; TUNTASUVAN et al., 1996; EL-SAWALHY and SEED, 1999; AL-RAWASHDEH et al., 2000; PACHOLEK et al., 2001). This is contrary to observations in Africa and South America, where there is little evidence to suggest that domesticated livestock other than camels and horses, respectively, are clinically affected or infected with *Trypanosoma evansi*. Nevertheless, there are reports of serological evidence of infection in goats and sheep from the Sudan and in cattle from Brazil (BOID et al., 1981; FRANKE et al., 1994; LUCKINS, 1998) and both goats and cattle have been considered as potential reservoirs of infection (DENNING, 1989; LUCKINS, 1998). In Nigeria, camels are found in the northern part of the country, most commonly in Borno, Kano, Katsina, Kebbi, Sokoto, Jigawa and Yobe states, where they are utilized considerably as sources of meat. Most of the camels found in these areas are traded from the neighbouring Niger and Chad Republics (OCHAPPA, 1988).

Natural and experimental camel trypanosomosis have been described in different parts of the world (DIA et al., 1997; AL-RAWASHDEH et al., 2000; PACHOLEK et al., 2001; NJIRU et al., 2002). Severe outbreaks, which occurred in different parts of the world where several thousand animals died in the 1970s and, of late, in 1994 and 1995, for instance, in Pantamal, Brazil, have also been well documented (LUCKINS, 1998).These epidemics pose a major constraint to camel productivity given their importance as a source of meat, milk production, transportation and draught power, as well as by-products (wool, hair, skin and hides). In addition, they also provide foreign currency to their owners from their export (ELAMIN et al., 1999).

Because of the range of agro-ecological zones and the diverse farming systems in which the disease occurs, as well as its debilitating effects on a variety of livestock, surra has attracted international attention in recent years, with the hosting of an international symposium on strategies for research and control of surra held 19-22 August, 1998, at

Obihiro University, Japan, (OBIHIRO, 1998). The symposium was timely, given the recent epidemics. Therefore, formulating and implementing strategies for effective control of surra as expected will result in decreased production costs, increased productivity, as well as a decrease in morbidity and mortality due to the disease.

In this review we will attempt to summarize the present state of our knowledge based on available literature, as well as drawing attention to gaps in our knowledge of camel trypanosomosis.

Etiology

Trypanosoma evansi is a species belonging to the subgenus Trypanozoon and is the causative agent of camel trypanosomosis. It is hypothesized that *Trypanosoma evansi* originated from *Trypanosoma brucei* by adaptation to a non cyclical mode of transmission and loss of ability to undergo growth and differentiation in the fly vector (LUCKINS, 1998). Camels that came into contact with tsetse flies acquired infections, and when such camels moved to non-tsetse areas, transmission was spread by other haematophagous flies. Other species of trypanosomes, e.g. *Trypanosoma congolense*, *Trypanosoma brucei* and *Trypanosoma vivax* have also been isolated from camels in Sudan, but their role in camel trypanosomosis is insignificant (MAHMOUD and GRAY, 1980; ELAMIN et al., 1999).

The disease was further disseminated by camel caravans travelling to North Africa, the Middle East and further east into South Asia. In a similar manner, horses were probably the means by which surra reached America, principally by movement of the animals from West Africa in the 16th century. Evidence from isoenzyme studies and characterization of nuclear and kinetoplast DNA support this hypothesis and suggest only a limited evolutionary origin of *Trypanosoma evansi* (GIBSON et al., 1983; SONGA et al., 1990). The most notable molecular difference is that while the kinetoplast DNA (KDNA) of *Trypanosoma evansi* lacks maxicircle DNAs and possess only interlocked 1-kb minicircle DNAs, the KDNA networks of *Trypanosoma brucei* has both.

In addition to the name surra, other names such as 'murrina', 'mal de caderas' or 'derrengadera' are used to describe similar diseases caused by trypanosomes indistinguishable from *Trypanosoma evansi* in South America. The collective name American surra has since been proposed for adoption in South America due to the need to maintain uniformity in nomenclature (LOSOS, 1980; LUCKINS, 1998).

The vector

Trypanosoma evansi lacks the genes necessary for mitochondrial development (GIBSON et al., 1983; BORST et al., 1987; SONGA et al., 1990) and is therefore unable to undergo growth and differentiation in the insect vector. Nevertheless, this has not precluded

transmission by insects. It is speculated that the widespread occurrence of *Trypanosoma evansi* is largely due to its being spread mechanically by the bites of haematophagous flies, e.g. Tabanus. Stable flies (Stomoxys) have also been incriminated, but based on experimental transmission between horses, guinea pigs and dogs, they do not appear to be important vectors (LOSOS, 1980).

More than 20 different species of Tabanus have been shown experimentally to transmit *Trypanosoma evansi* (LUCKINS, 1998). Furthermore, surveys of Tabanus in the various tropical areas have shown a definite correlation between the seasonal outbreaks of *Trypanosoma evansi* infections and the increase in number of Tabanus during the rains (MAHMOUD and GRAY, 1980; NJIRU et al., 2002). Thus, rainfall, suitable moisture-retaining clay soil and surface water pools also support the development of suitable camel browsing conditions, where Acacia senegal shrubs grow in abundance. The prevalence of some Tabanus spp. all year round ensures that transmission of the parasite occurs wherever reservoir hosts, vectors and susceptible hosts co-exist. This finding may explain the sporadic occurrence of the disease during the dry season and outbreaks during the rainy season (NJIRU et al., 2002). However, the efficiency of the different flies to transmit *Trypanosoma evansi* appears to vary in different geographic conditions and is also dependent on the interval between two successive feeds and intensity of the fly challenge (LUCKINS, 1998).

Although the mode of mechanical transmission is well established, its dynamics are not understood. Therefore, considerable experimentation is still required to attempt to define quantitatively the effect of the host species, the duration of infection in the host and the level of parasitaemia, the period between feeds and the relative efficiency of different vector species in ensuring successful transmission (OBIHIRO, 1998).

Transmission by biting flies is not the sole means by which infection is perpetuated; ingestion of meat from infected carcasses by carnivores can result in infections, and in South America, vampire bats are said to be of importance both as reservoirs of infection and as vectors (LUCKINS, 1998). However, there no definitive study has ever been conducted to confirm their role in the epidemiology of trypanosomosis, and it is therefore not really clear how important they are.

Surra distribution

Surra is widespread in different parts of the world and poses a major constraint to camel productivity (ELAMIN et al., 1999). Available information on the prevalence of surra caused by *Trypanosoma evansi* in many countries of the world as reported, are: Nigeria (27%), Chad (30%) (LOSOS, 1980); Mauritania (24%) (DIA et al., 1997); Niger (29%) (PACHOLEK et al., 2001); Kenya (28%) (NJIRU et al., 2001); Ethiopia (21%) (ZELEKE and BEKELE, 2001); Jordan (33%) (AL-RAWASHDEH et al., 2000); India (22%) (PATHAK et al., 1999); Sudan (33%) (ELAMIN et al., 1999); Iran (10%) (ZARIF-FARD and HASHEMI-

FESHARKI, 2001), and South America (27% in carrier state in capybras) (FRANKE et al., 1994), 35.4 and 43.3%, respectively, in the Tafilalet and Ouarzazate provinces of Morocco (RAMI et al., 2003).

Clinical manifestations

Trypanosoma evansi can infect a variety of hosts and causes a species-specific pathology. The following descriptions are taken from the accounts of MAHMOUD and GRAY (1980) and LUCKINS (1998). In camels the disease is manifested by elevation of body temperature which is directly associated with parasitaemia. Infected animals show progressive anaemia, marked depression, dullness, loss of condition, and often rapid death. Anaemia was observed to be a major clinical finding in camel trypanosomosis in Morocco (RAMI et al., 2003). Milder cases develop recurrent episodes of fever. Some camels develop oedema in their dependent parts of the body, urticaria plaques and petechial haemorrhages in serous membranes. Death finally ensues if untreated. However, some may harbour trypanosomes for 2-3 years thus constituting reservoirs of infection to susceptible camels and hosts. Other well documented field reports are death (TUNTASUVAN et al., 1997); abortion (LOHR et al., 1986); weight loss, reduced draught power (LUCKINS, 1998) and nervous signs like circling movement and trembling, unusual aggressiveness, running aimlessly and sudden collapse in severely stressed and over worked animals (MANUEL, 1998). At post mortem, necrotic foci in the liver and spleen as well as generalised lymphoid tissue hyperplaisia are common in camels suffering from surra (ROTTCHER et al., 1987).

Pathology and pathogenesis

Anaemia is a major component of the pathology of surra and of African trypanosomosis generally. Anaemia in *Trypanosoma evansi* infections of camels is reportedly macrocytic and hypochromic (JATKAR and PUROHIT, 1971). In the early phases of infection the anaemia is haemolytic and haemophagocytic. The mechanism(s) responsible for this increased erythrophagocytic activity are not fully understood. Several have been proposed, viz, immune complexes, expanded mononuclear phagocytic system per se, haemolytic factor produced by the trypanosome, fever and disseminated intravascular coagulation (Food Agricultural Organization, 1979). In the late stages, anaemia continues to be a major factor, with probably additional causes. However, irrespective of the cause of anaemia the primary abnormality of function are the anoxic conditions created by the persistent anaemia. Following this are signs of dysfunction which appear in the various organs. An increase in cardiac output due to increases in stroke volume and heart rate and a decrease in circulation time are obvious manifestations. The central nervous system is reported to be most susceptible to anoxia with consequent development of cerebral anoxia. The marked depression observed in camel trypanosomosis is a mental state and is a manifestation of

depression of cerebral cortical function in various degrees. Other nervous signs reported, such as circling movement, incoordination and dullness, appear to be the results of brain tissue disturbance or damage by the parasites. Evidence of *Trypanosoma evansi* being found in the cerebrospinal fluid has been presented (ROTTCHER et al., 1987).

Tissue damage

The atypical lesions of multiple necrotic foci found in the liver and spleen, as well as generalized lymphoid tissue hyperplasia in camels suffering from surra on post mortem, could be attributed to pathological events that occur in the tissues of animals infected with *Trypanosoma evansi*. The degenerative changes thus observed could be due to tissue anoxia, possibly caused by anaemia, which results in a fall in tissue pH and vascular damage. Other mechanisms may also be involved.

It is known that *Trypanosoma evansi* is a member of the Brucei group of trypanosomes, which have a known preference for connective tissues of a host, where they disrupt the collagen bundles and destroy the fibroblasts which produce and maintain the collagen (BOID, 1980).This disruption of host connective tissues, along with the vascular damage attributable to brucei group trypanosomes (BOID, 1980), would be expected to release large quantities of cytoplasmic and mitochondrial enzymes into the serum, thereby causing further tissue damage.

Indeed, a two-step process in the pathology of infection with *Trypanosoma evansi* in camels based on studies of changes in serum enzymes has been proposed (BOID, 1980). The first step coincides with the appearance of trypanosomes in the host bloodstream and is characterized by a sharp and as yet unexplained rise in sorbitol dehydrogenase (SDH) activity. The second step occurs later in the infection and is characterized by a large increase in serum levels of glutamic oxaloacetic transaminase (GOT) now known as aspartate alanine transferase (AST) (DE LA RUE et al., 1997) and a smaller rise in glutamic pyruvic transaminase (GPT), now known as alanine amine transferase (ALT) (DE LA RUE et al., 1997). The rise in AST level can be attributed partly to cellular damage caused by the trypanosomes lysis, while the increase in ALT probably results from host destruction of trypanosomes. AST is found mostly in cell organelles and rises when there is a great damage to the heart, kidney, skeletal muscles and liver. ALT is a specific liver enzyme found in the cell cytoplasm and its rise is associated with cell membrane damage. The reported increases in these enzymes, especially AST, is not surprising as it is indicative of organ damage and supports the post mortem reports of necrotic foci in the liver and spleen of camels suffering from surra.

The fever characterized by high temperature might be due to the effects of toxic metabolites produced by dying trypanosomes (TIZARD et al., 1978). In addition, the oedema reported in the dependant parts of the body during the chronic stage could be due

to a significant decrease in the albumin levels, resulting in alterations in osmotic pressure of the blood. This leads to excessive accumulation of fluid in tissue spaces caused by a disturbance in the mechanism of fluid interchange between capillaries, the tissue spaces and the lymphatic vessels. All this possibly indicates great liver damage (DE LA RUE et al., 1997). The haemorrhage and serous exudates that occurred could be caused by haemolysis involving the expanded mononuclear phagocytic system. This has also been observed in Trypanosoma brucei-infected donkeys, while the frequent abortions reported may be attributed to endocrine dysfunction (LOSOS, 1980).

Immune response

Pronounced immune changes occur in African trypanosomosis. An increase in gamma-globulin (IgM) during both acute and chronic *Trypanosoma evansi* infections in camels has been reported (BOID et al., 1981) but this is not protective, as the majority of the antibodies are auto antibodies. Leucocytosis, neutrophilia and eosnophilia have been reported in *Trypanosoma evansi* infections of camels (ANOSA, 1988). These changes occur as a result of an increase in the activity of the mononuclear phagocytic system. The eosinophilia observed is a feature of parasitic infections and is associated with immediate-type hypersensitivity reactions. The cells are expected to accumulate in tissue in response to tissue injury. In the acute phase of the disease, lymph nodes and spleen are remarkably reactive, with plasma cells predominating. This may account for the generalized lymphoid tissue hyperplasia characteristic of *Trypanosoma evansi* infections, while in the late stages the immune system becomes depleted of lymphoid cells (LOSOS, 1980).

Circulating and tissue-mediated immune complexes have been demonstrated in laboratory animals infected with *Trypanosoma brucei* species, and much of the antibody found in them is directed against the trypanosome (Food Agricultural Organisation, 1979). Complement has also been found in association with them. These immune complexes are likely to have wide varying pathological effects including anaemia, complement activation, tissue damage and interference with both induction and effector mechanisms of the immune response (Food Agricultural Organisation, 1979).

Hypocomplementaemia (decreased alteration of the complement system) has been reported, including elevated levels of immunoconglutinin and deposition of complement in the tissues. Further possible evidence of complement reactivity is found in the demonstration that the kinin system becomes activated with the release of pharmacologically active substances, thus causing microvascular dilatation and increased permeability (Food Agricultural Organisation, 1979).

A state of immunosuppression later develops associated with these changes. The host immune response to a variety of antigens has been found depressed in animals under experimental conditions (BALTZ et al., 1981; ANENE et al., 1989; ENWEZOR and

EKEJINDU, 1998). Several hypotheses have been put forward to explain trypanosome-induced immunosuppression, and the most favoured appeared to be the action of trypanosome enzymes. Trypanosome enzymes, such as phospholipases (TIZARD et al., 1978), neuraminidases (ESIEVO, 1983) and proteases (LONSDALE-ECCLES and GRAB, 1986) have all been implicated in membrane fluidity and cellular damage. Moreover, the phospholipases generate free fatty acids (FFAs) and these have been reported to not only have a haemolytic effect, thus contributing to anaemia, but also to control lymphocyte reactivity through their role as prostagladin precursors. The net effect of immunosuppression is lack of pre-immunity to other diseases. Little wonder then that in most cases secondary infections, e.g. bacteria bronchopneumonia, often sets in and death may ensue if untreated.

Diagnosis

There are no pathognomonic signs of surra and so laboratory diagnosis has to be carried out to confirm infection. Traditionally, this involves parasitological and serological diagnosis. Parasitological diagnosis is mainly carried out by the direct microscopic examination of blood or buffy coats and/or sub-inoculation of camel blood into rodents such as mice or rats. However, the test has a poor sensitivity, often less than 50% (MONZON, 1990; NANTULYA et al., 1989; NANTULYA, 1990; LUCKINS, 1992; YADVENDRA et al., 1998). The implication of this is that in most situations *Trypanosoma evansi* is under-diagnosed and the level of infection is greater than frequently reported. On the other hand, serological techniques, e.g. immunfluorescent antibody test (IFAT), enzyme Linked Immunosorbent Assay (ELISA) and the Card Agglutination Test for Trypanosomosis (CATT), although sensitive, cannot distinguish current from cured infections (LUCKINS, 1988).

Recent tests, e.g. latex agglutination test (LAT) or Surratex based on trypanosome-antigen detection in blood or serum, are more reliable and have shown a high correlation with patent or sub-patent disease in camels (OLAHO-MUKANI et al., 1996). A comparative sensitivity test for *T. evansi* in camels in Kenya revealed 68.8% sensitivity for CATT / *T. evansi* and 58.8% for SURATEX, although the two techniques showed no significant difference (NGAIRA et al., 2003). Similarly, at prevalence values between 10 and 100%, CATT / *T. evansi*, as well as SURATEX, had infinitely high positive predictive values, whereas SURATES had a lower NPV than CATT / *T. evansi* and the two techniques were more sensitive than parasitological methods in revealing the true extent of trypanosomosis in camel herds (NGAIRA et al., 2003). Similarly, ATARHOUCH et al. (2003) diagnosed a prevalence of 14.1% via CATT and 18.2% via ab-ELISA in provinces located in the South and East of the Atlas mountain chain in Morocco. Also, DELAFASSE and DOUTOUIN (2004) using Buffy coat Technique (BCT) and CATT revealed a prevalence rate of *T. evansi* of 5.3 and 30.5%, respectively, in Chad. However, non-validation, standardization, application and deployment are factors militating against their use in the field.

Treatment, prevention and control

Treatment with trypanocidal drugs is the usual method of control of *Trypanosoma evansi* and quinapyramine has been used in camels, and only recently melarsomine (cymelarsen) (Rhone Merieux, France) was introduced for the treatment of surra in camels because of the problem of drug resistance (LUCKINS, 1998; BOURDICHON, 1998). Treatment of *T. evansi* infected camels in Morocco with melarsomine (Cymelarsan (R). Rhonemerieux) reduced the sero prevalence level from 58 to 19% within a year (RAMI et al., 2003) Resistance up to 500 ng/ml has been found in camels against quinapyramine sulphate (BOURDICHON, 1998). Cymelarsen is also effective against *T. evansi* infections in cattle and horses (RAYNAUD et al., 1989) and animals with surra are commonly treated at different stages of the disease. However, relapses in camels after treatment have been reported (OTSYULA et al., 1992).

Another drug, Trypan, which is a formulation containing diminazene-di-aceturate (diamidinophenyltriazene diaceturate tetrahydrate), phenazone and procaine hydrochloride is effective against *T. evansi* infections, as well as infections with *Trypanosoma Congolese*, *Trypanosoma vivax*, and *Trypanosoma brucei*. The drug is also effective against *Babesia bigemina*, *Babesia canis* or other Babesia and *Theileria annulata* (BOURDICHON, 1998). It has a synergistic and an additive effect in comparison with other trypanocidal drugs and is reported to have a painless, antipyretic and long-lasting effect. It has also been adjudged as being the most effective trypanocidal drug to date (BOURDICHON, 1998).

With regard to prevention, it has been confirmed that a single injection of 15 ml of Trypan affords an animal protection against a new re-infestation over a period of three months (BOURDICHON, 1998). Trypan can be used for curative and preventive treatment. On the whole, control of surra requires treatment of infected animals with effective drugs and reducing blood sucking flies by regular insecticide treatment.

Conclusion

Camel trypanosomosis is a disease of major economic importance in many countries in Africa, Asia and South America. The devastating epidemics caused by the disease several years back is not frequently seen nowadays, but they do still occur. Although camel trypanosomosis has been recognized as the most important single cause of economic losses in camel rearing areas, yet there have been no planned campaigns to control *T. evansi* using modern methods of fly control or chemotherapy (LUCKINS, 1998). In most cases, control is limited to treating those animals that are considered to be infected on the basis of unreliable clinical signs.

Therefore, a number of research issues need to be considered:

1. Application of validated, standardized diagnostic tests (particularly penside tests) at both local and regional levels
2. Identification of the principal vector species responsible for transmission in different ecological situations
3. Improving understanding of the dynamics of mechanical transmission in endemic and epidemic situations
4. Investigating the potential for vector control in the management of this disease
5. Determining the efficacy of different trypanocidal drugs and the extent of drug resistance, as well as ascertaining the causes of relapses of cymelarsin in the chemotherapy of camel trypanosomosis
6. Determining which factors that precipitate epidemic outbreaks of disease
7. Determining the role of wild animals in the epidemiology of camel trypanosomosis.

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SAŽETAK

Tripanosomoza deva (Surra) uzrokovana vrstom *Trypanosoma evansi* najvažniji je uzrok pobola i smrtnosti deva. Bolest neciklički prenose hematofagni insekti (npr. Tabanus sp.), a endemska je u Africi, Aziji i Južnoj Americi. Osim deva ugrožene su i druge vrste domaćih životinja. Zbog velike zemljopisne proširenosti, kontrola bolesti privukla je međunarodnu pozornost s težištem na oblikovanju i uvođenju djelotvornih strategija u svrhu povećanja proizvodnosti te smanjenja pobola i smrtnosti. U radu su opisani kliničko-patološki nalazi bolesti s ciljem njezina djelotvornog suzbijanja. Glavni znak bolesti je anemija. Razvoj i trajanje anemije tijekom bolesti dovodi do anoksije što se očituje znakovima poremećene funkcije različitih organa kao rezultat pada pH u tkivu i oštećenja krvnih žila. Zatim slijedi oslobađanje velikih količina citoplazmatskih i mitohondrijskih enzima, naročito aspartat aminotransferaze (AST) i alanin aminotransferaze (ALT) u serum, uzrokujući daljnje stanično i tkivno oštećenje. Usporedno s ovim promjenama dolazi i do imunosupresije koja povećava osjetljivost životinja za druge infekcije i dovodi do smrti ako se ne liječi. Kontrola bolesti temelji se na njezinoj točnoj dijagnozi, liječenju djelotvornim tripanocidnim lijekovima kao što je tripan i primjeni metoda suzbijanja vektora.

Ključne riječi: tripanosomoza, surra, *Tripanosoma evansi*, Tabanus, anoksija, imunosupresija