















ASSESSMENT OF HISTOPATHOLOGY IN RABBITS EXPERIMENTALLY INFECTED WITH *TRYPANOSOMA EVANSI*

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Abstract

Trypanosoma evansi is a vector-borne protozoan parasite responsible for surra, a disease that affects a wide range of domestic and wild animals, causing severe health consequences and significant economic losses. This study aimed to investigate the histopathological effects of *T. evansi* infection in rabbits under controlled experimental conditions and assess the potential of rabbits as a suitable model for studying this disease. Sixteen rabbits were divided into infected and control groups. The infected group received an inoculation of *T. evansi* isolated from cattle. Clinical signs were monitored, and tissue samples from the liver, lungs, spleen, myocardium, and kidneys were collected for histopathological evaluation. Microscopic analysis revealed marked pathological changes in infected animals, including granular and fatty degeneration of hepatocytes, hyperemia, emphysema and exudative inflammation in the lungs, myocardial dystrophy, spleen hyperplasia, and degenerative changes in renal tissue. These alterations reflect systemic involvement, indicating the pathogenicity of *T. evansi* and its ability to affect multiple organ systems. The experimental model reproduced classical features of the disease, such as anemia, tissue edema, and hemorrhages, supporting the relevance of rabbits as a viable model for trypanosomiasis research. This study contributes to a better understanding of host-pathogen interactions and pathogenesis in surra. The results offer valuable insights for the development of diagnostic tools, treatment strategies, and vaccine research. Moreover, the established rabbit model may facilitate future investigations into the biology and virulence of *T. evansi* strains circulating in Kazakhstan and beyond.

Keywords: Trypanosomosis, *Trypanosoma evansi*, rabbits, histopathology.

Introduction

Trypanosomes are unicellular flagellated protozoa belonging to the family *Trypanosomatidae* and the genus *Trypanosoma* (Sobhy et al., 2017). The genus *Trypanosoma* comprises many species that cause diseases known as trypanosomoses in domestic and wild animals, as well as in humans (Mekata et al., 2013). Livestock trypanosomoses, caused by *Trypanosoma brucei*, *T. equiperdum*, and *T. evansi*, all of which belong to the subgenus *Trypanozoon*, have a significant socio-economic impact and limit animal productivity worldwide (Desquesnes et al., 2013).

Trypanosoma evansi is a protozoan parasite responsible for the disease known as surra, which affects various domestic and wild mammals. Discovered in 1880 by British veterinarian Griffith H. Evans in India, *T. evansi* was initially observed in horses suffering from surra. The parasite is characterized by a single flagellum running along its undulating membrane, facilitating movement. Unlike other trypanosomes transmitted by tsetse flies, *T. evansi* is primarily spread mechanically by blood-feeding insects such as horseflies (*Tabanidae*) and stable flies (*Stomoxys*). The parasite infects a wide range of hosts, including horses, camels, cattle, buffaloes, deer, elephants, and tigers. In South America, vampire bats and capybaras serve as reservoirs, contributing to the parasite's persistence in the environment (Sazmand & Joachim, 2017).

Trypanosoma evansi is mechanically transmitted by hematophagous insects (*Tabanidae* and *Stomoxys*)

and affects several tropical regions worldwide. This parasite causes the disease commonly known as 'Mal de Cadeiras' in Brazil and 'Derrengadera' in Venezuela (Desquesnes et al., 2013).

Although *T. evansi* is considered a hemoflagellate, it is a protozoan parasite that inhabits both intra- and extravascular fluids. One of the most intriguing aspects of *T. evansi* is its ability to periodically switch its major variant surface glycoprotein (VSG), leading to recurrent waves of parasitemia. These parasitemic waves have been observed in experimental infections with Brazilian *T. evansi* isolates (Aquino et al., 1999; Queiroz et al., 2000; Herrera et al., 2001).

The severity of *T. evansi* infections varies considerably across different geographical regions, depending on the virulence of the strain and the susceptibility of the host. Anemia is the primary consequence of infection. Consequently, it has been suggested that resistance to anemia, as well as the control of parasitemia, reflects the degree of host tolerance to infection (Trail et al., 1990). Affected animals may exhibit fever, general deterioration of health, and immunosuppression (Holland et al., 2001). Diagnosing *T. evansi* infection remains challenging due to the varied and non-specific clinical signs. In enzootic areas, natural hosts often present mild, chronic forms of the disease, further complicating diagnosis (Roberto et al., 1994).

Trypanosomosis in domestic animals is known to cause various pathological changes in vital visceral and reproductive organs. This study aimed to assess

the pathological changes in visceral organs of rabbits experimentally inoculated with *Trypanosoma evansi*.

Materials and Methods

Trypanosoma evansi isolate

For this research, a *T. evansi* isolate of cattle origin was propagated in guinea pigs at the laboratory of LLP 'Kazakh Scientific Research Veterinary Institute'.

Animal Studies

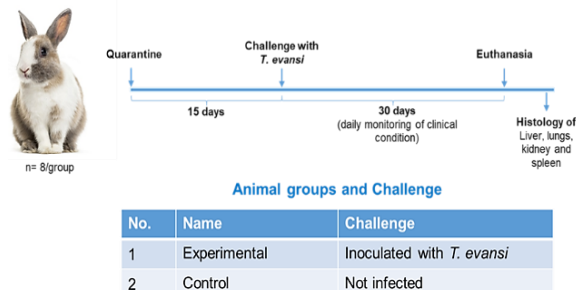
The study involved 16 clinically healthy adult rabbits (average weight 2–2.5 kg), which were randomly assigned into two groups (n = 8 per group):

- Group I (Experimental group): Rabbits were inoculated with *Trypanosoma evansi* on day 0.
- Group II (Control group): Rabbits were not infected. Before the experiment, all animals underwent a 15-day quarantine period to ensure they were free from clinical signs of disease. After challenge with *T. evansi*, rabbits in the experimental group were observed for 30 days, with daily monitoring of clinical condition. On day 30 post-challenge, all animals were humanely euthanized for sample collection and pathological assessment.

The experimental timeline and group distribution are shown in Figure 1.

Figure 1

Experimental design and animal grouping



Histology Tests

The rabbits were euthanized by intraperitoneal administration of 10% chloral hydrate (Acros Organics, Geel, Belgium). Following necropsy, a macroscopic examination of internal organs was performed to assess the presence of lesions. Whole lungs, liver, kidneys, and spleen were fixed in 10% neutral formalin (Sigma, Taufkirchen, Germany) for histological examination (Figure 1). Tissue sections were prepared and stained with hematoxylin and eosin (Sigma, Taufkirchen, Germany).

Ethics statement

The study was conducted by national and international animal handling laws and recommendations. The study protocol was approved by the Local Bioethics

Commission of Kazakh Scientific Research Veterinary Institute LLP (Protocol №10, dated 28 September 2024).

Results and Discussion

Histological examination results

Liver. In most cases, hepatic lobules exhibited granular dystrophy (Figure 2a) at varying degrees of severity. The affected hepatocytes were enlarged and rounded, with poorly defined lobular boundaries. Eosinophilic granules were observed in the cytoplasm. The nuclei of these hepatocytes displayed morphological changes, including karyopyknosis and karyolysis.

In rare cases, granular and fatty dystrophies co-occurred in the middle region of the hepatic septa (Figure 2b). In some hepatocytes, nuclear dispersion and dissolution were observed. Blood congestion was noted in the cavities of the septa, central veins, and sinusoidal capillaries. The endothelial layers of many vessels appeared enlarged and swollen. In certain areas, the structural arrangement of hepatic cords was disrupted, particularly around the central vein Figure 2c.

Figure 2a

Granular liver dystrophy. Hematoxylin-eosin staining

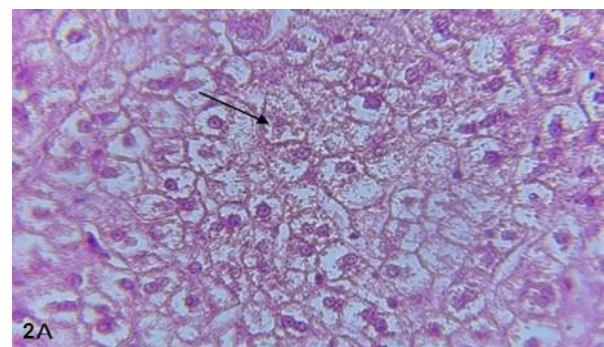


Figure 2b

Granular (a) and fatty (b) liver dystrophy. Hematoxylin-eosin staining

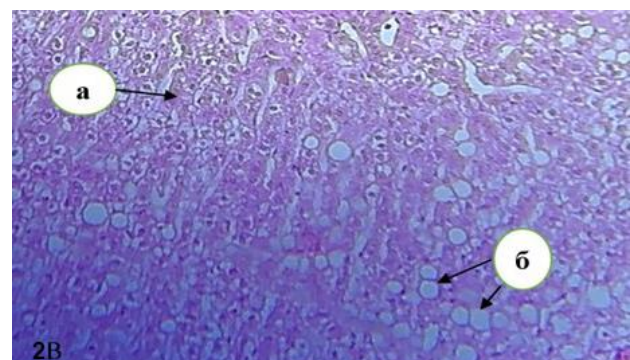
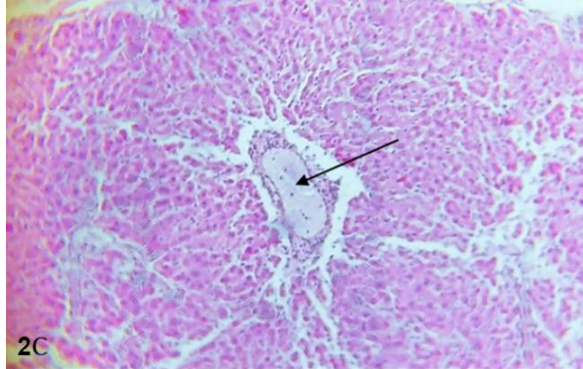


Figure 2c

Liver hyperemia. Hematoxylin-eosin staining



Lungs. Microscopic changes in the lungs were heterogeneous, with varying degrees of hyperemia, edema, emphysema, and atelectasis, which often overlapped. In large portions of the lung tissue, the capillary cavities within the interalveolar connective tissue were dilated and congested with blood, while the alveolar spaces contained eosinophilic fluid, which included erythrocytes, leukocytes, and alveocytes. Serous exudate accumulated around some bronchi. In certain areas, characteristic features of emphysema were observed (Figure 3a).

In emphysematous regions, alveolar cavities appeared expanded, with thinned and ruptured walls. The alveolar epithelium was stretched and attenuated, and blood vessels within the alveolar walls exhibited hemorrhagic changes. Focal areas of distal atelectasis and interalveolar septal thickening, due to cellular infiltration, were present against the background of acute alveolar emphysema. The blood vessels were moderately engorged, and in some areas, vascular walls were thickened with surrounding cellular infiltration. In certain vessels, plasma and cellular elements of the blood were separated. Additionally, many large and medium-sized bronchi appeared dilated without signs of inflammation or sclerosis (Figure 3b).

Figure 3a

Pulmonary emphysema. Hematoxylin-eosin staining

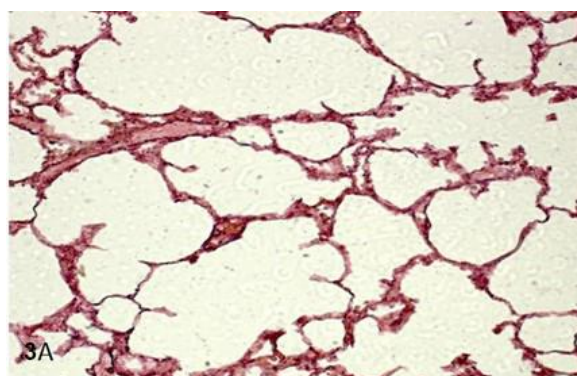
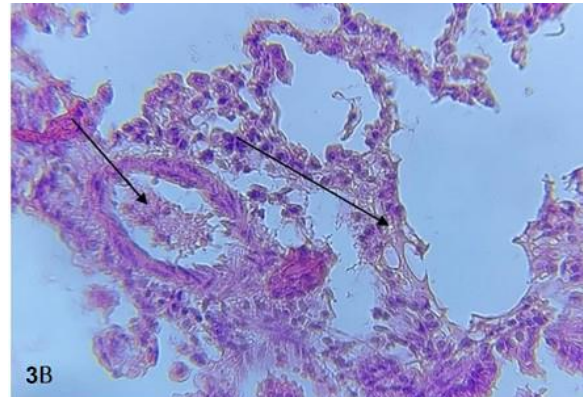


Figure 3b

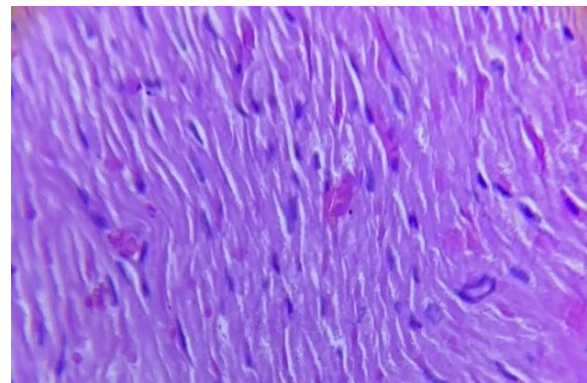
Exudative inflammation of the lungs. Pulmonary vessels filled with blood. Hematoxylin-eosin staining



Myocardium. Numerous muscle fibers appeared swollen, with some exhibiting indistinct cross-striations and granular material accumulation in the cytoplasm. The nuclei were enlarged, intensely stained with hematoxylin, and displayed peripheral convexity. Between the muscle fibers, areas of swelling, occasional hemorrhagic foci, and scattered reticular infiltrates were observed. The endothelial cells of the vessel walls were enlarged and swollen. In rare cases, focal fragmentation of muscle fibers was noted (Figure 4).

Figure 4

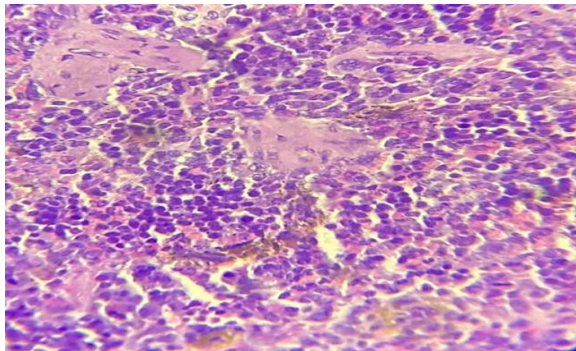
Myocardial dystrophy. Hematoxylin-eosin staining



Spleen. In all cases, the splenic pulp was congested with blood, with dilated vascular cavities. In some capillaries, erythrocytes were densely packed, forming a homogeneous mass. The follicles remained intact and in a normal state. Some trabeculae appeared swollen and thickened (Figure 5).

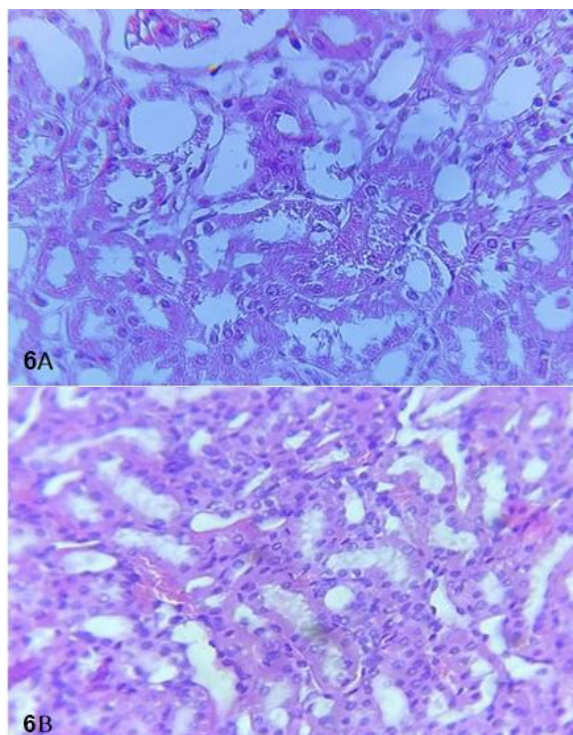
Kidneys. The vascular cavity was dilated and filled with blood, with erythrocytes undergoing hemolysis, resulting in a uniform size and the formation of a homogeneous mass. Some glomerular vessels were enlarged, the capillaries were engorged with blood, and a yellowish watery exudate accumulated in the capsule cavity.

Figure 5
Spleen hyperplasia. Hematoxylin-eosin staining



The nephrocytes lining the convoluted tubule walls appeared swollen, with well-defined boundaries. The cytoplasm was darkened and contained small eosinophilic granules stained red. The tubular lumen was narrowed, and in some cases, nephrocytes detached from the basement membrane, floating freely within the tubule lumen. The nuclei of nephrocytes exhibited karyopyknosis and karyolysis. In the juxtamedullary region of the kidneys, small foci of hemorrhage were frequently observed. The nuclei of some cuboidal epithelial cells lining the vertically oriented tubules were in a pyknotic state (Figure 6).

Figure 6
A - Granular and fatty degeneration of the kidney
B - Hematoxylin and eosin staining



Invasive diseases of animals, particularly trypanosomiasis (surra and dourine), are characterized by debilitating symptoms such as intermittent fever, anemia, jaundice, hemoglobinuria, dysfunctions of the digestive, cardiovascular, nervous, and motor systems, paresis and paralysis of individual motor nerves, decreased productivity, severe emaciation leading to cachexia, and a high mortality rate. The prevalence of trypanosomiasis ranges from 15% to 50% (Sabanshiev, 1993; Shabdarbayeva et al., 2014).

Trypanosomes are a distinct group of unicellular organisms, first discovered by Gruby in the blood of frogs. In 1880, the English veterinary surgeon Evans established that the causative agent of a widespread animal disease in India at the time, known as 'surra', was a trypanosome, later named *Trypanosoma evansi*. In 1894, Bruce demonstrated that the causative agent of the African animal disease 'nagana' was *Trypanosoma brucei*. Today, numerous species of trypanosomes are known to infect mammals, fish, birds, reptiles, and amphibians.

Among pathogenic trypanosome species, two parasites are found in the Republic of Kazakhstan: *Trypanosoma equiperdum*, the causative agent of dourine in horses, and *Trypanosoma evansi*, the causative agent of surra in animals. This has been confirmed through serological studies of camel and horse blood sera across the country (Sabanshiev, 1993; Shabdarbayeva et al., 2014). The urgency of combating trypanosomiasis is due to the fact that this infection hinders the development of horse and camel breeding in Kazakhstan. As a vast Central Asian country with a long-standing tradition of using horses and camels for agriculture, transportation, food production, and cultural identity, where these practices are deeply embedded in the historical, cultural, and economic landscape, trypanosomiasis remains a significant problem.

In animals infected with trypanosomiasis (surra and dourine), symptoms include emaciation, anemia, remittent fever, edema in the chest area, limbs, and genital organs, as well as watery discharge from the eyes and nose. The disease lasts from 1.5 to 2 months, sometimes extending to 4–6 months, with some animals succumbing to the infection. Post-mortem examination reveals anemia of the mucous membranes, gelatinous infiltration of subcutaneous tissue, hemorrhages on serous and mucous membranes, in the kidneys, and in the urinary bladder, along with significant enlargement of lymph nodes and the spleen. Diagnosis is based on detecting the pathogen through microscopy of a crushed drop of peripheral blood or a lymph node aspirate. If initial results are negative, a bioassay is conducted using mice, rats, rabbits, guinea pigs, or dogs. In suspected cases, the blood serum of animals is tested using the complement fixation test (CFT) and X-ray fluorescence analysis (XRF).

Trypanosoma evansi, the causative agent of surra in camels, is also the etiological agent of dourine, which primarily affects horses (Gizaw, 2017). Unlike other trypanosomal infections, equine trypanosomiasis is not transmitted by insect vectors but exclusively by the sexual route (Desquesnes, 2013). Among pathogenic trypanosomes, *Trypanosoma evansi* has the broadest host range and the widest geographic distribution (Radwanska, 2018). *Trypanosoma evansi* is observed in domestic and wild animals in acute, subacute, and chronic forms, depending on the virulence of the strain and host-parasite interactions. Susceptible hosts include horses, camels, donkeys, mules, llamas, dogs, cats, cattle, and buffaloes (Desquesnes et al., 2016). In laboratory conditions, experimental infection has been successfully induced in dogs, rabbits, rats, and mice (Perrone et al., 2018). Biswas et al. (2001) described *Trypanosoma evansi* as highly pathogenic to laboratory animals (mice, rats, and rabbits). The parasite utilizes glucose and oxygen for its growth and reproduction, leading to the depletion of these metabolites, which causes degenerative changes in the host. Further disease progression is associated either with toxins released by the parasite or with immunological reactions (Bal et al., 2012).

Our histological studies revealed hyperemia, edema, emphysema, and varying degrees of atelectasis in the lungs of infected rabbits, findings similar to those reported by Chandra et al. (1999) and Ngeranwa et al. (1993). In contrast, our histological examination of the liver in infected rabbits showed that affected hepatocytes were enlarged, had a rounded shape, and exhibited poorly defined lobular boundaries. Studies by Uche and Jones (1992) identified fatty degeneration followed by hepatocyte necrosis as a common cytological finding associated with *T. evansi* infection in rabbits (Uche & Jones, 1992).

Histopathological lesions caused by trypanosomiasis were also studied in the myocardium, spleen, and kidneys of infected rabbits, where varying degrees of dystrophy were observed. Although the pathology of the disease caused by different species of *Trypanosoma* in domestic and laboratory animals is well known, the knowledge gained from studying the histopathology of rabbits experimentally infected with animal trypanosomiasis could serve as a starting point for identifying key features of the infection's progression and the properties of the isolate found in our country. Additionally, these findings could be valuable in testing new drugs. The acquired

knowledge will expand research capabilities to counteract emerging *Trypanosoma* isolates, making it a significant step toward future advancements.

Conclusions

Based on the results of this study, the following key conclusions can be drawn:

1. Rabbits are a suitable experimental model for studying animal trypanosomiasis caused by *Trypanosoma evansi*. The infection reliably reproduces the clinical and pathological features of the disease observed in natural hosts.
2. Histopathological examination revealed characteristic lesions in multiple organs, including granular and fatty degeneration of hepatocytes in the liver, emphysema and exudative inflammation in the lungs, myocardial dystrophy, hyperplasia of the spleen, and degenerative changes in the kidneys. These findings confirm the systemic nature of the infection.
3. The experimental infection model allows detailed observation of disease progression, tissue damage, and organ involvement, thereby providing a basis for evaluating the pathogenicity of different *T. evansi* strains.
4. The data support the use of rabbits in biological testing, especially in cases where traditional microscopic diagnostics yield inconclusive results despite the presence of clinical signs.
5. This research contributes to the broader understanding of trypanosomiasis pathogenesis and highlights the need for further studies to explore therapeutic and preventive strategies, including drug efficacy trials and vaccine development.

The findings are particularly relevant for Kazakhstan, where *T. evansi* is endemic and threatens the productivity and health of horses, camels, and other livestock species.

In summary, this study not only confirms the pathological potential of *T. evansi* in a controlled setting but also establishes a practical and reproducible platform for future research on surra and related trypanosomal diseases.

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