

құрылымына, лактация айына байланысты маститпен аурушандықты анықтау. Зерттеулер 2023 жылдың сәуірінен желтоқсанына дейін жүргізілді.

Көктемде 90 ірі қара мал тексерілді, оның ішінде 14 ірі қара мал немесе 16 %-ға жуығы ауырғандығы анықталды, ауырғандардың жалпы саны 25,5 пайыз немесе 4 ірі қара мал деп есептелген. Жазда маститке тексерілген ірі қара мал саны 88 басты құрады және оның 6 ауырған, бұны пайыз бойынша есептейтін болсақ, 6,8 % құрайды. Осылардың ішінде бір бас маститтің клиникалық түрімен ауырған. Күз кезеңінде 87 бас маститке тексерілді, 3 бас ірі қара мал ауырғаны анықталды. Бұл пайыз көрсеткіші бойынша 3,4 % құрайды және клиникалық түрі анықталмады. Қыста, күз мезгіліндегідей, мастит ауруы салыстырмалы түрде төмен 2,3 % құрайды, ал көктеммен 13 пайызға аз. Барлық кезеңде сүт безінің ауруына 349 бас тексерілді және оның 25 басы мастит болып шықты және 5 бас аурудың клиникалық түрін жұқтырғандығы анықталды. Тостаған тәрізді сиырлардың саны – 113 бас, оның 13,2 %-ы ауруға шалдыққан, ешкі бойынша 20 бастың ішінде 4-і маститке сезімтал, яғни 20 % құрайды.

Лактацияның екінші айында сүт безі ауруына тексерілген 25 сиырдың 12-сі немесе 48 %-ы ауру болып шықты, оның 4-і маститтің клиникалық түрі, бұл барлық ауырғандардың 16 % құрады. Лактацияның төртінші айында ауырғандардың саны 4 басқа азайып, зерттелген жануарлардың жалпы санының 32 % құрады және маститтің клиникалық түрімен тек 4 % анықталды. Аурудың төмендеуі лактацияның 6-шы және 8-ші айларында болады.

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## DYSTROPHIC PROCESSES IN ORGANS AND TISSUES OF SAIGA CALVES IN CASE OF EXHAUSTION

### ANNOTATION

This paper describes the pathanatomical picture in organs and tissues of emaciated saiga calves that fell during mass calving of their mothers in Janybek district, West Kazakhstan region.

Pathanatomical changes in emaciated saigas were characteristic of alimentary dystrophy (emaciation, cachexia), accompanied by various metabolic disorders, enzymopathy, the development of protein dystrophy, and atrophic processes in organs. Young saigas developed progressive emaciation, dehydration, and a lack of fat in the subcutaneous tissue, pericardium, and mesentery. Visible mucous and serous membranes were dry and anemic. Curvature of the spine in the lumbar region, rickets, and damage to deciduous teeth were detected. Atrophic processes were found in all internal organs: liver, kidneys, and spleen. The color varied from light red to brown.

The liver is reduced in volume and dark brown in color; the edges of the liver are pointed; the consistency is flabby; and brown atrophy of the liver is observed. Spleen reduced in size, color purple-reddish, capsule shriveled, soft consistency on section, scraping is insignificant. Whitish overgrowths of connective tissue are seen under the capsule; the pericardium lacks fat; the coronary vessels are translucent; and the myocardium is flabby and light brown in color. There is no peri-renal fat in the

kidney, the kidneys are reduced in size and flabby, the capsule is easily removed, and the color of the kidneys is light brownish. On section, the boundaries of the organ are smoothed, the cortical layer is thin, and the renal vessels are empty.

In saigas under one year of age, nematodes were found in the gastrointestinal tract in isolated numbers, and in other young animals, the lungs were inflamed with severe catarrhal inflammation and bronchopneumonia due to decreased general body resistance. In severe pneumonia, the tissue consistency of the lung is compacted, the pleura is tense, and turbid exudate flows from the cut surface. In catarrhal bronchopneumonia, the lung areas are red and thickened, with cloudy fluid flowing from the incision surface and a thick, mucus-like exudate from the bronchi.

In one saiga head, a brain autopsy revealed a diagnosis of cerebral cenurosis, with single, oval, and elongated cenuroses up to 3–4 cm in length in the left hemisphere, injection of blood capillaries in the brain, atrophied connective tissue in the areas where the parasite was found, and congestion in the bottom of the eye.

**Key words:** *saiga; dystrophy; organs; tissues; West Kazakhstan region*

**Introduction.** The family Bovidae and subfamily Antilopinae contain the genus Saiga (Gray, 1843) [1]. This antelope is distinguished by two enlarged, closely spaced nostrils that point downward and create a short proboscis [2,3]. Males also have prominently ringed, robust horns that are slightly twisted. Saiga is an Ice Age remnant that thrives in temperate to cold climates. It was widely spread in the Pleistocene from Western Europe across central and northern Eurasia up to North America, although it is currently limited to a few isolated locations in Central Asia [4, 5]. Millions of saiga herds once roamed the earth, but because of ongoing population declines, the species is now listed as critically endangered [6,7,8]. According to Kahlke [5] and Sher [9], the Olyorian Complex in Yakutia is home to the earliest known occurrence of Saiga, which dates from the late Early to early Middle Pleistocene (1.2–0.6 Ma). This animal spread its range during the Middle Pleistocene, initially inhabiting Western Siberia and Europe [4,5]. During the Last Glacial era (MIS 5d–MIS 2, 115–12 ka), the saiga attained its maximum distribution. This was particularly true at the conclusion of the period (MIS 3 and MIS 2), when its geographic range extended longitudinally from the Pyrenees to Alaska and northwest Canada [10-16].

Saiga's distinction resulted from their extensive dispersion throughout space and time. Numerous writers identified various physical forms and purposefully gave them the status of species or subspecies. The majority of current saiga populations are categorized as belonging to the species *Saiga tatarica*, although there are two subspecies that have been genetically confirmed: *S. tatarica tatarica* and *S. t. mongolica* [17]. While the latter is native to western Mongolia, the former is found in Kazakhstan and the Republic of Kalmykia [18].

There is a lot more ambiguity about the Pleistocene forms. due to the fact that numerous researchers have identified several Saiga species, including *S. prisca* from Moravia [19] *S. ricei* from Alaska [20], *S. binagadensis* from Azerbaijan [21], and *S. borealis* from Yakutia [22]. Furthermore, numerous writers used various taxonomy classifications for saiga. Its taxonomic status and history are therefore still hotly contested. Sher [9] made the assumption that both the extinct *S. ricei* and the surviving *S. tatarica* existed in an attempt to try and arrange the classification. He said that *S. borealis* was halfway between the other two species, but because it lacked key diagnostic indices and measurements, he was unable to determine its classification. Presently, however, only one species, *S. tatarica*, has been defined by R.D. Kahlke [23], comprising three subspecies: *S. tatarica borealis* from northeastern Siberia, which became extinct at the start of the Holocene, the extant *S. tatarica*, which also includes the fossil saiga from the Crimea and Eastern Europe, and the extant *S. tatarica mongolica*. Scholars once regarded *Saiga prisca* and *S. ricei* as junior synonyms of *S. tatarica borealis*.

The saiga antelope (*Saiga tatarica*) is a nomadic species that inhabits the desert, semi-desert, and steppe ecosystems of Central Asia, particularly in Mongolia and Kazakhstan [24]. The Mongolian saiga, a critically endangered subspecies, has faced significant challenges, including habitat degradation, climate change, and human persecution, leading to a decline in its population [25]. Furthermore, the saiga has been affected by disease outbreaks, such as the Peste des Petits Ruminants virus, which caused a mass mortality event in Eastern Asia, impacting two-thirds of the endangered saiga population [26]. The saiga's migratory behavior makes the protection of its migratory corridors crucial for its

conservation, as these corridors are essential for a large proportion of the population during specific life cycle stages [27, 28].

Efforts to raise public awareness and promote conservation attitudes among local communities have been explored, highlighting the importance of engaging stakeholders in conservation initiatives [29, 30]. Furthermore, the effectiveness of biodiversity-conservation marketing and public awareness campaigns in influencing attitudes and behavioral intentions towards saiga conservation has been evaluated, emphasizing the need for targeted interventions [31, 32]. The saiga's ecological significance as a nomadic herding species in the desert, semi-desert, and steppe ecosystems of Central Asia underscores the importance of understanding and managing its habitat to ensure its survival [33]. Additionally, the genetic diversity of saiga populations has been studied to provide insights into conservation strategies, emphasizing the need for genetic conservation efforts to maintain the species' viability [34]. The saiga's ability to rapidly recover, as evidenced by stabilized or increased populations in certain areas, underscores the potential for successful conservation measures, provided that persistent threats such as disease outbreaks and trade-driven impacts are effectively addressed [35]. Overall, the conservation of the saiga antelope requires a multifaceted approach that addresses ecological, social, and economic factors to ensure the species' long-term survival.

The spread of diseases among saiga populations has been linked to interactions with livestock, highlighting the importance of understanding disease transmission dynamics between wildlife and domestic animals [36]. Furthermore, the illegal trade of saiga antelope horn for traditional medicine poses a significant threat to the species, as it can lead to increased human-wildlife interactions and disease transmission risks [37]. The involvement of multidisciplinary approaches and the One Health framework has been emphasized in managing disease outbreaks affecting saiga populations, as demonstrated by the challenges posed by the Ebola outbreak in West Africa and the saiga antelope die-off in Kazakhstan [38].

Conservation efforts for the saiga antelope must prioritize disease management and surveillance, including seroprevalence studies to understand the prevalence of infectious diseases within saiga populations [39]. Additionally, the eradication of diseases such as PPR through vaccination programs has been identified as a crucial strategy to protect saiga populations from disease outbreaks [40]. Understanding the epidemiology of diseases affecting saigas and implementing measures to mitigate disease transmission risks are essential for the long-term conservation and survival of this endangered species.

**Materials and Methods.** This paper describes the pathanatomical picture in the organs and tissues of emaciated saiga calves, which also died as a result of the mass mortality of the mothers of saigas during the mass calving period in the rainy season. The adult saiga population was diagnosed with infectious enterotoxemia complicated by pasteurellosis. In areas of mass migration of saigas in the Janybek district of West Kazakhstan province.

The study was conducted in the histology laboratory of the Graduate School of Veterinary Clinical Sciences, Institute of Veterinary Medicine and Animal Husbandry, Zhangir Khan West Kazakhstan Agrarian and Technical University (Uralsk, Kazakhstan). A post-mortem examination of fallen saigas was carried out using the Shore method [41].

The collected materials underwent histological examination. The following method was used to prepare a histological slide: tissues obtained from digestive parts were fixed in a 10% formalin solution for 24 hours and subjected to a process of dehydration through different sections, dehydrated with increasing concentrations of ethanol, cleared in xylene, and finally impregnated in paraffin [19, 20]. Blocks were cut with a rotary microtome to 4-5  $\mu\text{m}$  thickness and stained with hematoxylin and eosin dye [42].

**Results and discussion.** During prolonged starvation, animals develop alimentary dystrophy (emaciation, cachexia), a disease accompanied by profound metabolic disorders, enzymopathy, and the development of dystrophic and then atrophic processes leading to the progressive emaciation of animals. The disease usually develops gradually, with the first symptoms appearing 3–4 weeks to 2–3 months after the onset of malnutrition [43]. When an animal does not eat for a long time, it lives only at the expense of its body reserves; glycogen reserves in the liver and muscles, fat reserves in different parts of the body, and subsequently muscle and organ protein are consumed. Loss of 10–20% of body weight fluid is fatal for mammals [44].

Pathological and anatomical changes in emaciated saigas were characterized by dehydration and

a lack of fat in the subcutaneous and retroperitoneal tissues, pericardium, mesentery, and omentum. The mucous and serous membranes were dry and anemic. Curvature of the spine in the lumbar region, fractures, and damage to deciduous teeth were also detected. The color of the internal atrophied organs (liver, kidneys, and spleen) varied from light red to brown. The liver was reduced in volume and dark brown in color; its edges were devoid of parenchyma and sharp; and its consistency was flabby (brown liver atrophy). The spleen was ribbon-shaped, reduced in size, and violet-red in color; the capsule was moderately shriveled and soft on the section; and scraping was scanty. Whitish overgrowths of connective tissue in the trabecular zone were observed under the capsule. Pericardial fat was absent, and coronary vessels were translucent. Myocardium was light brown in color. Perirenal fat was absent. Kidneys were reduced in size, their consistency was more often flabby, the capsule was easily removed, and the color of kidneys varied from light brown to red-brown. On the section, the border between the cortical and cerebral substances was smoothed, the cortical layer was thinned, and the renal vessels were empty.



Figure 1 – Necropsy of a six-month-old saiga

In a six-month-old saiga with signs of alimentary dystrophy, a section of the cortical layer showed translucent tubules (granular dystrophy). The stomach was reduced in size, usually without content, and red spots were found in the submucous layer, mainly along the course of blood vessels (Vishnevsky's spots). The gastric mucosa was pale pink in color, collected in deep longitudinal and transverse folds, and covered with viscous, thick mucus of yellow-milk color (Figure 1).



Figure 2 – Necropsy of a yearling saiga antelope

Saigas under one year of age had nematodes in single numbers in the gastrointestinal tract. All young animals with alimentary dystrophy had inflammatory processes in lungs, namely serous catarrhal and purulent bronchopneumonia, which developed at decrease of general resistance of the organism. In serous pneumonia, the lung tissue is thickened, red or blue-red, the pleura is tense, and slightly turbid fluid flows from the cut surface. In catarrhal bronchopneumonia, the affected area of the lung had a red, compacted consistency. Turbid fluid was squeezed out from the surface of the incision and thick mucus from the bronchi. Depending on the size of the lung lesion, lobular or lobar pneumonia was observed (Figure 2).



Figure 3 – Necropsy of the digestive system of a three-month-old saigas

Examination of the digestive system of a three-month-old saiga, which had been starved for a long time and showed signs of alimentary dystrophy, showed that the stomach was reduced in size, contained a brown coloured frothy liquid, the mucosa was swollen, with pronounced folding, and the folds were poorly spread. Small intestine without contents, mucous membrane grayish, swollen (Figure 3).



Figure 4 – Necropsy of a saiga brain

One saiga head was diagnosed with cerebral cenurosis at a post-mortem examination of the brain (Figure 4). The pathomorphological diagnosis of saiga cenurosis includes a complete autopsy of saiga carcasses, a thorough examination of all internal organs: anatomo-physiological cavity of the skull, blood vessels of the brain, capillaries of the retina and nipple of the eye, and the liver, differing in that the examination on the first day of the clinical and pathomorphological manifestation of the disease reveals the localisation in the brain of single, oval and elongated shaped cenuroses up to 3-4 cm long in the left hemisphere, the injection of blood capillaries of the brain, connective tissue is atrophied in places of parasite location, congestion in the area of the bottom of the eye, overflow of blood vessels, hemorrhage on the retina and nipple of the eye, their colour change, in the liver pathological changes in the form of parenchymatous granular protein dystrophy, small whitish-yellow necrotic foci, destructive changes in ganglion cells of cortical and basal areas, lysis of neuroplasma and presence of shadow cells are revealed.

**Conclusion.** Thus, during the period of mass calving, saigas suffer from diseases of various etiologies, up to and including mass mortality of adults and young animals.

During this period, it is necessary to carry out monitoring surveys of the saiga population and to reinforce organizational veterinary and sanitary measures to prevent the mortality of wild saiga fauna.

Veterinary medicine has a major role to play in addressing these problems, and the use of innovative methods of diagnosis and prevention of various saiga diseases are vital elements in our society.

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### **ТҮЙІН**

Бұл мақалада Батыс Қазақстан облысы Жәнібек ауданында киіктердің жаппай төлдеу кезінде өлген киіктер ағзалары мен тіндеріндегі патанатомиялық көрініс сипатталған.

Киіктердің қахексия кезіндегі патанатомиялық өзгерістері негізінен алиментарлы сипатта болды: дистрофия (қондылығын жоғалту, қахексия), әртүрлі метаболикалық бұзылулармен, ферментопатиямен, ақуыз дистрофиясының дамуымен және ағзалардағы атрофиялық процестермен бірге жүреді. Киік төлдері біртіндеп қондылығын жоғалтып, дегидратацияға ұшырап, тері астындағы тіндерде, перикардияда және мезентерияда майдың болмауын дамытты. Көрінетін шырышты және серозды қабықтар - құрғақ, анемиялық. Бел аймағында омыртқаның қисаюы, рахитикалық розарий және сүт тістерінің зақымдануы анықталды. Барлық ішкі органдарда атрофиялық процестер анықталды: бауыр, бүйрек, көкбауыр ағзаларының түсі ашық қызылдан қоңырға дейін өзгерді.

Бауыр көлемі кішірейіп, түсі қою қоңыр, шеттері үшкір, консистенциясы бос, оның қоңыр атрофиясы байқалады. Көкбауырдың мөлшері кішірейген, түсі күлгін-қызыл, капсула мыжылған, жұмсақ консистенциялы болды. Капсуланың астында дәнекер тіннің ақшыл өсінділері көрініп, перикардия майсыз, коронарлық тамырлар мөлдір, миокард ашық қоңыр түсті күйде кездесті. Бүйректе бүйрек маңы майы жоқ, бүйрек мөлшері кішірейген, бос, капсула оңай жойылады, бүйрек түсі ашық қоңыр болады. Ағзаның ұштары кесіндісінде тегістелген, кортикальды қабат жұқарған, бүйрек тамырлары бос.

Бір жасқа дейінгі киіктерде асқазан-ішек жолында нематодтар бірнеше мөлшерде табылды, ал қалған жас төлдерде бронхопневмония мен серозды-катаральды сипаттағы қабыну процестері ағзаның жалпы төзімділігінің төмендеуіне байланысты өкпеде анықталды. Серозды пневмонияда өкпенің консистенциясы тығыздалған, қызғылт түсті, плевра шиеленіскен, кесілген бетінен бұлыңғыр экссудат ағып кетеді. Катаральды бронхопневмониямен жеңіл қызыл, шырыш тәрізді экссудат бронхтардан ағып кетеді.

Жарып-сою барысында бір киіктің басынан бас миын тексеру кезінде айқын дистрофиялық өзгерістері бар церебральды ценуроз диагнозы анықталды, ал бас миында көлемі 3-4 см-ге дейін сол жақ жарты шарда біркелкі, сопақша және ұзартылған ценурлар табылды. Паразиттер табылған аймақтарда мидың қан капиллярларының инъекциясы, дәнекер тіндері атрофияланған және көз түбінің аймағында тоқырау айқын көрінеді және ақуыздың дегенерациясы байқалды.

### **РЕЗЮМЕ**

В данной статье описывается патанатомическая картина в органах и тканях истощенных сайгачат павших во время массового окота матерей сайги в Жаныбекском районе Западно-Казахстанской области.

Патанатомические изменения при истощении сайгачат были характерны в основном алиментарной дистрофия (истощение, қахексия), сопровождающаяся различными нарушениями обмена веществ, ферментопатией, развитием белковой дистрофии и атрофическими процессами в органах. У молодняка сайгаков развивалось прогрессирующее истощение, обезвоживание, отсутствием жира в подкожной клетчатке, перикарде и брыжейке. Видимые слизистые и серозные оболочки сухие, анемичны. Обнаруживали искривление позвоночника в поясничном отделе, рахитические четки и повреждения молочных зубов. Во всех внутренних органах обнаруживались атрофические процессы в органов: печени, почек, селезенки, окраска варьировала от светло - красной до бурой.

Печень уменьшена в объеме, цвет темновато-бурый, края печени заострены, консистенция дряблая наблюдается бурая атрофия печени. Селезёнка уменьшена в размере, окраска фиолетово-красноватого цвета, капсула сморщенная, на разрезе мягкой консистенции, соскоб незначительный. Под капсулой видны беловатые разрастания

соединительной ткани, в перикарде отсутствует жир, коронарные сосуды полупрозрачны, миокард дрябловатый светло-коричневого цвета. В почке отсутствует окологпочечный жир, почки уменьшены в размере, дряблая, капсула снимается легко, почки светло-коричневатой окраски. На разрезе границы между корковым и мозговым слоем сглажены, корковый слой истончен, сосуды почек запустевшие.

У сайгаков в возрасте до одного года в желудочно-кишечном тракте были обнаружены нематоды в единичном количестве, а у остальных молодых животных в легких обнаруживались воспалительные процессы серозно-катарального характера с бронхопневмонией, вследствие снижения общей резистентности организма. При серозной пневмонии ткань консистенция легкого уплотнена, красноватого цвета, плевра напряженная, с поверхности разреза стекает мутноватый экссудат. При катаральной бронхопневмонии участки легкого красноватого цвета, уплотненной консистенции, с поверхности разреза стекает мутная жидкость, а из бронхов тягучий слизеподобный экссудат.

У одной головы сайгака при вскрытии головного мозга установлен диагноз ценуроз головного мозга с явными дистрофическими изменениями, при этом в головном мозге обнаружены одиночные, овальной и вытянутой формы ценурусы длиной до 3-4 см в левом полушарии, инъецированность кровеносных капилляров головного мозга, в местах обнаружения паразита соединительная ткань атрофирована и ярко выражены застойные явления в области дна глаза, выражена белковая дистрофия.

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