MINISTRY OF HIGHER AND SECONDARY SPECIAL EDUCATION OF THE REPUBLIC OF UZBEKISTAN

SAMARKAND STATE UNIVERSITY OF VETERINARY MEDICINE, LIVESTOCK AND BIOTECHNOLOGIES

DEPARTMENT OF "PARASITOLOGY AND ORGANIZATION OF VETERINARY AFFAIRS"

"I APPROVE"

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EDUCATIONAL AND METHODICAL COMPLEX ON THE SUBJECT

"PARASITOLOGY AND INVASIVE DISEASES"

Area of expertise:	400000 - Agriculture and water management
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The educational and methodological complex of the subject was developed in accordance with the approved curriculum, the working curriculum, the curriculum and the working curriculum.

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EDUCATIONAL AND METHODICAL COMPLEX ON THE SUBJECT "PARASITOLOGY AND INVASIVE DISEASES"

The educational and methodological complex of the subject was discussed at the 1st meeting of the "Parasitology and Veterinary Work Organization" department on August 25, 2022 and was recommended for discussion at the Faculty Council.

Head of the Department _____ **Sh.Kh.Kurbanov**

The educational and methodological complex of the science was discussed and recommended for use at the Council of the "Veterinary Diagnostics and Food Safety" Faculty (Report No. 1 of August 26, 2022).

Chairmanof the Faculty Council, Professor _____ **Davlatov R.B.**

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5. Characteristics of parasite hosts
6. Invasive diseases.

Keywords: Parasitology (parasites – freeloader, parasite, logos – teaching), parasitism, zooparasites, phytoparasites, helminthology, protozoology, arachnology, entomology, symbiosis, friendly, hostile, synonymy, indeferent, mutualism, commensalism, parasitism, hostile, permanent, temporary, definitive, intermediate, additional, optional, reservoir, ecto- and endoparasites.

1. A brief history of the development of Veterinary parasitology. Before the revolution, parasitology as an independent discipline did not exist. There was not a single institute in the whole of Russia where questions of parasitology were developed; this discipline was not taught either in higher and secondary veterinary and medical schools, or in advanced medical courses. However, some scientists in the field of zoology, veterinary medicine and medicine (K.A.Rudolfi, R.Leykart, G,F, Kuhenmeister, P.S.Pallas, I.I.Mechnikov, E.A.Ostrovsky, L.F.Borovsky, A.D.Fedchenko, D.L.Romanovsky, S.N.Kamensky, N.A.Kholodkovsky and others), along with other issues, were interested in parasitology issues.

In 1910, at the All-Russian Congress of Veterinarians, K.I.Scriabin raised the question of the need to organize departments of parasitology.

In 1917, on the basis of the Veterinary Institute in Novocherkassk (now the Don Agricultural Institute), the first department of Parasitology in the former Soviet Union was organized, of which K.I.Scriabin was elected professor. In 1920, K.I.Scriabin moved to Moscow and organized the Department of Parasitology and Invasive Animal Diseases at the Moscow Veterinary Institute (now MBA) and so, the development of parasitology after the revolution is directly connected with the name of academician K.I. Scriabin. He created helminthology on new foundations. Numerous expeditions organized by K.I.Scriabin made it possible to study the helminth fauna of the former Soviet Union in an unprecedented short time. The researchers described a large number of new types of helminths, revealed the cycles of their development and initiated the development of means to combat helminthiasis. K.I.Scriabin organized the All-Union Institute of Helminthology, which was named after him (VIGIS), and a helminthological laboratory at the Academy of Sciences.vshego Union.

In the development of vet.Professor V.L. Yakimov played an important role in protozoology in the post-revolutionary period. In 1919, he organized the Department of Parasitology at the Leningrad Veterinary Institute, where he began training protozoologists. Together with his students, he described over 120 types of pathogens

of protozoal diseases, studied the epizootology of these diseases, therapy and prevention. In addition to the V.L.Yakimov school, protozoologists were trained on the basis of the protozoological laboratory of RES under the guidance of A.V. Belitzer, and then A.A.Markov.

Arachnoentomological direction in parasitology was developed under the guidance of Academician E.N. Pavlovsky. His numerous expeditions made it possible to find out the spread of arthropods as carriers of vector-borne diseases and to create a new doctrine about diseases with natural foci.

Currently, in the former Soviet Union and other countries, parasitology comprehensively studies theoretical and practical issues in this specialty. Domestic veterinary parasitology faces the task of solving the problem of reducing the incidence of invasive diseases of animals on the basis of modern scientific achievements, and in the future to achieve their recovery.

2. General parasitology. Parasitology (Greek. Parasites-freeloader,

parasite, logos – teaching) is a complex science that comprehensively studies both the parasites themselves and the diseases caused by them and methods of combating them in humans, animals, birds and plants.

Parasitism is a historical association of genetically heterogeneous organisms based on food connections and interchange, when one (parasite) uses the other (host) as a habitat and food source, and both partners are in antagonistic relations of varying degrees of severity.

Of the total number of animal species close to 1.5 million, about 60-65 thousand (4-5%) are parasites. The widespread and diverse types of parasitism suggests that this life form with a special type of relationship to the environment arose in various systematic groups of animals independently of one another.

Parasites are the cause of many diseases of humans, agricultural, commercial and wild animals. In addition, parasites cause enormous damage to some agricultural crops and other plants.

Parasites related to animals are called zooparasites, and diseases are invasive or parasitic, in contrast to diseases caused by parasites-plants - phytoparasites, which are called infectious.

Depending on whether the “hosts” belong to the animal or plant kingdom, zooparasitology is divid

ed into zooparasitology of plants and zooparasitology of animals.

K.I.Scriabin, considering that all living organisms at that time were classified into two kingdoms: animals and plants, proposed to call the science of animal parasites zooparasitology and gave the following scheme of its content.

Zooparasitology, or as it is commonly called parasitology proper, consists of protozoology – the science of parasitic protozoa and diseases caused or caused; helminthology – studying parasitic worms and diseases caused by them; arachnology and entomology, which study arachnids and insects, both pathogens and carriers of infectious and invasive diseases.

According to the objects of parasitization of pathogens, parasitology is divided into medical, veterinary and agronomic. Veterinary parasitology refers to

zooparasitology; as a biological discipline, it is closely related to zoology. Knowledge of the morphology and systematics of parasites is necessary for the diagnosis of invasive diseases, and research on biology, ecology are the basis for carrying out a complex of preventive measures. In addition, parasitology is in close contact with epizootology, physiology, biochemistry, pharmacology, immunology, pathological anatomy, private pathology of infectious and non-infectious diseases, vet.san.expertise, ATS, as well as with other clinical, zootechnical and agronomic disciplines.

Types of relationships of organisms in nature. Organisms., inhabiting-

living on our planet, they live at the expense of nutrients obtained in two ways. Some of them synthesize nutrients – autotrophs; these include plants and some bacteria. Others absorb nutrients synthesized by autotrophs – these are heterotrophs (this is the type of nutrition in animals and most bacteria). Heterotrophs include organisms called "parasites" (para- about, sitos – nutrition). In the biological sense, parasites are organisms that use another living host organism as a food source and habitat. Their existence is determined by their relationship with the owner.

All organisms inhabiting our planet are in

more or less close communication with each other, often forming various cohabitations.

There are the following categories of biological relationships:

1). Indeferent; 2). Symbiotic; 3). Hostile.

An indeferent cohabitation is a community with such relationships of the organisms that make up it, in which each of them is completely independent of the others, but at the same time is closely connected with the life of the whole collective as a whole.

The symbiotic relationship is divided into two: friendly and hostile relationships.

Friendly cohabitation can be mutually beneficial (mutualism) or unilaterally beneficial (commensalism).

Mutualism is a cohabitation of organisms in which each side benefits from such a relationship.

Commensalism (freeloading) is a type of relationship between organisms in which one organism uses the remains of another organism's food without harming it.

Sinoikia (from sin- together, oikos- house, dwelling) is a type of relationship between organisms in which one organism uses another organism as a place of settlement, without harming it, and receives a certain benefit from it.

Hostile mutual relations of organisms are characterized by the fact that one (or both) organisms cause harm to another. The varieties of such relationships are predation and parasitism.

Predators are usually stronger than their prey, which they immediately kill and eat.

Parasitism is organisms that are maximally adapted to their hosts and special conditions of existence. Parasitism developed as a result of the joint evolution of cohabiting organisms. The parasite cannot exist without another host organism.

In addition to the concept of parasitism, there is also hyperparasitism (superparasitism) – a form of life of one parasite in another. An example is the protozoan flagellate (Histomonasmeleagridis), which can parasitize the eggs of the nematode Heterakisgallinarum.

Types of parasites. Animal organisms leading a parasitic lifestyle are usually divided into temporary and stationary.

Temporary parasites are organisms that complete the entire cycle of their development, from the egg to the adult stage, outside the host organism (they use the latter only for nutrition). These include horseflies, mosquitoes, bed bugs and some ticks. These parasites periodically attack animals (their hosts) for the purpose of nutrition, they are ectoparasites (external).

Stationary parasites infest the host for a long time, i.e. throughout their entire life and use it not only for food, but also for habitat. Stationary parasites are endoparasites. These parasites are divided into permanent and periodic.

Topic No. 2. VETERINARY HELMINTHOLOGY AND ITS COMPONENTS.

Helminthology (gelmins – worm) is a complex science that studies the morphology of various helminths in order to differentiate them and the diseases they cause for the organization of scientifically based measures to combat them. Helminthology is distinguished between medical, veterinary and agronomic, but there are common diseases for animals and humans and they are called anthropozoonoses. However, some helminthic diseases are known in which there is no specific place of their habitat in the host body, but their effect on the body is necessarily pathogenic.

Systematics of parasites

type of Plathelminthes flatworms cl. Trematoda – trematodes

Monogenea – monogeneia

Cestoda – cestodes

type of Acanthocephales

scrapers cl. Acantocephala – acanthocephala

type Nemathelminthes

roundworms of the Nematoda cl . – nematodes

of the Arthropoda type

arthropods cl . Insecta – insects

Arochnoidea – arachnids

type Protozoa Protozoa cl. Sporozoa – spores

Mastigophora – flagellates

Ciliata – infusoria

Helminths are multicellular organisms ranging in length from several mm to 30 meters or more. Their body is covered with a shell (tigument), the genitals and

excretory system are developed. The nervous and digestive systems are less developed. Cestodes and acanthocephalians have no digestive system.

Helminths have a bilateral symmetry of the body, a well-developed skin-muscle sac. There is no circulatory system. Most helminths belong to endoparasites (only monogeneans are ectoparasites).

According to epizootology, epidemiology and biology are divided into:

Geohelminths – they have the source of the spread of eggs and larvae are environmental factors. The epizootological chain in geohelminthiasis includes the host, elements of dead nature (soil, manure, water and care items) and the pathogen (eggs and larvae). Biohelminthiasis – intermediate and additional hosts are involved in their development. The links of the epizootological chain in biogelminthiasis are definitive and intermediate (sometimes additional) hosts and the causative agent of the disease. Trematodoses are endoparasites of animals, humans, rodents. According to scientist Petrichenko, there are ≈ 390 species of trematodes in mammals, ≈ 78 species in birds, and ≈ 30 in humans. Localization: bile ducts of the liver, digestive canal, pancreas, circulatory system.

Morphology. The body is mostly flattened in the dorso-ventral direction. The body shape is different: threadlike, pear-shaped. Fixation organs: oral and abdominal nipples. They have the following systems: nervous excretory digestive genital NS is represented by 2 ganglia located on both sides of the pharynx from which the nerve trunks depart.

The excretory system is located on both sides of the body. It has tubules that flow into a common channel. At the end there is a hole that is located on the back of the body.

The digestive system consists of an oral opening located at the bottom of the oral cavity, behind the oral opening is the pharynx, esophagus and intestinal trunks, which end blindly in the back of the body. Therefore, undigested food is thrown out through the mouth. The outside of the body is covered with a shell, which is called a tigument (outer cover). It participates in metabolic processes.

The reproductive system is well developed and complex. Almost all flukes are hermaphrodites (bisexual).

The male reproductive system consists of testes, vas deferens, which are enclosed in a muscle sac and is called a muscle sac – bursa. The genital system ends with the vas deferens and it is called cyrus (sexual organ). It opens next to the vagina in front of the abdominal sucker. The female reproductive system consists of an ootype (eggs are fertilized and formed there). From the ootype, the oviduct departs and connects with the ovary and with the ovary (contains sperm).

Biology. Biohelminths. Imaga (sexual outlook) is localized in vertebrates.

Larval stage in mollusks (intermediate hosts). Additional hosts: annelids, fish, mollusks, crustaceans, arthropods. They reproduce with the help of eggs (oviparous). When the eggs come out, they have an embryo that is surrounded by yolk cells. This suggests that they are invasive.

By this time, hundreds of different types of helminths in humans and animals were already known.

The founder of the scientific classification of plants and animals, the Swedish botanist Carl Linnaeus, included all organisms known by his time (1758) with the appearance of worms in the class Vermes (Latin name). As a result, fly larvae, earthworms and a number of other lower animals were mistakenly attributed by Linnaeus to this class too. The Australian zoologist Zeder (1800) corrected this error of Linnaeus. He subdivided the Vermes class into five distinct classes: roundworms, flatworms, suckers, spiny-headed and bubble worms.

Rudolfi (1819) did not recognize the independence of these classes, considering them to be units of the Vermes class, assigning them the following scientific (Latin) names, which have essentially not lost their meaning to the present. He named the flukes Trematoda, flat (ribbon) — Cestoidea, spiny—headed - Acanthocephala, round — Nematoidea and bubble — Cystica.

During the first half of the XIX century. helminths were studied mainly by zoologists. Since the beginning of the second half of the same century, scientists have begun to conduct experiments on the study of helminth development cycles. Kuhenmeister (1851) experimented on dogs and rabbits for the first time proved that the helminth of the mesentery of rabbits Cysticercus pisiformis is the larval stage of Taenia pisiformis, parasitizing in the intestines of dogs. Later, he also experimentally established that from Cysticercus cellulosae (pork finn), parasitizing in the muscles of pigs, develops into Taenia solium in the human intestine.

After feeding the dog echinococcus bubbles, Siebold (1853) infected her with the ribbon stage of the parasite of the same name. He also experimentally proved that Multiceps multiceps is formed in the intestines of dogs as a result of eating the brains of sheep suffering from vertyachka, i.e. having a larva of this cestode in the form of a bubble in the brain, known as Coenurus cerebralis.

Leykart (1861) proved that calves become infected with finnosis as a result of ingestion of individual eggs, or whole mature segments of bovine tapeworm (Taeniarhynchus saginatus), parasitizing in the human intestine. A number of other researchers repeated the above experiments and invariably established that bubble worms are larval stages of tapeworms. On this basis, the class of bubble worms has completely lost its independence since the second half of the XIX century.

The Danish scientist Steenstrup (1842) experimentally proved that flukes develop during the alternation of sexual and asexual reproduction with the obligatory change of hosts. Leucart (1859) studied this process in particular detail for the causative agent of fascioles of sheep (Fasciola hepatica). Tsenker (1860) proved for the first time that a person becomes infected with Trichine/la spiralis when eating raw (not neutralized) pork containing encapsulated larvae of this parasite in the muscles. He also found that pigs become infected with trichinella by eating rats, which, in turn, become infected with trichinella when eating kitchen and household waste. He also proved that dogs are also infected with trichinella in a similar way.

Experiments on the development cycles of cestodes and trichinella for the first time allowed us to recommend scientifically-based preventive measures for helminthiasis transmitted to humans through meat and meat products. To this end, in a number of countries in Europe, America, as well as in Russia, veterinarians of

slaughterhouses in the 1870s and 1880s were entrusted by special government orders with the duty to examine meat for finnosis, and in Germany, Denmark, France and England - also for trichinosis. At the same time, the manual on human and animal parasites (Germany) began to be published. In a revised form, it was published in Russian by E.K. Brandt (1873), (professor of the Medical and Surgical Academy in St. Petersburg) under the title "Animals - parasites of humans and domestic animals".

At the beginning of the current century , Fulleborn et al . infection with strongyloides was proved by penetration of larvae through intact skin with their subsequent migration through the host's bloodstream. A little later, Stewart, Nance, and Fulleborn (1923-1925) established the phenomenon of migration of human and animal ascaris larvae through the bloodstream into the lungs and then back into the intestines, which was a very major discovery in the field of helminthiasis pathology.

Since that period, the most productive scientific research on pathology and the development of anti-helminthic measures in the field of medicine and veterinary medicine began to be conducted in our country under the leadership of Academician Konstantin Ivanovich Scriabin. During the fifty-year period (1917-1967), the works of K.I. Scriabin and the works of his numerous students have won major achievements in the field of helminthology:

• a cadre of highly qualified helminthologists-physicians, veterinarians, biologists and agronomists, including dozens of doctors and hundreds of candidates of sciences, including such outstanding researchers in the field of veterinary helminthology as: D.N. Antipin, S.N. Boev, P.A. Velich Kin, V. S. Ershov, L. A. Losev, I. V. Orlov, A.M. Petrov, V.I. Petrochenko, V.I. Pukhov, R.S. Shultz, E.E. Shumakovich and many others;

• K.I. Scriabin proposed (1925) a special method of combating helminthiasis in humans and animals, which he called deworming;

• • K.I. Scriabin and his students have published dozens of major monographs on helminthiasis of all types of farm animals and humans;

• • original textbooks on veterinary parasitology have been created for students of veterinary universities and students of veterinary technical schools, some of them have been translated into a number of foreign languages.

Topic No. 3. TREMATODES AND TREMATODOSES. ANIMAL FASCIALOSIS,

Lecture plan:

1. Veterinary helminthology.
2. Flukes and flukes (morphology, biology and systematics)
3. Fascioles of animals

Keywords: Helminthology, helmins, logos, Plathelminthes, Trematoda Nemathelminthes, Acantocephalis, Anntlides, Fasciolata, Paramphistomatata, Heterophyata, Echinostomatata, Schistosomatata, Fasciola, Fasciola hepatica, F.gigantica, Lymnaea, L.auricularia, L.truncatula, biohelminth , final, intermediate

host, deworming, biothermic treatment, planned, 4-carbon chloride. rolenol, combitrem, dertil, facocide.

1. Flukes and flukes.Trematodes are parasitic worms belonging to the type of flatworms Plathelminthes, a class of Trematoda – flukes. All flukes are parasites localized in various organs and tissues of animals and humans.

Trematodes are most often leaf-shaped, their size varies from 0.1 mm to 10-15 cm in length.The body is covered with a musculoskeletal sac containing internal organs.At the front end of the body there is a mouth sucker, at the bottom of which there is a mouth opening. The mouth is followed by a pharynx (pharynx) and an elongated esophagus. The intestine usually consists of two blindly ending trunks. As a rule, there is no anal opening in flukes. The remains of undigested food are thrown out through the mouth. On the ventral side of the body there is an abdominal sucker – an organ of fixation. The excretory system is represented by a complex network of tubules ending in an extra tube with an opening in the back of the parasite's body. The nervous system consists of nerve nodes lying under the glottis and nerve trunks extending to other parts of the body.

The reproductive system of trematodes is well developed, it is very difficult to build. Trematodes (with the exception of representatives of the order Schistosomatata) are hermaphrodites.

The male reproductive apparatus includes two testes, from which one vas deferens (Vasefferens) depart, the vas deferens are connected to a common vas deferens (Vasdeferens). It is usually included in a special muscular sac – the genital bursa ((Bursacirri) and opens with an external male genital opening on the ventral surface of the body of the fluke. The final part of the vas deferens is the copulatory organ (Cirrus), there are prostatic glands (Glandulaeprostatae) in the genital bursa, they surround the vas deferens.

The female reproductive apparatus includes the ootype, where fertilization and egg formation take place. The ovary, which secretes germ egg cells, as well as the ovary, are connected to the ootype through the oviduct, In addition, the ootype communicates with the yolk cells, which produce nutrient material for eggs.Often the ootype communicates with the ventral surface of the body through the laurel canal (canalisLaureri). An excess of yolk cells is removed through this channel, and sometimes it serves for copulation, playing the role of a vagina. The uterus has the form of a convoluted tube, with one end it connects to the ootype, the other communicates with the external environment through the external female genital opening. Special glands (the body of Melissa) secrete fluid that washes the ootype and uterus and facilitates the free sliding of eggs along the genital tract to the opening of the uterus. The uterus also performs the function of the vagina. External genital openings are located in different flukes in very different parts of the body, most often along the midline of the body, in the gap between the suckers.

Biology of the pathogen. Flukes are biohelminths, the first intermediate hosts are mollusks, both aquatic and terrestrial (land). In addition to the intermediate host, additional hosts take part in the development of many trematodes, which may be fish, amphibians, insects or mollusks.The fertilized eggs of flukes secreted by marita (a

sexually mature parasite) are covered with a shell, inside which are the fertilized egg and the yolk cells surrounding it. The eggshell has four shells: three outer ones protect the embryo from mechanical damage, and the fourth inner one protects it from chemical influences.

In the future, a miracidium is formed in the egg, which either comes out of the egg, actively attacks the corresponding mollusk and invades its tissues, or is swallowed by the mollusk. Sometimes the intermediate host swallows an egg, inside of which there is a miracidium. In the mollusk, the miracidium develops into the next larval stage – the maternal sporocyst (it is a bag filled with germ cells), the latter parthenogenetically gives rise to the next generations – pre-modern sporocysts, or redia. Cercariae are formed from redia. If the trematode develops with the participation of one intermediate host, then the cercariae, coming out of the mollusk, lose their tail, encyst and pass into the invasive stage- the adolescarium. Cercariae of trematodes, in the development cycle of which there is an ass-filling host, actively or passively penetrate into it, encyst and develop into invasive metacercariae. In the body of a definitive host, the shell of invasive larvae is destroyed and young flukes reach the parasitizing site in various ways and develop into marita.

In some cases, the development cycle of trematodes becomes more complicated (daughter sporocysts, redia) or, conversely, individual stages may fall out.

Thus, the reproduction of trematodes occurs both sexually (in the marita stage) and asexually - parthenogenetic (sporocyst, redia).

Taxonomicatrematodes. Representatives of five sub-orders of trematodes are of the greatest veterinary and medical importance.

Fasciolata – develop with one or two intermediate hosts. The most common species are Fasciola hepatica, Fasciola gigantica, Dicrocoelium lanceatun – parasites of the bile ducts of the liver; Eurytrema pabcreaticum –pancreas;, Hastilesia ovis - the small intestine of sheep and goats; various species of the genera Prosthogonimus and Plagiorchis – flukes of the oviduct of birds, etc.

Paramphistomatata (Paramphistomatata) is characterized by the absence of an oral sucker and an abdominal sucker shifted to the posterior end of the body. Development with the participation of one intermediate host – an aquatic mollusk, parasitized in the rumen of agricultural and wild animals.

Heterophyata (Heterophyata) – small and medium-sized flukes, develop with the participation of two intermediate hosts, fish and crustaceans serve as additional hosts. In carnivores, representatives of the Opisthochidae family – species Opisthorchis felineus, Clonorchis sinensis - are often found in the bile ducts of the liver.

Echinostomatata (Echinostomatata) – trematodes with an armed and unarmed adoral disc, most species parasitize waterfowl. Development takes place with the participation of two intermediate hosts, and additional hosts can be mollusks, amphibians, fish. The most common species of wild and domestic ducks is Echinostomarevolutum.

Schistosomatata (Schistosomatata) – bisexual trematodes. Suckers are poorly developed, eggs are usually provided with a spike, parasitize in the circulatory system of birds and mammals, develop with the participation of one intermediate host – a mollusk. Invasive larvae are cercariae that actively enter the body of a definitive host through the outer integuments.

FASCIOLES are trematodous diseases of sheep, goats, cattle, as well as numerous domestic and wild mammals caused by trematodes of two species: Fasciola hepatica and Fasciola gigantica, family Fasciolidae. A person also suffers from fascioliasis. The sexually mature fasciole is parasitized in the bile ducts of the liver. The disease proceeds chronically, less often acutely and is expressed by a violation of the general metabolism with damage mainly to the liver.

Distribution. Everywhere.

Economic damage. In animals with fascioliasis, productivity is significantly reduced, in cows, milk yields decrease by 25-40%. The quality of meat and wool deteriorates, there is a case, a large number of the liver of sick animals is culled with VSE carcasses, as well as veterinary costs for the treatment and prevention of the disease.

Pathogens. Fasciola hepatica - Fasciola vulgaris. Its length is 2-3 cm, width is 1 cm. There are spikes on the cuticle of the anterior part of the body from the dorsal and ventral sides. The front part of the body is elongated in the form of a proboscis with two suckers, the intestine with lateral processes, the testes are tree-branched, the uterus is located in the front part of the body, followed by the ovary, the lateral fields of the prasite are densely filled with clusters of yolks.

Fasciola gigantica – giant fasciola, differs from the common fasciola in size and body shape, in the details of the development cycle and pathogenicity. Its length is 4-7.5 cm, width 0.6-1.2 cm, the body is elongated, the lateral edges of the parasite are parallel, they are a pathogenic species.

Biology of pathogens. The parasite is a biohelminth, developing with the participation of an intermediate host. The intermediate host is the small pond - Lymnaea truncatula, and for Fasciola gigantica-the ear-shaped pond - Lymnaea auricularia.

Together with bile through the common bile duct, fasciole eggs enter the duodenum, and then into the external environment. For the further development of eggs, oxygen, appropriate temperature and humidity are necessary first of all.

Under favorable conditions, after two weeks, a miracidium develops in the egg, which leaves the egg into the water and penetrates into the body of the intermediate host – mollusks. In the body of the mollusk, the miracidium sucks cilia, migrates to internal organs, mainly in the liver, and after a few hours becomes a maternal sporocyst. Sporocysts, through asexual development, give rise to a further generation – redia, and cercariae are already emerging from them. From the mollusk, the cercariae go out into the external environment, and there they float freely for some time, and then they attach to some solid object and whether it remains on the surface of the water and is encysted. The tail appendage disappears, the cystogenic glands

envelop the body of the larva, forming a dense shell – a cyst. A cercarium covered with a cyst is called an adolescarium (an invasive larva).

Topic No. 4: PARAMPHISTOMATOSIS AND ORIENTOBILCHARCIOSIS OF ANIMALS.

Lecture plan:

1. Paramphistomatosis
2. Orientobilkhartsioz

Keywords: Ruminants, freshwater mollusks, Planorbis, Planorbis planorbis, Gyraulus filiaris, Segmentina nitida, deworming, bithionol, carbon 4-chloride, copper sulfate.

PARAMPHISTOMATOSIS is a trematodous disease of cattle, buffaloes, less often sheep and goats, as well as reindeer and some wild animals, caused by trematodes of the Paramphistomatata suborder, parasitizing in the small intestine, rumen and less often in the mesh of animals. The disease is acute and chronic. Acute paramphistomatosis is observed in calves aged 1.5-2 years and is caused by trematodes migrating into the mucous membrane of the small intestines and rennet. Chronic paramphistomatosis is caused by adult helminths parasitizing in the pancreas, mainly on the mucous membrane of the scar.

Distribution. Paramphmstomatosis is widespread everywhere, including in our Republic. On the coast of the rivers Amu Darya, Sirdarya, Zerafshan and other rivers, where intermediate hosts of the parasite are found.

Economic damage. Paramphistomatosis causes great economic damage: with an acute course, up to 50-100% of young cattle die, the growth and development of calves slows down sharply. And with chronic – the productivity of animals decreases, as well as veterinary expenses for the treatment and prevention of the disease.

Pathogens. From paramphistomat at krp.rog.cattle and sheep are parasitized by several species belonging to different genera: Paramphistimum, Calicophoron, Gastrotylax, Liorchis. Of these, the most widespread are the Chlorchisscotiae, P.ichikawai, C.calicophorum, G.crumenifer.

Their body is fusiform or cylindrical, and has the shape of a circle on the cross sections. The length of the flukes is 5-20 mm, there is no oral sucker, a powerful pharynx follows the oral opening, the abdominal sucker is well developed and splits near the posterior end of the body.

Biology of pathogens. In principle, the development cycle of all inputs of the Paramphistomatata suborder is the same. So, for example, P.cervi eggs, together with the animal's phacalia, are released into the external environment, where the miracidia come out of them. The term of development of the latter depends on the temperature of the medium. At 19-27oC, the miracidium hatches after 12-13 days and begins to swim in the water, then the miracidium penetrates into the body of the mollusk, forms into a sporocyst and develops up to 9-11 redia in it, in which dechernia redia or

cercariae can form. The cercarium leaves the mollusk after 1.5-3 months and encyst in the water on plants, becoming invasive adolecramia.

Definitive hosts become infected by ingestion of adulterants during watering or grazing. Young paramphistomas are embedded in the thickness of the intestinal wall of the animal, primarily the duodenum, pass through the tissue stage of development in it and migrate into the scar retrograde through the gastrointestinal tract. The shortest period of growth and development of paramphmistomes to the sexually mature stage in cattle and sheep is 3.5-4 months.

Epizootological data. Animals are infected on low-lying pastures throughout the grazing season. Invasive mollusks overwinter and are the main source of infestation of reservoirs. Adolescariums are unstable and die when biotopes dry out. Usually, outbreaks of acute paramphistomatosis are observed one month after the calves are driven to pasture. The patent period of development of the parasite is several years, on average 4-5 years. Wild ruminants can also be a source of invasion.

Pathogenesis. Pathogenic influence is exerted by both sexually mature paramphistomas and young parasites during migration from the intestine to the rennet and scar. The influence of parasites on the body of the animal is composed of moh: mechanical, toxic and open the gate to infections.

Immunity. Non-sterile. During the disease period, antibodies appear in the blood of infected animals, after the organisms are freed from helminths, the antibodies also disappear.

Symptoms of the disease. At the croup. horn.the acute course of cattle is associated with the parasitization of young migrating parasites; in most cases, the infection ends fatally or acquires a chronic course. The clinical picture manifests itself 2-4 weeks after the animals are driven to unfavorable pastures and is observed for 17-35 days. They note the pallor of the visible mucous membranes, moaning and gnashing of teeth, loss of appetite, progressive exhaustion and sedimentation, hypotension and atony of the pre-ventricles, panoses, periodically replaced by constipation, swelling in the submandibular space and underbelly. Protein appears in the washing of animals. The number of erythrocytes and hemoglobin content in the blood decreases, leukopenia and leukocytosis are noted. Chronic course in kr.rog.cattle are characterized by prolonged incessant diarrhea, progressive emaciation, anemia.

Pathoanatomic changes are mainly observed in the habitats of young immature helminths: the 12-duodenum and the pyloric part of the abomasum. The nature of the changes depends on the degree of infection and is expressed by swelling and catarrhal hemorrhagic inflammation of the duodenum, the pyloric part of the abomasum and an increase in mesenteric lymph nodes, exhaustion, anemia of visible mucous membranes. There are gelatinous infiltrates in the area of the submandibular space and the breast. Thousands of paramphistomats are found in the 12-pert intestine. With chronic paramphistomatosis, atrophy of the villi of the scar is noted at the sites of detection of trematodes.

Diangosis and differential diagnosis. The diagnosis during life is made on the basis of the clinical picture, epiz. Data and results of fecal examination by the

methods of helminthoscopy tgnlmintoovoskopicheskimi. The final diagnosis is clarified after finding young flukes in the small intestine and rennet.

Paramphistomatosis should be distinguished from fascioliasis, strongylatosis of the gastrointestinal tract, paratuberculosis, alimentary dystrophy.

Treatment. In the chronic course of paramphistomatosis of the croup. horn.cattle use bitionol at a dose of 0.07 g / kg after an 8-12-hour fasting diet. In the acute course of paramphistomatosis, bithionol is used twice with an interval of 48 hours.

4-carbon chloride – in a dose of 2-5 ml per head in gelatin capsules or diluted in parts of cottonseed oil and injected intramuscularly or injected directly into the scar.

1-2% solutions of copper sulfate in a dose for calves – 80-120 ml per head, for adults - 150-200 ml per head.

Resorantel only croup.horn.cattle at a dose of 0.065 g / kg 2 times with an interval of 2 weeks.

Gilomite – at a dose of 0.05 g / kg 2 times with an interval of 2 weeks.

Kerosene (white) -15-20 ml per head only for calves and fattening animals, is it impossible to milk a cow?

Prevention and control measures. Basically, the same set of measures is envisaged as for fascioliasis. Plan planned deworming of the entire affected croup.horn.cattle in the stable period. If necessary, deworming is repeated two weeks after the first one. In summer, during the period of possible outbreaks of invasion, 3-4 weeks after pasture, two-fold preimaginal deworming of young animals will be carried out with intervals of 10 days of young animals under the age of two years.

Manure is disinfected by biothermic means, the fight against intermediate hosts by mechanical, physical, chemical and biological methods, as well as full-value feeding of animals according to the diet.

Of great importance is the protection of susceptible animals from infection with paramphistomatosis. Therefore, animals should not be pasted on poor low-lying, swampy and rich in small bodies of water areas of pastures, it is impossible to drink from sewage ponds, small ponds, machazhin, puddles, ditches, feed with mown grass from areas of pastures that are poor in paramphistomatosis. It is advisable to practice changing pastures.

Upon admission to the farm of a new batch of cereals.horn.cattle are carried out their selective helminthoprological examination. When infected animals are detected, they are dewormed. And then admission to the general herd.

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ORIENTOBILHARZIOSIS is a focal disease of cattle, sheep and goats caused by trematodes from the genus Orientobilharzia of the suborder Histosomatata. These are peculiar bisexual trematodes, which in the sexually mature stage live in the blood of vertebrates – in the vessels of the mesentery, liver, pancreas, submucosa of the intestine.

Distribution. Orientobilkhartsioz is widespread everywhere, in our republic it occurs in the Khorezm region and the Autonomous Republic of Karakalpakistan.

Economic damage consists of the death of infected animals, stunting in growth and development, veterinary costs for the treatment and prevention of the disease. is a focal disease of cattle, sheep and goats caused by trematodes from the genus Orientobilharzia of the suborder Histosomatata.These are peculiar bisexual trematodes, which in the sexually mature stage live in the blood of vertebrates – in the vessels of the mesentery, liver, pancreas, submucosa of the intestine.

The pathogen. Orientobilharzia turkestanica is a bisexual trematode, the male is 6.4-12.9 mm and 0.40-0.64 mm wide. The oral sucker is round, the testes are oblong-oval in the amount of 78-80 are located in 2 rows between the intestinal trunks. The female is 4.8-6.8 mm long and 0.08-0.14 mm wide. Suckers are rudimentary. In the uterus, one egg is elongated-oval in shape, has one tweezer on each of the poles: one of them has the appearance of a slightly curved process, and the other is a curved appendage with a constriction on the border with the body of the egg itself. The length of mature eggs is 0.13-0.14 mm and the width is 0.04-0.06 mm. They contain motile miracidium.

Biology of the pathogen. The definitive host is cattle, sheep, goats, reindeer and other wild ruminants. The intermediate host is the ear–shaped pond mollusk – Lymnaea auricalaria.

Orientobilharans penetrate into the smallest veins in close proximity to the intestinal wall of definitive hosts and here they lay not yet fully mature eggs in the lumen of small vessels. Further development and formation of myracimdii in the egg occurs in the host tissue. Mature eggs penetrate into the intestines, with feces they are released into the external environment, where the miracidium comes out of them, penetrating into the mollusk. In the mollusk, the miracidium forms into a maternal sporocyst, which, multiplying parthenogenetically, gives rise to daughter sporocysts,

and then to cercariae. Cercariae come out of the mollusk into the water and can enter the body of definitive hosts by active introduction through the skin or mucous membranes and reach the sexually mature stage within 35-40 days. Orientobilharans live in the body of the main host for an average of 4-5 years.

Epizootological data. The definitive host is infected at any age. Orientobilkhars are found in all months of the year, but the maximum peak reaches the autumn-winter period. Animals become infected during watering and grazing in places with a fresh water spill, where mollusks live. Of ruminants, cattle are more susceptible to the disease.

Pathogenesis and symptoms of the disease. Orientobilharans are extremely pathogenic flukes and cause severe disease in endemic foci, sometimes accompanied by death.The pathogenic effect of the pathogen on the animal's body is caused by the mechanical destruction of the host tissue by cercariae in the process of introduction and migration, the impact of sexually mature parasites and their eggs, the migration of eggs through the host tissues, intoxication of the host. Allergic phenomena cause a diverse clinical manifestation.

Orientobilchartsii are especially potogenic for sheep and goats. The animals have depression, and in the future progressive diarrhea with a premix of blood and fibrin films. The visible mucous membranes are anemic. The temperature is 41,2-41,4 ^{o}C, the pulse and breathing are rapid. This period often ends with the death of animals, especially young ones.

Pathoanatomic changes. In the small intestine, intense hyperemia and swelling of the mucosa, single and multiple nodules When viewed to light in mesenteric blood vessels, it is easy to notice orentobilchartsii. In scraping off the intestinal mucosa of sheep affected by orientobilcharciosis, under a microscope, the eggs of the parasite.

Diagnosis. For lifetime diagnostics, the method of detecting miracidia (Berman-Orlov) is used.

Treatment. It is recommended twice (every other day) intramuscular administration of fadin at a dose of 0.3 ml / kg. Asiomv D.A. (1986) indicates the high efficacy of albilgaoa in doses: sheep and goats 0.02-0.025 g /kg, calves 0.03-0.04 g / kg orally twice with an interval of 5 days.

Prevention is based on the destruction of the ear-shaped pond in the same ways as with fascioliasis. When using molluscocides in the most intense foci of invasion, all precautionary rules should be observed when working with them.

TOPIC NO. 5. DICROCELIOSIS AND EURYTHREMATOSIS ANIMALS.

Lecture plan:

1. Dicroceliosis
2. Eurythrematosis

Keywords: chronic, definitive, intermediate, additional, land mollusks, ants, Dicrocoeliidae, Dicrocoeliumlanceatum, Helicella, Zebrina, Theba, Fruticola, Formica, Proformica, deworming, panacur (fenbendazole), tafen.

DICROCELIOSIS is a widespread trematode disease caused by the Dicrocoeliumlanceatum trematode of the Dicrocoeliidae family, parasitizing the bile ducts and gallbladder of more than 70 species of domestic and wild mammals. Mainly ruminants (sheep, goats, cattle, buffaloes, camels, deer and others) are ill. Dicroceliosis also occurs in humans.

Distribution. Dicroceliosis is widespread everywhere, especially in steppe areas, where there are all conditions for the development of intermediate and additional hosts. The disease occurs most often in the desert-pasture zone than in the irrigation and foothill-mountain zones.

Economic damage. The damage from dicroceliosis consists of a slowdown in growth and development, a decrease in the productivity of animals, an increase in feed consumption as well as the expense for carrying out therapeutic and preventive measures.

The pathogen. Dicrocoelium lanceatum is a very small fluke, reaches 1-1.2 cm in length with a width of 1.5-2.5 mm. The lanceolate formatela, the oral and abdominal suckers are close together, the intestinal trunks are smooth, located on the lateral parts of the parasite's body. In the front part of the fluke body behind the abdominal sucker are two testes, then the ovary, the ovary, the Melissa body. In the middle part of the body, to the side of the intestine, there are bunchy yolks, the uterus is tree-branching, occupies the entire posterior half of the fluke. Egg dicrocelia are small, 0.038-0.045 mm long with a width of about.023-0.030 mm, dark brown in color with a thick shell. They are asymmetrical, contain miracidium, and a lid is located at one pole.

Biology of the pathogen. The parasite is a biohelminth, developing with the participation of definitive, intermediate and additional hosts. Eggs of the pathogen with already developing miracidia with bile enter the intestines of animals, and then with feces - into the external environment, where they are swallowed by various terrestrial mollusks.In the intestine of the mollusk, the miracidium leaves the egg, penetrates into the connective tissue between the liver follicles, sheds cilia and becomes a maternal sporocyst, after which it splits into a number of daughter sporocysts, in which, in turn, cercariae are formed.

After the appearance of mature cercariae, the process of formation of new cercariae in daughter sporocysts does not stop, so mollusks have been parasitic carriers for a long time. From the daughter sporocysts, cercariae are introduced by blood into the respiratory cavity of the mollusk, where they are enveloped in a sticky mucous substance. The formed mucous lumps (100-200 or more in each) are pushed out by respiratory movements from the mollusk and stick to plants. The development of dicrocelium in the body of a mollusk lasts 3-6 months. In the external environment, mucous lumps with cercariae are eaten by various types of ants. In the ant's body, the cercariae are freed from mucus and actively penetrate from its

intestines into the abdominal cavity and there they lose their tail, begin to encyst and after 1-2 months develop into invasive metacercariae.

Definitive hosts become infected with dicroceliosis during grazing, swallowing ants infested with metacercaria along with grass. It was found that after infection of ants, one of the cercariae from the abdominal cavity of the insect penetrates into the head part - they fall into a state of stupor. Such ants hold tightly to plants with their jaws and almost do not react to external stimuli, which facilitates the infection of definitive hosts.

Metacercariae after digesting ants are released from cysts, actively penetrate into the liver of the animal through the common bile duct and after 72-85 days become sexually mature and secrete eggs.

Epizootological data. Dicroceliosis is spread almost everywhere, but especially in the south, in the steppe regions of the country. Animals get infected. As a rule. On pastures, most intensively in spring and autumn. Ants frozen in the grass are more often found in the morning and evening.

Dicrocelia eggs are very resistant to various environmental influences. They tolerate drying at a temperature of 18-200 C for a week . The temperature does not kill them up to 500C. They are also very resistant to low temperatures. Thus, eggs and larval forms of parasites can overwinter in mollusks and ants without losing their invasiveness.

Wild artiodactyls can also play a role in the spread of dicroceliosis among domestic animals, if they use the same pastures.

Pathogenesis. Mechanical, toxic and opens the gates to secondary infections.

Immunity. As a result of primary infection with dicroceliosis, short-term immunity is created (but not in all animals) with a slight tension.

Symptoms of the disease. With a weak invasion, there are no clinical signs of the disease. With the accumulation of a large number of helminths, a serious disease is observed. The animals are depressed, they note a violation of the digestive function, diarrhea alternates with constipation. The coat becomes brittle, bare areas appear on the body. Swelling in the chest and underbelly. Cows have reduced milk yield. Clinically, dicroceliosis is manifested in sheep older than three years. They develop emaciation, followed by emaciation, a decrease in all types of productivity. Death in the animal is almost not observed, and with unsatisfactory conditions of feeding and maintenance, the disease sometimes ends in death.

Pathoanatomic changes. With a strong invasion, the liver is enlarged, the bile ducts are dilated, they are dense and have the appearance of white strands filled with a semi-liquid brown-greenish mass containing many parasites. The gallbladder has thick bile and a large number of parasites. At autopsy, exhaustion, anemia, gelatinous infiltration of the subcutaneous membrane are observed in such animals.

The diagnosis during life is made on the basis of the detection and feces of parasite eggs, for which ovoscopy methods are used. The postmortem diagnosis is based on the detection of dicrocelia in the liver, taking into account pathoanatomic changes.

Treatment. A highly effective drug is hexychol in doses of 0.3 g / kg to a large horn.cattle, calves and sheep and goats 0.4 g / kg, hexinol C can be used at a dose of 0.3 g / kg, panacur (fenbendazole) twice with a daily interval at a dose of 0.1 g / kg, cattle - at a dose of 0.15 g / kg, tafen at a dose of 0.21 g / kg, once in the morning feeding with a large amount of compound feed by the method of free feeding.

Prevention consists of a complex of anti-mycoceliosis measures: deworming of adult livestock, helminthological assessment of pastures, organization of stall-walking keeping of young animals, destruction of intermediate hosts, veterinary and sanitary restrictions.

Preventive deworming of animals is best carried out in the autumn-winter months.

In the fight against intermediate hosts, the plowing of virgin lands with subsequent sowing with cultivated grasses, the destruction of shrubs, the cleaning of stones on pastures, and, where permissible, the burning of dry grass are effective. Cattle are not grazed in areas that are unfavorable for dicroceliosis.

The effectiveness of the health measures carried out is checked annually. To do this, 20-30 animals from each farm or flock that is not affected by the disease are examined helminthically.

Euritrematosis is an invasive disease of sheep, goats, cattle, camels, pigs, deer, as well as many wild ruminants caused by the trematode Euritremapancreaticum. Dicrocoeliidae.Euritremapancreaticum is parasitic in the pancreas and very rarely in the liver.

Distribution. Everywhere.

The pathogen and its biological development. Euritremapancreaticum is 8-16 mm long, 5.5-8.5 mm wide, with large, strongly protruding oral and abdominal suckers. Live specimens are bright red in color, the testes are oval, arranged symmetrically on the sides of the abdominal sucker. The ovary is spherical, lying behind the abdominal sucker several times smaller than the testes. The uterus is tree-branching, occupies the entire posterior half of the fluke.

Intermediate hosts are a land mollusk from the genus Eulotalantzi, additional insects are grasshoppers from the genus Conocephalus and crickets –Oecanthus.

Eurythreme eggs isolated with feces are swallowed by land mollusks, in whose intestines the miracidium comes out and, penetrating into the liver of the mollusk, develops into a maternal sporocyst. A month later, daughter sporocysts are formed in it, leaving the maternal individual through the "birthing season". After about a year, cercariae form in them. Containing cercariae, migrate from the liver into the mantle cavity of the mollusk, and then come out and fall on the grass and soil. In the future , cercariae swallow grasshoppers of various species (), and after about 2 months they reach the stage of metacercariae.

Definitive hosts become infected with eurythremes by eating infested grasshoppers with grass, they develop sexually mature stages of the parasite in 2-3 months. The patent development period does not exceed one year.

Epizootological data. In the former Soviet Union, eurythrematosis was registered in the republics of Central Asia, Kazakhstan, the Far East, Altai, Crimea

and the Caucasus. The source of the invasion is infected animals and pastures and water infected with infested grasshoppers, The infection of animals occurs in pasture conditions in spring and increases in summer.

Pathogenesis Localized inside the excretory ducts of the pancreas, eutritremes cause their deformation, expansion, and sometimes blockage. The glandular part of the parenchyma of the gland atrophies.

Symptoms of the disease. With intensive invasion, the animals are severely depleted, they have chronic diarrhea and swelling in the head, neck, chest. Sometimes, with the phenomena of cachexia, death occurs.

Pathoanatomic changes. The pancreas is enlarged, red-pink in color, flabby consistency. The ducts of the pancreas are dilated, with a large number of trematodes. Deep necrotic processes of parenchyma are noted.

The diagnosis during life is made on the basis of ovoscopy: sequential washing of feces or the Darling method. Eurythreme eggs are dark brown, 0,044-0,048 mm long, 0,032-0,036 mm wide, there is a lid on one pole, an appendage in the form of a button on the opposite pole; they must be differentiated from dicrocelia eggs.

Treatment and prevention have not been developed.

Topic No. 6: CESTODES AND CESTODOSES. CYSTICERCOSIS CATTLE AND PIGS.

Lecture plan:

1. *General morphology of cestodes*
2. *Development cycle*
3. *Systematics of cestodes*
4. *Cysticercosis of the croup.horn.cattle*
5. *Cysticercosis of pigs*

Keywords: Class Cestoda, type Plathelminthes, order Cyclophyllidea - tapeworms and Pseudophyllidea of lentets,scolex, neck, body (strobila), segment (proglottid), sucker (botria), cysticercus, echinococcus, cenurus, alveococcus, cysticercoid, strobilocercus, tetratidium, procercoid, plerocercoid , biohelminth, egg, larva, teniata, anoplocephalata, daveniata, mesocestoidata, hymenolepidata.bubble, armed, unarmed.

General morphology of cestodes. Cestodes belong to the type of flatworms, the class of Cestodes- tapeworms. Of veterinary and medical importance are representatives of two orders: chainworms - Cyclophyllidea and tapeworms – Pseudophyllidea, belonging to the subclass of true tapeworms (Eucestoda).

In the uterine stage, cestodes live in the digestive organs of vertebrates.

Their body is usually ribbon-shaped, flattened in the dorsoventral direction, consists of a head (scolex), a neck and a strobila consisting of segments (proglottids). The length of the entire cestode, depending on the species, ranges from a few

millimeters to 10 meters. The number of proglottids can be from one to several thousand.

Scolex in the form of a compact formation of iemmt has a variety of shape, size, structure. The scolex chains are usually more or less rounded in shape with 2-4 suckers with muscular walls, which can be armed with hooks. At the top, the scolex is often equipped with a special muscular outgrowth-a proboscis, carrying weapons in the form of one or more rows of hooks. The number, size and shape of hooks, the ratio of the length of their morphological elements (blades, handles and root process), as well as the nature of the location of hooks are essential in the species diagnosis of chains. In lentets, the scolex is elongated and equipped with two or four suction pits (botria). Behind the scolex there is an unsegmented part of the body - the neck – the growth zone, in which the segments are formed. Since new segments are formed between the neck and the preceding segment, the older segments are moved to the rear, and the youngest segment is located at the neck.

The body of the cestode is covered by a musculoskeletal layer consisting of a cuticle, a basement membrane and a subcuticle. The muscle layer is located under the subcuticular layer, and the entire space between the internal organs is filled with parenchyma.

The cestodes have well-developed nervous, excretory and genital organs, but the cestodes have no digestive, respiratory and circulatory organs.

The nervous system consists of several nerve nodes located in the scolex, and longitudinal trunks extending from it, which pass through the entire body of the cestode and are interconnected by transverse branches.

The excretory system is built in the same way as in flukes.

Ribbon parasites do not have a morphologically expressed digestive system, and they feed by sucking food with the entire surface of the body.

Genitals. The youngest segments are honest asexual. As they develop, the male reproductive system (male segments) begins to form in them, and then the female one. The segments in which the male and female reproductive systems are fully developed and functioning are called hermaphrodite. After cross-fertilization or self-fertilization of such segments, the male genitals degenerate, the female ones develop as much as possible and the segments become female. Finally, all parts of the female reproductive system degenerate, and in the last segments there remains one uterus filled with fertilized eggs. Such segments are called mature. Mature segments are released into the external environment with the feces of the host, either one by one, or in the form of scraps of strobila. Eggs are released after the destruction of a mature segment in the intestines of the host or in the external environment (cepni) and through independent excretory opening of the uterus (lentets).

The male reproductive system consists of one or more, and more often of numerous testes, from which the vas deferens flow into the vas deferens. The latter forms a seminal vesicle and ends with a cirrus surrounded by a sexual bursa.

The female reproductive system consists of the ovary, the oviduct, the yolks, the body of the Melissa, the ootype, the vagina and the uterus. The ovary is often single and lobed. It is connected to the ootype by the oviduct, the latter is surrounded

by the glands of the taurus Melis. The female genital opening leads to the vagina, the narrow duct of which forms the seminal receptacles flows into the ootype, where the yolk duct opens. The yolk is usually compact in the chains, and in the tapeworms it is divided into follicles. The uterus originates in the ootin. It can have an independent outlet opening through which fertilized eggs (tapeworms) are released, or it can be closed and takes a different shape as it is filled with eggs (tapeworms).

Male and female genital openings usually open side by side in the area of the genital tubercle from the lateral surface of the segment (cepni) or on its wind surface (lentets), as a result of which individual segments often have self-fertilization.

The uterine wall is more tenacious sometimes forms special formations. These formations can be of two types:

1. Egg capsules that are formed by the uterus itself,

2. Paruterine organs, which are formed due to denser areas of the parenchyma near the uterus, into which eggs fall or they are formed due to uterine extensions, in which eggs remain after degeneration of the body of the organ itself, or eggs can fall from these extensions into fibrous formations formed by the parenchyma, the so-called egg cocoons. Regardless of the method of formation, egg capsules, egg cocoons and paruterine organs play a protective role for fertilized eggs.

Chainworm eggs are rather monotonous in their morphology, so it is practically impossible to determine the species by their structure. Mature eggs are oval, rounded or spherical in shape, covered with an extremely delicate transparent shell. Through which the oncosphere inside shines through well. The oncosphere consists of a thick radially striated shell (embryophore), inside which there is an embryo with six embryonic hooks, surrounded by its own, tightly fitting shell.

Oncospheres are more often colorless, less often colored in yellow and yellow-brown colors. The egg shell is rapidly destroyed and oncospheres are detected when examining feces.

In the representatives of the order of the sluggards, the eggs have a lid like the eggs of flukes.

All cestodes are biohelminths, and their development occurs with the participation of one (cepni) or two (lazy) intermediate hosts. In the body of intermediate hosts, the larva of various cestodes have a diverse structure.The main types of these larvae in the tapeworms are: cysticercus, cenurus, echinococcus, alveococcus, strobilocercus, cysticercoid and tetratidium, and in the tapeworms – procercoid and plerocercoid.

The development cycle. Tsepni, as it was noted, develop with the participation of two hosts: definitive and intermediate. From the oncospheres that have got into the digestive tract of the intermediate host with food or water, the embryo is released, which migrates with blood, getting into various internal organs, where, depending on the type of cestode, it develops into the appropriate type of larva. Some of these lavocysts (for example, cenura, echinococci, alveococci) in an intermediate host are able to reproduce asexually. Definitive hosts become infected by eating organs or tissues or by ingesting intermediate hosts containing an invasive larvocyst.

As for the sluggards, their development can be reduced to five phases: 1- an egg whose embryogenesis occurs in water; 2- a coracidium hatching from an egg and leading a free-swimming lifestyle; 3- a procercoid developing from a coracidium in the body of paddlefoot crustaceans; 4- a phase, a plerocercoid developing from a procercoid in fish and a 5–adult cestode developing from a plerocercoid in the intestines of warm-blooded animals.

The causative agents of cestodoses of farm animals belong to five suborders of chains.

Taeniata (Taeniata) - with a single family Taeniidae, as a rule, large species, with the length of mature segments exceeding their width. The proboscis is usually armed with large hooks arranged in 2 rows, forming a so-called crown. The uterus is in the form of a longitudinal trunk with numerous branches, sac-shaped or spherical. In the ribbon stage, cestodes parasitize in the intestines of mammals (carnivorous carnivores or humans), larvocysts (cysticercus, cenurus, echinococcus, alveococcus and strobilocercus) - in various organs and tissues of mammals.

Anoplocephalates (Anoplocephalata) are also large cestodes, but they have an unarmed scolex without a proboscis and hooks, and the segments are elongated in the transverse direction. Adults parasitize in the intestines of mammals (artiodactyls, ungulates and rodents), larvocysts of the cysticercoid type –in arthropods.

Hymenolepidates (Hymenolepidata) are small and medium–sized cestodes, the scolex is equipped with a proboscis and hooks, often arranged in one row. Ribbon stages live in the intestines of birds and mammals, larvocysts of the cysticercoid type live in the body of various invertebrates.

Davaineata (Davaineata) are small or medium–sized cestodes, the proboscis of the scolex is armed with hooks of a characteristic axe-shaped shape, arranged in one or more rows. Adult cestodes usually parasitize birds, larvocysts of the cysticercoid type – in mollusks and arthropods.

Mesocestoidates (Mesocestoidata) are medium–sized cestodes with a chkolex, without a proboscis and hooks. The sexual openings are located along the middle laziness of the segments, the eggs contain the oncosphere and are folded into the paruterine organ. Adult cestodes parasitize in the intestines of predatory mammals and birds.Development takes place with the participation of two intermediate hosts. Cysticercoid is formed in the body of oribatid mites, and tetratidium is formed in an additional (various vertebrates) host.

Diseases caused by cestode larvae. teniidoses.Teniidoses unite a large group of widespread and clinically diverse helminthiasis of rural, domestic, commercial and wild animals caused by numerous representatives of the Taeniata suborder.

The causative agents of teniidoses are both ribbon – imaginal and larval – larval, cestode stages. Therefore, imaginal and larval teniidoses are distinguished. Among teniidoses there is a large number of anropozoohelminthiasis, but the degree of human participation in the spread of these invasions varies. With some anthropozoonoses (teniosis, tenirinhosis), a person serves as an obligate host, he is the only source of infection of animals with larval cestodoses (cysticercoses). On the other hand, there are anthropozoonoses in which a person is an intermediate host and

does not participate in the spread of invasion (echinococcosis, alveococcosis, cenurosis), and predatory mammals act as obligate-final hosts. Taking into account the specific weight of individual links in the biological cycle of teniidosis pathogens in epizootology and epidemiology, these invasions can be divided into two groups:

1. Teniidosis, in which a person is the final host of pathogens.

2. Teniidosis, in which the dog and other predatory mammals are the final host of pathogens.

Teniidosis, in which a person is the final host of pathogens. Two anthropozoonoses belong to this group of teniidoses: bovis cysticercosis and cellulose cysticercosis. These helminthiasis are caused in animals by larval stages of teniids, while sexually mature cestodes are parasitic in humans.

CYSTICERCOSIS (FINNOSIS)

CATTLE

Cattle cysticercosis is caused by Cysticercusbovis, the larval stage of the cestode Taeniarhynchussaginatus, which is parasitic in the human intestine. Cysticerci are localized in the intermuscular connective tissue of the skeletal muscles, heart, tongue, less often in the tissues of parenchymal organs.

Distribution. Cattle cysticercosis is widespread everywhere, and in our Republic about 10% of the examined animals with a rare disease are infected with cysticercosis, of which about one percent are culled. Thus, the disease occurs in cattle farms from 2.6 to 8.9%, and in livestock complexes – from 1.3 to 10.3%. In Karakalpakistan, cattle are infected with an average of 7.7%. Samarkand -7.3%, Bukhara -9.8%, Kashkadarya -9.3%, Ferghana - 12%, Navoi -11.5%, Namangan - 13.5%, Surkhandarya 10.3%.

In Uzbekistan, when examining the population for infection with teniarinhosis, 24 out of 10,000 people were infected with teriarinhosis.

Economic damage. Cysticercoses cause significant economic damage due to the rejection of severely affected carcasses, a decrease in the quality of the infected meat and the cost of its disinfection. Bovine and pork tapeworm parasitizing in the human intestine, significantly reduce his ability to work, slow down his development. In addition, cysticerci of pork tapeworm, parasitizing in the brain and eyes of a person, can cause significant visual and mental disorders, and sometimes cause death.

The pathogen. Cysticercusbovis is a grayish-white bubble, transversely oval in shape, 5-15 mm long, 3-8 mm wide. It consists of two shells, the outer one is connective and the inner one is germinative. On its inner shell there is a large scolex, 1,5-2 mm in diameter, equipped with four suction cups.

The sexually mature stage. Taeniarhynchussaginatus reaches 10 m (or more) in length and 12-14 mm in width, the scolex is unarmed, large. Proboscis in the form of a rudimentary formation resembling an apically located sucker. The diameter of the suction cups is 0,8 mm. Sexual openings alternate incorrectly, the ovary is two-lobed. The blades are rounded in shape, equal in size, there is no seed collector. In mature segments, the uterus is in the form of a medial trunk, from which 18-32 lateral branches branch off to the right and left. There are 2 long filaments on the outer shell

of the egg. Oncospheres have a thick two-contour, radically striated shell of yellow-brown color, 0,03-0,04 mm long and 0,02-0,03 mm wide.

Biology of the pathogen. The parasite is a biohelminth.The main-definitive host is a human, and intermediate hosts are cattle, buffalo, yak, zebu and reindeer.

From the human intestine invaded by Taeniarhynchussaginatus, mature segments are released into the external environment passively with excrement, or actively crawling out of the anal opening. Infection of intermediate hosts occurs when they swallow oncospheres and, in some cases, proglottids of teniarynchus.

In the intestines of cattle, the embryo leaves the egg and, with the help of six hooks, is introduced into the capillaries of the intestine, and in the future, it can be hematogenically introduced into any organs, where an invasive cysticercus is formed after 4.5 months. Cysticerci develop mainly in the intermuscular connective tissue, in some cases they reach invasiveness in the subcutaneous tissue, adipose tissue, brain, liver, lungs, heart in the eyes of cattle, in the brain and cerebellum of reindeer.

A person becomes infected with teniarynchosis when eating cattle meat or the brain of reindeer affected by cysticerci.This is usually observed in the process of cooking, when eating dishes from raw or insufficiently cooked meat (stroganina, shish kebab, basturma, steak, etc.). When cysticerci enter the human digestive tract, they, under the influence of gastric juice and bile, boil out the scolex, which is glued to the intestinal wall with suckers. In the future, the parasite grows and reaches puberty after 3 months. The lifespan of teniarynchus is more than 10 years.

J.K.Strom, as a result of long and careful observations, came to the conclusion that the rate of departure of proglottids is kept at the same level in the amount of 6-8 segments per day, that is, about 2500 proglottids are isolated per year by a person with teniarynchosis. If we take into account that the number of oncospheres contained in one proglottis reaches 145-175 thousand, then in the presence of even one cestode, the helminth carrier can allocate from 175 thousand to 4 million 900 thousand oncospheres per day, during the year – about 440 million.

Epizootological data. The intensity of invasion in cattle is usually weak, Calves are more susceptible to cysticercosis than adult animals, and intrauterine infection is often observed. Cysticercus and from different geographical zones differ both in survival rate and in the duration of life in the body of cattle, which gives reason to assume the existence of different geographical strains of Cysticercusbovis. The invasion of animals occurs under unsanitary conditions of livestock, as well as if the maintenance staff does not use toilets.

Pathogenesis. The helminth has a particularly strong pathogenic effect during migration, and fully formed cysticerci do not have a noticeable pathogenic effect.

Immunity. Parasitization of C.bovis in the body of cattle causes the development of relative immutability, manifested by a decrease in survival rate and a shortening of the life of cysticerci. Repeated infection increases the tension of immunity. Young calves, after the interruption of cystic leukemia, acquire low-voltage immunity and are able to become infected again, adult animals are resistant to re-infection.

Symptoms of the disease. Clinically, cysticercosis of cattle is not manifested. When experimentally infecting calves, many scientists observed severe pain in animals during the mirgation period with signs of acute general disease and fatal outcome.

Pathoanatomic changes. In acute cysticercosis, massive spot hemorrhages are detected on the subcutaneous tissue, muscles, and heart. Small gray-colored bubbles-cysticerci are visible in the muscles.

Diagnosis. During his lifetime, various methods of immunological diagnostics were tested, but they have not yet entered into widespread practice, so the reaction of latex agglutination requires refinement in terms of increasing sensitivity and the use of a more specific antigen.

Postmortem diagnostics is based on the detection of cysticerci in cattle carcasses during post-slaughter examination.

The luminescent method significantly increases the percentage of detection of cysticerci in meat.

Treatment. Panacur (22.2% fenbendazole granulate) in a total dose of 0.05 g / kg of ADV weight and droncit (praziquantel) in a total dose of 0.01 g / kg of ADV when used for 2 consecutive days individually with a small amount of moistened feed.

However, due to the lack of sufficiently reliable methods for the diagnosis of cysticercosis, treatment for this invasion is still practically not used.

PIG CYSTICERCOSIS (CELLULOSE)

Porcine cysticercosis is an anthropozoonosis caused by Cysticercuscellulosae, the larval stage of the Taeniasolium cestode parasitizing in the human intestine. The cysticerci themselves are localized in the muscles, heart, brain, eyes, liver and lungs; in humans – in the brain and eyes.

The pathogen. Larval stage.Cysticercuscellulosae are transparent ellipsoid–shaped bubbles, measuring 6-20 mm in length and 5-10 mm in width. The bubble wall is two-layered, filled with a slightly opalescent liquid, in which there is a scolex attached to the inner shell. The scolex is screwed in, shines through the wall of the mound in the form of a white speck, and has the same structure as the scolex T. Solium.

The ribbon stage - Taeniasolium does not exceed 3 m in length. The Skolex is armed with a double crown of hooks, the number of which ranges from 22 to 32. The hooks of the large row are 0.16-0.18 mm long, and the small one is 0.11-0.12 mm long.Sexual openings alternate incorrectly. A characteristic feature of hermaphrodite segments is the presence of an additional third lobe of the ovary. In the mature segment, 7-12 lateral branches depart from the median trunk of the uterus on each side, forming, in turn, a small number of blades.

Biology of the pathogen. The parasite is a biohelminth. The definitive owner is only a person, and the intermediate owners are a domestic pig, wild boar, bear, camel, dog, cat, rabbit, hare, and also a person.

Man is the only definitive host that periodically releases mature segments with feces. In the external environment, the segments make active movements, while the eggs are pushed out of the uterus through the destroyed edge of the proglottode.

Intermediate hosts become infected by ingesting Taeniasolium eggs with food or water. In the intestines of pigs, the oncosphere comes out of the egg, its shell is destroyed and the embryo penetrates into the crown or lymphatic vessels of the intestinal wall, then they are usually brought with blood into the intermuscular connective tissue, brain, eyes and other organs and after 2-4 months they finish their development. The lifespan of cysticerci in pigs is 3-6 years, after which they shrivel, become soaked in lime and die.

It should be taken into account that a person for Taeniasolium is not only a definitive, but also an intermediate host.Human infection with cysticerci occurs in two ways: either by ingesting oncospheres with food, or by internal self-infection. Therefore, persons infected with the ribbon stage are always at risk of cysticercosis.

The final development of the parasite occurs in the human body, which becomes infected by ingestion of cysticerci located in undigested or unroasted meat. In the human gastrointestinal tract, the membranes of the bladder are digested, and the scolex is turned out in the 12-person intestine, which causes bile, then the scolex attaches to the intestinal mucosa, burrowing into it with its hooks begins to grow and after 2-3 months reaches the sexually mature stage and mature segments are released out. The lifespan of Taeniasolium in the human body is calculated in years.

Epizootological data. Cysticercosis of pigs is registered in Ukraine, Belarus, the central regions of the Non-Chernozem zone. The source of infection of animals with cysticercosis is a person infected with pork tapeworm. Due to the long life of the parasite, one patient can infest a large area with eggs. This is facilitated by the lack of well-maintained toilets, their unsanitary condition. Pigs willingly eat human feces, swallowing in place of si and tapeworm larvae. Isobaki are easily invaded, which are also characterized by coprophagia. Taeniasolium eggs can be brought into the pasture by the wind, on the shoes of service personnel, on the feet of animals, insects, etc.

Wastewater and pastures in irrigation fields will play a special role in the spread of cysticerco.

Pathogenesis. Mechanically, toxic and open the gate to secondary infections. The degree of pathogenic action depends on the localization of cysticerci and the intensity of invasion.

Symptoms of the disease. Pig cysticercosis is usually asymptomatic, with severe invasion, edema, cachexia, epileptic seizures develop, the disease can end in death. When localized in the brain - nervous phenomena, in the eyes – blindness may occur.

Pathoanatomic changes. The tissues are compressed and in a state of atrophy, with intensive invasion, the muscles are degenerated, watery, sprouted by connective tissue.

Diagnosis. It is difficult to establish cysticercosis in pigs during their lifetime. In recent years, they have been trying to diagnose the disease using

immunobiological methods that have not yet received practical application, a postmortem diagnosis is made based on the detection of cysticerci in muscles and various internal organs.

Prevention of cysticercosis of cattle and pigs. Measures to combat cysticercosis of animals, in which a person is the only definitive host, that is, the source of infection of farm animals, are unthinkable without coordinated actions of veterinary and medical organizations. Economic and public institutions, broad strata of the entire population should take an active part in the implementation of these measures. However, despite the closest contact and interconnection in the conduct of events, veterinary and medical science and practice have their own specific tasks that need to be solved, based on the specific conditions of the struggle, to take into account the peculiarities of the epidemiology of teniarynchosis and teniosis.

Veterinary measures are aimed at preventing human infection with teniosis and theriariasis, which is ensured by conducting a thorough veterinary examination of cattle and pig meat and preventing the possibility of infestation of animals with cysticercoses.

In accordance with the current rules of the vet.san.examination of meat and meat products, carcasses of cattle and pigs are subjected to a special examination for cysticercosis. They check the meat of animals of all ages. For this purpose, first of all, the external and internal life-giving muscles, as well as the heart, are opened. They probe the tongue, making incisions in doubtful cases. In the presence of cysticerci, two parallel incisions are additionally made in cattle of the neck muscles, deep lumbar muscles and diaphragm, and in pigs – lumbar, occipital muscles and diaphragm. In addition, the scapular, elbow, spinal and pelvic limb muscles are cut and examined.

If more than three living or dead cysticerci are found on an area of 40 cm2 of the muscles of the head and heart and on one of the cuts of the muscles of the carcass, the entire carcass (except the intestines) is sent for technical disposal or destroyed. The internal fat is melted for food purposes.

If more than three live or dead cysticerci are found on the same area of cuts of the muscles of the heart and head, and in the absence or presence of no more than three cysticerci on the remaining sections of the above muscles, the head and heart are sent for disposal, and the carcass and other organs (except the intestines) are disinfected by boiling, freezing or salting, followed by use for the preparation of minced sausage products or canned minced meat. The decontaminated sub-products are sent for industrial processing. Internal fat is melted.

Farmyard slaughter is prohibited, for which, along with the construction of meat processing plants, slaughterhouses, meat control stations, the network of slaughterhouses and sites in rural areas is being expanded, so that all miao and all organs from livestock killed in these conditions undergo veterinary and sanitary control. Cysticercose carcasses should be disinfected only with the knowledge of veterinary supervision. At slaughter sites, markets organize points for the disinfection of conditionally fit and cysticercose meat, Carcasses affected by cysticerci are strictly forbidden to return to the owners.

All cases of detection of cysticercosis in animals are immediately reported to the relevant medical organizations (district health departments, city health departments) for examination and carrying out therapeutic and preventive measures among persons who are the source of infection of animals with cysticercosis. To do this, it is necessary to organize an accurate accounting of slaughtered cattle. Mandatory labeling of all animals entering for slaughter will make it possible to identify foci of cysticercosis of cattle and pigs and at the same time signal this to the appropriate veterinary and medical organizations.

Considering that the source of human infection with teniosis can be the meat of not only pigs, but also wild boars, camels, roe deer, bears, hares and rabbits, and teniarinkhoz – meat of cattle, zebu, yaks, the brain of deer, carcasses of these animals and deer heads should be subjected to vet.san.examination for exceptions of cysticerci.

The main condition providing for the infection of cattle and pigs with cysticercoses is the proper maintenance and feeding of animals. They do not allow vagrancy of animals. The admission of livestock to the territory of human habitation must be closed.

A complex of medical measures. The measures that are leading in the complex of combating these helminthiasis should be aimed at improving the health of the population from teniosis and teniarynchosis, protecting the external environment of its invasion by proglottids and oncospheres of teniids, which prevents infection of intermediate hosts with cysticercoses. First of all, it is necessary to exclude the ingress of bovine and pork tapeworm segments into pastures, livestock premises, watering sources, feed storage sites, and to prevent contact with cysticercosis, with human experiments. To do this , the following is necessary:

1. Systematic examination of people in order to identify all patients with teniosis and teniarynchosis, followed by their mandatory deworming, which should ensure the complete removal of teniids from the human body. First of all, these activities should be carried out among livestock workers (shepherds, reindeer herders, milkmaids, pigs, stockmen, etc.) as well as among the rural population in livestock areas and food industry workers. During deworming, the isolated teniids and feces containing fragments and oncospheres of cestodes should be completely destroyed.

2. Systematic protection of soil and water bodies from contamination by human feces. Arrangement of public and individual toilets, systematic monitoring of their sanitary condition. Conduct the most careful monitoring to ensure that the population uses toilets, preventing the dispersion of excrement in the external environment.

3. Sanitary control over the disposal of human excrement and the state of wastewater, excluding the possibility of the spread of invasion.

4. Compliance with personal preventive measures by a person. Do not eat raw, unboiled or fried meat and internal organs. During cooking, be careful when processing raw meat in order to avoid accidental ingestion of cysticerci on kitchen items and other products. Since a person is not only the final, but also the intermediate host of pork tapeworm, thorough general hygienic measures are necessary to exclude the possibility of ingestion of oncospheres by a person.

Sanitary and helminthological education of the population. Sanitary and educational work should be systematically carried out among the general population by both veterinary and medical and school workers. In order to involve the population in the active fight against helminthiasis, they use all the opportunities that are provided in specific conditions (lectures, conversations, speeches in print, on radio and television, demonstration of popular science films, publication of popular science literature, the release of posters, leaflets, etc.). Special seminars are systematically organized for veterinary and medical workers, first of all it is necessary in areas that are disadvantaged by teniidoses.

PIC No. 7. ECHINOCOCCOSIS AND CENUROSIS OF ANIMALS.

Lecture plan:

1. Echinococcosis
2. Cenurosis

Keywords: Echinococcus granulosus larvae (E.unilocularis), single-chamber bladder, biohelminth, Echinococcus veterinorum, Echinococcus hominis, Echinococcus acephalocysticus, Taeniidae, Multiceps multiceps, Coenurus cerebralis, germinative, cuticular membrane, planned deworming, anthelmintics.

ECHINOCOCCOSIS is an anthropozoonosis, a cestodosis disease that usually occurs asymptomatically in sheep, goats, cattle, pigs, camels, less often horses, donkeys and other mammals that are intermediate parasite hosts. Echinococcosis also affects a person, sometimes with a fatal outcome. The disease is caused by the larval stage of the cestode from the family.Taeniidae-Echinococcusgranulosus, localized in the liver, lungs, less often in other parenchymal organs and tissues.

The ribbon stage of echinococcus parasitizes in the small intestine of dogs, wolves, jackals and less often foxes (definitive hosts).

Distribution. The disease is widespread almost everywhere, especially it is registered in the southern regions of the former Soviet Union, in Central Asia, Kazakhstan, Siberia.

In our Republic, echinococcosis is spread on average from 26 to 57%., so, 38.6% of slaughtered cattle are infected with echinococcosis, sheep – 57-76%, goats – 12%, pigs - 16%, camels -9%, horses -12%.

Of the 10,000 people examined, 6-9 respond positively. An average of 350-360 people are operated on annually in the Samarkand City Hospital for echinococcosis.

Economic damage. Echinococcosis occurring chronically, invasion significantly reduces productivity in animals, causes a delay in the growth and development of young animals, reduces the resistance of animals to other diseases. During vet.san.examination of internal organs, organs affected by echinococcosis, and sometimes whole carcasses are destroyed, as well as vet. expenses for preventive measures.

The pathogen. Echinococcusgranulosuslarvae (E.unilocularis) is a single–chamber bubble filled with fluid. Echinococcal fluid is a product of the blood of an

intermediate host, it plays the role of a protective and nutrient medium for scolexes. The wall of the bladder consists of two shells: the outer cuticle and the inner germinal.

The cuticle shell is milky-white, sometimes with a slightly yellowish tinge, in older bladders it becomes cloudy, taking on a jaundiced appearance.

The germinal or germinal membrane lining the inside of the bladder cavity is thin, delicate, and represents a kind of embryonic tissue capable of producing brood capsules with the simultaneous formation of embryonic scolexes and secondary (daughter) bladders in them. Sometimes the elements of this shell penetrate between the layers of the cuticle to the outer surface, which creates conditions for the exogenous development of secondary bubbles.

Brood capsules are outgrowths of the germinal shell, where germinal scolexes are formed in the future.

Sometimes secondary (daughter) bubbles are formed inside larvocysts, and in them, in turn, tertiary (grandchild) bubbles, where brood capsules and scolexes can also develop. These bubbles form not only in the cavity of the maternal bladder (endogenously), but also outside it (exogenously) with further budding.

The size of echinococcal bladders can be from evda visible to the head of a newborn child, the shape of the bladders is usually round, although it may vary depending on the organ and localization in it. The number of bladders in individual animals ranges from single to several tens, and sometimes even thousands of specimens.

Outside, the echinococcal bladder is covered with a connective tissue capsule in the form of a powerful fibrous formation formed as a result of chronic inflammation from host tissues. It is dense, relatively thin, is a grayish-white shell that adheres to the cuticle of the parasite and repeats the shape of the latter.

In the organism of the intermediate host, morphological modifications of the echinococcal bladder are distinguished, which have received many diverse names.

Echinococcus veterinorum. Bubbles of this modification are characterized by the presence of brood capsules with scolex and echinococcal fluid, secondary and tertiary bubbles do not develop either inside or outside the maternal larvocyst. In cattle and sheep, they make up the main mass of larvocysts. This form of echinococcus is also recorded in humans.

E.hominis are characterized by the fact that in addition to echinococcal fluid, brood capsules with scolex, they also develop secondary (daughter) and tertiary (grandchild) bubbles.

E.acephalosysticus. The acephalocyst is characterized by the fact that there is only liquid in the echinococcal bladder. These are larvocysts in which daughter and grandchild bladders can develop, but in them, as in the maternal bubble, there are no brood capsules and scolexes. It suggests that acephalocysts are formed as a result of creating unfavorable conditions for the development of the parasite in the host body, and this is considered as one of the forms of manifestation of immunity in animals against echinococcus.

Echinococcus granulosus- echinococcus in the ribbon stage is a very small cestode, the strobila of which has a length of up to 9 mm and consists of 3-4 segments. The mature segment usually exceeds the rest of the strobila. The Scolex is equipped with a proboscis armed with 36-40 hooks. The hollow opening is located in the posterior half of the lateral edge of the segment. In mature segments there is a uterus in the form of a longitudinal trunk with lateral protrusions. Oncosphere with a radially striated shell, its diameter is 0.030-0.040 mm.

Biology of the pathogen. The final hosts (dogs, wolves, jackals, foxes) in a place with experiments secrete mature echinococcus segments into the external environment, which contain a uterus filled with eggs. The number of eggs in a mature segment averages about 800, and sometimes reaches several thousand. In the external environment, echinococcus segments are actively moving, they crawl out of experiments, spreading to a distance of 15-20 cm and sometimes climbing up the stems of plants. During the movement, their walls are torn and the eggs stand out.

Intermediate hosts are infected by ingesting oncospheres or mature segments of the parasite together with food or water. In the stomach, the shells of the oncospheres are destroyed, the embryo is embedded in the intestinal wall, reaches the capillaries and with blood or lymph they are introduced into various organs and tissues, where they form very slowly into a single-chamber bubble. The larvocyst of echinococcus reaches the invasive stage no earlier than 6 months later and depends on the type of intermediate host. So,....

Dogs and other carnivores become infected by eating organs affected by echinococcal blisters, which are localized most often in the liver and lungs. In dogs, the prepatent period of parasite development lasts on average 2-3 months and depends on the season of the year and the intermediate host.

The life expectancy of echinococcus in the dog's body is 5-6 months and also depends on the season and the intermediate host.

Epizootological data. Despite the wide range of intermediate hosts, sheep (mostly adults) are of the greatest importance in epizootology and epidemiology of echinococcosis.The case of animals is more common in winter and spring. The source of infection with echinococcosis of animals and humans, as a rule, are dogs, primarily neglected, priotarny and shepherds. People become infected with echinococcosis through direct contact with dogs affected by echinococci, or by eating vegetables and fruits contaminated with echinococcus eggs.

Pathogenesis. Echinococcus larvae have mechanical, toxic and allergic effects. Sometimes they occur in huge numbers, reach large sizes, put pressure on the affected organ and cause atrophy of adjacent tissues. Depending on the localization, number and size of echinococcal bladders, various disorders occur in the body of the intermediate host, both local and general.

Immunity in different animals is expressed to varying degrees. In pigs, compared with cattle, it is weak, in sheep – of considerable tension.

Symptoms of the disease. For a long time after infection, echinococcosis proceeds without pronounced signs. The general condition and fatness of the animals are considered satisfactory. In the future, clinical signs gradually appear, which

depend on the location and the number of bubbles. In most cases, severe exhaustion occurs, the productivity of animals decreases, sheep wool becomes disheveled, often falls out, cows reduce milk yield. With liver damage, digestive disorders develop, the area of hepatic bluntness increases, palpation of this area is painful. With echinococcosis of the lungs – cough, difficulty breathing. Animals can die from cachexia during intensive invasion.

Pathoanatomic changes. Echinococcal vesicles are usually found in the liver and lungs, less often in the kidneys, spleen, sometimes they are located near the surface of the organ, protruding above its serous membrane. With intensive invasion, the affected organs significantly increase in size and weight, the tissue of these organs atrophies. Sometimes echinococci are located inside the lungs or liver and then they can be detected by probing.

Diagnosis. According to the clinical picture, it is difficult to diagnose echinococcosis. Fluoroscopy is not widely used. Sometimes immunobiological methods are used, which are very promising. For example, scolexo-precipitation reactions, RNGA, RDSK, Kazoni reactions.

It should be borne in mind that when opening fallen animals and finding only single specimens of parasites, they cannot be considered the cause of death.

Treatment of animals with larval echinococcosis has not been developed.

CENUROSIS (PINWHEEL) is a disease of sheep and goats caused by the larval stage (Coenuruscerebralis) of the cestodyMulticepsmulticeps. Cattle, yaks, rarely camels, pigs, horses, wild ruminants and very rarely humans also suffer from cenurosis. Cenures are localized in the brain and less often in the spinal cord. In the ribbon stage, it parasitizes in the small intestine of dogs, wolves, jackals, foxes and arctic foxes.

Distribution. The disease is widespread everywhere, especially often it is registered in sheep and goat farms. Every year, about an average of 8-12% of lambs of the current year of birth are infected with cenurosis.

Economic damage. Cenurosis causes significant economic damage to the farm, which consists of the mass death of sheep and forced cutting of sick animals. Carcasses of sick animals are severely depleted and are sent for technical disposal. As well as veterinary expenses for preventive measures.

The pathogen. Coenuruscerebralis has the appearance of a bubble up to 10 cm in diameter, round or oval in shape, the size of which depends on the degree of development, localization and type of animal. The bubble wall consists of two layers. On the inner – germinative translucent shell, scolexes are located in separate groups close to each other. Their number increases as the censorship develops, sometimes there are several tens and hundreds of them. In their structure, they are identical to the scolex of the sexually mature stage.

Multicepsmulticeps reaches up to 1 m in length and consists of 200-250 segments with a maximum width of about 5 mm. The poorly developed proboscis is equipped with 22-32 hooks arranged in 2 rows. There are up to 200 testes in the hermaphrodid segment, located mainly in the middle part of the segment. In mature

segments, 9-26 lateral branches depart from the median trunk of the uterus on each side.

Biology of the pathogen. The parasite is a biohelminth. Intermediate hosts of the parasite are sheep, goats, cattle, less often pigs, horses, etc.

With the excrement of definitive hosts (mainly priotarny and shepherd dogs), mature segments of the multiceps are allocated, filled with eggs and fall on the grass, feed, litter and into the water. Eggs are very resistant and can remain viable in the external environment for a long time.

Intermediate hosts become infected on pastures by ingesting parasite eggs together with food or water. In the intestines of animals, the shells of eggs and oncospheres are destroyed and the embryo is embedded with its hooks into its wall, and then penetrates into the blood vessels and spreads through the body with blood. After reaching the brain or spinal cord, it loses its hooks and begins to grow, developing into a bubble-shaped larva. The bubble grows slowly, after a month, scolexes begin to form in the censorship, and later the rudiments of hooks appear in them. Invasive censures can be detected after about 3 months.

Dogs, wolves, foxes, eating the brains of sheep with cenurosis, become infected with multiceptosis. In the intestines of the final hosts, as many parasites are formed as there were scolexes in the bladder. The prepatent period of Multicepsmulticeps development in a dog is 40-50 days, and the cestode develops faster in puppies than in adult dogs. The lifespan of multiceps in the body of dogs is on average 6-8 months.

Epizootological data. Cenurosis is widespread, it is very often diagnosed in the North Caucasus, Siberia, the Lower and Middle Volga, the Urals, Kazakhstan, the republics of Central Asia, less often in Ukraine and Transcaucasia.

Cenurosis affects animals up to one year old, less often sheep older than two years. Animals die from cenurosis at all seasons. Parasite eggs are very resistant to external influences. The main source of infection with cenurosis of animals are priotarny and shepherd dogs. Wolves, jackals do not play a big role in the epizootology of cenurosis.The fox is an optional host for the parasite, i.e. oncospheres do not have an invasive ability.

Pathogenesis is caused by both mechanical action and intoxication. Parasites act mechanically during the period of exit from the capillaries and introduction into the brain, as well as developing censorship. Which put pressure on the brain and the bones of the skull.

Immunity. With cenurosis, age-related immunity is sharply expressed. Besides. Sheep have individually expressed immunity, since not all animals that have swallowed Multicepsmulticeps eggs become infected and get sick. Lambs are the most susceptible to the disease.

The symptoms of the disease in sheep cenurosis depend on which part of the brain the bubble is localized, which of the brain areas is most changed by the number of bubbles and their size, as well as on the physiological state of the animal. The symptom complex of cenurosis, in accordance with the biology of the parasite, consists of three stages.

The first stage is caused by the migration of the embryo from the intestines of the animal to the brain. Sheep are in a state of depression or excitement, note timidity, convulsive twitching, aimless movements. The mucous membranes of the eyes are hyperemic. Sometimes on the 4th-5th day after the first symptoms of the disease appear, sheep die. In the acute course of cenurosis, the peculiarity of clinical signs and the rapidity of their increase make them vet.workers can anticipate an infection and make a variety of diagnoses.

The second stage is determined by the formation and slow growth of the price tag and lasts 2-6 months. There are no clinical manifestations of the disease, the sheep seem healthy in appearance.

The third stage is characterized by a sharp deterioration in the general condition of sheep. Animals sometimes refuse to go into a coma, suddenly stop, stand for a long time with their heads down, rest their forehead on an object, do not react to their surroundings, often make manege movements to the side of the affected half of the brain. Coordination of movements is disrupted, sometimes sheep throw their heads, run forward or back away, tonic convulsions appear. In some animals, complete or partial loss of vision occurs. The painful sensitivity of the scalp is disturbed, the animals refuse to feed, gradually lose weight, weaken, lie more. In this state, the animal can stay 1-2 months. and then dies.

When the spinal cord is affected, a shaky gait is noted, later complete paralysis of the hind limbs develops, and the skin in the affected area loses sensitivity.

Pathoanatomic changes. When opening a fallen or killed sheep, one or more bubbles are found in the brain, the brain is so compressed that destruction and death of nerve cells occur in a large area, and sometimes atrophy of the skull bones.

The diagnosis is made comprehensively: based on epizootological data, clinical signs and animal studies. With percussion of the cranial bones at the location of the censor, bluntness is detected, palpation establishes thinning and deflection of the skull bones at the location of the censor.

An allergic intradermal test for sheep and cattle cenurosis (KazNIVI) has been developed.

Differential diagnosis. Most often, cenurosis must be distinguished from estrosis: sheep of all ages suffer from estrosis, whereas cenurosis usually affects lambs of the current year of birth. Circular movements of sheep with estrosis are not characteristic and are not performed in one direction; with estrosis, serous-mucous, purulent or bloody discharge from the nasal cavity is noted with the formation of crusts around the nostrils, with cenurosis, nasal discharge is not noted. Thinning of the cranial bones is detected only with cenurosis. Ophthalmoscopy makes it possible to differentiate cenurosis from estrosis, since with cenurosis there is a change in the form of a stagnant nipple in the bottom of the eye, and with estrosis, the fundus remains normal.

Sheep cenurosis should be differentiated from listeriosis, rabies, Scottish encephalomyelitis, bradzot, Aujesky's disease; from non–contagious diseases - poisoning with feed and some chemicals, functional disorders of the central nervous system, dropsy of the ventricle of the brain, trauma, etc.

Treatment. Surgical, but does not always give the desired result.

Measures to combat teniidosis, in which a dog and other predatory animals are the final hosts of pathogens (echinococcosis, cenurosis and cysticercosis).

The links of the biological cycle of development of pathogens of this group of cestodoses are as follows:

1. In the ribbon stage, the cestodes are localized in the small intestine of dogs and other predatory mammals;
2. Eggs (oncospheres) are preserved, depending on the type of pathogen and its area, for quite a long time in the external environment;

Larvocysts of cestodes live in different organs and tissues of a wide range of intermediate hosts - various mammals, mainly ungulates and rodents, and in some cases humans.

The principle of combating teniidosis consists in interrupting the biological chain of pathogens by the active accommodation of a person in order to destroy individual links or separate them. The success of the fight against teniidosis can be achieved by carrying out a set of measures at the level of modern helminthological science, taking into account local epizootological and epidemiological features. This complex consists of activities:

1. To prevent infection of farm animals with larval cestodoses;
2. To prevent infection of dogs and wild predatory mammals with the ribbon stages of teniid.

Measures aimed at preventing infection of farm animals with larval cestodoses.Fighting stray dogs. Trapping of stray dogs should be organized in all localities without exception.Both veterinary and medical workers, as well as police authorities, as well as the general public are required to take part in this event.

The order of keeping dogs. In each district, a detailed plan is being developed with the participation of local conditions regulating the maintenance of dogs belonging to both individual farms and private individuals. This plan is approved by the hakim of the district and brought to the attention of the persons responsible for their maintenance.

All economically useful dogs are registered, they are issued a passport and a license plate, as well as special cards where they make notes about the deworming carried out.

On the territory of the location of the flock, a minimum number (1-2) of dogs are left, and where economic conditions allow, they are generally released from dogs.

It is strictly prohibited to allow dogs that are in the personal use of citizens to enter the territory of livestock farms, slaughterhouses, meat processing plants, markets, in feed storage areas.

In areas that are unfavorable for echinococcosis, regions, edges of all service dogs of farms, including those guarding grain, and in rural areas and all dogs belonging to the population, it is necessary to undergo diagnostic deworming every 3 months. Dogs whose feces contain echinococcal segments are immediately isolated and dewormed.

Deworming of dogs should be planned and systematic, with mandatory coverage of all dogs of the farm, since even one non-dewormed animal can play a significant role in the spread of invasion.

Currently, it is recommended to use hydrobromic arecoline, phenasal, phenalidone, yomezan, droncite (praziquantel), azinox, cestel, cestan and others for deworming dogs.

There is no consensus on the timing of deworming of dogs, the number of deworming in teniidoses.Some authors recommend deworming dogs 4 times a year (quarterly), others - 2 times a year, and some researchers have come to the conclusion that in order to completely exclude larval teniidoses of priotarny dogs, it is necessary to deworm every 1.5 months. In areas that are unfavorable for teniidosis, dogs must be dewormed from December to April every 45 days, and from May to November – every 30 days.

Deworming is carried out on a specially designated (protected from the wind), fenced area. Veterinary staff work in protective clothing (closed dark robe, headdress, gauze bandage on the mouth and nose). No unauthorized persons are allowed on the site. The place from where the feces are collected is burned with a blowtorch fire for at least 1 minute.

Measures aimed at preventing infection of dogs and wild carnivores with ribbon stages of teniid. In order to prevent infection of the final hosts, it is necessary to slaughter animals only under strict vet.- sanitary supervision and in specially designated places. Vet.-san are organized in a number of farms.the blocks are 10x20 m sections fenced with a grid with concrete or iron posts, where there is a cemented platform for the degassing of dogs, a room for autopsy of corpses, a sectional table, a corpse incinerator and a water tank.

Animal organs affected by echinococci, censuses and cystic cells should not be released from the slaughterhouse without prior treatment, much less fed to dogs. The affected organs, depending on the degree of invasion and pathoanatomic changes, are subject to sterilization or disposal, the best way to destroy animal corpses is burning.

Once a decade, a clinical examination of sheep should be carried out to identify and isolate patients with cenurosis. All sheep with cenurosis are allocated to special groups for surgical treatment, promptly culled, preventing their death. Animals with echinococcosis and cenurosis are sent to slaughter in separate groups. A person, as you know, can get infected from dogs when the teniid oncosphere is swallowed, so it is necessary to observe the rules of personal hygiene.

The success of the devastation of larval teniidoses and other helminthioses is possible only with systematic, well-staged cultural and educational work. Local authorities, livestock workers and the general public should be involved in carrying out measures to combat larval teniidoses.

TOPIC No. 8. ANOPLOCEPHALATOSIS OF ANIMALS.

Lecture plan:

1. Anoplocephalatoses (moniesiosis, moniesiosis, avitellinosis, stillesiosis) of ruminants (we explain in order);

2. Anoplocephalidosis of horses;

Keywords: Anoplocephalata, Moniezia, M.expansa, M.benedeni, M.autumnalia,Avitellina centripunctata, Stillesiaglobipunctata, Thysanieziagiardi, oribatid(soil, shell) mites of the genus Scheloribates, hay eaters, eggs, oncosphere,, interglottid glands, pear-shaped apparatus, deworming, phenasal, copper vitriol, panacur (fenbendazole), rintal (febantel), fencur, fendaz

From the suborder **Anoplocephalata,** sheep, goats and cattle in the imaginal stage are parasitized by representatives of two families: Anoplocephalidae and Avitellinidae. The causative agents of these cestodoses belong to four genera:Moniezia,Thysaniezia, Avitellina and Stillesia. Cestodes are localized in the small intestine and cause comorbid diseases: moniesiosis, tizanesiosis, avitellenosis and stellesiosis.

Moniesiosis is a cestodosis disease of sheep, goats, cattle and other wild ruminants caused by M.expansa and M.benedeni cestodes, parasitizing in the small intestine and characterized by digestive disorders, diarrhea, emaciation, sometimes neuronal phenomena and the case of yangyat, goats, calves.

Distribution. The disease is widespread everywhere. In Uzbekistan, the disease is spread in the range of 20-21%.

Economic damage. The disease causes significant economic damage to the economy. Of the sick animals, 5-6% mostly young animals die, the rest of the animals lag behind in growth and development, meat and wool productivity decreases and the costs of treatment and prevention of the disease.

Pathogens. The genus Moniezia is a large cestode. Scolex with four suction cups, unarmed. Sexual openings open on both sides of each segment. There are interproglottid glands, the oncosphere is surrounded by a pear-shaped apparatus.

M.expansa has a milky-white strobila, dense, opaque up to 10 m long. Width up to 16 mm. The scolex is spherical, the segments are short and wide, and the interproglottid glands are grouped along their anterior edge in the form of annular bodies. In mature segments there are branches of the uterus filled with eggs.The genital openings open on both sides of the segment and often protrude above its lateral edges, forming genital papillae.

In M.benedeni, the strobila is yellow–white, translucent, reaches 4 m in length, and a width of up to 2.6 cm. This cestode differs from M.expansa by the greater width of the segments, as well as by the fact that its interproglottid glands are located in a strip along the width of the segment, mainly in the middle part of the anterior margin, and are not grouped in rosettes.

In M.autumnalia – the strobila is white, dense, opaque, its length is up to 3 m, the maximum width is 8 mm, the interproglottid glands, as in M.benedeni, linear type, are located on the ventral and dorsal sides of the strobila along the midline,

however, the length of hermaphrodite and mature segments is equal to or exceeds the width, while as a proglottid, M.benedeni is very short, but wide and the number of testes is 3 times larger.

Biology of pathogens. The parasite is a biohelminth. They develop with the participation of an intermediate host. Intermediate hosts are oribatid (soil, shell) mites of the genus Scheloribates.

Infected animals, along with feces, secrete eggs and mature segments filled with eggs into the external environment. Inside the egg is an oncosphere equipped with six hooks. Eggs are swallowed by intermediate hosts – oribatids. In the tick, the oncosphere comes out of the eggs, actively penetrates through the wall of the digestive tube into the body cavity, where the further development of the larval stages of monies to the invasive cysticercoid takes place. In summer and autumn, at a temperature of 16-200C, the oncospheres of moniesias of both types develop to a formed cysticercoid in 65-90 days, in autumn-winter – in 95-114 days, and at a constant temperature of +260C – in 51-52 days. M.benedeni completes the prepatent period of development in 42-49 days, and M.expansa in 39-8-40 days. Calves develop monies in 47-50 days. The patent period for the development of paprazitis is on average 2-7 months.

Epizootological data. Animals become infected on pasture by ingestion of invasive oribatid mites. In some places, these ticks are found in very large quantities. Depending on the season of the year, temperature, humidity, the intensity of settlement of pastures by ticks varies. Ticks can be found on sufficiently moist pastures, plowing areas significantly reduces the number of ticks, cultured areas are much less covered than virgin ones.

Lambs can become infected with monieziosis in the adjacent territory and pastures of a permanent type. Grass-sown areas of the first and second years of use are most suitable for grazing young animals, since oribatid cliches are almost absent here. Lambs, calves and calves, first released to pastures in the spring, become infected with M.expansa, the intensity and extensiveness of the invasion lasts for 2-3 months. At a high level. So, in some farms, the EI of lambs reaches 90%, and in calves – about 40-60%. Then the disease is on the wane, but from about August the animals become infected with M.benedeni (autumn moniesiosis).

The pathogenesis is caused by the mechanical action of cestodes, which have an irritating effect on the walls of the small intestine. The width of mature segments of moniesia is approximately equal to the diameter of the lumen of the small intestines in 2-2-month-old lambs, and therefore the accumulation of a large number of helminths often leads to obturation and intussusception of the intestine, a sharp violation of the motility of digestion and absorption. Ecto and endotoxins of monies have allergic and toxic effects on the body, causing inflammatory and degenerative processes in various tissues and organs. Under the influence of toxins, the activity of the nervous system, heart and other organs is disrupted. As a result of damage to the integrity of the intestinal mucosa, second infections may occur. With moniesiosis, the stability of the body of lambs and calves decreases, their protective reactions weaken.

Immunity. With moniesiosis of sheep, there is age-related immunity, especially it is expressed in lambs that do not become infected with M. until 3-4 months.benedeni, however, acquired immunity to low-voltage anesthesia and persists for no more than 2 years. Lambs who have had moniesiosis caused by M.expansa may later become infected with M.benedeni. Therefore, acquired immunity is specific.

The symptoms of the disease depend on the intensity of the invasion, the age and general condition of the animals. Young animals are sick, in adult animals, invasion, as a rule, is not clinically manifested. In lambs, lethargy is first noted, they lose weight, lag behind the flock. The feces become softened, and then diarrhea develops; there is an abundant amount of mucus in them, in many cases moniesia segments are found. 10-15 days after the beginning of the separation of the segments, clinical signs increase, sometimes pronounced nervous phenomena appear. Such a course can stimulate some infectious and invasive diseases, especially cenurosis.

Summer monesiosis caused by M.expansa is characterized by the fact that the disease begins some time in the spring or summer after the pasture of sheep and goats and continues in the fall.First, there is a violation of digestion, liquid feces with mucus are released, on the surface of which you can notice white stripes resembling noodles – that is, these are segments of monesias filled with eggs. Sick lambs in the summer lose weight, lag behind in development, become weak, sluggish, anemia develops. In separate animals, signs of central nervous system damage are observed, animals make aimless movements, their gait is short, there are convulsions and can end in death. In some cases, death occurs from intestinal blockage by tangles of cestodes or from intoxication during their decomposition in the body of animals. The course of the disease and the severity of the observed clinical signs depend on AI and primarily on the physicalthe state of the animal's body, as well as the presence of concomitant diseases.

Autumn moniesiosis of sheep and goats is caused by the M.benedeni cestode, which mainly manifests itself from July and grows by autumn.

Pathoanatomic changes are different. Usually the corpse is emaciated, the mucous membranes are anemic, exudate in the abdominal and thoracic cavities. Possible intussusception, inversion of the intestines. There are proliferative-degenerative processes in mesenteric lymph nodes, intestinal mucosa, kidneys, spleen. Hemorrhages on the intestinal mucosa. There are hemorrhages in the brain, infiltrates, and ionesias in the intestine.

The diagnosis during life is made by the detection of moniesias in the feces of eggs. Moniesia eggs are characterized by the fact that the oncosphere is surrounded by a pear-shaped apparatus, M.expansa eggs are triangular, and M.benedeni eggs are quadrangular. The size of the eggs varies from 0,05 to 0,09 mm.

In sheep and goats, in addition to moniesias, cestodes of three more species belonging to the family are parasitized in the small intestine: Avitellinidae: Thysanieziagiardi, Avitellinacentripunctata and Stilesiaglobipunctata.

The final diagnosis is clarified posthumously after the presence of anesthesia in the small intestine and on the basis of pathological and anatomical changes.

Treatment. They recommend phenasal, phenalidone, bithionol, copper sulfate, panacur (fenbendazole), fendaz, fencur, rintal (febantel), albenzazole and its analogues, moniesin.

Prevention. In farms with poor moniesiosis, it is necessary to carry out both pasture prophylactic (isolated rearing of young animals, the use of stall, stall-camp and stall-walking keeping of young animals) and special measures (preventive deworming and chemoprophylaxis) taking into account local conditions.

Calves of the current year of birth are processed 35-40 days after the animals are driven to pasture and again 35-40 days after the first one. 30 days after the second, the final deworming is carried out.

Lambs are dewormed for the first time 14-16 days after pasture, the second time – 15-20 days after drinking, and the third time –25-30 days after the second deworming. These methiodes prevent the development of the disease caused by M.expansa, since M. benedeni animals become infected later. Therefore, at the end of September, if necessary, another deworming of lambs against M. benedeni is recommended. A month after the transfer of sheep to stable maintenance, the entire livestock is dewormed.

Recently, lambs of all ages and adult sheep have been fed salt-phenothiazine-copper-vitriol mixture without restriction (1 part of copper sulfate, 10 parts of phenothiazine and 89 parts of feed salt).

Anoplocephalidosis of horses. Anoplocephalidosis of horses is caused by three species of cestodes from the family Anoplocephalidae: Anoplocephalamagna – localized in the jejunum and ileum, A.perfoliata – in the blind and obochnaya, and Paranoplocephalamillana - in the small intestine. In addition to horses, these species parasitize mules and donkeys.

Pathogens.Anoplocephala magna-up to 520 mm long and 25 mm wide, spherical scolex, unarmed, 2.8-3.0 mm in diameter, with powerful spherical suction cups, no neck, single genital apparatus, one-sided sexual opening. Eggs measuring 0.072x0.084 mm, with a poorly developed pear-shaped apparatus.

Anoplocephala perfoliata is up to 70 mm long and 8-14 mm wide, the scolex is almost cubic in shape, about 3 mm in diameter, with well–developed suckers. Each sucker is connected from the dorsal and ventral sides with two ear-shaped processes, segments are short and wide, the genital apparatus is single, the genital openings are one-sided, eggs are 0.08-0.096 mm in diameter with a pear-shaped apparatus.

Paranoplocephala mamillana- 10-40 mm long,5-6 mm wide, unarmed scolex, 0.7-0.8 mma in diameter,single genital apparatus, one-sided genital openings, eggs 0.05-0.06 mm in diameter, pear-shaped apparatus larger than the radius of the egg.

Biology of pathogens. The parasite is a biohelminth. Intermediate hosts are oribatid mites. Invasive larva is a cysticercoid, cysticercoids reach the invasive stage after 140–15h0 days.

Epizootological data. Anoplocephalidosis is widespread everywhere, and mainly young animals from 5-7 months of age to 2-3 years are sick. Cases of the disease have been reported in suckling foals. The main source of infection of

intermediate hosts is a diseased animal and carriers of the invasion. Horses become infected by eating oribatid mites infested with cysticercoids together with grass. After -1-1.5 months, adult cestodes form in the intestines of definitive hosts. The highest percentage of invasion by Anoplocephalamagna species falls at the end of summer and autumn, Paranoplocephalamillana – at the end of summer and beginning of autumn, A.perfoliata is less common, invasion by this species is kept almost at the same level throughout the year.

The pathogenesis is caused by both mechanical irritation of the intestine and toxic effects. With the accumulation of a large number of anoplocephalids, intestinal intussusception was noted. The products of the cestode metabolism act on the hematopoietic organs and the nervous system.

Symptoms of the disease. With intensive infection, enteritis and colicky phenomena develop. Foals lose weight quickly, lag behind in growth. Anemia of the mucous membranes is pronounced, sometimes the foals have edema of the extremities, underbelly, indicating involvement in the painful process of the cardiovascular system. A.perfoliata is considered the most pathogenic species.

Pathoanatomic changes. At the autopsy of horses, exhaustion, anemia of the mucous membranes are noted, a large number of anoplocephalids are found in the intestines.

The diagnosis is made on the basis of fecal examination by the Fulleborn method - mature eggs or anoplocephalid eggs are found.

Treatment. A highly effective drug is phenasal. Its dose at a single dacha for young animals is 0.2 g / kg, for foals up to two years 0.25 g / kg and for adult horses 0.3 g / kg. The drug is given in a mixture with moistened concentrated feed at the rate of 300-500 g of feed per animal. Male fern extract is also effective in doses from 5 to 20 g in gelatin capsules, after 3-4 hours. A saline laxative is prescribed.

Prevention. In farms that are disadvantaged by anoplocephalitis, preventive deworming is carried out before transferring the cattle to stable maintenance. Preimaginal treatment is recommended 20 days after possible infection of foals. It is necessary to clean the manure daily, followed by its biothermal disinfection.

TOPIC No. 9. NEMATODES AND NEMATODES. PARASCARIDOSIS OF HORSES AND ASCARIASISPIGS.

Lecture plan:

1. Anatomical and morphological characteristics, biological development and systematics of nematodes.
2. Ascariasis of pigs
3. Parascaridosis of horses.

Keywords: Nematodes, Nematoda, Nemathelminthes, cuticle, spicula, rudder, genital papillae, genital bursa, Oxyurata, Ascaridata, Strongylata, Trichocephalata, Spirurata, Filariata, Dioctophymata, Rhabditata, biohelminth, geohelminth, eggs, larva, ovipositors, larva.

Nematodes are diseases caused by helminths from the class of round parasitic nematode worms (Nematoda). This is the largest group of helminths from veterinary helminthology.

1. Anatomical and morphological structure, biological development and systematics of nematodes. Nematodes are characterized by a long, thread-like or spindle-shaped body. The body length of various types of nematodes ranges from 1 mm to 10 m, the outside of the body is covered with a dense layer of cuticle, forming, together with the underlying muscle tissue, the so-called musculoskeletal sac, in which the internal organs are located. The surface of the cuticle is uneven, it can be striated in different directions, some types of nematodes have cuticle formations of various shapes –shields, ridges, spikes that serve to fix nematodes.

The digestive system consists of the esophagus, originating from the mouth opening at the anterior end, and the intestine, located along the entire length of the body; near the posterior end of the body - the anal opening.

The oral opening in many species of nematodes is surrounded by special cuticular formations-lips, and in some nematodes the oral capsule is surrounded by a corolla of petals. Certain types of nematodes have a mouth capsule of various sizes and shapes. Sometimes the oral capsule is equipped with teeth or cutting plates.

The esophagus is a tube of various lengths, sometimes with a bulbous thickening at the posterior end or with a spherical expansion (bulbulm), in which there may be chewing plates, in spirulata and filariata, the esophagus consists of muscular and glandular parts. Anisakids have a blind outgrowth (glandular ventricle) on the border of the esophagus and intestines.

The intestine is a straight tube that opens with an anal opening on the ventral side of the posterior end of the body of the nematode.

The excretory system is represented by tubules, they begin in the back of the body, then merge into one channel, which opens in the front of the body.

The nervous system consists of a central nervous ring surrounding the esophagus with nerve trunks extending from it, and a number of nerve ganglia. The peripheral nervous system is the nerve endings embedded in the superficial cuticular papillae at the head and tail ends of the parasite. Their location and numberare of diagnostic importance.

The sexual system. With rare exceptions, all nematodes are bisexual animals; females are much larger than males.

The sexual apparatus of females is represented by two ovaries, two uterus with oviducts and one vagina, which opens on the ventral side of the body in the form of a hollow slit (vulva), closer to the head or tail end. In some species of nematodes, the cuticle forms a kind of valve above the genital slit, which apparently plays a role in the copulation of helminths.

The male's sexual apparatus consists of a testis, a strongly convoluted vas deferens consisting of a seminal vesicle and an ejaculatory canal. The vas deferens opens into the anal opening of the intestine - the cloaca. Near it, in some species of nematodes, there is a complex of auxiliary male genitalia. Important among them are spicules – chitinized formations of various shapes (paired or single), more often

pigmented. Some nematodes have an unpaired chitinous organ on the dorsal wall of the cloaca-the gubernaculum (rulek), spicules slide along it when they are extended. In trichostrongylids, there is a similar organ on the ventral wall of the cloaca – a telamon. The nerve papillae connected to the nerve ring orient the male relative to the female and her sexual opening. The sexual papillae of males can be sessile and stalked in their shape, they are located in front and behind the cloaca. The number of papillae in individual representatives of ascaridates can reach 20-30 pairs.

In males of the strongylata suborder, the tail end of the body ends with a special cuticle formation - a sexual bursa having a fan-shaped shape and consisting of three lobes: two symmetrical lateral and median. Smaller in size. In the blades of the bursa, from its base to the edges, there are radially neuromuscular ridge-like formations- ribs. In some strongylates, the bursa is two-lobed, the bursa of males performs binding and fixing functions. The presence of a sexual bursa in males is characteristic of nematodes of the strongylate suborder.

Egg nematodes are very diverse in their shape, size, structure and color. The size of the eggs varies between hundredths and tenths of a millimeter. The egg shell protects the egg from adverse environmental factors, primarily from drying out, from the outside it can be colored brown, yellowish or another color. In some species of nematodes on one of the poles there is a kind of lid (oxyurids), or a cork at both poles (in trichocephalates). Some species of ascaridates have a shell of cellular structure.

The biological cycle of nematode development is characterized by a large diversity. As among all other parasitic worms, geohelminths are distinguished among nematodes. Developing in a direct way, without the participation of intermediate hosts, and biohelminths, the development cycle of which is carried out with the mandatory participation of intermediate hosts. In addition, many species of nematodes have reservoir hosts.

Female nematodes secrete eggs or larvae through sexual opening, according to which they are called oviparous or viviparous. Depending on the type of egg-laying nematodes, the eggs they secrete contain either an already formed larva or individual blastomeres. In some cases, larvae hatch from eggs when they pass through the intestines, which are thrown out. Eggs or larvae of egg-laying nematodes are released into the external environment with feces or urine. If eggs with an embryo are released into the external environment, then in any case, a larva of the first stage necessarily develops in the egg. In the future, the larva can leave the egg shells or develop in it to the invasive stage.

Larvae of viviparous nematodes, depending on the localization of the latter, are released either into the external environment through the gastrointestinal tract, with outflows from the eyes, or enter the blood, and from it into the body of blood–sucking insects - intermediate hosts (suborder filariata). The exception to this provision is trichinella. Female trichinella penetrate deeply into the intestinal mucosa, where they spawn a large number of live larvae, which are introduced into the striated musculature with a blood flow and encapsulated in it. In this case, the same animal is the definitive and intermediate host.

The invasive stage of nematode larvae usually reaches after two molts in the external environment (in the egg, on the soil, grass) – geohelminths or in the body of an intermediate host – biohelminths.

With the development of nematodes in a direct way, animals become infected after ingesting invasive eggs or larvae. Before developing into an invasive stage, larvae in the external environment lead a free lifestyle: they grow, move, migrate along the fox and stelby plants. When animals are infected with invasive eggs, the larva hatches in the intestine. Eggs and larvae of nematodes enter the digestive tract of animals with food or water, and larvae of some species of nematodes (strongyloids, ankylostomas, uncinariae) they are able to actively penetrate through the intact skin of the animal. Further development of nematode larvae occurs directly in the digestive tract, or they sometimes undertake a complex migration in the host body before reaching the place of their development to the sexually mature stage.

When developing indirectly, many species of invertebrates and vertebrates can be intermediate hosts of nematodes. All types of nematodes of the suborders filariate, spirurate, dioctophimate, and some species of trichocephalates, strongylates and oxy-murates belong to the biohelminths. The larvae of nematodes, having got from the body of an intermediate host into the body of a definitive host, also, as a rule, make a very diverse migration in it, depending on the type of helminth.

Taxonomy of nematodes. Type **Nemathelminthes**, class **Nematoda-nematodes** includes eight suborders of veterinary significance: **Oxyurata,Ascaridata, Strongylata, Trichocephalata, Spirurata, Filariata, Dioctophymata and Rhabditata.** Each suborder has its own morphological and biological characteristics.

In Oxyurata, the mouth opening is surrounded by three or six lips, at the posterior end of the esophagus there is a spherical expansion (bulbus) with chewing plates. The vulva opens at the front of the body. The male has one or two spicules, they differ sharply in size and shape. Eggs in most species are asymmetrical. Geo- and bio-helminths.

In Ascaridata, the mouth opening is surrounded by three lips. The esophagus is cylindrical, there is no bulbus. On the tail end of the male there may be small wings, preanal and postanal papillae. Spicules are two, equal, there is no steering wheel. The vulva of the female in the anterior half of the body is slightly noticeable, the eggs with a multilayer dense shell are smooth or bumpy. Geo- and bio-helminths.

In Strongylata, the head end is toothless, the oral capsule is small or powerful, sometimes armed with cutting plates or teeth, the esophagus in the back of the body is somewhat dilated. The most characteristic feature is the presence of a sexual cuticular bursa with ribs in males. Two identical spicules, rarely different in size and structure. The vulva opens in the middle part or in the anterior third of the body, can be surrounded by lips, covered by a cuticle valve. Eggs with a thin shell, of various sizes. Geo- and bio-helminths.

In trichocephalates (Trichocephalata), the anterior end of the body is long, threadlike, the posterior end is either much thicker (in trichocephaluses), or of the same thickness (in capillaries). The esophagus is in the form of a thin tube, the

esophageal glands are well expressed, giving the impression of single cells arranged in a row. The male has one spicule, thin, long, or it is absent. The vulva opens in the posterior third or anterior part of the body, oviparous, rarely viviparous (trichinella), barrel-shaped eggs with corks at both poles. Geo- and bio-helminths.

In Spirurata, the mouth opening is mostly surrounded by two lips divided into lobes. The oral cavity passes into the pharynx (pharynx), the esophagus is divided into two sections:muscular (((anterior) and glandular (posterior). Chspicles are usually two, unequal or equal. On the lateral sides of the tail end of the male, cuticular wings, stalked or sessile papillae are most often located. The vulva often opens near the middle of the body, slightly noticeable. The eggs are small, with a thick shell, they leave the body with a ready larva. Biohelminths.

Filariata have a characteristic biological feature: they are localized in closed systems and body cavities of the definitive host (thoracic and abdominal cavities, in blood vessels, tendons, muscles). The head end is simple, often without lips, the esophagus is cylindrical, consists of muscular and glandular parts. There are two spicules. Unequal, the vulva is located at the anterior end of the body. Oviparous and viviparous. Biohelminths.

In Dioctophymata, the oral end is simple or equipped with a muscular sucker. The cuticle is transversely outlined, the esophagus is simple, without bulbus. Males have a dense, bell-shaped bursa without ribs at the tail end. The spicule is single, elongated, without appendages. The anus in females is at the tail end, the vulva opens not far from it; in some species, the vulva is in the front part of the body. The eggs have a powerful shell with a complex pattern on the surface. Biohelminths.

Rhabditata has a characteristic structure of the esophagus: it has two bulbuses - anterior (prebulbus) and posterior. Such an esophagus is called a rhabdito-prominent.Small thin nematodes, among which there are free-living organisms. The eggs are small, after entering the external environment, larvae develop in them in a few hours. Geohelminths.

ASCARIASIS OF PIGS is a disease caused by the nematodes Asagissiiim. Ascaridae of the Ascaridata suborder, young and mature forms of which parasitize in the small intestine of pigs (boars), and larvae affect internal organs (lymph nodes, lungs, liver), causing pathological changes of an allergic nature, eosinophilic filtrates in the lungs, bronchopneumonia, pleurisy, respiratory disorders.

Distribution. Ascariasis of pigs is widespread everywhere, especially in pig farms where a walking area is used, and in pig-breeding complexes the disease is very rarely observed. Suckling pigs, young animals under the age of 6-7 months are most susceptible to ascariasis. Adult pigs rarely suffer from ascariasis, they are usually parasitic carriers.

Economic damage. Ascariasis of pigs causes great economic damage, which consists of the death of animals, stunting of growth and development of young animals, a sharp decrease in animal productivity.

The pathogen.Ascarissuum is a large, white nematode, its oral end is equipped with three lips. The male is 10,5-22 cm long, has two equal spicules 1,2-2 mm long. Females are 23-30 cm long, the opening of the vulva is located in the

anterior third of the body. The eggs are dark brown, 0,050-0,075 mm long and 0,049-0,059 mm wide, covered with a very thick, coarse-browned outer shell.

Biology of the pathogen. The development cycle is direct, without the participation of an intermediate host. Mature female ascaris lay eggs in the small intestine of the animal (one ascaris can lay up to 200 thousand eggs). eggs per day), which are thrown out together with feces. In the external environment, under favorable conditions, mobile invasive larvae develop within 2-3 weeks inside the eggs. When ingesting such eggs, pigs become infected with ascariasis. In their intestines, larvae hatch from eggs, which enter the liver through the portal vein system with blood flow. From the liver, larvae enter the capillaries of the lungs through the vena cava, right heart and pulmonary artery, where they linger for a while, grow up, penetrate the pulmonary alveoli, migrate to the bronchioles, bronchi, and then cough into the oral cavity and are swallowed. After 1.5-2.5 months in the small intestine, they develop into adult ascariids. The term of parasitization of ascaris is 7-10 months, after which they spontaneously depart.

Epizootological data. Ascariasis of pigs is widespread everywhere. The source of the invasion is pigs infested with ascarids. Animals are infected mainly in or near a pigsty (less often on a pasture), swallowing ascaris eggs together with contaminated feed and water.

The role of earthworms in the spread of the disease has been established. Earthworms swallow ascarid eggs, and larvae come out of eggs in their intestines, which can remain viable and invasive for a long time. Poor, irregular cleaning of pigsties, untimely manure harvesting contribute to the spread of infestation.

In ascariasis-affected households, weanlings and weanlings aged 3-5 months are most affected (up to 97-98%). With age, EI and AI decrease.

Pathogenesis. Ascarides have a mechanical, toxic, allergic effect on the animal's body and open the gates to secondary infections.

Immunity to ascariasis in pigs is acquired, therefore, with the increase of fatty acids, the susceptibility to this helminthosis decreases, immunity develops due to the production of antibodies during the migration of larvae, increased reactivity of the body and mobilization of its protective devices. When infected with ascariasis, antibodies appear in the blood after 5-10 days and disappear 90-100 days after infection. They are detected in the blood serum by the precipitation reaction on live ascarid larvae. The highest concentration of the antibody was noted in the initial period of the disease. In addition, sensitization of pigs by migrating larvae is accompanied by the occurrence of allergic reactions.

Symptoms of the disease. The onset of the disease is characterized by ascariasis pneumonia, in which the body temperature rises to 41.50 C, appetite decreases, breathing becomes more frequent, anxiety and cough appear. This lasts from 6 to 15 days. Uporost-suckers note digestive disorders, vomiting. The animals develop severe shortness of breath, general depression, drooling, they gel, burrowing into the litter. Sometimes piglets have intermittent convulsions (more often during feeding), piglets suddenly squeal, are fed to the floor, they start convulsing, breathing becomes faster, skin turns red, eyes open wide. The attack lasts for 1-2 minutes, after

which the animal lies motionless for some time, then they hardly rise, their gait is shady, they remain sluggish, the weakness of the ass is noticeable.

On the skin of piglets, as a result of allergies, rash, urticaria, papules the size of a lentil grain or more may appear, in place of which scabs form after 5-6 days. Surrounded by a belt of brownish or black color. There is no visible clinical picture in adult pigs.

Pathoanatomic changes. During migration, larvae are found in lymph nodes, liver, lungs. Characteristic are white spots on the liver of 2-5 cm in size (white-spotted liver). Hemorrhage foci and pneumonic foci in the lungs also give them a spotty appearance. Small foci with necrosis in the center and spot hemorrhages, expansion and infiltration by eosinophils of the interlobular connective tissue are detected on histopremes from the surface of the liver. Histological changes in the liver are characterized as acute interstitial eosinophilic hepatitis. After the migration of the larvae stops, the infiltrates gradually dissolve and a thickening of the interlobular connective tissue remains, leading to interstitial cirrhosis.

Sexually mature ascarids can cause catarrhal inflammation of the intestinal mucosa.

The diagnosis during life is made on the basis of a study of feces by the method of Fulleborn or Shcherbovich.

The early stage of ascariasis can only be determined immunobiologically.For this purpose, an allergic diagnosis is proposed:!A 5-solution of the antigen prepared from ascaris is injected intradermally in the ear area, in piglets infected with ascariasis, a purple-red rim appears on the skin after 5 minutes, which persists for 30-40 minutes, and then disappears.

Posthumously, the diagnosis is made on the basis of the detection of ascarids in the intestine, as well as the result of the examination of pieces of lung and liver tissue for ascarid larvae using the Berman-Orlov method.

Treatment. For pig deworming, piperazine and its salts, piavetrine, nilverm, chlorophos, panacur (fenbendazole), rintal (febantel) and other anthelmintics are used.

Prevention. In farms that are unfavorable for ascariasis of pigs, scheduled preventive deworming, sanitation, biothermal manure treatment, farm territory, paddock yards are carried out annually, after thorough cleaning of manure, they are plowed, leveled with a roller and abundantly sprinkled with lime.

Maintenance and proper feeding of pregnant queens and piglets are of great preventive importance.

EQUINE PARASCARIDOSIS is a disease of ungulates caused by nematodes, Parascarisequorum of the Ascaridae family, parasitic in the small intestine, whose larval forms migrate in the body along the hepatopulmonary pathway.

Distribution. The disease is widespread everywhere, suckling foals and foals up to one year old are most susceptible to paraskaridosis, and adult horses are also strongly infected (up to 46-50%), but they are usually parasitic carriers. With age, the percentage of infection and the intensity of invasion decrease.

Economic damage. Paraskaridosis of horses causes great economic damage, which consists of the death of animals, stunting of growth and development of young animals, a sharp decrease in animal productivity.

The pathogen. Parascarisequorum is a white nematode with an elastic spindle–shaped body. The mouth is surrounded by three lips, the edges of which are equipped with teeth; small intermediate lips are located between the large lips, the male is 15-28 cm long, externally differs from the female in smaller size and a curved tail end, where there are small wings and 79-105 pairs of sexual papillae. The female is 18-37 cm long, the opening of the vulva is at the level of the anterior quarter of the body length. Paraska-reed eggs are round, 0.09-0.10 mm in diameter, with a thick shell, dark brown in color, unfertilized eggs are light.

Biology of the pathogen. The development cycle is direct – according to the ascarid type. The term of mirgation of the parascarid larva along the hepatopulmonary pathway is 23-25 days, the prepatent period of development is 2-2.5 months, parascarids live in the body of ungulates up to one year or more.

Epizootological data. Parascaridosis mainly affects young animals under the age of one year. Invasion of suckling foals is especially difficult, with fatal cases. Who can become infected in the first days and weeks of their life.adult horses are also highly infected (up to 46-50%), but they are usually parasitic carriers. With age, the percentage of infection and the intensity of invasion decrease.

EI and AI largely depend on the conditions of keeping, caring and feeding animals. Incomplete feeding lowers the body's resistance and therefore contributes to a more intense infection of foals.The source of invasion are sick animals that secrete paraskarid eggs into the external environment together with feces, polluting the surrounding area and care items – machines, feeders, shovels, brooms, etc. Irregular cleaning of rooms, feeders, feeding hay from the floor contribute to the invasion of animals with paraskarid eggs. Horses can become infected not only in the premises, levades, on the tilled territory, but also on the pasture when ingesting parasite eggs together with grass or water. Suckling foals have a habit of sniffing, gagging various objects, dried feces contaminated with helminth eggs.

Paraskarid eggs develop at a temperature not lower than 10^0C and not higher than 38-40^0C, Drying has a detrimental effect on them, fecal matter 20-25% and relative humidity below 70% eggs do not develop and die. On pastures in summer, paraskarid eggs usually die after 26-65 days from drying out and the action of direct sunlight. The most favorable conditions for paraskaridosis infection in stables and pastures are from May to September.

Pathogenesis is reduced to traumatization of tissues by sexually mature worms and their larvae, toxic effects and inoculation of pathogenic microflora by larvae during their migration, In addition, parasitization of paraskarids is associated with the development of allergic phenomena, as with ascariasis of pigs.

Acquired immunity manifests itself in the form of a decrease in susceptibility to parascaridosis as the age of the animals increases.

The symptoms of the disease are very diverse and largely depend on the age of the animals and the intensity of the invasion. Adult animals, as a rule, are parasitic

carriers, so their disease is asymptomatic. In foals at the beginning of the disease, during the migration of larvae, enteritis and diarrhea are noted (3-4 days). After that, signs of bronchopneumonia develop:short-term fever, cough, rapid breathing, serous-mucous discharge from the nose, sometimes there are attacks of nervous excitement. These phenomena are most pronounced on the 9th-16th day after infection and usually last 4-7 days, then gradually disappear.

The disease in the stage of parasitization of mature paraskarids is characterized by constant emaciation of sick animals, periodic diarrhea, alternating constipation, foals lag behind in growth, their abdominal volume increases, sometimes there are colic. The visible mucous membranes are pale. In the nervous form, tetanic convulsions, paresis of the ass, violent seizures of excitement, stimulating signs of rabies.

When examining blood, a decrease in the number of erythrocytes and hemoglobin content, an increase in ESR, and eosinophilia are established.

Pathoanatomic changes. Upon autopsy, local mechanical damage to the mucous membrane of the small intestine is noted: foci of superficial destruction of the epithelial cover, sometimes hemorrhage, ulceration. General changes are characterized by acute and chronic catarrhal. Sometimes fibrinous or hemorrhagic inflammation is associated with congestive edema of the submucosal and subsurface layers of the lining of the small intestine, mucosal degeneration of the epithelium and infiltration by lymphoid cells, eosinophils, polymorphonuclear leukocytes and histiocytes.

During the mass migration of larvae, multiple spot hemorrhages and inflammation are found in the lungs. So-called parasitic nodules with necrosis foci in the center are formed around the larvae that died along the migration path. They are most often found in the liver and lungs, less often in lymph nodes, kidneys, etc. Over time, these nodules are surrounded by a connective tissue capsule and then calcified.

The diagnosis during life is made on the basis of coprological studies using the Fulleborn method. Sometimes paraskarides are excreted together with feces; the discharge of these helminths can be caused by diagnostic deworming. Posthumously, the disease is established when parascarids are detected in the intestine.

Treatment. Piperazine and its salts, piavetrin, panacur (phenobendazole), fencur, fendaz, rintal (febantel), mebendazole, morantel tartrate, carbon tetrachloride are used.

Prevention. In farms that are disadvantaged by paraskaridosis, mandatory preventive deworming of horses is carried out in the following terms:

-Foals of the current year of birth – the first time in August, the second time after weaning;

-Young animals 1-2 years old and adult horses – in March-April and in October-November.

In herd horse breeding farms, suckling foals, starting from the age of 3 months, are fed monthly for 2 consecutive days in a group way, 10 g of piperazine per reception. From August until the end of the year, its dose is increased to 15 g per reception; this drug is given 2 days in a row every 2-3 months.

Horses at racetracks are dewormed only with piperazine every 2-3 months. After the treatment of the animals, the stables are deinivazied. If deworming was on the pasture, then the horses are kept for 3-4 days on a separate plot, after which it is replanted.

In addition to deworming, in dysfunctional farms, it is recommended: timely cleaning of manure, systematic cleaning of premises, feeders, care items with subsequent disinfection of them. Adobe floors in stables, sheds, looms and stalls are tamped at least once a year (in summer) and covered with a fresh layer of clay 10-15 cm thick. A separate mill is fixed for each horse.

TOPIC No. 10. OXYUROSIS AND INTESTINAL STRONGYLATOSES OF HORSES.

Lecture plan:

1. *Oxyurosis of horses*
2. *Delafondiosis*
3. *Alfortiosis*
4. *Strongylosis*
5. *Trichonematosis*

Keywords: Oxyurata, Oxyuridae, Oxyurisequi, geohelminth, direct path of development, prepatent, patent period, planned deworming, biothermic treatment of manure.

EQUINE OXYUROSIS is a chronically occurring disease caused by nematodes Oxyurisequi from the family Oxyuridae, parasitizing mainly in the large colon. The disease is manifested by a violation of the digestive tract and a lesion of the skin at the root of the tail ("combing" of the tail). Other ungulates, such as donkeys, mules, and zebras, also suffer from oxy-cirrhosis in horses. Oxyurosis is common everywhere.

The pathogen. The mouth opening is hexagonal in shape, opens into a short oral capsule (pharynx), surrounded by six lips. The esophagus in the back has a bulbus.

The male is 6-15 mm long, the tail end is blunt, with a cuticular membrane supported by several pairs of stalked papillae. There is one sharp spicule about 167-0.223 mm long, there is no rudder.

The female reaches 40-160 mm in length, is thick, has a relatively short head and a long thin tail. The vulva is located in the anterior half of the body. Eggs with a size of 0.085-0.099x0.040-0.045 mm, colorless, translucent, asymmetrical, on one of the poles – a kind of "lid". By the time of egg laying, they are close to the mature invasive stage.

Biology of the pathogen. Development goes on without the participation of an intermediate host. After fertilization, the males die, and the females form and mature

eggs, which they do not lay in the intestines, as it is noted in other types of helminths. Overflowing with eggs, females descend together with feces, passively. They come out of the intestines, some of them fall to the ground and lay eggs on the surface of the intestines, and some, lingering with the help of a long thin tail in the folds of the mucous membrane around the anus, secrete eggs in the perianal area, under the tail.A sticky mucous mass containing eggs forms a grayish coating on the perianal folds. After the allocation of the entire stock of eggs, the females also die. Under the tail and in the perineum area there are favorable conditions for the development of eggs (humidity, oxygen, suitable temperature). After 2-3 days, larvae develop in them, and the eggs become invasive. In females thrown on the surface of feces, egg laying begins after 1-2 minutes, in the depth of the feces of the female, eggs are not excreted.

When drying out in the perianal folds, when the tail moves, mature eggs fall into the external environment, polluting feeders, walls, crossbars, in stalls, litter, grass, horse care items.

Animals become infected with oxyurosis when ingesting invasive eggs of oxyuris. Larvae come out of their eggs in their intestines, which after 3-4 weeks grow into sexually mature parasites.

Epizootological data. Oxyurosis mainly affects young animals up to one year old and old horses. Damp dirty rooms, poor feeding contribute to the spread of oxyurosis. Horses are more often invaded when eating food from the floor, where most of the eggs of oxyuris are located. The greatest number of sick horses is observed in the stable period, from November to May months.

Pathogenesis. Oxyuris have a mechanical, toxic effect on the animal's body. As a result, catarrhal inflammation of the intestine develops. Females and their eggs isolated in the perianal folds cause severe itching. To get rid of it, horses rub against hard objects, resulting in dermatoses, baldness in the area of the root of the tail.

Symptoms of the disease. In sick horses, the so-called "combing" of the tail is formed, which is a characteristic clinical sign, Animals, experiencing itching, show anxiety, often scratch against the walls, crossbars of the stall and other objects. At the root of the tail, the hair falls out, eczema appears. Patients gradually lose weight, they have various disorders of the gastrointestinal tract.

Pathoanatomic changes have not been studied.

The diagnosis is made on the basis of clinical signs and the detection of eggs by scraping from the perianal folds.

Treatment. 4-carbon chloride, salts of piperazine, phenothiazine, panacur, rintal, tetramisole granulate 20% and mebenvet granulate 10%, ivomek, baymek, ivermectin are used.

Prevention. In a dysfunctional farm, planned deworming of horses is carried out every 1-1.5 months.Cleaning and bleaching of the premises, biothermal treatment of manure, full feeding of animals according to the diet. Young animals are raised separately from adult animals.

Intestinal strongylatosis of horses is a complex of helminthic diseases caused by representatives of the suborder Strongylata of the family Strongulidae and

Trichonematidae, parasitizing in the mature stage in the large intestine of animals.Larval stages are localized in various tissues depending on the type of pathogen. To date, there are about 45 species of nematodes – pathogens of intestinal strongylatoses of horses and other ungulates (donkeys, mules) animals. All of them belong to geohelminths, have similar development in the external environment and are characterized by a common patented pathogenic effect on the body, consisting of the pathogenic influence of numerous individual species of strongylates, which, as a rule, do not occur in horses in the form of pure invasion.

Distribution. Intestinal strongylatoses are widespread, ubiquitous helminthiasis. Almost all horses, starting from a very young age, are completely infected with these diseases. AI depends on the age, conditions of keeping and feeding of animals (from several hundred parasites to tens, and sometimes hundreds of thousands).

Economic damage. Intestinal strongylatoses bring significant economic damage, which consists of a sharp decline in the growth and development of sick foals, a decrease in working capacity, direct losses - animal deaths, as well as veterinary costs for the treatment and prevention of the disease.

Pathogens. Males of all representatives of strongylates have a well-developed cuticular genital rib bursa. Their body length is 0.5-4.5 cm. Of the representatives of the Strongylidae family, the species of genera are of the greatest importance: Genus: Strongylus, species - Strongylus (Delafondia)vulgaris, S. (Alfortia) edentatus, S.(Strongylus) equinus, a characteristic feature that distinguishes from trichonematodes is the presence of a hemispherical oral capsule, Of the representatives of the Trichonematidae family, the genera: Trichonema, Gyalocephalus and Poteriostomum are of the greatest importance. They differ from strongylids by the cylindrical or annular shape of the oral capsule.

The eggs are oval in shape, 0.07-0.1 mm long, 0.04-0.05 mm wide, with a thin shell, light gray in color.

Biology of pathogens. Fertilized females secrete a large number of eggs in the intestines of animals, which, together with feces, fall on the soil, litter, manure. At a favorable temperature, in the range of 8-380C, a larva forms in the egg, which breaks the egg shell, bursts into the external environment, where it molts twice and develops to an invasive stage. Invasive larvae developed in feces migrate horizontally and vertically in the soil and along the stems of plants with sufficient humidity (rain, dew).

Horses become infected by ingesting invasive ones together with grass or water (when drinking from puddles and other small standing reservoirs), indoors when eating coarse feed from the floor, fresh grass mown on pastures contaminated with larvae, with irregular cleaning and changing the litter in the walls.

The development of various types of strongylates in the horse's body proceeds differently, as a result of which pathoanatomic changes and clinical signs manifest themselves in different ways. In accordance with zhtim and taking into account some epizootological features, separate nosological names are accepted in the general strongyl-yatosis complex of horses: delafondiosis of blood vessels, alfortiosis of the

bruschina, strongylosis of the pancreas, nodular trichonematosis and intestinal strongylatosis caused by parasitization of mature strongylates. Effective methods of treatment against the above-mentioned diseases have not yet been developed, only a palliative method of treatment is being carried out. However, prevention and measures to combat them are common and are directed against sexually mature stronglings.

DELAFONDIOSIS OF HORSES or thromboembolic colic – the causative agent of this disease are the larvae of Longylusvulgaris–Delafondiavulgaris, which parasitize in the arteries of the intestinal wall and mesentery root, cause inflammation of the walls of blood vessels, the formation of anervism, atrophy of nerve cells and nerve trunks, innervating certain areas of the intestine with the development of hemorrhagic infarcts in them. With severe invasion, delafondiosis is clinically manifested by thromboembolic (delafondious) colic.

Strongylusvulgaris – parasitize in the cecum and the lower knee of the colon. Male is 14-16 mm, female is 20-24 mm long. Distinctive feature: two ear-shaped teeth are located in a large rounded oral capsule at the base of the dorsal esophageal trough.

Invasive larvae of delafondia that have entered the intestine shed their cover and penetrate into the thickness of the mucous membrane, penetrate further into the blood vessels, migrate between the muscular and serous layers or enter the lymphatic vessels.

Larvae trapped in the lymphatic vessels are introduced into the liver and there they quickly die.

Larvae trapped in the intestinal and mesenteric arteries continue to develop. From small blood vessels (capillaries and arterioles) they actively move through the blood flow into the larger vessels of the anastomotic network of the mesentery root and the aorta with its branches. Here the larvae form blood clots, in which they linger and develop within 5-6 months.

After this period, they reach 2 cm, shed, dropping the cuticle, are easily released from the thrombus into the light of blood vessels. Then, with the blood flow, the larvae are again transferred to the branches of the arteries of the caecum and colon, from where they penetrate into the intestinal wall, where they linger for 3-4 weeks, forming parasitic nodules the size of a pea. From the nodules, the larvae exit into the intestinal lumen and grow into sexually mature males and females. **The prepatent period** of parasite development is 6-7.5 months, and the period of parasitization is about one year.

Immunity is weakly expressed, reinvention and superinvasion are possible.

Pathogenesis. Delafondium affects the animal's body by mechanical, toxic effects and opens the gates to secondary infections.

The symptoms of the disease are very diverse and depend on AI, the stage of development of larvae, primary infection or superinvasion. In foals infected for the first time, the disease is acute and is characterized by an increase in body temperature to 41^0C, anemia and disorders of the gastrointestinal tract. Repeated infection of animals, as a rule, does not manifest itself clinically.

The chronically occurring disease is characterized by periodic attacks of colic with clinical signs of intussusception, intestinal twisting or thromboembolic process. The greatest number of cases of delafondiosis colic is noted in February-March in animals aged five to ten years.

According to the severity of the clinical manifestation, depending on the degree of circulatory disorders and the outcome of the disease, there are two forms of delafondious colic: **mild and severe.** In both forms, colic attacks are usually not associated with any errors in feeding and keeping horses.

The mild form lasts from one to several hours, less often up to a day and ends with recovery. At first, the sick horse is very worried, then falls and rolls unrestrainedly on the ground in strong excitement, while defecation occurs; pulse and breathing are normal. After a while, the colic gradually weakens, then begins again. In the intervals between attacks, the animal feels normal and even takes food and water.

In severe form, in addition to severe attacks of colic, animals take unnatural positions: lying on their backs with outstretched limbs, the position of a sitting dog, sometimes the volume of the abdomen increases. Over time, colic attacks become more violent and prolonged, and the interval between them is shorter. In the intervals between attacks of colic, horses are in a state of depression and deafening, grabbing hay with their teeth, holding it in their mouth without worrying, there is frequent yawning. The onset of the disease is characterized by increased peristalsis and frequent defecation, feces of a liquid consistency, sometimes with an admixture. Gradually, the peristalsis weakens and stops completely, the feces are delayed, after 2 hours flatulence occurs, strong tension of the abdominal walls. Rectal examination reveals a strong swelling of the blind and colon. Pulse and breathing become more frequent. With the onset of complications (enteritis, peritonitis), the body temperature rises, the pulse decreases, muscle tremors appear, sometimes turning into general convulsions. The severe form lasts 1-2 days and ends with the death of the animal.

In rare cases, the disease takes on a protracted character and lasts up to 15-20 days.

Pathoanatomic changes. Upon autopsy, mass spot hemorrhages are found on the mucous and serous membranes of the intestine, on the mesentery, inflammation of the mesentery root, abscesses in it; a characteristic feature is the presence of anervism of various sizes and shapes. In anervisms, larvae of delafondia are found, the number of which can reach 300 .kz. On the intima of the aorta, the anterior mesenteric artery, its branches, you can see mass elevations intertwining with each other – filamentous passages – traces of larval migration.

In horses that have fallen from delafondiosis, the mucous and serous membranes of the large colon and caecum are colored black and red; in the affected area of the intestine - bloody tar–colored fluid. In the abdominal cavity, serous fluid is yellowish or reddish in color, in some cases, the presence of fibrous exudate. Blood clots, emboli, purulent arteritis, periarteritis are found in the affected blood vessels.

The diagnosis is made according to clinical signs, quite characteristic of delafondious colic. In case of severe form of the disease, the inversion and

infringement of the intestines should be excluded by rectal examination. It is recommended to probe the anterior mesenteric artery through the rectum, in sick animals at the same time they feel the trembling of the artery walls. Posthumously, the disease is diagnosed on the basis of pathoanatomic changes.

Palliative treatment. It should be aimed at restoring collateral circulation. For this purpose, camphor oil is injected subcutaneously at 20-50 g every 2-3 hours, and the animal is wired. As an analgesic and to prevent intestinal rupture, morphine is prescribed subcutaneously in a dose of 0.1-0.4 g.

You can use mebenvet, which is administered through the mouth in the form of a bolus individually, once at a dose of 0.15 g / kg or at a dose of 0.12 g / kg twice with an interval of 24 hours.

Prevention is reduced to improving the health of horses from sexually mature intestinal strongylates.

ALFORTIOSIS OF HORSES. The causative agents of goiter are the larvae of the nematode Strongylusedentatus - Alfortiaedentates.

Strongylusedentates –parasitize in the caecum and colon, sizes: male 23-26 mm, female 32-40 mm–In alfortia, the oral capsule is cup-shaped, teeth are missing, the head end is somewhat swollen and does not have a post-gradual narrowing, it seems to be cut off.

The development of alfortia in the horse's body. Invasive larvae of alfortia, having dropped the cover, penetrate into the intestinal wall into the subserozoic layer, from where they migrate between the mesentery leaves to its root, and then down under the peritoneum leaf, concentrating in the area of the costal arch, groin, (mainly in the back). In these places, the larvae are located in small cavities-hematomas, develop within 5-6 months, growing to 3-4 cm. After that, they return back up to the mesentery root and descend down to the intestinal wall; between the muscular and mucous layers, the larva forms a parasitic nodule (the size of a bean), in which it stays for 3-4 weeks, after which it enters the intestinal lumen, attaches to the mucous membrane and develops into a sexually mature alfortia.

The prepatent period of development of the parasite is 8-9, and sometimes 9.5-10 months, and the period of parasitization of strongylates is equated to one year.

Pathogenesis. Migrating larvae injure tissues, cause hemorrhages. Progressive anemia develops from the toxic action of the larvae. Cachexia and the death of the animal occurs. The larvae of alphorthia introduce pathogenic microflora into the host organism along the migration path. Septic peritonitis develops when complicated by purulent microflora.

Symptoms of the disease. Alfortiosis is acute and chronic.

The acute course is peculiar exclusively to foals, develops within a few days and is manifested by depression, weakness, severe anemia. After 6 days, intermittent mild colic is noted. Body temperature rises to 40-41.90C. The pulse is weak, rapid, breathing is frequent, tense and painful. When palpating the abdominal wall, foals experience pain, moan, look around on their stomach, bend excessive movements, often lie down or stand hunched over. Patients die with the phenomena of amenia and cachexia.

The chronic course lasts 1.5-2 or more months. It happens in foals and old horses. The signs are the same as in the acute course (depression, anemia), but less pronounced.

It is not difficult to make **a diagnosis** during life, one can only suspect peritoneal alfortiosis on the basis of clinical signs.

Posthumously, the diagnosis is made according to the characteristic pathoanatomical changes that are detected during autopsy (alfortious peritonitis): on the diffusely reddened peritoneum, a large number of hematomas in the form of dark red and bluish spots the size of a five-kopeck coin. Through the serous membrane, curved or elongated larvae of alfortia are visible reddish or milky in color. During the incision, small cavities (hematomas) with bloody fluid and a thick mass with alfortia larvae up to 4 cm in length are found. With a large number of larvae, the peritoneum is edematous. In the abdominal cavity 1.5-2 liters of yellowish-red liquid.

In differential diagnosis, it is necessary to take into account infectious anemia, which is excluded or established by hematological or other research methods.

Prevention is reduced to improving the health of horses from sexually mature intestinal strongylates.

STRONGYLOSIS OF HORSES. The causative agents of this disease are the larvae of the nematode **Strongylusequines - Strongylusequines.**

Mature strongiluses parasitize in the large intestine and are the largest strongilids of horses: the male is 25-35 mm long, the female is 35-45 mm. At the bottom of the powerful spherical oral capsule there are four teeth: two thin and long and two short.

The development of strongiluses in the body of horses. The larvae, released from the cover, penetrate through the intestinal mucosa and migrate between the mesentery leaves into the pancreas, where they develop for 8 months, increasing to 4-4.5 cm. After that, they return back to the large intestine, develop into adult parasites. The total period of development of strongiluses in the host body is at least 10 months.

Pathogenesis. The pathogenic effect of strongiluses in the pancreas is reduced to a violation of blood circulation, an overgrowth of connective tissue, which leads to a weakening of the function of the pancreas.

The diagnosis is made posthumously, based on the detection of strongilus larvae during the opening of the pancreas.

TRICHONEMATOSIS OF HORSES. The causative agents of this disease are the larvae of numerous species of nematodes of the genus Trichonema, parasitizing in the wall of the caecum and colon.

Trichonemas are very small nematodes whose size ranges from 5-7 to 12-16 mm in length, the oral capsule is spherical or cylindrical in shape. Trichonematid larvae parasitize under the mucous membrane of the large intestine, and adults parasitize in the lumen of the caecum and colon.

The development of trichonemas in the horse's body. Invasive larvae are introduced into the thickness of the intestinal mucosa, curl there into a ring and nodules of the size from poppy to millet grain ("poppy intestines") are formed around them, there can be 30-50 copies per 1 cm2 of such nodules. The larvae grow

gradually for 1.5-2 months, reaching 5-10 mm in length by this time. Then they tear the wall of the cysts out into the lumen of the caecum and colon and develop into mature males and females.

Pathogenesis. Trichonematous nodular process lasts 1-2 months. However, in the mucous membrane of the blind and colon, there is always a certain number of trichonematous nodules with larvae in them as a result of a new infection. The larvae of trichonemas injure the mucous membrane, resulting in serous, fibrinous or hemorrhagic colitis. This "opens the gates" to pathogenic microbes that cause septic process, the formation of metastases, peritonitis, arthritis, etc. can develop. Incised larvae cause general intoxication of the body.

Pathoanatomic changes. The corpse is exhausted. Catarrhal, sometimes fibrinous or hemorrhagic inflammation of the mucous membrane of the large intestine. Diffuse hemorrhagic foci are being treated, the mucous membrane is as if dotted with poppy seeds (trichonematous nodules). It should be borne in mind that this picture is similar to mass spot hemorrhages, so as not to make a mistake, you can give out trichonem larvae from the nodules. Necrotic foci, small ulcers, crater–like openings are often found on the mucous membrane - traces of the former stay of larvae. In the lumen of the intestine – liquid contents of a bloody color, in which you can surround a mass of young trichonemas that have emerged from cysts. With the complication of pathogenic microflora, peritonitis, abscess under the skin is possible.

Symptoms of the disease. With a mild form, emaciation, anemia, fatigue at work, diarrhea, delay in molting, sometimes a slight increase in temperature (39,2-39,3^0 C. Under good feeding and keeping conditions, the animals recover in 1,5-2 months.

In severe form, the temperature rises (39-40,5^{0}C), progressive emaciation, anemia occur. They note general weakness, loss of appetite, shaky gait, lying down. The severe form is characterized by debilitating diarrhea, feces with a hollow smell, an admixture of blood, sometimes with a mass of young trichonemas of red color, there may be slight swelling of the limbs, weak colic. Mares sometimes have ovulation. There are significant changes in the blood: a decrease in hemoglobin content to 30-40%, a decrease in the number of red blood cells, eosinophilia, an increase in ROE.

The diagnosis is only postmortem, based on pathoanatomic changes and the detection of trichonem larvae inside nodules in the thickness of the intestinal mucosa.

Prevention is common for all intestinal strongylatoses of horses.

STRONGYLATOSES OF HORSES CAUSED BY ADULT PARASITES. These diseases, caused by parasitization in the large intestine of sexually mature strongylids and trichonematids, are characteristic of horses of all ages, but mainly animals older than five years.

Pathogenesis. Strongylides and trichonematides have a mechanical, toxic effect on the animal's body and open the gates to secondary infections.

Pathoanatomic changes are noted in the large intestine: inflammation of the mucous membrane, sometimes of a gangrenous nature, ulceration of it, necrotic foci, reveal a huge number of strongylates attached to the mucosa or freely located in the

contents of the intestine. It has been established that strongylates of the same kind are fixed, concentrating in groups in certain areas of the intestine.A vivid picture of this phenomenon can be seen when triodontophores are affected. Trichonemas during normal autopsies are visually difficult to notice in the contents of the intestine due to their small size.

Larvae of delafondia and alfortia, in addition to blood vessels, are sometimes found in other organs and tissues. So, the larvae of delafondia getting into the lungs, form the so-called nodular delafondiosis of the lungs.

The symptoms of the disease are not characteristic. With a low AI, the disease proceeds asymptomatically (strong infection), and with strong invasion, a decrease or lack of appetite, post-severe emaciation, anemia, fatigue, decreased performance of adult horses, lag in growth and development in foals are noted. Sometimes there is diarrhea, occasionally the temperature rises to 39,5-40^{0}C. In the faeces of horses, there is often a large number of strongylates, especially trehonem.

The diagnosis of life can be made by a coprological method – the study of feces by the Fulleborn method.

For a more accurate diagnosis, invasive larvae are grown from eggs, according to which the genera and species of strongylates are determined. The main differences between these larvae are the shape, location and number of intestinal cells. In alfortia larvae, the intestinal tube contains 20 shapeless, poorly expressed cells, in delafondia – 32 cells; in strongylus – 16 elongated cells; in trichonem larvae, the intestine consists of 8 clearly defined triangular cells.

Posthumously, the diagnosis is made based on the presence of strongylates in the intestine and pathoanatomic changes.

Prevention consists of a whole range of measures. The main ones are preventive deworming 2 times a year: in spring, before the start of the pasture season, and in autumn, in September. Therapeutic deworming is carried out at any time of the year.

For the deworming of horses, carbon 4-chloride, piperazine salts, phenothiazine, fenbendazole (panacur), rintal, mebendazole in the form of mebenvet 10% granulate, morantel tartrate are used.

TOPIC No. 11. STRONGYLATOSES OF THE GASTROINTESTINAL TRACT OF RUMINANTS.

Lecture plan:

1. Systematics of strongylatosis of the gastrointestinal tract of ruminants
2. Morphology and biology of strongylatosis of the digestive tract.
3. Hemonchosis
4. Marshallagiosis
5. Nematodirosis
6. Habertiosis

Keywords: Strongylata, Strongylidae, Trichostrongylidae, Trichonematidae, Ancylostomatidae, Chabertia, Nematodirus, Haemonchus, Marshallagia, Ostertagia, Cooperia Trichostrongylus, Mecistocirrus, prepatent and patent period of development, bio- and geonematodes, deworming, direct path of development, i.e. without the participation of an intermediate host.

In the digestive tract of ruminants, a large number of species of nematodes from the suborder Strongylata, the family Strongylidae, the genus Chabertia, the species Chabertia ovina, the family Trichostrongylidae, the genera Chis, Marshallagia, Trichostrongylus, Nematodirus, Ostertagia, Cooperia, Mecistocirrus and others are parasitized; the family Trichonematidae, the genus Oesophagostomum, family Ancylostomatidae, genus Bunostomum.

The unification of all these types of nematodes is based on the common localization, development cycles of the helminths themselves, epizootology, pathogenesis and clinical manifestations of the diseases caused by them, as well as treatment and preventive measures, which are approximately the same. At the same time, there are a number of significant differences that make it possible to distinguish individual diseases from this general strongylatous complex, the most studied and specific. These include: hemonchosis, marshallagiosis, nematodirosis, trichostrongylosis, esophagostomiasis, bunostomiasis, habertiosis and others.

HEMONCHOSIS. Hemongosis is caused by nematodes of the genus Haemonchus (the most widespread species of Haemonchuscontortus), parasitizing sheep and goats, as well as cattle (Haemonchusplacei), reindeer and many wild animals in the abomasum.

The pathogen. Compared with other trichostrongylids, the largest nematodes (length 1,8-3,4 cm) have a thinned head end equipped with a rudimentary oral capsule with a chitinized tooth inside and a pair of cervical papillae. The male has a powerful sexual bursa with an assimitrically located dorsal rib, spicules are short and massive, 0,3-0,5 mm long, there is no rudder.

Biology of the pathogen. The development proceeds according to the general scheme. At temperatures below $5,5^0$ C and above 40^0C, hemonchus eggs do not develop and die. The total period of development from eggs to invasive larvae at a temperature of 26^0C is 4 days, at a temperature of 17-18^0C-6-8 days. Non-invasive larvae are poorly resistant and die quickly when drying out and sudden temperature changes. Invasive larvae tolerate drying for 1-1,5 years, remain viable during overwintering, withstand temperatures up to 50^0C in a humid environment, and slightly higher in a dry one.

Once in the host's abomasum, the larvae develop within 17-20 days to the sexually mature stage. In the body of animals, hemonchuses can parasitize for 6-8 months.

Epizootological data. Infection with hemonchosis occurs mainly on pasture in the summer-spring and autumn seasons. AI can be so high that helminths completely cover the mucous membrane of the abomasum. The seasonal dynamics of the hemonkhoz depends on the climatic and geographical zone and economic conditions.

In lambs that have gone out to pasture for the first time, the disease manifests itself in late summer and early autumn.

Pathogenesis. The pathogenesis is based on injury to the mucous membrane of the rennet, inflammatory processes and capillary bleeding. Hemonchuses feed on the host's blood, which leads to the development of anemia. Intoxication with parasite waste products leads to pathophysiological and pathomorphological changes throughout the body, including in the hematopoietic organs, endocrine glands and the nervous system.

Symptoms of the disease. Depression, decreased appetite, pallor of the mucous membranes, in some cases the temperature rises to 40-40,5^{0}C. The hemoglobin content in the blood decreases, ESR increases, severe leukocytosis develops (14,6-20 thousand in 1 mm^3). Young animals and animals that are in poor feeding and maintenance conditions are particularly hard to get over.

Pathoanatomic changes. Anemia and severe exhaustion of corpses are characteristic. In the abdominal and thoracic cavities, the liquid is slightly yellowish in color. The mucous membrane of the abomasum is covered with parasites, dotted with hemorrhages, thickened. The contents of the abomasum are liquefied, brown in color, the mucous membrane of the small and large intestines is inflamed. The phenomena of nephrosis, edema of the intermuscular connective tissue, degeneration of the muscle fibers of the heart, atrophy of the liver parenchyma, atrophic and degenerative changes in the brain and spinal cord, endocrine glands, hematopoietic organs are noted.

The diagnosis is vital according to invasive hemonchus larvae, postmortem – according to autopsies.

Treatment. The following drugs are used: phenothiazine, naphthamone, copper sulfate, nilverm, fenbendazole (panacur), rintal (febantel), albendazole and its analogues, mebenvet, tafen and tividin.

Prevention. Preventive deworming in temperate zones is carried out during the winter stall period, forced – at any time of the year. In dysfunctional farms, regular preventive deworming, biothermic treatment of manure, feeding of animals according to the diet are necessary, in the pasture season, a mixture of 1 part phenothiazine and 9 parts of feed salt is fed daily to their heart's content. As far as possible, pasture prevention measures are used – grazing lambs isolated from adult animals, providing lambs with late lambing of high-altitude or dry pastures.

MARSHALLAGIOSIS. Marshallagiosis is caused by nematodes of the genus Marshallagia (Marshallagiamarshalli), parasitic in the rennet and small intestine of sheep and goats, as well as cattle, reindeer and many wild animals.

The pathogen. Marshallagiamarshalli is a light yellow nematode, male 10-12 mm long, female 12-20 mm long, the head end is equipped with small chitinized lips inside. The male has a powerful sexual bursa with an assimitrically located dorsal rib, spicules are short, 0.20-0.23 mm long, there is no rudder.

Biology of the pathogen. The parasite is a geohelminth, the development proceeds according to a scheme common to all strongylids, without the participation

of an intermediate host. The prepatent development period is 22-25 days, the patent period is 6-8 months.

Epizootological data, pathogenesis, clinical signs, pathoanatomic changes, diagnosis, treatment and prevention are the same as in hemonchosis.

NEMATODIROSIS. Nematodirosis is caused by nematodes from the genus Nematodirus (the most common species are Nematodirusfilicollis, N.spathiger), parasitizing the small intestine of sheep and goats, as well as cattle and many wild animals.

The pathogen. Nematodirusfilicollis is a light yellow nematode with a body length of 0,7-3 cm. At the head end, the expanded cuticle forms a vesicle. The bursa consists of two wide lobes and a barely noticeable dorsal lobe, the spicules are long, filiform, connected to each other by a membrane, 07-1,1 mm long, there is no rudder.

Biology of the pathogen. Nematodiruses are a geohelminth, the development proceeds according to the scheme common to all strongylids, but with the difference that the larvae are formed inside the egg before the invasive stage.The prepatent development period is 24-26 days, the patent period is 5 months.

Epizootological data. Nematodyrosis with clinically pronounced signs occurs mainly in lambs, and often in the form of enzooties. Mortality can be significant. In sick animals, growth and development are sharply slowed down.

Pathogenesis. Nematodiruses, penetrating deep into the intestinal wall, injure tissues, resulting in erosion on the mucosa, necrosis of villi and epithelium, inflammation of the intestine. Mature helminths feed on the host's blood, causing anemia and exhaustion.

Immunity. Older lambs and adult animals develop age-related immunity to nematodirosis.

The symptoms of the disease are not characteristic. The first clinical signs appear 11-14 days after infection: shortness of breath, diarrhea, increased thirst. In the acute period of the disease, death may occur a few days after the appearance of the first clinical signs. With low AI, the disease proceeds sluggishly and is characterized by intermittent diarrhea, decreased appetite, depression, emaciation, lag in growth and development.

Pathoanatomic changes. The mucous membrane of the small intestine is thickened and inflamed, there are multiple hemorrhages on it. The corpse is anemic, emaciated, with pronounced atrophy of skeletal muscles. There are dystrophic and inflammatory changes in the peripheral nervous system, lymph nodes, myocardium, jejunum and ileum.

The diagnosis during life can be made by the Fulleborn method, since the eggs of nematodiruses differ in size and shape from the eggs of other strongylates: they stand out sharply with their large size (0,231-0.238 x 0,119-0,136 mm), light, and contain several large germ cells in the center.

Treatment and prevention is the same as with hemonosis

HABERTIOSIS. Habertiosis is caused by nematodes Chabertiaovina of the Strongylidae family, parasitizing in the large intestine of sheep, goats, cattle, camels and some wild ruminants.

The pathogen. Habertia have a rather thick, whitish-colored body up to 26 mm long. The head end is obliquely cut off, the mouth opening opens ventrally. In a large spherical oral capsule there are many small teeth, the mouth opening is surrounded by a crown of sharp triangular petals. The male's bursa is short, as if cut off. The spicules are thin. 1,3-1,7 mm, there is a steering wheel.

Biology of the pathogen. Larvae emerge from eggs isolated with feces during the day, which develop to an invasive stage in the external environment for about five days. The prepatent period of development in the host body is 32-60 days, and sexually mature parasites live in the intestine for no more than a year.

Epizootological data. Habertiosis is widespread everywhere, but clinically pronounced disease is more often registered in areas of developed sheep breeding. Sheep of all ages are susceptible to habertiosis, but as a clinically pronounced disease, it is noted mainly in young animals. The high resistance of invasive larvae to drying and low temperatures contributes to the mass infection of animals. Invasive larvae can persist in the external environment for more than a year.

Pathogenesis. Attaching a powerfully developed capsule to the intestinal mucosa, habertia injure it, thereby contributing to the penetration of pathogenic microorganisms and toxins. Young habertia are hematophagous, so anemia develops due to blood loss.

Symptoms of the disease. In animals, exhaustion, anemia, diarrhea, swelling in the submandibular space, dryness and fragility of the coat, increased thirst with reduced appetite are noted. There is protein in the urine, eosinophilia in the blood, a shift in the leukocyte formula to a shelf of nuclear neutrophils. In sheep, especially before the age of 6 months, the disease can be very severe and often leads to the death of animals.

Pathoanatomic changes are found mainly in the large intestine: anemia and swelling of the mucous membrane, the presence of mucus on it, spot hemorrhages and attached haberti. Liver degeneration, gelatinous degeneration of intermuscular, pericardial and pericardial adipose tissue are also noted.

The diagnosis during life can be made by invasive larvae, posthumously – based on the detection of haberti and pathological changes in the intestine caused by them.

Treatment and prevention is the same as with other intestinal strongylatoses of ruminants.

BUNOSTOMIASIS. Bunostomiasis is caused by nematodes Bunostomum trigonocepalum and Bunostomum phlebotomum of the Ancylostomatidae family. The species Bunostomum trigonocepalum is specific to sheep, and the species Bunostomum phlebotomum is specific to cattle. Parasites are localized in the small intestine.

The pathogen. Nematodes are whitish, up to 2,6 cm thick. Bunostomes are characterized by a curved dorsally head end, the presence of a funnel-shaped oral capsule with a dorsal groove and two semilunar cutting plates at the edge of the oral opening. The male has a bursa with an asymmetric dorsal lobe, spicules are equal,

brown in color, Bunostomum trigonocepalum 0,6-0,64 mm, Bunostomum phlebotomum 3,5-4 mm in length, there is no rudder.

Biology of pathogens. In the external environment, larvae hatch from eggs, which mature to the invasive stage within 3-7 days.

Animals become infected by ingesting larvae or by penetrating them through intact skin (most often). In the host body, the parasite develops for about 40-46 days, and according to some data -103 days. The patent development period is a year or more.

Epizootological data. Bunostomiasis occurs in sheep everywhere, but with different extent and intensity of invasion depending on natural and clinical conditions. EI in Uzbekistan is 83.1%, the intensity of infection can be up to 5-6 thousand copies, sheep of all ages are infected, but young animals are more intensively infected. Animals get infected in the pasture. In some foci, the disease occurs in the form of enzooties, the case of animals at the same time can reach 60-80% of the number of cases.

Pathogenesis. Bunostomes are hematophagous, attaching to the mucus lining of the intestine, injuring it, thereby contributing to the penetration of pathogenic microorganisms and toxins, as in other intestinal strongylatoses of ruminants.

Symptoms of the disease. Debilitating diarrhea, anemia and edema, progressive emaciation. Young animals lag behind in development.

Pathoanatomic changes. At the autopsy of corpses, a sharp picture of exhaustion, hydremia and cachexia, serous infiltration of the subcutaneous fangs are noted. Bunostomiasis is characterized by acute or chronic inflammation of the mucous and muscular membranes, sometimes purulent or phlegmonous in nature. There are massive small ulceration in the places of fixation of parasites.

The diagnosis during life is made by the method of growing and determining invasive larvae, postmortem – based on the detection of bunostomas and pathoanatomic changes.

Treatment. Phenothiazine is used at a dose of 1.0 g / kg, 4-carbon chloride (3 ml per adult sheep, 1-2 ml for young animals), naphthamone, copper sulfate, nilverm, fenbendazole (panacur), rintal (febantel), albendazole and its analogues, mebenvet, tafen and tividin.

Prevention. Given the possibility of infection through the skin, it is not recommended to keep sheep in wet weather in fecal-contaminated pens, it is necessary to change the rear more often, using dry elevated places for this purpose. Preventive deworming and chemoprophylaxis also give a positive result.

ESOPHAGOSTOMIASIS OF RUMINANTS. Esophagostomiasis is caused by nematodes of the genus Oesophagostomum of the family Trichonematidae. Sheep have O.venulosum and O. columbianum, cattle have O. radiatum. They parasitize in the large intestine.

Pathogens. Esophagostomes are thick, white-colored nematodes reaching 20 mm in length. The oral opening is located terminally and is provided with a small narrow oral capsule at the head end of the cuticular vesicle, separated from the body

by a ventral furrow. Males have equal spicules, relatively thin, O.venulosum 1.1-1.5 mm,O. columbianum 0.77-0.86 mm and O.radiatum 0.7-0.8 mm in length.

Biology of pathogens. The formation of larvae before the invasive stage in the external environment lasts 7-8 days. In the host body, different types of esophagostomes develop in different ways. The larvae.venulosum is introduced into the mucous membrane of the small intestine and, after lingering in it for a day, enters the lumen of the intestine, moves into the large intestine, where after 24-30 days they develop into adult parasites. The larvae of O.columbianum and O.radiatum follow the same path, but some of them encyst in the mucous membrane of the small intestine, forming nodules. Some larvae remain viable in these nodules for a long time (years); they can leave the cysts and develop until puberty. Similar nodules with larvae are formed in calves in the caecum and colon, while the larvae enter the lumen of the large intestine and reach puberty 32-43 days after infection.

Epizootological data. Animals are infected mainly on pasture in the early spring and summer seasons. In some dysfunctional farms, the number of livestock affected by esophagostomiasis can reach 100% with AI up to 400 copies. Lambs younger than 3 months of age do not suffer from esophagostomosis.

Pathogenesis. The invasion is most difficult during the period when the parasites are located in the intestinal wall (nodular disease) and gradually exits it into the lumen. Uzeks can be suppressed due to the introduction of pyogenic microflora into them by the larvae of esophagostomes. The mechanical and toxic influence of parasites leads to inflammation of the intestine, violation of its physiological functions.

Symptoms of the disease. There are two periods of the disease: larval – parasitization of larvae in the intestinal mucosa and imaginal –parasitization of mature helminths in the intestine. In the first period, digestive disorders are noted, a decrease or lack of appetite, the release of liquid feces with an admixture of blood and mucus, emaciation, sometimes signs of mild colic appear (animals worry, push their hind limbs). Sometimes the body temperature rises slightly, anemia develops. In the second period, the disease proceeds subclinically, sometimes diarrhea appears in animals.

Pathoanatomic changes. Upon autopsy, hyperemia and swelling of the intestinal mucosa are detected, nodules ranging in size from poppy seeds to beans and hazelnuts; nodules in the mucous and submucosal layers often have a necrotic dark brown or black apex. The largest number of nodules is observed along the mesentery attachment site; with a very strong lesion, the nodules are located throughout the intestine, sometimes merging into conglomerates. With high AI, there are thousands of nodules that can cause intestinal intussusception. When cutting nodules having a thin connective tissue capsule, they reveal a caseous or purulent mass with esophagostom larvae.

The diagnosis during life is made by the method of growing and determining invasive larvae in feces, posthumously – by finding characteristic nodules and parasites themselves on the mucosa.

Treatment. Phenothiazine is used at a dose of 0,5 g / kg, thiabendazole, nilverm, fenbendazole (panacur), rintal (febantel), albendazole and its analogues, mebenvet, tafen and tividin.

Prevention. Preventive deworming and chemoprophylaxis, as with other strongylatoses of ruminants. If possible, lambs are grazed separately from sheep for them, pastures that have not been used for adult animals for a year.

ESOPHAGOSTOMIASIS OF PIGS. Pig esophagostomiasis is caused by nematodes Oesophagostomumdentatum from the family Trichonematidae, parasitizing in the large intestine.

The pathogen. Esophagostomes are small nematodes, 7-14 mm long, white in color, the mouth opening is equipped with a small oral capsule. In males, the spicules are equal, thin 1,16-1,3 mm in length.

Biology of the pathogen. The development of larvae to the invasive stage in the external environment lasts 7-8 days. Invasive larvae, once in the intestine, penetrate into the thickness of the mucous membrane. A nodule forms around the larva. After 23 days, the larvae enter the intestinal lumen and reach sexual maturity in 1,5-2 months.

Epizootological data. In pigsties, the infection of animals is insignificant, since manure is constantly irrigated with urine and larvae do not develop in it. Adult pigs are most intensively infected, 2-4 month-old young are infected less. Invasive larvae are resistant to environmental factors, they can live in water for up to a year.

Pathogenesis. Basically similar to that of esophagostomosis of ruminants.

Symptoms of the disease. At the beginning of the disease, the appetite of animals decreases, diarrhea appears, later blood and mucus are contained in the fecal masses. Pigs are exhausted and die. This is observed during the period when helminths are in nodules (nodular disease). With low AI, the disease proceeds chronically, without pronounced symptoms.

Pathoanatomic changes. Upon autopsy, hyperemia and swelling of the mucous membrane of the large intestine are detected. In the early period, the nodules are reddish in color, later they become stormy. When the nodules are crushed, a larva can be found in them. Erosion and ulcers often appear on the site of the formed nodules.

The diagnosis during life is made by growing and identifying larvae that have a double cuticle cover and a long awl–shaped tail, and posthumously - when the parasites themselves and nodules are found on the intestinal mucosa.

Treatment. Tiabendazole is prescribed at a dose of 0,05 g / kg for 5 consecutive days; nilverm – sows 1,0 g per head for 5 days in a row; piperazine adipinate – 50 g per animal once a day, 3 days in a row, chlorophos – as with ascariasis.

Prevention is basically the same as with ascariasis.

TOPIC № 12. STRONGYLATOSIS OF THE RESPIRATORY ORGANS. DICTYOCULOSIS.

Lecture plan:

1. Strongylatoses of respiratory organs
2. Dictyoculosis
3. Dictyoculosis of sheep and goats

Keywords: Strongylata Dictyocaulidae, Protostrongylidae, Metastrongylidae, Crenosomatidae, Syngamidae, Dictyocaulus, D. filarial, D. viviparous, D.eckerti, D.arnfieldi, D.cameli, bronchus.trachea, prepatent, patent, deworming, planned, forced.

In the respiratory organs of agricultural animals, nematodes of the Strongylata suborder belonging to the family parasitize: Dictyocaulidae, Protostrongylidae, Metastrongylidae, Crenosomatidae and Syngamidae.

Dictyocauluses parasitize in the trachea and bronchi of cattle and small cattle, reindeer, camels and horses. Males are characterized by a well-developed rib bursa with short cellular spicules. Geohelminths.

Protostrongylids are thin nematodes, the sexual bursa in males is strongly recoded or even absent. The spicules are equal, often with flattened extensions at the ends. The vulva in females is located near the anus. They parasitize in the alveoli, bronchioles and parenchyma of the lungs in small cattle. Biohelminths, intermediate hosts–terrestrial mollusks.

The metastrongylids at the head end have two powerful three-lobed lips. The bursa of the male is well developed, but with shortened ribs. The vulva in females is located at the posterior end of the body near the anus. They are parasitic in domestic and wild pigs in the bronchi. Biohelminths, intermediate hosts – earthworms.

Crenosomes are characterized by cuticular ring–shaped folds in the anterior part of the body, the bursa of the male is well developed, they parasitize in the bronchi and trachea of fur-bearing animals 6 foxes, Arctic foxes, Ussuri raccoons, sables, minks, cupids, etc., biohelminths, intermediate hosts -nasmy mollusks.

Syngamuses in the sexually mature form are constantly in a mated state. The male is several times smaller than the female, the spicules are very short, the vulva of the female is located in the anterior half of the body, parasitic in the tran of domestic and wild birds. Geohelminths develop with or without the participation of reservoir hosts.

DICTYOCULOSES. Dictyocaulosis of animals is caused by parasitization in the trachea and bronchi of strongylates of the genus Dictyocaulus, D. filaria parasitizes in sheep and goats, D. viviparous in cattle, D. eckerti in reindeer, D. arnfieldi in horses and donkeys, D. Cameli in camels.

Dictyocauloses are widespread all over the world and cause significant economic damage. Losses consist of the death and forced slaughter of sick animals, the sick young develop poorly, lag behind in growth, productivity decreases in adult animals, the quality of meat, fat, wool and skin deteriorates. A decrease in

productivity occurs not only with clinically pronounced dictyoculosis, but also with a subclinical form. Invaded animals are more susceptible to other diseases and are more difficult to tolerate.

Dictyoculosis of sheep and goats. Dictyoculosis of small cattle is caused by nematodes D. filaria, parasitizing in the trachea and bronchi of sheep, goats and wild ruminants.

The causative agent is a large filamentous nematode, milky white in color, the intestine shines through in the form of a dark line. Male 3-8 cm and female 5-15 cm in length.They have 4 small lips and a very small oral cavity. Spicules are short, closely brown, boot-shaped, 04-0.64 mm long. The vulva in females is located slightly behind the middle of the body.Eggs in size 0,112-0,138-0,069-0,090 mm and contain fully formed larvae when laid.

Biology of the pathogen. When coughing, the eggs fall into the mouth, and the animals swallow them. Larvae from eggs can hatch in the lungs, but usually this happens in the host's VZHTK. Larvae of the I –stage of D. filaria with a length of 0.54-0.55 (0.58) mm are excreted with feces. They are easily differentiated by the presence of a small button-shaped thickening at the head end and by numerous brownish-colored food granules in intestinal cells.After 1-2 days, the larvae molt for the first time, and then after 4-5 days – for the second time. The faces of the III stage are covered with two covers. Larvae reach the invasive stage at a temperature of 270C in 6-7 days, at a temperature of 5-100C, this period increases to 15-17 days.

Hosts become infected by ingesting invasive larvae with food. The larvae penetrate the intestinal wall for three days and then through the lymphatic vessels into the mesenteric lymph nodes, where they develop and molt for the third time on the 4th day after infection. Larvae migrate through the lymphatic and blood vessels to the lungs, where they linger in the capillaries and destroying them, exit into the alveoli, bronchioles and bronchi. Development to the sexually mature stage is about 4 weeks. Sometimes this period stretches up to 3-4 months. Dictyocauluses parasitize in the body of sheep from several months to 1.5-2 years.

Epizootological data. Dictyoculosis mainly affects young animals up to one year old. Adult animals are parasitic carriers and serve as a source of invasion. The spread of the disease is facilitated by the joint maintenance and grazing of adult animals and young animals. Chgnchta become infected with dictyoculosis when grazing and keeping in the fields where sick animals used to be. When eating green fodder mown in meadows where the infected cattle grazed. In the republics of Central Asia, including Uzbekistan, there is one peak of invasiveness.

Pathogenesis. The effects of dictyocauluses on the host organism are composed of mechanical and toxic effects and open the gates to secondary infections.

Immunity. Some lambs have primary individual innate immunity, which is expressed in weak primary infection and rapid release from parasites. Age-related immunity is also well expressed - in animals older than a year, the body's resistance to dictyoculosis increases. Acquired immunity after a natural illness or artificial immunization can be quite persistent. It is expressed either in the form of complete immunity to re-infection, or in a decrease in the survival rate, lengthening the

development of the pathogen to the sexually mature stage, shortening the life of the pathogen and a sharp decrease in oviposition by females. Immunity acquired as a result of natural illness, with proper feeding, can last 2.5 years or more in sheep, and with artificial infection – for several months.

The symptoms of the disease develop gradually and the degree of their severity depends on the dose of infection and the state of the animal's body. At first, patients have a weak cough. Which increases and becomes painful as bronchitis develops. The cough increases during movement after the previous rest, as well as at night. Serous or serous-purulent outflows are released from the nostrils, which, drying out, form crusts. Animals experience severe itching and rub their noses on the ground. Breathing becomes difficult and rapid, the temperature is normal, but in severe form, complicated by infection, atypical fever develops, visible mucous membranes become cyanotic, swelling appears in the eyelids, lips, underbelly and less often legs. Animals gradually lose weight, weaken. Death can occur as a result of complete exhaustion and blockage of the airways by tangles of helminths or due to the development of irreversible pathological processes with complications of secondary infection.

Pathoanatomic changes. The infected lambs have an acute and chronic course of pathohistological processes.

With an acute course on the 9th-10th day after infection, hyperemia is noted in the lungs. Hemorrhages, thickening of alveolar hemorrhoids, serous hemorrhagic edema of tissue. On the 14th-15th day, microbronchitis, bronchitis and peribronchitis develop. The process intensifies and turns into acute catarrhal hemorrhagic focal bronchopneumonia, often complicated by secondary infection with the development of purulent catarrhal pneumonia.

During the transition to a chronic form, catarrhal-desquamative phenomena develop in the bronchi. In the future, connective tissue grows around the bronchi, atelectasis and pulmonary ephmysema develop.

Diagnosis. A preliminary diagnosis based on clinical signs and epizootological data is clarified by helmintholaroscopic examination of animals using the Berman-Orlov, Vida method or the UsNIIV method. The larvae have a button-like thickening at the head end, and the tail end is blunt-rounded. The size of the larvae is 0,54-0,55 x 0.025 mm.

A postmortem diagnosis is made based on the detection of sexually mature dictyocauluses in the trachea and bronchi, their larval forms in the lung tissue and characteristic pathomorphological changes. If no dictyocauluses are detected during autopsy, then the lung tissue is examined for the presence of larvae of parasites according to Berman-Orlov.

Treatment. For deworming, nilverm, levomizole, mebendazole, fenbendazole (panacur), rintal (febantel), ivomek, baymek, ivermectin and an aqueous solution of iodine are used according to the instructions.

Prevention. Carry out a set of measures aimed at destroying the invasive principle in the host body, as well as in the external environment. The complex of preventive measures includes therapeutic and prophylactic deworming, pasture and

chemical prevention and measures of a general sanitary order aimed at strengthening the animal's body.

TOPIC № 13. TELYAZIOSIS OF CATTLE AND TRICHINOSIS OF PIGS.

Lecture plan:

1. Telyaziosis of cattle
2. Pig trichinosis

Keywords: Spirurata, Thelaziidae, Thelazia, Th. rhodesi, Th. gulosa, Th. skrjabini, Trichocephalata, Trichonellidae, Trichinella, Trichinellaspiralis, geohelminth, biohelminth, prepatent, patent period of development, eggs, viviparous, larva, deworming.

Telyazioses are helminthic diseases of cattle, manifested by conjunctivitis-keratitis. These diseases are caused by three species of nematodes of the genus Thelazia of the family Thelaziidae from the suborder Spirurata

Th. rhodezi, Th. gulosa, Th. Skrjabini localized in the conjunctival ieshka and under the third eyelid; Th. Gulosa and Th. Skrjabini – in the ducts of the lacrimal gland and the lacrimal nasal canal.

Telazia also parasitize horses – Thelazia lacrimalis, pigs - Thalazia erschovi, dogs - Thalazia callipaeda.

Pathogens are small nematodes up to 21 mm long. Rhodezi is a body surface with rough transverse striation, which gives the parasite a jagged appearance. The mouth opening leads to a small oral capsule. Males have two unequal spicules: the right one is 0.11 mm, the left one is 0.624-0.846 mm long. Th. Gulosa without rough transverse striation of the cuticle, but differs in the presence of a wide oral capsule, the male spicules are unequal left 0.129-0.165 mm, right - 0.608-0.692 mm in length. Th. Skrjabini is also devoid of rough transverse striation of the cuticle, the oral capsule is very small. Males have almost equal spicules: right -0.082 mm, left -0.113-0.185 mm in length.

Biology of pathogens. The biohelmitt parasite develops with the participation of an intermediate host, which are various species of flies: Muscaautumnalis,M. convexifrons and M. amis.

Fertilized female telyazii spawn a large number of live mobile larvae that fall into the lacrimal outflow and are swallowed by flies. Larvae develop in the body of flies to the invasive stage within about 3-4 weeks. Invasive larvae enter the abdominal cavity of the fly and move into its proboscis. When the fly is near the eye, the larvae independently crawl out of its proboscis and penetrate into the conjunctival sac, where after 15-20 days they grow into sexually mature females and males. Telyazia live in the eye for several months, and individuals live for more than a year.

Epizootological data. Telaziosis enzooties among cattle are observed in summer, in June-August. Animals become infected by direct contact with intermediate hosts in the pasture or ward. Telazii can be found in the eye of animals

at any time of the year, but the greatest number of them are observed in summer. Therefore, telyaziosis is a seasonal disease. It is found everywhere.

The pathogenesis is insufficiently studied. Telyazii have a mechanical effect on the conjunctiva and cornea, which is accompanied by the introduction of banal microflora and the development of conjunctivitis of a serous or purulent nature. The damaged cornea becomes cloudy, and the inflamed conjunctiva is so much lowered that the eyelids completely cover the diseased eye. Erosions form on the cornea. There may be corneal perforation, lens damage and the development of fibrinous hemorrhagic iridocyclitis.

With the development of inflammatory processes, a rounded or oval ulcer forms in the clouded cornea, the eyeball protrudes strongly. Over time, the cornea gradually heals, diffuse opacity resolves, the eyes acquire a normal appearance, a white spot of various sizes remains in place of the former ulcer.

Immunity in sick animals does not develop.

Symptoms of the disease. The most characteristic signs: lacrimation, photophobia. Redness and swelling of the conjunctiva, edema of the eye, in advanced cases – keratitis, ulcers on the cornea, an eyesore. The disease usually lasts 1-2 months, especially it occurs in young animals up to 4 months and older. In addition, the animals worry, shake their heads, they note a weakening of appetite and a decrease in milk yields.

The degree of clinical manifestation does not always correspond to AI, since in some cases, in the presence of a large number of parasites, there are no symptoms of the disease.

Pathoanatomic changes are characterized by the presence of conjunctivitis, keratitis, opacification and ulceration of the cornea, damage to the lens.

The diagnosis is made on the basis of clinical signs and studies of flushes from conjunctival cavities. To identify sick animals in the summer, a monthly clinical examination of cattle is carried out.

Treatment. It is recommended to wash the eye cavity with one of the following means: 2-3% boric acid solution, 3% lysol emulsion, 3% ichthyol emulsion, ditrazine citrate can be applied at a dose of 0.015 g / kg, subcutaneously twice with an interval of 1 day or in the periorbital also with an aqueous solution of iodine (crystalline iodine –th g, potassium iodide – 2 g and boiled water – 2 liters).

In case of complication of telyaziosis with a second infection (purulent conjunctivitis, keratitis) prescribe symptomatic treatment (penicillin, sulfonamide preparations, eye ointments and eye medicinal films (GLP).

Prevention. Preventive deworming of the entire livestock is carried out during the period of stable maintenance or in the spring before the beginning of summer of cow flies, repeated every 7-10 days. In summer, during the hot time of the day, during the period of the greatest activity of flies, it is recommended to keep animals indoors or under a canopy. Night grazing of animals, biothermal treatment of manure, full feeding of animals according to the diet.

TRICHINELLOSIS, Trichinellosis is an acute and chronically occurring anthropozoic, naturally focal, invasive disease with pronounced allergic phenomena

caused by nematodes from the family Trichonellidae of the suborder Trichocephalata, The genus Trichinella species Trichinella spiralis (pork trichinella), Trichinella native (trichinella from predatory animals of Eurasia,Trichinella nelsoni (from predatory Africa), Trichinella pseudospiralis (from the striped raccoon from Dagestan). Trichinella pseudospiralis differs from the previous three by its smaller body size and the fact that its larvae are not encapsulated in the muscles, in addition, Trichinella pseudospiralis completely completes the development in the body of birds (chickens, ducks, partridges, sparrows, etc.).

Adult trichinella parasitize in the small intestine of animals and humans, and larvae – in the striated muscles of these the same organisms. To date, more than 100 species of mammals that are hosts of trichinella have been registered.

The most common trichinosis occurs in pigs, dogs, wolves, foxes, cats, bears, rats, mice. Wild boars, badgers, arctic foxes, minks, sables, ferrets, hedgehogs, tigers and many wild carnivores and rodents are susceptible to it. Helminthiasis has been recorded in marine mammals: whales, walruses, seals.

Economic damage. The economic damage from trichinosis is very great: trichinosis carcasses of animals, regardless of the degree of infection, are destroyed. This helminthiasis is also very dangerous for humans: trichinosis in humans is very difficult, difficult to treat and very often ends in death.

The pathogen. These are very small nematodes. Male 1.4-1.6 mm long, 0.14 mm wide. Two pairs of papillae are located at the posterior end of the body and in the gap between the two lobes behind the cloaca. Females are twice as large as males -3-4 mm, viviparous

Biology of the pathogen. The parasite is a biohelminth.The same animal is initially a definitive, and then an intermediate host of the parasite. Animals become infected by eating trichinella meat, which contains live encapsulated larvae of trichinella. In the stomach, the capsule is destroyed, a larva about 1 mm long comes out of it.

These larvae develop rapidly in the small intestine until puberty. Mature eggs in the ma of samou appear after 44 hours after infection.

Female trichinella are introduced with their head end into the mucosa, and on the 4th day after infection, live larvae hatch. One uterus spawns up to 2,100 larvae 1.2 mm long and 0.006 mm wide. There is a stiletto on the head end. The larvae penetrate into the lymphatic system, then into the circulatory system and are carried throughout the body by the blood current. They linger in striated muscles, penetrate under the sarcolemma of muscle fibers, grow, then curl into a spiral. Larvae become invasive 17.5 days after infection. From the 4th to the 12th week, capsules are formed around the larvae, which after 6 months in pigs begin to become deified; this process ends completely after 15-16 months.

In the muscles, encapsulated trichinella larvae can remain viable for 25 years. Female trichinella in the intestines of the host lag behind for up to 8 weeks, after which they die.

Epizootological data. Trichinosis is common everywhere. Trichinosis of pigs is of great practical and health importance. Pigs become infected by eating the

corpses of infected rats, cats, wild animals, as well as raw or poorly cooked slaughter confiscated and kitchen waste. A person is infected with trichinosis by eating pork, bear meat or other meat with trichinella larvae.

Pathogenesis. Trichinella have mechanical and toxic effects, as well as sensitization of the body with pronounced allergic phenomena.

Immunity. The formation of immunity in trichinosis is very difficult and depends on a number of hormonal and cellular factors.

Symptoms of the disease. In pigs, trichinosis occurs without pronounced clinical signs. Note a short-term increase in body temperature, depression, muscle soreness, intermittent lameness. More serious changes occur in the blood: pronounced eosinophilia and an increase in ESR.

Pathoanatomic changes. The normal structure of muscle fibers is disrupted: at the locations of capsules with larvae, the fibers expand, acquire a fusiform appearance, their transverse striation gradually disappears, the nuclei increase and the muscle fibers disintegrate, turning into a granular mass. With high AI, small, whitish-colored seals with poppy seeds are noticeable in skeletal muscles, representing capsules with trichinella larvae.

Trichinella are localized mainly in the legs of the diaphragm, in the muscles of the tongue, esophagus, larynx, intercostal, thoracic, in rare cases they are found in parenchymal organs, in the muscle layers of lard.

Diagnosis. Lifetime diagnosis of trichinosis can be carried out by immunobiological methods: serological reaction of indirect immunofluorescence and reaction of ring precipitation in the capillary.

Postmortem diagnosis is performed by trichinelloscopy. After a copressor study of pork meat samples (24 slices from the legs of a diaphragm the size of a wheat grain), it is used as the most accurate method of digesting muscle samples in artificial gastric juice, which allows detecting trichinella with weak invasion, which are not always detected by conventional trichinelloscopy.

I**n the differential diagnosis** of trichinella, it should be distinguished from cysticerci and Mischer sacs, which are the simplest parasites – sarcocysts, sarcocysts are not enclosed in capsules, but have their own thin shell. The shape of the Mishera pouches is very diverse: from elongated, spindle-shaped, sickle-shaped to oval. Their sizes range from 0,005 to 4-5 mm in length. Capsules of trichinella have a lemon-shaped shape, reach a length of 0,68 mm and a width of 0,37 mm; one, rarely two spirally coiled trichinella are visible inside.

When the capsules are desaturated, the muscle sections are enlightened with 5-10% hydrochloric acid at an exposure of 1-2 hours, followed by glycerin.

Animal treatment has not been developed. In humans, thiabendazole and other benzimidazoles are used.

Prevention. To prevent infection of people with trichinosis, all carcasses of pigs, wild boars, bears, and nutria, without exception, are necessarily examined for this helminthiasis.

At any intensity of neighing, trichinellosis of animal carcasses is subject only to technical disposal, they are strictly forbidden to be used for food. By-products

having muscle tissue are also disposed of. The fat is peretapvilayut at 100^0C for 20 minutes. Internal fat is sold without restrictions.

If a trichinella carcass is found, the farm from which the animal came is informed about it in order to take appropriate preventive measures. On animal farms, it is forbidden to feed carcasses of animals, dogs, cats without first examining them for trichinosis and disinfection, the corpses of rats are burned. It is not necessary to feed pigs and fur-bearing animals slaughterhouse waste without thorough decontamination.

Strict deratization measures at pig farms, slaughterhouses, meat processing plants prevent the spread of trichinosis. For the destruction of rodents, zookumarin and other means are used. It is necessary to combat the vagrancy of pigs on the territory of settlements.

Any dysfunctional point for trichinosis is considered as a natural focus of the disease, a complex of veterinary-saninary and medical preventive measures is carried out in it.

TOPIC № 14. VETERINARY PROTOZOOLOGY. PYROPLASMOSIS, FRANCAIELLOSIS AND BABESIOSIS OF CATTLE.

Lecture plan:

1. General characteristics of veterinary protozoology, the main stages of its development.
2. Morphology, biology and systematics of parasitic protozoa
3. Piroplasmidoses of animals (Piroplasmosis, babesiosis and francaiellosis).

Keywords: Veterinary protozoology, Protozoa, protozoosis, nucleus, cytoplasm, cytoplasmic network, cilia, flagellum, pseudopodium, pinocytosis, phagocytosis, aerobic, anaerobic, chemotaxis, thermotaxis, asexual, sexual, copulation, conjugation, Protozoa, Sarcomastigophora, Mastigophora, Apicomplexa, Ciliophora, Piroplasmida, Babesiidae, Piroplasma, Piroplasma bigeminum, Babesia bovis, Francaiella colchica, Boophillus calcaratus, Haemaphysalis punctata, Rhipicephalus bursa, azidine, diamidine, DAT, norotrip, sulfantrol, hemosporidine, polyamidine, ethidine.

Protozoology (Protozoa type) is the science of the simplest single-celled animal organisms. Veterinary protozoology studies protozoa that parasitize animals and cause diseases in them – protozooses. Its task is to study the morphology and biology of the parasite, determine its type and ways of infecting animals. At the same time, protozoology studies the pathogenic effect of the pathogen on the animal body, methods of diagnosing diseases, specific and pathogenetic therapy, as well as the prevention of these diseases.

I.The main stages of the development of veterinary protozoology. Single-celled animals as microscopic organisms were first studied by microbiology. Later, at the end of the XIX and the beginning of the XX century, the protozoans were

separated into an independent discipline – protozoology. In those years, remarkable discoveries were made that were of great importance in the development of the doctrine of pathogenic protozoa. Pathogens of trypanoses, malaria, pyroplasmidosis, eimeriosis and other diseases have been identified.

Protozoological research in the field of medicine and veterinary medicine has led to a number of discoveries of pathogens of protozoal diseases that caused epidemics and epizootics in many countries. Thus, the "surra disease" was widely spread in India. Veterinarian Evans in 1880 Revealed the etiology of this disease and established that its causative agent is trypanosoma. In 1888, Babes in Romania discovered a pathogen called "bloody urine" in cattle. In 1889 Smith and Kilborn also found pyroplasmas in the blood of cattle, thereby proving that Texas fever in America is caused by the blood parasite P.bigeminum. In 1893 These authors have established that pyroplasmas are transmitted from sick animals to healthy pasture ticks. It was a new idea about unicellular organisms as pathogens of human and animal diseases.

In 1891. Russian scientist D.L.Romanovsky created a special method of coloring protozoa, which was a kind of impetus in the development of protozoology.

The works of I.I.Mechnikov and D.L.Romanovsky had a huge influence on the development of protozoological science, which for the first time proved that the causative agent of human malaria belongs to the protozoa. A certain role in the development of therapy for protozoan diseases of humans and animals was played by the works of P. Ehrlich and D.L.Romanovsky on the study of the mechanism of action of medicinal preparations on pathogens. I.I. Mechnikov's research on immunity also contributed to this.

In Russia, the work of well-known researchers V.Y.Danilevsky, E.N.Dzhunkovsky and I.M. Luz, E.I.Marcinovsky, V.L.Yakimov, A.V.Blitzer, N.A.Saarov and others had a significant impact on the development of protozoology, who identified new pathogens of protozoal and spirochaete animal diseases. In 1898, A.I. Kachinsky first described the pyroplasmosis of cattle in Russia. In 1903, E.P.Dzhunkovsky and I.M.Luz discovered theileriosis of cattle in Transcaucasia. In 1096, A.V. Blitzer and E.I. Marcinovsky discovered pyroplasmosis of horses in Ryazan. In 1911 Student Dementiev first found the causative agent of pyroplasmosis in pigs.

In 1913, V.L.Yakimov led an expedition to Turkistan, where he conducted extensive research on trypanosomiasis, leishmaniasis, priroplasmidosis, collected and described the fauna of vector ticks. The main research on veterinary protozoology by V.L.Yakimov and his numerous students was done after the October Revolution. In the twenties, V.L. Yakimov created a large scientific center in Leningrad for the study of protozoal animal diseases.

At the same time, a second scientific center is being created in Moscow under the leadership of first A.V. Belitzer, and then A.A.Markov, where protozoal diseases of horses, camels, cattle and small cattle are studied. Subsequently, the issues of systematics, pathogenesis, immunity, ultrastructure of protozoa, etc. are being developed.

Currently, the All-Union Society of Protozoologists is working under the leadership of Yu.I.Polyansky. In many research institutes and universities, veterinary protozoology studies pathogens of protozoal diseases and measures are being developed to combat these animal diseases. Such veterinary protozoologists as A.G.Gafurov work in our Republic. Kuldoshev O., Rasulov U., Abdurasulov Sh.

II. Systematics of pathogenic protozoa. Based on data on the morphology and biology of protozoa, the **International Committee on Protozoan Toxonomy** in 1980 recommended combining Protozoa into a sub-kingdom. The latter is divided into 7 types. Pathogens of animal diseases belong to 3 types:

1. **Sarcomastigophora** - flagellates-

2. **Apicomplexa** – its representatives at the anterior end of the body have a so-called apical complex.

3. **Ciliophora** – ciliated.

Veterinary parasitology also considers unicellular

the organisms are spirochaetes and anaplasmas, which are currently classified as Rickettsi–like organisms.

III. Piroplasmidiosis of animals (Piroplasmosis, babesiosis and francaiellez). The class Sporozoa type Apicomplexa includes several orders, the orders Piroplasmida and Coccidiida have veterinary significance. The order Piroplasmida consists of the family Babesiidae and Theileriidae. **The Babesiidae** families are united by the genera: Babesia, Piroplasma and Francaiella, and the Theileriidae families are Theileriidae Theileria and Nuttalia.

The family Babesiidae includes three genera Babesia, Piroplasma and Francaiella (abroad and some of our scientists refer this genus to the genus Babesia). Two species belong to the genus Babesia: Babesia bovis, Babesia ovis – parasites of pear-shaped, rounded or ring-shaped pear-shaped, their size is less than the radius of the erythrocyte, paired pear-shaped forms are located at an obtuse angle, often along the periphery of the erythrocyte.

The genus **Piroplasma** includes: Piroplasma bigeminum (parasite of cattle and buffaloes), Piroplasma caballi (parasite of horses and mules), Piroplasma ovis (parasite of sheep and goats), Piroplasma canis (parasite of dogs), Piroplasma traurmanni (parasite of domestic and wild pigs). Parasites of this genus are pear-shaped rounded annular or irregular, the size of single and paired pear-shaped pyroplasmas is larger than the radius of the erythrocyte, paired forms, connected by narrow ends, are located at acute angles in the center of the erythrocyte.

The genus Francaiella unites: Francaiella colchica (cattle parasite) and Francaiella tarandi rangiferis (reindeer parasite). Parasites of this genus are pear-shaped, oval or lanceolate, the size of single and paired pear-shaped francaiella is approximately equal to the radius of the erythrocyte or less, paired forms are located at an obtuse angle in the center of the erythrocyte.

Piroplasmosis of cattle is an acute disease caused by Piroplasma bigeminum and it is accompanied by fever, anemia, jaundice and hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems.

Distribution. Pyroplasmosis is widespread everywhere, their infection rate is 5-15, sometimes up to 40% and is a seasonal disease, found mainly in the southern part of the former Soviet Union, mainly within the North Caucasus, Transcaucasia, Crimea, the republics of Central Asia, including in our Republic, and the south of Kazakhstan, in the hot season of the node, that is the spring-summer period of the year depends on the range of vector ticks.

Economic damage. The economic damage from pyroplasmosis is very great: the productivity of animals decreases, milk yield of milk cows decreases by 25-40%, the quality of meat deteriorates. There are abortions in the second half of pregnancy, as well as the costs of treating sick animals.

The pathogen. Piroplasma bigeminum is oval, rounded, pear–shaped, paired-shaped, ring-shaped or elongated–oval, the size of single forms is from 2.2 to 6 microns, paired pear-shaped - up to 4.5 microns. There are usually one, two and rarely more parasites in the erythrocyte. Paired pear-shaped ones are connected by narrow ends and are located in the erythrocyte at an acute angle. At the beginning of the disease, single forms are more common, and then paired ones prevail. Infection of erythrocytes is 5-15%, sometimes up to 40%.

Biology of the pathogen. In erythrocytes, pyroplasmas multiply by simple division into two or budding. In the future, pyroplasmas multiply in ticks, where schizogonal division occurs. Carriers of pyroplasmas are **single-host mites – Boophilus calcaratus,two-host mites - Rhipicephalus bursa,and three-host mites - Haemaphysalis punctata.** Transmission of Piroplasma bigeminum by ticks proceeds according to the transovarial type. Boophilus calcaratus mites transmit pyroplasmas in the nymph phase, and Rhipicephalus bursa, Haemaphysalis punctata – in the imago phase.

Epizootological data. Pyroplasmosis is widespread mainly in the southern part of the country.The pathogen is transmitted by pasture mites Boophilus calcaratus, Rhipicephalus bursa and Haemaphysalis punctata. Pyroplasmosis is susceptible except for cattle, buffaloes and zebu.

Pyroplasmosis is an enzootic disease limited to the area of vector ticks. Biotopes of Boophilus calcaratus are found on untilled pastures with sufficient soil moisture, herbaceous or shrubby vegetation or sparse forest. Pyroplasmosis belongs to pasture summer, seasonal diseases. Cattle are infected on natural pastures. In the range of Boophilus calcaratus ticks, one pyroplasmosis in cattle is relatively rare, most often it occurs together with Francaiella - Francaiella colchica.

Symptoms of the disease. With natural infection with pyroplasmosis, the incubation period in animals is 9-14 (and sometimes up to 24) days, and with experimental infection of animals, the incubation period lasts 7-8 days. In young animals up to one year old and native adult livestock, pyroplasmosis manifests itself in an atypical form.

In adult cattle that do not have premunition, the symptoms of pyroplasmosis are typical. During the first day, the body temperature rises to 41-42.40 C and remains at this level throughout the entire period of the disease. A pronounced depressed state is characteristic, animals take food reluctantly, but thirst

on the first day can be intensified. Cows have sharply reduced milk yields. The pulse in patients is up to 100-120 minutes, heart tremors are felt by the hand, breathing is rapid, intestinal peristalsis is intensified on the 1-2 day of the disease, the mucous membranes of the eyes first turn red, then become anemic, with a jaundice tinge. On the 2nd day of the disease, the symptoms are most pronounced. The animals stand dejectedly, with their heads down, tears often flow from their eyes. Feed and water are taken sluggishly, sometimes they refuse to feed at all. Intestinal peristalsis weakens, and scar contractions become rare. Urine is initially yellow in color, but with an acute course of pyroplasmosis, it becomes red on the 2nd day. Hemoglobinuria is accompanied by frequent urination.

On the 3rd-4th day, the symptoms of the disease reach their culminating manifestation. At this time, the animals seem emaciated, they lie more, they do not take water, sometimes they grind their teeth. The mucous membranes are pale with a jaundice tinge or small-point hemorrhages. The scar stops contracting altogether or its movements are extremely slow. The heart beats are pounding, an arrhythmia appears. Urine is dark red in color, it is excreted often, in small portions. With these symptoms, the disease lasts 5-7 days and ends fatally. Death occurs at subnormal temperature, foam is released from the nostrils, convulsions of the extremities periodically appear.

With an atypical course of pyroplasmosis, an increase in temperature is noted, but the depression is expressed to an average degree. Appetite is reduced, the water animals are nervous, the movements of the scar are initially intensified, then its contraction slows down. The mucous membranes are pale, with a slightly yellowish tinge. Urine is yellow, and if it happens to be red, then for a short time.

Deterioration of the general condition may occur as a result of placing or grazing sick animals in the sun. And also when driving over a considerable distance.

Pathoanatomic changes. Mucous and serous membranes are pale, with a jaundice tinge and small-point hemorrhages, lymph nodes are enlarged, the spleen is dark red, significantly enlarged, hemorrhages are noticeable on the surface, the pulp consistency is flabby, the liver is enlarged, dark red, sometimes clay with a yellow tinge. The kidneys are enlarged, the border of the cortical and cerebral layers is smoothed. The bladder is usually filled with urine of a dark red color and rarely dark yellow. The book is hard, its contents are dry and crumbling. There is swelling in the lungs of the side on which the corpse was lying. There is a foamy pink liquid in the bronchi. On the surface of the heart, especially in the area of the atrium and its apex, spot and striated hemorrhages. There are numerous hemorrhages on the endocardium, blood clotted.

Diagnosis. The diagnosis is made comprehensively: on the basis of epizootological data, clinical signs and on the basis of the results of microscopic examination of blood smears.

Pyroplasmosis should be differentiated from teileriosis, babesiosis, francaiellosis, anthrax and leptospirosis.

Treatment. Sick animals are not driven out to pasture. They are provided with water, easily digestible. With juicy feeds, it is advisable to add a cup of milk or fresh

milk to the diet. In practical conditions, treatment is carried out taking into account the possibility of mixed invasion of pyroplasmosis and francaiellosis. For this purpose, specific chemotherapeutic drugs and symptomatic agents are used. Initially, the animal needs to undergo symptomatic treatment in order to prevent further changes in the heart muscle. Of the specific drugs I recommend azidine (berenyl), diamidine, DAT, norotrip, babenil, tripanil, hemosporidine, sulfantrol, polyamidine, etdin.

Prevention. Sick animals are necessarily subjected to specific treatment, otherwise the disease can end fatally. Therefore, when cases of piroplasmosis occur, the patient immediately undergoes a mitigating chemoprophylaxis with azidine or other specific preparations. After 10-14 days, if the animals remain on the same pastures, this treatment is repeated.

The main task in the elimination of pyroplasmosis in the enzootic zone is to carry out a set of preventive measures. Which include chemoprophylaxis and control of vector ticks.

Francaiellosis is an acute disease caused by Francaiella colchica (Babesia colchica). It is manifested by high fever, anemia, jaundice and hemoglobinuria.

The causative agent is Francaiella colchica (Babesia colchica) - their shape is rounded and pear-shaped, in the erythrocyte they are most often located in the center of one or two, rarely more. Paired francaiella are interconnected by sharp ends at an obtuse angle in the form of "glasses", which is characteristic of this pathogen, their magnitude reaches up to 2.8 microns. The incidence of erythrocytes is on average 4-5%. The accumulation of francaiella is also found in the corpse - in the capillaries of the brain, adrenal glands and other internal organs, therefore this parasite multiplies in the internal organs.

Biology of the pathogen. Fransaiella differ somewhat from pyroplasmas, in particular, their development in the tick takes place differently, since their invasion of animals will be confused already 3.5 days after the start of sucking of Boophilus calcaratus larvae. Therefore, the incubation period for francaiellosis is shorter. Non-sterile immunity against francaiella in cattle after active recovery is more resistant than against pyroplasmas, and lasts about a year.

Epizootological data. Francaiella colchica tolerates only Boophilus calcaratus ticks. Since the development of Francaiella colchica is similar to the development of Piroplasma bigeminum, the data on the epizootology of francaiellosis are similar to the epizootology of piroplasmosis.

The symptoms of the disease are similar to symptoms of pyroplasmosis, the incubation period is 13-14 days. Unlike pyroplasmosis in animals with francaiellosis, hemoglobinuria is less common, although francaiellosis is more severe.

Treatment and prevention are the same as with pyroplasmosis.

Babesiosis is an acute vector–borne disease caused by unicellular parasites Babesia bovis, manifested by fever, hemoglobinuria, anemia and jaundice of the mucous membranes, functional disorders of the cardiovascular and digestive systems.

The pathogen. Babesias are localized inside erythrocytes, merozoites have lanceolate and pear–shaped forms, and trophozoites are rounded. Diagnostic forms

are considered paired pear-shaped, connected by thin ends, located at an obtuse angle along the periphery of red blood cells. Their magnitude 1,0-2,4-0,5-1,5 microns. The erythrocyte lesion reaches 7-15%, rarely 40%.

Biology of the pathogen. In the body of cattle, babesias multiply in erythrocytes by simple division into two or budding, forming three individuals. Sometimes babesia can be seen outside of red blood cells. Their development in the body of ticks has not been studied enough. Babesia bovis is transmitted by the three-host mites Ixodes ricinus and Ixodes perculcatus. Ticks, feeding on a sick or sick animal, perceive babesia. Which first multiply in the intestine by simple division, then form cells with 2-4-6 nuclei, club-shaped (perhaps they are the result of the merger of two individuals). Such parasites penetrate into the intestinal epithelium and multiply, forming multinucleated cells. The latter disintegrate and divide several more times. After that, the club-shaped cells penetrate into the hemolymph and migrate to various organs and tissues of the tick, including egg follicles, where they still multiply. After leaving the eggs of the larvae ticks and their subsequent transformation into nymphs and imagos, babesias migrate in their body and enter the salivary glands. Nymphs and imago Ixodes, attacking the animal, introduce the pathogen with saliva into the blood. Since ticks of the genus Ixodes do not occur in our country, in this regard, the diseases babesiosis also do not occur.

TOPIC № 15. BOVINE TAILERIOSIS.

Lecture plan:

1. Teileriidoses, characteristics, distribution and economic damage.
2. Biological development of pathogens.
3. Immunity.
4. Taileriosis of cattle.
5. The causative agent
6. Epizootological data.
7. Symptoms of the disease, pathoanatomic changes.
8. Diagnosis, differential diagnosis, treatment, prevention and control measures.

Keywords: Piroplasmida, Theileriidae, Theileria and Nuttallia, Theileria annulata, Theileria sergenti, Theileria mutans Theileria orientalis. Theileria ovis, Theileria tarandi-rangiferis, Nuttallia equi, schizogonia, garnet bodies, Hyalomma, Hyalomma detritum-two-host pasture mites H.anatolicim- three-host mites.

1. Theileriidosis of animals is caused by protozoa from the order Piroplas-mida, the family Theileriidae. This family unites two genera: Theileria and Nuttallia. The Teileriids in the orgasonym of warm-blooded animals are.t is a complex development cycle. They multiply by multiple division or schizophrenia in RES-e, resulting in the formation of schizonts, or garnet bodies.

There are 4 types of teileria in cattle in our country, two of them are virulent – Theileriaannulata, Theileriasergenti, and two types of weakly virulent – Theileriamutans and Theileriaorientalis. Theileriaovis is the main exciter of the cycle,

and Theileriatarandi is rangiferis in reindeer. Nuttallia are represented by one species –Nuttalliaequi in horses.

Distribution. Teileriosis is a seasonal disease, common in the Northern Caucasus, Transcaucasia, the republics of Central Asia, including in our republic. in the south of Kazakhstan.

Economic damage. Teileriosis causes great economic damage to farms. It consists of losses from the death of animals, which can reach 40-80% of the number of cases of tailerism. Cows lose milk yield and it does not recover until the next calving. In sick bulls, spermatogenesis is disrupted, its recovery lasts for a year or longer, individual animals remain infertile. Animals brought back from the safe zone are especially seriously ill. As a result, breeding work to improve local livestock is hampered, as well as the costs of treatment and prevention of the disease.

Biology of pathogens. Teileriids develop in the body of warm-blooded animals and in carrier ticks.

Ticks, sucking on the body of animals, inoculate small mononuclear sporozoites. The latter penetrate into the lymphocytes, and with them – into the nearby (regional) lymph node. In lymphatic cells and outside of them, sporozoites multiply by schizogony, resulting in the formation of multinucleated schizonts (garnet bodies or Koch balls). At the beginning of schizogony, schizonts with large, irregularly shaped nuclei are formed – macrochizonts, which break up into separate individuals of macromerozoites and re-embed into healthy lymphoid cells. The process of schizogony repeats several times, the last time they divide and form microschizonts that differ from macrochizonts in the structure of the nucleus. Microschizonts have small, round, regular-shaped nuclei. In microschizonts, merozoites are formed by budding, which are embedded in red blood cells, in which they multiply by simple division.

Ticks, sticking to the body of a sick or sick animal, swallow red blood cells together with parasites. Merozoites, once in the body of a tick larva or nymph, develop in the intestines, hemolymph and salivary glands.

Currently, there is evidence of sexual multiplication of theileria in the tick body (deacons and Schein with the authors).

In the body of sick animals, taileria and nuttallia are preserved for up to 6 years.

Immunity. Animals that have been ill with teileriosis and nuttalliosis acquire non-sterile immunity or preimunition. Immunity to subsequent infection in animals is created by both cellular and humoral factors.

Cellular immunity is poorly understood, however, at the beginning of the disease, macro- and microchizonts can be found in phagocytes. Phagocytosis in teileriosis develops in the primary focus, but despite this, the pathogens of the disease violate the lymphatic barrier and settle into all organs and tissues of the animal.

Humoral immunity is caused by the production of antibodies. Moreover, antibodies in the body of sick animals are produced of two types: antibodies against tayleria from lymph nodes and antibodies against tayleria from erythrocytes. Antibodies are detected in the RSC. The first to appear on the 3rd-4th day are

antibodies of protic taileria from pomegranate bodies, and on the 7th-10th day after the attachment of ticks – antibodies against erythrocyte forms of the pathogen. Antibodies are specific to each type of pathogen. For example, serum from a sick animal infected with Theileriaannulata does not react with reactions with Theileriasergenti antigens, and vice versa.

The intensity and duration of immunity depends on the severity of the disease and the virulence of the pathogen. If animals are not reinvested, their immunity persists for 2-4 years, in case of reinvasion, the titer of antibodies increases in animals and the duration of immunity is prolonged.

4. Teileriosis is an acute or subacute vector-borne disease of cattle, zebu and buffaloes, caused by non-pigmented protozoa and manifested by an increase in lymph nodes, high fever, anemia, disruption of the cardiovascular and digestive systems, exhaustion and a high percentage of mortality.

The pathogen. The morphology of the pathogen depends on the stage of development. Sporozoite, which has entered the animal's body with tick saliva, multiplies in the lymph nodes and forms macro- and microschizonts, which have a diverse shape, their size ranges from 8 to 20 microns. The cytoplasm of schizonts turns blue, and the nucleus is dark ruby. The black macrochizonts have an irregular shape and relatively large sizes. The nuclei of microschizonts are small dot-like.

Microschizont breaks down into micromerozoites, which are embedded in red blood cells, they are called erythrocyte forms. In erythrocytes, taileria appear on the 2nd-3rd day, and sometimes later, after the temperature rises, they have rounded, oval, rod-shaped, cruciform and anaplasmoid forms and they are located at the edge of the cell. The size of rounded shapes is 0.5-1.5 microns. In one erythrocyte there may be 1-7, but more often 2-3 parasites, the infection of erythrocytes reaches 80-95%.

Epizootological data. Carriers of the invasion are ticks of the genus Hyalomma. The main importance is Hyalommadetritum(two-host mites) and Hyalommaanatolicum (three-host mites). Tick attacks occur on pastures and in stockyards. These types of ticks have adapted to life indoors, so there are cases of teileriosis in stable maintenance. Animals suffer from teileriosis in the warm season, the disease occurs from March-April to October. The season and dynamics of taileriosis depend on the climatic features of the year, the species composition of vectors and the condition of animals.

Symptoms of the disease. The duration of the incubation period is 12-21 days, sometimes more. The course of the disease is acute and subacute.

With an acute course, which is more often crowded in imported animals, an unpaired increase in the superficial lymph node is first noted, more often a pre-scapular, supra-named or popliteal (depends on the place of attachment of the tick). The lymph node increases 2-4-fold, becomes dense, painful when pressed. 1-3 days after the lymph node enlargement, the temperature rises to 410C and higher in animals, while appetite decreases and milk yield decreases. With the development of the disease, after 3-4 days, appetite almost completely disappears, chewing gum stops. With the cessation of food intake, the work of the intestines slows down,

peristalsis is poorly listened to, feces become thick, contain a lot of mucus, sometimes there is a blood oxide, urination is difficult, urine is released in a thin trickle, its color is normal or somewhat darker. Animals lose weight quickly, there is a decline in strength, the stomach sags, thirst appears (they drink often, little by little).

On the first days of the temperature rise, the mucous membranes of the eyes, nasal cavity are hyperemic, with individual spot hemorrhages. In the future, they become pale, slightly jaundiced, with multiple hemorrhages. As the painful process develops in separate animals, hemorrhages appear in the skin of the inner surface of the ear, udder, scrotum, root of the tail in the form of dots or specks.

During the period of high temperature, the breathing of animals increases to 40-80 movements, the pulse rate is up to 80120 beats per minute, a moderate pulse is often visible. There is a dry and intermittent cough, lacrimation (tears are often pinkish in color and they contain garnet bodies). The coat is ruffled, dull. The animals are standing. Legs wide apart. More often they lie, they rise with difficulty. A tremor appears in the muscles of the extremities. Pregnant cows can abort.

The number of erythrocytes in the blood decreases to 1,5 million in 1 mm^3, hemoglobin to 3-2 g%, leukocytes reaches 11 thousand in 1 mm^3, mainly due to lymphocytes.

In the subacute course, the lymph nodes also increase, the temperature rises to 41^0 and above, decreases after 2-3 days, then rises again and with slight fluctuations lasts until the end of the disease. The disease lasts 2-4 weeks or more. The visible mucous membranes are slightly hyperemic, then they become anemic and spot hemorrhages appear on them. Animals refuse to feed, at the beginning of the disease, peristalsis is increased, diarrhea may occur, but soon it is replaced by constipation, as intestinal atony develops.

Patients lie, moan, become completely indifferent to the environment, heart weakness develops and animals die. Local animals with a relatively mild course of the disease slowly recover.

Pathoanatomic changes. The corpse is exhausted. The skin in thin and pigmented places is sometimes weakly yellow, you can see spotty hemorrhages. The mucous membranes are pale, with a jaundiced tinge, hemorrhages are visible. The subcutaneous patch is jaundiced, sometimes infiltrated with hemorrhages. The superficial lymph nodes are enlarged, juicy with hemorrhages. The muscles are flabby, pale.

There is a small amount of clear liquid in the chest cavity. The pleura is jaundiced, covered with spot hemorrhages, Light emphysema, multiple hemorrhages on the mucous membranes of the bronchi. The heart is enlarged, flabby, there are massive hemorrhages on the epicardium and endocardium.

The peritoneum is slightly jaundiced, with hemorrhages, the liver is enlarged, flabby, yellow-sandy or red-brown, with hemorrhages under the capsule and in depth. The gallbladder is enlarged, filled with viscous bile. The spleen is enlarged, softened, under the hemorrhage capsule. The kidneys are enlarged, the border of the cerebral

and cortical layers is erased, massive hemorrhages. There is clear, light or dark yellow urine in the bladder, on the mucous hemorrhage.

The book contains dry, dense, fodder masses. Changes in abomasum are characteristic: ulcers with equal edges, 2-10 mm in size, are scattered on the surface of the mucous membrane, as well as many hemorrhages, nodules ranging in size from millet grain to a corn pea. In the small intestine, the mucosa is swollen, hyperemic, covered with mucus. In the duodenum there are small nodules (less often ulcers). In the large intestine, the mucosa is swollen, hyperemic, covered with mucus, hemorrhages. In bulls, granule-like eruptions and massive hemorrhages are found in the testes.

The diagnosis is made comprehensively taking into account epizootological data, clinical signs, pathoanatomic changes and laboratory studies. At the beginning of the disease, a punctate from the lymph nodes is examined to detect garnet bodies, and during the period of clinical signs, blood smears from peripheral vessels are examined to detect erythrocyte forms of taileria.

Teileriosis should be differentiated from pyroplasmosis, babesiosis, franciella and anaplasmosis, as well as from anthrax and emcar.

Treatment. Sick animals are provided with rest, they are not driven out to pasture, they are constantly provided with clean water, easily digestible, juicy feeds: green grass, tops of garden crops, melons, crushed root crops, bran or compound feed, it is desirable to add milk whey or fresh milk, fresh yogurt or curdled milk to the diet and carry out symptomatic treatment.

From specific chemotherapy drugs, butach, telex, telemax, buparvacon, buparvalek, polyamidine, etdin and others are prescribed to sick animals.

For the treatment of teileriosis, we have recommended (by the Department of **"Parasitology and ATS"** under the guidance **of Associate Professor Hakberdiev P.S.** 3-day treatment of teileriosis, which has been successfully tested in production over the past 15 years, the scheme of which is attached.

Prevention involves increasing the resistance of animals by improving feeding and maintenance, as well as the fight against ticks.

In the autumn-winter period (2-3 months before the appearance of vector ticks), susceptible young animals are vaccinated with a vaccine prepared from schizont taileria grown in the culture of lymphatic cells of organs and tissues of cattle.The vaccine is administered subcutaneously at a dose of 1 ml, regardless of the weight of the calf. In vaccinated animals, after 14-22 days, the temperature may hang by 0,5-2^{0}C and the regional lymph node may increase. Immunity in calves occurs on 30-35 days and persists for life if they are attacked by invasive ticks.

TOPIC № 16. EIMERIOSES (COCCIDIOSES). AIMERIOSIS OF RABBITS.

Lecture plan:

1. Coccidiosis of animals
2. Eimeriosis of rabbits

Keywords: Apicomplexa, Sporozoa, Coccidiida, Eimtriidae, Eimerinae, Eimeria, E.stiedae, E.magna, E.perforans, E. tenella, E.necatrix, E.maxima, E.acervulina, sporogonia, merogonia, gametogonia.

1. Coccidiosis of animals. Coccidioidoses unite a large group of animal and human diseases caused by protozoa belonging to the Apicomplexa type, the Sporozoa class, the Coccidiida order and the Eimtriidae family. The family Eimeriidae is divided into two subfamilies: Eimeriinae and Isosporinae.

Representatives of the Eimeriinae subfamily live only in the body of one species and complete the entire life cycle in it, are localized in the intestines of the host, a mature oocyst of the eimerioid type, that is, contains 4 spores and 2 sporozoites in each. One genus Eimeria belongs to this subfamily.

Representatives of the Isosporinae subfamily can live in the host organism of several species, and need 2 hosts to complete the life cycle: definitive and intermediate. Asexual reproduction – merogony and sexual process – gametogony most representatives of the isosporia perform in the intestines of definitive hosts, sporogony – the formation of 8 sporozoites – in the intestine or outside the host body. In intermediate hosts, isosporins multiply by endodiogeny or endopolygony with the formation of cysts. The mature oocyst is of the isosporoid type, that is, it contains 2 spores and 4 sporozoites in each of them.

The pathogens of the genera Cystoisospora, Toxoplasma, Sarcocystis, Besnoitia have veterinary significance.

Eimerioses of animals. Eimerioses are a large group of diseases of agricultural and wild mammals, birds, and fish. They mainly affect young animals and manifest themselves with diarrhea, emaciation, anemia.

Eimeria parasitize in intestinal epithelial cells, the exception is E.stiedae, which parasitizes in the bile ducts in rabbits and E.truncuta–in the mucous membrane of the renal pelvis of geese.

Eimerioses can cause devastating enzooties, causing great damage to farms. The prevalence of eimeriosis is very high, for example, in Kazakhstan among calves there are patients from 40 to 76%, among cows – 16-36%. Calves lose up to 27 kg of weight, lambs – up to 8.5 kg, chickens weigh 2-2.5 times lower than healthy ones. The infection rate of rabbits varies from 70 to 100%, the incidence of the number of cases reaches up to 40%. At poultry farms, chickens suffer from eimeriosis up to 70%, without preventive measures, the case can reach 100%. However, the damage from eimeriosis is not limited to the case, animals lose in weight gain, product quality. The meat of rabbits and birds that have been ill contains an increased amount of free amino acids, which reduces its nutritional value, they deteriorate faster during storage. A chicken that has been ill with eimeriosis begins egg laying with a delay of 1-2 months. Sheep lose weight, milk yield and wool shearing decrease, as well as the cost of purchasing preparations for treatment, prevention and disinfection of premises.

Biology of eimeria. In the life cycle of parasites, there are 3 phases of development: merogony – asexual division – ends with the formation of merozoites, gametogony – sexual process – ends with the formation of oocysts, both phases

develop in the host body; sporogony – reproduction in the oocyst – ends with the formation of sporozoites in the external environment.

Pathogenesis. Aimeria affects the host's body mechanically, toxic and open the gates to the development of secondary infections.The development of the painful process begins with the penetration of sporozoites into the intestinal epithelial cells of the host. As a result of merogony, mass destruction of epithelial cells occurs. From 6 to 680 million oocysts are allocated daily in one patient of the abdomen. Microflora penetrates through the damaged intestinal wall and causes an additional focus of inflammation and, subsequently, necrosis. Putrefactive microflora multiplies in the intestinal epithelial cells, the metabolic products of which increase the intoxication of the body. Inflammation of the intestinal wall makes it difficult to absorb fluid from the intestinal lumen, which leads to increased peristalsis and diarrhea, impaired absorption of nutrients, and leads to fasting of the body.

1. Eimeriosis of rabbits. Rabbits get sick with eimeriosis mainly in the period from weaning to 4-5 months of age, more adults are less likely to get sick. The disease is characterized by anemia, sometimes jaundice of the mucous membranes, rapid emaciation, an increase in abdominal volume, sometimes diarrhea, convulsions and mass death of animals.

Pathogens. Rabbits are parasitized by 8 species of eimeria, of which one species of E.stiedae is localized in the epithelium of the bile ducts, the rest – in the small intestine and only E.perforans - in the large intestine. Three types are more common:

E.stiedae – oval oocysts, yellowish-brown in color, at the narrow end there is a noticeable micropile, in the mature oocyst and sporocyst there are residual bodies, size 30-40x16-25 microns, sporulation 3-4 days.

E.perforans – oval or cylindrical oocysts, colorless, micropiles are noticeable in large forms, residual bodies in oocysts and sporocysts, size 20-25x12-15 microns, sporulation 1-2 days.

E.magna – oval-shaped oocysts, brownish with a pronounced micropile, around which there is a thickening, size 32-37x21-25 microns, residual bodies in the oocyst and sporocysts, sporulation 3-5 days.

Epizootological data. Eimeriosis is widespread, infection rate is 70-100%. Baby rabbits are infected from the first days of life, but while they receive their mother's milk, clinical signs almost do not appear. AI is affected by crowding, dampness in cages and rooms, poor quality of feed, colds, recruitment of groups of different ages, as well as the degree of infection with uterine eimeria. The more intensely the females are infected, the more rabbits die from eimeriosis.

Symptoms of the disease. The incubation period is 4-12 days. According to the localization of pathogens, intestinal, hepatic and mixed forms are distinguished. In natural conditions, as a rule, there is a mixed form. The course of the disease can be acute, subacute and chronic. The manifestation of signs of the disease depends on the type of pathogens, AI and resistance of the organism. Even before the appearance of oocysts in the feces, the mucous membranes of the eyes, nasal and oral cavities become anemic, later jaundice appears. Rabbits are reluctant to eat food or

completely refuse it, sluggish, lie more on the abdomen. The volume of the abdomen is increased, painful on palpation, feces retain consistency, but may also be liquid. Rabbits have frequent urination, some have rhinitis, conjunctivitis, increased salivation, sometimes convulsions, paralysis of the neck muscles, pelvic limbs are observed. Females are poorly fertilized, give small litters of 2-3 rabbits, that is, weak, since females are low-dairy. In sick rabbits, molting is delayed, the fattening period is prolonged. Mortality reaches 70%. Recovered rabbits secrete oocysts for a long time.

Pathoanatomic changes. The corpse is exhausted. The blood vessels of the intestine are injected. The small intestines contain a lot of mucus and are filled with gases. The mucous membrane is hyperemic, there are multiple hemorrhages on it, the total mass of the intestines is increased. Numerous hard whitish nodules containing eimeria are visible on the mucous membranes.

The liver is enlarged, the bile ducts are dilated, their walls are thickened. On the surface and inside the liver there are rounded or irregularly shaped nodules of dirty white or yellowish color, the size of a millet grain to a pea. There are a large number of oocysts in the nodules. Mesenteric lymph nodes are enlarged.

VSE carcasses, forcibly killed rabbits. Inexhaustible carcasses are used without restriction, depleted ones are checked for salmonellosis. If the result is negative, it is used for food.

The diagnosis is made comprehensively: based on epizootological data, clinical signs, autopsy results and microscopic examination of feces by the Darling or Fulleborn method.

Aimeriosis of rabbits should be differentiated from pseudotuberculosis, pasteurellosis, listeriosis and encephalitozoonosis.

Treatment. Sick animals are isolated, given food rich in carbohydrates (beets, oats, cereal hay).

Sulfadimethoxine is used for sick rabbits: 320 g is added to 100 kg of feed on the first day, and in the next 4 days -160 g., then they make breaks for 5 days and repeat the course.

Norsulfazole is prescribed at a dose of 480 g together with phthalazole at a dose of 160 g per 100 g of feed. Therapeutic food is used for 5 days, then take a break for 5 days and repeat the course.

Eimeria oocysts can be spread by rodents, birds, maintenance personnel on shoes, clothing, and care items. Rabbits get sick during the warm season. But if the rooms where the cages are insulated, then the disease appears in winter.

Immunity. Recovered rabbits acquire non-sterile immunity, which, with constant reinvasion by oocysts, persists for life. Immunity is specific, that is, antibodies are produced against those types of eimeria with which the rabbit was infected.

Furazolidone in a dose of 50 g per 100 kg of compound feed for 7 days.

Chemococcid in a dose of 0.03 g / kg is used with feed in 2-5-day courses with a break of 3 days. The effectiveness increases with the introduction of yogurt, ABC, and whey into the diet.

Prevention. Rabbits need to be kept in cages with a mesh floor (do not let it get stuck) outdoors or in non-insulated rooms. It is necessary to change the litter daily and scald the feeders and drinkers with boiling water. The food should be full-fledged with the addition of vitamins and trace elements.

During weaning of young animals from mothers, chemoprophylaxis is prescribed: sulfadimethoxine at a dose of 0.1 g and monomycin at a dose of 25,000 units / kg with feed in 2-5 day courses with an interval of 3 days, norsulfazol at a dose of 0.4 g with monomycin at a dose of 25,000 units / kg in 2-5 day courses with a break of 3 days, furazolidone at a dose of 0.02g/kg with feed according to the same system.

Aimeriosis of chickens. Eimeriosis is a widespread disease of chickens up to 2-3 months of age, manifested by anemia, emaciation and diarrhea, a high percentage of bird deaths.

Pathogens. In chickens, 9 species of eimeria are parasitized, the most virulent of them are: E. tenella, E.necatrix, E.maxima and E.acervulina.

E. tenella are oval–shaped oocysts, covered with a double-contour shell, colorless, there is no micropile, there is a granule on one of the poles, the size is 22.9—x19.1 microns, sporogonia 24-48 hours, localized in the caecum.

E.necatrix - oval or ovoid oocysts, colorless, a granule is visible on one of the poles, size 16.7 x 14.2 microns, sporogony 24-48 hours, localized in the middle part of the small intestine.

E.maxima – ovoid oocysts, less often oval, yellowish-brown in color, the shell is slightly rough, at the narrowed end of the micropile and granule, size 20x30mkm, sporogony 30-48 hours, localized in the anterior and middle parts of the intestine.

E.acervulina – ovoid oocysts, colorless, at the pointed end slightly noticeable micropile and idna or several granules, size 16.4x12.7 microns, sporogony 1-2 days, localized in the duodenum.

Epizootological data. All breeds of chickens are susceptible. The frequency of eimeriosis in poultry farms is not pronounced, since the temperature and humidity in poultry houses are maintained at the same level regardless of the time of year.

When walking, eimeriosis manifests itself from the end of spring to autumn. The sources of invasion are sick and sick birds. They are infected through infected food and water. Oocysts can be carried by wild birds, rodents, insects, maintenance personnel on clothes and shoes. Oocysts are very resistant to low temperature and disinfectants. Damp rooms, crowded maintenance, untimely cleaning of litter, feeding disorders contribute to the occurrence of the disease.

Immunity. Recovered birds acquire non-sterile immunity, which, with constant reinvention by oocysts, persists for a lifetime, and without reinvention it persists for only 45-60 days. Immunity is strictly specific to the type of eimeria that caused the disease.If, for the purpose of prevention, drugs were used that delay the development of merozoites at the first stage of merogony, then they do not develop immunity and they remain susceptible to eimeriosis even after the withdrawal of chemotherapy drugs at 2-3 months of age. The immunogenic stages of eimeria in the body of chickens are merontes of the II and III generations.

Symptoms of the disease. The incubation period is 4-7 days. The course of eimeriosis can be acute, subacute, chronic and asymptomatic (in adult birds). Sick chickens sit hunched up, lose their appetite, they get thirsty, they lose weight quickly. The comb and earrings are pale, the feathers are ruffled, around the cloaca are stained with liquid feces, which are initially whitish-green, then dark brown with streaks of mucus and blood. Chickens sit, as if leaning on their wings, stretch at times, coordination of movement is disrupted, trembling and paresis of the limbs are observed.

In the blood, the number of red blood cells and the value of hematocrit decreases, the number of white blood cells increases. The total protein index is decreasing. By the 6th-7th day after infection, the content of albumins and globulins decreases as much as possible. Protein deficiency in the body of chickens, apparently, is caused by starvation, as well as poor absorption of it, blood loss during intestinal bleeding and impaired liver systesis.

With an acute course, chickens die on the 5th-7th day and mortality reaches 100%.

In the subacute course, the clinical signs are the same, but they are weaker and the disease lasts 10 or more days, and mortality is lower - up to 50%.

Pathoanatomic changes. The corpse is emaciated, anemic, the musculature is flabby, In chickens affected by E. Tenella, the caecum is enlarged, thickened, the serous shell is bluish-red, the contents are colored red. When chickens are infected with eimeria parasitizing in the small intestine, the changes are similar, the lumen is filled with a curd-like bloody mass. The mucous membrane is hyperemic with hemorrhages, grayish-white nodules with a pinhead can be seen on its surface, they can also be seen from the serous membrane, they contain eimeria at different stages of development.

Changes in the small intestine are often diagnosed as enteritis of non-infectious etiology or poisoning. To clarify the diagnosis, it is necessary to examine the scraping from the mucous membrane and the removal of nodules, where oocysts, merozoites or gametes can be found.

The diagnosis is made on the basis of epizootological data, clinical signs, changes in the corpse and laboratory studies of faeces using Darling or Fulleborn methods for detecting oocysts.

Aimeriosis of chickens should be differentiated from histomonosis, trichomoniasis, pasteurellosis, pullorosis and colibacteriosis.

Treatment. Protoeimeriotic drugs that prevent the creation of immunity include: chemococcid, clopidol, koiden, stenorol, rigeococcin, larbec and some others.

The group of drugs that do not interfere with the creation of immunity includes: coccidiovit, ardinone, amprolium, coccidine, iramine, claramine, coccicol (at a dose of 1 kg / t of feed). In addition, sulfadimethoxine, sulfamomethoxine (dimethone), sulfadimesine and others are used in breeding and egg farms.

Prevention. Of great importance in prevention is the full feeding of not only chickens, but also breeding stock. It is impossible to let dampness in the premises,

untimely cleaning of the litter, overcrowding of chickens both with cellular and outdoor maintenance.

In dysfunctional farms, chemoprophylaxis is carried out with the same drugs as for treatment, but in smaller doses.

A method of immunochemoprophylaxis has been developed. Chickens at the age of 10 days are fed three types of oocysts and at the same time, for 20 days, a chemical preparation is given with food that does not interfere with the formation of immunity.

TOPIC № 17 VETERINARY ARACHNOLOGY. DISEASES OF PSOROPTOSIS, SARCOPTOSIS AND CHORIOPTOSIS.

Lecture plan:

1. Veterinary arachnology

2. Systematics, morphological structure and biology of arthropods.

3. Psoroptosis of sheep

Keywords: Veterinary arachnology, Arachnologiya, Arachne, Arachnoidea, Arthropoda, Insecta, Crustacea, Parasitiformes, Acariformes, Ixodoidea, Gamasoidea, Argasidae, Argasinae, Ornithodorinae, Sarcoptiformes, Trombidiformes, Oribatei, Sarcoptoidea, Analgesoidea, Tyrogliphoidea , Psoroptidae, Sarcoptidae, Psoroptes, Psoroptes ovis, scabies, wet, dry, acaricidal drugs.

1. Veterinary arachnology (Arachnologiya) – the science of arachnids (Greek. agashpe – spider, logos –teaching). The Arachnologiya class is included in the Arthropoda type – arthropods. Among the animal world, the Arthropoda type stands out with a huge number of species – there are about 1.5 million of them. Insects are especially numerous, which make up more than 90% of the number of arthropod-type species.

Arthropods are bilaterally symmetrical segmented animals, their body is covered with a cuticle that performs the function of a skeleton.

In arachnids, the head is fused with the thoracic region and forms a cephalothorax. In ticks, the body is not divided into departments – it is merged. The legs of arthropods consist of segments (hence the name – arthropods). The number of legs in adults varies, which is important in determining class membership. For example, insects have three pairs of legs, arachnids have four.

Arachnids have well-developed nervous, circulatory, digestive, excretory and reproductive systems.

Arthropods are bisexual, rarely hermaphrodite. They usually reproduce by fertilization, but some species also reproduce parthenogenetically. Most species lay eggs, and only some species give birth to larvae.

Growth in arthropods occurs abruptly, according to the phases of development, when molting from a larva to a nymph, from a nymph to an imago – male or female.This process in each phase of development is accompanied by a change of cuticle, histolysis of tissues and embryonic development of the next phase. Moreover,

in many arthropods, during molting, there is a greater or lesser change in the structure of the body and its increase. Such a transformation in the development of arthropods is called metamorphosis.

The type of arthropods includes five classes, of which veterinary parasitology considers three: Arachnoidea (arachnids), Insecta (insects) and Crustacea (crustaceans).

In arachnids, the body is divided into a cephalothorax and abdomen (spiders) or fused (ticks), they have four pairs of limbs. Veterinary arachnology studies ticks from the order Parasitiformes (parasitoform mites) and Acariformes (acariform mites). Parasitoform mites are important as carriers of pathogens of invasive, infectious diseases and as ectoparasites. Acariform mites are the causative agents of scabies diseases. Some mites from this order (oribatids) belong to saprophytes, but at the same time they are intermediate hosts of some species of tapeworms.

Acariform mites. The order Acariformes unites two-thirds of all known ticks, among which there are free-living and parasitic species. The mites are 0,1-0,3 mm long, their body is divided by a transverse groove into two sections: a proterosome carrying the oral apparatus and two front pairs of legs, and a hysterosome carrying the third and fourth pairs of legs, sexual and anal openings. There are no chitinous shields on the dorsal and ventral surfaces. Ticks breathe through the entire surface of the body, in addition, the respiratory organs – stigmas – imperceptibly open at the base of the legs or oral apparatus. The development cycle includes the stages of eggs, larvae, protonymphs, teleonymphs and imagos.

The order Acariformes is represented by the suborders Sarcoptiformes, Trombidiformes and Oribatei.

The suborder Sarcoptiformes includes the superfamily Sarcoptoidea – sarcoptoid (scabies) mites, the superfamily Analgesoidea – feather mites and the superfamily Tyrogliphoidea – tyroglyphoid mites. Representatives of the Tyrogliphoidea superfamily are small mites that live in grain, flour, hay and other feed and food products, and are often the culprits of feed poisoning.

Sarcoptoid mites. Ticks of the superfamily Sarcoptoidea are causative agents of sarcoptoid animal diseases, it includes two families: Psoroptidae and Sarcoptidae.

Psoroptosis of animals. Mites of the Psoroptidae family are larger than those of the Sarcoptidae family. Parasites live on the epidermal layers of the skin.

The Psoroptidae family includes three genera: Psoroptes, Chorioptes and Otodectes. Ticks of the genus Psoroptes are skinners, their body length is up to 0.8 mm. Visible to the naked eye. Their body is oval, the proboscis is long, adapted for piercing the epidermis and sucking lymph. Females have suckers on long segmented rods, on the 1st, 2nd and 4th pairs of legs. There are two bristles on the 3rd pair of legs. In males, the suckers are located in the same way as in females, but on the 4th pair of legs they are rudimentary. In addition, the males have well-defined opisthomal lobes and two sexual suckers.

The skinners have adapted to parasitism in sheep (Psoroptes ovis), cattle (Psoroptes bovis), horses, donkeys and mules (Psoroptes equi) and rabbits (Psoroptescuniculi). They do not reproduce on non-specific hosts.

Biology of pathogens. Skinworms belong to permanent parasites, since they reproduce only on animals, and they do not last long in the external environment. Development occurs in phases: egg, larva, protonymph, teleonymph, imago. The duration of metamorphosis in males and females of skin mites is different. Under optimal conditions, males develop within 14-16 days, females - 18-20 days.

Fertilization in ticks goes in two stages. Initially, the male copulates with the teleonymph, and after turning it into a female, the latter is fertilized by sexual products that were introduced by the male into the sexual opening of the second nymph.

Skinners under optimal conditions on the skin of sheep can give a very numerous reproduction. It is believed that one pair of ticks during the year gives birth to a multimillion offspring. The number of eggs laid and the development of offspring from them depend on the humidity of the subcutaneous air and the coat layer, as well as temperature. Females lay eggs on the surface of the skin, attaching them with uterine secretions. The lifespan of females is up to 60 days. The skinners are relentless to the effects of the external environment. Both low temperatures and dry air have a detrimental effect on them. Under all optimal conditions, ticks remain in the external environment for up to 65 days, and only two days in the pasture.

PSOROPTOSIS OR CUTANEOUS SCABIES OF SHEEP is an acute or chronically occurring disease characterized by itching of the skin, hair loss and exhaustion of the body, and sometimes the disease ends fatally.

Epizootological data. All breeds of sheep are susceptible to psoroptosis, but thin-armed and semi-thin-armed are especially sensitive. The disease affects sheep (both adults and young) in the winter period of the year. Of great importance in the spread of invasion are increased skin moisture, excessive contact of sick animals with healthy ones, as well as inadequate feeding, helminthiasis and other diseases.

Among adult animals, the largest number of patients appears during the cold season. The disease spreads rapidly and in a month the entire flock is affected by psoroptosis - an acute course. In summer, after shearing sheep, the psoroptotic process gradually subsides (latent flow), but in autumn it resumes again with renewed vigor.

In lambs, the invasion develops differently, they become infected at the age of 1.2-2 months when they are kept together with sheep. The disease proceeds chronically, and in autumn, when the lambs grow wool, the chronic course turns into acute.

The source of the invasion is sick sheep. Newly arriving sheep are brought into healthy flocks of skinners in the summer. The external environment can serve as a reservoir of excitability only under favorable conditions for ticks. In the cold, the skinners freeze and die. Other types of animals that come into contact with sick sheep can also be carriers of ticks.

Pathogenesis. The first lesions of mites occur more often on the sides of the trunk. Crawling on an animal, ticks with their bristles on their paws and suckers irritate skin receptors and cause itching. Itchy places of sheep are injured with teeth,

resulting in injury and moistening of the skin with saliva, all this contributes to the reproduction of ticks in the primary focus of psoroptosis.

Due to the ingress of toxic saliva of skinners into the wounds that are formed when punctured by the proboscis of ticks, the skin becomes inflamed and lymph accumulates on its surface, which eventually thickens, dries out and, together with the dead cells of the epidermis, forms dense crusts. It is in them that foreign microbes are introduced. Ticks and their waste products further enhance anti-inflammatory phenomena, which affects the general condition of animals. Violate the functional functions of the skin. Absorption of metabolic products and lysis of dead mites, decomposition products of skin tissues, microbial toxins affects the central nervous system, RES, metabolism, blood. As a result, eosinophilia and other hemodynamic shifts develop.

In the presence of factors that increase the resistance of the sheep's body, which is noted after shearing in the summer, the psoroptotic process is reversed or proceeds slowly.

Symptoms of the disease. The incubation period depends on a number of factors. In experimental infection of adult sheep, the first symptoms (itching, inflammatory phenomena) are detected after 10-14 days, and in lambs – after 1.5-2 months. The course of psoroptosis can be acute, chronic and latent.

With an acute course, itching first of all attracts attention. Sick animals try to get itchy places with their teeth, feet, or scratch them on hard objects. In the affected area, the wool is tangled and easily pulled out. Such places are called slaughterhouses by sheep breeders. In the future, this wool falls out. It is very characteristic that the bundle of fallen wool is retained and is clearly visible against the background of a dark fleece, like a lump of cotton wool. The center of the hearth is compacted. When new lesions appear, animals begin to itch intensely. The itching becomes especially intense at night, after driving and rain. After 6-8 weeks, almost the entire skin of the animal can be affected, except for the legs and head. At the same time, the wool falls off most of the body. With extensive skin damage, the condition of animals worsens. They lose weight quickly, the mucous membranes become anemic and with insufficient nutrition, the disease ends fatally.

The chronic course of scabies is observed in lambs in the summer. Constant contact with diseased uterus is the main reason for their infection. The disease in lambs develops less intensively than in adult sheep in the cold season. Short hair, low humidity, sun rays, intensive skin growth – all this is unfavorable for the development of skinners. The most characteristic signs: mild itching at the site of the lesion, crumpled wool. The affected areas are noticeable by the altered coat. When scratching such lambs make peculiar movements with their lips and tongue. The skin becomes inflamed, but without seals.

In September-October, the wool grows back, and the painful process becomes more active, that is, it proceeds acutely.

The latent flow is facilitated by unfavorable conditions for the reproduction of skinners: sheep shearing, solar irradiation. Ticks can persist in the folds of the skin and cause minor symptoms in the form of light scratching.

Diagnosis and differential diagnosis. Diagnosis in the acute course of the disease in sheep does not cause difficulties. It is put on the basis of characteristic clinical signs (scratching of the affected areas and the subsequent response of the animal, expressed in the movement of the head, lips and tongue). In doubtful cases and with an asymptomatic course, studies of scrapings on the border of affected and healthy skin areas are necessary.

When making a diagnosis, it is necessary to exclude diseases in which there is itching, hair loss and dermatitis.

It should be borne in mind that itching in sheep is also possible with the bites of ixodic lice mites. But at the same time there is no hair loss, and with careful examination, parasites can be detected. With monotonous feeding (silage) during winter keeping, sheep may also have itching in the lower abdomen by spring without hair loss. Scratching in sheep is also noted when the seeds of thyrsa (feather grass) get into the skin. However, animals react to this painfully, and at the same time with multiple skin lesions, they also observe damage to the digestive organs. Wool falls out with mastitis, with high fever and exhaustion, but there is no inflammation of the skin in place of the fallen wool.

Psoroptosis should be differentiated from chorioptosis and sarcoptosis. With chorioptosis, the limbs are affected, whereas with psoroptosis, the legs are not affected, sarcoptosis occurs in rough-haired sheep, initially the process develops in the head, tail and other places where the skin has a slight coat. The skin of priscoptosis is covered with crusts.

Treatment and prevention. The whole flock is considered unfavorable for psoroptosis when at least one sick animal is isolated, the unfavorable flock is transferred to such conditions as to exclude the possibility of transferring ticks to healthy livestock.Depending on the air temperature, sick sheep are treated with a wet and dry method.For the treatment of patients with psoroptosis of animals, recently recommended: ivomek, baymek, rustomectin, PIVSA, ivermectin is administered subcutaneously at a dose of 1 ml per 50 kg of live weight (at the rate of 2 mg of dry matter per kg of live weight, twice with an interval of one week.

In farms where sheep-heads do not suffer from psoroptosis, a complex of preventive measures is carried out against the introduction of skinners; sheep entering the farm are first quarantined, and then bathed in acaricidal emulsions or treated with the above preparations.

Pastures for sheep should not be adjacent to the pastures of a neighboring farm. Premises, bases where sick sheep were located, as well as equipment, uod items are subjected to deacarization.

Keeping dogs, cattle and horses from other farms in flocks is allowed only after counter-current treatment.

Chorioptosis of animals. Ticks of the genus Chorioptes are skin-eaters, they feed on exfoliated cells of the epidermis. They parasitize cattle (Chorioptesbovis), horses (Chorioptesequi), goats (Chorioptescaprae) and sheep (Chorioptesovis).

Ticks of the genus Chorioptes have an oval body, 0.3-0.5 mm in length, a proboscis in the form of a blunt cone, since the skin-eaters are equipped with

powerful chelicerae for chewing the scales of the epidermis. Suckers in females on the first. The second and fourth pairs of limbs sit on short non-segmented rods. Males have suckers on all limbs.

The biology of ticks of the genus Chorioptes is similar to that of ticks of the genus Psoroptes.

Chorioptosis of cattle. The skin of the extremities, the root of the tail and the udder mirror are affected. The disease is more common simultaneously with psoroptosis. Diagnosis and treatment are the same as for psoroptosis.

Chorioptosis of sheep is manifested by the fact that preventive anti-flow treatments are not carried out. At first, skinworms affect the limbs in the area of the put joints, and their parasitism is accompanied by peeling of the epidermis, itching, followed by the formation of crusts and thickening of the skin. The diagnosis is made according to these signs and the results of microscopic examination of scrapings. Treatment and prevention, as in sheep psoroptosis.

Chorioptosis of goats. A relatively rare disease. The first signs of the disease are noted in the extremities, in rare cases the process spreads to the rest of the body. In the affected areas, severe itching develops, abundant peeling of the epidermis and hair loss, then the skin thickens, shrinks and becomes covered with scabs.

Chorioptosis of horses is characterized by a lesion of the skin of the extremities in the area of the brushes, the inner thigh area and the disease is accompanied by itching, which manifests itself to a greater extent at night and after work. The most significant symptoms are hair loss in the area of the ibeder brushes and frequent stepping with your feet at rest.The diagnosis is the same as with psoroptosis, but the scraping is taken in the center of the lesion. Treatment and prevention, as with psoroptosis.

Sarcoptoidosis of animals. The Sarcoptoidea superfamily includes the Sarcoptidae family, includes two genera:Sarcoptes and Notoedres.

The mites of the genus Sarcoptes have a spherical body, 0.25-0.5 mm long, short and thick legs with bell-shaped suckers on long rods on the first and second pairs in females, on the first, second and fourth pairs in males.

Biology of the pathogen. Itching parasitizes in the epidermal layer of the skin. Females make moves under the epidermal layers, in each of which they lay 2-8 eggs. In total, she lays 40-60 eggs. For the full development of ticks under optimal conditions, it takes an average of 15-19 days. In the external environment, itches do not multiply and they retain mobility for no more than 15 days.

Ticks of the genus Sarcoptes are causative agents of sarcoptosis in horses, donkeys and mules –Sarcoptesequi, pigs –Sarcoptessuis and Sarcoptespalvula, goats - Sarcoptescaprae, reindeer – Sarcoptestarandi-rangiferis, rabbits –Sarcoptescuniculi, dogs –Sarcoptescanis. When switching from a specific host to a non–specific one, itches do not multiply, but cause a short-term disease - pseudo-fever.

Equine sarcoptosis (itchy scabies) is an acute or chronic disease with symptoms of itching, inflammation of the skin, hair loss and progressive exhaustion.

Epizootological data. Itches parasitize horses, donkeys, mules and may temporarily reside on cattle. and also on a person.

Pathogenesis. Itching causes a more severe disease than skin diseases. They make numerous moves in the subepidermal layer in the horizontal and vertical directions. Skin damage leads to disorders of many body systems, in particular, the central nervous system, RES, cardiovascular, etc. Extensive damage to the skin by itching, scratching leads to its thickening, baldness and folding. As a result, skin respiration is disrupted and oxygen deficiency increases, heat transfer increases. With reduced resistance of the body, irreversible pathological processes occur and the sick animal dies.

Symptoms of the disease. 12-20 days after infection, itching and inflammation of the skin are noted. In some cases, the process develops rapidly (acute course) – after 4-6 weeks, most of the skin is affected and the animals have a pronounced intoxication. In other cases, the disease proceeds slowly (chronically), accompanied by a gradual emaciation of the general condition. But if, under the influence of certain factors, the resistance of the body decreases, then the chronic course can turn into acute.

The first signs of sarcoptosis are noted more often on the head and less often in the area of the shoulders and back, where a yoke and saddle are applied. In places where skin moisture is increased. Nodules appear, the hair is glued and pulled out in bunches, the animals feel a strong itch. Lesions spread to neighboring areas of the skin, old foci are characterized by compaction, peeling, hair loss and skin folding. The parts of the body that the animal can scratch are heavily wiped, covered with cracks and pustules. Horses are worried, especially in warm stables, after work, which is associated with increased tick activity. The temperature in patients is recorded only with inflammation of the skin, complicated by a purulent process.

In the summer, sick animals are exhausted, their molting is delayed. Depletion of the body is accompanied by a decrease in the number of red blood cells, eosinophilia and leukocytosis.

The diagnosis is made on the basis of epiz. data and cl.signs, as well as when itching is detected in skin scrapings and it is made with a scalpel before the appearance of a sucrose in the center of the hearth from several places, the material is placed in kerosene or 10% caustic alkali (sodium or potassium), and then examined under a microscope.

Treatment. For the treatment of patients with sarcoptosis of horses, recently recommended: ivomek, baymek, rustomectin, PIVSA, ivermectin.

Pig sarcoptosis is a chronically occurring disease with symptoms of itching, inflammation and the formation of skin folds and dense crusts. Adult animals are asymptomatic and are parasitic carriers.

Epizootological data. The source of the invasion is adult pigs. Itching is transmitted from patients to susceptible animals through direct contact, through care items, as well as through the clothing of service personnel. Outside the host's body, itches survive up to 3 weeks, and at a temperature of - 5-9^{0}C -5 days.

Symptoms of the disease. Sarcoptosis of pigs is observed in two forms: ear and total. The ear shape (Sarcoptespalvula) is clinically manifested mainly in old sows and boars. This form is characterized by damage only to the inner surface of the

auricles and the skin around the ear. The total form (Sarcoptessuis) occurs in different age groups of pigs, mainly in young animals aged 3-6 months) at the same time, the skin of various parts of the body, including the auricles, is affected and proceeds according to the type of allergic reaction with three periods of disease development: an asymptomatic period – from the introduction of ticks to the appearance of the first signs of itching, the period of initial allergic reaction – the appearance of itching and the period of pronounced clinical signs- papules, vesicles, crusts on the skin, itching. Itching of both types can penetrate into human skin and mimic the symptoms of sarcoptosis, but they do not reproduce, do not lay eggs and die.

The diagnosis is relatively easy due to the presence of nodules, crusts and pronounced itching. In pigs, this is the only sarcoptoid disease. To clarify the diagnosis, a scrape is taken from the skin (along the periphery of the focus) from the inner surface of the ears and the affected areas and examined under a microscope to detect ticks.

The treatment and prevention of the disease are identical with equine sarcoptosis.

Sarcoptosis of camels manifests itself with the onset of the cold period, caused by Sarcoptescameli. Young people get sick more often. The first foci of scabies in adult camels are found on the neck, the inner surface of the thighs, and then on the scalp, corolla, in the interdigital fissure, and the process becomes generalized. In camels, the scalp is first affected, and then other parts of the body. At the beginning of the disease, severe itching appears in the foci, and then nodules and pustules develop. Itchy places are combed by animals, as a result of which cracks form on the skin, and it becomes crusted, becomes folded, the wool falls out and the animals lose weight quickly. In winter, sarcoptosis is acute, and in summer-chronically. If animals are not treated, they die.

The diagnosis, treatment and control measures are the same as in equine sarcoptosis.

Sarcoptosis of goats is caused by Sarcoptescaprae, it is often found among domestic goats, wild goats are also sick – teke (Capraesibirica). The initial and most frequent lesions in goats are found on the lips, nostrils and ears. Subsequently, the itching process spreads to the rest of the trunk. At the same time, bran-like scales are found in the affected areas, then scabs, wool falls out and the skin thickens, becomes folded. When animals are exhausted, they die.

Diagnosis, treatment and prevention are carried out by the same methods as with chorioptosis of goats.

Sarcoptosis of dogs is caused by Sarcoptes canis, manifested by skin lesions in the croup, groin, earlobe and is characterized by severe itching, anxiety and itching of these places. In the affected areas, the skin becomes covered with a bran-like coating, crusts appear, hair falls out. The skin becomes dry, there is a lot of dandruff, cracks appear, where the purulent microflora is introduced. Dogs have individual sensitivity, so, for example, not all puppies are infected in one litter. Without treatment, animals become exhausted and die.

Diagnosis, treatment and prevention are carried out by the same methods as with sarcoptosis of other animals.

Sarcoptosis of cattle and sheep is very rare. When itching is detected on animals, treatment and prevention are carried out by the same methods as with psoroptosis.

TOPIC № 18. VETERINARY ENTOMOLOGY. HYPODERMATOSIS OF CATTLE.

Lecture plan:

1. Veterinary entomology and its brief history
2. Structures, biology and taxonomy of insects
3. Hypodermatosis of cattle

Keywords: Custom, logos, heads, breasts, abdomen, cuticle, facets,segment, ovogenesis, eggs, larvae, pupae, imago, complete, incomplete metamorphosis, Arthropoda, Tracheata, Insecta, Diptera, Brachycera, Hypodermatidae, Oestriide, Gastrophilidae, Hypoderma, Hypoderma ovis, Hypoderma lineatum.

1. Veterinary entomology and its brief history. Entomology (from the Greek Entom – insect orlogos – teaching, science) is a science that studies the world of insects. Entomology is divided into a number of specialized disciplines - general entomology, which is a theoretical scientific discipline, agricultural and forestry, engaged in the study of beneficial insects and the development of methods for combating insect pests of plants, as well as medical and veterinary.

Veterinary entomology studies insects that cause harm to animal health and develops measures to combat them. Insects can harm the health of animals as parasites and as carriers of pathogens of infectious and invasive diseases. Some insects are intermediate hosts of animal helminths, others cause damage to livestock products. Diseases caused by insects are called entomoses.

The arachno-entomological direction in parasitology was developed under the leadership of Academician E.N. Pavlovsky, His numerous expeditions made it possible to find out the spread of arthropods as carriers of transmissive diseases and to create a new doctrine about diseases with natural foci.

Currently, entomological studies have been conducted and are being conducted in our republic by scientists such as Uzakov U.Ya., Ruzimuradov A.R., Enileeva N.H. and a number of others.

Morphology of insects. The body of insects consists of 3 sections: the head, chest and abdomen, it is covered with a cuticle and performs the functions of an external skeleton. The head of insects is movably articulated with the chest, consists of 6 segments forming a strongly compacted cranial box, oral organs and one pair of antennae. Antennae (antennae) are located between the eyes on the front surface of the head, consists of segments, are mobile, serve as organs of touch and smell. The structure of the antennae differs from insect to insect, which is used to recognize them. The mouth organs are attached to the head from the front and bottom. It

consists of an upper lip, 3 pairs of oral limbs (jaws) and a pharynx. Depending on the method of nutrition and food intake, the oral organs can be gnawing, sucking and wheelchair-sucking type.

On the sides of the head there are compound eyes consisting of many small simple eyes (facets), in connection with which they are also called facet, in addition, some insects have 1-3 simple eyes on the crown.

The chest is represented by 3 segments (anterior chest, middle chest and posterior chest), each of which carries a pair of legs from the lower side, and the segments of the middle chest and posterior chest also have a pair of wings.

The abdomen is built of 12 segments, but some of them are modified into components of the reproductive apparatus, so the visible number of abdominal segments in different insect species can range from 5 to 11.

The oviposition of females consists of components of the 8th-9th segments, the external sexual appendages of the male (hypopygium) – of the 9th-10th segments. The last 11th segment of the abdomen in many insects is reduced, but its appendages (cerci) remain attached to the 10th segment. The anal opening is located on the same segment, and males also have slates. The sexual opening is located behind the 8-9 sternites.

The outer integuments of the body form appendages, glands, hairs, which form a dense cover in some insects.

The internal structure of insects. The muscular system consists of striated fibers, is well developed, provides high mobility of insects.

There are internal organs in the insect's body cavity. The upper and lower diaphragms divide the cavity into 3 sections: the upper (there is a blood vessel in it), the lower (there is an abdominal nerve chain in it) and the middle (with the organs of digestion, excretion, reproduction and fat body located in it). The respiratory system is represented by spiracles opening on the sides of the chest and abdomen, strongly branching tracheae and tracheoles suitable for all internal organs, through which gas exchange occurs in tissues.

The circulatory system is open, represented by a single blood vessel (heart) located on the dorsal side of the abdomen. The heart is divided by valves into chambers. It passes in the thoracic department into the aorta, which opens into the body cavity at the head end. The blood is red, yellowish or greenish in color, contains colorless amoeboid cells (hemocytes), among which there are phagocytes. Along the spinal vessel, the blood moves from behind to the front, and in the body cavity – in the opposite direction. It enters the heart through valve-equipped openings (awns) available in each segment of the body.

The digestive apparatus of insects consists of 3 sections: the anterior and posterior are lined with chitinous cuticle, the middle one is lined with glandular epithelium.

The excretory organs - the malpighian vessels – open into the posterior intestine. The metabolic products in them come from the hemolymph, which washes the blind ends of the vessels.

The fatty body in the form of lobules is densely braided with tracheae. It fills all the gaps between the internal organs, supports them and serves as the main place of depositing nutrient reserves.

The nervous system is highly advanced, divided into central, peripheral and sympathetic. The central narva system consists of a head (supragloteal ganglion) located above the intestine and an abdominal nervous chain consisting of paired ganglia and located under the intestine in the thoracic and abdominal parts of the body. Several ganglia of the abdominal chain are located in the head and form a subgloteal ganglion connected to the supra-pharyngeal ganglion commissures. The peripheral nervous system is formed by nerves running from the ganglia of the central and sympathetic nervous system to organs and tissues. The sympathetic nervous system consists of 3 divisions: the oropharyngeal, the abdominal and the caudal. It regulates the work of the muscles and internal organs of insects.

The basis of the sense organs in insects are sensillae, consisting of a perceiving structure and adjacent sensitive cells. Insects distinguish between mechanical sense, hearing, chemical sense, hypothermic sense and vision. Sensitive sensors can be located in the skin or protrude above it in the form of bumps, bristles, hairs and other formations.

Insects are segregated. The male's reproductive system consists of a pair of testes, a pair of vas deferens, an ejaculatory canal, accessory sex glands and a male sexual appendage. The female's reproductive system includes a pair of ovaries, a pair of oviducts, an unpaired oviduct, accessory sex glands, an ovipositor and an anterior ovipositor.

Reproduction and development of insects. In some insects, the ability to reproduce appears immediately after birth, in others - after some time, which occurs due to the unequal degree of sexual maturity. Egg maturation (ovogenesis) is regulated by brain harmons and juvenile. After fertilization, the larva develops in the egg. This period is called embryonic. All further development, including the adult insect, is called postembryonic.

Insect growth occurs only in the larval phase. In postembrionic development, there are 2 types of metamorphosis: complete, when the insect goes through the phases of larva, pupa and imago, and incomplete - development occurs without the pupa phase. In insects with complete transformation, the larva differs sharply from the imago in appearance, nutrition and habitat (gadflies, flies, mosquitoes, horseflies and others). The pupa does not feed, but exists due to the nutrients accumulated by the larva. In it, histolysis first occurs, and then histogenesis – the formation of new organs. After histogenesis is completed, the imago emerges from the pupa. In insects with incomplete transformation (bedbugs, lice) the larva emerging from the egg is similar to an adult insect and differs from it only in the sexual appendages and wings. They are formed later in the molting process.

Gadflies and diseases caused by them. According to the systematic position of gadflies, they are included in the Diptera order (Diptera), the short-whiskered suborder (Brachycera).They are combined into 3 families:

1. Hypodermatidae – subcutaneous gadflies, including 9 genera, of which Hypoderma, Oedemagen and Grivellia have veterinary significance:

2. Oestridae– nasopharyngeal gadflies, consisting of 5 genera, of which representatives of the genera Oestrus, Cephenomyia, Rhnoesrtus and Ctphalopina have veterinary significance

3. Gastrophilidae – gastric gadflies with one genus Gastrophilus.

3. HYPODERMATOSIS OF CATTLE.

Hypodermatosis is a chronic disease of cattle caused by larvae of subcutaneous gadflies, characterized by inflammatory phenomena in habitats, general intoxication of the body and a decrease in animal productivity.

Two species parasitize cattle: Hypodermabovis – common subcutaneous gadfly (string), the other - Hypodermalineatum - southern subcutaneous gadfly (esophagus).The main host for both types of gadflies is cattle. String larvae can develop in zebu, horse, sheep and goat, and esophageal larvae – in the body of yak, zebu, horse, goat and sheep.

Morphology of gadflies. Imago are large, up to 2 cm long insects resembling bumblebees in appearance. The body consists of a head, chest and abdomen, covered with thick hairs of yellow, orange and black colors. The esophagus differs from the string by its slightly smaller size and color of the chest and abdomen hair. The eggs are small, with a diameter of 0.85-0.86 mm, and together with the attached appendage -1.09 mm.

The face of the I-stage at the exit from the egg has a length of 0.6 mm, before the first molt - 17 mm, the color is white or pale yellow. At this stage, the larvae of the string and the esophagus differ in the structure of the oral hooks: in the string they are separated at an acute angle in the form of a fork, and in the esophagus the front end is pointed and has a tooth-shaped protrusion.

Stage II larvae are larger (18-20 mm) than stage 1 larvae. In place of the mouth hooks, they have only small pigmented areas.

Larvae of the III stage of the string, up to 28 mm long, esophagus -16-26 mm. In the row, the posterior spiracles have an irregular oval contour from the outside, the scar is located at the bottom of the funnel and is usually completely closed. The height of the spiracles is greater than that of the esophagus, reaching 1,2 mm. On the posterior edge of the penultimate abdominal segment from the dorsal side of the larva there is no zone with thorns. The esophagus has this zone, although small, but there is. In addition, the larvae of stage III of the esophagus differ in that they have flat, rounded, up to 1 mm high posterior spiracles, around which there is a zone of 10-15 rows of small thorns. The row has almost twice as many rows.

Pupae are darker in color than larvae, but retain all their signs. The dorsal side of the pupa is almost straight, with a pronounced cap at the anterior end.

Biology of gadflies. Gadfly refers to insects with a complete transformation. The full development cycle ends within a year.

The exit of the imago gadflies from the pupae occurs very quickly – within 2-3 seconds and after 30-80 seconds the fly is able to fly and mate. Adult gadflies do not feed and live off the nutrients accumulated in the larval phase, so their life is short 3-

10 days, and when the temperature drops – up to 28 days. An adult gadfly loses up to 36% of its mass by the end of life.

On sunny days, the gadfly years are observed at a temperature of +6-80C, and on cloudy days – at +13-140C. Males gather annually in the same places where females arrive for mating. After mating, the cut-off females go in search of animals for laying eggs.

When attacking animals, female gadflies behave differently, the string flies around the herd, chasing animals and making specific sounds. Animals are very worried, they try to escape from persecution, and the female esophagus gets close to the animals by short flights, crawling, falling behind unnoticed, she calmly lays eggs on the hairline. The string lays one hair at a time, the esophagus lays 5-20 eggs at a time.

Female gadflies are very prolific, laying up to 800 eggs. For laying eggs, females prefer areas with a short spine and abundant undercoat in the area of the hungry pit, the soft wall of the abdomen, groin, and the front of the thighs. The formation of the larva inside the egg lasts 3-7 days in the row, 3-6 days in the esophagus. The larvae hatched from the eggs penetrate through the skin into the host's body. Then the larvae of the string migrate along the large vessels and nerves to the spine and through the intervertebral openings enter the adipose tissue of the spinal canal, and the larvae of the first stage of the esophagus migrate towards the esophagus and are localized in its submucosal layer.

The total duration of stay of larvae in the esophagus and spinal canals is 5-6 months. After that, the larvae migrate to the back and lumbar region, where they form connective tissue capsules. For further development, they need oxygen from the atmospheric air, for which the larvae form fistula openings in the skin. After 1-8 days, the larvae molt and enter stage II. Mature stage III larvae exit the capsule through fistula openings in the skin (mainly in the morning and afternoon) and fall to the ground, where their pupation takes place. The fallen larvae are inactive, pupation occurs within 1-2 days, less often within 7 days. The duration of pupal development is 20-40 days.

Epizootological data. The density of livestock settlement is one of the main factors affecting the number of gadflies and the infestation of animals with larvae. The extensiveness and intensity of gadfly infestation of young animals is higher than that of adult animals. The movement of cattle infected with subcutaneous gadfly larvae inevitably leads to the spread of hypodermatosis. A much greater danger is the loss to pupation of even a small number (10-15) of stage III larvae from animals accidentally untreated in autumn, leading already during the first season and over-infection of 40-50% of the stage livestock.

Pathogenesis. The larvae of horseshoe gadflies have a mechanical and general toxic effect on the body of cattle and open the gates to the development of secondary infections.

Symptoms of the disease. The first clinical signs of hypodermatosis appear when gadfly larvae that have emerged from eggs are introduced into the skin. Animals develop itching, swelling of the subcutaneous fangs, soreness of the areas

affected by the faces. These symptoms can be observed during the whole period of the gadfly summer.

When the larvae approach the skin of the back, the symptoms are clearly manifested in all affected animals. At first, small seals form under the skin of the back, barely felt during palpation, after a while they turn into faintly noticeable bumps with a hole in the center. When pressing on them, the animal has a painful reaction. As the larva grows, the discharge of serous fluid from the fistula opening increases, which glues the hair. The skin covering the fistula capsule is inelastic, with fever and painful.

The hair on the back of a sick animal is in patches or completely tousled. At the locations of fistula capsules, bundles of hair glued together with dried exudate are directed in different directions. With abundant discharge of purulent exudate from fistulas, the hairline is contaminated with greenish-yellow secretions.

Pathoanatomic changes. When opening animals during the period of infection with larvae, small bubbles can be found in the subcutaneous patch, in which larvae from 1 to 5 mm in length are viewed. The latter, upon careful examination, are also found on the superficial fascia of the trunk, in the connective tissue layers of the muscles, dirty green stripes of secretions are noticeable on the migration routes of the larvae. Areas of the esophagus affected by larvae are hemorrhagic, edematous both from the mucous membrane and serous. There are hemorrhages in places where larvae accumulate in the spinal canal.

During the period of parasitization of larvae of stages II and III, the greatest changes are noted in the skin, subcutaneous tissue and muscles. Fistula capsules are clearly visible here, surrounded by a gelatinous mass of light or pink color, permeated with numerous blood-filled vessels. With a strong lesion of larvae, serous or serous-hemorrhagic inflammation of the muscles is noted, covering extensive areas of the back.

Diagnosis. The diagnosis is made on the basis of examination and palpation of the animal's skin in the places of accumulation of larvae of the II and III stages of subcutaneous gadflies – along the entire length of the back from the withers to the sacrum.

Treatment and prevention. The main method of combating hypodermatosis is the destruction of stage I larvae of subcutaneous gadflies in the body of animals. To do this, systemically acting insecticides are used: hypodermin-chlorophos, chlorophos, neguvon, neguvon N, ivomek, ivermectin, baymek, rustomectin, PIVSA and others. Animals are treated in autumn after the end of the gadfly summer: in August-September-October. Moreover, all cattle grazed on pasture are subject to processing, in order to achieve 100% effectiveness of treatment, animals are treated twice in autumn with an interval of 30 days. The drugs are applied to the back from the dispenser in a thin trickle on both sides of the vertebral column from the withers to the sacrum: hypodermin-chlorophos to animals weighing up to 200 kg in a dose of 16 ml, more than 200 kg -24 ml, ivomek and its analogues are administered once, subcutaneously in the neck, at a dose of 200 mcg / kg of the body.

In the spring, in order to identify sick animals, a single livestock survey is carried out. Animals affected by stage II and III larvae are treated with the above drugs in the same doses as with early chemotherapy.

Since the spread of pathogens of hypodermatosis occurs mainly by sick animals, cattle not treated with insecticides, infected with larvae, should not be allowed to pastures.

TOPIC No. 19. ESTROSIS OF SHEEP, RHINESTROSIS AND GASTROPHYLOSIS OF HORSES.

Lecture plan:

1. Estrosis of sheep
2. Rhinestrosis of horses
3. Gastrophylosis of ungulates

Keywords: Nasopharyngeal, gastrointestinal gadfly, Oestrus, Oestrusovis, Rhinoestrus, Rhinoestruspurpureus, Rh.latifrons, Rh.usbekistanicus, Gastrophilus, G.intestinalis, G.veterinus, G.haemorrhoidalis, G.pecorum, G.inermis, G.nigricornis, G.magnicornis, G.flavipes, chlorophos, chlorophos with snow, Bravermectin, disinsection of animals.

1. Estrosis of sheep – caused by larvae of the sheep gadfly Oestrusovis, parasitizing in the nasal cavity, frontal and paranasal sinuses of the head, accompanied by inflammation of the mucous membranes in the places of their parasitization. The sheep gadfly belongs to the family of nasopharyngeal viviparous gadflies (Oestriide), the Diptera order (Diptera) and the insect class (Insecta). The only owner of this species is a sheep, although it is quite common in domestic goats.

Morphology of the pathogen. The winged gadfly is yellow-brown or yellow-gray color, 10-12 mm long. Females are slightly larger than males. The body is covered with sparse short hairs sitting on small dark-colored bumps. The head is large, hemispherical in shape. Dark green shiny faceted eyes are separated by a forehead, on which 3 tubercles are arranged in a triangle. There is no oral opening, in place of its light plate, through which the rudiments of the jaws and proboscis shine through. The egg is white, oblong, up to 1 mm long, arched curved, with rounded ends. Female gadflies are viviparous. After 14-18 days after mating, grayish-white, small (1.27-1.35 mm), spindle-shaped, very mobile larvae consisting of 12 segments appear in the uterine expansion. Before molting, stage I larvae reach 4-5 mm in length and 0.34 mm in width. The larva of stage II is white, 5-12 mm long, up to 3 mm wide. The first thoracic and eighth abdominal segments are armed with small conical spines. The larva of stage III is 10-30 mm long . The rear horse of the larva is wider than the front one. Young larvae are white, with brown mouth hooks, adult larvae have dark hair on the dorsal side. The pupa has a length of 12 mm, a width of 5 mm, the lower end is blunt, the upper end, on which the notch is located, is beveled at acute angles, the color is initially dark gray, clouded brown.

The biology of gadfly. Sheep gadfly imagos do not feed, but live off the reserves of nutrients accumulated in the larva phase. At the pupal phase, this reserve is economically distributed for the formation of an adult insect and the creation of fat deposits in the body consumed during mating, maturation of larvae and flights.

The imago's exit from the pupa most often occurs in the morning in warm sunny weather. Mating lasts 2-3 minutes. After fertilization, the formation of larvae begins in the uterine receiver. During this period, the females fly, then for 10-20 days they sit in the recesses, crevices of buildings. Females with mature larvae dramatically change their behavior. They become active, mobile, begin to fly. Injection of larvae into the nasal cavity of an animal can occur both on the fly and from the ground from a distance of up to 40 cm. At one time, the female throws out 8-12, sometimes 20-39 larvae.

The female's attack on sheep lasts 2-4 days, with a decrease in temperature – 5-6 days, after which she dies. During her lifetime, the female gives birth to about 600 larvae.

In the south, two generations develop during the year, and the autumn-spring generation develops in the usual time, and the summer-autumn generation develops in a shorter time. The life span of the imago is 12-13, maximum 46 days. The sheep gadfly lives near the breeding grounds, if there are sheep there. In search of animals for laying larvae, female gadflies travel distances of up to 30 km .

The main mass of stage I larvae is localized on the inner and outer surfaces of the lower nasal shells, nasal septum and the wall of the nasal cavity. Larvae of stages II and III develop in areas more isolated from the external environment – in the frontal sinuses and cavities at the base of the horns. The duration of larval development in sheep in zones with one generation per year is 8-11 months, and in zones with two generations of larvae of the autumn generation – 8-10 months, spring – from 20-30 days to 3-6 months.

Mature, well-pigmented larvae of stage III migrate from the frontal sinuses to the nasal cavity and during sneezing sheep are thrown to the ground. The departure of stage III larvae for pupation occurs more often in the morning. Pupation of larvae occurs in the soil at a depth of 1-5 cm . In areas with dense soil, larvae can pupate under a leaf, a chip, a stone. The pupae of the sheep gadfly tolerate low temperatures well. The duration of the pupal phase ranges from 14-17 to 46 days.

Epizootological data. The disease has been registered in many countries of the world, but mostly in steppe and semi-steppe areas with well-developed sheep breeding. The disease is characterized by high extensiveness, reaching 70, 90 and even 100% in most zones with a high intensity of invasion by larvae, which number several dozen copies in each sheep. Old, sick with other diseases, weak animals are more intensively affected by larvae than healthy ones.

The highest intensity of infection of sheep with larvae is noted by the end of summer gadfly. During the winter period, some of the larvae die and the intensity of invasion decreases, in the period from March-April to May-June it is minimal due to the loss of larvae for pupation.

Pathogenesis. Gadfly larvae have local mechanical and general toxic effects. Once in the nasal cavity of a sheep, gadfly larvae wound the mucous membrane with their thorns, which causes its inflammation. The mucous membrane ulcerates, swells, and an abundant exudate begins. Inflammation can be complicated by microflora, turn into purulent-necrotic and spread to the membranes of the brain. The inflammatory process in the nasal cavity makes breathing difficult, and when larvae enter the trachea, aspiratory dyspnea develops.

Symptoms of the disease. During the disease, there are 3 periods. The first period begins after the invasion of sheep by gadfly larvae. The movement of larvae trapped in the nasal cavity and their attachment to the mucous membrane is very painful and causes protective reactions. Sheep sneeze, snort, shake their heads, rub their noses against legs and hard objects, trying to get rid of extraneous stimuli. On the 2nd-3rd day, serous-mucous discharge is released from the nostrils, sometimes with streaks of blood. Sick sheep have difficulty breathing, often digestive disorders. With the onset of the idle years, the gadfly stops and the symptoms of the disease disappear. There comes a second, hidden period of the disease. The third period develops closer to spring, when the intensive growth of larvae resumes and the disease worsens again. There is an abundant serous or serous-purulent discharge from the nasal shells, animals cough convulsively, trying to get rid of mucus, difficulty breathing, animals often snort. Along with the serous-purulent contents, the larvae of the III stage also fall out for pupation. During this period, the clinical signs of estrosis are most pronounced. Lambs are especially difficult to tolerate the disease, because of severe rhinitis, they breathe through their mouths, which prevents them from sucking queens. They grow slowly, switch to foot food early, and therefore the number of gastrointestinal diseases and waste among young animals increases.

Often, inflammatory processes from the mucous membrane of the nasal cavity spread to the pharynx, the mucous membranes of the latticed bone, frontal sinuses and the lining of the brain. Purulent rhinitis appears, often accompanied by the phenomena of meningial syndrome, called "false vertyachka". The general condition of the animals deteriorates sharply, they become sluggish, refuse to feed, lose weight quickly and die.

Pathoanatomic changes depend on the AI, duration and severity of the disease. In the first days of the disease, ulceration, hyperemia, catarrhal inflammation and swelling of the mucous membranes of the nasal cavity, often the trachea and bronchi, are noted. In winter, the changes are less pronounced and remain only in the locations of the larvae in the form of limited inflammatory areas.

During the transition of larvae to the II and III stages, the mucous membrane of the sieve bone is inflamed, the labyrinths are filled with mucous or purulent contents with an ichorous odor. Similar changes are not uncommon in the frontal sinuses and near the horn spaces. In sheep with signs of false vertebra, the vessels of the cerebral membranes are injected, the anterior parts of the large hemispheres are swollen and pigmented, there is infiltration in the ventricles and ammonium horns.

The diagnosis is established based on clinical signs and autopsy results of killed and fallen animals. Epizootic data are necessarily taken into account (seasonality of the disease and zonal features of the ecology of the sheep gadfly).

Estrosis of sheep should be differentiated from cenurosis, listeriosis and rabies.

Treatment and prevention. To combat estrosis, general preventive measures are carried out, in the fall – early chemotherapy and in the spring – treatment of clinical sick animals.

The spread of the causative agent of estrosis is mainly by sick animals. Therefore, it is unacceptable to export sheep to prosperous farms without preliminary therapeutic and preventive treatment of them, it is also not allowed to release sick sheep to pastures without treating them with insecticides. The autopsy of fallen animals should be carried out on a specially equipped site, and the fallen larvae are destroyed. In the spring, once every 2 weeks, koshary and tyr are thoroughly cleaned of manure and manure is stored for biothermal disinfection. In dysfunctional farms, sheep and goats grazed on pastures are subjected to early chemotherapy with an aerosol of DDVF and chlorophos or a solution of chlorophos is evaporated. A 0,1% solution of chlorophos is used by the method of free soldering, once. In animals with an obvious clinic of estrosis affected by stage II and III larvae, the nasal cavity is irrigated in spring with a 4% aqueous solution of chlorophos.

Recently, the following drugs have been recommended for the treatment and prevention of estrosis:ivomek, ivermectin, baymek, rustomectin, PIVSA and others. These drugs are administered subcutaneously at a dose of 0,2 mg per kg of live weight according to ADV, once.

RHINESTROSIS OF HORSES. Rhinestrosis is a chronically occurring, widespread disease caused by larvae of nasopharyngeal gadflies parasitizing in the nasal and adjacent cavities of the head and the disease is characterized by local inflammatory processes and general pathological phenomena.

The causative agents of rhinestrosis are 3 species of gadflies belonging to the family Oestridae genus Rhioestrus. These are Rh.purpureus-white-headed or Russian gadfly, Rh.latifrons - short gadfly and Rh.usbekistanicus - small-chip gadfly.

Morphology of pathogens. The Russian gadfly (Rh.purpureus) is very similar to the sheep gadfly. It is purple-brown in color, 10-12 mm long. The head is large, naked, swollen, especially in the transverse direction, white. The female's forehead is very wide, the male's is narrower. The oral organs are underdeveloped. On the brown with a reddish tinge of the back there are 4 shiny black longitudinal stripes, interrupted at the transverse dorsal suture. The abdomen is egg-shaped, almost naked, dirty violet-gray with a silvery sheen, dotted with black dots along the middle part. The legs are light to dark brown in color. The wings are 8-12 mm long, transparent, with 3 black dots at the base, distinguishing gadfly from other species.

The I–stage larva is about 1 mm long, oval in shape, wide in front and tapering in back. Its body is almost flat, slightly convex from the dorsal side. The head end is equipped with two powerful sharp curved hooks of light brown color. At the base of their mouth opening. The body is covered with spikes, on the sides of the body, on segments, there are 4-5 rather long hairs, directed in an arc, with the tops backwards.

On the rounded last segment there are spiracles and several curved spikes. The length of the larva before molting is 3,5 mm (stage II larva). The larva of stage III reaches a length of 17,5 mm, the front end of its body is narrowed and equipped with two screw hooks. The convex upper and flat lower sides are armed with spikes. The color of the larva is red at first, but becomes white as it matures. The pupa is 12,5 mm long and 6 mm wide, on the 10th segment, elongated-egg-shaped, slightly thicker at the back.

The short gadfly is 11-13 mm long, the longitudinal stripes on the midrib are red-brown, the body of the larva of stage III is wide, which differs from other species.

The gadfly is small - up to 9 mm long, the sides and bottom of the chest are covered with reddish hairs.

The biology of the causative agents of oestrinestrosis is typical for gadflies of the family Oestridae. In the phase of adult insects, they are found throughout the summer: in our Republic, the flight of nasopharyngeal gadflies of horses goes from early May to mid-June and in autumn during September-October. The life expectancy of Russian gadfly females is 30 days, shorty - 46-52 and small-40 days, males – much shorter. In the conditions of Central Asia, all kinds of gadfly gives 2 generations per year.

The diurnal behavior of males is characterized by increased activity in the first half of the day. Females are active from hatching to mating, after which they sit in secluded places in the crevices of mud-brick buildings and duvals. This lasts for about 14 days. After the larvae mature, the intensive flight of females and infection with animal larvae begin again. In the hot hours of the day, gadflies rest on the branches of plants, stones, hills, roofs of buildings. The fertility of females is very high: the Russian gadfly has 700-792, the shorty has 640-1074 and the small-chip has 425-560 larvae.

To lay the larvae, female gadflies rapidly fly up to the horse's nostrils and, after injecting a portion of 8 to 40 or more larvae, fly away, However, this process does not always happen successfully for the larvae, since the horse, sensing the presence of its enemy, immediately begins to snort and shake its head up and down until the danger passes. Larvae trapped in the nasal cavity move deeper and are firmly attached to the mucous membrane. The habitats of stage 1 larvae are the inner surface of the shells and the labyrinth of the lattice bone. Here, in the labyrinth of the lattice bone, the larvae molt. Stage 3 larvae fall out of the nasal cavity, penetrate into the surface layer of the soil and pupate. Pupation takes place within a period of 24 to 48 hours . The duration of the pupal phase is 15-30 days and depends on the ambient temperature.

Epizootological data. The source of the invasion is infected horses, donkeys, mules. The area of the Russian gadfly is wider than shorty and maloship. EI and AI are higher in areas with a larger number of hosts. Larvae are more strongly infested and young and old animals are more seriously ill. The number of sick horses in dysfunctional farms reaches 75%. The intensity of infection with larvae is: average - 66, maximum – 416 copies.

Pathogenesis and symptoms of the disease. Gadfly larvae injure the tissues of the nasal cavity of animals with near-mouth hooks and spikes, which leads to catarrhal inflammation of the nasal mucosa and underlying tissues. Rhinitis and laryngitis develop. Infiltration and edema often occur in the places of attachment of larvae, the act of swallowing is violated. When watering animals, water flows out of the nostrils. The development of purulent-necrotic processes is possible. Horses often cough, snort, soreness is noted when feeling the throat. The general condition of the animals is depressed, the submandibular and parotid lymph nodes are enlarged.

Pathoanatomic changes. Gadfly larvae are found on the mucous membrane of the nasal cavity and frontal sinuses. The mucous membrane is inflamed, dark purple in color, ulcerated.

The diagnosis is made on the basis of clinical signs, the results of a pathoanatomic examination and the detection of gadfly larvae in the nasal cavity and pharynx. It is necessary to differentiate rhinestrosis from diseases of the upper respiratory tract, glanders, myta and gastrophylosis.

Treatment. The simplest method is washing the nasal cavities with solutions of drugs that kill larvae of nasopharyngeal gadflies. Apply in autumn and spring a 2% aqueous solution of chlorophos with a flow rate of 50-100 ml. Preparations of vivomek, ivermectin, baymek, rustomectin, PIVSA, bravomethin and others have high efficiency against all stages of larvae. These drugs are administered subcutaneously at a dose of 0.2 mg per kg of live weight according to ADV, once.

Prevention is poorly developed. It is recommended to eliminate imagos, larvae and pupae or to prevent female gadflies from attacking horses. Since the spread of pathogens can occur with horses coming from disadvantaged farms, it is necessary to carry out preventive treatment during the quarantine period with means that kill the larvae described above.

GASTROPHYLOSIS OF UNGULATES. Gastrophylosis is a widespread chronic disease of horses and donkeys caused by larvae of gastrointestinal gadflies and characterized by inflammatory processes in the places of attachment of larvae and the general pathological condition of the body.Gastrointestinal gadflies belong to the family Gastrophilidae, and the genus Gastrophilus, and species: G.intestinalis – large gastric gadfly, G.veterinus-duodenum, G.haemorrhoidalis-barbel, G.pecorum-herbivore, G.inermis –small gastric gadfly, anchor, G.nigricornis - chernous, G.magnicornis – big–moustached gadfly, goloshei, G.flavipes - donkey gadfly.

Morphology and biology of pathogens. The large gastric gadfly is large, yellowish-brown in color. The head is large, covered with hairs. There are faceted eyes on its sides, and 3 more simple eyes on the crown. The midrib is dark, covered with light yellow or brownish hairs. The wings are transparent, with a pattern of dark spots with light veins. The legs are slender, brownish-yellow. The abdomen is covered with hairs, brownish-yellow above, with dark spots. Eggs are yellowish, transversely striated, large - up to 1,25 mm long, wedge-shaped.

Larvae of stage I at birth are 1,05-1,1 mm long, white, spindle-shaped. Head armament – 2 movable curved hooks and a median point. A mouth opening is located down between the hooks. Stage II larvae are up to 16 mm long. The body is slender,

pointed in front, slightly expanding back. The posterior spiracles have the shape of rounded plates with two slits. Larvae of stage III are oval-cylindrical, up to 20 mm long. The segments of the larva's body from the 2nd to the 10th are covered with two rows of thorns, larger in the first row. The posterior spiracles are in the form of rounded plates with three slits.

Larvae of the large gastric gadfly parasitize horses and donkeys, among other types of gastric gadflies, it is the most numerous and harmful. The average life expectancy of the imago is 10-20 days, the fertility of females is from 887 to 1052 eggs. Immediately after hatching, it sits on elevated places, sun-sanctified rocks and plants, or flies without attacking horses. His behavior changes after mating and females start looking for horses to lay eggs. Years are marked here, in the south – for a longer time, starting from the end of June to September-October months.

Egg laying takes place on the fly. The female attaches one egg to the host's hairline in those places where he can reach with his teeth, in particular on the front legs, shoulders and sides. Externally, horses do not react to the approach of a gadfly. The number of eggs laid per animal reaches 3000-5000. The development of larvae in eggs lasts 7-16 days, but their hatching does not occur immediately. They can remain viable in an egg for 40-50 or even 90 days. In order for the larva to come out of the egg, it is necessary to influence a number of factors: humidity, heat and the touch of a post-root object. Such conditions are created when combing the places of attachment of eggs with teeth. In this case, the lid of the egg bounces off and the larva crawls out.

The larvae that have emerged from the eggs enter the oral cavity of the horse, attach to the mucous membrane of the tongue and develop in it for 21-28 days, after which they molt and pass into stage II. The development of larvae of stages I and II occurs in the stomach. Only isolated instances of them are found in the duodenum and esophagus. In the stomach, they attach their strong screw hooks to its left cardiac part. In the spring of the following year, the mature larvae of stage III leave the stomach and, together with the experiments, go outside. Pupation takes place in the faeces or on the surface layer of the earth. Depending on the temperature, the pupal phase lasts 18-52 days, after which an adult gadfly hatches from it.

Morphology and biology of pathogens. The large gastric gadfly is large, yellowish-brown in color. The head is large, covered with hairs. There are faceted eyes on its sides, and 3 more simple eyes on the crown. The midrib is dark, covered with light yellow or brownish hairs. The wings are transparent, with a pattern of dark spots with light veins. The legs are slender, brownish-yellow. The abdomen is covered with hairs, brownish-yellow above, with dark spots. Eggs are yellowish, transversely striated, large - up to 1.25 mm long, wedge-shaped.

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Treatment. To combat gastrophylosis, chlorophos is used at the rate of 40 mg / kg. Irrigate with a 5% solution of chlorophos grain fodder or hay for 1 kg per animal and feed to horses. A group method of feeding chlorophos with snow for 10-12 hours has been developed. The drug is dissolved in hot water, sprayed into feeders on snow and mixed. Before feeding, horses are kept without water and snow for a day. Free group drinking of 0,1% aqueous solution of chlorophos is also recommended for horses. Medicinal feed pellets containing 1% chlorophos are used. They are fed to adult horses at the rate of 1-1,2 kg, to young animals – 0,5 kg, once.Preparations of vivomek, ivermectin, baymek, rustomectin, PIVSA, bravomethin and others have high efficiency against all stages of larvae. These drugs are administered subcutaneously at a dose of 0.2 mg per kg of live weight according to ADV, once.

To destroy the larvae of gastric gadflies, horses are injected with trolen at a dose of 90 mg / kg and amidophos at a dose of 48 mg / kg through a probe. Anti-gadfly treatment of horses in our republic with two generations of gadflies at the end of July-August and October-November.

Prevention involves the release of animals from larvae and prevention of the spread of infestation with relocated horses.

3.2 TRAINING MATERIALS FOR PRACTICAL CLASSES

Topic No. 1. FASCIOLOSIS OF ANIMALS.

The purpose of the classes: To teach students the morphological structure and biological development of pathogens of paramphistomatosis and orientobilcharciosis of animals, methods of diagnosis of these diseases and their differentiation from other diseases, treatment, prevention and measures to combat paramphistomatosis and orientobilcharciosis of animals.

Materials and equipment. Water, bucket, fecal samples from large and small cattle, saturated solutions of table salt, saltpeter, magnesia sulfate, Darling solution, microscope, slide, cover glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelminitic samples,

FASCIOLIASIS OF ANIMALS AND HUMANS is an acute and chronically occurring trematodous diseases of sheep, goats, cattle, as well as numerous domestic and wild mammals caused by trematodes of two species: Fasciola hepatica and Fasciola gigantica, the family Fascioliidae, which parasitize in the liver and in the bile ducts of the liver and gallbladder. A person also suffers from fascioliasis. The disease is characterized by a disorder of the digestive organs, metabolism, depression, progressive pallor of the conjunctiva, jaundice of visible mucous membranes, bloody panos followed by constipation, the appearance of edema, severe emaciation, decreased productivity, sometimes death of the animal is observed.

Taxonomy. The causative agent of fascioliasis according to the systematics are arranged as follows: type – Plathelminthes, class- Trematoda, subclass- Digenia, order- Fasciolata, families- Fascioliidae, genus- Fasciola and pathogens: Fasciola hepatica and Fasciola gigantica.

Anatomical and morphological structures of pathogens. Fasciola hepatica is an ordinary fasciola, its length is 2-3 cm, width is 0,8–1,2 cm. There are spines on the anterior part of the body on the dorsal and ventral sides. The anterior part of the body is elongated in the form of a proboscis with two suckers, the intestine with lateral processes, the testes are tree-branched, the macta is located in the front part of the body, followed by the ovary, the lateral fields of the parasite are densely filled with clusters of yolks.

Fasciola gigantica- giant fasciola- differs from ordinary fasciola in size and body shape, in the details of the development cycle and pathogenicity. Its length is 4-7,5 cm, width 0,6-1,2 cm, the body is elongated, the lateral edges of the parasite are parallel, are more pathogenic than the common fasciola.

Biology of pathogens. Parasite biohelminths develop with the participation of two hosts: The definitive host of the parasite are numerous domestic and wild mammalian animals, as well as humans. Intermediate hosts are freshwater mollusks of the genus Lymnea, species: Lymnea truncatula and L.auricularia. The prepatent period of development of the parasite is on average from 2.5 months to 4 months, the patent period of development is on average 4-5 years.

Diagnosis and differential diagnosis. The diagnosis is made in a complex manner: based on epizootological data, clinical signs, pathologic and anatomical

changes and an accurate diagnosis in laboratory conditions based on the results of helminthoprological studies of animal feces (helminthoscopy and helminthoscopy).

Fascioliasis should be differentiated from paramphistomatosis, dicroceliosis, orientobilcharciosis, anaplocephalidosis, hemonchosis, marshallagiosis, leptospirosis, paratuberculosis, mechanical hepatitis, gastroenteritis.

Treatment, prevention and control measures. The following anthelmintics are used for deworming: hexachlorparaxylene, hexychol, hexychol C, hexachloroethane, carbon tetrachloride, Dertil B and O, filinsan, faskoverm, fazinex, combitrem, closantal, rolenol and other drugs in recommended doses. Albendazole preparations and its analogues in fascioliasis are less effective (only 15-20% effectiveness).

Prevention. For the complete elimination of fascioliasis, a complex of measures is carried out on the farm aimed at destroying the sexually mature stages of helminths (located in the body of animals), eggs and larvae of fascioles, as well as intermediate hosts. They create conditions that exclude the possibility of infection of the final, intermediate hosts and the introduction of invasion from the outside, and also provide for full-fledged feeding and sanitary and hygienic maintenance of animals.

Preventive deworming is carried out in dysfunctional farms at least twice a year. It is advisable to treat animals 3 months after their transfer to stable maintenance, i.e. when the bulk of the fascioles in the liver reaches puberty.

Security questions:

1. Characteristics of the disease?
2. How are the pathogens of fascioliasis located according to systematics?
3. What is the difference between ordinary fasciola and giant fasciola?
4. When does the infection of animals with fascioles occur and in what ways do young fascioles migrate to the liver?
5. Laboratory methods for diagnosing fascioliasis?
6. From which diseases should fascioliasis be differentiated?
7. What anthelmintics are used for fascioliasis, their doses and methods of application?
8. How are fascioliasis prevented?

TOPIC No. 2. PARAMPHISTOMATOSIS OF ANIMALS.

The purpose of the classes: To teach students the morphological structure and biological development of pathogens of paramphistomatosis of animals, methods of diagnosing these diseases and their differentiation from other diseases, treatment, prevention and measures to combat paramphistomatosis and orientobilcharciosis of animals.

Materials and equipment. Water, bucket, fecal samples from large and small cattle, saturated solutions of table salt, saltpeter, magnesia sulfate, Darling solution, microscope, slide, cover glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

PARAMPHISTOMATOSIS – these are acute and chronically occurring trematodous diseases of cattle, sheep, goats, as well as reindeer and some wild ruminants caused by trematodes of the Paramphistomatata suborder, parasitizing in the small intestine, rumen and less often in the ruminant mesh, characterized by depression, digestive disorders, pallor of visible membranes, groans and gnashing of teeth, loss of appetite, progressive exhaustion, diarrhea, periodically replaced by constipation, swelling in the area of the submandibular space and underbelly, decreased productivity, upholding the growth and development and death of young animals.

Systematics of pathogens. Pathogens of paramphistomatosis according to systematics are arranged in the following order: type- Plathelminthes, class- Trematoda, subclass- Digenia, order- Paramphistomatata, family Paramphistomatidae and Gastrotylacidae 4 genera: Paramphistomum, Liorchis Gastrotylax, Colicophoron and pathogens: P.cervi, P.ichikawai, L. scotiae, G.crumenifer and Colicophoron colicophorm.

Anatomical and morphological structure of pathogens. Of the above pathogens, P.ichikawai, L. scotiae, G.crumenifer and Colicophoron colicophorm are the most widespread in our Republic. Their body is fusiform or cylindrical, pear-shaped, 5-20 mm long, 5-6 mm wide, there is no oral sucker, a powerful pharynx follows the oral opening, passing into the esophagus and intestines, ending in the back of the body. Two large testes lie one behind the other, the ovary is located behind the testes, the yolks are located on the sides of the entire length of the body. The genital openings open in the front of the body, near the intestinal bifurcation. The abdominal sucker is well developed and located near the posterior end of the body, i.e. it is bipolar. The eggs of paramphistomatoids are pale gray, oval in shape, with a well-marked lid at one pole. Their length is 0.120-0.172 mm, width 0.069-0.095 mm, yolk cells do not completely fill the egg cavity.

Biological development of pathogens. Parasites –biohelminth, develop with the participation of freshwater mollusks from the family Planorbidae, species: Planorbis planorbis, P.caritanys, Anisus albus and others. Definitive hosts become infected by eating adulterants. Young forms of the parasite migrate to the submucosa of the duodenum 12 and after 1.5-2 months return to the pre-ventricles (mainly the scar and less often the mesh), where they reach puberty, i.e. the prepatent period of development is 3.5-4.5 months, and the period of parasitization (patent period of development) is on average 2-3 years.

Diagnosis and differential diagnosis. The diagnosis is made on the basis of epizootological data, clinical signs, helminthological examination of feces and the detection of parasite eggs, pathoanatomic changes and the detection of helminths themselves.

Epizootological data. The source of the invasion is adult animals that become infected in areas with low-lying swampy pastures and shallow reservoirs where intermediate hosts live. Sometimes infection also occurs in the stall when feeding grass mown in unfavorable areas. Animals that come out to pasture in the spring become infected with last year's population of larvae. The second summer-autumn rise of the disease is observed in August and September from the generation of fluke larvae this year.

Clinical signs. Cattle are acutely or chronically ill. The acute intestinal form manifests itself in young animals at the age of 1-2 years with a high intensity of invasion. At first, lethargy, decreased appetite are observed, animals lie down, diarrhea occurs, most often with an admixture of blood, wool is ruffled, dull, visible mucous membranes are pale, ulceration on the nasal mirror. Initially, the body temperature rises by 1-1.50 C, but with deterioration, on the contrary, it decreases, edema appears in the area of the interdigital space and the breastbone. The death of animals begins on the 7-8 th day after the first signs of the disease.

The chronic course is caused by the accumulation of a large number of parasites in the pancreas. At the same time, the functions of the gastrointestinal tract are disrupted, the pallor of the mucous membranes is noted. The animal progressively loses weight, feces of a fetid smell.

Helminthoprological studies. Helminthoprological studies are carried out by two methods:

1. Helminthoscopy. Helminthoscopy is performed during the acute course of the disease. For this purpose, successive flushes of feces from sick animals are made to detect young forms of paramphistomats.

2. Helminthoscopy. The method of successive flushes is used, in which parasite eggs are detected.

Pathoanatomic changes. When opening a fallen animal, attention should be paid to the nature of the course of the disease. The acute process is characterized by pronounced swelling and catarrhal hemorrhagic inflammation of the duodenum and the pyloric part of the abomasum, inflammation of the gallbladder with an increase in the volume of the bile duct. Young flukes are detected when examining the intestinal and stomach mucous membranes, and sometimes examine the fluid of the abdominal cavity and the contents of the gallbladder. In the chronic course of adult disease, paramphistomat is found when examining the cavity of the pre-ventricles. In cold corpses, parasites are fixed with the back end to the villi of the scar, book or grid cells. Atrophy of the villi is noted at the attachment sites.

Paramphistomatosis should be differentiated from fascioliasis, orientobilcharciosis, colibacteriosis, paratuberculosis, gastroenteritis.

Treatment, prevention and control measures. The following anthelmintics are recommended: hexachloroethane, 4-carbon chloride, bithionol, resorantel (only for cattle) at a dose of 0.065 g / kg 2 times with an interval of 2 weeks; gilomite – 0.05 g / kg 2 times with an interval of 2 weeks, 1-2% copper sulfate at a dose of 30-40 ml to 120150 ml/head depending on the live weight. Light (white) kerosene in a dose of 15-20 ml per head (except for dairy cows).

Prevention. Prevention consists of a complex of antiparamphistomatous measures:

1. Planned deworming of all affected cattle during the stall period. If necessary, deworming is repeated, but not earlier than two weeks after the first one. In summer, during the period of possible outbreaks of invasion, 3-4 weeks after pasture, preimaginal deworming of young animals under the age of two years is carried out twice with intervals of 10 days;
2. Biothermal disinfection of manure in manure storage facilities;
3. Fight against intermediate hosts of the parasite;
4. Do not graze susceptible animals on unfavorable low-lying, swampy and rich in shallow reservoirs and drainage areas of pasture, you can not drink from standing ponds, small ponds, puddles, ditches, feed grass mown from unfavorable areas of pastures;
5. It is advisable to practice changing pastures;
6. Full feeding of animals according to the diet.

Security questions:

1. Tell us about the characteristics of the disease.
2. Tell the taxonomy of the pathogen.
3. Representatives of how many families and genera belonging to the order Paramphistomatata parasitize in the pre-ventricles of cattle.
4. Briefly describe the anatomical and morphological structure of the pathogen.
5. How does the causative agent of the disease develop?
6. How is the diagnosis made and from which diseases should it be differentiated?
7. Tell us the methods of treatment of paramphistomatosis?
8. How are the disease prevented?
9. Tell us about the health measures for paramphistomatosis?

TOPIC № 3. ORIENTOBILKHARTSIOZ ANIMALS.

The purpose of the classes: To teach students the morphological structure and biological development of pathogens of paramphistomatosis of animals, methods of diagnosing these diseases and their differentiation from other diseases, treatment, prevention and measures to combat paramphistomatosis and orientobilcharciosis of animals.

Materials and equipment. Water, bucket, fecal samples from cattle and small cattle, saturated solutions of table salt, saltpeter, magnesia sulfate, Darling's solution, microscope, slide, cover glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelminitic samples.

ORIENTOBILHARZIOSIS is a focal, chronically occurring disease of sheep, goats, cattle, caused by trematodes from the genus Orientobilharzia Schistosomatata, which is parasitized in the vessels of the mesentery, liver, pancreas,

submucosa of the intestine and the disease is characterized by depression, increased body temperature, increased pulse and respiration, progressive diarrhea with blood and fibrin films, decreased productivity of adult animals, upholding in the growth and development and death of young animals.

Systematics of the pathogen. The causative agent of the disease according to the systematics are arranged as follows: The type is Plathelminthes, the class is Trematoda, the subclass is Digenia, the suborder is Schistosomatata, the families are Schistosomatidae, the genus is Orientobilharzia and the species is Orientobilharzia turkestanica.

Characteristics of the pathogen. Orientobilharzia turkestanica is a kind of bisexual trematodes, the male is 6.4–12.9 mm long and about 48-0.64 mm wide, the oral sucker is rounded, the testes are oblong-oval in the amount of 78-80, arranged in two rows between the intestinal trunks. The female is 4.8-6.8 mm long and 0.08-0.14 mm wide. The suckers are rudimentary, in the uterus one egg is elongated-oval in shape, has one spike on each of the poles, one of them has the appearance of a slightly curved process, and the other is a curved appendage with a constriction on the border with the body of the egg itself. The length of mature eggs is 0.13-0.14 mm and the width is 0.04-0.06 mm, they contain mobile miracidium.

Biology of the pathogen. The parasite is a biohelminth, the intermediate host is freshwater mollusks of the species Lymnea auricularia. The invasive larva is a cercarium. The prepatent development period is 35-40 days, the patent period is more than one year.

Diagnosis and differential diagnosis. The diagnosis is made comprehensively: based on epizootological data, clinical signs, pathoanatomic changes and the detection of parasites themselves, an accurate diagnosis based on laboratory studies of animal feces by the Berman-Orlov method on the detection of orientobilchartsia miracidia, for which first fecal samples are placed in a thermostat for 24-48 hours for growing larvae (miracidia), after which the Berman-Orlov method is used toOrlov is being examined for feces.

Orientobilchartsmosis should be differentiated from fascioliasis, paramphistomatosis, paratuberculosis, gastroenteritis.

Treatment, prevention and control measures. Until recently, the treatment of orientobilcharciosis was not developed, in 1986, Professor Asimov D.A. tested a number of anthelmintics and recommended for treatment: fuadin at a dose of 0.3 ml / kg of animal weight twice with an interval of one day; ambilgar at a dose of 0.03-0.04 g / kg twice orally with an interval of 5 days.

Prevention is based on: 1. Introduction of intermediate hosts by all available (mechanical, chemical, physical and biological) methods;

1. Planned deworming of animals 2 times a year (April-May and September-December);

2. Full feeding of animals according to the diet.

TOPIC No. 4. DICROCELIOSIS AND EURYTHREMATOSIS OF ANIMALS.

The purpose of the classes: To teach students the morphological structure and biological development of pathogens of dicroceliosis and eurythrematosis of animals, methods of diagnosis of these diseases and their differentiation from other diseases, treatment, prevention and measures to combat dicroceliosis and eurythrematosis of animals.

Materials and equipment. Water, bucket, fecal samples from cattle and small cattle, saturated solutions of table salt, saltpeter, magnesia sulfate, Darling's solution, microscope, slide, cover glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelminitic samples.

DICROCELIOSIS is a widely distributed helminthica disease caused by the trematode Dicrocoelium lanceatum, family Dicrocoeliidae, parasitizing the bile ducts and gallbladder in more than 70 species of domestic and wild ruminants. Dicroceliosis also occurs in humans.

Systematics of the pathogen. The causative agent of the disease according to the systematics are arranged in the following order: type - Plathelminthes, class - Trematoda, subclass - Digenia, suborder –Fasciolata, family –Dicrocoeliidae, genus - Dicrocoelium, species - Dicrocoeliumlanceatum.

The pathogen. Dicrocoeliumlanceatum is a lanceolate trematode, length – 7-10-15 mm, width – 1.5-2.5 mm. In the front part of the body there are oral and abdominal suckers. Sexual openings open between them. Behind the abdominal sucker are two testes, an unpaired ovary and a uterus filled with mature eggs. In the middle part of the body, to the side of the intestine, there are cluster-shaped yolks. Dicrocelium eggs are 0.038-0.045 mm long and 0.023-0.030 mm wide, dark brown, asymmetrical, with a thick shell, there is a lid on one pole, a small spike on the other, there is a miracidium inside the egg.

Biology of the pathogen. The parasite is a biohelminth, developing with the participation of three hosts. Intermediate hosts are various species of terrestrial (land) mollusks of the genera Helicella, Zebria, Theba, Fruticola, and additional ants of the genera Formica and Proformica.The prepatent period of development is on average 72-8 6 days, and the period of parasitization is over 4 years.

Diagnostic methods. The diagnosis is made in a complex way: based on epizootological data, clinical signs, pathologic-anatomical autopsy of the animal, during life it is made on the basis of the detection of parasite eggs in the feces, for which helminthioscopy methods are used (Fulleborn, Shcherbovich method)

Dicroceliosis should be differentiated from other trematodous diseases, from gastrointestinal strongylatoses, trichocephalosis.

Treatment. Hexachlorparaxylene hexichol, hexichol C, panacur (fenbendazole), rintal (febantel), bithionol, tafen are used.

Hexychol - to cattle in a dose of 0.3-0.4 g / kg, sheep and goats -0.4 g / kg of animal weight; hexachlorparaxylene – to cattle – in a dose of 0.4-0.5 g / kg, sheep and goats – 0.6 g / kg of animal weight three times with an interval between treatments of at least 1 month, hexychol C – at a dose of 0.3 g / kg; panacur

(fenbendazole) – at a dose of 0.1 g / kg twice with a daily interval; tafen at a dose of 0.21 g / kg of animal weight, once.

Prevention and control measures. For the complete elimination of dicroceliosis, a complex of measures is carried out on the farm aimed at destroying the sexually mature stages of helminths (located in the body of animals), eggs, as well as intermediate and additional hosts. Profilactic deworming is carried out in the autumn-winter months. In the fight against intermediate hosts, the plowing of virgin lands, followed by sowing them with cultivated herbs, the destruction of shrubs, the cleaning of stones on pastures, and where available, the burning of dry grass are effective.

When organizing the fight against dicroceliosis, it is necessary to identify areas of pastures where animals are infected. This can be established by examining mollusks and mainly ants for infection with dicrocelia larvae: in early spring they can be detected throughout the day, and in summer – in the morning and evening. Cattle are not grazed in areas that are unfavorable for this invasion. And also regularly carry out biothermal disinfection of manure and feeding animals according to the diet.

EURITREMATOSIS is an invasive disease of sheep, goats, cattle, camels, pigs, deer and many wild ruminants caused by the trematode Euritremapancreaticum. Dicrocoeliidae, parasitic in the pancreas and very rarely in the liver. The disease is characterized by severe exhaustion, swelling in the head, neck, chest and sometimes death of the animal when cachexia is detected.

Taxonomy. The causative agent of the disease according to the systematics are arranged in the following order: type - Plathelminthes, class - Trematoda, subclass - Digenia, suborder –Fasciolata, families –Dicrocoeliidae, genus - Euritrema, species - Euritremapancreaticum.

The instigator of the disease. Euritremapancreaticum is 8-16 mm long and 5.5-8.5 mm wide, with large, strongly protruding oral and abdominal suckers. Live specimens are bright red in color. The testes are oval, arranged symmetrically on the sides of the abdominal sucker, the ovary is spherical, lies behind the abdominal sucker, several times smaller than the testes, the uterus is tree-branching occupies the entire posterior half of the fluke. Eurytrem eggs are dark brown in color, 0.044-0.048 mm long, 0.032-0.036 mm wide, a lid is formed on one pole, an appendage in the form of buttons on the opposite.

Biology of the pathogen. The parasite is a biohelminth, the intermediate host is a land mollusk of the species Eulotalantzi, an additional host is insects grasshoppers from the genus Conocephalus and crickets Oecanthus. The prepatent period of development, i.e. the period of reaching puberty is 2-3 months, and the period of parasitization (patent) in the body of animals is not more than one year.

Diagnostic methods. The diagnosis is made on the basis of epizootological data, clinical signs, pathological and anatomical changes, and during life it is made on the basis of helminthioscopy: sequential washing of feces or the Darling method on the detection of eurytrem eggs.

Eurythrematosis should be differentiated from pancreatitis, dyspepsia, gastroenteritis.

The treatment has not been developed.

Prevention. For the complete elimination of eurythrematosis, a set of measures is carried out on the farm:

1. Planned systematic deworming of animals;
2. Biothermal treatment of manure;
3. Fight against intermediate and additional hosts;
4. Feeding animals according to the diet.

Security questions:

1. Tell the characteristics of dicroceliosis.
2. Tell the taxonomy of the pathogen.
3. Briefly tell us the anatomical and morphological structure of the pathogen and its biological development?
4. How do we diagnose and from which diseases should we differentiate?
5. Tell us the method of treatment of dicroceliosis?
6. How is the disease prevented?
7. What are the health measures for dicroceliosis?
8. Tell us the characteristics of eurytrematosis?
9. Tell us the taxonomy of Eurytrema pancreaticum?
10. Tell us the anatomical and morphological structures of Eurytrema pancreaticum?
11. How does Eurytrema pancreaticum develop?
12. How do we diagnose eurytrematosis and from which diseases should it be differentiated?
13. Tell us about the health measures of the farms from eurythrematosis?

TOPIC No. 5. BOVINE CYSTICERCOSIS CATTLE AND PIGS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of causative agents of cysticercosis of cattle and pigs, methods of diagnosis, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, samples of affected and healthy carcasses of cattle and pigs, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

CYSTICERCOSIS (FINNOSIS) CATTLE is a subclinically occurring, anthropozoic, cestodose disease of cattle caused by Cysticercus bovis by the larval stage of the cestode Taeniarhynchus saginatus, parasitizing in the human intestine. Cysticerci are localized in the intermuscular connective tissue, skeletal muscles, heart, tongue, less often in the tissues of parenchymal organs.

Systematics of the pathogen. The causative agent of bovine cysticercosis according to the systematics are arranged in the following order: type –

Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Taeniata, families - Taeniidae, genus - Taeniarhynchus: 1. The ribbon form is Taeniarhynchus saginatus2. The larval form is Cysticercus bovis.

The pathogen. Cysticercus bovis is a fully formed cysticercus of grayish-white color, transversely oval shape, 5-15 mm long, 3-8 mm wide. On its inner shell there is a large scolex, 1,5-2 mm in diameter, equipped with four suction cups. Outside, the cysticercus is surrounded by a delicate connective capsule.

The sexually mature stage -Taeniarhynchus saginatus reaches up to 10 m (or more) in length and 12-14 mm in width. On the inner shell of the cysticercus there is an unarmed, large scolex, located in an inverted position, the diameter of the scolex is 1,5-2,0 mm. Proboscis in the form of a rudimentary formation, filling the apically located sucker. The diameter of the suction cups is 0.8 mm. The genital openings alternate very incorrectly, the ovary is two-lobed, the blades are rounded, equal in size, there is no ovary. There is a vaginal sphincter, mature segments are 16-20 mm long, they move energetically, In mature segments the uterus is in the form of a median trunk, from which 18-32 lateral branches depart to the right and left. There are 2 long filaments on the outer shell of the egg. Oncospheres have a thick two-contour radially striated shell of yellow-brown color, 0.03-0.04 mm long and 0,02-0.03 mm wide.There are up to 145-175 thousand eggs in one segment. In the presence of one cestode, the helminth carrier can allocate from 175 thousand per day . Up to 4 million 900 thousand oncospheres, and at the end of the year – about 440 million oncospheres.

Biology of the pathogen. The parasite is a biohelminth, the definitive host is only a human, and the intermediate hosts are cattle, buffalo, yak, zebu and northern deer. The prepatent period of development is on average 3 months, the life expectancy of the teniarynchus is more than 10 years. The period of reaching the invasive stage is 4.5 months.

Diagnosis and differential diagnosis. The diagnosis is made on the basis of epizootological data, clinical signs (not characteristic), pathoanatomic changes, during life on the basis of immuno-biological diagnostics, the reaction of latex agglutination, which has not yet entered into widespread practice.

Post-mortem diagnosis is based on the detection of cysticerci in cattle carcasses during post-slaughter examination. The luminescent method significantly increases the percentage of detection of cysticerci in meat.

Bovine cysticercosis should be differentiated from sarcocystosis, in which Mischer's sacs are formed.

Treatment. Panacur (fenbendazole in the form of 22.2% granulate) at a total dose of 0.05 g / kg of body weight (according to ADV), droncit (praziquantel) at a dose of 0.01 g / kg, twice, individually (Bessonov and others, 1980), nilverm at a dose of 0.025 g/ kg twice with an interval of 5 days, individually (M.V., Alferova, 1987).

Pig cysticercosis (cellulose) – this is chronically or subclinically occurring, anthropozoonosis-cestodosis, caused by Cysticercus cellulosae – the larval stage of the Taenia solium cestode, parasitizing in the human intestine, and the cysticerci

localize themselves in various organs and tissues, they are most often found in pigs in muscles, heart, brain, eyes, liver and lungs, and in humans – in brain and eyes.

Systematics of the pathogen. The causative agent of pig cysticercosis according to the taxonomy is arranged in the following order: type –Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Taeniata, family - Taeniidae, genus - Taenia: 1. Ribbon form - Taeni solium2. The larval form is Cysticercus cellulosae.

The pathogen.Cysticercus cellulosae – fully formed cysticerci – red ellipsoidal bubbles, measuring 6-20 mm in length and 5-10 mm in width, the wall of the vesicle is double-layered, filled with a slightly spalescent liquid, in which the scolex attached to the inner shell, in the screwed state, shines through the wall of the cysticercus in the form of a white speck and has the same structure, as the scolex of the sexually mature Taeni solium.

The ribbon stage of Taeni solium does not exceed 3 m in length.Skolex is armed with a double crown of hooks, the number of which ranges from 22-32. Hooks of a large row of 0,16-0,18 mm in length, small 0,11-0,12 mm in length. A characteristic feature of hermaphrodite segments is the presence of an additional third lobe of the ovary. In the mature segment, 7-12 lateral branches depart from the median trunk of the uterus on each side, forming, in turn, a small number of lobes.

Biology of the pathogen. The parasite is a biohelminth, the definitive host is a human, and the intermediate hosts are a domestic pig, wild boar, bear, camel, dog, cat, rabbit, hare, and also a human. The invasive larva reaches maturity in 2-4 months, the life span of the cysticerci in the body of intermediate hosts is 3-6 months, after which they shrivel, are soaked in lime and die. The prepatent period for the development of the sexually mature stages of the pathogen is 2-3 months, and the period of parasitization in the body of the definitive host is calculated in years.

Diagnostic methods. The diagnosis is made comprehensively: based on epizootological data, symptoms of the disease and pathoanatomic changes. From epizootological data: the only source of infection with pig cysticercosis is a person, the invasion is spread mainly in areas of highly developed pig breeding, a person becomes infected with teniosis when eating insufficiently cooked or fried meat containing invasive cysticerci, and sometimes when eating bacon, ham, brisket, in which viable cysticerci can be preserved with weak salting and cold smoking.

Clinical signs. The disease usually proceeds without clinical signs, so it is impossible to register it, even with a strong intensity of invasion, sick pigs, as a rule, seem perfectly healthy. In some cases, pigs have disorders in the coordination of movements, and with the localization of cysticerci in the eye and brain, specific signs may appear, edema, cachexia, epileptic seizures develop; blindness, the disease can end in death. With the localization of cysticercus in the tongue and under the mucous membrane of the anus, pathological changes can be diagnosed during life. In highly invasive pigs, cysticerci are found in the perineal region.

Pathoanatomic changes are based on detection in pig carcasses during post-mortem examination. In pigs, cysticerci are more often localized in the occipital and shoulder-shoulder muscles, in the heart, tongue and brain, and very rarely in parenchymal organs.

Pig cysticercosis should be differentiated from trichinosis and sarcocystosis.

The treatment has not been developed. With the localization of cysticerci in the eyes, surgical treatment is performed.

Prevention of cesticerciasis of cattle and pigs. Measures to combat cysticercosis of animals, in which a person is the sole owner, i.e. the source of infection of animals, are unthinkable without coordinated actions of veterinary and medical organizations.

Veterinary line measures are aimed at preventing human infection with theriosis and teniarinchosis, which is ensured by conducting a vet.examination of cattle and pig meat and preventing the possibility of animal invasion by cysticercoses. Animal carcasses are examined for cysticercosis; backyard slaughter is prohibited; cysticercose carcasses should be disinfected only with the knowledge of veterinary supervision; organize slaughter points, market points for decontamination of conditionally fit and cysticercose meat; categorically do not return carcasses burned with cysticercose to owners; all cases of detection of cysticercosis should be immediately reported to the appropriate medical organizations to identify sick persons affected by teniosis and teniarinchosis and immediately carry out therapeutic and preventive measures among persons who were the source of infection of animals; carry out mandatory labeling of animals entering for slaughter and keep accurate records of slaughtered livestock; organize proper maintenance and feeding of animals; prevent vagrancy of animals;aategarically prohibit the access of livestock to the territory of human habitation;

A complex of medical measures. To do this, the following is necessary:

1. Systematic examination of people in order to identify all patients with teniosis and teniarinchiasis, followed by deworming;

2. Systematic protection of soil and water from contamination by human faeces. Arrangement of public and individual toilets, carrying out systematic monitoring of their sanitary condition. Conduct the most careful monitoring to ensure that the population uses toilets, preventing the scattering of excrement on the external environment.

3. Sanitary control over the disposal of human excrement and the state of wastewater, excluding the possibility of the spread of invasion.

4. Compliance with personal preventive measures by a person. Do not eat raw, unboiled or deep-fried meat and internal fats

Sanitary-helminthological education of the population. Sanitary and educational work should be systematically carried out among the general population by both veterinary and medical and school workers. In order to involve the population in an active fight against gklmintosis, they use all the opportunities that are provided in specific conditions (lectures, talks, speeches in print, on radio and television, demonstration of popular science films, publication of scientific and popular literature, the release of posters, leaflets, etc. Special seminars are systematically organized for veterinary and medical workers, first of all it is necessary in areas that are disadvantaged by teniidoses.

Security questions:

1. How is the systematics of the exciterscysticercosis of cattle and pigs located?
2. Tell us the anatomical and morphological structures of Cysticercus bovis and its differences from Cysticercus cellulosae?
3. Tell us the anatomical and morphological structures of Taeniarhynchus saginatus and its differences from Taenia solium?
4. Tell us the biology of the causative agent of cattle cysticercosis and its differences from pig cysticercosis?
5. How do we diagnose cysticercosis during the life of the animal?
6. How do we treat sick people with teniarynchosis and teniosis?
7. How do we prevent cysticercosis?
8. If sick animals with cysticercosis of cattle or pigs appear on the farm, what should we do, what measures should we take to improve the farms from the disease?

TOPIC № 6. ANOPLOCEPHALATOSIS OF RUMINANTS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of anoplocephalatoses of ruminants, methods of lifetime and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from sheep, goats and cattle, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

Anoplocephalatosis. Representatives of the suborder Anoplocephalata in the ribbon stage parasitize in the intestines of numerous species of domestic and wild animals.

A characteristic feature of anoplocephalitis is the absence of chitinous hooks on the scolex, sexual openings overlap on the lateral surface of the segment, and the development cycle occurs with the participation of oribatid mites, in which an invasive larva – cysticercoid develops.

Anoplocephalatosis of ruminants. Representatives of the Anoplocephalidae and Avitellinidae families parasitize from the suborder Anoplocephalata in sheep, goats and cattle in the imaginal stage. The causative agents of these cestodoses belong to four genera:Moniezia, Thysaniezia, Avitellina and Stilezia.

They are localized in the small intestine and cause independent diseases: moniesiosis, tizanesiosis, avitellinosis and stylesiosis.

Moniesiosis of ruminants is a chronically occurring cestodosis disease of sheep, goats, cattle and wild animals caused by cestodes of the Anoplocephalidae family: Monieziaexpansa, Monieziabenedeni and Monieziaautumnalia, parasitizing in the small intestine and characterized by depression, gastrointestinal tract disorders,

severe diarrhea, emaciation and decreased productivity and feeding of young animals , especially lambs and goats.

Systematics of pathogens of the disease. The causative agents of moniesiosis according to the systematics are arranged in the following way: type – Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Anoplocephalata, families: Anoplocephalidae,genus- Moniezia, species: 1. Monieziaexpansa, 2. Monieziabenedeni and 3. Monieziaautumnalia.

Pathogens of the disease. Monieziaexpansa- strobila milky white, dense, its length reaches 10 m, the segments are wide but short, the scolex with four suckers is not armed. At the leading edge of the segment, the interproglottid glands are arranged evenly in groups in the form of rosettes, in the hermaphrodite segment from 150 to 327 testes and two sets of female sex glands. The sexual openings are closed on each side of the segment. A young uterus in the form of a network of intricately intertwining thin tubes. Growing, it fills all the middle fields of the segment and the side fields. Later, the tubes of the uterus expand greatly, and the mature segment takes on the character of a bag filled with eggs.

Moniezia benedeni - strobi is yellow-white, wide, thin, translucent, its length is up to 4 m, with a width of 1.6 cm, a scolex with four suction cups, unarmed. In the anterior part of the segments, starting from the 12th-15th cm of the scolex, the interproglottid glands are located in the form of a flat strip along the middle line of the strobila.Two sets of female genital glands are located on the sides of the segment, the opening is two-sided. The uterus initially looks like a network, and with the arrival of eggs, it turns into a wide sac that occupies the entire segment.

Moniezia autumnalia. Strobila is white, dense. Opaque, its length is 2.5 m, maximum width is 8 mm, interproglottid glands. The same as in Monieziabenedeni, linear type, located on the ventral and dorsal sides of the strobila along the midline, however, the length of hermaphrodite and mature segments is equal to or exceeds the width, while the proglottids of Monieziabenedeni are very short but wide, and the number of testes is 3 times greater.

Biology of pathogens. Paravzites are biohelminths, develop with the participation of an intermediate host. Intermediate hosts are oribatid (soil, shell) mites of the genus Scheloribates. The invasive larva is a cysticercoid. Prepatent period of development: in Monieziaexpansa – 38-40 days, and in Monieziabenedeni – 42-49 days. The life span of moniesias is 2-7 months.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs, helminth-coprological studies (helminthoscopy, helminthoscopy and preventive deworming), and posthumously on the basis of pathologic and anatomical changes and the detection of the cestodes themselves in the small intestine.

Helminthoscopy of lambs' feces for infection with monies is usually carried out in animal parking areas. It is better to inspect them early in the morning or after a day's rest. The segments of moniesias are yellow-white, lie on the surface of the feces and usually contain mature eggs of moniesias. These segments should be distinguished from the segments of the tizanesias and avitellia. Avitellinids

(tizanesium and avitellinae) differ from moniesia, firstly, by a single sexual apparatus, while moniesia have a double one; secondly, avitelinids lack interproglottid glands in hermaphrodite segments; thirdly, in avitellinids, the oncosphere does not have a pear–shaped apparatus, and the eggs in mature segments are covered with a capsule (there are many capsules in tizanesias, one in avitellia).

For a faster and more accurate diagnosis of moniesiosis, fecal samples taken directly from the rectum are examined for the presence of segments. P.N. Vimbe suggested taking fecal samples using a vaginal mirror. To do this, 100-150 ml of water is poured into the rectum from a rubber syringe to remove feces and mucous membrane. After that, a vaginal mirror is inserted. Fecal balls are quickly poured into the substituted cuvette, where they are examined.

Helminthoscopy. The Fulleborn method is usually used. Monieziaexpansa eggs are hexagonal in shape with rounded edges, under a microscope they are visible in the form of an irregular triangle. Mature eggs of Monieziabenedeni are ten– and very rarely twelve-sided, under the microscope they have figures of an irregular quadrilateral (almost square) or pentagon, with slightly rounded corners.

Diagnostic deworming is carried out 14-16 days after the animals are driven out to pasture. To do this, 50 lambs are usually taken from the flock and dewormed with copper sulfate.

Tizanesiosis is a chronic ongoing cestodosis disease of sheep, goats, cattle and other ruminants caused by cestoda Thysanieziagiardi, family Avitellinidae, parasitized in the small intestine and characterized by depression, impaired gastrointestinal function, diarrhea, anemia of the mucous membrane,nervous phenomena, decreased productivity and sometimes the death of animals older than one year.

Systematics of the pathogen. It is arranged according to the taxonomy in the following order: – Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Anoplocephalata, families - Avitellinidae, genus -Thysaniezia, species - Thysanieziagiardi.

The causative agent is Thysaniezia giardi - a white cestode, 4.3 m long, with a maximum width of 8.7 mm, an unarmed scolex with four suckers. The genital openings are one-sided, they alternate incorrectly, the uterus in the form of a transverse tube extends across the entire width of the anterior half of the segment, the eggs lie in groups in the protrusions of the uterus, which gradually increase in size and eventually occupy the entire uterus, many parotid organs are formed, each of them contains 5-15 eggs.Eggs without a pear-shaped apparatus.

Biology of the pathogen. The parasite is a biohelminth, the development cycle is not deciphered, it is assumed that the intermediate host is hay-eating insects or oribatid mites. Invasive cysticercoid larva, the prepatent development period is 45-50 days, the patent period is 6-8 months.

Avitellinosis is a chronic ongoing cestodosis disease of sheep, goats, cattle and other ruminants caused by cestoda Avitellinacentripunctata, family Avitellinidae, parasitized in the small intestine and characterized with signs of central nervous system damage, then depression, gastrointestinal dysfunction, diarrhea, anemia of the

mucous membrane, nervous phenomena, decreased productivity and unexpected death of animals.

Systematics of the pathogen. It is arranged according to the taxonomy in the following order: – Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Anoplocephalata, families- Avitellinidae, genus – Avitellina, species Avitellinacentripunctata.

The pathogen. Avitellina centripunctata- strobila up to 3 m long, narrow, fills the cord, the scolex is unarmed with four suction cups, the last segments are clearly visible in the form of rosaries, the sexual openings are one-sided, incorrectly alternate. The uterus is surrounded by a perinatal organ, breaks up into capsules containing eggs, the oncosphere is large with three pairs of embryonic hooks.

Biology of the pathogen. The parasite is a biohelminth, the development cycle has not been deciphered until recently, in 1988 P.T. Tverdokhlebov and others established that the intermediate hosts are insects-collemboles from the genus Entomobrya. Invasive larva cysticercoid, prepatent period of development about 1.5 months, patent period – 6-8 months.

Stilesiosis is a chronically occurring cestodosis disease of sheep, goats, cattle and other ruminants caused by cestoda Stilesiaglobipunctata, family Avitellinidae, parasitizing in the small intestine/

Systematics of the pathogen. The taxonomy is arranged in the following order: – Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Anoplocephalata, families- Avitellinidae, genus – Stilesia, species - Stilesiaglobipunctata.

The pathogen. Stilesia globipunctata – strobila is very thin and delicate, its length is about 60 cm, segmentation is invisible from the outside, the sexual apparatus is single, the uterus is in the form of a thin transverse tube, two paruternal organs in the mature segment, eggs are 0.027x0.014 mm, have the shape of a small thick spindle.

The biology of the pathogen has not been deciphered. It is believed that the intermediate host is oribatid mites. Invasive larva – cysticercoid, prepatent period of development -1.5 months, patent period – 6-8 months.

Diagnosis and differential diagnosis. The diagnosis is made during life: on the basis of epizootological data, clinical signs and helminthological studies. The methods of helminthoscopy are used (segments of the tizanesias up to 10 mm long and 2 mm wide, white in color, resemble a grain of rice in appearance, sexual openings are clearly visible in it, which open on one side of the segment and alternate incorrectly), the Fulleborn method (in the segments of the tizanesias eggs (3-8 pieces) are included in characteristic capsules, they can only be found in freshly separated segments; when drying, the segments bend sickle–shaped, the capsules are pulled out and appear on the surface of sheep feces in the form of a white plaque) and diagnostic deworming (the same as with moniesiosis), and posthumously - based on the autopsy of fallen animals and the detection of the cestodes themselves in the small intestine.

Treatment, prevention and measures to combat anoplocephalatoses of ruminants. They recommend phenasal, phenalidone, bithionol, arsenic acid tin,

copper sulfate, panacur (fenbendazole), rintal (febantel), albendazole, monesin, monezol and other anthelmintics in recommended doses and application regimens.

In unfavorable anoplocephalic farms, it is necessary to carry out both pasture prevention (isolated rearing of young animals, the use of stall, stall-camp and stall-walking keeping of young animals) and special measures (preventive deworming and chemoprophylaxis) taking into account local conditions.

Calves of the current year of birth are processed 35-40 days after the animals are driven to pasture and again 35-40 days after the first one. A month after the second treatment, the final deworming is carried out.

Lambs are dewormed for the first time 14-16 days after they are driven to pasture, for the second time 15-20 days after the first and for the third time 25-30 days after the second deworming. At the end of September, if necessary, another deworming is recommended. A month after the transfer of sheep to stable maintenance, the entire livestock is dewormed.

From October to May months, it is recommended to feed animals with a salt-phenothiazine-copper sulfate seed (1 part copper sulfate, 10 parts phenothiazine and 89 parts feed salt) for 8 months at a dose of 1.0 per head, and it is also necessary to carry out regularbiothermal disinfection of manure and full-value feeding of animals according to the diet.

Security questions:

1. How many representatives of the families of the order Anoplocephalata are parasitic in ruminants?
2. How many genera of the Anoplocephalidae family are parasitized in ruminants?
3. How do representatives of the Anoplocephalidae family distinguish from Avitellinidae?
4. How do we distinguish Moniezia expansa from Moniezia benedeni?
5. How do we distinguish Moniezia expansa from Thysaniezia giardi.
6. How do we distinguish Thysaniezia giardi from Avitellina centripunctata and Stilesia globipunctata?
7. Tell us the biological development of the causative agents of moniesiosis and its differences from the causative agents of tizanesiosis, avitellinosis and styleziosis?
8. How is the diagnosis made with anoplocephaliasis of ruminants?
9. From which diseases should anoplocephaliasis of ruminants be differentiated?
10. What anthelmintics are used for ruminant anoplocephalitis?
11. Which of these anthelmintics are the most effective?
12. How do we prevent anoplocephalatosis in animals?
13. Tell us the methods of chemoprophylaxis of anoplocephalatosis of ruminants and the mixtures used?

TOPIC № 7. ANOPLOCEPHALOSIS OF UNGULATES.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of anoplocephalatoses of ungulates, methods of in vivo and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from horses and donkeys, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

Anoplocephalatoses of horses are caused by three types of cestodes of the Anoplocephalidae family: Anoplocephalamagna, Anoplocephalaperfoliata and Paranoplocephalamillana, localized in the small and large intestines, characterized by impaired digestive function, enteritis, colic, settling in growth and development, the appearance of edema, sometimes symptoms of peritonitis and the case of young animals.millana, localized in the small and large intestines, characterized by impaired digestive function, enteritis, colic, settling in growth and development, the appearance of edema, sometimes symptoms of peritonitis and the case of young animals.

Systematics of pathogens.Pathogens of anoplocephalidosis of ungulates according to the taxonomy are arranged as follows: type-Plathelminthes, class-Cestoda, order-Cyclophyllidea, suborder - Anoplocephalata, families-Anoplocephalidae, genera: Anoplocephala and Paranoplocephala and species: Anoplocephala magna, Anoplocephala perfoliata and Paranoplocephala mamillana.

Pathogens.Anoplocephalamagna –up to 52 cm long and 2.5 cm wide, the scolex is spherical, unarmed, 2.8-3.0 mm in diameter, with powerful spherical suckers, there is no neck. The sexual apparatus is single, the sexual openings are one-sided, the eggs are 0.072-0.084 mm in size, with a poorly developed pear-shaped apparatus.It is localized in the jejunum and ileum.

Anoplocephalaperfoliata - up to 70 mm long and 8-14 mm wide.The scolex is almost cubic in shape, about 3 mm in diameter, with well-developed suckers.Lokazil is used in the blind and colon

Paranoplocephalamamillana-10-40 mm long, 5-6 mm wide, unarmed scolex, about 7-0.8 mm in diameter. The sexual apparatus is single, the sexual openings are one-sided. The eggs are 0.05-0.06 mm in diameter, the pear-shaped apparatus is larger than the radius of the egg. It is localized in the small intestine.

Biology of the pathogen. The parasites are biohelminths, the intermediate host is oribatid (shell) mites, the invasive larva is a cysticercoid. The prepatent period of development is 1-1.5 months, the period of parasitization is about one year.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and results of helminthoprological studies (methods of helminthoscopy and Fulleborn) of animal feces and the detection of mature segments or anoplocephalid eggs, and posthumously on the basis of pathoanatomical changes and the detection of the cestode itself.

Anoplocephalidosis of horses should be differentiated from parascaridosis, oxyurosis, intestinal strongylatosis, enteritis, colic.

Treatment. Phenasal, phenalidone, panacur (fenbendazole), rintal (febantel), albendazole preparations and its analogues are used.

Prevention is based on:

1. Planned preventive deworming;
2. The regularistic struggle with the intermediate host of the parasite;
3. Regular cleaning of stables from manure, followed by its biothermal disinfection:
4. Complete feeding of animals according to the diet.

Security questions:

1. Give a description of anoplocephalidosis of horses?
2. Tell us the systematics of pathogens of anoplocephalidosis of horses?
3. Tell us the anatomical and morphological structures and biological development of pathogens of anoplocephalidosis of ungulates?
4. How many representatives of the genera of the Anoplocephalidae family parasitize ruminant horses?
5. Tell us the methods of lifetime diagnosis of equine anoplocephalidosis?
6. From which diseases should equine anoplocephalidosis be differentiated?
7. Treatment of equine anoplocephalidosis, which of the recommended drugs is the most effective anthelmintic?
8. How do we prevent equine anoplocephalidosis, if the disease has arisen, then we should carry out what health measures?

TOPIC № 8. ASCARIASIS. PARASCARIDOSIS OF HORSES AND ASCARIASIS OF PIGS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of paraskaridosis of horses and ascaridosis of pigs, methods of in vivo and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from horses and pigs, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

PARASCARIDOSIS OF HORSES is a disease of ungulates: horses, donkeys, mules, caused by nematodes Parascarisequorum,family Ascaridae, parasitic in the small intestine, larval forms of which migrate in the body along the hepatopulmonary pathway and are characterized at first by depression, disorders of the gastrointestinal tract, diarrhea, then there is a violation of respiratory function, cough, expiration from nose and then again a violation of the digestive function,

diarrhea, a decrease in fatness and emaciation of young animals, and sometimes sudden deaths of young pigs –weaning pigs.

Systematics of the pathogen: According to the systematics, the pathogen is arranged in the following order: type- Nemathelminthes, class - Nematoda, suborder – Ascaridata, families-Ascaridae, genus-Parascaris, species - Parascarisequorum.

Morphological structures of the pathogen. Parascarisequorum is a white nematode with an elastic spindle–shaped body. The mouth is surrounded by three large lips, the edges of which are equipped with teeth: small intermediate lips are placed between the larger lips. The male is 15-28 cm, outwardly differ from the female in smaller size and a curved tail end, where there are small lateral wings and 79-105 pairs of sexual papillae. The female reaches 37 (45) cm in length: the opening of the vulva at the level of the anterior quarter of the body length. Paraskarid eggs are round, 0.09-0.10 mm in diameter, with a thick shell, dark brown in color, unfertilized eggs are light.

Biology of the pathogen. The parasite is a geohelminth, develops without the participation of an intermediate host. Isolated eggs in the external environment at a favorable temperature and sufficient humidity ripen in 7-8 days . Animals become infected by ingesting invasive eggs with food or water. The larvae leave the egg in the intestines of the horse and penetrate into the blood vessels, then the parascarids migrate along the hepatopulmonary pathway and after 22-23 days they again enter the small intestine. The prepatent period of development is 2-2.5 months, and the period of parasitism in the body is on average up to a year.

Diagnosis and differential diagnosis. The diagnosis during the life of the animal is made on the basis of epizootological data, clinical signs and helminthological studies.Feces are examined by the Fulleborn flotation method in order to detect paraskarid eggs. Paraskarid eggs are round, 0.09-0.10 mm in diameter, with four thick shells, (the outer shell is smooth), dark brown, unfertilized eggs are light.

Posthumously, the diagnosis is made on the basis of pathoanatomic changes and the detection of helminths themselves.

Parascaridosis of horses should be differentiated from anoplocephalidosis, intestinal strongylatosis, oxyurosis, enteritis, sometimes from peritonitis.

Treatment. Piperazine, piavetrin, fenbendazole (panacur), febantel (rintal), carbon tetrachloride, mebendazole, marantel tartrate are used in the recommended doses and application schemes.

Prevention. Preventive deworming of horses 2 times a year: foals of the current year of birth - the first time in August, the second time – after weaning; adult animals - the first time in March-April and the second time in October-November: After the treatment of animals, the stables are disinfected. If deworming was on the pasture, then the horses are kept for 3-4 days on a separate plot, after which it is plowed.In addition, preventive measures are carried out: timely manure cleaning, systematic cleaning of premises, feeders, care items, followed by their disinfection. Adobe floors in stables, sheds, walls and stalls are re-stamped at least once a year and covered with a fresh layer of clay, 10-15 cm thick. A separate machine is fixed for

each horse. In addition, animals should be fed full-fledged food regularly to increase the body's resistance.

PIG ASCARIASIS is a disease caused by nematodes of the Ascaridae family, Ascaridata suborder, parasitizing in the small intestine and characterized by an increase in body temperature to 41.50 C, pneumonia, decreased appetite, increased breathing, the appearance of anxiety and cough of the animal, settling in the growth and development of young animals and a decrease in the productivity of adult animals, sometimes 6-7-month-old piglets are suddenly fed and they die (they squeal, they are served on the floor, they start convulsing, their breathing quickens, their skin turns red, their eyes open wide).

Systematics of the pathogen: According to the systematics, the pathogen is arranged in the following order: type- Nemathelminthes, class - Nematoda, suborder– Ascaridata, families-Ascaridae, genus-Ascaris, species–Ascarissuum.

Anatomical and morphological structure of the pathogen.–Ascarissuum is a relatively large nematode of pinkish–white color, wind-shaped. The mouth opening is surrounded by three lips, the esophagus is simple, cylindrical. The male is 10.5-22 cm long and about 3 mm wide, has two equal spicules 1.2-2 mm long. Females are 23-30 (35) cm long and 5-6 mm wide, the opening of the vulva spreads in the anterior third of the body.The eggs are dark cinnamon-colored, 0.050-0.087 mm long, 0.040-0.055 mm wide, covered with a very thick coarse-browned shell. There is one germ cell in a freshly separated egg.

Biological development of the pathogen. The development cycle is direct, i.e. without the participation of an intermediate host. In the external environment, under favorable conditions (with the onset of oxygen, temperature and sufficient humidity), mobile invasive larvae develop within 2-3 weeks inside the eggs. The hepatopulmonary migration period is 22-23 days. The prepatent period of development is 1.5-2.5 months, and the period of parasitization is 7-10 months.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and the results of helminthological studies of feces by the Fulleborn or Shcherbovich method in order to detect the eggs of the pathogen.

Posthumously on the basis of pathoanatomic autopsies of animals and the detection of ascarids themselves in the small intestine.

Ascariasis of pigs should be differentiated from metastrogylosis, trichocephalosis, esophagostomosis, bunostomosis, colibacteriosis, paratyphosis and dyspepsia.

Treatment. For pig deworming, piperazine, piavetrine, nilverm, chlorophos, panacur (fenbendazole), rintal (febantel), albendazole and others are used in the indicated doses and application schemes.

Prevention. 1. Carry out regular preventive deworming of animals taking into account the biological development of the pathogen. Weaning piglets are used 1.5-time deworming, which are dewormed the first time - on the 55-60 th day of life, the second time 25-30 days after the first deworming;

2. Biothermal disinfection of manure;

3. Regular cleaning, disinfection and whitewashing of premises ;
4. Fighting the reservoir host;
5. Do not keep adults and young animals together:
6. Full feeding of animals according to the diet.

Security questions:

1. Give a description of pig ascariasis?
2. Tell us the taxonomy of the causative agent of ascariasis?
3. Tell us the morphological structure of Ascarissuum?
4. Tell us the biological development of Ascarissuum?
5. Coprological method for diagnosing ascariasis of pigs?
6. From which invasive and non-infectious diseases should ascariasis be differentiated?
7. How do we treat ascariasis in pigs, tell us the doses and schemes of anthelmintics used?
8. Describe the parascaridosis of horses?
9. Tell us the taxonomy of the causative agent of parascaridosis?
10. Tell us the morphological structure of Parascarisequorum?
11. Tell us the biological development of Parascarisequorum?
12. Coprological method of diagnosing horse parascaridosis?
13. From which invasive and non-infectious diseases should parascaridosis be differentiated?
14. How do we treat parascaridosis, tell us the doses and schemes of anthelmintics used?
15. How to prevent ascariasis of pigs and ascariasis of horses in dysfunctional farms for this helminthiasis?

TOPIC No. 9. OXYUROSIS AND INTESTINAL STRONGYLATOSIS IN HORSES

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of equine oxyurosis, methods of lifetime and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from horses and donkeys, various volumetric cups, saturated salt solutions, magnesia sulfate and saltpeter, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

Oxyurosis is a chronically occurring nematode disease of single–hoofed animals caused by nematodes Oxyuris equi of the Oxyuidae family, parasitizing mainly in the blind and large colon and characterized by the manifestation of a violation of the digestive tract, skin lesions at the root of the tail ("combing" of the tail).

Systematics of the pathogen. The causative agent of the disease according to the systematics are arranged in the following order: type - Nemathelminthes, class - Nematoda, suborder - Oxyuirata, families – Oxyuidae, genus – Oxyuris, species - Oxyuris equi.

Anatomical and morphological- structure. Oxyuris equi – Nematodes are whitish-yellowish in color, the mouth opening is hexagonal in shape, opens into a short oral capsule (pharynx), surrounded by six lips, the esophagus expands in the back, forming a bulbus, inside which there are chitinous clusters that serve for mechanical grinding of food. The male reaches 6-15 mm in length, and 0.8-1.0 mm in width. The caudal end is blunt, with a cuticular membrane, supported by several pairs of stalked (or rib-shaped) papillae, There is one thin sharp spicule 0.167-0.223 mm long, there is no rudder.

The female reaches 40160 mm in length, thick, has a relatively short head and a long thin tail, the vulva is located in the anterior, thickened part of the body at a distance of 7-10 mm from the head end. Eggs measuring 0.085-0.099 mm long and 0.040-0.045 mm wide, gray, slightly asymmetrical, covered with a thick, double shell, giving the impression of a lid. By the time the egg is laid, the glare has reached the mature invasive stage.

Biological development of the pathogen. The parasite is a geohelminth, development occurs without the participation of an intermediate host, i.e. in a direct way. The prepatent period of development is 3-4 weeks, and the period of parasitization is at least 6-8 months.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and helminth-ovoscopic studies of scrapings from the perianal folds, the inner side of the root of the tail and from the perineal region of the horse on the formation of helminth eggs.

The method of performing scraping. With a small wooden spatula or match moistened with a 50% glycerin solution in water, scraping is done from the perianal folds, from the inner side of the tail root and from the perineum area of the horse and transferred to a slide in 2-3 cans of glycerin solution, covered with a cover glass and examined under a microscope for the presence of oxyuris eggs. Eggs measuring 0.085-0.099 mm long and 0.040-0.045 mm wide, gray, slightly asymmetrical, covered with a thick, double shell, giving the impression of a lid.

Oxyurosis of horses should be differentiated from metabolic disorders, from hypo- and vitamin deficiency, which manifest themselves in the form of dermatitis and eczema and from a disease of a non-contagious nature.

Treatment. Carbon tetrachloride, piperazir, piavetrin, panacur (fenbendazole), rintal (febantel), 20% tetramisole in the form of granules and 10% mebenvet granulate and other drugs are used in the recommended doses and application schemes.

Prevention. 1. Planned deworming of all horses, regardless of age, 3-4 times with an interval of 1-1.5 months;

1. Biothermal disinfection of manure;
2. Full feeding of animals according to the diet.

Intestinal strongylatosis of horses is a complex of helminthic diseases caused by representatives of the Strongyla suborder, families: Strongyliidae and Trichonematidae parasitizing in the mature stage in the large intestine of animals, larval stages are localized in various tissues and organs depending on the type of pathogen. Currently, there are about 50 species of nematodes belonging to different genera of the two families. Of the Strongyliidae family, the most important species are Strongylus (Delafondia) vulgaris, Strongylus (Alfortia) edentatus, Strongylus (Strongylus) equinus, and of the Trichonematidae family, the only genus is Trichonema, but there are a lot of species.

Systematics of pathogens. According to the taxonomy, the pathogens of intestinal strongylatoses of loashadei are arranged in the following order: type – Nemathelminthes, class - Nematoda, suborder Strongylata, families Strongyliidae and Trichonematidae, genus: Strongylus and Trichonema and species Strongylus (Delafondia) vulgaris, Strongylus (Alfortia) edentatus, Strongylus (Strongylus) equinus, and from the family Trichonematidae is the only genus -Trichonema.

Characteristics of the exciter. Strongylus (Delafondia) vulgaris is a nematode of light yellow color, its head end is equipped with a powerfully pronounced oral capsule, inside of which there is a dorsal groove, on the basis of which two ear–shaped teeth are located. The male is 14-16 mm long, 0.7-0.95 mm wide, two equal spicules reach 2.1 mm in length, males have a well-developed cuticular genital bursa in the back of the body, which is equipped with nerve outgrowths. The female is 20-24 mm long and 1.25-1.5 mm wide. The vulva opens in front of the anal opening, which is located subterminally. Eggs are oval in shape, 0.070 mm long and 0.040 mm wide, strongylid type. Mature forms of strongylids are localized in the colon, and larvae are localized in the arteries of the intestinal wall and root.

Biology of the pathogen. The parasite is a geohelminth, develops without the participation of an intermediate host. Fertilized females secrete a large number of eggs in the intestines of animals, which, together with feces, fall on the soil, litter, manure. At a favorable temperature, a larva forms in the egg, which breaks the egg shell, enters the external environment, where it molts twice and develops to an invasive stage. Invasive larvae developing in feces migrate horizontally and vertically in the soil and along plant stems with sufficient humidity (rain, dew).

Horses become infected by ingesting invasive larvae together with grass or water. Invasive larvae of delofondia, which have got into the intestine, shed the thick cover and penetrate into the thickness of the mucous membrane, penetrate further into the intestinal and mesenteric blood arteries. From small ones (capillaries and arterioles) they actively dig against the blood flow into the larger vessels of the anastomotic network of the mesentery root and the aorta with its branches, and here the larvae form blood clots, in which they linger and develop for 5-6 months. After this period, they reach a length of 2 cm, shed, dropping the cuticle, are easily released from the thrombus into the lumen of the blood vessels and with the blood flow, the larvae are again transferred to the branches of the caecum and colon, from where they penetrate into the intestinal wall, where they stay for 3-4 weeks. Fomenting parasitic

nodules the size of a pea. From the nodules, the larvae exit into the intestinal lumen and grow into mature males and females.

The pre–patent development period is 6-7.5 months, and the patent period is more than one year.

Strongylus (Alfortia) edentatus is a dark yellow nematode. Male 23-26 mm, two equal spicules measuring 1.9 mm. Female 32-40 mm . They differ from the previous species, there is no ear-shaped tooth in the oral capsule at the base of the dorsal groove, the larvae of the 3-4 stage are localized in the subserous tissue, under the perietal leaf of the peritoneum, in the nodules of the intestinal wall thickness. In these places, small hematomas are formed and there the larvae develop for 5-6 months, after which they return back up to the mesentery root and descend down to the intestinal wall, between the muscular and mucous layers the larva forms a parasitic nodule in which it is 3-4 weeks, after which it enters the lumen of the large intestine, attaches to the mucous membrane and develops into a sexually mature alfortia. The pre–patent development period is 8-9, sometimes 9.5-10 months, and the patent period is over one year.

Strongylus equinus is a grayish-yellow nematode with a powerful oral capsule with four teeth (two long and narrow and two short, wedge-shaped) at the head end. The male is 25-35 mm and 1.1-1.35 mm wide, two equal spicules up to 3 mm in length. The female is 35-45 mm long and 1.75-2.5 mm wide. The eggs are oval in shape, 0.085 mm long and 0.050 mm wide. The larvae released from the cover penetrate through the intestinal mucosa and migrate between the mesentery leaves into the pancreas, where they develop for 8 months, increasing to 4-4.5 cm, then they return to the large intestine and develop into adult parasites. The prepatent development period is 10 months, the patent period is more than one year.

Trichonematidoses are caused by numerous representatives of the Trichonematidae family belonging to 12 different genera; horses are most often infected with various species of the genus Trichonematidae. Trichonemas are small nematodes from 5-7 mm to 12-16 mm in length, the oral capsule is pronounced, its width exceeds its length. At the base of the capsule, small chitinized lanceolate plates or teeth depart from the esophageal funnel. Males have two equal hair-like spicules. In females, the tail end is straight or dorsally curved, the eggs are oval-shaped, strongylid type, Invasive larvae are embedded in the thickness of the intestinal mucosa, curl there into a ring and nodules of the size from poppy to millet grain are formed around them, there the larvae grow gradually for 1.5-2 months, reaching 5-10 mm in length by this time. Then they tear the wall of the cysts, go out into the lumen of the blind and colon and develop into sexually mature parasites. The pre–patent development period is 3 months, and the patent period is about one year.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and helminthic-coprological studies of animal feces.

The method of helminthoscopy. Investigations are carried out according to the Fulleborn method. The eggs of all intestinal strongylates of horses are oval, with a thin two-layer shell. Crushing balls are visible in freshly extracted eggs.

The method of helmintholavoscopy. To establish an accurate diagnosis, the method of helmintholaroscopy of feces is used, which are kept in a thermostat at a temperature of 24-280 for 7-8 days, in order to obtain invasive larvae of strongylids and trichonematids. Larvae differ in their shape, location and colicity of intestinal cells. In the larvae of delafondia, the intestine consists of 32 cells, in alfortia – of 20 shapeless, weakly expressed cells, in strongilus - of 16 elongated cells, and in trichonemas – of 8 clearly defined triangular cells.

Posthumously on the basis of pathoanatomic changes and the detection of the parasites themselves in the caecum and colon.

Intestinal strongylatoses should be differentiated from parascaridosis, from gastroenteritis, colitis and colic of a non-infectious nature, pancreatitis.

Treatment. Diseases caused by larval stages of pathogens should be treated pallitively, i.e. it should be aimed at restoring collateral circulation. For this purpose, camphor oil is injected subcutaneously at 20-50 g every 2-3 hours, and the animal is wired. As an analgesic and to prevent intestinal rupture, morphine is prescribed subcutaneously at a dose of 0.1-0.4 g per head. Mebenvet granulate can be used, which is administered through the mouth in the form of a bolus individually, once at a dose of 0.15 g / kg or at a dose of 0.12 g / kg twice with an interval of 24 hours.

Prevention. Preventive measures of a general sanitary nature are the same as for paraskaridosis:

1. Planned deworming of all horses, regardless of age, 2 times a year;
2. Do not keep young animals with adult animals;
3. Biothermal disinfection of manure;
4. Full feeding of animals according to the diet.

Security questions:

1. Tell us the taxonomy of pathogens of intestinal strongylatoses of ungulates?
2. Tell us the biological development of pathogens of intestinal strongylatoses of horses, on the example of a specific species?
3. Methods of lifetime diagnosis of intestinal strongylatoses of horses?
4. Postmortem diagnosis of strongylatoses of the digestive organs of ungulates?
5. How to make an accurate generic diagnosis of pathogens of intestinal strongylatoses of horses?
6. How do we treat intestinal strongylatoses of ungulates, using the example of a specific disease?
7. How are preventive measures carried out for intestinal strongylatoses of horses?

TOPIC № 10. STRONGYLATOSES OF THE DIGESTIVE ORGANS OF RUMINANTS

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of strongylatoses of the digestive

tract of ruminants, methods of lifetime and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from cattle, sheep, goats, various volumetric cups, saturated solutions of table salt, magnesia sulfate and saltpeter, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

In the digestive tract of ruminants, a large number of nematode species from the suborder Strongylata, families Strongyliidae (genus Chabertia), Trichostrongylidae (genera - Trichostrongylus, Haemonchus, Marshallagia, Ostertagia, Cooperia, Nematodirus, Mecistocirrus and others), Trichonematidae (genus - Oesophagostomum), parasitize Ancylostomatidae (genus - Bunostomum).

The unification of all these types of nematodes is based on the common localization, development cycles of the helminths themselves, epizootology, pathogenesis and clinical manifestations of the diseases caused by them, as well as treatment and preventive measures, which are approximately the same.

Anatomical and morphological signs of pathogens of intestinal strongylatoses of ruminants are generally similar to those of intestinal strongylates of horses and vary according to the affiliation of individual nematode species to a particular family and genus of the Strongylata suborder.

Intestinal strogilatoses of ruminants belong to geohelminths, that is, they develop in a direct way, without the participation of an intermediate host. Their development in the external environment proceeds in the same way as in intestinal strongylates of horses.

Systematics of pathogens. According to the taxonomy, the pathogens of intestinal strongylatozoa of ruminants are arranged in the following order:6 Type-Nemathelminthes, class- Nematoda, Strongylata, families-Strongyliidae, genus-Chabertia, Chabertia ovina, families – Trichostrongylidae, genera- Trichostrongylus, Haemonchus, Marshallagia, Ostertagia, Cooperia, Nematodirus, Mecistocirrus and others, species: H.contortus, O.ostertagi,Marsnallaia marshalli, Ntmatodirus filicollis and N.spathiger, family Trichonematidae,genus - Oesophagostomum, species: Oesophagostomum radiatum (in cattle), Oe.venulosum, Oe.columbianum (in sheep), Oe.dentatum (in pigs), Ancylostomatidae, genus – Bunostomum, species – Bunostomumtrigocephalum and Bunostomum phlebotomum.

Anatomical and morphological structure of pathogens: Haemonchus contortus are nematodes larger than other trichostrongylids, reddish in color, filamentous in shape. The length of the male is 18-23 mm, the female is 26-35 mm. The head end is a rudimentary oral capsule equipped with a lanceolate tooth. The cervical papillae protrude in the form of spikes, the male's genital bursa is powerful, with an asymmetrically located dorsal lobe. Two equal spicules are short, massive, complex structure, brown in color, the length of which reaches 0.3-0.5 mm. The vulva of the female is located in the back of the body and is covered with a cuticular valve. The eggs are 0,070-0,085x0,041-0,048 mm in size, the so-called strongylid type. Localized in the rennet and the initial part of the small intestine.

Biology of the pathogen. The parasite is a geohelminth, development proceeds according to a strongylid scheme, larvae come out of the egg and molt twice in the external environment for 5-10 days and become invasive. The prepatent period of development is 15-25 days, and the period of parasitization is no less than 6-8 months.

Marsnallaia marshalli is a light yellow nematode. The length of the male is 10-12 mm, the female is 12-20 mm. The head ends with a rudimentary oral capsule, the sexual bursa of the male is powerful, with an asymmetrically located dorsal lobe. Two equal spicules are short, massive, complex structure, brown in color, the length of which reaches 0,20-0,23 mm. The vulva of the female is located in the back of the body. The eggs are 0,070-0,085x0,040-0,045 mm in size, the so-called strongylid type. Localized in the rennet and the initial part of the small intestine.They are localized in the rennet and the small intestine.

Biology of the pathogen. The parasite is a geohelminth, development proceeds according to a strongylid scheme, larvae come out of the egg and molt twice in the external environment for 6-8 days and becomes invasive. The prepatent period of development is 3-4 weeks, and the period of parasitization is no more than one year.

Nematodirus filicollis and Nematodirusspathiger are nematodes with a filiform anterior end, the oral cavity is small, there is one tooth on its dorsal surface. The length of the male is 7,5-15 mm, the width is 0,090-0,130 mm, and the length of the female is 19-21 mm, at the head end the expanded cuticle forms a vesicle, the bursa of the male consists of two wide lateral lobes and a barely noticeable dorsal lobe, the spicules are thin, long, connected at the distal end by a membrane, there is no rudder. The vulva opens in the posterior third of the body. These species differ in the shape of the distal end of the spicules. The eggs of nematodiruses are significantly larger in size than the eggs of other trichostrongilids (0,229-0,272x0,085-0,153 mm) and when examining freshly excreted feces, an eight-cell embryo is found in the eggs). They are localized in the small intestine.

Biology of the pathogen. The geohelminth parasite develops like all trichostrongylids, with the difference that the larva forms inside the egg in 24-28 days before the invasive stage. In the host organism, the larvae penetrate deeply into the intestinal wall, molt there twice, and then exit into the intestinal lumen and develop into sexually mature helminths after 24-26 days (prepatent period). Nematodiruses live in the host body for up to 5 months (patent period).

Chabertia ovina- have a rather thick, whitish–colored body length: the male is 14-18 mm, and the females are 14-26 mm, the head end is obliquely cut and curved to the windward side, the mouth opening opens ventarl, in a large spherical oral capsule there are many small teeth in the form of a crown, the mouth opening is surrounded by a crown of sharp triangular petals, the bursa of the male it is short, as if cut off, spicules are thin, 1,3-1,8 mm in length there is a roll.The vulva of the female is located in the back of the body. Eggs size 0,100-0,120x0,039-0,058 mm).

Biology of the pathogen. Larvae emerge from eggs isolated with feces during the day, which develop to an invasive stage in the external environment for about 5

days. The prepatent development period is 32-60 days, and the patent development period is no more than a year.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and helminthic-coprological studies of animal feces.

The method of helminthoscopy. Investigations are carried out according to the Fulleborn method. The eggs of all intestinal strongylates of ruminants are oval, with a thin two-layer shell. Crushing balls are visible in freshly extracted eggs.

The method of helmintholavoscopy. To establish an accurate diagnosis, the method of helmintholaroscopy of feces is used, which are kept in a thermostat at a temperature of 24-280 for 7-8 days, in order to obtain invasive larvae of strongylid and trichostrongylid. Larvae differ in their shape, location and colicity of intestinal cells. In hemonchus larvae, the intestine consists of 32 cells, in marshallagia – of 20 shapeless, weakly expressed cells, in trichostrongylus - of 16 elongated cells, and in nematodiruses – of 8 clearly defined triangular cells.

Posthumously on the basis of pathoanatomic changes and the detection of the parasites themselves in the rennet and intestines.

Strongiatoses of the digestive tract of ruminants should be differentiated from fascioliasis, paramphistomatosis, orientobilcharciosis, anopolephaliasis, trichocephalosis, colibacteriosis, paratuberculosis paratyphoid, enteritis, gastroenteritis, dyspepsia and other infectious and non-infectious diseases of ruminants.

Treatment. The following anthelmintics are used: phenothiazine (0.5 g / kg), copper sulfate in the form of a 1% aqueous solution, nilverm 0.015 g / kg, fenbendazole (panacur) at a dose of 0.01 g / kg, febantel (rintal) -0.005-0.007 g / kg, mebendazole - 20 mg / kg, albendazole and its analogues – 12-15 mg / kg and a number of other anthelmintics.

Prevention. In farms that are unfavorable for strongylatosis, it is necessary to carry out both pasture prevention (isolated rearing of young animals, the use of stall, stall-camp and stall-walking keeping of young animals) and special measures (preventive deworming and chemoprophylaxis) taking into account local conditions.

From October to May, it is recommended to feed animals with a salt-phenothiazine-copper sulfate mixture (1 part copper sulfate, 10 parts phenothiazine and 89 parts feed salt) for 8 months at a dose of 1,0 per head, and it is also necessary to carry out regular biothermal disinfection of manure and full feeding of animals according to the diet.

Security questions:

1. Tell us the systematics of pathogens of strongylatoses of the digestive organs of ruminants?
2. Tell us the biological development of pathogens of strongylatoses of the digestive organs, on the example of a specific species?
3. Methods of lifetime diagnosis of strongylatoses of ruminants?
4. Postmortem diagnosis of strongylatoses of the digestive organs of ruminants?

5. How to make an accurate generic diagnosis of pathogens of strongylatoses of ruminants?

6. How do we treat strongylatoses of the digestive organs of ruminants, on the example of a specific disease?

7. How do we prevent intestinal strongylatoses of ruminants?

8. How and with what laughter is chemoprophylaxis of strongylatoses of the digestive organs of ruminants carried out?

TOPIC № 11. DICTYOCULOSIS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of strongylatoses of the respiratory organs of animals, methods of lifetime and postmortem diagnostics, their differentiation, treatment, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, fecal samples from cattle, sheep, goats, various volumetric cups, saturated solutions of table salt, magnesia sulfate and saltpeter, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

The respiratory organs of farm animals are parasitized by nematodes of the Strongylata suborder belonging to five families: Dictyocaulidae, Protostrongylidae, Metastrongylidae, Crenosomatidae and Syngamidae.

Dictyocauluses parasitize in the trachea and bronchi of cattle and small cattle, reindeer, camels and horses. Males are characterized by a well-developed rib bursa and short cellular spicules, all representatives are geohelminths.

Protostrongylids are thin nematodes. The sexual bursa in males is greatly reduced or even absent, the spicules are equal, often with flattened extensions at the ends, the vulva in females is located near the anus, parasitic in the alveoli, bronchioles and lung parenchyma in small cattle. Biohelminths, intermediate hosts – terrestrial mollusks.

Metastrongylids – at the head end have two powerful three-lobed lips, the male's bursa is well developed, but with shortened ribs. the vulva in females is located at the posterior end of the body near the anus. They are parasitic in domestic and wild pigs in the bronchi. Biohelminths, intermediate hosts – terrestrial (earthworms).

Crenosomes are characterized by cuticular ring-shaped folds in the anterior part of the body. The male's bursa is well developed. Parasitic in the bronchi and trachea of fur–bearing animals: foxes, Arctic foxes, Ussuri raccoons, sables, minks, martens, etc. Biohelminths, intermediate hosts - terrestrial (earthworms).

Syngamuses in the sexually mature form are constantly in a mated state. The male is several times smaller than the female, the spicules are very short, the vulva of the female is located in the anterior half of the body. They parasitize in the trachea of domestic and wild birds. Geohelminths develop with or without the participation of reservoir hosts.

Dictyocaulesis. Dictyocaulesis of animals is caused by parasitism in the trachea and bronchi of strongylates of the genus Dictyocaulus: in sheep and goats - Dictyocaulus filaria, in cattle - Dictyocaulus viviparus, in reindeer - Dictyocaulus eckerti, in horses and donkeys - Dictyocaulus arnfieldi and in camels - Dictyocaulus cameli.

Dictyocaulosis of sheep and goats is caused by nematodes Dictyocaulus filaria, parasitic in the trachea and bronchi and characterized by impaired respiratory function, the appearance of cough, bronchitis, bronchopneumonia, serous-purulent discharge from the nose, difficulty breathing, the appearance of edema in the interdigital space, underbelly, in the eyelids, lips and less often legs, standing in growth-the development of young and a decrease in the productivity of adult animals and the case.

Systematics of the pathogen. According to the taxonomy, the causative agent of the disease are arranged in the following order: type - Nemathelminthes, class - Nematoda, order Strongylata, family - Dictyocaulidae, genus - Dictyocaulus and species Dictyocaulus filaria and Dictyocaulus viviparus.

Characteristics of the pathogen. Dictyocaulus filaria is a filamentous nematode, whitish in color. The male has a length of 3-8 cm, a width of 0,352-0,464 mm. At the tail end of the bursa, supported by the ribs, there are two equal spicules of yellow color, 0,4-0,6 mm long. The female is 5-15 cm long and 0,528-0,592 mm wide, the opening of the vulva is located near the middle of the body.

Dictyocaulus viviparus is whitish in color, male length is 17-44 mm long, 0,20–0,70 mm. Width, at the tail end of the bursa, supported by ribs and two equal spicules with a length of 0,22-0,27 mm, the female is 23-73 mm long and 0,27-0,67 mm wide, the vulva opening is located near the middle of the body.

Biological development of pathogens. They develop without the participation of an intermediate host, i.e. they are geohelminths. Prepatent period of development: Dictyocaulus filaria - 4 weeks, sometimes this period stretches up to 3-4 months. The patent development period is 1,5-2 years; Dictyocaulus viviparus – respectively 21-30 days, 50-70 days, sometimes months, even years.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and detection of larvae in faeces by the Berman-Orlov method. Dictyocaulus filaria larvae have a size of 0,54-0,55x0,025 mm, there is a button-like thickening at the head end, and the tail end is obtusely rounded; the size of the larvae in Dictyocaulus viviparous is 0,31-0,36x0,016-0,019 mm, the head end is rounded, the tail end is short and pointed, the middle part of the larva is filled with grayish grains or lumps, and the head and tail ends remain light. Larvae can be mobile, but their translational movements are poorly expressed.

Post-mortem diagnosis is carried out by autopsy of animals that have fallen or been killed for diagnostic purposes. Take into account the characteristic changes in the lungs and the presence of dictyocauluses in the lumen of the bronchi and trachea. Young forms localized in the lung tissue are detected by grinding the latter and examining using the Berman-Orlov method.

Dictyoculosis should be differentiated from echinococcosis, bronchopneumonia, pneumonia, bronchitis, pleurisy.

Treatment. For deworming animals use: nilverm, levomizole, tetramizole, mebendazole, fenbendazole (panacur), febantel (rintal), ditrazine citrate, divezide, an aqueous solution of iodine.

Nilverm (levomizole, tetramizole) in the form of 5-10% aqueous solution (for sheep and goats) and 1% (for cattle) or in powder mixed with feed. In emaciated animals with high intensity of invasion, nilverm is prescribed orally or subcutaneously for 2 consecutive days at a dose of 0,01 g / kg twice.

Levomizole is administered subcutaneously once at a dose of 0,01 g / kg; mebendazole is used in the form of mebenvet (10% granulate), given with feed once, at a dose of 0,02 g / kg of ADV weight; fenbendazole in subclinical form is used once through the mouth at a dose of 0,005 g / kg or 0,023 g / kg ADV the paracura of the granulate is 22,2%, with a clinical form of -0,015 and 0,068 g / kg, respectively, the drug is given individually or mixed with loose ends. Feed in a group way; rintal (febantel) is used individually or in a group method in a mixture with concentrated feed in doses of 0,01 g / kg ADV and in the form of 0,1 g / kg granulate; ditrazine citrate is used in the form of a 25% solution that is sterilized in a water bath, a freshly prepared solution is prescribed to sheep and goats 4 ml per 10 kg of weight animal (0.1 g / kg of dry matter), subcutaneously, twice with an interval of one day; divezid is prescribed to sheep in the form of a 33.3% aqueous solution once subcutaneously in doses: for therapeutic purposes – 3 ml / 10 kg of animal weight and for preventive purposes 2 ml per 10 kg of weight.

An aqueous solution of iodine is prepared according to the recipe: crystalline iodine 1,0 g, potassium iodide 1,5 g, distilled water 1500 ml. The freshly prepared solution is injected with a syringe intratracheal. The needle is inserted into the inter-cartilaginous space of the upper third of the trachea. To administer the solution, the sheep is given a dorsal-lateral position with the front part of the body raised at an angle of 300. For therapeutic deworming, the solution is injected first into one lung, then a day later into another, and for preventive purposes – at the same time, first into one lung, then, turning the animal, into another. For lambs of the current year of birth, an aqueous solution of iodine is prescribed 5-8 ml, for young animals from one to two years – 10 ml, for adult sheep, older than two years – 15-20 ml in each lung.

Prevention. Carry out a set of measures aimed at destroying the invasive principle in the host body, as well as in the external environment. The complex of preventive measures includes therapeutic and preventive deworming, pasture and chemical profiling and measures of a general sanitary order aimed at strengthening the animal's body.

The chemoprophylaxis of dictyoculosis gives good results by daily free feeding during the pasture season of a mixture of salt-phenothiazine-copper sulfate mixture (1 part copper sulfate, 10 parts phenothiazine and 89 parts feed salt) at a dose of 1,0 g per head, and it is also necessary to carry out regular biothermal disinfection of manure and full feeding of animals according to the diet.

Security questions:

1. Tell us the systematics of pathogens of strongylatosis of the respiratory organs of animals?

2. Tell us the anatomical and morphological structure of Dictyocaulus filaria and its differences from Dictyocaulus viviparous?

3. Tell us the biological development of pathogens of strongylatosis of the respiratory organs, using the example of a specific species?

4. Methods of lifetime diagnosis of dictyoculosis of sheep and cattle?

5. Postmortem diagnosis of dictyoculosis of sheep and cattle?

6. How to make an accurate species diagnosis of pathogens of strongylatosis of the respiratory organs of animals?

7. How do we treat dictyoculosis of sheep and cattle?

8. How do we prevent dictyoculosis of sheep and cattle?

9. How and with what laughter is chemoprophylaxis of dictyoculosis of animals carried out?

TOPIC № 12. TELYAZIOSIS OF CATTLE AND TRICHINOSIS OF PIGS.

Lecture plan:

1. Telyaziosis of cattle

2. Pig trichinosis

Keywords: Spirurata, Thelaziidae, Thelazia, Th. rhodesi, Th. gulosa, Th. skrjabini, Trichocephalata, Trichonellidae, Trichinella, Trichinellaspiralis, geohelminth, biohelminth, prepatent, patent period of development, eggs, viviparous, larva, deworming.

Telyazioses are helminthic diseases of cattle, manifested by conjunctivitis-keratitis. These diseases are caused by three species of nematodes of the genus Thelazia of the family Thelaziidae from the suborder Spirurata

Th. rhodezi, Th. gulosa, Th. Skrjabini localized in the conjunctival ieshka and under the third eyelid; Th. Gulosa and Th. Skrjabini – in the ducts of the lacrimal gland and the lacrimal nasal canal.

Telazia also parasitize horses – Thelazia lacrimalis, pigs - Thalazia erschovi, dogs - Thalazia callipaeda.

Pathogens are small nematodes up to 21 mm long. Rhodezi is a body surface with rough transverse striation, which gives the parasite a jagged appearance. The mouth opening leads to a small oral capsule. Males have two unequal spicules: the right one is 0,11 mm, the left one is 0,624-0,846 mm long. Th. Gulosa without rough transverse striation of the cuticle, but differs in the presence of a wide oral capsule, the male spicules are unequal left 0,129-0,165 mm, right – 0,608 - 0,692 mm in length. Th. Skrjabini is also devoid of rough transverse striation of the cuticle, the oral capsule is very small. Males have almost equal spicules: right -0,082 mm, left - 0,113-0,185 mm in length.

Biology of pathogens. The biohelmitt parasite develops with the participation of an intermediate host, which are various species of flies: Muscaautumnalis,M. convexifrons and M. amis.

Fertilized female telyazii spawn a large number of live mobile larvae that fall into the lacrimal outflow and are swallowed by flies. Larvae develop in the body of flies to the invasive stage within about 3-4 weeks. Invasive larvae enter the abdominal cavity of the fly and move into its proboscis. When the fly is near the eye, the larvae independently crawl out of its proboscis and penetrate into the conjunctival sac, where after 15-20 days they grow into sexually mature females and males. Telyazia live in the eye for several months, and individuals live for more than a year.

Epizootological data. Telaziosis enzooties among cattle are observed in summer, in June-August. Animals become infected by direct contact with intermediate hosts in the pasture or ward. Telyazii can be found in the eye of animals at any time of the year, but the greatest number of them are observed in summer. Therefore, telyaziosis is a seasonal disease. It is found everywhere.

The pathogenesis is insufficiently studied. Telyazii have a mechanical effect on the conjunctiva and cornea, which is accompanied by the introduction of banal microflora and the development of conjunctivitis of a serous or purulent nature. The damaged cornea becomes cloudy, and the inflamed conjunctiva is so much lowered that the eyelids completely cover the diseased eye. Erosions form on the cornea. There may be corneal perforation, lens damage and the development of fibrinous hemorrhagic iridocyclitis.

With the development of inflammatory processes, a rounded or oval ulcer forms in the clouded cornea, the eyeball protrudes strongly. Over time, the cornea gradually heals, diffuse opacity resolves, the eyes acquire a normal appearance, a white spot of various sizes remains in place of the former ulcer.

Immunity in sick animals does not develop.

Symptoms of the disease. The most characteristic signs: lacrimation, photophobia. Redness and swelling of the conjunctiva, edema of the eye, in advanced cases – keratitis, ulcers on the cornea, an eyesore. The disease usually lasts 1-2 months, especially it occurs in young animals up to 4 months and older. In addition, the animals worry, shake their heads, they note a weakening of appetite and a decrease in milk yields.

The degree of clinical manifestation does not always correspond to AI, since in some cases, in the presence of a large number of parasites, there are no symptoms of the disease.

Pathoanatomic changes are characterized by the presence of conjunctivitis, keratitis, opacification and ulceration of the cornea, damage to the lens.

The diagnosis is made on the basis of clinical signs and studies of flushes from conjunctival cavities. To identify sick animals in the summer, a monthly clinical examination of cattle is performed.

Treatment. It is recommended to wash the eye cavity with one of the following means: 2-3% boric acid solution, 3% lysol emulsion, 3% ichthyol emulsion, ditrazine citrate can be applied at a dose of 0.015 g / kg, subcutaneously

twice with an interval of 1 day or in the periorbital also with an aqueous solution of iodine (crystalline iodine –th g, potassium iodide – 2 g and boiled water – 2 liters).

In case of complication of telyaziosis with a second infection (purulent conjunctivitis, keratitis) prescribe symptomatic treatment (penicillin, sulfonamide preparations, eye ointments and eye medicinal films (GLP).

Prevention. Preventive deworming of the entire livestock is carried out during the period of stable maintenance or in the spring before the beginning of summer of cow flies, repeated every 7-10 days. In summer, during the hot time of the day, during the period of the greatest activity of flies, it is recommended to keep animals indoors or under a canopy. Night grazing of animals, biothermal treatment of manure, full feeding of animals according to the diet.

TRICHINELLOSIS, Trichinellosis is an acute and chronically occurring anthropozoic, naturally focal, invasive disease with pronounced allergic phenomena caused by nematodes from the family Trichonellidae of the suborder Trichocephalata, The genus Trichinella species Trichinella spiralis (pork trichinella), Trichinella native (trichinella from predatory animals of Eurasia,Trichinella nelsoni (from predatory Africa), Trichinella pseudospiralis (from the striped raccoon from Dagestan). Trichinella pseudospiralis differs from the previous three by its smaller body size and the fact that its larvae are not encapsulated in the muscles, in addition, Trichinella pseudospiralis completely completes the development in the body of birds (chickens, ducks, partridges, sparrows, etc.).

Adult trichinella parasitize in the small intestine of animals and humans, and larvae – in the striated muscles of these the same organisms. To date, more than 100 species of mammals that are hosts of trichinella have been registered.

The most common trichinosis occurs in pigs, dogs, wolves, foxes, cats, bears, rats, mice. Wild boars, badgers, arctic foxes, minks, sables, ferrets, hedgehogs, tigers and many wild carnivores and rodents are susceptible to it. Helminthiasis has been recorded in marine mammals: whales, walruses, seals.

Economic damage. The economic damage from trichinosis is very great: trichinosis carcasses of animals, regardless of the degree of infection, are destroyed. This helminthiasis is also very dangerous for humans: trichinosis in humans is very difficult, difficult to treat and very often ends in death.

The pathogen. These are very small nematodes. The male is 1,4-1,6 mm long, 0,14 mm wide. Two pairs of papillae are located at the posterior end of the body and in the gap between the two lobes behind the cloaca. Females are twice as large as males -3-4 mm, viviparous

Biology of the pathogen. The parasite is a biohelminth.The same animal is initially a definitive, and then an intermediate host of the parasite. Animals become infected by eating trichinella meat, which contains live encapsulated larvae of trichinella. In the stomach, the capsule is destroyed, a larva about 1 mm long comes out of it.

These larvae develop rapidly in the small intestine until puberty. Mature eggs in ma in samou appear already 44 hours after infection.

Female trichinella are introduced with their head end into the mucosa, and on the 4th day after infection, live larvae hatch. One uterus spawns up to 2,100 larvae 1,2 mm long and 0,006 mm wide. There is a stiletto on the head end. The larvae penetrate into the lymphatic, then into the circulatory system and are carried throughout the body by the blood current. They linger in striated muscles, penetrate under the sarcolemma of muscle fibers, grow, then curl into a spiral. Larvae become invasive 17,5 days after infection. From the 4th to the 12th week, capsules are formed around the larvae, which after 6 months in pigs begin to become deified; this process ends completely after 15-16 months.

In the muscles, encapsulated trichinella larvae can remain viable for 25 years. Female trichinella in the intestines of the host lag behind for up to 8 weeks, after which they die.

Epizootological data. Trichinosis is common everywhere. Trichinosis of pigs is of great practical and health importance. Pigs become infected by eating the corpses of infected rats, cats, wild animals, as well as raw or poorly cooked slaughter confiscated and kitchen waste. A person is infected with trichinosis by eating pork, bear meat or other meat with trichinella larvae.

Pathogenesis. Trichinella have mechanical and toxic effects, as well as sensitization of the body with pronounced allergic phenomena.

Immunity. The formation of immunity in trichinosis is very difficult and depends on a number of hormonal and cellular factors.

Symptoms of the disease. In pigs, trichinosis occurs without pronounced clinical signs. Note a short-term increase in body temperature, depression, muscle soreness, intermittent lameness. More serious changes occur in the blood: pronounced eosinophilia and an increase in ESR.

Pathoanatomic changes. The normal structure of muscle fibers is disrupted: at the locations of capsules with larvae, the fibers expand, acquire a fusiform appearance, their transverse striation gradually disappears, the nuclei increase and the muscle fibers disintegrate, turning into a granular mass. With high AI, small, whitish-colored seals with poppy seeds are noticeable in skeletal muscles, representing capsules with trichinella larvae.

Trichinella are localized mainly in the legs of the diaphragm, in the muscles of the tongue, esophagus, larynx, intercostal, thoracic, in rare cases they are found in parenchymal organs, in the muscle layers of lard.

Diagnosis. Lifetime diagnosis of trichinosis can be carried out by immunobiological methods: serological reaction of indirect immunofluorescence and reaction of ring precipitation in the capillary.

Postmortem diagnosis is performed by trichinelloscopy. After a copressor study of pork meat samples (24 slices from the legs of a diaphragm the size of a wheat grain), it is used as the most accurate method of digesting muscle samples in artificial gastric juice, which makes it possible to detect trichinella with weak invasion, which are not always detected by conventional trichinelloscopy.

In the differential diagnosis of trichinella, it should be distinguished from cysticerci and Mischer sacs, which are the simplest parasites – sarcocysts, sarcocysts

are not enclosed in capsules, but have their own thin shell. The shape of the Mishera pouches is very diverse: from elongated, spindle-shaped, sickle-shaped to oval. Their sizes range from 0.005 to 4-5 mm in length. Capsules of trichinella have a lemon-shaped shape, reach a length of 0.68 mm and a width of 0.37 mm; one, rarely two spirally coiled trichinella are visible inside.

When the capsules are desaturated, the muscle sections are enlightened with 5-10% hydrochloric acid at an exposure of 1-2 hours, followed by glycerin.

Animal treatment has not been developed. In humans, thiabendazole and other benzimidazoles are used.

Prevention. To prevent infection of people with trichinosis, all carcasses of pigs, wild boars, bears, and nutria, without exception, are necessarily examined for this helminthiasis.

At any intensity of neighing, trichinosis of animal carcasses is subject only to technical disposal, they are strictly forbidden to be used for food. By-products having muscle tissue are also disposed of. The fat is peretapvilayut at 1000 C for 20 minutes. Internal fat is sold without restrictions.

If a trichinella carcass is found, the farm from which the animal came is informed about it in order to take appropriate preventive measures. On animal farms, it is forbidden to feed carcasses of animals, dogs, cats without first examining them for trichinosis and disinfection, the corpses of rats are burned. It is not necessary to feed pigs and fur-bearing animals with slaughterhouse waste without thorough decontamination.

Strict deratization measures at pig farms, slaughterhouses, meat processing plants prevent the spread of trichinosis. For the destruction of rodents, zookumarin and other means are used. It is necessary to combat the vagrancy of pigs on the territory of settlements.

Any dysfunctional point for trichinosis is considered as a natural focus of the disease, a complex of veterinary-saninary and medical preventive measures is carried out in it.

TOPIC № 13. PYROPLASMOSIS, FRANCAIELLOSIS AND BABESIOSIS ANIMALS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of piroplasmidosis of animals, methods of in vivo – laboratory (taking a drop of blood, preparation of smears, fixation and staining, their examination under a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Piroplasmosis of cattle is an acute and subacute, transmissible, seasonal, protozoal disease caused by unicellular parasites of the species Piroplasma bigeminum, parasitizing in the erythrocytes of the blood of cattle and is characterized by an increase in body temperature to $41,5^0$ C, depression, violation of the digestive, respiratory and cardiovascular system, the appearance of hemoglobinuria on the 2nd-3rd day of illness, severe emaciation and loss of productivity and death of animals.

Systematics of the causative agent of pyroplasmosis. Pyroplasmas belong to the sub-kingdom - Protozoa, type-Apicomplexa, order - Piroplasmida, family - Babesiidae and genus – Piroplasma. In cattle, Piroplasma bigeminum parasitizes, in sheep - P.ovis, in horses - P.caballi, in dogs - P.canis, in pigs - P.trautmanni.

Morphological structures of Piroplasma bigeminum. Pyroplasmas are parasites that live in red blood cells. They distinguish between the nucleus and cytoplasm. When stained according to Romanovsky, the nucleus turns pink-ruby color, the cytoplasm – bluish. The shape of the pyroplasmas is polymorphic, i.e. ring-shaped, oval, amoeboid, pear-shaped and paired, the size of which is from 1,5-3 microns (rounded) to 3,5-5,2 microns (paired-pear-shaped), single pear-shaped -1,7-3,5 microns. Pyroplasmas in the amount of 1-2 (rarely 3-4) are located in the center of the erythrocyte. The percentage of infection of red blood cells averages 10-15% and very rarely reaches 40%.

Biology of the pathogen. Carriers of the pathogen are single-host mites Boophylus calcaratus, two–host mites - Rhipicephalus bursa and three-host mites - Haemaphysalis punctata and H.rossicus. The disease season coincides with the period of tick attacks on animals, and the first outbreak of the disease appears in April-May –June, and the second outbreak – in August-September, sometimes in October-November, depending on weather conditions.

Pyroplasmas are transmitted transovarially by ticks, so larvae and nymphs infect animals. If pyroplasmas are transmitted by Boophylus calcaratus ticks, then infection of animals occurs through nymphs, and if pyroplasmas are transmitted by Rhipicephalus bursa and Haemaphysalis punctata ticks, then infection of animals occurs through imago (adult) ticks.

Francaiellosis is a rapidly acute, transmissive, seasonal, protozoal disease caused by unicellular parasites of the species Francaiella colchica (Babesia colchica), parasitizing in red blood cells of cattle and characterized by an increase in body temperature to 41.8^0 C, depression, violation of the digestive, respiratory and cardiovascular system, jaundice of the visible mucous membranes, the appearance of hemoglobinuria on the 4th-5th day of the disease, severe emaciation and loss of productivity and death of animals.

Systematics of the causative agent of francaiellosis. Francaiella belong to the sub-kingdom - Protozoa, type-Apicomplexa, order - Piroplasmida, family – Babesiidae and genus - Francaiella and species - Francaiella colchica (Babesia colchica).

Morphological structures of Francaiella colchica. Fransaiella, colored according to Romanovsky, has a bluish cytoplasm, and the nuclei are reddish in color. In erythrocytes, they are most often located in the center of one or two, rarely

more. Their shape is rounded and pear-shaped, the size of the parasites is up to 2,8 microns. Paired francaielles are connected to each other by sharp ends at an obtuse angle in the form of "points"what is characteristic of this pathogen. Single parasites in red blood cells are more common, their number in the peripheral blood at the beginning of the disease is very small, and in the middle and severe course of the disease they affect up to 4-5% of red blood cells. The accumulation of Francaiella is also found in the corpse – in the capillaries of the brain, adrenal glands and other organs, therefore Francaiella colchica multiplies in the internal organs.

Biology of the pathogen. Fransaiella differ somewhat from piroplasmas, in particular, their development in the tick proceeds differently than piroplasmas, since their invasion of animals occurs 3,5 days after the beginning of suckling of Boophylus calcaratus larvae. Therefore, the incubation period for francaiellosis is shorter. Non-sterile immunity against francaiella in cattle, the field of active disease is more resistant, even against pyroplasmas and lasts about a year.

The carrier of the pathogen is single-host mites of the species Boophylus calcaratus, the pathogen is transmitted by the nymphs of the tick, then the infection of animals occurs through nymphs, therefore, mixed invasion is more often observed in cattle than pure.

Babesiosis is an acute, transmissible, seasonal, protozoal disease caused by unicellular parasites of the species Babesia bvis, parasitizing in red blood cells of cattle and characterized by an increase in body temperature to 42^0C, depression, violation of the digestive, respiratory and cardiovascular system, anemia and jaundice of the mucous membranes, the appearance of hemoglobinuria on the 3rd-4th day of illness, severe emaciation and loss of productivity and death of animals.

Systematics of the causative agent of babesiosis. Babesias belong to the sub-kingdom - Protozoa, type-Apicomplexa, order - Piroplasmida, family - Babesiidae and genus Babesia, and the causative agent - Babesia bovis

Morphological structures of Babesia bovis. Babesias are localized inside erythrocytes, merozoites have lanceolate and pear–shaped forms, and trophozoites are rounded. Diagnostic forms are considered paired pear-shaped, connected by thin ends, located at an obtuse angle along the periphery of the erythrocyte. Their size is 1,0-2,4 x 0,5-1,5 microns. In smears, single ring-shaped parasites are more common, sometimes in the form of a trefoil, the incidence of erythrocytes reaches 7-15%, rarely 40%.

Biology of the pathogen. In the body of cattle, babesias multiply in erythrocytes by simple division into two or budding, forming three individuals. Sometimes babesia can be seen outside of red blood cells. Their development in the body of ticks is not enough. Babesia bovis is transmitted by the three-host mites Ixodes ricinus Ixodes perculcatus. Ticks, feeding on a large or sick animal, perceive babesias, which first multiply in the intestine by simple division, then form cells with 2-4-6 nuclei, club-shaped (perhaps they are the result of the merger of two individuals). Such babesias penetrate into the intestinal epithelium and multiply, forming multinucleated cells that disintegrate and divide several more times. After that, the club-shaped cells penetrate into the hemolymph and migrate to various

organs and tissues of the tick, including egg follicles, where they still multiply. After leaving the eggs of the larval ticks and their subsequent transformation into nymphs and imagos, babesias migrate in their body and enter the salivary glands. Nymphs and imago Ixodes, attacking an animal, introduce the pathogen with saliva into the blood (transovarial transmission of the pathogen).

Diagnosis and differential diagnosis. The diagnosis during the life of the animal is made on the basis of epizootological data, clinical signs and on the basis of microscopic studies. The material for microscopic examination is a thin smear of blood stained according to Romanovsky. The characteristic form of pathogens for diagnosis is considered to be: for piroplasmas, a paired pear-shaped, connected by narrow ends at acute angles, located in the center of red blood cells, the size of which is greater than the radius of red blood cells; for fransaiella, rhapsodies located in the center of the erythrocyte, which are connected at an obtuse angle and fill the frame of "glasses", the size of which is equal to the radius of the erythrocyte; for babesia, paired pear-shaped forms, in which the angle of divergence is obtuse, they are often located on the periphery of the erythrocyte, as if sitting "astride" the erythrocyte.

Postmortem diagnosis of bovine piroplasmidoses is based on pathogenoanatomic changes. A characteristic pathologic-anatomical change in pyroplasmosis is the state of the book, in which a solid dry mass is found, which crumbles.

Piroplasmidoses should be differentiated from teileriosis, anaplasmosis, leptospirosis, anthrax and mechanical hepatitis.

Treatment. Sick animals are not driven out to pasture, they are provided with clean water, easily digestible, juicy feeds, it is desirable to add milk whey or fresh milk to the diet. After that, he proceeds to symptomatic and specific treatment. Azidine (berenyl), babenil, norotrip, DAT, dimizone and others are used in specific chemotherapy drugs with mixed invasion (piroplasmosis and francaiellosis) at a dose of 3.5 mg / kg of live weight in the form of a 7% solution administered intramuscularly or subcutaneously. Diamidine (dimizole) in a dose of 1-2 mg / kg of animal weight in the form of a 4% solution, intramuscularly or subcutaneously. Sulfantrol at a dose of 30-40 mg / kg in the form of a 1% alcohol (330) solution is administered intravenously or in the form of a 10% aqueous solution is administered intramuscularly. Polyamidine in a dose of 5 ml / 100 kg of live weight intramuscularly or subcutaneously.

Prevention. Scheduled mitigating chemoprophylaxis is carried out (for prolongation) with azidine, diamidine or polyamidine: if azidine is carried out every 12-14 days, diamidine - every 20-22 days, and polyamidine -30-35 days, as well as the fight against vector ticks, i.e., bathing animals every 7-10 days during the period of active attack vector ticks (from March to September months) acaricidal drugs.

Security questions:

1. Give a description of the pyroplasmosis of cattle?
2. How is the causative agent of piroplasmosis of bovine cattle located according to the taxonomy?

3. Tell us the morphology of Piroplasma bigeminum, its biological development in the body of animals and vector ticks?

4. Tell us the laboratory methods of lifetime diagnosis of piroplasmidosis?

5. How do we distinguish Piroplasma bigeminum from other types of piroplasmids?

6. In our Republic, there is babesiosis of cattle, if not, then why, explain?

7. How do we treat piroplasmidosis in cattle?

8. The main pathoanatomic changes in piroplasmosis?

9. How do we fight against piroplasmidosis of cattle?

10. Tell us the essence of the mitigating chemoprophylaxis of pyroplasmidoses of animals?

TOPIC № 14. TEILERIOSIS OF CATTLE.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of the causative agents of bovine taileriosis and their biological development, methods of in vivo –laboratory (taking a drop of blood from peripheral vessels and punctate from lymph nodes, preparation of smears, fixation and staining of their dissection under a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Bovine taileriosis is an acute and subacute, transmissible, seasonal, protozoal disease, caused by pigmented unicellular protozoa, parasitic in the reticular-endothelial systems and in the erythrocytes of animal blood and is characterized by depression, unilateral enlargement of the superficial lymph nodes, high fever (up to 420 and higher), anemia, cardiac dysfunction-vascular and digestive systems, severe exhaustion and a high percentage of mortality.

Taxonomy of the causative agents of teileriosis. The causative agents of teileriosis belong to the sub-kingdom - Protozoa, type-Apicomplexa, order – Piroplasmida, family - Theileriidae, genus - Theileria, species: Theileria annulata, Theileria sergenti, Theileria mutans and Theileria orientalis.

Morphology of Theileria annulata. The morphology of the pathogen depends on the stage of development. Sporozoite, which has entered the animal's body with tick saliva, multiplies in the lymph nodes and forms macro and microschizonts, their size ranges from 8 to 30 microns. In the stained smears, the cytoplasm of schizonts turns blue, and the nucleus turns dark ruby. Microschizont breaks down into micromerozoites, which are embedded in red blood cells, they are called erythrocyte forms. In erythrocytes, taileria appear on the 2nd-3rd day, sometimes later, after the temperature rises. They have rounded, oval, rod-shaped, cruciform and anaplasmotid shapes. The size of the rounded forms is 0,5-1,5 microns, oval 0,6-2,0, rod-shaped -

0,7-2,5 microns, comma–shaped – 0,6 and anaplasmoid 0,6 microns. In stained smears, the cytoplasm of the taileria is also colored blue, and the nucleus is dark ruby. There may be 1-7 in one erythrocyte, but more often 2-3 taileria. Infection of erythrocytes reaches 80-95%.

Theileria sergenti. The morphology of Theileria sergenti differs from Theileria annulata. Garnet bodies are somewhat smaller, and their cytoplasm is paler according to Raman, the number of nuclei in schizonts is greater. Erythrocytic forms of taileria are larger and have several clusters of chromatin. Elongated forms predominate over rounded ones, the size of comma–shaped and rod-shaped 1--4 microns, pear-shaped – 2,7-3,1 microns, oval and round -1,4-1,7 microns, dot-shaped – 0,5, cruciform -0,8-1.5 microns, localization is central, the incidence of erythrocytes reaches 40-50%.

Biology of pathogens. Taileria develop in the body of warm-blooded animals and in carrier ticks.

Ticks, sucking on the body of animals, inoculate small mononuclear sporozoites, which penetrate into lymphocytes, and with them - into the adjacent (regional) lymph node. In lymphatic cells and outside, sporozoites multiply by schizogony, resulting in the formation of multinucleated schizonts (garnet bodies or Koch balls). At the beginning of schizogony, schizonts are formed with large, irregularly shaped nuclei – macrochizonts, which break up into individual macromerozoites and are introduced into healthy lymphoid cells. The process of schizogony repeats several times, the last time they divide and form microschizonts that differ from macrochizonts in the structure of the nucleus. Microschizonts have small, round, regular-shaped nuclei. In microschizonts, merozoites are formed by budding, which are embedded in red blood cells, in which they multiply by simple division.

Ticks, sticking to the body of a sick or sick animal, swallow red blood cells together with parasites. Merozoites, once in the body of a tick larva or nymph, develop in the intestines, hemolymph and salivary glands. Currently, there is evidence of sexual reproduction of taileria in the tick body (Diakonov and Schein with the authors).

Diagnosis and differential diagnosis. The diagnosis during the life of the animal is made on the basis of epizootological data, clinical signs and on the basis of microscopic and serological studies.

Microscopic studies. For early diagnosis of the disease, a punctate is taken from enlarged lymph nodes, a thin smear is prepared and stained according to Romanovsky and examined under the immersion system of a microscope and garnet bodies are searched for, and during the period of the disease, taileria is found in a thin smear of blood from the capillaries of the auricle. In erythrocytes, small round or other forms of parasites are found, in which the nucleus and a small rim of the cytoplasm are clearly visible.

Serological studies. Serological studies are based on the renaction of complement binding with a specific antigan. Early diagnosis is carried out with an antigen from pomegranate bodies, and later – with an antigen from erythrocyte forms.

Postmortem diagnosis of bovine taileriosis is based on pathogenoanatomic changes. A characteristic pathological and anatomical change in teileriosis is the condition of rennet, the mucous membrane that is hyperemic and ulcerated, ulcers with smooth edges, 2-10 mm in size, are scattered on the surface of the mucous membrane, as well as many hemorrhages, nodules ranging in size from millet grain to large pea.

Differential diagnosis. Teileriosis should be differentiated from pyroplasmosis, francaiellosis, babesiosis, anaplasmosis, anthrax, leptospirosis.

Treatment. Sick animals are not driven out to pasture, they are provided with clean water, easily digestible feeds are given: green grass, tops of garden crops, melons, crushed root crops, bran or compound feed, it is desirable to add milk whey, fresh yogurt or curdled milk to the diet. After that, they begin specific treatment. Several treatment regimens have been developed, proposed by the staff of scientists of UzNIIV. KazNIVI, TajNIVI, All-Russian Institute of Experimental Veterinary Medicine.

Hakberdiev P.S. developed a 3-day course of treatment for teileriosis and put it into practice, the effectiveness of which is 98-100%. The scheme of application of the 3-day course of treatment is as follows:

Morning procedure: azidine (berenyl) or other specific drugs are administered intramuscularly at the recommended dose (3,5 mg per kg of live weight in the form of a 7% aqueous solution). After that, caffeine sodium benzoate, vitamin B_6 and B_{12}, ascarbic acid (vitamin C), antibiotics from the tetracycline group are introduced. After 30 minutes, polyamidine (etdin) is injected intramuscularly or subcutaneously at the recommended dose.

Evening procedure: Polyamidine (etdin) is first injected. After that, caffeine sodium benzoate, vitamin B_1 and B_{12}, ascarbic acid (vitamin C), antibiotics from the tetracycline group are introduced. After 30 minutes, azidine (berenyl) or other specific drugs are injected intramuscularly or subcutaneously at the recommended dose (3,5 mg per kg of live weight in the form of a 7% aqueous solution).

On the 2nd and 3rd day, the above procedures are repeated. This 3-day course of treatment has been put into practice over 500 heads of cattle of the Samarkand region.

Recently, the following drugs have been recommended for the treatment of bovine taileriosis: Butach (telex, telemax, buparvalek, buparvacon) at a dose of 1 ml per 20 kg of live weight, once, administered intramuscularly. After 72 hours, the drug can be repeated, at the same dose, if the body temperature of the animal is not normalized.

Prevention presupposes an increase in the resistance of animals by improving feeding and maintenance, as well as the fight against vector ticks. For this, the premises are treated both inside and outside, once every 1.5-2 months, and the animals are treated every 7-10 days with acaricidal drugs, i.e. during an active attack of vector ticks from the second half of March to September months.

In the autumn-winter period (2-3 months before the appearance of vector ticks on the pasture), susceptible young animals are vaccinated with a vaccine prepared

from schizont taileria grown in the culture of lymphatic cells of organs and tissues of cattle. The vaccine is administered subcutaneously at a dose of 1 ml, regardless of the weight of the animal. In vaccinated animals, body temperature may drop by 0,5-2^{0}C after 14-22 days and the regional lymph node may increase. Immunity in calves occurs on the 30-35 th day and persists for life if they are attacked annually by invasive ticks.

In addition, planned mitigating chemoprophylaxis is carried out (for prolongation) with azidine, diamidine or polyamidine: if carried out with azidine every 12-14 days, diamidine - every 20-22 days, and polyamidine -30-35 days.

Security questions:

1. Give a characteristic of bovine teileriosis?
2. How are the pathogens of bovine teileriosis located according to the taxonomy?
3. Which pathogens of teileriosis are very pathogenic for animals?
4. Tell us the morphology of Theileria annulata and its biological development in the body of animals and vector ticks?
5. Tell us the morphology of Theileria sergenti and its biological development in the body of animals and vector ticks?
6. Tell us laboratory methods of lifetime diagnosis of teileriosis?
7. The main pathoanatomical changes in teileriosis?
8. Tell us the atypical form or course of teileriosis and how is the diagnosis made in this course of the disease?
9. How do we treat cattle teileriosis?
10. Tell us the 3-day course of treatment for teileriosis?
11. How do we fight with bovine taileriosis?
12. Tell us the essence of vaccination of calves against teileriosis?

TOPIC № 15. EIMERIOSIS OF RABBITS.

Aimeriosis of rabbits. Rabbits get sick with eimeriosis mainly in the period from weaning to 4-5 months of age, more adults are less likely to get sick. The disease is characterized by anemia, sometimes jaundice of the mucous membranes, rapid emaciation, an increase in abdominal volume, sometimes diarrhea, convulsions and mass death of animals.

Pathogens. Rabbits are parasitized by 8 species of eimeria, of which one species of E.stiedae is localized in the epithelium of the bile ducts, the rest – in the small intestine and only E.perforans - in the large intestine. Three types are more common:

E.stiedae - oval oocysts, yellowish-brown in color, at the narrow end there is a noticeable micropile, in the mature oocyst and sporocyst there are residual bodies, size 30-40x16-25 microns, sporulation 3-4 days.

E.perforans – oval or cylindrical oocysts, colorless, micropiles are noticeable in large forms, residual bodies in oocysts and sporocysts, size 20-25x12-15 microns, sporulation 1-2 days.

E.magna - oval-shaped oocysts, brownish with a pronounced micropile, around which there is a thickening, size 32-37x21-25 microns, residual bodies in the oocyst and sporocysts, sporulation 3-5 days.

Epizootological data. Eimeriosis is widespread, infection rate is 70-100%. Baby rabbits are infected from the first days of life, but while they receive their mother's milk, clinical signs are almost not manifested. AI is affected by crowding, dampness in cages and rooms, poor quality of feed, colds, recruitment of groups of different ages, as well as the degree of infection with uterine eimeria. The more intensely the females are infected, the more rabbits die from eimeriosis.

Eimeria oocysts can be spread by rodents, birds, maintenance personnel on shoes, clothing, and care items. Rabbits get sick during the warm season. But if the rooms where the cages are insulated, then the disease appears in winter.

Immunity. Recovered rabbits acquire non-sterile immunity, which, with constant reinvasion by oocysts, persists for life. Immunity is specific, that is, antibodies are produced against those types of eimeria with which the rabbit was infected.

Symptoms of the disease. The incubation period is 4-12 days. According to the localization of pathogens, intestinal, hepatic and mixed forms are distinguished. In natural conditions, as a rule, there is a mixed form. The course of the disease can be acute, subacute and chronic. The manifestation of signs of the disease depends on the type of pathogens, AI and resistance of the organism. Even before the appearance of oocysts in the feces, the mucous membranes of the eyes, nasal and oral cavities become anemic, later jaundice appears. Rabbits are reluctant to eat food or completely refuse it, sluggish, lie more on the abdomen. The volume of the abdomen is increased, painful on palpation, feces retain consistency, but may also be liquid. Rabbits have frequent urination, some have rhinitis, conjunctivitis, increased salivation, sometimes convulsions, paralysis of the neck muscles, pelvic limbs are observed. Females are poorly fertilized, give small litters of 2-3 rabbits, that is, weak, since females are low-dairy. In sick rabbits, molting is delayed, the fattening period is prolonged. Mortality reaches 70%. Recovered rabbits secrete oocysts for a long time.

Pathoanatomic changes. The corpse is exhausted. The blood vessels of the intestine are injected. The small intestines contain a lot of mucus and are filled with gases. The mucous membrane is hyperemic, there are multiple hemorrhages on it, the total mass of the intestines is increased. Numerous hard whitish nodules containing eimeria are visible on the mucous membranes.

The liver is enlarged, the bile ducts are dilated, their walls are thickened. On the surface and inside the liver there are rounded or irregularly shaped nodules of dirty white or yellowish color, the size of a millet grain to a pea. There are a large number of oocysts in the nodules. Mesenteric lymph nodes are enlarged.

VSE carcasses, forcibly killed rabbits. Inexhaustible carcasses are used without restriction, depleted ones are checked for salmonellosis. If the result is negative, it is used for food.

The diagnosis is made comprehensively: based on epizootological data, clinical signs, autopsy results and microscopic examination of feces by the Darling or Fulleborn method.

Aimeriosis of rabbits should be differentiated from pseudotuberculosis, pasteurellosis, listeriosis and encephalitozoonosis.

Treatment. Sick animals are isolated, given food rich in carbohydrates (beets, oats, cereal hay).

Sick rabbits use sulfadimethoxine: on the first day, 320 g is added to 100 kg of feed, and in the next 4 days -160 g., then they make breaks for 5 days and repeat the course.

Norsulfazole is prescribed at a dose of 480 g together with phthalazole at a dose of 160 g per 100 g of feed. Therapeutic food is used for 5 days, then take a break for 5 days and repeat the course.

Furazolidone in a dose of 50 g per 100 kg of compound feed for 7 days.

Chemococcide in a dose of 0.03 g / kg is used with feed in 2-5-day courses with a break of 3 days. The effectiveness increases with the introduction of yogurt, ABC, and whey into the diet.

Prevention. Rabbits need to be kept in cages with a mesh floor (do not let it get stuck) in the open air or in non-insulated rooms. It is necessary to change the litter daily and scald the feeders and drinkers with boiling water. The food should be full-fledged with the addition of vitamins and trace elements.

During weaning of young animals from mothers, chemoprophylaxis is prescribed: sulfadimethoxine at a dose of 0.1 g and monomycin at a dose of 25,000 units / kg with feed in 2-5 day courses with an interval of 3 days, norsulfazole at a dose of 0.4 g with monomycin at a dose of 25,000 units / kg in 2-5 day courses with a break of 3 days, furazolidone at a dose of 0.02g/kg with feed according to the same system.

TOPIC № 16. TRICHOMONIASIS OF CATTLE.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of flagellated organisms and diseases caused by them, methods of in vivo –laboratory (taking a drop of blood, preparing smears, fixing and staining them with a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Cattle trichomoniasis is a chronically contagious, protozoal disease of cattle caused by Trichomonas foetus and is characterized in cows by abortions in the early

stage of pregnancy, vaginitis, metritis, and in bulls by balyanoposthitis and impotence.

Systematics of the causative agent of the disease. The causative agent of the disease according to the systematics are arranged as follows: Protozoa sub-kingdom, Sarcomastigophara type, Mastigophora subtype, Zoomastigophora class, Kinotoplastida order, Trichomonodida, Trichomonas, Trichomonas foetus suborder.

Morphology of the pathogen. The shape of Trichomonas foetus is oval, pear-shaped, fusiform, round, the size of which varies between 8-25 microns in length, 3-15 microns in width. The body of trichomonads consists of a cytaplasm, a nucleus, a kinetoplast, a wave-like membrane, flagella and an axostyle. In the stained Romanovsky-Giemse smears, the cytoplasm turns blue, the nucleus turns red, the kinetoplast and flagella are intensely red. Along the entire body of the trichomonas, an axostyle runs through the middle, it turns pale blue. The undulating membrane bordered by a flagellum resembles a hexagon and consists of 4-6 teeth, painted in pale blue color.

Biology of the pathogen. Trichomonads in cows live on the mucous membrane of the vagina, uterus, in the fetus and amniotic fluid, and in bulls – in the prepuce, penis and accessory glands. They have rheotaxis, which ensures their passage through the cervix together with sperm. Trichomonads feed on mucus, microbes and red blood cells. They multiply by simply dividing the maternal individual into two daughter ones, and sometimes by multiple division on nutrient media.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and on the basis of microscopic studies and the cultural method.

Microscopic examinations. To detect trichomonads, fresh material is taken. In cows, discharge or flushing from the vagina, amniotic fluid of the aborted fetus, scraping from the placenta, the contents of the abomasum, abdominal and thoracic cavities, fetal heart are examined; in bulls, flushing from the prepuce, the secret of the accessory glands and sperm. Flushes are obtained in various ways. In cows - with the help of a spoon - a Korchaga catheter, a vaginal mirror, tampons-sticks and other devices. In bulls - with a Korchaga scarifier, a Bozhevolny loop, a rubber pear with a catheter, etc. For flushing, take 1-25 ml of sterile saline solution. The resulting material is examined in a crushed drop, which is better to take from the bottom. Trichomonas are found under medium magnification of a microscope in a darkened field. You can apply vital coloring with a weak solution of eosin, methylene blue, safranin. It is better to conduct research on the farm. With a negative result, sowing is done on a nutrient medium from the washout sediment. From the flush, you can prepare a smear and paint it according to Romanovsky.

Cultural method. This method is used during the chronic course of the disease, when the clinical signs of trichomoniasis are not pronounced and the pathogen is difficult to detect microscopically. Trichomonads grow on artificial nutrient media. They are more often used by the medium proposed by V.V. Petrovsky. To liver water + 1% peptone + 0.5% table salt) add 1% maltose. The broth

is poured into 9 ml test tubes, layered with vaseline oil and sterilized in an autoclave at 110-1120 for 30 minutes; pH 7.2-7.4. Before sowing, 10% sterile blood serum of 1 ml (preferably horses) and antibiotics - penicillin 100 units and streptomycin 1500 units per 1 ml of medium are applied to vaseline oil on Wednesday. In the finished medium, sediment is sown in an amount of 0.3-0.5 ml. Cultured in a thermostat at 370 . The result of sowing is checked after 48-72 hours. The crops are monitored for two hundred days. When microscopy of cultures, it is better to first use a small magnification lens (8x) with a strong eyepiece (15x). This achieves a wide field of view and there is a great opportunity to detect trichomonads, if there are few of them.

Cattle trichomoniasis is differentiated from infectious follicular vestibulitis, a characteristic feature of which is the presence of yellow-white nodules on the mucous membrane of the vestibule of the vagina: from vibriosis - according to epizootological and clinical signs, microscopic examination and detection of vibrions; from listeriosis - based on a complex of studies and reactions of listeria agglutinations; from leptospirosis – except epizootological, clinical, pathoanatomic data, leptospira is detected in blood and urine; from toxoplasmosis – according to RDSK and RSK.

Treatment. Treatment and prevention of the su-aura of camels, horses and donkeys. Naganin is used for sick animals: camels at a dose of 0.03 g / kg, horses and dogs – 0,01-0,015 g / kg. After 10 days, naganin is re-administered to camels and dogs. When establishing the naganin resistance of trypanosomes, 7% azidine is recommended at a dose of 3,5 mg / kg. The solution is prepared on a distilled dose or on a 5% glucose solution (for horses), it is administered intramuscularly, twice with an interval of 24 hours. The finished solution is administered at the rate of 5 ml per 100 kg of animal weight. If the animal's weight is 400 kg or more, then the total dose of the drug is divided in half and injected into different places. For camels, the solution is injected intramuscularly in the area of the shoulder blades, for horses and donkeys – in the croup area.

Complex treatment with azidine (berenyl), tryponyl, DAT and naganin is also used. A positive result was obtained from the use of piraldine, an antricide analog, tripamidium and some others. In addition to trypanocidal drugs, animals are injected with cardiac drugs that stimulate digestion and hematopoiesis. Sick animals improve feeding and maintenance.

Prevention. After treatment, all animals are kept in isolation for 6 months. After 4,5 and 6 months, they are subjected to clinical examination, microscopic and serological studies. Animals found to have trypanosomes or a high titer of antibodies are treated repeatedly.

During the summer, blood-sucking insects of animals are distilled to pastures free of vectors. Msnao slaughtered animals are used for food in boiled form. The skins are disinfected by drying and salting. The corpses of animals that have fallen from trypanosomiasis are disposed of.

Treatment and prevention of accidental horse disease. Naganin is applied at a dose of 0,01-0,015 g / kg in 10% dilution in saline solution, intravenously. After 30-40 days, the administration of the drug is repeated. To prevent complications.

Horses 1-2 days before treatment and up to 7-10 days after the introduction of the drug are prescribed dressage to a light sweat (up to 3 times a day).

A good result is given by double administration of azidine (berenyl), triponyl, DAT, at a dose of 3,5 mg / kg in the form of a 7% solution on a 5% glucose solution, intramuscularly, a day later, so that there are no edema at the injection site. After treatment for the 4th-6th month, the animals are examined by all diagnostic methods. Horses that have given negative results are considered healthy, combined treatment is used for relapses. In addition to specific treatment, sick animals are prescribed t symptomatic (cardiac) remedies, improve feeding and maintenance.

Prevention. On the farm, individual patients are destroyed as a source of invasion. If it is not economically feasible, then patients and suspected of infection are treated. The rest of the horses are treated with triponyl or naganin in therapeutic doses in order to improve the horse's health as quickly as possible.

Treatment and prevention of trichomoniasis of cattle. In the early stage of the disease, cows are relatively easy to treat. When trichomonads penetrate into the uterus and ovaries, combination therapy is recommended. It is necessary not only to destroy the pathogen, but also to restore the normal state of the genitals. To do this, they improve the feeding and maintenance of a sick animal, as well as stimulate (with pituitrin, synestrol, etc.) uterine contraction to remove foreign contents.

In case of primary disease, douching of the genitals is prescribed with 8-10% ichthyol solution on glycerin or water, an aqueous solution of iodine (1:500), flavacridine (1: 1000). Etacridine lactate (1:2000), a 1% solution of metronidazole is administered intravenously three times at a dose of 80-100 ml. A.A.Sunaykin suggests irrigating the uterus with nitrofuran seed (0,1 g furazolidone, 0,2 g furacillin and 1000 ml of saline solution). Before changing, the seeds are brought to a boil and cooled.

In case of chronic course, combined treatment is recommended – 2 ml of 0,5% solution of proserin or 1% solution of synestrol in a dose of 2-4 ml twice a day, followed by irrigation of the vagina and uterus with disinfectant solutions in the amount of 500-600 ml. In the presence of pyometritis and endometritis, in addition, the uterus is massaged. The course of treatment lasts 5-7 or more days.

In bulls, a 6-day course of treatment is effective, consisting of subcutaneous injections of a 1% solution of furamon in an amount of 1,5-2 ml (with an interval of 48 hours) and four intramuscular injections of a 10% suspension of furazolidone prepared in olive oil or fish oil at a dose of 10 ml per 100 kg the mass of the animal. Simultaneously solution of nitrofuran mixture. After 10-12 minutes, 25-30 ml of 5% furazolidone oil emulsion is injected into the prepuce, then a long massage is performed.

A 5-day course of treatment with metronidazole (trichopol, flagil) gives a good result. It is administered intramuscularly or subcutaneously at a dose of 0,05 g / kg in saline solution or in a water-glycerin mixture (1: 3) and simultaneously washed the preputial cavity with a 3% tricho-floor emulsion on distilled water or a 1% suspension on fish oil.

After the course of treatment in bulls, flushes from the prepuce or sperm are examined five times every 10 days, and in cows, flushing from the vagina is examined three times. The study is carried out by the cultural method.

Prevention. The main thing is to prevent a splinter of invasion into the farm. Since infection occurs sexually, with artificial insemination with sperm from sick bulls, as well as through care items, it is necessary to observe control when new animals enter the farm. In case of artificial insemination, all tools and objects of care for bulls should be only individual.

If the disease has already appeared on the farm, then only artificial insemination is recommended. Sick cows are isolated from the general herd and treated. All bulls are examined, patients are also isolated and, depending on the age of the disease and the tribal value, they are treated or handed over for slaughter. Sperm from bulls that have undergone cruz treatment is used after a five-time study or after a bioassay on healthy heifers.

A 20% solution of freshly slaked lime is used to disinfect the places where sick animals are kept, as well as zhizhestoks. Litter and manure are treated biothermically.

CONTROL QUESTIONS:

1. Tell us the characteristics of trichomoniasis of cattle
2. How is the causative agent of trichomoniasis located according to the systematics, tell us the morphology and biology of the pathogen?
3. Laboratory methods for diagnosing trichomoniasis?
4. Trichomoniasis of cattle should be differentiated from what diseases?
5. How do we treat cows and bulls with trichomoniasis, specific and symptomatic methods and treatment regimens used?
6. How to improve the economy if the disease has already appeared?
7. How do we prevent the farm from trichomoniasis?

TOPIC № 17. SU-AURA AND ACCIDENTAL HORSE DISEASE.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of flagellated organisms and diseases caused by them, methods of in vivo –laboratory (taking a drop of blood, preparing smears, fixing and staining them with a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Su-aura of camels, horses and donkeys. Su-aura – estro or chronically occurring transmissible, seasonal protozoal disease of animals caused by protozoa Trypanosoma ninaekolhjakimovae belonging to the Sarcomastigophara type, characterized by fever, edema, enlarged lymph nodes and severe exhaustion of the animal.

Systematics of the causative agent of the disease. The causative agent of the disease according to the systematics are arranged as follows: Protozoa sub-kingdom, Sarcomastigophara type, Mastigophora subtype, Zoomastigophora class, Kinotoplastida order, Trypanosomatina suborder, Trypanosoma genus, Trypanosoma ninaekolhjakimovae species

Morphology of the pathogen. Trypanosoma ninaekolhjakimovae (some researchers consider it a synonym of Trypanosoma evansi), morphologically similar to other trypanosoma species. The shape of trypanosomes is elongated, fusiform, gimlet-shaped (), the size of the parasite varies between 20,9-32,0x1,4-2,8 microns, the body of trypanosomes consists of cytoplasm, nucleus, kinetoplast and flagellum.the draw is rounded, located in the middle part of the body, the kinetoplast – in the form of a small grain is located at the posterior end of the body, the flagellum originates from the kinetoplast, runs along the body and ends at the anterior end freely. Trypanosomes are localized in blood plasma, lymph nodes, internal organs, and the nervous system. It moves briskly, progressively, reproduces by longitudinal division into 2, 4 and possibly 6 daughter individuals. In natural conditions, camels, horses, donkeys, mules, dogs, cattle and wild mammals are susceptible to the disease. Laboratory animals are artificially infected: (white mice, rats, guinea pigs and rabbits).

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and on the basis of microscopic and serological studies and a biopsy.

Microscopic studies. Blood taken from peripheral vessels (skin, auricle) is examined by the crushed drop method. Mobile trypanosomes are visible between red blood cells under medium magnification of a microscope in a darkened field. This method identifies up to 20% of patients. You can examine a thin smear of blood stained according to Romanovsky. At the same time, the cytoplasm of trypanosomes turns bluish-purple, the nucleus and flagellum are red–purple, the kinetoplast is pale pink.

Serological studies. In camels, they are carried out with the help of a formalin reaction. In an animal, blood is taken from the cervical vein into a clean bacteriological tube up to three-quarters of its volume, and maintained at room temperature for 1-2 days. The separated serum is carefully sucked with a pipette and 1 ml is poured into a test tube. The test tube is closed with a cotton plug, shaken and left in the room for two days for observation. Reaction results: if the serum has become jelly–like and does not drain when the test tube is overturned, the reaction is positive; if the serum has thickened, but when tipping or tilting the test tube very slowly drains, the result is doubtful; if the serum remains liquid, the result is negative. Camels that have shown positive and questionable results are considered suspicious due to the su-aura disease.

Currently, an agglutination reaction (RA) has been developed to identify sick camels, RSC and RDSC with an antigen are used in horses and dogs to diagnose accidental illness. In addition to these methods, an indirect immunofluorescence

reaction and an indirect immunofluorescence reaction with complement binding have been developed.

B i o p r o b a. Blood is taken from animals suspected of su-aura disease and laboratory animals (mice or rats) are infected, which are acutely ill and die on the 6th-8th day after infection. Trypanosomes are found in their blood. Group diagnostics is also carried out. To do this, blood from several animals suspected of su-aura disease is mixed and injected into laboratory animals. Even if one of the animals turned out to be sick or a carrier of trypanosomes, the laboratory animal becomes ill.

Postmortem diagnosis is based on pathoanatomic changes. The corpse is emaciated, the mucous membranes and serous membranes are anemic. There is serous fluid in the thoracic and abdominal cavities. Lymph nodes and spleen are enlarged. Blood coagulates poorly, the heart muscle is flabby, hemorrhages. The intestinal mucosa is swollen, hemorrhages in places, congestive hyperemia in the kidneys, liver.

Differential diagnosis. The aura of animals should be differentiated from helminthiasis, accidental disease, pyroplasmosis, nuttaliosis, infectious anemia, tuberculosis and other diseases.

Accidental equine disease is a chronically occurring, contagious, protozoan disease of ungulates caused by Trypanosoma etquiperdum, characterized by damage to the genitals, the formation of thaler plaques on the skin, unilateral facial nerve paresis, squatting and sometimes death of the animal.

Systematics of the causative agent of the disease. The causative agent of the disease according to the systematics are arranged as follows: Protozoa sub-kingdom, Sarcomastigophara type, Mastigophora subtype, Zoomastigophora class, Kinotoplastida order, Trypanosomatina suborder, Trypanosoma genus, Trypanosoma etquiperdum species.

Morphology of the pathogen Trypanosoma etquiperdum. Morphologically, Trypanosoma etquiperdum does not differ from Trypanosoma ninaekolh-jakimovae. The size of her body is 22-28 microns in length and 1,4-2,6 microns in width. It is localized in the capillaries of the mucous membrane of the genitals, only ungulates are susceptible, laboratory animals and dogs are infected in exceptional cases, it is not cultivated on nutrient media, reproduction is typical for all trypanosomes.

Diagnosis and differential diagnosis. The diagnosis during life is made on the basis of epizootological data, clinical signs and on the basis of microscopic and serological studies.

Microscopic studies. Scrapings are taken from the mucous membrane of the urethra and vagina: in mares with a sterile spoon, a Korczak catheter or a slide, in stallions with a sterile urethral spoon or a Volkman spoon. Moreover, in addition to mucus, there should be a small amount of blood in the scraping. It is examined immediately by the crushed drop method at an average magnification of the microscope. They are looking for live mobile trypvanosomes.

Serological studies are carried out with the antigen from trypanosomes in the RSC. From horses suspected of having a disease, 2-3 ml of fresh blood serum is sent to the laboratory.

Security questions:

1. Tell us the characteristics of the su-aura?
2. How is the pathogen of the su-aura located according to the systematics, tell the morphology and biology of the pathogen?
3. How is the diagnosis made during the life of the animal on the su-aura?
4. Postmortem diagnosis of the su-aura?
5. The Su-aura should be differentiated from what diseases?
6. How do we treat patients with trypanosomiasis of animals, specific and symptomatic treatment methods used?
7. How to improve the economy if the disease has appeared?
8. How do we prevent the farm from trypanosomiasis?
9. Tell us the characteristics of the accidental illness of horses?
10. How is the causative agent of accidental horse disease located according to the taxonomy, tell us the morphology and biology of the pathogen?
11. How is the diagnosis made during the life of an animal on a case of horse disease?
12. Accidental horse disease should be differentiated from what diseases?
13. How do we treat patients with accidental animal diseases, specific and symptomatic treatment methods used?
14. How to improve the economy if the disease has appeared?
15. How do we prevent the farm from accidental illness?

TOPIC № 18: PSOROPTOSIS, SARCOPTOSIS ANDCHORIOPTOSIS OF ANIMALS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of arthropods, methods of in vivo –laboratory and postmortem diagnostics of psoroptosis, sarcoptosis and chorioptosis of animals.

Materials and equipment. Water, bucket, microscope, slide glass, samples of scrapings from the border of affected and healthy skin areas, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Psoroptosis (cutaneous scabies) of sheep is an acute or chronic disease caused by mites from the genus Psoroptes, parasitizing the epidermal layer of the skin and characterized by itching of the skin, hair loss and exhaustion of the body, sometimes in some animals the disease ends in death.

Systematics of the pathogen. The causative agent of sheep sporoptosis according to the systematics are arranged in the following order: type – Arthropoda, class Arachnoidea, order - Acariformes, suborder Sarcoptiformes, superfamily – Sarcoptoidea.

Sarcoptoidosis (Sarcoptoidea) is caused by scabies, representatives of two families: Psoroptidae and Sarcoptidae. These are small mites, 0.2-0.8 mm in length, their body is round or oblong-oval, slightly flattened in the dorsoventral direction. The cuticle is dense, often striated. There are protrusions and bristles on it. In front of

the body there is a proboscis, the shape and structure of which are different depending on the genus. There are four pairs of five-jointed legs, and the larvae have three: the first two pairs are located in the anterior third of the body and are directed forward. The legs of the legs are armed with one or two claws and suckers on rods. Ticks do not have eyes and special respiratory organs, they breathe with the entire surface of the body. Phases of development: egg, larva, protonymph, teleonymph and imago.

Morphology of ticks of the family Psoroptidae - genus - Psoroptes, species: Psoroptes ovis (sheep), Psoroptes bovis (cattle), Psoroptes equi (horses, donkeys and mules) and Psoroptes cuniculi (rabbits) Body shape is oblong-oval, the oral apparatus of the piercing type, adapted for piercing the epidermis and sucking lymph. The legs are long, the development, the suckers are on long jointed or short non-jointed rods. The eggs are oblong-oval, asymmetrical. The flat side of the uterine secret is fixed to the host's body. Ectoparasites. Ticks of this family cause psoroptosis (cutaneous scabies), chorioptosis (leathery scabies and otodectosis (ear scabies) in various pitchforks of domestic animals.

Mites of the genus Psoroptes are the largest among scabies mites. Mature individuals can be seen with the naked eye. They are gray-yellow in color, the length of the male is up to 0.5 mm, the female is up to 0.8 mm, the oral apparatus is adapted for piercing the skin and sucking lymph. The legs are developed, at their free end there are thin-walled black-walled rods ending in suckers. In males, these suckers are on the first, second and third pair of legs. Females lay eggs on the surface of the host's skin. Eggs are oblong-oval in shape, up to 0.3 mm long, blunted at both poles, asymmetrical. The flat side is adjacent to the host's skin, being held on it thanks to the uterine secret.

Ticks of the genus Sarcoptidae. These are the smallest-sized scabies mites. Their body is rounded, the oral apparatus is gnawing, horseshoe-shaped, their legs are short, thickened with non-jointed rods. The egg is strictly oval. Ectoparasites live inside the skin.

The family Sarcoptidae is divided into two genera: the genus Sarcoptes and Noroedres.

The genus Sarcoptes causes sarcoptosis in animals. In horses, Sarcoptes equi is parasitized by uloshadei, in cattle - Sarcoptes bovis, in camels - Sarcoptes camellia, in pigs - Sarcoptes suis, in sheep - Sarcoptes ovis, in dogs - Sarcoptes canis, in rabbits - Sarcoptes cuniculi and in reindeer Sarcoptes rangiferi. Ticks of this genus (itches) are small, the body length of males is up to 0.2 mm, the length of females is 0.45 mm, the body is rounded, dirty gray in color, there are scales and thorns on the dorsal side, the proboscis is developed, horseshoe–shaped, adapted for gnawing passages in the skin of the host.The legs are short, cone-shaped, the front pairs of legs are the most developed. Females have unsigmented rods on the front pairs of legs, ending in suckers, and the rear pairs of legs without suckers, but with long bristles. In the male, the first, second and fourth pairs of legs end with rod suckers, and the third pair with a long bristle. The posterior end of the body of the female and male is rounded. The male has no copulatory suckers. Anal opening at the posterior end of the body.

The eggs are oval in shape, slightly blunted at the poles, 0,15-0,25 mm long, with a two-layer shell. A six-legged larva up to 0,15 mm long emerges from the egg after 3 to 6 days.

The genus Noroedres causes notohedrosis in animals. In dogs, cats and rats, Noroedres cati parasitizes, in rabbits - Noroedres cuniculi. Morphologically, Noroedres have many similarities with ticks of the genus Sarcoptes, they are distinguished by the location of the anal opening. The original place of settlement of ticks in cats is in the area of the base of the ears, forehead and upper part of the neck, in rats – the edges of the ears and tail.

In cats and dogs, as well as fur-bearing animals, in the absence of treatment, ticks spread to other parts of the body, and a severe generalized form of notohedrosis occurs.

Ticks of the genus Chorioptes parasitize on the surface of the skin of animals. The causative agent of choryptosis in horses is Chorioptes equi, in cattle - Chorioptes bovis, in sheep - Chorioptes ovis, in goats - Chorioptes caprae and in rabbits - Chorioptes cuniculi.

Morphologically, skin-eating mites occupy an intermediate position between pruritus and skinworms. They are relatively large, the length of the male is up to 0.3 mm, the female is 0.4-0.5 mm, the body shape is oblong-oval, the proboscis is short, rounded, the oral apparatus is gnawing, the chelae are wedge-shaped, they feed on the scales of the epidermis and the products of inflammation caused by them. These mites are characterized by wide tulip-shaped suckers on their legs, located on short non-pigmented rods.

Sexual diformism is well expressed, in the male the posterior end ends with two abdominal lobes bearing four long bristles, of which two are flat. Suckers are present in all legs, the fourth pair of legs is three times smaller than the third pair of legs. In the female, the posterior end of the body is obtusely rounded, there are no suckers on the third pair of legs, instead of them there are long bristles.

Otodectosis is an ear scabies, the causative agent of which is Otodectes cynotis, parasitizing in the external auditory canal, on the eardrum and in the auricle of carnivores. The disease is severe in foxes and arctic foxes. Ticks of this genus bear some resemblance to skin-eating ticks. The womb of the body is oval, gray-yellow in color, males up to 0.3-0.4 mm in length, females up to 0.5 mm. The proboscis is weakly protruding, rounded in shape, the oral apparatus of the gnawing type. Sexual diformism is well expressed, the male has no abdominal processes, but there are two tubercles on the posterior edge of the body, from which two long and short scutes depart.The fourth pair of legs is poorly developed, the free end of each leg ends with a tulip-shaped sucker sitting on a short rod. In the female, the fourth pair of legs is poorly developed and does not even protrude beyond the edges of the body, suckers are available only on the second pair of legs.

Diagnostic methods. The diagnosis is made on the basis of epizootological data, clinical signs and laboratory studies.

Epizootological data. Sarcoptoidosis of domestic animals is widespread. Animals are infected by direct and indirect contact of patients with healthy ones.

Young emaciated animals get sick more often and hard. Scabies mites find favorable conditions for their development in the cold and humid season, when the animals' hairline lengthens and becomes denser and the humidity of the subcutaneous air increases; the maximum spread of these mites is winter and early spring.

With the onset of the warm season, the molting of animals, the number of ticks on the host's body decreases sharply, which leads to the appearance of a veiled course of the disease. The exception is ear scabies, which manifests itself at different times of the year. However, without medical intervention, self-recovery of animals with sarcoptoidosis does not occur. Itchies live on the body of animals for 4-6 weeks, outside the body of the host for 3-4 weeks. The skinners are more stable, they live up to 60 days on the host's body, up to 65 days outside the body, and only 2 days on pastures in summer.

Clinical signs of the disease. Sarcoptosis is most severe in horses, goats, pigs, reindeer, and notohedrosis – in cats and fur-bearing animals. With sarcoptosis, the incubation period is 2-3 weeks. Young animals become infected more often and are more seriously ill. The main symptoms: itching of the skin, local increase in body temperature, the appearance of small nodules and bubbles on the skin. Bubbles burst when combing, their contents pour out onto the surface, dry out, as a result, a flaky layer forms on the skin, sweat and sebaceous glands, as well as hair sacs, are injured and destroyed at the same time. In such areas of the body, hair falls out, the stratum corneum is thinned, the skin loses elasticity, cracks in places. At the same time, the animals develop anemia and progressive exhaustion.

Ticks initially settle on the animal's head, the upper part of the neck, the inner surface of the earlobe (in pigs), on the root of the tail, legs and other parts of the body. The skin thickens and gathers into folds at the places of contact.

With psoroptosis, the initial signs of the disease are noted in areas of the body covered with a denser long hair cover. Unlike sarcoptosis in psoroptosis, the skin folding is weakly pronounced or completely absent. When combing, the hair falls out in clumps and forms bald spots. Often the bald areas merge, as a result of which the thermoregulation of the body is disrupted. Animals lose weight quickly, become susceptible to colds and die.

Psoroptosis – cutaneous scabies is most severe in fine-fleeced sheep and young cattle. The foci of the initial settlement of ticks are the area of the sacrum, back and neck. In horses, psoroptosis begins at the withers, and from there spreads to the neck, back, upper parts of the shoulder blades and other areas of the body. In cattle, changes are first noted on the skin in the neck, tail root, sacrum and at the base of the horns. In goats and rabbits, skin mites are localized on the inner surface of the auricle and the external auditory canal, where layers of brown crusts are formed. Here they find favorable conditions for the development of the pyogenic microflora. Such patients, especially rabbits, develop inflammation of the middle and then the inner ear. The head is turned to 450 (crooked head).

Chorioptosis manifests itself in the form of focal skin lesions. It is covered with rejected hyperpidermal cells and dried lymph. There are ticks under such a crust. In horses, the primary foci of the settlement of skin–eating mites are the area of the fetal

joint (for which this disease is called "foot scabies"), in cattle – the area of the root of the tail and limb, more often in the area of the fetal joint or on the inner surface of the thighs, in sheep – the head, limbs and scrotum, in goats – neck, head, the lateral surfaces of the trunk and the root area of the tail.

Laboratory studies are based on the detection of scabies mites or their eggs in skin scrapings. Scraping is taken with an abdominal scalpel from a freshly infected area or from an old one, but on the border with a healthy area of the skin, since the largest number of ticks accumulate in these places. Due to the fact that ticks can be ecoto- and endoparasitic, scrapings should be made deep (in an amount of at least 0.5 cm3) so that there is blood in them.

The resulting material is examined on site or in the laboratory. The sent material is placed in a laboratory cup, at the bottom of which moistened filter paper is placed. The grooves between the glasses of the cup are filled with paraffin or smeared with plasticine. Scrapings can be packed (separately from each animal) in a well-closed stopper or in a cellophane bag, which is wrapped in thick paper or placed in a closing glass or plastic jar.

The scraping material is examined for the detection of dead ticks or their fragments (mortal methods) or for the appearance of living mobile ticks (vital methods).

To establish a primary diagnosis, usually use mortal methods, of which the following two methods are the most common6

1. The scraping is placed on a watch glass or in a laboratory cup, or on the center of the slide. A double amount of 10% sodium or potassium solution is added there by volume. All this is stirred and left for 25-40 minutes to soften and dissolve the crusts. To speed up the study, the resulting seed is heated to 60-700.Then the material is distributed in small portions between the slide and cover glasses and examined under a small magnification microscope with a slightly darkened field of view.

2. The method of M.P.Dobychin. In a test tube with 1 ml of 10% sodium hydroxide or potassium solution, skin scraping is applied and heated for 1-2 minutes. After 3-5 minutes, the tube is filled with 55% sugar solution or 60% hyposulfite solution and left alone for 5 minutes. Then, drops are taken from the surface of the solution with a wire loop and transferred to a slide, covered with a cover glass and examined under a microscope.

Vital methods are aimed at detecting live ticks, which is important not only for diagnosis, but also for evaluating the effectiveness of the treatment.

1. The method of D.A.Priselkova. The scraping is placed in a laboratory cup or on a watch glass, or on a slide and a double amount of kerosene is added to it. The scraping crusts are thoroughly stirred with a dissecting needle, scalpel or slide edge. Crushed droplets are prepared from the resulting material, which are viewed under a small magnification microscope. Scabies mites in kerosene retain their viability for up to 4 hours.

2. Skin scraping is placed on a watch glass, an eight-fold amount of water is poured in there and everything is thoroughly mixed. Then the material is placed for

15 minutes in a thermostat at a temperature of 35-400, and then the hour glass is placed on the microscope slide and examined under low magnification. Ticks in thermal water make active movements and become noticeable.

3. Scraping is put into a laboratory cup, turned upside down and put it in this position on a heat source (up to 450) After 5-10 minutes, skinners and skinheads come out of the scraping crusts, and after 12-15 minutes itches. Then the lid of the cup is viewed under a microscope or under a magnifying glass.

4. A fresh scrape taken from an animal suspected of having a disease of cutaneous or leathery scabies is put on black paper and heated to 350 from below. Under the influence of heat, the mites crawl out of the crusts after 3-5 minutes. They move on paper and on a dark background are noticeable even to the naked eye.

Treatment, prevention and control measures. The whole flock is considered to be dysfunctional for psoroptosis if at least one sick animal is detected.

Depending on the air temperature, sick sheep are treated with a wet and dry method. The wet method is more efficient and less time-consuming.

Hexachlorane emulsions are used for wet treatment. Containing 0.023-0.030% gamma isomer. The emulsion is prepared from various preparations, in particular from a 16% mineral-oil emulsion of the gamma isomer of HCG. It is most advisable to use enriched hexachlorane (92% of the gama isomer) or lindane (99-100% of the gamma isomer) to combat psoroptosis. The best effect is obtained by double treatment of a dysfunctional flock with a 10-12-day interval. In the process of bathing sheep, wool and fat are adsorbed from the emulsion of creolin and gamma isomer. Therefore, to compensate for the "depletion" of the working emulsion, a liquid with a double content of gamma isomer is added to the bath.

After repeated bathing in 2-3 weeks, a thorough analysis of the treatment is carried out. If by this time the itching in the affected areas has not stopped, then scrapings are re-examined in such animals. With a positive result, other drugs are used: aqueous emulsions of neocidol, vetionol, cydrin, cymbush, karate, alpha and others.

In winter, when the wet method cannot be used, it is recommended to use colloidal sulfur dust-the dry method, twice, as well as organophosphorus preparations in aerosol and non-repellent packages: acrodex, dermatosol, cyodrin, psoroptol and others. Animals treated with dust, regardless of the results of treatment, are bathed twice in hexachlorane emulsion in the spring after shearing

In addition, subcutaneous injections of ivomek, baymek, PIVS, and ivermectin at a dose of 0.2 g / kg of body weight have recently been recommended.

For the prevention of the disease, it is important that the animals are grazed on pastures isolated from the livestock of other farms, new animals are allowed into the farm after preliminary quarantine, if there is a threat of skidding in the farm, it is treated with colloidal sulfur for preventive purposes or subcutaneously injected with ivomek, imermectin.

Security questions

1. What is the veterinary significance of parasitoform and acariform mites, give examples?

2. The longevity of acariform mites in different species?

3. Tell us the systematics and anatomical and morphological structures of ticks of the Psoroptidae family

4. Measures to combat psoroptosis of animals?

5. Tell us the characteristic clinical signs of psoroptosis?

6. How to diagnose sarcoptosis and chorioptosis?

7. What is the difference between psoroptosis and sarcoptosis and chorioptosis of animals?

TOPIC 19 № HYPODERMATOSIS OF CATTLE.

Hypodermatosis is a chronic disease of cattle caused by larvae of subcutaneous gadflies, characterized by inflammatory phenomena in habitats, general intoxication of the body and a decrease in animal productivity.

Two species parasitize cattle: Hypodermabovis – common subcutaneous gadfly (string), the other - Hypodermalineatum - southern subcutaneous gadfly (esophagus).

The main host for both types of gadflies is cattle. String larvae can develop in zebu, horse, sheep and goat, and esophageal larvae – in the body of yak, zebu, horse, goat and sheep.

Morphology of gadflies. Imago are large, up to 2 cm long insects resembling bumblebees in appearance. The body consists of a head, chest and abdomen, covered with thick hairs of yellow, orange and black colors. The esophagus differs from the string by its slightly smaller size and color of the chest and abdomen hair. The eggs are small, with a diameter of 0,85-0,86 mm, and together with the attached appendage -1,09 mm.

The face of the I-stage at the exit from the egg has a length of 0.6 mm, before the first molt - 17 mm, the color is white or pale yellow. At this stage, the larvae of the string and the esophagus differ in the structure of the oral hooks: in the string they are separated at an acute angle in the form of a fork, and in the esophagus the front end is pointed and has a tooth-shaped protrusion.

Stage II larvae are larger (18-20 mm) than stage 1 larvae. In place of the mouth hooks, they have only small pigmented areas.

Larvae of the III-stage of the string, up to 28 mm long, esophagus -16-26 mm. In the row, the posterior spiracles have an irregular oval contour from the outside, the scar is located at the bottom of the funnel and is usually completely closed. The height of the spiracles is greater than that of the esophagus, reaching 1.2 mm. On the posterior edge of the penultimate abdominal segment from the dorsal side of the larva there is no zone with thorns. The esophagus has this zone, although small, but there is. In addition, the larvae of stage III of the esophagus differ in that they have flat, rounded, up to 1 mm high posterior spiracles, around which there is a zone of 10-15 rows of small thorns. The row has almost twice as many rows.

Pupae are darker in color than larvae, but retain all their signs. The dorsal side of the pupa is almost straight, with a pronounced cap at the anterior end.

Biology of gadflies. Gadfly refers to insects with a complete transformation. The full development cycle ends within a year.

The exit of the imago gadflies from the pupae occurs very quickly – within 2-3 seconds and after 30-80 seconds the fly is able to fly and mate. Adult gadflies do not feed and live off the nutrients accumulated in the larval phase, so their life is short 3-10 days, and when the temperature drops – up to 28 days. An adult gadfly loses up to 36% of its mass by the end of life.

On sunny days, the gadfly years are observed at a temperature of +6-80C, and on cloudy days – at +13-140C. Males gather annually in the same places where females arrive for mating. After mating, the cut-off females go in search of animals for laying eggs.

When attacking animals, female gadflies behave differently, the string flies around the herd, chasing animals and making specific sounds. At the same time, the animals are very worried, they try to get away from persecution, and the female of the esophagus gets close to the animals by short flights, crawling, falling behind unnoticed, she calmly lays eggs on the hairline. The string lays one hair at a time, the esophagus lays 5-20 eggs at a time.

Female gadflies are very prolific, laying up to 800 eggs. For laying eggs, females prefer areas with a short spine and abundant undercoat in the area of the hungry pit, the soft wall of the abdomen, groin, and the front of the thighs. The formation of the larva inside the egg lasts 3-7 days in the row, 3-6 days in the esophagus. The larvae hatched from the eggs penetrate through the skin into the host's body. Then the larvae of the string migrate along the large vessels and nerves to the spine and through the intervertebral openings enter the adipose tissue of the spinal canal, and the larvae of the first stage of the esophagus migrate towards the esophagus and are localized in its submucosal layer.

The total duration of stay of larvae in the esophagus and spinal canals is 5-6 months. After that, the larvae migrate to the back and lumbar region, where they form connective tissue capsules. For further development, they need oxygen from the atmospheric air, for which the larvae form fistula openings in the skin. After 1-8 days, the larvae molt and enter stage II. Mature stage III larvae exit the capsule through fistula openings in the skin (mainly in the morning and afternoon) and get to the ground, where their pupation takes place. The fallen larvae are inactive, pupation occurs within 1-2 days, less often within 7 days. The duration of pupal development is 20-40 days.

Epizootological data. The density of livestock settlement is one of the main factors affecting the number of gadflies and the infestation of animals with larvae. The extensiveness and intensity of gadfly infestation of young animals is higher than that of adult animals. The movement of cattle infected with subcutaneous gadfly larvae inevitably leads to the spread of hypodermatosis. A much greater danger is the loss to pupation of even a small number (10-15) of stage III larvae from animals

accidentally untreated in autumn, leading already during the first season and over-infection of 40-50% of the livestock of the stage.

Pathogenesis. The larvae of horseshoe gadflies have a mechanical and general toxic effect on the body of cattle and open the gates to the development of secondary infections.

Symptoms of the disease. The first clinical signs of hypodermatosis appear when gadfly larvae that have emerged from eggs are introduced into the skin. Animals develop itching, swelling of the subcutaneous fangs, soreness of the areas affected by the faces. These symptoms can be observed during the whole period of the gadfly summer.

When the larvae approach the skin of the back, the symptoms are clearly manifested in all affected animals. At first, small seals form under the skin of the back, barely felt during palpation, after a while they turn into faintly noticeable bumps with a hole in the center. When pressing on them, the animal has a painful reaction. As the larva grows, the discharge of serous fluid from the fistula opening increases, which glues the hair. The skin covering the fistula capsule is inelastic, with fever and painful.

The hair on the back of a sick animal is in patches or completely tousled. At the locations of the fistula capsules, bundles of hair glued together with dried exudate are directed in different directions. With abundant discharge of purulent exudate from fistulas, the hairline is contaminated with greenish-yellow secretions.

Pathoanatomic changes. When opening animals during the period of infection with larvae, small bubbles can be found in the subcutaneous patch, in which larvae from 1 to 5 mm in length are viewed. The latter, upon careful examination, are also found on the superficial fascia of the trunk, in the connective tissue layers of the muscles, dirty green stripes of secretions are noticeable on the migration routes of the larvae. Affected larvae areas of the esophagus are hemorrhagic, edematous both from the mucous membrane and serous. In places where larvae accumulate in the spinal canal, hemorrhages.

During the period of parasitization of larvae of stages II and III, the greatest changes are noted in the skin, subcutaneous tissue and muscles. Fistula capsules are clearly visible here, surrounded by a gelatinous mass of light or pink color, permeated with numerous blood-filled vessels. With a strong lesion of larvae, serous or serous-hemorrhagic inflammation of the muscles is noted, covering extensive areas of the back.

Diagnosis. The diagnosis is made on the basis of examination and palpation of the animal's skin in the places of accumulation of larvae of the II and III stages of subcutaneous gadflies – along the entire length of the back from the withers to the sacrum.

Treatment and prevention. The main method of combating hypodermatosis is the destruction of stage I larvae of subcutaneous gadflies in the body of animals. To do this, systemically acting insecticides are used: hypodermin-chlorophos, chlorophos, neguvon, neguvon N, ivomek, ivermectin, baymek, rustomectin, PIVSA and others. Animals are treated in the autumn after the end of the gadfly summer: in

August-September-October. Moreover, all cattle grazed on pasture are subject to processing, in order to achieve 100% effectiveness of treatment, animals are treated twice in autumn with an interval of 30 days. The drugs are applied to the back from the dispenser in a thin trickle on both sides of the vertebral column from the withers to the sacrum: hypodermin-chlorophos to animals weighing up to 200 kg in a dose of 16 ml, more than 200 kg -24 ml, ivomek and its analogues are administered once, subcutaneously in the neck, at a dose of 200 mcg / kg of the body.

In the spring, in order to identify sick animals, a single livestock survey is carried out. Animals affected by stage II and III larvae are treated with the above drugs in the same doses as with early chemotherapy.

Since the spread of pathogens of hypodermatosis occurs mainly by sick animals, cattle not treated with insecticides, infected with larvae, should not be allowed to pastures.

TOPIC №20. SHEEP ESTROGEN.

Estrosis of sheep –caused by larvae of the sheep gadfly Oestrusovis, parasitizing in the nasal cavity, frontal and paranasal sinuses of the head, accompanied by inflammation of the mucous membranes in the places of their parasitization. The sheep gadfly belongs to the family of nasopharyngeal viviparous gadflies (Oestriide), the Diptera order (Diptera) and the insect class (Insecta). The only owner of this species is a sheep, although it is quite common in domestic goats.

Morphology of the pathogen. The winged gadfly is yellow-brown or yellow-gray color, 10-12 mm long. Females are slightly larger than males. The body is covered with sparse short hairs sitting on small dark-colored bumps. The head is large, hemispherical in shape. Dark green shiny faceted eyes are separated by a forehead, on which 3 tubercles are arranged in a triangle. There is no oral opening, in place of its light plate, through which the rudiments of the jaws and proboscis shine through. The egg is white, oblong, up to 1 mm long, arched curved, with rounded ends. Female gadflies are viviparous. After 14-18 days after mating, grayish-white, small (1.27-1.35 mm), spindle-shaped, very mobile larvae consisting of 12 segments appear in the uterine expansion. Before molting, stage I larvae reach 4-5 mm in length and 0.34 mm in width. The larva of stage II is white, 5-12 mm long, up to 3 mm wide. The first thoracic and eighth abdominal segments are armed with small conical spines. The larva of stage III is 10-30 mm long . The rear horse of the larva is wider than the front one. Young larvae are white, with brown mouth hooks, adult larvae have dark hair on the dorsal side. The pupa has a length of 12 mm, a width of 5 mm, the lower end is blunt, the upper end, on which the notch is located, is beveled at acute angles, the color is initially dark gray, clouded brown.

The biology of gadfly. The imago of the sheep gadfly do not feed, but live off the reserves of nutrients accumulated in the larva phase. At the pupal phase, this reserve is economically distributed for the formation of an adult insect and the

creation of fat deposits in the body consumed during mating, maturation of larvae and flights.

The imago's exit from the pupa most often occurs in the morning in warm sunny weather. Mating lasts 2-3 minutes. After fertilization, the formation of larvae begins in the uterine receiver. During this period, the females fly, then for 10-20 days they sit in the recesses, crevices of buildings. Females with mature larvae dramatically change their behavior. They become active, mobile, and begin to fly. Injection of larvae into the nasal cavity of an animal can occur both on the fly and from the ground from a distance of up to 40 cm. At one time, the female throws out 8-12, sometimes 20-39 larvae.

The female's attack on sheep lasts 2-4 days, with a decrease in temperature – 5-6 days, after which she dies. During her lifetime, the female gives birth to about 600 larvae.

In the south, two generations develop during the year, and the autumn-spring generation develops in the usual time, and the summer-autumn generation develops in a shorter time. The life span of the imago is 12-13, maximum 46 days. The sheep gadfly lives near the breeding grounds, if there are sheep there. In search of animals for laying larvae, female gadflies travel distances of up to 30 km .

The main mass of stage I larvae is localized on the inner and outer surfaces of the lower nasal shells, nasal septum and the wall of the nasal cavity. Larvae of stages II and III develop in areas more isolated from the external environment – in the frontal sinuses and cavities at the base of the horns. The duration of larval development in sheep in zones with one generation per year is 8-11 months, and in zones with two generations of larvae of the autumn generation – 8-10 months, spring – from 20-30 days to 3-6 months.

Mature, well-pigmented larvae of stage III migrate from the frontal sinuses to the nasal cavity and during sneezing sheep are thrown to the ground. The departure of stage III larvae for pupation occurs more often in the morning. Pupation of larvae occurs in the soil at a depth of 1-5 cm . In areas with dense soil, larvae can pupate under a leaf, a chip, a stone. The pupae of the sheep gadfly tolerate low temperatures well. The duration of the pupal phase ranges from 14-17 to 46 days.

Epizootological data. The disease has been registered in many countries of the world, but mostly in steppe and semi-steppe areas with well-developed sheep breeding. The disease is characterized by high extensiveness, reaching 70,90 and even 100% in most zones with a high intensity of invasion by larvae, which number several dozen copies in each sheep. Old, sick with other diseases, weak animals are more intensively affected by larvae than healthy ones.

The highest intensity of infection of sheep with larvae is noted by the end of summer gadfly. During the winter period, some of the larvae die and the intensity of invasion decreases, in the period from March-April to May-June it is minimal due to the loss of larvae for pupation.

Pathogenesis. Gadfly larvae have a local mechanical and general toxic effect. Once in the nasal cavity of a sheep, gadfly larvae wound the mucous membrane with their thorns, which causes its inflammation. The mucous membrane ulcerates, swells,

and an abundant exudate begins. Inflammation can be complicated by microflora, turn into purulent-necrotic and spread to the membranes of the brain. The inflammatory process in the nasal cavity makes breathing difficult, and when larvae enter the trachea, aspiratory dyspnea develops.

Symptoms of the disease. During the disease, there are 3 periods. The first period begins after the invasion of sheep by gadfly larvae. The movement of larvae trapped in the nasal cavity and their attachment to the mucous membrane is very painful and causes protective reactions. Sheep sneeze, snort, shake their heads, rub their noses against legs and hard objects, trying to get rid of extraneous stimuli. On the 2nd-3rd day, serous-mucous discharge is released from the nostrils, sometimes with streaks of blood. Sick sheep have difficulty breathing, often digestive disorders. With the onset of the idle years, the gadfly stops and the symptoms of the disease disappear. There comes a second, hidden period of the disease. The third period develops closer to spring, when the intensive growth of larvae resumes and the disease worsens again. There is an abundant serous or serous-purulent discharge from the nasal shells, animals cough convulsively, trying to get rid of mucus, difficulty breathing, animals often snort. Along with the serous-purulent contents, the larvae of the III stage also fall out for pupation. During this period, the clinical signs of estrosis are most pronounced. Lambs are especially difficult to tolerate the disease, because of severe rhinitis, they breathe through their mouths, which prevents them from sucking queens. They grow slowly, switch to foot food early, and therefore the number of gastrointestinal diseases and waste among young animals increases.

Often, inflammatory processes from the mucous membrane of the nasal cavity spread to the pharynx, mucous membranes of the latticed bone, frontal sinuses and the lining of the brain. Purulent rhinitis appears, often accompanied by the phenomena of meningial syndrome, called "false vertyachka". The general condition of animals deteriorates sharply, they become sluggish, refuse to feed, lose weight quickly and die.

Pathoanatomic changes depend on the AI, duration and severity of the disease. In the first days of the disease, ulceration, hyperemia, catarrhal inflammation and swelling of the mucous membranes of the nasal cavity, often the trachea and bronchi, are noted. In winter, the changes are less pronounced and remain only in the locations of the larvae in the form of limited inflammatory areas.

During the transition of larvae to the II and III stages, the mucous membrane of the sieve bone is inflamed, the labyrinths are filled with mucous or purulent contents with an ichorous odor. Similar changes are not uncommon in the frontal sinuses and near the horn spaces. In sheep with signs of false vertebra, the vessels of the cerebral membranes are injected, the anterior parts of the large hemispheres are swollen and pigmented, there is infiltration in the ventricles and ammonium horns.

The diagnosis is established based on clinical signs and autopsy results of killed and fallen animals. Epizootic data are necessarily taken into account (seasonality of the disease and zonal features of the ecology of the sheep gadfly).

Estrosis of sheep should be differentiated from cenurosis, listeriosis and rabies.

Treatment and prevention. To combat estrosis, general preventive measures are carried out, in the fall – early chemotherapy and in the spring – treatment of clinical sick animals.

The spread of the causative agent of estrosis is mainly by sick animals. Therefore, it is unacceptable to export sheep to prosperous farms without preliminary therapeutic and preventive treatment of them, it is also not allowed to release sick sheep to pastures without treating them with insecticides. The autopsy of fallen animals should be carried out on a specially equipped site, and the fallen larvae are destroyed. In the spring, once every 2 weeks, koshary and tyr are thoroughly cleaned of manure and manure is stored for biothermal disinfection. In dysfunctional farms, sheep and goats grazed on pastures are subjected to early chemotherapy with an aerosol of DDVF and chlorophos or a solution of chlorophos is evaporated. A 0.1% solution of chlorophos is used by the method of free soldering, once. In animals with an obvious clinic of estrosis affected by stage II and III larvae, the nasal cavity is irrigated in spring with a 4% aqueous solution of chlorophos.

Recently, the following drugs have been recommended for the treatment and prevention of estrosis:ivomek, ivermectin, baymek, rustomectin, PIVSA and others. These drugs are administered subcutaneously at a dose of 0.2 mg per kg of live weight according to ADV, once.

TOPIC № 21. RHINESTROSIS AND GASTROPHYLOSIS EQUINE RHINESTROSIS OF HORSES.

Rhinestrosis is a chronically occurring, widespread disease caused by larvae of nasopharyngeal gadflies parasitizing in the nasal and adjacent cavities of the head and the disease is characterized by local inflammatory processes and general pathological phenomena.

The causative agents of rhinestrosis are 3 species of gadflies belonging to the family Oestridae genus Rhioestrus. These are Rh.purpureus-white-headed or Russian gadfly, Rh.latifrons - short gadfly and Rh.usbekistanicus - small-chip gadfly.

Morphology of pathogens. The Russian gadfly (Rh.purpureus) is very similar to the sheep gadfly. It is purple-brown in color, 10-12 mm long. The head is large, naked, swollen, especially in the transverse direction, white. The female's forehead is very wide, the male's is narrower. The oral organs are underdeveloped. On the brown with a reddish tinge of the back there are 4 shiny black longitudinal stripes, interrupted at the transverse dorsal suture. The abdomen is egg-shaped, almost naked, dirty violet-gray with a silvery sheen, dotted with black dots along the middle part. The legs are light to dark brown in color. The wings are 8-12 mm long, transparent, with 3 black dots at the base, distinguishing gadfly from other species.

The I–stage larva is about 1 mm long, oval in shape, wide in front and tapering in back. Its body is almost flat, slightly convex from the dorsal side. The head end is equipped with two powerful sharp curved hooks of light brown color. At the base of their mouth opening. The body is covered with spikes, on the sides of the body, on

segments, there are 4-5 rather long hairs, directed in an arc, with the tops backwards. On the rounded last segment there are spiracles and several curved spikes. The length of the larva before molting is 3.5 mm (stage II larva). The larva of stage III reaches a length of 17.5 mm, the front end of its body is narrowed and equipped with two screw hooks. The convex upper and flat lower sides are armed with spikes. The color of the larva is red at first, but becomes white as it matures. The pupa is 12.5 mm long and 6 mm wide, on the 10th segment, elongated-egg-shaped, slightly thicker at the back.

The short gadfly is 11-13 mm long, the longitudinal stripes on the midrib are red-brown, the body of the larva of stage III is wide, which differs from other species.

The gadfly is small - up to 9 mm long, the sides and bottom of the chest are covered with reddish hairs.

The biology of the pathogens of rhinestrosis is typical for gadflies of the family Oestridae. In the phase of adult insects, they are found throughout the summer: in our Republic, the flight of nasopharyngeal gadflies of horses goes from early May to mid-June and in autumn during September-October. The life expectancy of Russian gadfly females is 30 days, shorty - 46-52 and small-40 days, males – much shorter. In the conditions of Central Asia, all kinds of gadfly gives 2 generations per year.

The diurnal behavior of males is characterized by increased activity in the first half of the day. Females are active from hatching to mating, after which they sit in secluded places in the crevices of mud-brick buildings and duvals. This lasts for about 14 days. After the larvae mature, the intensive flight of females and infection with animal larvae begin again. In the hot hours of the day, gadflies rest on the branches of plants, stones, hills, roofs of buildings. The fertility of females is very high: the Russian gadfly has 700-792, the shorty has 640-1074 and the small-chip has 425-560 larvae.

To lay the larvae, female gadflies rapidly fly up to the horse's nostrils and, after injecting a portion of 8 to 40 or more larvae, fly away, However, this process does not always happen successfully for the larvae, since the horse, sensing the presence of its enemy, immediately begins to snort and shake its head up and down until the danger passes. Larvae trapped in the nasal cavity move deeper and are firmly attached to the mucous membrane. The habitats of stage 1 larvae are the inner surface of the shells and the labyrinth of the lattice bone. Here, in the labyrinth of the lattice bone, the larvae molt. Stage 3 larvae fall out of the nasal cavity, penetrate into the surface layer of the soil and pupate. Pupation takes place within a period of 24 to 48 hours . The duration of the pupal phase is 15-30 days and depends on the ambient temperature.

Epizootological data. The source of the invasion is infected horses, donkeys, mules. The area of the Russian gadfly is wider than shorty and maloship. EI and AI are higher in areas with a larger number of hosts. Larvae are more strongly infested and young and old animals are more seriously ill. The number of sick horses in dysfunctional farms reaches 75%. The intensity of infection with larvae is: average - 66, maximum – 416 copies.

Pathogenesis and symptoms of the disease. Gadfly larvae injure the tissues of the nasal cavity of animals with near-mouth hooks and spikes, which leads to catarrhal inflammation of the nasal mucosa and underlying tissues. Rhinitis and laryngitis develop. Infiltration and edema often occur at the sites of attachment of larvae, the act of swallowing is violated. When watering animals, water flows out of the nostrils. The development of purulent-necrotic processes is possible. Horses often cough, snort, soreness is noted when feeling the throat. The general condition of the animals is depressed, the submandibular and parotid lymph nodes are enlarged.

Pathoanatomic changes. Gadfly larvae are found on the mucous membrane of the nasal cavity and frontal sinuses. The mucous membrane is inflamed, dark purple in color, ulcerated.

The diagnosis is made on the basis of clinical signs, the results of pathoanatomic examination and the detection of gadfly larvae in the nasal cavity and pharynx. It is necessary to differentiate rhinestrosis from diseases of the upper respiratory tract, glanders, myta and gastrophylosis.

Treatment. The simplest method is washing the nasal cavities with solutions of drugs that kill larvae of nasopharyngeal gadflies. Apply in autumn and spring a 2% aqueous solution of chlorophos with a flow rate of 50-100 ml. Preparations of vivomek, ivermectin, baymek, rustomectin, PIVSA, bravomethin and others have high efficiency against all stages of larvae. These drugs are administered subcutaneously at a dose of 0.2 mg per kg of live weight according to ADV, once.

Prevention is poorly developed. It is recommended to eliminate imagos, larvae and pupae or to prevent female gadflies from attacking horses. Since the spread of pathogens can occur with horses coming from disadvantaged farms, it is necessary to carry out preventive treatment during the quarantine period with means that kill the larvae described above.

GASTROPHYLOSIS OF UNGULATES. Gastrophylosis is a widespread chronic disease of horses and donkeys caused by larvae of gastrointestinal gadflies and characterized by inflammatory processes in the places of attachment of larvae and the general pathological condition of the body.Gastrointestinal gadflies belong to the family Gastrophilidae, and the genus Gastrophilus, and species: G.intestinalis – large gastric gadfly, G.veterinus-duodenum, G.haemorrhoidalis-barbel, G.pecorum-herbivore, G.inermis –small gastric gadfly, anchor, G.nigricornis - chernous, G.magnicornis – big–moustached gadfly, goloshei, G.flavipes - donkey gadfly.

Morphology and biology of pathogens. The large gastric gadfly is large, yellowish-brown in color. The head is large, covered with hairs. There are faceted eyes on its sides, and 3 more simple eyes on the crown. The midrib is dark, covered with light yellow or brownish hairs. The wings are transparent, with a pattern of dark spots with light veins. The legs are slender, brownish-yellow. The abdomen is covered with hairs, brownish-yellow above, with dark spots. Eggs are yellowish, transversely striated, large - up to 1,25 mm long, wedge-shaped.

Larvae of stage I at birth are 1,05-1.1 mm long, white, spindle-shaped. Head armament – 2 movable curved hooks and a median point. A mouth opening is located down between the hooks. Stage II larvae are up to 16 mm long. The body is slender,

pointed in front, slightly expanding back. The posterior spiracles have the shape of rounded plates with two slits. Larvae of stage III are oval-cylindrical, up to 20 mm long.The segments of the larva's body from the 2nd to the 10th are covered with two rows of thorns, larger in the first row. The posterior spiracles are in the form of rounded plates with three slits.

Larvae of the large gastric gadfly parasitize horses and donkeys, among other types of gastric gadflies, it is the most numerous and harmful. The average life expectancy of the imago is 10-20 days, the fertility of females is from 887 to 1052 eggs. Immediately after hatching, it sits on elevated places, sun-sanctified rocks and plants, or flies without attacking horses. His behavior changes after mating and females start looking for horses to lay eggs. Years are marked here, in the south – for a longer time, starting from the end of June to September-October months.

Egg laying takes place on the fly. The female attaches one egg to the host's hairline in those places where he can reach with his teeth, in particular on the front legs, shoulders and sides. Externally, horses do not react to the approach of a gadfly. The number of eggs laid per animal reaches 3000-5000. The development of larvae in eggs lasts 7-16 days, but their hatching does not occur immediately. They can remain viable in an egg for 40-50 or even 90 days. In order for the larva to come out of the egg, it is necessary to influence a number of factors: humidity, heat and the touch of a post-root object. Such conditions are created when combing the places of attachment of eggs with teeth. In this case, the lid of the egg bounces off and the larva crawls out.

The larvae that have emerged from the eggs enter the oral cavity of the horse, attach to the mucous membrane of the tongue and develop in it for 21-28 days, after which they molt and pass into stage II. The development of larvae of stages I and II occurs in the stomach. Only isolated instances of them are found in the duodenum and esophagus. In the stomach, they attach their strong screw hooks to its left cardiac part. In the spring of the following year, the mature larvae of stage III leave the stomach and, together with the experiments, go outside. Pupation takes place in the faeces or on the surface layer of the earth. Depending on the temperature, the pupal phase lasts 18-52 days, after which an adult gadfly hatches from it.

Epizootological data. The source of the invasion is sick horses. The spread of pathogens of the disease occurs after the acquisition of a horse from a disadvantaged area and when larvae fall out from transported horses.

Pathogenesis. The larvae of gadflies that have penetrated into the oral cavity injure the mucous membrane, cause inflammation and swelling of tissues. When larvae are localized in the pharynx and root of the tongue, the act of swallowing may be disrupted. In the stomach and intestines, the larvae form crater-like depressions, the tissue thickens due to chronic irritation, many small ulcers form, gastric and intestinal hemorrhages may occur. With a high number of larvae, the secretory and motor functions of the stomach and intestines change.

Larvae of the small gastric gadfly, usokleya and herbivore, parasitizing in the posterior gastrointestinal tract, cause catarrhal inflammation of the rectum. Through injuries, the penetration of microflora and the occurrence of infectious diseases are possible. Larvae cause intoxication of the body by metabolic products.

The symptoms of the disease with a small number of larvae are hardly noticeable. With high invasiveness, the animals are emaciated, the coat is ruffled, without shine, the mucous membranes are anemic, appetite is sharply reduced, chronic gastroenteritis. If it is difficult to evacuate food from the stomach and patency in the duodenum, colic occurs. When the larvae are localized in the pharynx and in the area of the soft palate, a cough appears, water flows out of the nose during watering, difficult chewing and swallowing of food. Larvae of chernous and small gastric gadfly, migrating in the skin of the cheeks, cause dermatitis, folds and ulcers on the lips. Larvae of anchovy and usokleya with significant accumulation can cause rectal prolapse.

Pathoanatomic changes. In the early periods of larval parasitism, a large number of larvae form on the gastrointestinal mucosa. In places of their accumulation, multiple crater-shaped depressions in the mucous membrane are clearly visible, its hyperemia and swelling, under the mucous layer is hyperplasized. In some places, large ulcers form with tissue damage up to the muscle layer. Numerous ulceration in the same places sometimes lead to keratinizing stomach cancer.

Diagnosis. It is possible to predict the infection of horses with larvae of gastric gadflies in the summer-autumn period by finding their eggs on the hairline. Gadfly larvae can be detected visually by examining the oral cavity and pharynx. In winter and early spring, the diagnosis of infection can be made by detecting larvae in the feces after giving 40-80 mg / kg of chlorophos in an aqueous solution to horses, causing death and mass isolation of larvae. Allergic and serological methods of diagnosis of gastrophylosis are proposed. The most accurate diagnosis can be made with a pathoanatomical autopsy of fallen horses.

Treatment. To combat gastrophylosis, chlorophos is used at the rate of 40 mg / kg. Irrigate with a 5% solution of chlorophos grain fodder or hay for 1 kg per animal and feed to horses. A group method of feeding chlorophos with snow for 10-12 hours has been developed. The drug is dissolved in hot water, sprayed into feeders on snow and mixed. Before feeding, horses are kept without water and snow for a day. Free group drinking of 0.1% aqueous solution of chlorophos is also recommended for horses. Medicinal feed pellets containing 1% chlorophos are used. They are fed to adult horses at the rate of 1-1.2 kg, to young animals – 0.5 kg, once.Preparations of vivomek, ivermectin, baymek, rustomectin, PIVSA, bravomethin and others have high efficiency against all stages of larvae. These drugs are administered subcutaneously at a dose of 0.2 mg per kg of live weight according to ADV, once.

To destroy the larvae of gastric gadflies, horses are injected with trolen at a dose of 90 mg / kg and amidophos at a dose of 48 mg / kg through a probe. Anti-gadfly treatment of horses in our republic with two generations of gadflies at the end of July-August and October-November.

Prevention provides for the release of animals from larvae and prevention of the spread of infestation with movable horses.

MATERIALS FOR THE LABORATORY LESSON.

TOPIC No. 1. LIFETIME DIAGNOSIS OF HELMINTHIASIS.

The purpose of the classes: To teach students methods of in-life diagnosis of helminthiasis (quantitative and qualitative helminthoprological methods, intensity and extensiveness of invasion, helminthoscopic methods (methods of native smear, Fulleborn, Kalantaryan, Shcherbovich, Darling), helmintholaroscopy (methods of Berman-Orlov, Vida, simplified method of helmintholaroscopy, immunobiological reactions).

Materials and equipment. Mmcroscope, premed glass, cover glass, solutions: sodium chloride, sodium nitrite, magnesium sulfate, glycerin, various volumetric cups, metal loop, tweezers, scissors, scalpel, cotton wool, gauze, animal fecal samples.

Helminthoprological diagnostics is the examination of animal feces for the presence of helminths and their fragments.

A). Macrohelminthoscopy

1. Sequential washing method (deposition method). A small portion of feces (5-10 g) is mixed with 10 times the amount of water. The mixture is settled for 5 minutes, after which the liquid layer is drained, and a clean portion of water is added to the sediment and again it settles for 5 minutes and so on until the top layer of liquid becomes transparent. Then the liquid is drained, and the sediment is examined under a microscope for the presence of fluke eggs. This method is used to diagnose fasciolosis and other trematodoses.

B). Helminthoscopy

1. The native smear method is the simplest method of helminthic removal of feces. It can be used in any conditions, in pets of all kinds. The technique of his Jacob. A small (about the size of a pea) piece of feces is taken with a glass or wooden stick, placed on a slide, 2-3 drops of a mixture of equal parts of glycerin and water are added, thoroughly mixed. After removal of solid particles, the contents are covered with a cover glass and examined under a microscope. Instead of glycerin, you can take a drop of plain water. Glycerin enlightens the drug, which facilitates the study and protects from rapid drying. It is recommended to prepare 2-3 preparations from one animal at the same time. The disadvantage of this method is that with a weak invasion, it gives a large percentage of negative results. This method can be used to examine feces for strongylidosis, ascariasis, fascioliasis, trichocephalosis, etc.

2. Flotation methods. a). The Fulleborn method. Technique of the method execution. To perform this method, we need a saturated solution of table salt, for which 350-400 g (380 g) of salt is dissolved in 1 liter of water, heated to a boil and filtered through cotton wool or gauze. The solution is used cold, its density is 1.18.

Take 10-20 g of feces and place it in a jar or cup with a capacity of 100-200 ml and carefully rub it with a glass or wooden stick in a saturated solution of table salt. The solution is poured gradually, stirring the feces all the time, and the total amount of the added solution should be about 20 times more than the amount of feces. Then the liquid is filtered through a metal sieve and set aside for 30-45 minutes, during which time it will float to the surface, since the saturated salt solution has a higher density than the egg. A film is removed from the surface of the exfoliated liquid with

a metal loop (no more than 1 cm in diameter) bent at a right angle, transferred to a slide and covered with a cover glass and examined under a microscope.

The Fulleborn method is recommended to be used mainly for the detection of eggs of round helminths and partially tapeworms (tenium, anaplocephalid).

b). The Shcherbovich method. The Shcherbovich method is used to detect eggs with a higher density (metastrongilid). To perform this method, we need a saturated solution of magnesium sulfate salt, for which 920.0 g of magnesium sulfate is dissolved in 1 liter of water, heated to a boil and filtered through cotton wool or gauze. The solution is used cold, its density is 1.45. The technique of performing the method is the same as Fulleborn.

c). The Kalantarian method. To perform this method, we need a saturated solution of sodium nitrate salt (saltpeter), for which 1 kg of saltpeter is dissolved in 1 liter of water, heated to a boil and filtered through cotton wool or gauze. The solution is used cold, its density is 1.4. The technique of performing the method is the same as Fulleborn.

d). The Darling method. The Darling method combines deposition and flotation procedures. Feces are mixed with water to a semi-liquid consistency and centrifuged for 3-5 minutes, as a result of which helminth eggs are deposited on the bottom. Then the liquid from the test tube is drained, and the Darling liquid (glycerin mixed in equal parts with a saturated solution of table salt) is added to the precipitate. The sediment is thoroughly stirred and centrifuged a second time for 3-5 minutes and after that the eggs of parasitic worms from the sediment float to the surface. With a metal loop, the film is removed on a slide, covered with a cover and examined under a microscope.

C). The method of helmintholaroscopy is the study of animal feces to detect helminth larvae.

1. The Vida method. Several balls of freshly extracted sheep and goat feces are placed on the slide or watch glass and a small amount of water is added at a temperature of about 400C. If the balls interfere with the slide, then a few drops of water are enough. After 40 minutes, the balls are removed, the remaining liquid on the glass is examined under a microscope for the presence of nematode larvae. The technique is effective if the feces are dense. It is used for the diagnosis of dictyoculosis, mulleriosis, protostrongylosis, cystioculosis of sheep and goats.

2. The Berman-Orlov method. Fecal samples (10 g) will be placed in the funnels of the Berman apparatus on a metal mesh or wrapped in pieces of gauze. Pre-funnels are filled with water at room temperature for 3-6 hours. During this time, the larvae of dictyocauluses crawl out of the sample into the liquid and descend through the tube to the bottom of the test tube. Then carefully disconnect the rubber trunks and with a quick movement, without shaking the sediment, drain the liquid from the test tube (you can suck out the water with a pipette). Test tubes are placed in a tripod. After shaking, the precipitate is poured onto slides and microscopized at low magnification. Nematode larvae are mobile, easily detected.

3. Simplified helmintholaroscopy method. Samples wrapped in gauze napkins are placed in cups of water. After 3-6 hours, the samples are taken out, the

liquid is defended for 10-15 minutes, after which the cups are filled and a transparent layer of liquid is sucked out of them with a pipette until the sediment begins to be absorbed. Then droplets of sediment are pipetted onto a slide for microscopy. If the sediment turns out to be thick, then water is poured into the cup and shaken, then stand for 10 minutes, after which the top layer of liquid is drained.

4. Sedimentation method with centrifugation (express method). Fecal samples (3-5 g) from sheep and goats are placed in test tubes with water at a temperature of 20-220C. The tubes are centrifuged at a speed of 1000-1500 rpm for 2 minutes. Then the samples are taken out with tweezers, the water is drained to a precipitate, and the precipitate, after shaking, is poured onto a slide and examined under a microscope for the presence of larvae of dictyocauluses, mullerius and other proto-trongylids.

Immunobiological diagnostics. Immunobiological diagnosis of helminthiasis in both veterinary and medical practice has not yet been widely used:

firstly, immunologists have not yet developed a technology for the production of appropriate antigens, the use of which would make it possible to obtain a specific reaction to a particular helminthiasis;

secondly, there is no generally accepted opinion on the most sensitive diagnostic reactions in helminthiasis;

thirdly, the technology and methods of cultivating helminths on artificial nutrient media have not been developed in order to obtain the necessary raw materials for the production of antigens.

Despite this, in practice, some reactions are used to diagnose so-called tissue helminthiasis-echinococcosis, cenurosis, cysticercosis, trichinosis, etc. To do this, RA, RSK, RNGA, RDID, RSkP, Kazoni reaction, Ronzhin method (KazNIVI) are used.

Security questions:

1. What is the helminthic method?
2. Tell us the method of sequential washing?
3. Tell us the method of native smear?
4. Tell the Fulleborn method?
5. Tell the Darling method?
6. Tell the Shcherbovich method?
7. Tell the Kalantarian method?
8. Tell the Berman-Orlov method?
9. Tell the simplified helmintholaroscopy method?
10. Tell us the method of sedimentation with centrifugation?
11. Tell us the method of a complete helminthological autopsy?

TOPIC No. 2. POSTMORTEM DIAGNOSIS OF HELMINTHIASIS.

Materials and equipment. Mmcroscope, premed glass, cover glass, solutions: sodium chloride, sodium nitrite, magnesium sulfate, glycerin, various volumetric cups, metal loop, tweezers, scissors, scalpel, cotton wool, gauze, animal fecal samples.

Postmortem diagnosis of helminthiasis consists in the detection of helminths of various stages of their development in the organs and tissues of the animal. Helminths parasitize in all organs and tissues of the animal body. Therefore, their collection and further determination is provided by special autopsy methods, which differ from the usual pathoanatomical autopsy of corpses. The most advanced technique of helminthological autopsies was developed by academician K.I. Scriabin.

There are:

- complete helminthological autopsy;
- incomplete helminthological autopsy;
- complete helminthological autopsy of individual organs.

A complete helminthological autopsy according to Academician K.I. Scriabin is the most reliable method that allows both quantitative and qualitative accounting of all helminths with which the animal is invaded. This method consists in the following: after removing the skin from the corpse, the subcutaneous patch is carefully examined, then the thoracic and abdominal cavities are opened and all organs of the systems are extracted: digestive, respiratory, circulatory, genitourinary, etc.

The organs of a particular system are separated and examined separately, while using the method of successive flushes. All tubular organs are opened along their length, the contents are placed in a basin, bucket or jar (depending on the volume of the organ), scraping is done from the mucous membranes, and the wall of the opened (sometimes) is viewed under the compressor.

Parenchymal organs (liver, lungs, pancreas, kidneys and others) are placed in a separate dish and turned into detritus (minced meat), torn by hands or cut into small pieces with a knife or scissors. Detritus, scrapings, the contents of organs are washed with water or saline solution, using the method of successive flushes. The obtained materials (sediments) are studied in small portions, first in black and then in white cuvettes or in Petri dishes on a black and white background. Large helminths are selected visually, and small ones are selected using a manual magnifying glass with 8-10–fold magnification. Helminths are collected only with brushes or preparation needles, but not with tweezers and not with fingers.

Incomplete helminthological autopsy. Incomplete helminthological autopsy is a simplified helminthological autopsy, in the process of which only individual helminths, sharply distinguished in size, are extracted from organs and tissues. Such an autopsy is of great importance both for the diagnosis of helminthiasis and for obtaining museum material.

Complete helminthological autopsy of individual organs. A complete helminthological autopsy of individual organs is carried out if it is necessary to have accurate data on the location of a particular helminth. For example, with fascioliasis, only the liver is examined, with dictyoculosis – the lungs, with ioniosis – the small

intestine, etc. This method is more practical, simple and quite accurate. These methods are most often used by students, scientific and practical specialists to test the effectiveness of new anthelmintics or to study the refinement of the biology of helminths.

Security questions:

1. When testing the effectiveness of new anthelmintics, a graduate student or doctoral student uses which autopsy method?
2. For the preparation of museum material, what method of autopsy of animals is necessary?
3. What is the difference between the method of complete helminthological autopsy from the method of complete helminthological autopsy of individual organs?

TOPIC No. 3. SYSTEMATICS, MORPHOLOGY AND BIOLOGY OF TREMATODES.

The purpose of the classes: To teach students the morphology and biology of trematodes, anatomical and morphological structure, biological development of Fasciola hepatica and F.gigantica, methods of diagnosis of fascioles and its differentiation from other trematode diseases, treatment, prevention and measures to combat fascioles of animals.

Materials and equipment. Water, bucket, liver of cattle or mrs, microscope, slide, cover glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, samples of anthelmintics,

Morphology of trematodes. Trematodes are parasitic worms belonging to the type of flatworms - Plathelminthes, class Trematoda – flukes. They are localized in various organs and tissues of animals and humans (liver, pancreas, pancreas, blood vessels, oviduct, sacrum). The body of the trematodes is flattened in the dorsoventral direction, their shape is leaf-shaped, pear-shaped, sometimes even approaching nematoid. The size of trematodes varies from fractions of a millimeter to several tens of centimeters, but most of them are from 0.1 mm to 15-20 mm in length. The body is covered with cuticles (skin). In some trematodes, the body is smooth, and in some trematodes, the body is covered with spines, which has a differential value for determining the type of trematodes. At the anterior end of the fluke body there is an oral sucker, and at the bottom of its mouth opening, passing into the pharynx (pharynx) and esophagus. The intestine consists of two blindly ending trunks, there is no anal opening, the remains of undigested food are thrown out through the mouth. On the ventral surface of the body there is an abdominal sucker - a fixation organ.

The excretory system is represented by a complex network of tubules ending in an excretory tube with an opening in the back of the parasite's body. The nervous system consists of nerve nodes lying under the glottis and nerve trunks extending to other parts of the body.

The reproductive system of trematodes is well developed, it is very difficult to build. Trematodes (with the exception of the order Schistosomatata) are hermaphordites, i.e. both male and female genitalia are located in the same body.

The male reproductive apparatus includes two testes (Testis), from which one Vas efferenz (Vas efferenz) departs, they connect with each other and form a common vas deferens (Vas deferens). It is usually enclosed in a special muscular sac – the genital bursa and opens with an external male opening on the ventral surface of the body of the fluke. The final part of the vas deferens is the copulatory organ (Cirrus). In the genital bursa there are prostatic glands (Glandule prostatae), they surround the vas deferens.

The female reproductive apparatus includesthe ootype (Ootyp), where fertilization and egg formation take place. The ovary (Ovarium) is connected to the ootype through the oviduct, which secretes germ egg cells, as well as the ovary. In addition, the ootype communicates with yolk cells that produce nutritious material for eggs. Often, the ootype communicates with the ventral surface of the body through the laurel canal (canalis Laureri). Excess yolk cells are removed through this channel, and sometimes it serves for copulation, playing the role of a vagina. The uterus has the appearance of a convoluted tube. One end connects it to the ootype, the other communicates with the external environment through the external female opening. Special glands (the body of Melissa) secrete fluid that washes the ootype and uterus and facilitates the free sliding of eggs along the genital tract to the opening of the uterus. The uterus also performs the function of the vagina. External genital openings are located in different representatives of suckers in very different parts of the body, most often along the midline of the body, in the gap between the suckers.

Biology of trematodes. All flukes are biohelminths, i.e. they develop with the participation of two or three hosts. The first intermediate hosts of trematodes are mollusks, both aquatic and terrestrial (land). In the development of many trematodes, in addition to the intermediate host, additional hosts also take part, which may be fish, amphibians, insects or mollusks. Fertilized eggs of flukes are covered with a shell, which has four shells: three outer ones protect the embryo from mechanical damage, and the fourth inner one from chemical influences.

In the future, a miracidium is formed in the egg, which either comes out of the egg, actively attacks the corresponding mollusk and invades its tissues, or is swallowed by the mollusk. In the mollusk, the miracidium develops into the next

larval stage – the maternal sporocyst (it is a bag filled with germ cells), the latter parthenogenetically gives rise to the next generations – daughter sporocysts, or redia. Cercariae are formed from redia.If the fluke develops with the participation of a single host, then the cercariae, coming out of the mollusk, lose their tail, encyst and pass into the invasive stage – the adolescarium. Trematode cercariae, in the development cycle of which there is an additional host, actively or passively penetrate into it, encyst and develop into invasive metacercariae. In the body of a definitive host, the shell of invasive larvae is destroyed and young flukes reach the place of parasitization in various ways, develop into marita.

Systematics of trematodes. According to the taxonomy, the trematodes are arranged as follows: Type- Plathelminthes, class- Trematoda, subclass- Digenia, orders - Fasciolata, Paramphistomatata, Heterophyata, Echinostomatata and Schistosomatata.

Topic 4: FLUKE PATHOGENS AND THEIR DIAGNOSTICS.

The purpose of the lesson: to study the structural features of the causative agents of trematodoses (fascioliasis, paramphistomatosis, dicroceliosis, opisthorchiasis) and their eggs.

Tasks: Implementation of zoohygienic, preventive and veterinary-sanitary measures.

Methods of zoohygienic, preventive and veterinary-sanitary measures.

Name of the work: determination of anatomical and morphological signs of the sexually mature stages (marit) on preparations and drawings and eggs of fascioles, paramphistomatids, dicrocelium and opisthorchis; familiarization with intermediate and additional hosts of trematodes.

Acquired skills: students will be able to diagnose and differential diagnose fascioliasis, paramphistomatosis, dicroceliosis, opisthorchiasis by anatomical and morphological signs of pathogens and the structure of trematode eggs.

Workplace: laboratory "Parasitology and invasive diseases".

Workplace equipment: microscope, stained total preparations of fascioles, paramphistomatides, dicrocelium; micro-preparations of fasciole eggs; samples of shells of freshwater and land mollusks – intermediate hosts of trematodes; drawings on the morphology of trematodes.

Workplace safety regulations: according to the laboratory instructions.

Sources of information:1. Akbaev M.Sh. et al. Parasitology and invasive diseases of farm animals, pp. 104-106.

2. Shevtsov A.A. et al. Parasitology, pp. 66, 91, 94-95.

3. Internet resources:

http://studopedia.ru/6_142347_klass-lentochnie-chervi--Cestoidea-.html

http://medbookaide.ru/books/fold9001/book1010/p11.php

Security questions:

1. List the characteristic (differential) signs of the sexually mature stages (marit) of fascioles, paramphistomatids, dicrocelium and opisthorchis.
2. What signs are common to fluke eggs?

TOPIC № 5. SYSTEMATICS, MORPHOLOGY AND BIOLOGY CESTOD.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of cestodes, their larval form and their differentiation.

Materials and equipment. Water, bucket, microscope, slide, cover glass, liver damage, lungs and other parenchymal organs of cattle and sheep, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, cestode samples.

Morphology of cestodes. Cestodes belong to the type of flatworms - Plathelminthes, class – Cestoda - tapeworms. Representatives of two orders have veterinary and sanitary significance: the chainworms - Cyclophyllidea and the lentets - Pseudophyllidea.

In the ribbon stage, cestodes live in the digestive organs of vertebrates.

The body of the cestode is usually ribbon-shaped, flattened in the dorsoventral direction, consists of a head (scolex), a neck and a strobila consisting of segments (proglottids). The length of the entire cestode varies in individual species from a few millimeters to 10 meters or more, and the number of proglottids can be from one to several thousand, depending on the type of cestode.

Scolex in the form of a compact formation has a different shape, size, structure. The scolex chains are usually more or less rounded in shape, with 2-4 suckers with muscular walls, which can be armed with hooks. At the top, the scolex is often equipped with a special muscular outgrowth- a proboscis, carrying weapons in the form of one or more rows of hooks. The number, size and shape of hooks, the ratio of the length of individual morphological elements (blades, handles and root process), as well as the nature of the location of hooks has a differential value for the diagnosis of chains. The scolex has an elongated shape and is equipped with two (or four) suction slits (botryas). Behind the scolex there is an unsegmented part of the body – the neck-the growth zone in which the segments are formed. As new segments begin to form from the neck, older segments gradually move back.

The cestodes have well-developed nervous, excretory and reproductive systems, but there is no respiratory, circulatory and digestive system.

The nervous system of the cestode consists of a central nerve node embedded in the scolex and longitudinal nerve trunks extending from it, which pass through the entire length of the cestode and are interconnected by transverse branches. The excretory system is built in the same way as in flukes. Tapeworms do not have a digestive system.

The body of the cestode covers the musculoskeletal layer consisting of cuticle, basement membrane and subcuticle. The muscle layer is located under the subcuticular layer, and the entire space between the internal organs is filled with parenchyma. The cuticle is a morphological analog of the intestinal tissues of higher animals and performs the functions of digestion, secretion and absorption.

The sexual system. Cestodes are hermaphrodites, in each segment there are male (testes, vas deferens, vas deferens, cirrus, genital bursa) and female (ovaries, oviduct, yolk, Melissa body, ootiv, vagina, uterus) reproductive systems. The youngest segments are initially asexual; then the rudiments of the male reproductive system appear in them. In the future, female sex glands develop in the same segments, as a result, the segment becomes hermaphrodite. After fertilization, the male sex glands degenerate, and the female ones reach their maximum development. Finally, in the most posterior segments, all the female glands disappear, with the exception of the uterus filled with eggs, which occupies the entire proglottis. Such a segment is already called female.

In tapeworms, the uterus is in the form of a looped channel communicating with the external environment by an outlet opening, and in chainworms - a longitudinal or transverse trunk with lateral diverticula, sometimes it can be sac–shaped, mesh-like or breaks up into separate sections - capsules.

Many representatives of tapeworms in each segment have a sexual apparatus in a double set, and the sexual openings are located either on the sides of the proglottids, or medially on their surface.

Cestode eggs are constructed differently. In the representatives of the order of the idlers, they are similar to the eggs of flukes. They are rounded, spherical or oval in shape, their shell is transparent and very delicate, contains an embryo – oncosphere with three pairs of embryonic hooks inside, which has its own radial striated shell –an embryophore.

In the organism of intermediate hosts, larval forms in various cestodes have various types of structure. The main types of these larvae in the tapeworms are: cysticercus, cysticercoid, cenurus, echinococcus, alveococcus, strobilocercus, dityridium.

Cysticercus (Cysticercus) is a bubble with a single embryonic scolex, armed with chitinous hooks or without them.Its size and shape varies depending on the type of cestode, age and location of the larva in the body of the intermediate host. Cysticerci usually live in mammalian animals.

A bubble filled with liquid, in which there is not one, but many scolexes, is called a Coenur, which is also common in milk-feeders.

Echinococcus (Echinococcus) is a bubble of complex structure filled with liquid. The inner germinal shell can produce brood capsules with the simultaneous formation of germinal scolexes and secondary (daughter) bladders in them. Echinococcus in the body of an intermediate host takes various morphological modifications.

Alveococcus (Alveococcus) is a conglomerate of small, irregularly shaped bubbles that do not contain liquids, in which germinal scolexes in the form of small dots can be seen

In some cestodes, the larval form forms an anterior swollen part with an invaginated scolex and a posterior one in the form of a caudal appendage, where embryonic hooks are located. This type of larva is called a **cysticercoid (Cysticercoid),** it usually develops in the body of invertebrate intermediate hosts.

Strobilocercus (Strobilocercus) has a well-defined, armed with large hooks, scolex, with a long false-jointed strobila extending from it, ending at the posterior end with a small bubble filled with liquid. Strobilocerci usually live in rodents.

The ditiridium is equipped with an unarmed scolex with four suckers and a relatively long caudal appendage without a caudal vesicle. It lives in the body cavities of rodents.

Two parasitic phases are distinguished in the life cycle of lentils – procercoid and plerocercoid.

Procercoid (Procercoid) – the body is elongated, 0.5-0.6 mm. At the anterior end there are primary botria (depressions), and at the posterior there is a cercomere – a spherical appendage with embryonic hooks. Procercoid inhabit intermediate hosts – crustaceans, mainly oarfishes.

Plerocercoid (Plerocercoid) is a larval stage of tapeworms, reaching 1 m in length, with two botryas at the anterior end of the body. It is localized in an additional host – in various species of fish, amphibians in the body cavity, muscles, musculature, liver and other organs.

Biology of cestodes. All cestodes, as a rule, are biohelminths, they develop with the help of one (chain) or two (lazy) intermediate hosts.

Tsepni, as noted above, develop with the participation of two hosts: definitive and intermediate. An embryo is released from oncospheres that have got into the gastrointestinal tract of an intermediate host with food or water, which migrates with blood, getting into various internal organs, where, depending on the type of cestode, the corresponding type of larva develops. Some of these larvocysts are able to reproduce asexually. Definitive hosts become infected by eating organs or tissues or by ingesting intermediate hosts containing an invasive larvocyst.

And as for the sluggards, their development can be reduced to five phases: 1 – eggs, the embryogenesis of which occurs in water; 2 – coracidium – hatching from an egg and leading a free–swimming lifestyle; 3 – procercoid, developing from coracidium in the body of oar-footed crustaceans; 4 - plerocercoid, developing from a procercoid in fish and a 5–adult cestode developing from a plerocercoid in the intestines of warm-blooded animals.

The causative agents of cestodoses of animals belong to five suborders of chains. These are: Teniata(Taeniata), Anoplocephalata (Anoplocephalata), Hymenolepidata (Hymenolepidata), Daveneata (Davaineata) and Mesocestoidata (Mesocestoidata).

SECURITY QUESTIONS:

1. What type and class do cestodes belong to?
2. Which detachments of cestodes have veterinary and medical significance?

3. How do these two detachments differ in morphological structures?
4. How do these two detachments differ in biological development?
5. The body of the cestode consists of how many parts?
6. What function do cuticles perform in cestodes?
7. Tell us about the biological development of the cestode?
8. Tell us the main stages of the larval form in the chains in the body of intermediate hosts?
9. Tell us the main stages of the larval form in the lentets in the body of intermediate hosts?
10. What is the difference between the eggs of the chain squad and the Lenzets squad?

TOPIC № 6. LARVAL FORMS OF CESTODES AND THEIR DIAGNOSTICS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of cestodes, their larval form and their differentiation.

Materials and equipment. Water, bucket, microscope, slide, cover glass, liver damage, lungs and other parenchymal organs of cattle and sheep, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, cestode samples.

Cysticercus (Cysticercus) is a bubble with a single embryonic scolex, armed with chitinous hooks or without them.Its size and shape varies depending on the type of cestode, age and location of the larva in the body of the intermediate host. Cysticerci usually live in mammalian animals.

A bubble filled with liquid, in which not one, but many scolexes, is called a **Coenur (Coenur),** which also inhabits milk-feeders.

Echinococcus is a bubble of complex structure filled with liquid. The inner germinal shell can produce brood capsules with the simultaneous formation of germinal scolexes and secondary (daughter) bladders in them. Echinococcus in the intermediate host body takes various morphological modifications.

Alveococcus is a conglomerate of small, irregular shaped bubbles that do not contain liquids, in which germinal scolexes in the form of small dots can be seen

In some cestodes, the larval form forms an anterior swollen part with an invaginated scolex and a posterior one in the form of a caudal appendage, where embryonic hooks are located. This type of larva is called a cysticercoid (Cysticercoid), it usually develops in the body of invertebrate intermediate hosts.

Strobilocercus (Strobilocercus) has a well-defined, armed with large hooks, scolex, with a long false-jointed strobila extending from it, ending at the posterior end with a small bubble filled with liquid. Strobilocerci usually live in rodents.

The ditiridium is equipped with an unarmed scolex with four suckers and a relatively long caudal appendage without a caudal vesicle. It lives in the body cavities of rodents.

Two parasitic phases are distinguished in the life cycle of lentils – procercoid and plerocercoid.

Procercoid (Procercoid) – the body is elongated, 0.5-0.6 mm. At the anterior end there are primary botryas (depressions), and at the posterior there is a cercomere

– a spherical appendage with embryonic hooks. Procercoid inhabit intermediate hosts – crustaceans, mainly oarfishes.

Plerocercoid is a larval stage of the tapeworms, reaching 1 m in length, with two botryas at the anterior end of the body. It is localized in an additional host – in various species of fish, amphibians in the body cavity, muscles, musculature, liver and other organs.

Biology of cestodes. All cestodes, as a rule, are biohelminths, they develop with the help of one (chain) or two (lazy) intermediate hosts.

Tsepni, as noted above, develop with the participation of two hosts: definitive and intermediate. An embryo is released from oncospheres that have got into the gastrointestinal tract of an intermediate host with food or water, which migrates with blood, getting into various internal organs, where, depending on the type of cestode, the corresponding type of larva develops. Some of these larvocysts are able to reproduce asexually. Definitive hosts become infected by eating organs or tissues or by ingesting intermediate hosts containing an invasive larvocyst.

And as for the sluggards, their development can be reduced to five phases: 1 – eggs, the embryogenesis of which occurs in water; 2 – coracidium – hatching from an egg and leading a free–swimming lifestyle; 3 – procercoid, developing from coracidium in the body of oar-footed crustaceans; 4 - plerocercoid, developing from a procercoid in fish and a 5–adult cestode developing from a plerocercoid in the intestines of warm-blooded animals.

The causative agents of cestodoses of animals belong to five suborders of chains. These are: Teniata(Taeniata), Anoplocephalata (Anoplocephalata), Hymenolepidata (Hymenolepidata), Daveneata (Davaineata) and Mesocestoidata (Mesocestoidata).

Security questions:

1. What type and class do cestodes belong to?
2. Which detachments of cestodes have veterinary and medical significance?
3. How do these two detachments differ in morphological structures?
4. How do these two detachments differ in biological development?
5. The body of the cestode consists of how many parts?
6. What function do cuticles perform in cestodes?
7. Tell us about the biological development of the cestode?
8. Tell us the main stages of the larval form in the chains in the body of intermediate hosts?
9. Tell us the main stages of the larval form in the lentets in the body of intermediate hosts?
10. What is the difference between the eggs of the chain squad and the Lenzets squad?

TOPIC № 7. DIAGNOSIS OF CATTLE AND PIG CYSTICERCA.

Cysticercosis (finnosis) of cattle is a disease of anthropozoonosis, cestodosis, which occurs in subclinical manifestations, which is caused by the fact that the larval form of Taeniarhynchus saginatus parasitizes Cysticercus bovis on the transverse – propagating muscles, heart, tongue, diaphragm of animals, the disease is characterized by the return of an allergic response to the organism.

Anatomy - morphology of the causative agent. Cysticercus bovis is a round-oval-shaped blue-whitish-pubescent larva of a whitish hue, with a clear liquid inside. The length of the cysticercks is 5-15 mm, and the width is 3-8 mm (7.5-9x5.5 mm). In the inner germinative shell is located in a state enclosed in a single unarmed scolex. The diameter of the scolex is 1.5-2 mm, armed with 4 muscular suction cups.

Development of the pathogen. The parasite is a biogelmint, the main master is people, and the intermediate master is cattle, the Buffalo zebular, the reindeer of the North. The duration of the parasite's sexual maturity is on average 3 months, and the duration of parasitizing is on average 10 years. The duration of reaching the stage of invasion of cysticercks is 4-4.5 months.(The biological development of the pathogen is fully stated in the lecture).

Pig cysticercosis (finnosis) is a disease of anthropozoonosis, cestodosis, which is caused by parasitizing on subclinical vision of the larval form of Taenia solium cestode Cysticercus cellulosae in the transverse – propagating muscles of animals, tongue, heart, head brain, eyes, lungs and liver, characterized by clinical manifestations depending on the location of the causative agent of the disease.

The structure of the pathogen. Cysticercus celulosae is an ellipsoid-shaped vesicle filled with clear liquid inside, the length of the vesicle is 6-20 mm, the width is 5-10 mm, the wall is made up of two layers, located in one, two rows to the inner germinative Shell, one long, one short, with a total of 22-32 chitinous loops, armed with a scaly

Development of the pathogen. Parasite biogelmint. The main boss is a man, intermediate bosses are a pig, a wild boar, a bear, a camel, a dog, a cat, a rabbit, and a man. The period of maturity of Taenia solium is 2-3 months, the period of parasitization is several years, the period of reaching the stage of invasion of cysticercus is 2-4 months, the period of residence is 3-6 years (full biological development is presented and outlined in the lecture).

Diagnosis and differential diagnosis. Diagnosis of cysticercosis of cattle and pigs is made in a complex way: taking into account epizootological data, depending on the clinical signs of the disease (not characteristic, subclinical), depending on the result of immunobiological reactions (latexagglyutination – it also does not give much good effect) and is laid on the basis of finding cysticercus after the death of the air, or when it is forcibly slaughtered,

We must be able to distinguish cysticercosis from trichinellez, sarcocystosis diseases.

In cattle, at the initial stage of the disease, an increase in body temperature to 40-41 ° C, weakness, deterioration of appetite, diarrhea is noted, which sometimes stops after

a few days. Later, atony of the pancreas, chewing, pain in the muscles of the back and abdomen, an increase in the scapular and inguinal lymph nodes, rapid breathing and palpitations are observed. After 8-12 days, the clinical signs disappear, and the animals look healthy. In the event that the animal dies from acute cysticercosis, petechial hemorrhages and small cisterns in the affected muscles, handrail, peritoneum and spleen are found in large quantities.

In the chronic form of finnosis, skeletal muscles are gray, gelatinous and degenerate (reborn).

A positive RSK with radiographic data, CT, MRI, as well as eosinophilia of the blood and cerebrospinal fluid, cysticercosis antigen will help in a successful diagnosis. Cysticercosis should be distinguished from brain tumors, neurosyphilis, meningoencephalitis, epilepsy, among others.

In the prevention and fight against the disease, specialists in the field of Veterinary Medicine and medicine are required to carry out complex measures together, in cooperation.

Measures taken by veterinary professionals:

- Vet all slaughtered or forcibly leaned cattle and pigs ' meat and meat products.you.subject to examination;
- Slaughter animals only at special slaughterhouses,slaughterhouses under the supervision of Veterinary Specialists;
- Detoxification of the affected limbs with cysticercks;
- Notification to regional medical professionals in order to determine the source of the disease if animals infected with cysticercus are detected;
- Not to include uncooked meat in the diet of pigs;
- Strict adherence to the rules of keeping and feeding animals;

The activities carried out by medical professionals are as follows:

- To examine people (especially workers in cattle and pig farms) against diseases of teniarynchosis and teniosis according to plan. If, in case of detection of the disease, their rapid hospitalization and treatment;
- To check the availability of closed-type toilets and compliance with their sanitary condition;
- Regularly neutralize water sources against parasitic oncospheres according to plan;
- Compliance with personal hygiene;
-Regular implementation of propaganda work among livestock breeders.

Security questions:

1. What is the difference between cattle and Pig cysticercosis diseases?
2. What is the difference in the structure of cattle and Pig cysticercus?
3. What is the difference between Taeniarhynchus saginatus and Taenia soliums? How they differ biologically.
4. What is the difference between parasite eggs and oncosphere?
5. Why is pork cysticercosis much more dangerous than cattle cysticercosis?
6. Why can people act as both the main and intermediate hosts in Pig cysticercosis, comment?

7. Meat and meat products in cattle and Pig cysticercosis disease
vet.you.tell me your expertise?
9. Prevention of the disease performed by veterinary specialists
what are the measures to be taken?
9. What are the todbirs performed by medical professionals?
10. What are the tasks facing Veterinary Specialists and scientists in the treatment of the disease?

TOPIC № 8. DIAGNOSIS OF ECHINOCOCCOSIS AND CENUROSIS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of pathogens of echinococcosis and cenurosis, methods of lifetime and postmortem diagnostics, their differentiation, prevention and measures to combat these diseases.

Materials and equipment. Water, bucket, microscope, slide, cover glass, samples of affected and healthy carcasses of cattle and pigs, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations, anthelmintic samples.

ECHINOCOCCOSIS is a chronically or subclinically occurring anthropozoonous, cestodose disease of sheep, goats, cattle, pigs, camels, deer, horses, donkeys and other mammalian animals caused by Echinococcus granulosus larva, the larval stage of the Echinococcus granulosus cestode, family Taeniidae, parasitic in parenchymal organs, mainly in the liver and lungs, less often in other organs, characterized by an allergic manifestation of the body.

Systematics of the pathogen. The causative agent of echinococcosis according to systematics is arranged in the following order: type –Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Taeniata, family - Taeniidae, genus – Echinococcus 1. Larval stage - Echinococcus granulosus larva, 2, Ribbon stage - Echinococcus granulosus.

The pathogen.Echinococcus granulosus larva (E.unilocularis) is a single-chamber bubble filled with liquid. Echinococcal fluid is a product of the blood of an intermediate host, it plays the role of a protective and nutrient medium for scolexes. The snad\ruzhi bubble is covered with a soyidinative capsule from the host tissue. The wall of the bladder consists of two shells: the outer cuticular and the inner germinal.

The cuticle shell is milky white, sometimes with a slightly yellowish tinge, in older bladders it becomes cloudy, taking on an jaundiced appearance.

The germinative, or germinal, shell lining the inside of the bladder cavity, current, tender, is a filamentous tissue capable of producing brood capsules with the simultaneous formation of germinal scolexes and secondary bladders in them. Sometimes elements of this shell penetrate between the layers of the cuticle to the outer surface, which creates conditions for the exogenous development of secondary bladders.

Brood capsules represent outgrowths of the germinative shell, where germinal scolexes are formed in the future. Brood capsules, as well as individual scolexes, in some cases break off from the germinal shell and float freely in the cavity of the maternal bladder or gather in large quantities at the bottom of it, forming the so-called hydatid sand. Bubbles develop not only in the cavity of the maternal belly (endogenously), but also outside it (exogenously), they can completely bud off from the maternal bladder.

The size of echinococcal bubbles varies from millet grain to the head of a newborn baby. The shape of the bubbles is usually round, but depending on the localization, the number of bubbles in individual animals varies from single specimens to tens, hundreds and even thousands.

The tape stage. Echinococcus granulosus – this small cestode, reaching 0.5-0.6 (0.9) cm in length, consists of 3-5 segments, of which 1-2 are asexual, one is germinate, and the last is mature, filled with eggs. The mature segment is usually longer than the rest of the strobila. The Scolex is equipped with a proboscis armed with 36-40 hooks. The length of the hooks of the 1st row is 0.040-0.045 mm, the 2nd row is 0.030-0.039 mm. The sexual openings are located in the posterior half of the lateral edge of the segment. In mature segments there is a sac-shaped uterus in the form of a longitudinal trunk, with noticeable lateral protrusions. In one mature segment there are up to 700-800 eggs. The oncosphere, covered with a radially striated shell, has a diameter of 0.030-0.040 mm of a weak yellow color.

Biological development of the pathogen. The parasite is a biohelminth, develops with the participation of two hosts. The prepatent period of development, depending on the type of intermediate host, season of the year, sex, conditions of keeping and feeding animals from 31 days to 118 days, the patent period of 735 days (observation period), larvae reach the invasive stage, depending on the type of animal from 12 months to 24 months.

Diagnosis and differential diagnosis. The diagnosis is made on the basis of epizootological data, clinical signs, pathoanatomic changes and immunobiological (RA, RSC, RNGA, RDID, RID, Kazoni reaction, X-ray, ultrasound, tomography) methods

Kazoni reaction (allergic reaction). Dry factory powder echinococcal allergen is diluted at a physiological level of 1:750 and injected into the thickness of the skin of the upper eyelid or tail fold: allergen dose: for sheep -0.2 ml, 0.5-0.75 ml for cattle. With proper administration of the allergen at the injection site, a swelling the size of a small pea in sheep and a large pea in cattle is formed, Accounting for the reaction (measurement of swelling, caliper along its width without squeezing) is carried out 2-4 hours after injection. The established standard for assessing the reaction in sheep is as follows: with a swelling size of up to 2 cm, the reaction is negative; from 2.1 to 2.4 cm – doubtful and from 2.5 cm higher - positive. For cattle, it is as follows: with a swelling width of up to 3.5 cm, the reaction is negative, from 3.5 to 4.5 – doubtful and from 4.6 cm and higher - positive.

For an allergic reaction, you can also use a liquid taken sterically from fresh echinococcal bladders, preferably from sheep. This liquid is used fresh, stored in the

refrigerator at a temperature below 2-40, as well as canned (with the addition of 1% phenol).

Larval echinococcosis should be differentiated from cenurosis and alveococcosis.

Coenurosis (cerebral or "pinwheel") of sheep is an anthropozoonosis, a cestodosis disease of sheep, goats, cattle and other wild ruminants caused by the larval stage of Coenurus cerebralis – cestodes Multiceps multiceps, family Taeniidae, localized in the brain and less often the spinal cord and characterized by depression or excitement, reduction or refusal of feed, timidity, convulsive twitching, aimless abrupt movements, violation of the cooridation of movement, emaciation and death of the animal.

Systematics of the pathogen. The causative agent of cerebral cenurosis or "pinwheel" according to the systematics is located in the following way: type – Plathelminthes, class - Cestoda, order - Cyclophyllidea, suborder - Taeniata, families - Taeniidae,genus – Multiceps, species: 1. Larval stage - Coenurus cerebralis, 2. Ribbon stage - Multiceps multiceps.

Larval stage of Coenurus cerebralis. The size of the bladder depends on the degree of development, localization in the brain and the type of animal. Usually mature censures are large in size, in sheep they reach 10 cm in diameter or more, rounded or oval in shape, filled with a transparent liquid. The circumference of the cenura is delicate, semi-transparent, on the inner germinal shell, white dense tubercles located in separate groups close to each other- germinal scolexes are clearly visible. There are 700 or more of them in one bubble. They have four suction cups and hooks (22-32) in two rows. The hooks of the 1st row reach 0.150-0.170 mm in length and are equipped with a sharp curved blade, the hooks of the 2nd row are 0.090-0.130 mm in length, with a slightly curved sharp blade.Localization of the censors is the brain, less often the spinal cord.

The ribbon stage - Multiceps multiceps – is localized in the small intestine of dogs and other carnivorous animals (foxes - is an optional host). Reaching 40-100 cm in length and 5 mm in width. The scolex of the tape stage corresponds to the scolex of the censors. In the germinate segment, there are up to 200 testes, incorrectly alternating genital openings are located laterally in the form of tubercles, both lobes of the ovary are almost equal in size and oval in shape, a small triangular yolk is located near the posterior edge of the segment. In mature segments, 9-26 branches depart from the median trunk of the uterus on each side, which usually form additional branches near the free edge.

Biology of the pathogen. The definitive host of the parasite is dogs and other carnivorous animals, and the intermediate host is sheep, goats, cattle, less often pigs, horses and other animals, as well as humans. The prepatent period of development is 45-60 days, patent (the period of parasitization in the body of dogs is 735 days (the period of observation). Larvae reach the invasive stage after 3 months.

Diagnosis and differential diagnosis. The diagnosis is made comprehensively: based on epizootological data, clinical signs, pathoanatomic changes, ophthalmoscopy and allergy method.

Ophthalmoscopy. Already 1-2 months before the pronounced symptoms of the disease in sheep, stagnant phenomena in the fundus of the eye are observed. These changes are noted in the eye opposite to the location of the censor in the brain, and they are expressed in the confluence of the boundaries of the nipple and retina. There is also a change in the color of the nipple and its shape, the color of the retina, an overflow of blood vessels. Hemorrhage on the retina and on the nipple. The fundus is examined using a reflector after artificial pupil dilation with 0.5% atropine solution. The study of the ocular day is not difficult and can be used by practitioners in the differential diagnosis of moniesiosis, listeriosis, estrosis, as well as tenuicolic cysticercosis in sheep that respond positively to the censure antigen.

Allergic method. Dry factory powder of the valuable allergen is diluted in a physiological 1:750 and injected into the thickness of the skin of the upper eyelid or tail fold: allergen dose: for sheep -0.2 ml, 0.5-0.75 ml for cattle. With proper administration of the allergen at the injection site, a swelling the size of a small pea in sheep and a large pea in cattle is formed, Accounting for the reaction (measurement of swelling, caliper along its width without squeezing) is carried out 2-4 hours after injection. The established standard for assessing the reaction in sheep is as follows: with a swelling size of up to 2 cm, the reaction is negative; from 2.1 to 2.5 cm – doubtful and from 2.6 cm higher - positive. For cattle, it is as follows: with a swelling width of up to 3.5 cm, the reaction is negative, from 3.6 to 4.5 – doubtful and from 4.6 cm and higher – positive.

For an allergic reaction, you can also use liquids and scolex censors, sterically taken from fresh censor bubbles. This allergenic liquid is used fresh, stored in the refrigerator at a temperature below 2-40, as well as canned (with the addition of 1% phenol).

Differential diagnosis. Cenurosis should be differentiated from moniesiosis, estrosis, listeriosis of animals.

Treatment, prevention and control measures. The treatment has not been developed. In the last stage of the disease, when the censorship reaches a significant size, surgical intervention is advisable. The operation consists in removing the cenures after preliminary trepanation or puncture of the cranial cavity.

Prevention and control measures against larval cestodoses of animals (See the topics “Differential diagnosis of pathogens of teniidoses of dogs and other carnivorous animals”).

Security questions:

12. Give a characterization of echinococcosis?
13. How is Echinococcus granulosus located according to the taxonomy?
14. Anatomical and morphological structure of Echinococcus granulosus?
15. Biological development of the pathogen of echinococcosis?
16. Lifetime diagnosis of echinococcosis?
17. Give a description of the price t**a**g?
18. How is the causative agent of cenurosis located according to the taxonomy?

19. Anatomical and morphological structure of Coenurus cerebralis and Multiceps multiceps?

20. Biological development of the causative agent of sheep cenurosis?

21. How do we diagnose cenurosis during life?

22. How should we deal with echinococcosis and cenurosis of animals?

TOPIC № 9. TAXONOMY, MORPHOLOGY AND BIOLOGY OF NEMATODES

The purpose of the classes: To teach students the systematics, morphological structure and biological development of nematodes and their differentiation from other parasitic worms (worms).

Materials and equipment. Bucket, water, microscope, slide, cover glass, affected parenchymal organs and intestines of animals, magnifying glass, various volumetric cups, scissors, scalpel, cotton wool, gauze, tables, museum preparations nematode samples.

Morphology of nematodes. The nematode class, type unites roundworms, among which there are free-living forms, as well as parasites of animals and plants.

Nematodes usually have an elongated, non-segmented body, thread-like or spindle-shaped, with a length of 0.3 mm to 8 m.

The essential difference between nematodes and trematodes and cestodes is as follows:

1. Nematodes have a well-developed digestive system, which have two openings (oral and anal openings);

2. All nematodes are bisexual helminths with pronounced sexual dimorphism;

3. Among nematodes there are geohelminths, i.e. developing in a direct way, without the participation of an intermediate host, and there are biohelminths developing with the participation of an intermediate host.

4. Nematodes have adapted to parasitize in all organs and tissues of the animal;

5. Nematodes are oviparous and viviparous;

The body of nematodes is covered with a dense cuticle, smooth or striated in the longitudinal or transverse directions. The outer cuticle layer passes to the oral cavity, esophagus, rectum and distal parts of the genital ducts, lining them from the inside. There may be wound–like formations on the cuticle - hooks, spikes, ridges, hairs, thickenings that serve to fix the parasite. Under the cuticle are the hypoderm and the muscle layer forming the body cavity.

Nematodes lack respiratory and circulatory systems, but the digestive, nervous, excretory and reproductive systems are well developed.

The digestive system. The digestive system begins with an oral opening located terminally, subventrally or subdorsally. The oral opening of nematodes can be: open and closed. In some nematodes, the mouth is closed, i.e. surrounded by cuticular formations – the so-called lips. The number of guts can be different: depending on the type of nematodes, from 3 to 12 lips, which have a differential value in determining the type of nematodes. In some nematodes, the mouth is an open

type, i.e. the lips are completely absent, the place of them is armed with teeth, corollas, also has a differential value. The mouth opening usually leads to the oral capsule, which has various shapes. The oral capsule opens into a cylindrical pharynx surrounded by a bear layer, or directly into the esophagus. The esophagus of different nematodes has a different shape and structure, which also has a differential value. At the end of the esophagus there may be a bulbar (spherical) expansion – a bulbus (ventricle), in which there is a valve apparatus or without it. The esophagus passes into the intestine and ends with the rectum. The sexual duct opens into the rectum in males, so they call it a cloaca, and in females it is called an anal opening, which is in a terminal or subterminal position.

The nervous system. The nervous system consists of numerous ganglia connected by fibers and forming a nerve ring around the esophagus, from which six nerve trunks branch back and forth. Single ganglia are also found in other parts of the body: anal, genital and others.

Excretory system. The excretory system is represented by a pair of unbranched lateral longitudinal channels that begin at the posterior end of the body, and merge in front into the excretory sinus, which opens with an excretory pore on the ventral surface near the anterior end of the body of nematodes.

The sexual system. All nematodes are a single-sex helminth with pronounced sexual dimorphism. Males are usually smaller than females, the genitals of males and females have a tubular structure.

The female genitals consist of two convoluted ovaries connected through tubular ovaries to two tubular uterus. Eggs from the ovary through the oviduct enter the ovum (a small expanded part of the uterus), in which sperm are preserved and fertilization occurs, after which the fertilized eggs enter directly into the uterus, where the egg shell is formed and the embryo begins to develop. The uterus is connected to the vagina, the vulva is usually covered at the posterior end of the body, sometimes at the anterior or in the middle of the body, which is of differential importance for determining the type of nematodes.

The male's reproductive system is represented by a single testis and a vas deferens (subdivided into a somewhat swollen section – the seminal vesicle and the vas deferens), which opens into the ventral part of the rectum, forming a cloaca. Near the excretory canal of the male sex glands there is a complex of auxiliary organs – spicules with a knuckle (gubernaculum) and sometimes a genital cone with a supporting apparatus and nerve endings. During copulation, they are inserted into the female's vagina and fix it, sperm flows through them.

In addition, there is often a sclerotic cuticular thickening on the dorsal wall of the cloaca - a rudder (gubernaculum), which serves to give the proper direction to the movement of the spicules. The numbers, size and shape of the spicules have diagnostic significance, the Telamon (supporting apparatus) is a fixed compacted part in the ventral and lateral walls of the cloaca, serves to prevent cloaca ruptures during the movement of the spicules and is a permanent morphological sign of trichostrongylid and srotostrongylid. In some nematodes, the tail end of the male forms a kind of proliferation of lateral areas consisting of two symmetrical lateral and

one dorsal lobes, which are equipped with neuromuscular formations - ribs, and some male nematodes are equipped with genital papillae, which are a fixation organ and serves for strong fixation of females during copulation.

The biological cycle of nematode development is very diverse. Among nematodes, geohelmites are distinguished, developing in a direct way, without the participation of intermediate hosts, and biohelminths, whose development cycle is performed with the obligatory participation of intermediate hosts, in addition. Many nematode species also have reservoir hosts.

Female nematodes secrete eggs or larvae into the external environment, in accordance with which they are called oviparous and viviparous. Depending on the type of egg-laying nematodes, the eggs they secrete contain either an already formed larva or individual blastomeres. In some cases, larvae hatch from eggs when they pass through the intestines, which are thrown out. Eggs or larvae of egg-laying nematodes are released into the external environment with feces or urine. If eggs with an embryo are released into the external environment, then in any case, a larva of the I-stage necessarily develops in the egg. In the future, the larva can leave the egg shells or develop in it to the invasive stage.

Larvae of viviparous nematodes, depending on the localization of the latter, are released into the external environment through the gastrointestinal tract, with outflows from the eyes or enter the bloodstream, and from it in the body of blood-sucking insects- intermediate hosts. The exception to this provision is trichinella. In trichinosis, the same animal is a definitive and intermediate host.The invasive stage of nematode larvae, as a rule, is half of two molts in the external environment (in the egg, on the soil, grass) – geohelminths or in the body of an intermediate host – biohelminths.

With the development of nematodes in a direct way, animals become infected after ingesting invasive eggs or larvae. Before the larvae develop into an invasive stage in the external environment, the larvae in the external environment lead a free, non-parasitic lifestyle: they grow, move, migrate along the stems of plants.

When animals are infected with invasive eggs, the larvae hatch in the intestines. Eggs and larvae enter the digestive tract of animals with food or water, and larvae of individual nematodes (strongyloids, hookworms, uncinaria) they are able to penetrate through the intact skin of the animal. Further development of larvae occurs directly in the digestive tract, or they sometimes undertake a complex migration in the host body before reaching the place of their development before puberty.

When developing indirectly, many species of invertebrates and vertebrates can be intermediate hosts. The biohelminths include the following types of suborders filariate, spirurate, dioctophimate, some types of trichocephalates, strongylate and oxyurate. The larvae of nematodes, having got from an intermediate host in the body of a definitive one, also perform a very diverse migration in it, depending on the type of nematodes.

Taxonomy of nematodes. Nematodes by taxonomy belong to the type of roundworms – Nemathelminthes, class - Nematoda, includes eight suborders of

veterinary and medical significance: Oxyurata, Ascaridata, Strongylata, Trichocephalata, Spirurata, Filariata, Dioctophymata and Rhabditata.

In oxyurates– the oral opening is surrounded by three or six lips, on the posterior end of the esophagus there is a spherical expansion (bulbus) with chewing plates, the vulva opens on the front part of the body, in the male one or two, which differ sharply in size and shape, and in most species are asymmetric.Geo- and bio-helminths.

In ascaridates, the mouth opening is surrounded by three lips, the esophagus is cylindrical, there is no bulbus, there may be small wings at the tail end of the male, preanal and postanal papillae, two equal spicules, there is no rudder, the vulva of the uterus in the anterior half of the body is faintly noticeable, eggs with a multilayer dense shell are smooth or bumpy. Geo- and bio-helminths.

Strongylates have a head end without a tooth. The oral capsule is small or powerful, sometimes armed with cutting plates or teeth. The esophagus in the back of the body is somewhat expanded, a characteristic feature is the presence of a sexual cuticular bursa with ribs in males, two identical spicules, less often different in size and structure. The vulva opens in the middle part or anterior third of the body, can be surrounded by lips, covered by a cuticle valve. Eggs with a thin shell, of various sizes. Geo- and bio-helminths.

In trichocephalates, the anterior end of the body is long, threadlike, and the posterior is much thicker or of the same thickness. The esophagus is in the form of a thin tube, the esophageal glands are well vented, giving the impression of single cells arranged in a row. The spicule is single, short, long or it is absent, the vulva opens in the posterior third or in the anterior part of the body, oviparous, rarely viviparous (trichinella), barrel-shaped eggs with "corks" at both poles. Geo- and bio-helminths.

In spirulata, the oral opening is mostly surrounded by two lips divided into lobes, the oral cavity passes into the pharynx, the esophagus is divided into two sections: muscular (anterior) and glandular (posterior). There are two spicules, equal or unequal. On the lateral sides of the tail end of the male, cuticular wings, stalked or seated papillae are most often located, the vulva often opens near the middle of the body, the side is noticeable, the eggs are small, with a thick shell, leaves the body with a ready larva. Biohelminths.

Filariates have a characteristic biological feature: they are localized in closed systems and body cavities of the definitive host (thoracic and abdominal cavities, in blood vessels, tendons, muscles). The head end is simple, often without lips, the cylindrical esophagus consists of mouse and glandular parts. The spicules are two, unequal, the vulva is located at the anterior end of the body, oviparous and viviparous. Biohelminths.

In dictyophymates, the oral root is simple or equipped with a muscular sucker, the cuticle is transversely striated, the esophagus is simple, without a bulbus. The male has a dense colloidal bursa on the caudal ring without ribs, one spicule, elongated, without appendages. The anus in females is at the tail end, the vulva opens not far from it, in some species the vulva is in the front part of the body. The eggs have a powerful shell with a complex pattern on the surface. Biohelminths.

The rhabdidata has a characteristic structure of the esophagus: it has two bulbuses – anterior (prebulbus) and posterior, such an esophagus is called a rhabditoid. Small thin nematodes, among which there are free-living organisms. The eggs are small, after entering the external environment, larvae develop in them in a few hours. Geohelminths.

Security questions:

1. What type and class are roundworms?
2. How many sub-orders of the nematode class have veterinary and medical significance?
3. Tell us the anatomical and morphological structure of nematodes?
4. Tell us the biological development of nematodes?
5. Tell us the biological features of the development of hematodes?
6. Tell us the biological features of the development of bioonematodes?
7. Representatives of which of the nematode suborders parasitize in the closed organs and tissues of the definitive host?
8. When parasitizing representatives of which suborder is the same organism a definitive and intermediate host?

TOPIC № 10. SYSTEMATICS, MORPHOLOGY AND DIAGNOSTICS OF PROTOZOANS.

Purpose of classes: To teach students the systematics, morphological structure and biological development of arthropods, methods of in vivo –laboratory and postmortem diagnostics of psoroptosis, sarcoptosis and chorioptosis of animals.

Materials and equipment. Water, bucket, microscope, slide glass, samples of scrapings from the border of affected and healthy skin areas, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Protozoology (Protozoa type) is the science of the simplest single-celled animal organisms. Veterinary protozoology studies protozoa that parasitize animals and cause diseases in them – protozooses. Its task is to study the morphology and biology of the parasite, determine its type and ways of infecting animals. At the same time, protozoology studies the pathogenic effect of the pathogen on the animal body, methods of diagnosing diseases, specific and pathogenetic therapy, as well as the prevention of these diseases.

I. The main stages of the development of veterinary protozoology. Unicellular animals as microscopic organisms were first studied by microbiology. Later, at the end of the XIX and the beginning of the XX century, the protozoans were separated into an independent discipline – protozoology. In those years, remarkable discoveries were made that were of great importance in the development of the doctrine of pathogenic protozoa. Pathogens of trypanosomiasis, malaria, pyroplasmidosis, eimeriosis and other diseases have been identified.

Protozoological research in the field of medicine and veterinary medicine has led to a number of discoveries of pathogens of protozoal diseases that caused epidemics and epizootics in many countries. So, in India, the "surra disease" was

widely spread. Veterinarian Evans in 1880 Revealed the etiology of this disease and established that its causative agent is trypanosoma. In 1888, Babes in Romania discovered a pathogen called "bloody urine" in cattle. In 1889 Smith and Kilborn also found pyroplasmas in the blood of cattle, thereby proving that Texas fever in America is caused by the blood parasite P.bigeminum. In 1893 These authors have established that pyroplasmas are transmitted from sick animals to healthy pasture ticks. It was a new idea about unicellular organisms as pathogens of human and animal diseases.

In 1891. Russian scientist D.L.Romanovsky created a special method of coloring protozoa, which was a kind of impetus in the development of protozoology.

The works of I.I.Mechnikov and D.L.Romanovsky had a huge influence on the development of protozoological science, which for the first time proved that the causative agent of human malaria belongs to the protozoa. A certain role in the development of therapy for protozoan diseases of humans and animals was played by the works of P. Ehrlich and D.L.Romanovsky on the study of the mechanism of action of medicinal preparations on pathogens. I.I.Mechnikov's research on immunity also contributed to this.

In Russia, the work of well-known researchers V.Y.Danilevsky, E.N.Dzhunkovsky and I.M. Luz, E.I.Marcinovsky, V.L.Yakimov, A.V.Blitzer, N.A.Saarov and others had a significant impact on the development of protozoology, who identified new pathogens of protozoal and spirochaete animal diseases. In 1898, A.I. Kachinsky first described the pyroplasmosis of cattle in Russia. In 1903, E.P.Dzhunkovsky and I.M.Luz discovered theileriosis of cattle in Transcaucasia. In 1096, A.V. Blitzer and E.I.Marcinovsky discovered pyroplasmosis of horses in Ryazan. In 1911 Student Dementiev first found the causative agent of pyroplasmosis in pigs.

In 1913, V.L.Yakimov led an expedition to Turkistan, where he conducted extensive research on trypanosomiasis, leishmaniasis, priroplasmidosis, collected and described the fauna of vector ticks. The main research on veterinary protozoology by V.L.Yakimov and his numerous students was done after the October Revolution. In the twenties, V.L. Yakimov created a large scientific center in Leningrad for the study of protozoal animal diseases.

At the same time, a second scientific center is being created in Moscow under the leadership of first A.V. Belitzer, and then A.A.Markov, where protozoal diseases of horses, camels, cattle and small cattle are studied. Subsequently, the issues of systematics, pathogenesis, immunity, ultrastructure of protozoa, etc. are being developed.

Currently, the All-Union Society of Protozoologists is working under the leadership of Yu.I.Polyansky. In many research institutes and universities, veterinary protozoology studies pathogens of protozoal diseases and measures are being developed to combat these animal diseases. Such veterinary protozoologists as A.G.Gafurov work in our Republic. Kuldoshev O., Rasulov U., Abdurasulov Sh.

II. Morphology, **biology and systematics of parasitic protozoa.** Protozoa are independent unicellular organisms that consist of inextricably linked nuclei and cytoplasm.

The nucleus in the life of a cell performs two functions: genetic and metabolic, it is usually located in a certain place of the cytoplasm, but sometimes it shifts.

The cytoplasm is an integral part of the cell, it is of a liquid or semi-liquid consistency, and it is covered with a cytoplasmic membrane (pellicle). A number of organelles (organoids) are distinguished in the cytoplasm: cytoplasmic network (reticulum), ribosomes, mitochondria, lysosomes, lamellar complex (golgi apparatus), as well as some other organelles inherent only in single cells.

The movement of protozoa is carried out by means of three main types of organelles: cilia, flagella and pseudopods. The cilia and flagella originate from the basal body lying in the cytoplasm near the cell surface. Movement with the help of pseudopods is inherent in amoebas and consists in the fact that the cytoplasm moves from place to place in their body (amoeboid movement).

Nutrition in protozoa is carried out through a special organelle of the cytostome by phagocytosis and pinocytosis.

Respiration in protozoa can be aerobic or anaerobic. The simplest, like all organisms, are characterized by irritability. Stimuli can be changed environmental conditions in the form of chemical, mechanical, thermal and other factors. The main reactions of the protozoa are called taxis or tropisms. Therefore, the response of protozoa to a chemical stimulus is called chemotaxis, to thermal irritation – thermotaxis, etc. Each taxi can be positive or negative.

Reproduction occurs asexually (agamous) or sexually. Asexual reproduction is carried out:

A). Division into two (monotomy) – uniform division of the body of the protozoan into two daughter individuals. Such reproduction is available in amoebas. Flagellates, and in certain periods of life and in representatives of sporovics;

B). Budding is an unequal division of the body – one or more daughter individuals bud off from a large maternal individual. This division is observed in flagellates. Some pyroplasmids;

C). Endodiogeny is the formation of two daughter individuals inside the mother cell, in contrast to simple division, daughter individuals are preserved for some time under the pellicle of the mother cell. Subsequently, the outer membrane of the mother cell covers the daughter individuals (toxoplasma).

D). Multiple fission (syntomy), in which repeated division of the nucleus occurs and the simplest becomes temporarily multicore. After that, cytoplasm forms around the new nuclei in the cell, organelles are formed, and then multiple division into a large number of new organisms begins. If asexual individuals are formed in the process of division, they are called meronts (schizonts), and the process of division itself is merogony (schizogony). New protozoa that have arisen as a result of merogony are called merozoites. If male and female individuals are formed in the process of division, then the multinucleated cell is called a gamont, and the process of division itself is called gametogony. Individuals formed as a result of gametogony are

called gametes: microgametes (males) and macrogametes (females). If multiple division occurs after sexual division – sporogony, and newly emerged individuals as a result of sporogony are called sporozoites.

During the sexual process, fertilization is carried out by copulation or conjugation.

During copulation, two heterosexual individuals (gametes) that outwardly look the same (isohemates) or sharply differ from each other (anisogametes) merge and form a zygote. The process ends with the fusion of nuclei, as a result, a nucleus of a dual nature is formed in the zygote, that is, with a diploid set of chromosomes. Subsequently, the reduction of the number of chromosomes (meiosis) occurs in the zitog, and a haploid set of chromosomes remains in each developing individual.

During conjugation, individuals, entering the sexual process, do not merge, but only temporarily connect, exchange parts of the nuclear apparatus and cytoplasm, and then disperse, returning to independent life. Conjugation is observed only in representatives of the Ciliata class.

Both copulation and conjugation are sexual processes, but not reproduction, because the number of individuals does not increase. The latter is noted only with asexual reproduction, which alternates with sexual procession.

Systematics of pathogenic protozoa. Based on data on the morphology and biology of protozoa, the International Committee on Protozoan Toxonomy in 1980 recommended combining Protozoa into a sub-kingdom. The latter is divided into 7 types. Pathogens of animal diseases belong to 3 types:

1. Sarcoma stigophora - flagellates
2. Apicomplexa – its representatives at the anterior end of the body have a so-called apical complex.
3. Ciliophora – ciliated.

Veterinary parasitology also considers unicellular organisms – spirochaetes and anaplasmas, which are currently classified as Rickettsi-like organisms.

Topic№11. DIAGNOSIS OF BOVINE TAILERIOSIS.

Theileriosis is a severe obligate-transmissible disease of cattle, occurring mainly in the spring-summer period, caused by parasites from the genus Theileria Bettencourt, Franca et Borges and occurring with symptoms of enlargement of the superficial lymph nodes, fever of a permanent type, a sharp violation of the heart, digestive system, multiple hemorrhages in internal organs and on the mucous membranes. Teilariosis caused by Th. Annulata (synonyms: tropical pyroplasmosis, Russian taileriosis, Transcaucasian taileriosis, Egyptian fever, tropical gonderiosis). It is widespread in Russia in Transcaucasia, some areas of the North Caucasus, Astrakhan region, in the countries of Central Asia, it occurs mainly in the spring and summer period. Abroad, teileriosis is common in North Africa, Southern Europe, Central, Eastern and Asia Minor.

Etiology. Erythrocyte forms of the causative agent of the disease multiply in the body of a susceptible animal, while not causing him any clinical signs of the

disease, but have the ability to infect ticks. When examining the blood of infected animals, we find a predominance of rounded parasites, in which the ratio of elongated parasites to rounded ones ranges from 1:3,38 to 1: 6,56. The size of the rounded forms of the parasite is 0,6-1,8 microns, oval -0,7-1,8, pear–shaped – 0,7-1,1, rod-shaped -0,8-2,3 and comma-shaped – 0,9-2,3 microns.

The development cycle. Taileria in the body of animals and ticks go through a complex development cycle. Sexual development of teileria occurs in the intestinal wall, hemolymph, hemocytes of ticks during metamorphosis and culminates in the formation of sporozoites in the salivary gland of nymphs and imagos. Sporozoites are also detected in the salivary gland of non-feeding imagos. Epizootological data. Animals of all ages and breeds are susceptible to the disease. At the same time, calves up to 6 months of age carry the disease hard, buffaloes and zebu usually get over it in a mild form. Enzootia in animals is possible if there is a source of the pathogen of invasion (sick animals and parasite carriers, vector ticks), vector ticks and susceptible animals.

Carriers of parasites are: Hyalomma anatolicum, H. Detritum,H. mauritnicum, H. scupense, H. plumbeum,H. asiaticum. The main role in the epizootology of teileriosis belongs to the first two types. Seasonal dynamics of the disease is associated with the presence of active phases of vector mites in nature. Sick animals begin to stand out from the beginning of May to October. Most animals get sick from the end of May, in June and July. Sporadic cases of the disease can also occur in the winter months.

Pathogenesis. Invasive forms of tick-inoculated taileria with lymph and blood enter the regional lymph nodes and internal organs of the animal, where their development occurs. Sporozoites give rise to macrochizonts, which are found in punctate from affected lymph nodes or organs outside cells and in cells (lymphocytes, histiocytes, RES cells). Macrophages are able to phagocytize taileria and "garnet bodies", however, as a result of intensive division of parasites, the number of affected cells increases. The development of taileria can be suppressed in the primary focus. Otherwise, "garnet bodies" flood the host's body, the red blood cell damage progresses. The waste products of the "pomegranate bodies" and the cells destroyed by them lead to intoxication of the body. A sick animal develops hyperthermia, the activity of the cardiovascular system and gastrointestinal tract is disrupted, deep changes in metabolism (protein, carbohydrate, mineral, etc.) occur.

In the acute febrile period of the disease, a sick animal has an increase in glucose in the blood. At the same time, with a severe and prolonged course of the disease, the amount of glucose in the blood decreases by 5-12 times compared to the norm. As the disease develops, the phenomena of hemorrhagic diathesis increase in the animal, in the presence of inflammatory processes in organs and tissues. In places where "garnet bodies" accumulate, granules, erosions and ulcers occur. The cells of the reticuloendothelium and the lymphoid– macrophasal system are blocked by "garnet bodies", therefore phagocytosis and decay of the invaded erythrocytes is slow. After 10-12 days, the number of "pomegranate bodies" in the affected organs

decreases, phagocytosis of the invaded erythrocytes increases. In a sick animal, deep anemia and oxygen starvation of tissues occur.

Clinical picture. The incubation period for infection through ticks is 5-12 days, sometimes 18; for blood infection with "garnet bodies" - up to 22 days. The course of the disease can be acute and subacute. In acute course, the first symptom of the disease is an increase in subcutaneous lymph nodes (inguinal, knee folds, supra-nominal and pre-scapular); with palpation, the lymph nodes are dense, painful.

Milk productivity stops completely after a significant decrease. A sick animal refuses food and water, there is no chewing gum. Pregnant cows can have abortions. With an acute course, the body temperature rises to 41 ° C, the fever in the sick animal is of a constant type, the high temperature lasts for 6-8 days. The condition of the animal is oppressed. During a clinical examination, we register the atony of the pre-ventricles. Constipation alternates with diarrhea. The pulse is rapid, the heartbeat is intensified. After 6-10 days, these symptoms in a sick cow become more pronounced. On the visible mucous membranes, less often on the delicate areas of the skin, we note spotty or spotty hemorrhages. On day 6-8, the body temperature of the sick animal drops sharply and the animal dies.

The subacute course of teileriosis is accompanied by an increase in lymph nodes, the body temperature rises to 40-41 °With and at this level it lasts 2-3 days, after which it decreases to normal. After 2-3 weeks, the body temperature rises again and with slight fluctuations it stays with the sick animal until the end of the disease. The general condition of sick animals is depressed. Appetite is weakened, chewing gum is sluggish, and then stops. The sick animal gradually loses weight and after 2-3 weeks we note exhaustion. Sick animals lie more, throw their heads on their sides, moan, and if treatment measures are not taken in a timely manner, then sick animals die from increasing heart weakness. The disease lasts 12-25 or more days.

Immunity. Animals that have been ill acquire intense immunity, which they retain for several years. Immunity in teileriosis is cell-dependent, macrophages, T- and B-lymphocytes take part in its formation. Antibodies are detected in the blood of sick and ill animals during the study.

In Russia, Uzbekistan, Tajikistan, cultural anti-teileriosis vaccines are used for the prevention of teileriosis. Pathoanatomic changes. When an animal falls on the 8th-15th day of the disease, fatness is from below average to average. The mucous membranes are hyperemic, slightly jaundiced, in them and serous membranes, under the endocardium, in muscles, in the liver, in lymph nodes, subcutaneous tissue, and often in the skin spot and spotty hemorrhages. The spleen is enlarged by 1.5 times, the pulp is softened, hemorrhages under the capsule.

The liver is enlarged in volume, flabby, under the capsule of hemorrhage. Lymph nodes are enlarged, edematous, hyperemic. In the mucous membrane of the abomasum, intestines and other organs, we find granule-like formations, in place of which ulcers and erosions are formed. There are signs of edema in the lungs, hemorrhages under the pleura, in the mucous membrane of the trachea and bronchi. The book is often filled with dense, dry fodder masses. In the case when the animal dies after 15-22 days or later, the corpses are usually exhausted, the mucous and

serous membranes are anemic, with a jaundice tinge; at autopsy, we register atrophy of skeletal muscles; liver, spleen and lymph nodes are slightly enlarged. Hemorrhages and granule-like formations are rare.

The diagnosis of teileriosis is made comprehensively taking into account epizootological, clinical, pathoanatomic data and on the basis of microscopy of blood smears, smears from punctures of lymph nodes, spleen, bone marrow and liver. When an animal falls, smears are microscopized-prints of parenchymal organs stained according to Romanovsky – Gimza. Differential diagnosis.

Veterinary specialists differentiate teileriosis from pyroplasmosis, francaiellosis, anaplasmosis and leptospirosis.

Treatment. The sick animal is left indoors or put under a canopy. Prescribe a diet of easily digestible feed. Several therapeutic schemes have been proposed for the treatment of teileriosis. The 1st scheme. On the first day, a sick cow is intramuscularly injected with a 7% solution of azidine at a dose of 0.0035 g / kg, after 4-6 hours, a 10% solution of sodium chloride is injected intravenously at a dose of 0.5 ml / kg and 1-2 g of ascorbic acid in 1-2% dilution. On the second day, terramycin is administered intravenously at a dose of 0.15 g / kg in a 5% dilution or intramuscularly olemorphocycline 7.5 mg / kg, or oxytetracycline 2000-5000 units per 1 kg of body weight. On the third day, caffeine and intramuscularly sulfadimesine (sodium salt) or sulfantrol at a dose of 0.0005 g / kg in a 10% dilution. If the animal has a relapse of an increase in body temperature, then after 2-3 days the course of treatment is repeated. 2nd scheme. Chiaocide once for three days at a dose of 1 mg / kg, on day 4-5, bigumal is prescribed at a dose of 12.5 mg / kg. These drugs are administered orally in a 1% aqueous solution. With a relapse of high body temperature, both drugs are prescribed simultaneously in the same doses and used until the body temperature drops and two days after the temperature drops. The general course of treatment is 5-6 days. At the same time, symptomatic treatment is carried out for sick animals: inside caffeine 3.0-5.0 g, phthalazole 10.0, cobalt chloride 50mg, copper sulphate 500mg – once a day. Vitamin B12 300-500mcg intramuscularly 1 time in 2-3 days.

Prevention. Regularly once every 1.5-2 months, anti-tick treatment of animals in the spring and autumn period. Stable keeping of animals is recommended for animal owners. If possible, the animals are transferred to camp or camp — pasture maintenance in places where there are no ticks – carriers of the causative agent of teileriosis. Animals are imported from safe farms in late autumn, placed in isolated areas and in rooms free of ticks.

TOPIC № 12. SYSTEMATICS AND DIAGNOSTICS OF IXODES AND ARGASS MITES.

Purpose of classes: To teach students the systematics, morphological structure and biological development of arthropods, methods of in vivo –laboratory and postmortem diagnostics of psoroptosis, sarcoptosis and chorioptosis of animals.

Materials and equipment. Water, bucket, microscope, slide glass, samples of scrapings from the border of affected and healthy skin areas, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Morphology of ticks of the family Psoroptidae - genus - Psoroptes, species: Psoroptes ovis (sheep), Psoroptes bovis (cattle), Psoroptes equi (horses, donkeys and mules) and Psoroptes cuniculi (rabbits) Body shape is oblong-oval, the oral apparatus of the piercing type, adapted for piercing the epidermis and sucking lymph. The legs are long, the development, the suckers are on long jointed or short non-jointed rods. The eggs are oblong-oval, asymmetrical. The flat side of the uterine secret is fixed to the host's body. Ectoparasites. Ticks of this family cause psoroptosis (cutaneous scabies), chorioptosis (leathery scabies and otodectosis (ear scabies) in various pitchforks of domestic animals.

Mites of the genus Psoroptes are the largest among scabies mites. Mature individuals can be seen with the naked eye. They are gray-yellow in color, the length of the male is up to 0.5 mm, the female is up to 0.8 mm, the oral apparatus is adapted for piercing the skin and sucking lymph. The legs are developed, at their free end there are thin-walled black-walled rods ending in suckers. In males, these suckers are located on the first, second and third pair of legs. Females lay eggs on the surface of the host's skin. Eggs are oblong-oval in shape, up to 0.3 mm long, blunted at both poles, asymmetrical. The flat side is adjacent to the host's skin, being held on it thanks to the uterine secret.

The genus Sarcoptes causes sarcoptosis in animals. In horses, Sarcoptes equi is parasitized by uloshadei, in cattle - Sarcoptes bovis, in camels - Sarcoptes camellia, in pigs - Sarcoptes suis, in sheep - Sarcoptes ovis, in dogs - Sarcoptes canis, in rabbits - Sarcoptes cuniculi and in reindeer Sarcoptes rangiferi. Ticks of this genus (itches) are small, the body length of males is up to 0.2 mm, the length of females is 0.45 mm, the body is rounded, dirty gray in color, there are scales and thorns on the dorsal side, the proboscis is developed, horseshoe–shaped, adapted for gnawing through the passages in the skin of the host.The legs are short, cone-shaped, the front pairs of legs are the most developed. In females, the front pairs of legs have unsigmented rods ending in suckers, and the rear pairs of legs without suckers, but with long bristles. In the male, the first, second and fourth pairs of legs end with rod suckers, and the third pair with a long bristle. The posterior end of the body of the female and male is rounded. The male has no copulatory suckers. Anal opening at the posterior end of the body.

The eggs are oval in shape, slightly blunted at the poles, 0.15-0.25 mm long, with a two-layer shell. A six-legged larva up to 0.15 mm long emerges from the egg after 3 to 6 days.

The genus Noroedres causes notohedrosis in animals. In dogs, cats and rats, Noroedres cati parasitizes, in rabbits - Noroedres cuniculi. Morphologically, Noroedres have many similarities with ticks of the genus Sarcoptes, they are distinguished by the location of the anal opening. The original place of settlement of ticks in cats is in the area of the base of the ears, forehead and upper part of the neck, in rats – the edges of the ears and tail.

In cats and dogs, as well as fur-bearing animals, in the absence of treatment, ticks spread to other parts of the body, and a severe generalized form of notohedrosis occurs.

Ticks of the genus Chorioptes parasitize on the surface of the skin of animals. The causative agent of choryptosis in horses is Chorioptes equi, in cattle - Chorioptes bovis, in sheep - Chorioptes ovis, in goats –Chorioptes caprae and in rabbits – Chorioptes cuniculi.

Morphologically, skin-eating mites occupy an intermediate position between pruritus and skinworms. They are relatively large, the length of the male is up to 0.3 mm, the female is 0.4-0.5 mm, the body shape is oblong-oval, the proboscis is short, rounded, the oral apparatus is gnawing, the chelae are wedge-shaped, they feed on the scales of the epidermis and the products of inflammation caused by them. These mites are characterized by wide tulip-shaped suckers on their legs, located on short non-pigmented rods.

Sexual diformism is well expressed, in the male the posterior end ends with two abdominal lobes bearing four long bristles, of which two are flat. Suckers are present in all legs, the fourth pair of legs is three times smaller than the third pair of legs. In the female, the posterior end of the body is obtusely rounded, there are no suckers on the third pair of legs, instead of them there are long bristles.

Otodectosis is an ear scabies, the causative agent of which is Otodectes cynotis, parasitizing in the external auditory canal, on the eardrum and in the auricle of carnivores. The disease is severe in foxes and arctic foxes. Ticks of this genus bear some resemblance to skin-eating ticks. The womb of the body is oval, gray-yellow in color, males up to 0.3-0.4 mm in length, females up to 0.5 mm. Proboscis weakly protrudes, rounded shape, gnawing type oral apparatus. Sexual diformism is well expressed, the male has no abdominal processes, but there are two tubercles on the posterior edge of the body, from which two long and short scutes depart.The fourth pair of legs is poorly developed, the free end of each leg ends with a tulip-shaped sucker sitting on a short rod. In the female, the fourth pair of legs is poorly developed and does not even protrude beyond the edges of the body, suckers are available only on the second pair of legs.

TOPIC № 13. TAXONOMY, MORPHOLOGY AND DIAGNOSTICS OF INSECTS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of protozoal diseases, methods of lifetime laboratory (taking pathological material, staining, their examination under a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Entomology is from the Greek word entomon – insect and logos-teaching) – a science that studies the world of insects. Veterinary entomology studies insects that cause harm to animal health and develops measures to combat them. Insects can harm the health of animals as parasites and as carriers of pathogens of infectious and invasive diseases. Some insects are intermediate hosts of animal helminths, others cause damage to livestock products, and insect-caused diseases are called entomoses.

Taxonomy of insects. According to the taxonomy of insects, it is arranged in the following order: type-Arthropoda- arthropods, subtype - Tracheata, subclass insects – Insecta - Hexapoda, class postcard–jawed, or real insects -Insecta-Ectognatha, section winged insects –Pterygota. This section is represented by the department of insects with a full sling – Holometabola and the department of insects with an incomplete transformation - Hemometabola. Insects with complete transformation include the order Diptera or flies and mosquitoes - Diptera, which is divided into suborders: long-whiskered diptera– Nemaetocera, short-whiskered straight-seam diptera -Brachycera-Orthorrhapha, and short-whiskered round–seam diptera -Brachycera - Cyclorrhapha.

The suborder dlmnous diptera includes the families: mosquitoes (Culicudae), midges (Simulidae), woodlice (Ceratopogonidae) and moths (Psychodidae).

The suborder of short-whiskered straight-seamed diptera includes the family of horseflies (Tabanidae), the suborder of short-whiskered round-seamed diptera includes the families: subcutaneous gadflies (Hypodermatidae), gastric gadflies (Gastrophilidae), nasopharyngeal gadflies (Oetridae), true flies (Muscidae), carrion flies (Calliphoridae), gray meat flies (Sarcophagidae) and Bloodsuckers (Hippoboscidae).

Insects with complete transformation also include the order of the flea (Siphonapthera). Insects with incomplete transformation includes the orders: down-eaters (Mallophaga), lice (Sophunculata or Anoplura), Hemiptera or bedbugs (Hemiptera) and cockroaches (Blattodea).

Morphology of insects. The body of insects is constructed of double-sided symmetry and is divided into three sections: the head, chest and abdomen, consisting of separate segments. On the outside it is covered with protective chitinized cuticles. The cuticle serves as a support for the muscles and consists of semicircles – dorsal (tergite) and ventral (sternite). These half-rings are connected by skin membranes, which provide the insect with flexibility and suppressiveness. At the junctions of the semicircles there are spiracles giving rise to the trachea.

The head is movably connected to the chest, the organs of touch and smell are located on it in the form of a pair of club-shaped, feathery or bristle-shaped antennae (ligaments, antennae). The eyes are more often faceted, less often simple, located on the sides of the head. Individual insects (flies, gadflies, etc.) have three more simple dot-shaped eyes on the crown. Some species (lice, down-eaters) have no eyes. The oral apparatus in the form of a proboscis, depending on the method of feeding, can be piercing-sucking (mosquitoes, etc.), cutting-sucking (horseflies), licking (non-blood-sucking flies), gnawing (down-eaters) or absent altogether (gadflies).

The breast consists of three segments: anterior chest, middle chest and posterior chest. In diptera, the wings are attached to the lateral edge of the middle breast, and in four-winged to the posterior breast. In diptera, buzzers are attached to the posterior chest instead of wings, usually having the form of straight rods with a flask-like thickening at the end. The wings are formed by two tightly folded plates, between which the veins pass. Shape, coloration and venation are essential in establishing the genus, species of insect. Three pairs of five-jointed legs ending in paws are attached to the ventral side of the thoracic segments. The distal end of the tray often carries claws, a suction pad and empodium, which provide fixation of the insect to the host body and other surfaces.

The abdomen often consists of 5-10 clearly or weakly expressed segments. At the posterior end of it are the anal opening, the ovipositor in females and the copulatory apparatus in males.

The internal organs are located in the chest and abdomen in three planes and are separated by partitions.In the upper cavity is the circulatory system, in the middle – the organs of digestion, reproduction and excretion, in the lower – the nervous system. In each cavity, the organs are surrounded by a fat body consisting of fat cells in the form of a curd mass. The fat body is an organ of paramount importance.

The circulatory system is unclosed. The blood travels part of the way through the blood vessels, then enters the lacunae, into the body cavity and re-enters the vessels. The heart is located on the dorsal side in the form of a tube, separated by valves into chambers. Blood cells are colorless, slightly colored in green, yellow or other colors, they look like animal leukocytes.

The digestive apparatus of insects is in the form of a long tube, divided into anterior, posterior and posterior intestines. The malpighian vessels, which act as excretory orpgans, flow into the posterior intestine. A number of jellies of various shapes and purposes adjoin the digestive tract, the main of which are salivary.

The respiratory system is represented by spiracles located on the sides of the thoracic and first eight peritoneal segments, they give rise to tracheae penetrating the body of insects.

The nervous system in the form of nerve nodes – two head, three thoracic and up to eight peritoneal with nerves extending from them.

The organs of touch in the form of sensitive hairs or bristles located on the antennae, tentacles and paws.

The olfactory organs are located on the antennae in the form of olfactory pits, hairs, they are important in finding food, places suitable for laying eggs or larvae, and for detecting individuals of the opposite sex, they are more developed in males than in females.

The male reproductive system consists of two testes, the vas deferens, the ejaculatory canal ending with the penis, and the female reproductive system consists of two ovaries, their excretory ducts-the oviducts and the vagina. In addition, there is a vas deferens and accessory glands.

Biology of insects. Radelnopole insects. Females after fertilization lay eggs or in rare cases give birth to larvae. In the subsequent development occurs according to the type of complete or incomplete transformation.

In most insects (horseflies, gadflies, midges, etc.), the development cycle proceeds by complete transformation, that is, there are four phases of development: egg, larva, pupa and imago. A larva develops in a fertilized egg (ovogenesis). This period is called embryonic, which passes into postembryonic. With complete metamorphosis, the larva performs several molts and turns into a pupa. In the pupal phase, insects are more often motionless, living off the nutrients accumulated by the larva. Deep changes (histolysis) occur inside the pupa, and then histogenesis occurs – the formation of a sexually mature individual. At the end of histogenesis, the imago hatches.

With incomplete metamorphosis in development, an egg, a larva and an imago are distinguished (the pupal phase falls out). The larva emerging from the egg is morphologically similar to the adult, but smaller in size. Having made two or more molts, it turns into an imago (down-eaters, lice, bedbugs, etc.).

Under unfavorable conditions (lowering of temperature, starvation, dryness or increased humidity) in the organs of insects, life processes slow down or are interrupted, immobility occurs, growth stops. This condition is called diapause – adaptation to existence in unfavorable conditions. Diapause occurs in wintering mosquitoes, flies and other insects. With the appearance of optimal conditions, vital processes are restored.

Of the numerous class of insects in veterinary medicine and medicine , the following detachments are of the greatest importance: 1). Diptera - Diptera (gadflies, horseflies, flies, bloodsuckers, mosquitoes, midges, woodlice and mosquitoes); 2). Shphunculata - lice; 3).Mallophaga - down-eaters; 4). Aphaniptera – fleas; 5). Hemiptera- bedbugs. In some cases, arthropods of the order Blattoptera – cockroaches and Dermestidae – leatherworms are important.

Insects are carriers and pathogens of diseases. During the warm pasture period, pets are subjected to a massive attack of ectoparasitic insects: horseflies, flies, mosquitoes, midges, midges, bloodsuckers and mosquitoes, which are colloquially known as "midges". Throughout the year, and especially during the stall period, stationary ectoparasites attack animals: hair-eaters, fluff-eaters, lice and runets, as well as fleas and colps.

Horseflies are the largest diptera hematophages from the order Diptera, suborder Brachycera, family Tabanidae. In veterinary medicine, horseflies of the genera are of the greatest importance: Tabanus, Atylotus, Hybomitra, Haematopoda (raincoats) and Chrysops (motley).

Morphology. According to the size of horseflies, they are divided into large, medium and small. Their body length is 6-30 mm, body color depending on the type is yellow, gray, brown, black. The head is large, rounded in front, on the sides of its large faceted eyes, which in living specimens shimmer with rainbow colors. Some species have three more simple eyes in the middle of the crown. In front of the head

there are 3-4-segmented antennae ending in shelf-like processes consisting of several (3-7) segments.

Only females suck blood. They have an oral apparatus of a piercing-sucking type (in males–a licking type), located on the lower edge of the head and has the appearance of a proboscis. Two-jointed tentacles covered with sparse hairs are attached to its sides. In females, the proboscis consists of a fleshy, dark-colored lower lip, at the distal end splitting into two lobes, dotted with grooves, paired stiletto-shaped lower jaws, sabre-shaped upper jaws, upper lip fused with the supraglottis, and the subglottis, inside which the salivary duct passes. In males, the upper jaws are reduced, they feed on plant juices.

The chest is broad, massive, the wings are large, red or slightly darkened with bandages or spots. There is a buzzer behind the wings. The legs are developed, hairy, end in a pair of claws and three suction pads, the abdomen is wide, sac-shaped, consists of seven segments, when swallowing blood, its volume increases.

Biological development of horseflies. Females, after fertilization and saturation with blood, lay eggs in heaps on the stems and leaves of plants growing near reservoirs. Eggs are cylindrical in shape, obliquely cut at the upper pole. The color of freshly laid eggs is whitish, becoming dark brown as they mature. After 6-10 days, spindle-shaped larvae hatch from the eggs. Their body consists of a head, three thoracic and nine abdominal segments, the length of the larva just hatched from the egg is 2.3-2.7 mm, in those maturing after six molts – up to 45 mm. The larvae, having molted, turn into pupae.

The genus Tabanus is the largest horseflies, their body length is 16-30 mm, faceted eyes are bright green, blue, less often brown. The antennae are shorter than the head, the wings are transparent, placed at rest. Lower legs of the hind legs without spurs. The most common species are Tabanus bovines, Tabanus bromius,Tabanus autumnalis.

The genus Hybomitra is a medium–sized horsefly, 12-22 mm long. The faceted eyes are green or blue, slightly hairy, with three transverse stripes, except for the faceted ones, there are three more simple eyes on the crown. The antennae are shorter than the head. Wings with dark spots and stripes, widely spaced at rest. Lower legs of the hind legs with spurs. Common species: Hybomitra tarandina, Hybomitra tropica, Hybomitra montana, etc.

The genus Haematopoda is small grey–colored insects. The body length is 6-11 mm, the antennae are longer than the head, the faceted eyes are dim, there are no eyes on the crown. The wings are gray, with a marble pattern, at rest they are folded over the abdomen. Unlike other species, they are active in cloudy weather. Common amd: Haematopoda pluvialis, Haematopoda hispanica and others.

Horseflies are widespread everywhere, numerous in areas rich in reservoirs, swamps and woody vegetation. The horsefly season begins in early June and lasts until September. Horseflies suck blood from different parts of the body, but more often in the head, neck and chest. During the summer, they tend to water bodies to quench their thirst. Their owners are horses, cattle, camels, deer, rarely sheep, dogs. Animals with dark coloration are more susceptible to attack. During blood sucking,

horseflies pierce the skin, saliva with toxic properties is injected into the wound. Horseflies swallow blood from corpses, which makes them dangerous carriers of pathogens of infectious and invasive diseases. Young animals are more difficult to tolerate insect injections compared to adults. With an intense attack of horseflies, animals worry. The horse has local swelling and soreness of the subcutaneous patch of the chest, abdomen and prepuce. Despite the abundant foot food, cows reduce milk yield by 15-20%, the efficiency and fatness of animals decreases. Camels become emaciated.

Midges are small diptera insects of the order Diptera, family Simuliidae. Many species of this family are pesky hematophages. The most important are S.galeratum, S.columbaczense and others. With a mass attack on warm-blooded animals, simulyotoxicosis occurs.

Morphology. The midges are small, the dina of the winged insect is 2-6 mm, the body color is more often black, gray or dark blue. The head is slightly flattened from front to back and bent towards the lower surface of the pile. Proboscis on the lower surface of the head of the piercing-sucking type in females, and the licking type in males. Antennas consisting of 11 hairless segments are attached to the head. Faceted eyes are located on the sides of the head. The upper chest is humped and slightly hairy. Adjacent to it is a pair of wide oval-shaped wings, which are transparent, without transverse veins. These wings provide insect planning in the air flow and moving them ten kilometers from the breeding sites. The legs are thick, short, and are bounded by a paw with a pair of claws. The abdomen is more often sac-shaped, capable of stretching when swallowing blood.

Biological development of midges. Only females suck blood, after fertilization and saturation with blood, they lay eggs in reservoirs with a fast current (rivers, streams). Bottle-shaped larvae hatch from the eggs after 4-12 days. At first they are white, but as they develop, they acquire a dark color. On the breast of the larva there is a double-jointed leg, at the distal end of which there are wings located at the end. On the rear segment there is a powerful suction cup, seated with hooks for fixing to underwater objects. The body length of the larva of the 1st stage is 1-2 mm, in the mature 4-10 mm. After five molts, the larvae weave a bashkakoid cocoon in which they pupate. Cocoons are expanded and open from above. 2-8 or more respiratory threads (fans) protrude from them. After 14-21 days, winged midges hatch from the pupae. They float on the surface of the water with an air bubble and for the first time live in coastal vegetation. Females in search of food move away from the breeding site for 10 or more kilometers. During the year they give 1-4 or more generations. Midges hibernate in the egg and larvae phase.

Midges attack animals outdoors in the daytime. They suck blood from different parts of the body, but more often in the eyes, nose, lips, ears, upper neck, underbelly and abdomen. During blood sucking, they secrete saliva that has general toxic properties. Horses, cattle, pigs, sheep and carnivores are the most sensitive to the injections of midges. The disease has the character of an epizootic outbreak and is often accompanied by the death of a significant number of animals.

Mosquitoes belong to the family Culicidae. They are widespread everywhere. Blood-sucking mosquitoes are united in the subfamily Culicinae, in which the genera are of the greatest importance: Aedes, Culex, Anopheles.

Morphology. Kamars have a slender elongated body, 4-11 mm, body color depending on the species is yellow, gray, brown or black. The head is small, rounded, a significant surface is occupied by faceted eyes, almost touching on the crown. The antennae are long, consisting of 15 segments, they are covered with short hairs from the sides in females, and long hairs in males. The oral apparatus of females of the piercing-sucking type. Piercing parts in the form of narrow long feelers.On the sides of the proboscis there are five-lobed tentacles. The chest is hump-shaped from the ventral side, with two pairs of spiracles on its sides. The wings are red, elongated, and there are buzzers behind them. The legs are long, the paws end in a pair of claws, the representatives of the genus Culex have nulvilles between the claws, the Aedes have their rudiments. The abdomen is elongated, consists of two hundred segments. Blood is sucked only by females who, after fertilization, lay 150-180 eggs or more in stagnant reservoirs (Culex, Anopheles) or on the surface of moist soil (Aedes). Freshly laid eggs are whitish, mature dark in color. They are oval in shape, slightly asymmetrical. In a number of species, they have appendages (floats).

The larvae are elongated, mobile. Their body is divided into a head, chest and abdomen. The cuticle is covered with hairs and bristles on the outside. They breathe atmospheric air. Having undergone four stages of development, it turns into pupae, whose body is darker than that of larvae, comma-shaped, mobile, and kept near the surface of the water. The following types of mosquitoes are most pathogenic for pets: Aedes vexans, Culex pipiens, Anopheles maculipennis.

Aedes vexans. The body length of the female is 6-7 mm, the tentacles are dark, the middle part is covered with red-golden scales on top. The wings are dark, the legs with transverse white stripes. Claws with teeth. Abdomen on top with transverse light stripes. Females have two protrusions on the posterior abdominal segment.

Culex pipiens. The female is up to 5 mm, the overall body color is light brown, the tentacles are 5-7 times shorter than the proboscis, the rear end of the tentacles is three-lobed, with three tufts of hair.

Anopheles maculipennis. The female is up to 7 mm, the overall body color is more often brown, in females the tentacles are almost equal in length to the proboscis, the wings are brownish, 4-5 dark spots. Midrib on top with a wide longitudinal stripe. The legs are lighter at the base. The legs are black, the abdomen is brown or sulfur.

Mosquitoes attack animals during the day, but are more active at dusk in late spring and summer. The flight range from the breeding sites reaches 2-3 km, but species of the genus Aedes are able to travel a distance of up to 35 km. With a mass attack of mosquitoes, animals worry, the regime of keeping them on pasture is violated, animals lose fatness, cows reduce milk yield, growth and development slow down in young animals. Animals of dark colors, with short and sparse hair, as well as with contaminated skin are more intensively attacked by insects.

The role of mosquitoes in the transmission of infectious and invasive diseases is significant.

Woodlice are small dusky diptera hematophages of the family Geratopogonidae. Females of the genera Culicoides, Leptoconops and Lasiochella are bloodsucking. It is widespread everywhere.

Morphology. Body length 0.8-3 mm, similar in appearance to mosquitoes, but differ from them in smaller size and stockier build. With respect to the longitudinal axis of the body, the head is tilted downwards, bears a pair of 15-segmented antennae, set with short hairs in females and long in males. The faceted eyes are large, bean-shaped, massive, piercing-sucking type, five-lobed mandibular tentacles are located on its sides. The chest is convex, the wings are wide, hairy, folded at rest over the abdomen. Representatives of the genus Culicoides have spots on the wings. Their pattern is typical for various inputs. The legs are slender, the paws end in a pair of claws. The abdomen is ovoid, consisting of ten segments.

After fertilization, females lay eggs in moist soil, silted banks of rivers and streams, in stagnant reservoirs. The eggs are whitish at first, but as they mature they acquire a dark color. After 2-5 days, the larva hatches from the egg, which, after the tread, reaches 15 mm in length and turns into a movable pupa 5 mm long, similar to the pupa of a mosquito. On the dorsal side of the anterior section there is a pair of breathing tubes. The following species are of the greatest importance in veterinary medicine.

Culicoides pulicaris. The body color is gray or brown. The length of the wings is 1.5-2.2 mm. They are whitish with dark spots. There are three distinct spots at the anterior edge, and a distal spot with a constriction in the middle. The edges of the wing are framed by long hairs almost along the entire length.

Culicoides nubeculosus. The largest of the woodlice, the body length is up to 2.5 mm, the middle part is dark brown above. The wing pattern is blurry. The number of dark and light spots is almost the same.

Leptoconops borealis. The length of the wings is 1.2 mm, the antennae are 13-segmented, the wings are spotless. They are active only in the light hours of the day. Their flight begins in early April and lasts until September.

Female woodlice are active in windless weather with an air temperature above 140, during sunrise and sunset. They suck blood from various parts of the body, but more often where the skin is thinner. They attack animals both in the open and indoors. The woodlice fly up to 400 m from the breeding sites . Injections of slimes are painful, cause itching, which leads to skin dissections, focal depilation and dermatitis. During a mass attack of slimes, animals stop taking food.

Violation of the diet, deprivation of rest, overexcitation of the nervous system negatively affect the fatness and productivity of animals.

Mosquitoes are small diptera hematophages of the family Psychodidae, found in southern and subtropical areas.

Morphology. The body length of mosquitoes is 1.5-3 mm, covered with dark brown hairs. The head is small, with a pair of festooned eyes, in front of which there are long antennae consisting of 16 members. Oral apparatus of the piercing-sucking

type. 4-5 segmented tentacles are adjacent to the proboscis. The chest is slightly humped from above, the wings are wide, hairy, at rest they are raised to the axis of the body at 400, which makes them sharply different from other diptera hematophages. The legs are long, thin, and consists of seven segments.

After fertilization, females lay eggs in rodent burrows or in moist organic waste. The egg is oblong-oval in shape, light brown in color, up to 0.38 mm in length. The larva is worm-like, black in color, hairy, depending on maturity, its length is from 0.8 to 2.6 mm. The pupa is brown, club-shaped, curved, up to 3 mm long.

The most common types of mosquitoes are: Phlebotomus papatasii, Phlebotomus sergenti. Mosquitoes, like woodlice, are twilight insects. During the day, they hide in rooms for animals and humans, and in open nature – in shaded shelters (more often in rodent burrows). Only females suck blood from various warm-blooded animals. Unlike other long-tailed diptera hematophages, they do not have a connection with water. From the breeding sites, they fly up to 1.5 km, willingly fly to artificial light. The flight takes place from April to October.In places of insect bites, itching of the skin appears in animals, dermatitis occurs. Horses, dogs and birds are the most difficult to tolerate their bites. Mosquitoes are biological carriers of pathogens of leishmaniasis of animals and humans.

Bloodsuckers are in the family Hyppoboscidae. The most important species are: Melophagus ovinus, Hyppobosca equina, Hyppobosca canis, Lipoptena cervi (on salt and moose) and species from the genus Ornithomia (on pitas).

Sheep melophagosis is a chronically occurring entomotic disease that occurs as a result of parasitizing on the body of Melophagus ovinus animals and is characterized by itching, dumping and loss of fleece, pallor of visible mucous membranes, anemia, decreased productivity and retention in growth and development, sometimes pasture of lambs.

Control questions:

1. What is the characteristic of the insect class?
2. How is the reproduction and development of insects?
3. Bloodsucking diptera insects (midges) and their main distinguishing features?
4. What are the main directions of combating midges and protecting animals from it?

Topic № 14. DIAGNOSIS OF MALLOPHAGOSIS AND SIPHUNCULATOSIS.

The purpose of the classes: To teach students the systematics, morphological structure and biological development of protozoal diseases, methods of lifetime laboratory (taking pathological material, staining, their examination under a microscope) and postmortem diagnostics.

Materials and equipment. Water, bucket, microscope, slide glass, blood samples from peripheral and lymphatic vessels, Romanovsky paint, alcohol, cotton wool, needle, scissors, gauze, tables, museum preparations.

Morphology. Melophagus ovinus is a wingless insect (runet) of yellow-brown color. The female leaves the pupa 4.4 mm long, and if there is a mature larva in her gonads up to 7.5 mm long. The body of the runet is slightly flattened in the dorsoventral direction, covered with hairs and bristles. The head is small, tightly adjacent to the chest, it has small faceted brown eyes. The proboscis is thin, long, of a piercing-checking type, adapted for swallowing blood, mandibular tentacles are adjacent to its sides, abundantly covered with hairs, creating the impression that the proboscis is in a case. The antennae are short, double-jointed, the legs are developed, widely spaced, the paws end in two-pointed claws. The abdomen is wide, its segmentation is weakly expressed. In females, the posterior edge of the abdomen is notched, in males it is rounded. Females are viviparous, larvae are white at birth, rounded in shape, up to 3,5 mm in length. The uterine secret is attached to the root part of the hair. At the rear end of it there is a pair of indistinguishable spiracles. By the 5th-7th hour, it acquires a dark brown color and turns into a pupa, outwardly similar to a larva, but darker in color and with a hard outer shell..

Prevention and treatment of mallophagosis. In order to prevent the spread of down-eaters from one farm to another, it is necessary to examine the spitting animals for the presence of ectoparasites. The same surveys should be carried out during on-farm movement of livestock. When down-eaters are detected, all animals in the room are subjected to disinsection. For this purpose, the testers are sprayed with 0,75% chlorophos solution, 0,5% carbophos or trichlorometaphos-3 emulsions, 0,15% cyodrine emulsion, 0,05% permethrin emulsion, 2% oxamate emulsion. The same drugs are used for disinfection of premises, equipment and inventory in livestock and poultry premises.The bird is sprayed with a 2% oxamate emulsion at the rate of 25-50 ml per head. Oxamate is highly effective against down-eaters, non-toxic to animals, including birds, is not excreted with egg in treated chickens. Other insecticides are also highly effective against down-eaters, but they cannot be used on egg-bearing poultry and treatment should be carried out no later than 30 days before slaughter. The consumption rates of drugs are 25-50 ml per bird.

Prevention and treatment of siphunculatoses. Animals should be kept in clean, dry rooms, provided with exercise, the skin should be regularly cleaned or washed with water. Do not move infected animals to other farms and to siphunculatosis-safe farms. Animals affected by lice are sprayed with insecticides: 0,75% chlorophos solution, 0,75% carbophos emulsion, 0,15% cyodrine emulsion, 0.01% permethrin emulsion, 5% oxamate emulsion until the hair is fully wetted; in aerosol cans, acrodex, hematopinazole, dermatozole, insectol are used at the rate of 40-60 g per animal. Treatments are carried out twice with an interval of 10-14 days.

Security questions:

1. What is the characteristic of the insect class?
2. How is the reproduction and development of insects?

animals?

3. Features of the biology of bloodsuckers and pathogens of mallophagosis, siphunculatosis and vermipsilesis of animals.

1.4 MATERIALS FOR INDEPENDENT STUDY

Topic № 1. History of the development of the science of parasitology and invasive diseases.

Lists of the proposed literature

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Topic No. 2. Sources and foci of invasive diseases.

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Topic No. 3. Economic and social harm of invasive diseases.

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Topic 4. Methods of laboratory research of invasive diseases.

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Topic 5. Larval development of trematodes.

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Topic 6. Epizootology of flukes and control measures.

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5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
6. Mirziyoyev Sh.M. Insonparvarlik, ezgulik va bunyodkorlik-milliy g‘oyamizning poydevoridir. Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 36 bet.
7.O‘zbekiston Respublikasi Prezidentining 2019-yil 28-martdagi “Veterinariya va chorvachilik sohasida davlat boshqaruvi tizimini tubdan takomillashtirish chora-tadbirlari to‘g‘risida”gi PF-5696-son Farmoni.
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Topic 7. Bioecology and epizootology of cestodes.

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4. Mirziyoyev Sh.M. Birlashgan millatlar tashkiloti bosh assambleyasi 75-sessiyasida so‘zlagan nutqini o‘rganish va keng jamoatchilik o‘rtasida targ‘ib qilish. O‘quv qullanma. Toshkent, “Ma’naviyat” NMIU, 2021 yil. – 280 bet.
5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
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Topic № 8. Measures against cestodes.

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3. Mirziyoyev Sh.M. Yangi O‘zbekiston taraqqiyot strategiyasi. Toshkent, “O‘zbekiston” nashriyoti, 2022 yil. – 416 bet.
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5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
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7.O‘zbekiston Respublikasi Prezidentining 2019-yil 28-martdagi “Veterinariya va chorvachilik sohasida davlat boshqaruvi tizimini tubdan takomillashtirish chora-tadbirlari to‘g‘risida”gi PF-5696-son Farmoni.
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Topic 9. Development, bioecology and epizootology of nematodes.

Lists of the proposed literature

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1. Qurbonov Sh.X. «Parazitologiya» fanidan amaliy va laboratoriya mashg‘ulotlari, O‘quv qo‘llanma,Toshkent, 2015.
2. Haqberdiev P.S., Ibragimov F.B. Veterinariya protozoologiyasi va araxnoentomologiyasi. O‘quv qo‘llanma, Toshkent, 2020
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4. Mirziyoyev Sh.M. Birlashgan millatlar tashkiloti bosh assambleyasi 75-sessiyasida so‘zlagan nutqini o‘rganish va keng jamoatchilik o‘rtasida targ‘ib qilish. O‘quv qullanma. Toshkent, “Ma’naviyat” NMIU, 2021 yil. – 280 bet.
5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
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7.O‘zbekiston Respublikasi Prezidentining 2019-yil 28-martdagi “Veterinariya va chorvachilik sohasida davlat boshqaruvi tizimini tubdan takomillashtirish chora-tadbirlari to‘g‘risida”gi PF-5696-son Farmoni.
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Topic number 10. Chemoprophylactic measures against nematodes.

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2. Haqberdiev P.S., Ibragimov F.B. Veterinariya protozoologiyasi va araxnoentomologiyasi. O‘quv qo‘llanma, Toshkent, 2020
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5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
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7.O‘zbekiston Respublikasi Prezidentining 2019-yil 28-martdagi “Veterinariya va chorvachilik sohasida davlat boshqaruvi tizimini tubdan takomillashtirish chora-tadbirlari to‘g‘risida”gi PF-5696-son Farmoni.
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Topic № 11. Taxonomy and morphology of protozoa.

Lists of the proposed literature

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5. Mirziyoyev Sh.M. Yangi O‘zbekistonda erkin va farovon yashaylik. “Toshkent, “Tasvir” nashriyot uyi, 2021 yil. – 52 bet.
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Topic № 12. Epizootology of protozoa and control measures.

Lists of the proposed literature

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Topic number 13. Systematics and morphology of pasture mites.

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Topic No. 14. Measures to combat infectious diseases.

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Topic number 15. Diseases caused by insects.

Lists of the proposed literature

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3.5 Scientific glossary (in Uzbek, Russian, English)

№	Ўзбекча	Русча	Инглизча	Луғавий маъноси	Инглизча маъноси
1.	Авителлина	Авителлина	Avitellina	Кавшовчи ҳайвонларнинг ичак цестодозлари.	Rumen intestinal tsestodozlari
2.	Авлод	Поколение	Generation	Бир оиладан тарқалган насил, зот, зурриёт,	How common a family of soybean seed,
3.	Автотроф	Автотроф	Avtotrofla	Ўзи озиқланувчи (ўсмлик, ҳайвон)	Nutrition (the animal)
4.	Adoleskariya	Адолескария	Adoleskariya	Трематодларнинг юқумли личинкаси.	Trematodes infectious larvae.
5.	Акулалар	Батареи	Batteries	Тоғайли балиқлар вакили	The representative of the fish of the cartilages
6.	Alveokokk	Альвеококк	Alveokokk	Йиртқич ҳайвонларнинг ичак цестоди.	Wild animal intestines tsestodi.
7.	Амниота	Амниотической	Amniotic	Эмбирион даврида муртак парда ҳосил қилувчи ҳайвонлар	During the embryo to make curtains of apostasy
8.	Аналтешик	Анальное отверстие	Anal hole	Озиқлангандан сўнг кераксиз маҳсулот чиқарувчи тешик	Nutrition unnecessary holes
9.	Anoplotsefalidlar	Аноплоцефалидлар	Anoplotsefalidlar	Отларнинг ичак цестодлари.	Tsestodlari intestine of horses.
10.	Araxnologiya	Арахнология	Araxnologiy	Ўргамчаксимонлар	Tsestodlari Intestine of

			a	ни ўрганувчи фан	horses.
11.	Araxnoentomo logiya	Арахноэнтом ология	Araxnoento mologiya	Ўргамчаксимон ва ҳашоратларни ўрганувчи фан	O'rgamchaksim on science and the study of insects
12.	Аскарида	Солдаты,	The soldiers,	Одам, уй ҳайвонлари, паррандаларнинг ичак нематодалари	People, livestock, poultry gastrointestinal nematodes
13.	Асосийёкидои мийхўжайин	Первичный или постоянный босс	Primary or permanent boss	Паразитларнинг вояга етган даврида паразитлик қилувчи ҳайвонлар ва одамлар	During the adult parasites parasitic animals and people
14.	Бачадон	Матка,	Uterus,	Тухум ҳужайраси ва уруғ ҳужайраси учрашиб зигота хосил қиладиган организм	Egg cells and seed cells in the body during the zygote formation
15.	Бинар номенклатура	Бинарная номенклатур а,	The binary nomenclatur e,	Систематик қонун	System of law
16.	Biogelmit	Биогельминт	Biogelmint	Икки хўжайин иштирокида ривожланувчи паразит чувалчанглар	Two master the participation of developing parasitic worms.
17.	Bionematoda	Бионематода	Bionematod a	Икки хўжайин иштирокида ривожланувчи паразит	Two master the participation of developing parasitic

				чувалчанглар.	worms.
18.	Biotsenoz	Биоценоз	Biotsenoz	Маълум ҳудудда биргаликда яшовчи ўсимликлар, ҳайвонлар, микроорганизмлар йиғмаси.	living together in certain plants, animals, microorganisms Build.
19.	Бирқаватлива кўпқаватли эпидермис	Однослойные и многослойные эпидермис,	The single-layer and multi-layer epidermis,	Бир қаватли ва кўп қаватли тери	A story and qavatliteri
20.	Бир хужайрали	Клетка	Cell	Бир ҳужайрадан тошкил топган содда ҳайвонлар	A simple cell composed of shells
21.	Бош мия		Brain,	Умуртқалиларга хос 5та бўлмтан ташкил топган	In vertebrates consisting of 5 bo'lmtan
22.	Умуртқалилар	Позвоночных животных,	Vertebrates,	Хордалилар кенжа типи умиртқа поғанаси ривожланган	Xordalilar sub-type umirtqa developed loins
23.	Бош скелетсиз	Скелет	Skeleton	Хордалиларнинг бош қисми аниқ ривожланмаган	The head of the Xordalilarning developed
24.	Буйлама	Около .	Around	Мускул тури	muscle type
25.	Булутлар	Облака,	Clouds,	Тубан кўп ҳужайралилар	Low number of cells
26.	Bunostomer	Буностомер	Bunostomer	Ҳайвонларнинг ичак нематодалари	Animal gastrointestinal nematodes
27.	Bo'g'imoyoqli	Бўғимоёқли	Bo'g'imoyoq	Оёқлари бўғинларга	Their feet, divided into

			li	бўлинган	joints
28.	Бўйинча	Шеи,	Neck,	Цестодларнинг ўсиш зонаси	Tsestodlarning zone
29.	Wolff канал,	**Во**лф канали	Wolff channel,	Сийдик канали	urinary channel
30.	Гельминтология	Приводит	Leads	Паразит чувалчангларни ўрганувчи фан	Parasitic worms Sciences
31.	Геогельминт	Гельминты	Sth	Бир хўжайин иштирокида ривожланувчи гельминтлар	To improve a host of worm
32.	Geonematodalar	Геонематода	Geonematodalar	Бир хўжайин иштирокида ривожланувчи нематодалар	A boss with the participation of developing nematodes
33.	Germafradit	Гермафрадит	Germafradit	Эркаклик ва урғочилик органлари битта организмда ривожланган ҳайвонлар	Male and female organs in one body developed
34.	Geterotroflar	Гетеротрофлар	Geterotroflar	Тайёр озиқалар билан озиқланувчи ҳайвонлар	Animals are fed with nutrients
35.	Gomonom	Гомоном	Gomonom	Бир хил тартибда жойлашган организм	The body is located in the same order
36.	Gonoidy тангачалар,	Гоноиджеочищенные	Gonoid have peeled	Суякли балиқларнинг тангачалари	Bony fish scales
37.	Dicroceliosis	Дикроцелиоз	Dicroceliosis	Dicroceulium lanceatum	Dicroceuliumlan call ceatum

				трематодаси чақирадиган касаллик..	trematodes disease
38.	Diksenli	Диксенли	Diksenli	Иккихўжайинли	two masters
39.	Diktiokaul	Диктиокаул	Diktiokaul	Йирик ва майда шохли ҳайвонларнинг нафас олиш органларида паразитлик қилувчи нематода	Large and small horned animal respiratory parasitic nematodes
40.	Diktokaulus	Диктокаулус	Diktokaulus	Йирик ва майда шохли ҳайвонларнинг нафас олиш органларида паразитлик	Large and small ruminants respiratory parasitic animals
41.	Difillobotrioz	Дифиллобот риоз	Difillobotrio z	Уч ҳўжайн иштирокда ривожланувчи цестодоз касаллик	Three cell disease tsestodoz improve
42.	Елка-қоринмускуллар	Плечаимышцыживота,	Shoulder and abdominal muscles,	Трематодаларнинг элка тамондан қорин томонга тортилган мускуллари	Trematodes shoulder, hand pull the abdominal muscles
43.	Нематодалар	Нематоды,	Nematodes,	Юмолоқ чувалчанглар	Yumoloq worms
44.	Ёнчизиқ	Стороны линии,	Side of the line,	Балиқларнинг ён томонида жойлашган сезги органлари	On the side of the fish's sensory
45.	Ёпиқ	Замкнутая	A closed	Организмда фақат қон, қон	Body only blood, semen or

	қонайланиш	циркуляция,	circulation,	томирларда оқса ёпиқ қон айланиш доираси дейлади	blood vessels closed circulation Dale
46.	jabrayoriqchalari,	Жабра ёриқчалариГарнитура	Jabra yoriqchalari	Тоғайли балиқларга хос нафас олишда иштирок этувчи ёриқлар	Cartilages involved in the fish breathing in the original cracks
47.	Jabra қопқоқлари	Крышка Жабра гарнитуры	Jabra cover	Суякли балиқларга хос нафас олишда иштирок этувчи қопқоқча	Bony fish breathing in the original cover
48.	jabra	Жабра Гарнитура	Jabra	Сувда яшовчиларнинг нафас олиш органи	Living in the water, breathing body
49.	Замбуруғлар дунёси	В мире грибов,	The world of fungi	Замбуруғларни қамроб олган олам	Qamrob world of fungi
50.	Заррача организмлар дунёси	Частицы мир организмов,	Particles world of organisms,	Заррачаларни қамровчи олам	Particles captivating world
51.	Zoonematodalar	Зоонематодалар	Zoonematodalar	Ҳайвонларда паразитлик қилувчи нематодалар	Animal parasitic nematodes
52.	Iksodit	Иксодит	Iksodit	Каналар тури	Canada
53.	Имага	Изображение	Image,	Вояга этган кана	*Adult mites*
54.	Ixtiozavr	Ихтиозавр	Ixtiozavr		
55.	Ixtiologiya	Ихтиология	Ixtiologiya	Балиқларни ўрганувчи фан	Fish Sciences
56.	Канна	Канны	Cannes	Ўргамчаксимонлар туркумлари	O'rgamchaksimonlar categories
57.	Кемирувчису	Грызуны	Rodents	Ҳашоратларнинг	The structure of

	рувчи	куколки.	dollies.	озиқланишга кўра оғз опаратининг тузилиши	the mouth by feeding on insects operator
58.	Кемирувчи	Грызуны,	Rodents,	Ҳайвонларнинг озиқланишга кўра оғз опаратининг тузилиши	The structure of the mouth of the feeding of the animals operato
59.	Qizilungach	Қизилунгач	Qizilungach	Ҳалқум билан ошқозон ўртасидаги ичак	Nasopharyngeal intestine between the stomach
60.	Қийшиқ(dioganal)	Косые (диоганал)	Oblique (dioganal)	Мускул тури	muscle type
61.	Qilbosh гижжа	Қилбошчервь.	Qilbosh worm.	Бош қисми қилга ўхшаш нематода	Like nematodes
62.	Kiprikchalar	Киприкчалар	Kiprikchalar	Ҳаракат ва ҳимоя вазифасини бажарувчи орган	And acting to protect the body
63.	Клоака	Клоаки,	Cloaca,	Айриш, кўпайиш ва овқат ҳазм қилиш органлари битта тешкдан	Some of the reproductive and digestive organs of a teshkdan
64.	Knidostporidiyalar	Книдостпориди ялар	Knidostporidiyalar	Содда ҳайвонлар синфи	A simple class
65.	Қорамол ва чўчқа solityori	Говядины и свинины солитёри	Beef and pork solityori	Личинкалик шакли қорамол ва чўчқада кечувчи цестод	Larvae of cattle and pigs tsestod
66.	Koratsidiy	Корацидий	Koratsidiy	Уч хўжайинли цестодларнинг личинкаси	Three master tsestodlarning larvae
67.	Larvotssta	Ларвоцста	Larvotssta	Цестод личинкаси.	Tsestod larvae.

68.	Марита	Марита	Marita	Воягаетган гельминт.	Voyagaetgan infection.
69.	Maritogoniya	Маритогония	Maritogoniya	Гельминтнинг вояга етиш даври.	Worm-adult period.
70.	Marshallagiya	Маршаллагия	Marshallagiya	Ошқозон-ичак нематодаси	Gastro-intestinal nematodes
71.	Metatserkariy	Метацеркарий	Metatserkariy	Трематодларнинг иккинчи оралиқ хўжайинида етилган юқумли личинкаси.	Trematodes second husband, infectious larvae.
72.	Miratsidiy	Мирацидий	Miratsidiy	Трематодаларнинг тухумларида етиладиган личинка.	Eggs will larval trematodes.
73.	Monieziyalar	Мониезиялар	Monieziyalar	Йирик ва майда шохли ҳайвонларнинг ичак паразити	Large and small shoxlihayvonlarningichakparaziti
74.	Monogneya	Моногнея	Monogneya	Бир сўрғичли трематода	A suction trematode
75.	Monofag	Монофаг	Monofag	Бир турдаги озиқа билан озиқланувчилар	A type of food oziqlanuvchilar
76.	Multitseps	Мультицепс	Multitseps	Йиртқич ҳайвонларнинг ичагида паразитлик қилувчи цестод	Wild animal intestine parasites tsestod
77.	Нематода	Нематода	Nematode	Юмолоқ чувалчанг	Yumoloq worm
78.	Нематодир	Нематода	Nematode	Ингичка ичак нематодаси	Intestinal nematodes

79.	Onkosfera	Онкосфера	Onkosfera	Цестодларнинг 6 илмоқчали юқумли личинкаси	Tsestodlarning 6 ilmoqchali infectious larvae
80.	Оралиқ хўжайин	В промежуточном хозяине,	In the intermediate host,	Икки ва ундан ортиқ тараққиёт босқичига эга бўлган паразитларнинг вояга етмаган даврини ўтовчи организмлар	Having two or more stage of development, it became a minor cycle of organisms, parasites
81.	Orientobilgartsiya	Ориентобильгарция	Orientobilgartsiya	Вена қонтомирлари трематодаси	Vienna qontomirlari trematodes
82.	Ostrakum	Остракум	Ostrakum	Отларнинг йғон ичак нематодаси	Horses yg'on intestinal nematodes.
83.	Паразитизм	Паразитизм	Parasitism	Текинхўрлик билан ҳаёт кечирувчи паразитлар.	Of life is detrimental interference.
84.	Paramfistomatidlar	Парамфистоматидлар	Paramfistomatidlar	Ковшовчи ҳайвонларнинг овқат ҳазм қилиш органларининг трематодалари.	Trematodes Kovshova animals' digestive organs.
85.	Partenogenez	Партеногенез	Partenogenez	Тухум ҳужайраларини оталанмасдан кўпайиши (қизлигичак ўпайиш).	An increase in egg cells otalanmasdan (qizligicha reproduction).
86.	Партеногенетик тараққиёт	Развитие partenogeneti	Partenogenetik	Трематодларнинг биринчи оралиқ хўжайин	Trematodlarningbirinchioraliqxo'jayinorganizm

		k	development	организмида кўпайиш даври.	idako'payishdavri.
87.	Partenogoniya	Партеногония	Partenogoniya	Жинссиз кўпайишнинг бир усули	One way to increase sexual
88.	Телоparuterinli	Парутеринли орган	Body paruterinli	Авителлинидларнинг (цестод) бачадонида ҳосил бўладиган паренхиматозлика псулалар (бачадондаги тухумлар тушадиган орган)	Avitellinidlarning (tsestod) womb parenximatozli capsules (body drop the egg in the uterus)
89.	Polifag	Плероцеркоид	Polifag	Уч хўжайинли (псевдофиллидиялар) цестдларнинг иккинчи оралиқ хўжайинида етилган юқумли личинка	Three owners (psevdofillidiyalar) tsestdlarning second husband, infectious larval
90.	Polixeta	Полихета	Polixeta	Кўп қиллилар	cials
91.	Пронефрос	Предпочка	Pronephros	Оддий буйрак	Normal kidney
92.	Protozoologiya	Протозоология	Protozoologiya	Содда ҳайвонлар типи	Simple type
93.	Protonefridial	Протонефридиал	Protonefridial	Паразит чувалчангларнинг айириш органлари.	Parasitic worm crash bodies.
94.	Protostrongilidlar	Протостронгилидлар	Protostrongilidlar	Қуй ва эчкиларнинг нафас олиш органининг бионематодалари.	Sheep and goats respiratory bionematodalari.
95.	Psevdofillidiya	Псевдофилли	Psevdofillidi	Цестодлар	Tsestodlar Class

		дия	ya	синфининг туркуми	Category
96.	Psoroptid	Псороптид	Psoroptid	Микроскопик кана тури	Type of microscopic mite
97.	Reding	Редия	Reding	Трематодаларнинг биринчи оралиқ хўжайинида етилган 3-чи авлод личинкаси.	The first owners of the 3rd generation larvae of trematodes.
98.	Рептилия	Рептилии,	Reptiles,	Судралиб юрувчилар	reptile
99.	Ришта (ddrakunkula)	Связь (ракункула)	The link (drakunkula)	Одам тери ости кличаткасида нематодаси	Subcutaneous klichatkasida nematodes
100	Сазан	Карп	Carp	Балиқ тури	type of fish
101	Sarig'don	Сариғдон	Sarig'don	Урғочилик органи	female body
102	Sarkodalilar	Саркодали	Sarkodalilar	Содда ҳайвон	simple animal
103	Sarkoptid	Саркоптид	Sarkoptid	Микроскопик кана тури	Type of microscopic mite
104	Сербаргижжа	Сербскихчервей,	Serbian worms,	Цестод тури	Tsestod
105	Синф	Класс,	Class,	Систематик категориялар	systematic Categories
106	Систематик категориялар	Систематические категории	Systematic categories	Систематик категориялар	systematic Categories
107	Skate	Скатлар	Skate	Тоғайли балиқлар туркуми	Underlie a series of fish
108	Skoleks	Сколекс	Skoleks	Цестоднинг	Tsestodningbos

				бошчаси	hchasi.
109	Spikula	Спикула	Spikula	Эркак нематодаларнинг қўшилиш жараёнида иштирок этувчи органи.	Male nematodes involved in the process of joining the body.
110	Sporalilar	Споралилар	Sporalilar	Содда ҳайвонлар синфи	A simple class
111	Stileziya	Стилезия	Stileziya	Кавшовчи ҳайвонларнинг ичак цестод	Rumen intestinal tsestod
112	Стробила	Стробила	Strobila	Цестод танаси	Tsestod body
113	Strongilidlar	Стронгилидлар	Strongilidlar	Нематодалар синфининг оиласи	Nematodes class families
114	Суякли балиқлар	Костейрыб,	Fish bones,	Балиқлар синфи	Fish Class
115	Teniatlar	Тениатлар	Teniatlar	Цестодлар синфинин кичкина туркуми	Tsestodlar a series of small sinfinin
116	Teniidlar	Тениидлар	Teniidlar	Цестодлар синфининг кичкина оиласи	Tsestodlar class of small family
117	Тери-мускул халта	Кожно-мускульный мешок,	Skin-muscular sac,	Териси мускул вазифасини бажаради	Skin, muscle function
118	Tizaniyziya	Тизаниезия	Tizaniyziya	Кавшовчи ҳайвонларнинг ичак цестоди	Rumen intestinal tsestodi
119	Тип	Класс	Class	Систематик катигория	systematickatigoriya

120	Трахия,	Трахеи,	Of the trachea,	Ҳашоратлар нафас олиш органи	Insects respiratory organ
121	Трематодалар	Трематоды	Trematodes	Ясси чувалчанглар типининг синфи.	Flat worms typical class.
122	Трихинелла	Трихинеллы	Trichinella	Одам, чўчқа, ит, каламушларда ривожланувчи нематода	Humans, Pig, dogs, rats in the developing nematode
123	Trixotsefallar	Трихоцефаллар	Trixotsefallar	Одам ичагида яшовчиқил бош нематода	People living in the intestine nematode
124	Tugarak og'izlilar	Тугаракоғизлилар	Tugarakog'izlilar	Оғзи тўғарак шаклда	The mouth of the round form
125	Тур	Видов,	Species,	Систематик катигория	systematic katigoriya
126	Туркум	Серия,	The series,	Систематик катигория	systematickatigoriya
127	Тухумдон	Яичники,	Ovaries,	Туҳум ишлаб чиқарувчи орган	Egg production unit
128	Uvildiriq	Увилдириқ	Uvildiriq	Балиқ нинг тухуми	Fish eggs
129	Умуртқа	Позвоночника,	Of the spine,	Ўқ склети	Bullet sclera
130	Уруғдон йўллари	Яйцеклеток,	Ovules,	Уруғ чиқарувчи йўл	The seed producer
131	Уруғдон	Яйцеклеток,	Ovules,	Эркаклик маҳсулоит ишлаб чиқарувчи орган	The production of men's body
132	Ўргимчак	Паук,	Spider,	Ўрмалаб юрувчи ҳайвон	Animal reptile
133	Ўсимликлар	Флора,	Flora,	Ўсмликлар олами	Accessories

	дунёси				Fashion
134	Ўсиш зона	Зона,	Zone,	Цестод бўйинчаси	the neck of the Tsestod
135	Фаланга	Паралич,	Paralysis,	Ўргамчаксмонлар вакили	representative O'rgamchaksmonlar
136	Фасциолалар	Фасциола	Fasciola	Жигар трематодалари	liver trematodes
137	Ascioliasis	Фасциолёз	Ascioliasis	Фасциолалар чақирадиган жигар трематодози	Call fasciola liver trematodozi
138	Финноз	Противкори	Measles	Қорамол ва чўчқа цистицеркозлари	Cattle and Pig cysticercosis
139	Fitogelmintlar	Фитогельминтлар	Fitogelmintlar	Ўсимлик гельминтлари	plant helminths
140	Fitomastigina	Фитомастигина	Fitomastigina	Ўсмликлар паразити	teenage parasite
141	Fitonematodalar	Фитонематодалар	Fitonematodalar	Ўсимлик нематодалари	plant nematodes
142	Fitofaglar	Фитофаглар	Fitofaglar	Ўсимликхўрлар.	O'simlikxo'rlar.
143	Ҳайвонот дунёси	Животные,	Animals,	Ҳайвонлар олами	The world of animals
144	Халқали чувалчанглар	Черви,	Worms,	Халқалардан ташкил топган чувалчанглар	Cycle of the worms
145	Xelitsera	Хелицера	Xelitsera	Тирноқсимон тиш	Tirnoqsimon tooth
146	Xivchinlilar	Хивчинлилар	Xivchinlilar	Ипли паразитлар	cotton parasites
147	Хитин	Хитин,	Chitin,	Қаттиқ пўст	peeling of the solid

148	Jordi,	Хорда	Jordi,	Йирик ҳайвонлар ўқ склетга эга	The largest lead sclera
149	Xordalilar	Хордалилар	Xordalilar	Йирик ҳайвонлар ўқ склетга эга	The largest lead sclera
150	Xorioptid	Хориоптид	Xorioptid	Микроскопик кана	microscopic mite
151	Ценур	Центов	Cents	Ёш ҳайвонларнинг бош (айрим ҳолларда орқа) мияларида ривожланувчи цестод multiceps multiceps нинг пуфагсимон личинкаси.	Young animals (in some cases, back) tsestod brain development multiceps multicepsning pufagsimon larvae.
152	Tserkariy	Церкарий	Tserkariy	Трематодаларнинг биринчи оралиқ хўжайинида етилган 4-чи авлод личинкаси.	The first owners of the 4th generation larvae of trematodes.
153	Tsestodlar	Цестодлар	Tsestodlar	Ясси чувалчанглар типининг синфи (лентасимон чувалчанглар).	Yassichuvalchanglar type of class (a strip of worms).
154	Tsikloid	Циклоид,	Tsikloid	Балиқ тангачаси	fish, peeled
155	Tsiklofillidiya	Циклофиллидия	Tsiklofillidiya	Цестодлар синфининг энг йирик туркуми	A series of the largest Tsestodlarsinfining
156	Tsirrus	Циррус,	Tsirrus	Эркаклик халтас	Men's Bag
157	Tsistitserk(фи	Цистицерк(ф	Tsistitserk	Цестодларнинг пуфагсимон	Tsestodlarning pufagsimon

	нна)	инский)	(finnish)	личинкаси	larvae
158	Tsistogoniya	Цистогония	Tsistogoniya	Трематодаларнинг сўнги личинкалик тараққиёт босқичи	The last larvae stage of development of trematodes
159	Чаён	Скорпион,	Scorpion,	Бўғиноёқлилар вакиллари	Bo'g'inoyoqlilar
160	Чиғаноқ	Оболочки,	Shell,	Қаттиқ пўст	peeling of the solid
161	Чин кожа тери	Натуральная тери-кориум	Genuine leather korium	Ҳақиқий тери	genuine leather
162	Shistosomatidlar	Шистосоматидлар	Shistosomatidlar	Трематодалар синфининг оиласи (шистосоматитлар кенжа туркумига оид).	Trematodes class families (shistosomatitlar sub-category).
163	Экскретор	Отходы за	Excrete	Чиқариш тешиги	anus
164	Эктодерма	Эктодерма,	Ectoderm,	Ташқи қават	the outer layer
165	Embriogoniya	Эмбриогония	Embriogoniya	Зиготанинг шакилланиш босқичи	The zygote formation stage
166	Эмбрион	Эмбриона,	Embryo,	Зиготанинг шакилланиши	the structure of the zygote
167	Энг содда ҳайвонлар	Простой,	Simple,	Бир ҳужайрали ҳайвонлар	Single-celled animals
168	Endoparazit	Эндопаразит	Endoparazit	Ички паразит	internal parasites
169	Entoderma	Энтодерма	Entoderma	Ички қават	interior floor
170	Энтомология	Энтомологи	Of entomology,	Ҳашоратларни ўрганувчи фан	Insects Sciences

171	Exinokokk	Эхинококк	Exinokokk	1) Цестоднинг ит ва бошқа йиртқич ҳайвонлардаги вояга етгани. 2) Ўтхўр ҳайвонларнинг ва одам ички органларининг (жигар, ўпка ва ҳокозо)даги вояга етган эхинококкнинг пуфагсимон личинкаси.	Tsestodning dogs and other wild animals adult. 2) herbivorous animal and human internal organs (liver, o'pkavahokozo) in adult echinococcus pufagsimon larvae.
172	Эхинококкоз	Эхинококковая	Echinococcus	Эхинококклар чақирадиган касаллик.	Calls on the echinococcus disease.
173	Exinostomatidlar	Эхиностоматидлар	Exinostomatidlar	Паррандаларнинг трематодалари	trematodes in birds
174	Юмалоқ чувалчанглар	Круглые черви,	Round worms,	Нематодалар	nematodes
175	Ясси чувалчанглар	Плоские черви,	Flatworms,	Яссилашган чувалчанг	flattened worm

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CERTIFICATION QUESTIONS ON THE SUBJECT:

IV. 1 INTERMEDIATE CONTROL ORAL QUESTIONS (120)

1-IC ORAL QUESTIONS

1. What is the helminthoprological method?
2. Tell us the method of sequential washing?
3. Tell us the method of native smear?
4. Tell the Fulleborn method?
5. Tell the Darling method?
6. Tell the Shcherbovich method?
7. Tell the Kalantaryan method?
8. Tell the Berman-Orlov method?
9. Tell the simplified helmintholaroscopy method?
10. Tell us the method of sedimentation with centrifugation?
11. Tell us the method of a complete helminthological autopsy?
12. Tell us about the incomplete helminthological autopsy?
13. Tell us a complete helminthological autopsy of individual organs?
14. When testing the effectiveness of new anthelmintics, a graduate student or doctoral student uses which autopsy method?
15. For the preparation of museum material, what method of dissection of animals is necessary?
16. What is the difference between the method of complete helminthological autopsy and the method of complete helminthological autopsy of individual organs?
17. Characteristics of fascioliasis?
18. How are the pathogens of fascioliasis located according to taxonomy?
19. What is the difference between ordinary fasciola and giant fasciola?
20. When does the infection of animals with fascioles occur and by what ways do young fascioles migrate to the liver?
21. Laboratory methods of diagnosis of fascioliasis?
22. From which diseases should fascioliasis be differentiated?
23. How is fascioliasis prevented?
24. Describe the characteristics of the disease.
25. Tell us the taxonomy of the pathogen.
26. Representatives of how many families and genera belonging to the order Paramphistomatata parasitize in the pre-ventricles of cattle.
27. Briefly describe the anatomical and morphological structure of the exciter.
28. How does the causative agent of the disease develop?
29. How is the diagnosis made and what diseases should be differentiated from?
30. How is the disease prevented?
31. Tell us about health-improving measures for paramphisto-matosis?
32. Describe the characteristics of orientobilcharciosis?
33. Tell us the systematics of Orientobilharzia turkestanica ?
34. How does Orientobilharziaturkestanica develop?
35. How do we diagnose orientobilharziosis and from which diseases should we differentiate?
36. Tell us about the wellness activities of the farms from orientobilkhartsioz?

37. Tell me the characteristics of dicrocenliosis.
38. Tell us the taxonomy of the pathogen.
39. Briefly tell us the anatomical and morphological structure of the exciter and its biological development?
40. How do we diagnose and from which diseases should we differentiate?
41. How is the disease prevented?
42. Tell us about health-improving measures for dicroceliosis?
43. Describe the characteristics of eurythrematosis?
44. Tell us the taxonomy of Euritremapancreaticum?
45. Tell us about the anatomical and morphological structure of Euritremapancreaticum?
46. How does Euritremapancreaticum develop?
47. How to diagnose euritrematosis and from which diseases should it be differentiated?
48. Tell us about the health-improving activities of farms from eurythrematosis?
49. What type and class do cestodes belong to?
50. Which detachments of cestodes have veterinary and medical significance?
51. How do these two detachments differ in morphological structures?
52. How do these two detachments differ in biological development?
53. The body of the cestode consists of how many parts?
54. What function do cuticles perform in cestodes?
55. Tell us about the biological development of cestodes?
56. Tell us about the main stages of the larval form in the chains in the body of intermediate hosts?
57. Tell us the main stages of the larval form in the lentets in the body of intermediate hosts?
58. What is the difference between the eggs of the order of the chain from the order of the lenzets?
59. How are the pathogens of cysticercosis of cattle and pigs located according to the systematics?
60. Tell us the anatomical and morphological structures of Cysticercus bovis and its differences from Cysticercus cellulosae?
61. Tell us the anatomical and morphological structures of Taeniarhynchus saginatus and its differences from Taenia solium?
62. Tell us the biology of the causative agent of cattle cysticercosis and its differences from pig cysticercosis?
63. How do we diagnose cysticercosis during the life of an animal?
64. How do we treat sick people with teniarynchosis and teniosis?
65. How do we prevent cysticercosis?
66. If a sick animal with cattle or pigs cysticercosis appeared on the farm, what should we do, what measures should we take to improve the farms from the disease?
67. Give a description of echinococcosis?
68. 68. How is Echinococcus granulosus located according to the taxonomy?

69. Anatomical and morphological structure of Echinococcus granulosus?
70. Biological development of the pathogen of echinococcosis?
71. Lifetime diagnosis of echinococcosis?
72. Give a characterization of the price tag?
73. How is the causative agent of cenurosis located according to the systematics?
74. Anatomical and morphological structure of Coenurus cerebralis and Multiceps multiceps?
75. Biological development of the causative agent of sheep cenurosis?
76. How do we diagnose cenurosis during life?
77. How should we deal with echinococcosis and cenurosis of animals?
78. How many species of teniids are parasitic in dogs?
79. How is the diagnosis made with teniidosis of dogs?
80. How is preventive deworming performed?
81. What anthelmintics are recommended for deworming dogs, name them, tell us their doses and application schemes?
82. How many times a year to carry out deworming of dogs against teniids?
83. Tell us the features of summer deworming?
84. How many representatives of the families of the order Anoplocephalata are parasitic in ruminants?
85. How many representatives of the genera of the Anoplocephalidae family parasitize ruminants?
86. How do representatives of the Anoplocephalidae family differ from Avitellinidae?
87. How do we distinguish Moniezia expansa from Moniezia benedeni?
88. How do we distinguish Moniezia expansa from Thysaniezia giardi.
89. How do we distinguish Thysaniezia giardi from Avitellina centripunctata and Stilesia globipunctata?
90. Will you tell us about the biological development of the causative agents of moniesiosis and its differences from the causative agents of tizanieziosis, avitellinosis and stilesiosis?
91. How is the diagnosis made with anoplocephalatosis of ruminants?
92. From which diseases should anoplocephalatosis of ruminants be differentiated?
93. How do we prevent anoplocephalatosis of animals?
94. Tell us the methods of chemoprophylaxis of anoplocephalatosis of ruminants and the mixtures used?
95. Give a description of anoplocephalidosis of horses?
96. Tell us the taxonomy of the pathogens of equine anoplocephalidosis?
97. Tell us the anatomical and morphological structure and biological development of pathogens of anoplocephalidosis of ungulates?
98. How many representatives of the genera of the Anoplocephalidae family parasitize ruminant horses?
99. Tell us the methods of lifetime diagnosis of anoplocephalid horses?
100. From which diseases should anoplocephalidoses of horses be differentiated?

101. How do we prevent anoplocephalidosis of horses, if the disease has arisen, what kind of health measures should we take?
102. What type and class are roundworms?
103. How many sub-orders of the nematode class have veterinary and medical significance?
104. Tell the anatomical and morphological structure of nematodes?
105. Tell the biological development of nematodes?
106. Tell the biological features
107. Tell us the biological features of the development of bioonematodes?
108. Representatives of which of the nematode suborders parasitize in the closed organs and tissues of the definitive host?
109. When parasitizing, which suborder representatives of the same organism is the definitive and intermediate host?
110. Describe the parascaridosis of horses?
111. Tell us the systematics of the causative agent of parascaridosis?
112. Tell us the morphological structure of Parascarisequorum?
113. Tell us the biological development of Parascarisequorum?
114. Coprological method of diagnosing parascaridosis of horses?
115. From which invasive and non-infectious diseases should parascaridosis be differentiated?
116. Give a description of pig ascariasis?
117. Tell us the systematics of the causative agent of ascariasis?
118. Tell us the morphological structure of Ascarissuum?
119. Tell us the biological development of Ascarissuum?
120. Coprological method for diagnosing pig ascariasis?

4.2 II-INTERMEDIATE CONTROL

Oral questions

1. Tell us the taxonomy of pathogens of protozoal animal diseases?
2. Tell us the morphological structure of the simplest organisms?
3. Tell us about the biological development of the simplest organisms in a specific example?
4. How is the diagnosis made during the life of an animal in laboratory conditions?
5. Tell us about microscopic methods of diagnosis of protozoal diseases?
6. How and where does blood for smears come from, methods of their fixation and coloring?
7. Which method is the most common method for staining smears?
8. How is the diagnosis made after the death of an animal? What do we send to the laboratory to clarify the diagnosis of protozoal diseases of animals?
9. What does veterinary arachnology study?
10. What type, class and unit do ticks belong to?
11. Are parasitoform mites the causative agents of the disease or their carriers?
12. In biological development, how many phases do ticks of the Ixodidae and Argasidae family go through?
13. Where are ticks of the Ixodidae family in animals?
14. Where are the ticks of the Argasidae family in birds?
15. Give a description of the pyroplasmosis of cattle?
16. How is the causative agent of piroplasmosis of cattle located according to taxonomy?
17. Tell us the morphology of Piroplasmabigeminum, its biological development in the body of animals and vector ticks?
18. Tell us the laboratory methods of lifetime diagnosis of piroplasmidoses?
19. How do we distinguish Piroplasmabigeminum from other types of piroplasmids?
20. In our Republic, there is babesiosis of cattle, if not, then why, explain?
21. How do we treat piroplasmidosis of cattle?
22. The main pathoanatomic changes in pyroplasmosis?
23. How do we fight against piroplasmidosis of cattle?
24. Tell us the essence of the mitigating chemoprophylaxis of pyro-plasmidosis of animals?
25. What is the characteristic of bovine taileriosis?
26. How is the causative agent of bovine taileriosis located according to taxonomy?
27. Which pathogens of teileriosis are very pathogenic for animals?
28. Tell us the morphology of Theileria annulata and its biological development in the body of animals and vector ticks?
29. Tell us the morphology of Theileria sergenti and its biological development in the body of animals and vector ticks?
30. Tell us the laboratory methods of lifetime diagnosis of teileriosis?

31. The main pathoanatomical changes in teileriosis?
32. Tell us the atypical form or course of teileriosis and how is the diagnosis made in this course of the disease?
33. How do we treat cattle teileriosis?
34. Tell us a 3-day course of treatment for teileriosis?
35. How do we fight with bovine taileriosis?
36. Tell us the essence of vaccination of calves against teileriosis?
37. Tell us the taxonomy of the causative agents of eimeriosis?
38. How do representatives of the Eimeriinae subfamily develop and how do they differ from representatives of the Isosporinae subfamily?
39. Where do the eimeria parasitize and tell the biological development of the eimeria in the external environment and in the animal body?
40. How many cycles differ in the life cycle of eimeria?
41. Tell us the characteristics of rabbit eimeriosis?
42. How are the pathogens of rabbit eimeriosis located according to taxonomy?
43. Methods of lifetime diagnosis of rabbit eimeriosis and from what diseases should it be differentiated?
44. How do we treat sick rabbits with eimeriosis, the drugs used, the dose and the schemes of its use?
45. How do we prevent eimeriosis of rabbits?
46. Eimeriosis appeared in the farm, how should we fight it, what are the health measures?
47. Tell us the characteristics of the su-aura?
48. How is the pathogen of the su-aura located according to systematics, tell the morphology and biology of the pathogen?
49. How is the diagnosis made during the life of the animal on the su-aura?
50. Postmortem diagnosis of the su-aura?
51. From which diseases should the su-aura be differentiated?
52. How to treat animals with trypanosomiasis, are specific and symptomatic methods of treatment used?
53. How to improve the economy if the disease has appeared?
54. How to prevent the farm from trypanosomiasis?
55. Tell us the characteristics of the accidental disease of horses?
56. How is the causative agent of accidental horse disease located according to taxonomy, tell us the morphology and biology of the pathogen?
57. How is the diagnosis made during the life of an animal of accidental horse disease?
58. Accidental horse disease should be differentiated from what diseases?
59. How to treat patients with accidental animal disease, specific and symptomatic treatment methods used?
60. How to improve the economy if the disease has appeared?
61. How to prevent the farm from accidental illness?
62. Tell us the characteristics of trichomoniasis of cattle

63. How is the causative agent of trichomoniasis located according to taxonomy, tell us the morphology and biology of the pathogen?
64. Laboratory methods of diagnosis of trichomoniasis?
65. From which diseases should trichomoniasis of cattle be differentiated?
66. How do we treat cows and bulls with trichomoniasis, the specific and symptomatic methods and treatment regimens used by them?
67. How to improve the economy if the disease has already appeared?
68. How to prevent the farm from trichomoniasis?
69. What is the veterinary significance of parasitoform and acariform mites, Give examples?
70. The life expectancy of acariform mites in different species?
71. Tell us the taxonomy and anatomical and morphological structures of ticks of the Psoroptidae family
72. Measures to combat psoroptosis of animals?
73. Tell us the characteristic clinical signs of psoroptosis?
74. How to diagnose sarcoptosis and chorioptosis?
75. What is the difference between psoroptosis and sarcoptosis and chorioptosis of animals?
76. How to diagnose demodicosis of dogs and cattle?
77. How do we treat demodecosis of dogs?
78. Preventive measures to combat demodicosis of dogs?
79. How to diagnose knemidocoptosis of birds?
80. Tell us about the health-improving activities carried out on the farm, with the appearance of knemidocoptosis of birds?
81. What is the difference between knemidocoptosis and syringophilosis and cytoditosis of chickens?
82. What is the characteristic of the insect class?
83. How is the reproduction and development of insects?
84. Blood-sucking diptera insects (gnus) and their main distinguishing features?
85. What are the main directions of the fight against midge and protection of animals from it?
86. Features of the biology of bloodsuckers and pathogens of mellophagosis, siphunculatosis and vermipsilesis of animals.
87. Biological justification of the fight against fleas and bedbugs.
88. Give a description of hypodermatosis of cattle?
89. How is the causative agent of hypodermatosis of cattle located according to the systematics?
90. Morphological structures of Hypodermabovis and its differences from Hypodermalineatum?
91. Biological development of pathogens Hypodermabovis and Hypodermalineatum?
92. Tell us the methods of lifetime diagnosis of hypodermatosis of cattle?
93. Tell us the methods of immunological diagnosis?

94. How do we treat animals with hypodermatosis, the insecticides used, their doses and methods of application?
95. How do we prevent cattle from hypodermatosis?
96. Give a description of sheep estrosis?
97. How is the causative agent of estrosis located according to the systematics?
98. Morphological structure of Oestrusovis?
99. Biological development of the pathogen Oestrusovis?
100. Tell us the methods of lifetime diagnosis of sheep estrosis?
101. What is the difference between sheep estrosis and cenurosis?
102. How do we treat animals with estrosis, the insecticides used, their doses and methods of application?
103. How do we prevent sheep from estrosis?
104. Give a description of rhinestrosis of horses?
105. Give a description of gastrophylosis of horses?
106. How is the systematics of the causative agents of horse rhinestrosis located?
107. How is the taxonomy of the pathogens of gastrophylosis of horses?
108. Morphological structure of Rhinoestrus purpureus?
109. Morphological structure of Gastrophilusintestinalis?
110. Biological development of the pathogen Rhinoestruspurpureus?
111. Biological development of the pathogen Gastrophilusintestinalis?
112. Tell us the methods of lifetime diagnosis of rhinestrosis and gastrophylosis of horses?
113. How do we treat patients with estrosis and gastrophylosis of animals, the insecticides used, their doses and methods of application?
114. How do we prevent horses from rhinestrosis?
115. How do we prevent horses from gastrophylosis?
116. Give a description of camel cephalopinosis?
117. How is the causative agent of camel cephalopinosis located according to the taxonomy?
118. Morphological structures of Cephalopinatitilator?
119. Biological development of the causative agent Cephalopinatitilator?
120. Tell us the methods of lifetime diagnosis of camel cephalopinosis?

4.3 FINAL CONTROL

Final oral questions

1. What is the helminthoprological method?
2. Tell us the method of sequential washing?
3. Tell us the method of native smear?
4. Tell the Fulleborn method?
5. Tell the Darling method?
6. Tell the Shcherbovich method?
7. Tell the Kalantaryan method?
8. Tell the Berman-Orlov method?
9. Tell the simplified helmintholaroscopy method?
10. Tell us the method of sedimentation with centrifugation?
11. Tell us the method of a complete helminthological autopsy?
12. Tell us about the incomplete helminthological autopsy?
13. Tell us a complete helminthological autopsy of individual organs?
14. When testing the effectiveness of new anthelmintics, a graduate student or doctoral student uses which autopsy method?
15. For the preparation of museum material, what method of dissection of animals is necessary?
16. What is the difference between the method of complete helminthological autopsy and the method of complete helminthological autopsy of individual organs?
17. Characteristics of the disease?
18. How are the pathogens of fascioliasis located according to taxonomy?
19. What is the difference between the common fasciola and the giant fasciola?
20. When does the infection of animals with fascioles occur and by what ways do young fascioles migrate to the liver?
21. Laboratory methods of diagnosis of fascioliasis?
22. From which diseases should fascioliasis be differentiated?
23. What anthelmintics are used for fascioliasis, their doses and methods of application?
24. How is fascioliasis prevented?
25. Describe the characteristics of the disease.
26. Tell us the taxonomy of the pathogen.
27. Representatives of how many families and genera belonging to the order Paramphistomatata parasitize in the pre-ventricles of cattle.
28. Briefly describe the anatomical and morphological structure of the exciter.
29. How does the causative agent of the disease develop?
30. How is the diagnosis made and from which diseases should we differentiate?
31. Tell us about the methods of treatment of paramphistomatosis?
32. How is the disease prevented?
33. Tell us about health-improving measures for paramphisto-matosis?
34. Describe the characteristics of orientobilcharciosis?
35. Tell us the taxonomy of Orientobilharzia turkestanica?

36. How is Orientobilharziaturkestanica developing?
37. How do we diagnose orientobilcharciosis and from which diseases should we differentiate?
38. Tell us the method of treatment of orientobilcharciosis?
39. Tell us the health measures of the farms from orientobilcharciosis?
40. Tell me the characteristics of dicrocenliosis.
41. Tell us the taxonomy of the pathogen.
42. Briefly tell us the anatomical and morphological structure of the exciter and its biological development?
43. How do we diagnose and from which diseases should we differentiate?
44. Tell us the methods of treatment of dicroceliosis?
45. How is the disease prevented?
46. Tell us about health-improving measures for dicroceliosis?
47. Describe the characteristics of eurythrematosis?
48. Tell us the taxonomy of Euritremapancreaticum?
49. Tell us about the anatomical and morphological structure of Euritremapancreaticum?
50. How does Euritremapancreaticum develop?
51. How to diagnose euritrematosis and from what diseases should it be differentiated?
52. Tell us about the health-improving activities of farms from eurythrematosis?
53. What type and class do cestodes belong to?
54. Which cestod detachments have veterinary and medical significance?
55. How do these two orders differ in morphological structures?
56. How do these two groups differ in biological development?
57. The body of the cestode consists of how many parts?
58. What function do cuticles perform in cestodes?
59. Tell us about the biological development of cestodes?
60. Tell us about the main stages of the larval form in the chains in the body of intermediate hosts?
61. Tell us the main stages of the larval form in the lentets in the body of intermediate hosts?
62. What is the difference between the eggs of the chain squad and the Lenzets squad?
63. How are the causative agents of cysticercosis of cattle and pigs located according to the systematics?
64. Tell us the anatomical and morphological structures of Cysticercus bovis its differences from Cysticercus cellulosae?
65. Tell us the anatomical and morphological structures of Taeniarhynchus saginatus and its differences from Taenia solium?
66. Tell us the biology of the causative agent of cattle cysticercosis and its differences from pig cysticercosis?
67. How do we diagnose cysticercosis during the life of an animal?
68. How do we treat sick people with teniarrhosis and teniosis?

69. How do we prevent cysticercosis?
70. If a sick animal with bovine or pig cysticercosis has appeared on the farm, what should we do, what measures should we take to improve the farms from the disease?
71. Give a characterization of echinococcosis?
72. How is Echinococcus granulosus located according to the taxonomy?
73. Anatomical and morphological structure of Echinococcus granulosus?
74. Biological development of the pathogen of echinococcosis?
75. Lifetime diagnosis of echinococcosis?
76. Give a characterization of the price tag?
77. How is the causative agent of cenurosis located according to the systematics?
78. Anatomical and morphological structure of Coenurus cerebralis and Multiceps multiceps?
79. Biological development of the causative agent of sheep cenurosis?
80. How do we diagnose cenurosis during life?
81. How should we deal with echinococcosis and cenurosis of animals?
82. How many species of teniids are parasitic in dogs?
83. How is the diagnosis made with teniidosis of dogs?
84. How is preventive deworming performed?
85. What anthelmintics are recommended for deworming dogs, name them, tell us their doses and application schemes?
86. How many times a year to carry out deworming of dogs against teniids?
87. Tell us the features of summer deworming?
88. How many representatives of the families of the order Anoplocephalata parasitize ruminants?
89. How many representatives of the genera of the Anoplocephalidae family parasitize ruminants?
90. How do they distinguish representatives of the Anoplocephalidae family from Avitellinidae?
91. How do we distinguish Moniezia expansa from Moniezia benedeni?
92. How do we distinguish Moniezia expansa from Thysaniezia giardi.
93. How do we distinguish Thysaniezia giardi from Avitellina centripunctata and Stilesia globipunctata?
94. Tell us about the biological development of the causative agents of monieziosis and its differences from the causative agents of tizanieziosis, avitellinosis and styleziosis?
95. How is the diagnosis made with anoplocephalatosis of ruminants?
96. From which diseases should anoplocephalatosis of ruminants be differentiated?
97. Which anthelmintics are used for ruminant anoplocephaliasis?
98. Which of these anthelmintics are the most effective?
99. How do we prevent animal anoplocephaliasis?

100. Tell us the methods of chemoprophylaxis of ruminant anoplocephaliasis and the mixtures used?
101. Describe the anoplocephalidosis of horses?
102. Tell us the systematics of the pathogens of anoplocephalidosis of horses?
103. Tell us the anatomical and morphological structure and biological development of the pathogens of anoplocephalidosis of ungulates?
104. How many representatives of the genera of the family Anoplocephalidae parasitize ruminant horses?
105. Tell us the methods of lifetime diagnosis of anoplocephalidosis of horses?
106. From which diseases it is necessary to differentiate anoplocephalidosis of horses?
107. Treatment of equine anoplocephalidosis, which of the recommended drugs is the most effective anthelmintic?
108. How do we prevent equine anoplocephalidosis, if the disease has arisen, what kind of health measures should we take?
109. What type and class do roundworms belong to?
110. How many sub-orders of the nematode class have veterinary and medical significance?
111. Tell anatomo-morphological structure of nematodes?
112. Tell us the biological development of nematodes?
113. Tell us the biological features of the development of hematodes?
114. Tell us the biological features of the development of bioonematodes?
115. Representatives of which of the nematode suborders parasitize in the closed organs and tissues of the definitive host?
116. When parasitizing, which suborder representatives of the same organism is the definitive and intermediate host?
117. Describe the parascaridosis of horses?
118. Tell the systematics of the causative agent of parascaridosis?
119. Tell the morphological structure of Parascarisequorum?
120. Tell the biological development of Parascarisequorum?
121. Coprological method of diagnosing parascaridosis of horses?
122. From which invasive and non-infectious diseases should parascaridosis be differentiated?
123. How do we treat parascaridosis, tell us the doses and schemes of anthelmintics used?
124. Describe the ascariasis of pigs?
125. Tell us the taxonomy of the causative agent of ascariasis?
126. Tell us the morphological structure of Ascarissuum?
127. Tell us the biological development of Ascarissuum?
128. Coprological method for diagnosing ascaridosis in pigs?
129. From which invasive and non-infectious diseases should ascaridosis be differentiated?
130. How do we treat pig ascariasis, tell us the doses and schemes, used anthelmintics?

131. How to prevent ascariasis of pigs and parascaridosis of horses in dysfunctional farms for this helminthiasis?
132. How many species of ascaridates parasitize in the body of dogs and fur-bearing animals?
133. Tell us the taxonomy of the causative agents of ascaridatoses of dogs?
134. Tell us the anatomical and morphological structure of Toxocaracanis and Toxascarisleonine and how they differ from each other?
135. Tell us about the biological development of Toxocaracanis and Toxascarisleonina?
136. Methods of lifetime diagnosis of ascariasis of dogs?
137. What is the basis for postmortem diagnosis of ascariasis of dogs?
138. How do we treat ascariasis of dogs, what and in what doses and schemes are anthelmintics used against ascariasis of dogs and other fur-bearing animals?
139. Tell us the preventive measures carried out with ascariasis of dogs?
140. Tell us the systematics of pathogens of intestinal strongylatoses of ungulates?
141. Tell us about the biological development of pathogens of intestinal strongylatoses of horses, for example a specific species?
142. Methods of lifetime diagnostics of intestinal strongylatoses of horses?
143. Postmortem diagnostics of strongylatoses of digestive organs of ungulates?
144. How to make an accurate generic diagnosis of the pathogens of intestinal strongylatoses of horses?
145. How do we treat intestinal strongylatoses of ungulates, for example, a specific disease?
146. How are preventive measures carried out for intestinal strongylatoses of horses?
147. Tell us the systematics of pathogens of strongylatoses of digestive organs of ruminants?
148. Tell us about the biological development of pathogens of strongylatoses of digestive organs, for example a specific species?
149. Methods of lifetime diagnosis of strongylatoses of ruminants?
150. Postmortem diagnosis of strongylatoses of digestive organs of ruminants?
151. How to make an accurate generic diagnosis of pathogens of strongylatoses of ruminants?
152. How do we treat strongylatosis of the digestive organs of ruminants, using the example of a specific disease?
153. How do we prevent intestinal strongylatosis of ruminants?
154. How and with what mixture is chemoprophylaxis of strongylatosis of the digestive organs of ruminants carried out?
155. Tell us the systematics of pathogens of strongylatosis of the respiratory organs of animals?
156. Tell us the anatomical and morphological structure of Dictyocaulusfilarialud and its differences from Dictyocaulus viviparous?

157. Tell us the biological development of pathogens of strongylatosis of the respiratory organs, using the example of a specific species?
158. Methods of lifetime diagnosis of sheep and cattle dictyoculosis?
159. Postmortem diagnosis of sheep and cattle dictyoculosis?
160. How to make an accurate species diagnosis of pathogens of strongylatosis of the respiratory organs of animals?
161. How do we treat dictyoculosis of sheep and cattle?
162. How do we prevent dictyoculosis of sheep and cattle?
163. How and with what mixture is chemoprophylaxis of dictyoculosis of animals carried out?
164. How many types of telyazia parasitize in the eyes of cattle?
165. Give a description of the telyaziosis of cattle?
166. Tell us the taxonomy of the causative agents of telyaziosis?
167. Tell us the anatomical and morphological structures of Th. Rhodesi?
168. Tell us the biological development of the causative agents of thesiosis?
169. How is the diagnosis made during life for telyaziosis and its differentiation from other diseases?
170. How do we treat telyaziosis, the drugs used, the dose and the schemes of their use?
171. How do we prevent diseases?
172. Describe the trichinosis of pigs?
173. How many types of trichinella parasitize animals in general, and the most common type of pathogen?
174. Tell us the anatomical and morphological structure of Trichinellaspiralis?
175. Tell us the biological development of the pathogen Trichinellaspiralis?
176. Tell us about the lifetime diagnosis of trichinosis?
177. Tell us about the postmortem diagnosis of trichinosis?
178. What control measures are carried out in case of pig trichinosis?
179. Give a description of horse parascaridosis?
180. Tell us the systematics of the causative agent of oxyurosis?
181. Tell us the morphological structure of O.equi?
182. Tell us the biological development of O.equi?
183. Coprological method of diagnosing equine oxy-urosis?
184. From which invasive and non-infectious diseases should oxyurosis be differentiated?
185. How do we treat oxyurosis, tell us the doses and schemes of anthelmintics used?
186. Tell us the systematics of pathogens of protozoal animal diseases?
187. Tell us the morphological structure of protozoan organisms?
188. Tell us about the biological development of protozoan organisms in a specific example?
189. How is the diagnosis made during the life of an animal in laboratory conditions?
190. Tell us about microscopic methods of diagnosis of protozoal diseases?

191. How and where does blood for smears come from, methods of their fixation and coloring?
192. Which method is the most common method for staining smears?
193. How is the diagnosis made after the death of an animal? What do we send to the laboratory to clarify the diagnosis of protozoal diseases of animals?
194. What does veterinary arachnology study?
195. What type, class and order do ticks belong to?
196. Are parasitoform ticks the causative agents of the disease or their carriers?
197. In biological development, how many phases do the ticks of the Ixodidae and Argasidae family go through?
198. Where are the ticks of the Ixodidae family in animals?
199. Where are the ticks of the Argasidae family in birds?
200. Give a description of the pyroplasmosis of cattle?
201. How is the causative agent of piroplasmosis of cattle located according to systematics?
202. Tell the morphology of Piroplasmabigeminum its biological development in the body of animals and vector ticks?
203. Tell the laboratory methods of lifetime diagnosis of piroplasmidosis?
204. How do we distinguish Piroplasmabigeminum from other types of piroplasmids?
205. Babesiosis of cattle occurs in our Republic, if not, then why, explain?
206. How do we treat piroplasmidosis in cattle?
207. The main pathoanatomical changes in pyroplasmosis?
208. How do we fight against pyroplasmidosis of cattle?
209. Tell us the essence of the mitigating chemoprophylaxis of pyro-plasmidosis of animals?
210. Give a characteristic of bovine teileriosis?
211. How is the causative agent of bovine teileriosis located according to systematics?
212. Which pathogens of teileriosis are very pathogenic for animals?
213. Tell us the morphology of Theileriaannulata and its biological development in the body of animals and vector ticks?
214. Tell us the morphology of Theileriasergenti and its biological development in the body of animals and vector ticks?
215. Tell us laboratory methods of lifetime diagnosis of teileriosis?
216. The main pathoanatomical changes in teileriosis?
217. Tell the atypical form or course of teileriosis and how it is diagnosed diagnosis with this course of the disease?
218. How do we treat bovine taileriosis?
219. Tell us a 3-day course of treatment for taileriosis?
220. How do we fight with bovine taileriosis?
221. Tell us the essence of vaccination of calves against teileriosis?
222. Tell us the taxonomy of the causative agents of eimeriosis?

223. How do representatives of the subfamily Eimeriinae develop and how do they differ from representatives of the subfamily Isosporinae?
224. Where do eimeria parasitize and tell us the biological development of eimeria in the external environment and in the animal's body?
225. How many cycles differ in the life cycle of eimeria?
226. Tell us the characteristics of rabbit eimeriosis?
227. How are the pathogens of rabbit eimeriosis located according to taxonomy?
228. Methods of lifetime diagnosis of rabbit eimeriosis and from which diseases should it be differentiated?
229. How do we treat sick rabbits with eimeriosis, the drugs used, the dose and the schemes of its use?
230. How do we prevent rabbit eimeriosis?
231. Eimeriosis appeared in the farm, how should we fight it, what are the health measures?
232. Tell us the characteristics of the su-aura?
233. How is the pathogen of the su-aura located according to systematics, tell us the morphology and biology of the pathogen?
234. How is the diagnosis made during the life of the animal on the su-aura?
235. Postmortem diagnosis of the su-aura?
236. From which diseases should the su-aura be differentiated?
237. How to treat animals with trypanosomiasis, are specific and symptomatic methods of treatment used?
238. How to improve the economy if the disease has appeared?
239. How to prevent the farm from trypanosomiasis?
240. Tell us the characteristics of the accidental disease of horses?
241. How is the causative agent of accidental equine disease located according to taxonomy, tell us the morphology and biology of the pathogen?
242. How is the diagnosis made during the life of an animal of accidental horse disease?
243. Accidental horse disease should be differentiated from which diseases?
244. How to treat patients with accidental animal disease, specific and symptomatic treatment methods used?
245. How to improve the economy if the disease has appeared?
246. How to prevent the farm from accidental illness?
247. Tell us the characteristics of trichomoniasis of cattle
248. How is the causative agent of trichomoniasis located according to systematics, tell us the morphology and biology of the pathogen?
249. Laboratory methods for diagnosing trichomoniasis?
250. From which diseases should trichomoniasis of cattle be differentiated?
251. How do we treat cows and bulls with trichomoniasis, specific and symptomatic methods and treatment regimens used by them?
252. How to improve the economy if the diseases have already appeared?
253. How to prevent the economy from trichomoniasis?

254. What is the veterinary significance of parasitoform and acariform mites, Give examples?
255. Life expectancy of acariform mites in different species?
256. Tell us the taxonomy and anatomical and morphological structures of mites of the Psoroptidae family
257. Measures to combat psoroptosis of animals?
258. Tell us the characteristic clinical signs of psoroptosis?
259. How to diagnose sarcoptosis and chorioptosis?
260. What is the difference between psoroptosis and sarcoptosis and chorioptosis of animals?
261. How to diagnose demodecosis of dogs and cattle?
262. How do we treat demodicosis of dogs?
263. Preventive measures to combat demodicosis of dogs?
264. How to diagnose knemidocoptosis of birds?
265. Tell us about the health measures carried out on the farm, with the appearance of knemidocoptosis of birds?
266. What is the difference between knemidocoptosis from syringophilosis and cytoditosis of chickens?
267. What is the characteristic of the insect class?
268. How is the reproduction and development of insects?
269. Bloodsucking diptera insects (midges) and their main distinguishing features?
270. What are the main directions of combating midges and protecting animals from it?
271. Features of the biology of bloodsuckers and pathogens of mellophagosis, siphunculatosis and vermipsilesis of animals.
272. Biological justification of the fight against fleas and bedbugs.
273. Give a characteristic of hypodermatosis of cattle?
274. How is the causative agent of hypodermatosis of cattle located according to the systematics?
275. Morphological structures of Hypodermabovis and its differences from Hypodermalineatum?
276. Biological development of pathogens Hypodermabovis and Hypodermalineatum?
277. Tell us the methods of lifetime diagnosis of hypodermatosis of cattle?
278. Tell us the methods of immunological diagnosis?
279. How do we treat animals with hypodermatosis, the insecticides used, their doses and methods of application?
280. How do we prevent cattle from hypodermatosis?
281. Give a description of sheep estrosis?
282. How is the causative agent of estrosis located according to the taxonomy?
283. Morphological structure of Oestrusovis?
284. Biological development of the pathogen Oestrusovis?
285. Tell us the methods of lifetime diagnosis of sheep estrosis?

286. What is the difference between sheep estrosis and cenurosis?
287. How do we treat animals with estrosis, the insecticides used, their doses and methods of application?
288. How do we prevent sheep from estrosis?
289. Give a description of horse rhinestrosis?
290. Give a description of horse gastrophylosis?
291. How is the systematics of the causative agents of horse rhinestrosis located?
292. How is the systematics of the pathogens of gastrophylosis of horses located?
293. Morphological structure of Rhinoestrus purpureus?
294. Morphological structure of Gastrophilus intestinalis?
295. Biological development of the pathogen Rhinoestrus purpureus?
296. Biological development of the pathogen Gastrophilus intestinalis?
297. Tell us the methods of lifetime diagnosis of rhinestrosis and gastrophylosis of horses?
298. How do we treat patients with estrosis and gastrophylosis of animals, the insecticides used, their doses and methods of application?
299. How do we prevent horses from rhinestrosis?
300. How do we prevent horses from gastrophylosis?

1.41 INTERMEDIATE CONTROL

Written questions

1. Subject "Parasitology", characteristics,
2. Historical development of the subject "Parasitology"
3. Vvklady Uzbek scientist in the development of "Parasitology"
4. Types of relationships in nature. Symbiosis and its forms.
5. Mutualism
6. Commensalism
7. An indifferent attitude
8. Parasitism
9. Innate
10. Endoparasites, give examples
11. Ectoparasites, give examples
12. Permanent parasites
13. Temporary parasites
14. Parasite hosts
15. A single-host parasite
16. Two-host parasite
17. The three-host parasite
18. Obligate
19. The main host
20. The intermediate host
21. Additional host
22. Tank host
23. Optional host
24. Nomenclature of invasive diseases
25. Economic damage caused by invasive diseases,
26. Vector-borne diseases and their natural foci.
27. Morphology of trematodes
28. Biological features of trematodes
29. K.I. Scriabin's doctrine of devastation
30. Pathogens of fascioliasis, their biological development, the source of invasion, ways of infecting definitive hosts.
31. Epizootic process in fascioliasis, development and course of the disease.
32. Pathogenesis, pathomorphology,
33. Immunity in fascioliasis
34. Diagnosis, differential diagnosis of fascioliasis
35. Treatment and measures to combat fascioliasis.
36. Pathogens of dicroceliosis, their biological development, the source of invasion, ways of infecting definitive hosts.
37. Epizootic process in dicroceliosis, development and course of the disease.
38. Pathogenesis, pathomorphological changes in dicroceliosis,
39. Immunity in dicroceliosis
40. Diagnosis, differential diagnosis of dicroceliosis

41. Treatment and measures to combat dicroceliosis
42. Dicroceliosis, characteristics, distribution and economic damage caused by dicroceliosis
43. Pathogens of paramphistomatosis, their biological development, the source of invasion, ways of infecting definitive hosts.
44. Epizootic process in paramphistomatosis, development and course of the disease.
45. Pathogenesis, pathomorphological changes in paramphistomatosis,
46. Diagnosis, differential diagnosis of paramphistomatosis
47. Treatment and measures to combat paramphistomatosis
48. Paramphistomatosis, characteristics, distribution and economic damage caused by paramphistomatosis
49. Orientobilcharciosis, characteristics, distribution and economic damage caused by orientobilcharciosis
50. The causative agent of orientobilcharciosis, their biological development, the source of invasion, ways of infecting definitive hosts.
51. Epizootic process in orientobilcharciosis, development and course of the disease.
52. Pathogenesis, pathomorphological changes in orientobilcharciosis,
53. Diagnosis, differential diagnosis of orientobilcharciosis
54. Treatment and measures to combat orientobilcharciosis
55. The causative agent of orientobilcharciosis, their biological development, the source of invasion, ways of infecting definitive hosts.
56. Epizootic process in eurythrematosis, development and course of the disease.
57. Pathogenesis, pathomorphological changes in eurythrematosis,
58. Diagnosis, differential diagnosis of eurythrematosis
59. Treatment and measures to combat eurythrematosis
60. Hastilesiosis, spread, economic damage, causative agent of eurythrematosis
61. Systematics, morphology of cestodes
62. Biological development of cestode
63. Larval form of cestodes
64. Economic and social damages in cestodose diseases
65. Cysticercosis of cattle
66. Cysticercosis of pigs.
67. Echinococcosis
68. Cenurosis
69. Anoplocephalatoses of ruminants (moniesiosis, tizaniesiosis, avitellenosis, stillesiosis)

70. Stillesiosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
71. Anoplocephalidosis of ungulates (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
72. Paranoplocephalosis of ungulates (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
73. Rayetinosis of birds (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
74. Teniidosis of dogs (disease symptoms, diagnosis, treatment and control measures)
75. Anthelmintics used in cestodoses
76. Morphology and biology of nematodes
77. Geo and bionematodes
78. Epizootology, pathogenesis and immunity of pr and nematode diseases
79. Pig ascariasis, characteristics, distribution and economic damage caused by the disease
80. Pig ascariasis, pathogen and its biological development
81. Ascariasis of pigs, epizootological data, pathogenesis, immunity.
82. Clinical signs and pathoanatomic changes in pig ascariasis.
83. Lifetime diagnosis of pig ascariasis
84. Treatment, prevention and control measures for pig ascariasis
85. Parascaridosis of horses (characteristics, distribution and economic damage).
86. Horse parascaridosis (pathogen and its biological development)
87. Parascaridosis of horses (epizootological data, pathogenesis, immunity)
88. Equine parascaridosis (clinical signs and pathoanatomic changes)
89. Equine parascaridosis (lifetime diagnosis and differential diagnosis)
90. Treatment, prevention and control measures for equine parascaridosis
91. Canine ascariasis
92. Toxocarosis (pathogen and its biological development)
93. Toxocarosis (clinical picture, pathanatomic changes)
94. Toxocarosis, diagnosis and differential diagnosis
95. Toxocarosis, treatment, prevention and control measures
96. Toxascariasis pathogen and its biological development)
97. Toxascariasis (clinical picture, pathanatomic changes)
101. Toxascariasis, diagnosis and differential diagnosis
102. Toxocarosis, treatment, prevention and measures to combat it
103. Oxyurosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
104. Intestinal strongylatoses of ungulates

105. Delafondiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
106. Alfortiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures) (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
107. Strongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
108. Trichonematosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
109. Strongylatoses of digestive organs of ruminants
110. Hemonchosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
111. Marshallagiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
112. Habertiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
113. Nematodirosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
114. Dictyoculosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
115. Pig metastrongylosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
116. Protostrongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
117. Mulleriosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
118. Cystocaulosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
119. Telyaziosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
120. Trichinosis (pathogen, biological development, symptoms of the disease, diagnosis and control measures)
121. Macrocontorhinchosis of pigs (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
122. Polymorphosis of ducks (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
123. Phyllicolysis (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
124. Parascaridosis of horses (characteristics, distribution and economic damage).

125. Parascaridosis of horses (pathogen and its biological development)
126. Parascaridosis of horses (epizootological data, pathogenesis, immunity)
127. Equine parascaridosis (clinical signs and pathological changes)
128. Parascaridosis of horses (lifetime diagnosis and differential diagnosis)
129. Treatment, prevention and control measures for equine parascaridosis
130. Ascariasis of dogs
131. Toxocarosis (pathogen and its biological development)
132. Toxocarosis (clinical picture, pathanatomic changes)
133. Toxocarosis, diagnosis and differential diagnosis
134. Toxocarosis, treatment, prevention and measures to combat it
135. Toxascariasis pathogen and its biological development)
136. Toxascariasis (clinical picture, pathanatomic changes)
137. Oxyurosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
138. Intestinal strongylatoses of ungulates
139. Delafondiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
140. Alfortiosis (causative agent, disease symptoms, diagnosis, treatment and control measures) (causative agent, disease symptoms, diagnosis, treatment and control measures)
141. Strongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
142. Trichonematosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
143. Strongylatoses of digestive organs of ruminants
144. Hemonchosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
145. Marshallagiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
146. Habertiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
147. Nematodirosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
148. Dictyoculosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
149. Pig metastrongylosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
150. Protostrongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)

1.5 2-INTERMEDIATE CONTROL

Written questions

1. Veterinary protozoology, history of development and contributions of Uzbek scientists in the development of science
2. Protozoa and diseases caused by them
3. Systematics of protozoa
4. Pyroplasmosis (characterization, spread and economic damage)
5. Pyroplasmosis (pathogen and its carriers)
6. Pyroplasmosis, epizootological data, pathogenesis and immunity.
7. Pyroplasmosis, symptoms of the disease, pathoanatomic changes
8. Pyroplasmosis, diagnosis and differential diagnosis
9. Pyroplasmosis, treatment, prevention and control measures
10. Francaiellosis (characteristics, spread and economic damage)
11. Francaiellosis (pathogen and its carriers)
12. Francaiellosis, epizootological data, pathogenesis and immunity.
13. Francaiellosis, symptoms of the disease, pathoanatomic changes
14. Francaiellosis, diagnosis and differential diagnosis
15. Frenmaiellosis, treatment, prevention and measures to combat it
16. Babesiosis (pathogen and its carriers)
17. Babesiosis, epizootological data, pathogenesis and immunity.
18. Babesiosis, symptoms of the disease, pathoanatomic changes
19. Babesiosis, diagnosis and differential diagnosis
20. Babesiosis, treatment, prevention and measures to combat it
21. Teileriosis (pathogen and its carriers)
22. Teileriosis, epizootological data, pathogenesis and immunity.
23. Teileriosis, symptoms of the disease, pathoanatomic changes
24. Teileriosis, diagnosis and differential diagnosis
25. Teileriosis, treatment, prevention and measures to combat it
26. Pyroplasmosis of horses (pathogen and its carriers)
27. Pyroplasmosis of horses, symptoms of the disease, pathoanatomic changes
28. Pyroplasmosis of horses, diagnosis and differential diagnosis
29. Pyroplasmosis of horses, treatment, prevention and measures to combat it
30.. Nuttaliosis of horses (pathogen and its carriers)
31. Nuttaliosis, diagnosis and differential diagnosis
32. Nuttaliosis, treatment, prevention and measures to combat it
33. Nuttaliosis, diagnosis and differential diagnosis
34. Nuttaliosis, treatment, prevention and control measures
35. Pyroplasmosis of sheep, goats and (pathogen and its carriers)

36. Pyroplasmosis of sheep and goats (epizootological data, pathogenesis and immunity).
37. Pyroplasmosis of sheep and goats, symptoms of the disease, pathoanatomic changes
38. Piroplasmosis of sheep and goats, diagnosis and differential diagnosis
39. Piroplasmosis of sheep and goats, treatment, prevention and control measures
40. Pig piroplasmosis (pathogen and its carriers)
41. Pig piroplasmosis (disease symptoms, pathoanatomic changes)
42. Pig pyroplasmosis (diagnosis and differential diagnosis)
43. Pig pyroplasmosis (treatment, prevention and control measures)
44. Pyroplasmosis of dogs (pathogen and its carriers)
45. Pyroplasmosis of dogs (symptoms of the disease, pathoanatomic changes)
46. Pyroplasmosis of dogs (diagnosis and differential diagnosis)
47. Pyroplasmosis of dogs (treatment, prevention and measures to combat it)
48. General characteristics of coccidia
49. Eimeriosis of cattle,
50. Eimeriosis of sheep and goats (pathogen, symptoms, treatment)
51. Eimeriosis of rabbits (pathogen, pathogenesis, diagnosis, treatment)
52. Toxoplasmosis (pathogen, biology, symptoms of the disease, diagnosis, treatment and methods of control)
53. Sarcocystosis (causative agent, symptoms, diagnosis, treatment and prevention)
54. Cystoisosporidiosis (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
55. Su-aura of camels (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
56. Accidental horse disease (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
57. Cattle trichomoniasis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
58. Leishmaniasis of dogs (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
59. Benzoithiosis (causative agent, symptoms, diagnosis, treatment and prevention)
60. Anaplasmosis of cattle(causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
61. Anaplasmosis of sheep (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
62. Borreliosis (spirochetosis) of birds
63. Balantidiosis of pigs (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)

64. Morphology, biological features of acariform mites
65. Psoroptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
66. Sarcoptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
67. Chorioptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
68. Otodectosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
69. Demodecosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
70. knemidocoptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
71. Gamazoid and argass mites
72. Tick-borne sheep paralysis (causative agent, disease symptoms, diagnosis, treatment and control measures)
73. Hypodermatosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
74. Estrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
75. Rhinestrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
76. Gastrophyllosis (pathogen, symptoms of the disease, diagnosis, treatment and control measures)
77. Crivelliosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
78. Cephalopinosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
79. Polymorphosis of ducks (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
80. Phyllicolysis (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
81. Veterinary protozoology, history of development and contributions of Uzbek scientists in the development of science
82. Protozoa and diseases caused by them
83. Systematics of protozoa
84. Pyroplasmosis (characteristics, distribution and economic damage)
85. Pyroplasmosis (pathogen and its carriers)
86. Pyroplasmosis, epizootological data, pathogenesis and immunity.

87. Pyroplasmosis, symptoms of the disease, pathoanatomic changes
88. Pyroplasmosis, diagnosis and differential diagnosis
89. Pyroplasmosis, treatment, prevention and measures to combat it
90. Francaiellosis (characteristics, distribution and economic damage)
91. Francaiellosis (pathogen and its carriers)
92. Francaiellosis, epizootological data, pathogenesis and immunity.
93. Francaiellosis, symptoms of the disease, pathoanatomic changes
94. Francaiellosis, diagnosis and differential diagnosis
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96. Babesiosis (pathogen and its carriers)
97. Babesiosis, epizootological data, pathogenesis and immunity.
98. Babesiosis, symptoms of the disease, pathoanatomic changes
99. Babesiosis, diagnosis and differential diagnosis
100. Babesiosis, treatment, prevention and measures to combat it
101. Teileriosis (pathogen and its carriers)
102. Teileriosis, epizootological data, pathogenesis and immunity
103. Teileriosis, symptoms of the disease, pathoanatomic changes
104. Teileriosis, diagnosis and differential diagnosis
105. Teileriosis, treatment, prevention and measures to combat it
106. Pyroplasmosis of horses (pathogen and its carriers)
107. Pyroplasmosis of horses, symptoms of the disease, pathoanatomic changes
108. Pyroplasmosis of horses, diagnosis and differential diagnosis
109. Pyroplasmosis of horses, treatment, prevention and measures to combat it
110. Nuttaliosis of horses (pathogen and its carriers)
111. Nuttaliosis, diagnosis and differential diagnosis
112. Nuttaliosis, treatment, prevention and measures to combat it
113. Nuttaliosis, diagnosis and differential diagnosis
114. Nuttaliosis, treatment, prevention and measures to combat it
115. Pyroplasmosis of sheep, goats and (pathogen and its carriers)
116. Piroplasmosis of sheep and goats (epizootological data, pathogenesis and immunity).
117. Piroplasmosis of sheep and goats, symptoms of the disease, pathoanatomic changes
118. Pyroplasmosis of sheep and goats, diagnosis and differential diagnosis
119. Pyroplasmosis of sheep and goats, treatment, prevention and measures to combat it
120. Pig pyroplasmosis (pathogen and its carriers)
121. Pig pyroplasmosis (disease symptoms, pathoanatomic changes)
122. Pig pyroplasmosis (diagnosis and differential diagnosis)
123. Pig pyroplasmosis (treatment, prevention and control measures)
124. Pyroplasmosis of dogs (pathogen and its carriers)
125. Pyroplasmosis of dogs (symptoms of the disease, pathoanatomic changes)

126. Pyroplasmosis of dogs (diagnosis and differential diagnosis)
127. Pyroplasmosis of dogs (treatment, prevention and measures to combat it)
128. General characteristics of coccidia
129. Eimeriosis of cattle,
130. Eimeriosis of sheep and goats (causative agent, symptoms, treatment)
131. Rabbit eimeriosis (pathogen, pathogenesis, diagnosis, treatment)
132. Toxoplasmosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
133. Sarcocystosis (causative agent, symptoms, diagnosis, treatment and prevention)
134. Cystoisosporidiosis (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
135. Su-aura of camels (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
136. Accidental horse disease (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
137. Cattle trichomoniasis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
138. Leishmaniasis of dogs (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
139. Benzoithiosis (causative agent, symptoms, diagnosis, treatment and prevention)
140. Anaplasmosis of cattle (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
141. Sheep anaplasmosis (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
142. Borreliosis (spirochetosis) of birds
143. Porcine balantidiosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
144. Morphology, biological features of acariform mites
145. Psoroptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
146. Sarcoptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
147. Chorioptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
148. Otodectosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
149. Demodicosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
150. Knemidocoptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)

1.6 FINAL CONTROL

1. Subject "Parasitology", characteristics,
2. Historical development of the subject "Parasitology"
3. Vvklady Uzbek scientist in the development of "Parasitology"
4. Types of relationships in nature. Symbiosis and its forms.
5. Mutualism
6. Commensalism
7. An indifferent attitude
8. Parasitism
9. Innate
10. Endoparasites, give examples
11. Ectoparasites, give examples
12. Permanent parasites
13. Temporary parasites
14. Parasite hosts
15. A single-host parasite
16. Two-host parasite
17. The three-host parasite
18. Obligate
19. The main host
20. The intermediate host
21. Additional host
22. Reservoir host
23. Optional host
24. Nomenclature of invasive diseases
25. Economic damage caused by invasive diseases,
26. Vector-borne diseases and their natural foci.
27. Morphology of trematodes
28. Biological features of trematodes
29. K.I. Scriabin's doctrine of devastation
30. Pathogens of fascioliasis, their biological development, the source of invasion, ways of infecting definitive hosts.
31. Epizootic process in fascioliasis, development and course of the disease.
32. Pathogenesis, pathomorphology,
33. Immunity in fascioliasis
34. Diagnosis, differential diagnosis of fascioliasis
35. Treatment and measures to combat fascioliasis.
36. Pathogens of dicroceliosis, their biological development, the source of invasion, ways of infecting definitive hosts.
37. Epizootic process in dicroceliosis, development and course of the disease.
38. Pathogenesis, pathomorphological changes in dicroceliosis,
39. Immunity in dicroceliosis
40. Diagnosis, differential diagnosis of dicroceliosis
41. Treatment and measures to combat dicroceliosis

42. Dicroceliosis, characteristics, distribution and economic damage caused by dicroceliosis
43. Pathogens of paramphistomatosis, their biological development, the source of invasion, ways of infecting definitive hosts.
44. Epizootic process in paramphistomatosis, development and course of the disease.
45. Pathogenesis, pathomorphological changes in paramphistomatosis,
46. Diagnosis, differential diagnosis of paramphistomatosis
47. Treatment and measures to combat paramphistomatosis
48. Paramphistomatosis, characteristics, distribution and economic damage caused by paramphistomatosis
49. Orientobilkhartsioz, characteristics, distribution and economic damage caused by orientobilkhartsioz
50. The causative agent of orientobilcharciosis, their biological development, the source of invasion, ways of infecting definitive hosts.
51. Epizootic process in orientobilcharciosis, development and course of the disease.
52. Pathogenesis, pathomorphological changes in orientobilcharciosis,
53. Diagnosis, differential diagnosis of orientobilcharciosis
54. Treatment and measures to combat orientobilcharciosis
55. The causative agent of orientobilcharciosis, their biological development, the source of invasion, ways of infecting definitive hosts.
56. Epizootic process in eurythrematosis, development and course of the disease.
57. Pathogenesis, pathomorphological changes in eurythrematosis,
58. Diagnosis, differential diagnosis of eurythrematosis
59. Treatment and measures to combat eurythrematosis
60. Hastilesiosis, spread, economic damage, causative agent of eurythrematosis
61. Systematics, morphology of cestodes
62. Biological development of cestode
63. Larval form of cestodes
64. Economic and social damages in cestodose diseases
65. Cysticercosis of cattle
66. Cysticercosis of pigs.
67. Echinococcosis
68. Cenurosis
69. Anoplocephalatoses of ruminants (moniesiosis, tizaniesiosis, avitellenosis, stillesiosis)
70. Moniesiozniesiosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)

71. Tizaniesiosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
72. Avitellenosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
73. Stillesiosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
74. Anoplocephalidosis of ungulates (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
75. Paranoplocephalosis of ungulates (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
76. Raietinosis of birds (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
77. Teniidosis of dogs (symptoms of the disease, diagnosis, treatment and control measures)
78. Anthelmintics used in cestodoses
79. Morphology and biology of nematodes
80. Geo and bionematodes
81. Epizootology, pathogenesis and immunity of pr and nematode diseases
82. Pig ascariasis, characteristics, distribution and economic damage caused by the disease
83. Pig ascariasis, the causative agent and its biological development
84. Ascariasis of pigs, epizootological data, pathogenesis, immunity.
85. Clinical signs and pathoanatomic changes in pig ascariasis.
86. Lifetime diagnosis of pig ascariasis
87. Treatment, prevention and control measures for pig ascariasis
88. Paraskaridosis of horses (characteristics, distribution and economic damage).
89. Paraskaridosis of horses (pathogen and its biological development)
90. Paraskaridosis of horses (epizootological data, pathogenesis, immunity)
91. Equine parascaridosis (clinical signs and pathological changes)
92. Equine parascaridosis (lifetime diagnosis and differential diagnosis)
93. Treatment, prevention and control measures for equine parascaridosis
94. Ascariasis of dogs
95. Toxocarosis (pathogen and its biological development)
96. Toxocarosis (clinical picture, pathanatomic changes)
97. Toxocarosis, diagnosis and differential diagnosis
98. Toxocarosis, treatment, prevention and measures to combat it
99. Toxascariasis pathogen and its biological development)
100. Toxascariasis (clinical picture, pathanatomic changes)
101. Toxascariasis, diagnosis and differential diagnosis

102. Toxocarosis, treatment, prevention and measures to combat it
103. Oxyurosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
104. Intestinal strongylatoses of ungulates
105. Delafondiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
106. Alfortiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures) (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
107. Strongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
108. Trichonematosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
109. Strongylatoses of digestive organs of ruminants
110. Hemonchosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
111. Marshallagiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
112. Habertiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
113. Nematodirosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
114. Dictyoculosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
115. Pig metastrongylosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
116. Protostrongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
117. Mulleriosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
118. Cystocaulosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
119. Telyaziosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
120. Trichinosis (pathogen, biological development, symptoms of the disease, diagnosis and control measures)
121. Macrocontorhinchosis of pigs (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)

122. Polymorphosis of ducks (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
123. Phyllicolysis (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
124. Veterinary protozoology, history of development and contributions of Uzbek scientists in the development of science
125. Protozoa and diseases caused by them
126. Systematics of protozoa
127. Pyroplasmosis (characteristics, distribution and economic damage)
128. Pyroplasmosis (pathogen and its carriers)
129. Pyroplasmosis, epizootological data, pathogenesis and immunity.
130. Pyroplasmosis, symptoms of the disease, pathoanatomic changes
131. Pyroplasmosis, diagnosis and differential diagnosis
132. Pyroplasmosis, treatment, prevention and measures to combat it
133. Francaiellosis (characteristics, distribution and economic damage)
134. Francaiellosis (pathogen and its carriers)
135. Francaiellosis, epizootological data, pathogenesis and immunity.
136. Francaiellosis, symptoms of the disease, pathoanatomic changes
137. Francaiellosis, diagnosis and differential diagnosis
138. Francaiellosis, treatment, prevention and measures to combat it
139. Babesiosis (pathogen and its carriers)
140. Babesiosis, epizootological data, pathogenesis and immunity.
141. Babesiosis, symptoms of the disease, pathoanatomic changes
142. Babesiosis, diagnosis and differential diagnosis
143. Babesiosis, treatment, prevention and measures to combat it
144. Teileriosis (pathogen and its carriers)
145. Teileriosis, epizootological data, pathogenesis and immunity.
146. Teileriosis, symptoms of the disease, pathoanatomic changes
147. Teileriosis, diagnosis and differential diagnosis
148. Teileriosis, treatment, prevention and measures to combat it
149. Pyroplasmosis of horses (pathogen and its carriers)
150. Pyroplasmosis of horses, symptoms of the disease, pathoanatomic changes
151. Pyroplasmosis of horses, diagnosis and differential diagnosis
152. Pyroplasmosis of horses, treatment, prevention and measures to combat it
153. Equine nuttaliosis (pathogen and its carriers)
154. Nuttaliosis, diagnosis and differential diagnosis
155. Nuttaliosis, treatment, prevention and measures to combat it
156. Nuttaliosis, diagnosis and differential diagnosis
157. Nuttaliosis, treatment, prevention and control measures

158. Pyroplasmosis of sheep, goats and (pathogen and its carriers)
159. Pyroplasmosis of sheep and goats (epizootological data, pathogenesis and immunity).
160. Pyroplasmosis of sheep and goats, symptoms of the disease, pathoanatomic changes
161. Pyroplasmosis of sheep and goats, diagnosis and differential diagnosis
162. Pyroplasmosis of sheep and goats, treatment, prevention and measures to combat it
163. Pig pyroplasmosis (pathogen and its carriers)
164. Pig pyroplasmosis (disease symptoms, pathoanatomic changes)
165. Pig pyroplasmosis (diagnosis and differential diagnosis)
166. Pig pyroplasmosis (treatment, prevention and control measures)
167. Pyroplasmosis of dogs (pathogen and its carriers)
168. Pyroplasmosis of dogs (symptoms of the disease, pathoanatomic changes)
169. Pyroplasmosis of dogs (diagnosis and differential diagnosis)
170. Pyroplasmosis of dogs (treatment, prevention and measures to combat it)
171. General characteristics of coccidia
172. Eimeriosis of cattle,
173. Eimeriosis of sheep and goats (causative agent, symptoms, treatment)
174. Rabbit emeriosis (pathogen, pathogenesis, diagnosis, treatment)
175. Toxoplasmosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
176. Sarcocystosis (causative agent, symptoms, diagnosis, treatment and prevention)
177. Cystoisosporidiosis (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
178. Su-aura of camels (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
179. Accidental horse disease (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
180. Cattle trichomoniasis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
181. Leishmaniasis of dogs (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
182. Benzoithiosis (causative agent, symptoms, diagnosis, treatment and prevention)
183. Anaplasmosis of cattle (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)

184. Sheep anaplasmosis (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
185. Borreliosis (spirochetosis) of birds
186. Porcine balantidiosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
187. Morphology, biological features of acariform mites
188. Psoroptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
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192. Demodicosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
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196. Hypodermatosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
197. Estrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
198. Rhinestrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
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201. Cephalopinosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
202. Subject "Parasitology", characteristics,
203. Historical development of the subject "Parasitology"
204. The Uzbek scientist's views in the development of "Parasitology"
205. Types of relationships in nature. Symbiosis and its forms.
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207. Commensalism

208. An indifferent attitude
209. Parasitism
210. Innate
211. Endoparasites, give examples
212. Ectoparasites, give examples
213. Permanent parasites
214. Temporary parasites
215. Parasite hosts
216. A single-host parasite
217. Two-host parasite
218. The three-host parasite
219. Obligate
220. The main host
221. The intermediate host
222. Additional host
223. Tank host
224. Optional host
225. Nomenclature of invasive diseases
226. Economic damage caused by invasive diseases,
227. Vector-borne diseases and their natural foci.
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229. Biological features of trematodes
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232. Epizootic process in fascioliasis, development and course of the disease.
233. Pathogenesis, pathomorphology,
234. Immunity in fascioliasis
235. Diagnosis, differential diagnosis of fascioliasis
236. Treatment and measures to combat fascioliasis.
237. Pathogens of dicroceliosis, their biological development, the source of invasion, ways of infecting definitive hosts.
238. Epizootic process in dicroceliosis, development and course of the disease.
239. Pathogenesis, pathomorphological changes in dicroceliosis,
240. Immunity in dicroceliosis
241. Diagnosis, differential diagnosis of dicroceliosis
242. Treatment and measures to combat dicroceliosis
243. Dicroceliosis, characteristics, distribution and economic damage caused by dicroceliosis
244. Pathogens of paramphistomatosis, their biological development, the source of invasion, ways of infection of definitive hosts.
245. Epizootic process in paramphistomatosis, development and course of the disease.
246. Pathogenesis, pathomorphological changes in paramphistomatosis

247. Diagnosis, differential diagnosis of paramphistomatosis
248. Treatment and measures to combat paramphistomatosis
249. Paramphistomatosis, characteristics, distribution and economic damage caused by paramphistomatosis
250. Orientobilcharciosis, characteristics, distribution and economic damage caused by orientobilcharciosis
251. Causative agent of orientobilcharciosis, their biological development, source of invasion, ways of infection of definitive hosts.
252. Epizootic process in orientobilcharciosis, development and course of the disease.
253. Pathogenesis, pathomorphological changes in orientobilcharciosis,
254. Diagnosis, differential diagnosis of orientobilcharciosis
255. Treatment and measures to combat orientobilcharciosis
256. The causative agent of orientobilcharciosis, their biological development, the source of invasion, the ways of infection of definitive hosts.
257. Epizootic process in eurythrematosis, the development and course of the disease.
258. Pathogenesis, pathomorphological changes in eurythrematosis,
259. Diagnosis, differential diagnosis of eurythrematosis
260. Treatment and measures to combat eurythrematosis
261. Hastilesiosis, spread, economic damage, causative agent of eurythrematosis
262. Systematics, morphology of cestodes
263. Biological development of cestode
264. Larval form of cestodes
265. Economic and social damages in cestodose diseases
266. Cysticercosis of cattle
267. Cysticercosis of pigs.
268. Echinococcosis
269. Cenurosis
270. Anoplocephalatoses of ruminants (moniesiosis, tizaniesiosis, avitellenosis, stillesiosis)
271. Stillesiosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
272. Anoplocephalidosis of ungulates (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
273. Paranoplocephalosis of ungulates (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
274. Raietinosis of birds (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)

275. Teniidosis of dogs (symptoms of the disease, diagnosis, treatment and control measures)
276. Anthelmintics used in cestodoses
277. Morphology and biology of nematodes
278. Geo and bionematodes
279. Epizootology, pathogenesis and immunity of pr and nematode diseases
280. Pig ascariasis, characteristics, distribution and economic damage caused by the disease
281. Pig ascariasis, pathogen and its biological development
282. Pig ascariasis, epizootological data, pathogenesis, immunity.
283. Clinical signs and pathoanatomic changes in pig ascariasis.
284. Lifetime diagnosis of pig ascariasis
285. Treatment, prevention and control measures for pig ascariasis
286. Parascaridosis of horses (characteristics, distribution and economic damage).
287. Horse parascaridosis (pathogen and its biological development)
288. Parascaridosis of horses (epizootological data, pathogenesis, immunity)
289. Equine parascaridosis (clinical signs and pathological changes)
290. Parascaridosis of horses (lifetime diagnosis and differential diagnosis)
291. Treatment, prevention and control measures for equine parascaridosis
292. Ascariasis of dogs
293. Toxocarosis (pathogen and its biological development)
294. Toxocarosis (clinical picture, pathanatomic changes)
295. Toxocarosis, diagnosis and differential diagnosis
296. Toxocarosis, treatment, prevention and measures to combat it
297. Toxascariasis pathogen and its biological development)
298. Toxascariasis (clinical picture, pathanatomic changes)
299. Oxyurosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
300. Intestinal strongylatoses of ungulates
301. Delafondiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
302. Alfortiosis (causative agent, disease symptoms, diagnosis, treatment and control measures) (causative agent, disease symptoms, diagnosis, treatment and control measures)
303. Strongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
304. Trichonematosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
305. Strongylatosis of the digestive organs of ruminants

306. Hemonchosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
307. Marshallagiosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
308. Habertiosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
309. Nematodirosis(pathogen, disease symptoms, diagnosis, treatment
310. Dictyoculosis (pathogen, symptoms of the disease, diagnosis, treatment and control measures)
311. Pig metastrongylosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
312. Protostrongylosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
313. Mulleriosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
314. Cystocaulosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
315. Telyaziosis (causative agent, biological development, symptoms of the disease, diagnosis, treatment and control measures)
316. Trichinosis (pathogen, biological development, symptoms of the disease, diagnosis and control measures)
317. Macrocontorhinchosis of pigs (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
318. Polymorphosis of ducks (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
319. Phyllicolesis (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
320. Parascaridosis of horses (characteristics, distribution and economic damage).
321. Parascaridosis of horses (pathogen and its biological development)
322. Equine parascaridosis (epizootological data, pathogenesis, immunity)
323. Equine parascaridosis (clinical signs and pathoanatomic changes)
324. Parascaridosis of horses (lifetime diagnosis and differential diagnosis)
325. Treatment, prevention and control measures for equine parascaridosis
326. Ascariasis of dogs
327. Toxocarosis (pathogen and its biological development)
328. Toxocarosis (clinical picture, pathanatomic changes)
329. Toxocarosis, diagnosis and differential diagnosis
330. Toxocarosis, treatment, prevention and control measures
331. Toxascariasis pathogen and its biological development)

332. Toxascariasis (clinical picture, pathanatomic changes)
333. Oxyurosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
334. Intestinal strongylatoses of ungulates
335. Delafondiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
336. Alfortiosis (pathogen, disease symptoms, diagnosis, treatment and control measures) (pathogen, disease symptoms, diagnosis, treatment and control measures)
337. Strongylosis(pathogen, disease symptoms, diagnosis, treatment and control measures)
338. Trichonematosis(pathogen, disease symptoms, diagnosis, treatment and control measures)
339. Strongylatoses of digestive organs of ruminants
340. Hemonchosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
341. Marshallagiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
342. Habertiosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
343. Nematodirosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
344. Dictyoculosis(causative agent, disease symptoms, diagnosis, treatment and control measures)
345. Pig metastrongylosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
346. Protostrongylosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
347. Veterinary protozoology, history of development and contributions of Uzbek scientists in the development of science
348. Protozoa and diseases caused by them
349. Systematics of protozoa
350. Pyroplasmosis (characteristics, distribution and economic damage)
351. Pyroplasmosis (pathogen and its carriers)
352. Pyroplasmosis, epizootological data, pathogenesis and immunity.
353. Pyroplasmosis, disease symptoms, pathoanatomic changes
354. Pyroplasmosis, diagnosis and differential diagnosis
355. Pyroplasmosis, treatment, prevention and measures to combat it
356. Francaiellosis (characteristics, distribution and economic damage)

357. Francaiellosis(pathogen and its carriers)
358. Francaiellosis, epizootological data, pathogenesis and immunity.
359. Francaiellosis, symptoms of the disease, pathoanatomic changes
360. Francaiellosis, diagnosis and differential diagnosis
361. Frenmaiellosis, treatment, prevention and measures to combat it
362. Babesiosis (pathogen and its carriers)
363. Babesiosis, epizootological data, pathogenesis and immunity.
364. Babesiosis, symptoms of the disease, pathoanatomic changes
365. Babesiosis, diagnosis and differential diagnosis
366. Babesiosis, treatment, prevention and measures to combat it
367. Teileriosis (pathogen and its carriers)
368. Teileriosis, epizootological data, pathogenesis and immunity.
369. Teileriosis, symptoms of the disease, pathoanatomic changes
370. Teileriosis, diagnosis and differential diagnosis
371. Teileriosis, treatment, prevention and measures to combat it
372. Pyroplasmosis of horses (pathogen and its carriers)
373. Pyroplasmosis of horses, symptoms of the disease, pathoanatomic changes
374. Pyroplasmosis of horses, diagnosis and differential diagnosis
375. Pyroplasmosis of horses, treatment, prevention and measures to combat it
376. Nuttaliosis of horses (pathogen and its carriers)
377. Nuttaliosis, diagnosis and differential diagnosis
378. Nuttaliosis, treatment, prevention and measures to combat it
379. Nuttaliosis, diagnosis and differential diagnosis
380. Nuttaliosis, treatment, prevention and measures to combat it
381. Pyroplasmosis of sheep, goats and (pathogen and its carriers)
382. Pyroplasmosis of sheep and goats (epizootological data, pathogenesis and immunity).
383. Pyroplasmosis of sheep and goats, symptoms of the disease, pathoanatomic changes
384. Pyroplasmosis of sheep and goats, diagnosis and differential diagnosis
385. Pyroplasmosis of sheep and goats, treatment, prevention and measures to combat it
386. Pig pyroplasmosis (pathogen and its carriers)
387. Pig pyroplasmosis (disease symptoms, pathoanatomic changes)
388. Pig pyroplasmosis (diagnosis and differential diagnosis)
389. Pig pyroplasmosis (treatment, prevention and control measures)
390. Pyroplasmosis of dogs (pathogen and its carriers)
391. Pyroplasmosis of dogs (symptoms of the disease, pathoanatomic changes)
392. Pyroplasmosis of dogs (diagnosis and differential diagnosis)

393. Pyroplasmosis of dogs (treatment, prevention and measures to combat it)
394. General characteristics of coccidia
395. Eimeriosis of cattle,
396. Eimeriosis of sheep and goats (pathogen, symptoms, treatment)
397. Eimeriosis of rabbits (pathogen, pathogenesis, diagnosis, treatment)
398. Toxoplasmosis (pathogen, biology, symptoms of the disease, diagnosis, treatment and methods of control)
399. Sarcocystosis (pathogen, symptoms, diagnosis, treatment and prevention)
400. Cystoisosporidiosis (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
401. Su-aura of camels (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
402. Accidental horse disease (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
403. Cattle trichomoniasis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
404. Leishmaniasis of dogs (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
405. Benzoithiosis (causative agent, symptoms, diagnosis, treatment and prevention)
406. Anaplasmosis of cattle (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
407. Anaplasmosis of sheep (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
408. Borreliosis (spirochetosis) of birds
409. Balantidiosis of pigs (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
410. Morphology, biological features of acariform mites
411. Psoroptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
412. Sarcoptosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
413. Chorioptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
414. Otodectosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
415. Demodicosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)

416. knemidocoptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
417. Gamazoid and argass mites
418. Tick-borne sheep paralysis (causative agent, disease symptoms, diagnosis, treatment and control measures)
419. Hypodermatosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
420. Estrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
421. Rhinestrosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
422. Gastrophyllosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
423. Crivelliosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
424. Cephalopinosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
425. Polymorphosis of ducks (pathogen, biological development, disease symptoms, diagnosis, treatment and control measures)
426. Phyllicolesis (pathogen, biological development, symptoms of the disease, diagnosis, treatment and control measures)
427. Veterinary protozoology, history of development and contributions of Uzbek scientists in the development of science
428. Protozoa and diseases caused by them
429. Systematics of protozoa
430. Pyroplasmosis (characteristics, distribution and economic damage)
431. Pyroplasmosis (pathogen and its carriers)
432. Pyroplasmosis, epizootological data, pathogenesis and immunity.
433. Pyroplasmosis, symptoms of the disease, pathoanatomic changes
434. Pyroplasmosis, diagnosis and differential diagnosis
435. Pyroplasmosis, treatment, prevention and measures to combat it
436. Francaiellosis (characteristics, distribution and economic damage)
437. Francaiellosis (pathogen and its carriers)
438. Francaiellosis, epizootological data, pathogenesis and immunity.
439. Francaiellosis, symptoms of the disease, pathoanatomic changes
440. Francaiellosis, diagnosis and differential diagnosis
441. Frenmaiellosis, treatment, prevention and measures to combat it
442. Babesiosis (pathogen and its carriers)
443. Babesiosis, epizootological data, pathogenesis and immunity.

444. Babesiosis, symptoms of the disease, pathoanatomic changes
445. Babesiosis, diagnosis and differential diagnosis
446. Babesiosis, treatment, prevention and measures to combat it
447. Teileriosis (pathogen and its carriers)
448. Teileriosis, epizootological data, pathogenesis and immunity.
449. Teileriosis, symptoms of the disease, pathoanatomic changes
450. Teileriosis, diagnosis and differential diagnosis
451. Teileriosis, treatment, prevention and measures to combat it
452. Pyroplasmosis of horses (pathogen and its carriers)
453. Pyroplasmosis of horses, symptoms of the disease, pathoanatomic changes
454. Pyroplasmosis of horses, diagnosis and differential diagnosis
455. Pyroplasmosis of horses, treatment, prevention and measures to combat it
456. Nuttaliosis of horses (pathogen and its carriers)
457. Nuttaliosis, diagnosis and differential diagnosis
458. Nuttaliosis, treatment, prevention and control measures
459. Nuttaliosis, diagnosis and differential diagnosis
460. Nuttaliosis, treatment, prevention and measures to combat it
461. Pyroplasmosis of sheep, goats and (pathogen and its carriers)
462. Pyroplasmosis of sheep and goats (epizootological data, pathogenesis and immunity).
463. Pyroplasmosis of sheep and goats, symptoms of the disease, pathoanatomic changes
464. Pyroplasmosis of sheep and goats, diagnosis and differential diagnosis
465. Pyroplasmosis of sheep and goats, treatment, prevention and measures to combat it
466. Pig pyroplasmosis (pathogen and its carriers)
467. Pig pyroplasmosis (disease symptoms, pathoanatomic changes)
468. Pig pyroplasmosis (diagnosis and differential diagnosis)
469. Pig pyroplasmosis (treatment, prevention and control measures)
470. Pyroplasmosis of dogs (pathogen and its carriers)
471. Pyroplasmosis of dogs (symptoms of the disease, pathoanatomic changes)
472. Pyroplasmosis of dogs (diagnosis and differential diagnosis)
473. Pyroplasmosis of dogs (treatment, prevention and measures to combat it)
474. General characteristics of coccidia
475. Eimeriosis of cattle,
476. Eimeriosis of sheep and goats (causative agent, symptoms, treatment)
477. Rabbit emeriosis (pathogen, pathogenesis, diagnosis, treatment)
478. Toxoplasmosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)

479. Sarcocystosis (causative agent, symptoms, diagnosis, treatment and prevention)
480. Cystoisosporidiosis (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
481. Su-aura of camels (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
482. Accidental horse disease (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
483. Cattle trichomoniasis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
484. Leishmaniasis of dogs (causative agent, biology, symptoms of the disease, diagnosis, treatment and control measures)
485. Benzoithiosis (causative agent, symptoms, diagnosis, treatment and prevention)
486. Anaplasmosis of cattle (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
487. Sheep anaplasmosis (causative agent, biology, disease symptoms, diagnosis, treatment and control measures)
488. Borreliosis (spirochetosis) of birds
489. Porcine balantidiosis (pathogen, biology, disease symptoms, diagnosis, treatment and control measures)
490. Morphology, biological features of acariform mites
491. Psoroptosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
492. Sarcoptosis (causative agent, disease symptoms, diagnosis, treatment and control measures)
493. Chorioptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
494. Otodectosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
495. Demodicosis (causative agent, symptoms of the disease, diagnosis, treatment and control measures)
496. cnemidocoptosis (pathogen, disease symptoms, diagnosis, treatment and control measures)
497. Francaiellosis (characteristics, distribution and economic damage)
498. Francaiellosis(pathogen and its carriers)
499. Francaiellosis, epizootological data, pathogenesis and immunity.
500. Francaiellosis, symptoms of the disease, pathoanatomic changes

1.7 1-INTERMEDIATE CONTROL

TEST QUESTIONS

1. What is the relationship of organisms in which both sides benefit from such cohabitation?

A. Predation
B. Sinoikia
C.Parasitism
D. Mutualism

2. What is the relationship of organisms, in which each of them is completely independent of the others, but at the same time is closely connected with the life of the whole collective as a whole?

A.Independent
B. Symbiotic
C.Mutualistic
D. Commensalistic

3. What is the relationship between organisms, in which one organism benefits, and the other neither benefits nor harms?

A.Sinoikia
B.Mutualism
C.Parasitism
D.Predation

4. What is the name of the relationship between two organisms, in which one of the organisms feeds on the remains of the other organism's food without harming it?

A.Commensalism
B.Mutualism
C.Sinoikia
D.Parasitism}

5. What is the relationship between two organisms, in which one organism is stronger than its prey, which it immediately kills and eats?

A.Predation
B.Parasitism
C.Mutualism
D.Kommensalism

6. What is the name of the relationship between two organisms, in which one organism, as a result of evolutionary development, has adapted to its hosts as much as possible and only harms it?

A. Parasitism
B.Commensalism
C.Mutualism
D.Sinoikia

7. In the diagnosis of which disease, animal feces are examined and when three, quadrangular eggs are found, the size of which varies between 0.05-0.09 mm?

A.Moniesiosis
B.Cysticercosis
C.Echinococcosis

D.Cenuroz

8. In the diagnosis of which nematode disease in birds, feces is examined by the Fulleborn or Darling method and when eggs are found, oval in shape, 0.070-0.086 mm thick, 0.047-0.051 mm wide?

A.Ascariasis
B.Davenioz
C.Prostogonimosis
D.Knemidocoptosis

9. When diagnosing which nematode disease of sheep, fecal samples should be examined by the Berman-Orlov method for the detection of parasite larvae?

A.Dictyocaulesis
B.Fascioliasis
C.Psoroptosis
D.Estroz

10. What kind of trematode is a leaf-shaped, 2-3 cm wide, 09.8-1.2 cm wide, and some have a ribbon-shaped, 4-7.5 cm wide, and 0.6-1.2 cm wide?

A.Fasciola hepatica, F.gigantica
B.Paramphistomum cervi
C.Dicrocoelium lanceatum
D.Euritrema pancreaticum

11. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, and a freshwater mollusk from the genus Lymnea as an intermediate?

A.Fascioles
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas

12.What kind of disease occurs if animals graze in spring and autumn in low-lying pastures, where freshwater mollusks from the genus Lymnea are widespread?

A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Prostogonimoz

13. In which disease is the pathogenic effect of the parasite caused by mechanical damage to the liver parenchyma, inoculation of a chronic infection and intoxication of the body with the products of the parasite's vital activity?

A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz

14. What kind of disease is acute and chronic, with an acute course there is pallor of the mucous membranes, an increase in body temperature, impaired digestive function, diarrhea, and with a chronic one – a depressed state, standing from the herd, the appearance of edema, severe emaciation and death?

A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz

15. In what disease are the corpses of fallen animals exhausted, fluid with an admixture of blood has accumulated in the abdominal cavity, lymph nodes and liver are enlarged, bile ducts are dilated, the gallbladder is overflowing with thick bile, and parasites are found in them?

A.Fasciolosis
B.Dicroceliosis
C.Orientobilkhartsioz
D.Paramphistomatosis

16. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of golden yellow eggs in faeces, oval shape, size 0.13-0.15 x0.07-0.09 mm?

A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz

17. In which disease animals are dewormed with one of the following drugs: hexachlorparaxylene, hexychol, 4-x carbon chloride, ursovermid, filixan, rolenol, closantel, combitrem, facocide?

A.Fasciolosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Paramphistomatosis

18. For the prevention of what disease of animals should be carried out planned deworming, biothermic treatment of manure and destruction of intermediate hosts by mechanical, physical, chemical and biological means?

A.Fasciolosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Dicroceliosis

19. Which disease is a widespread fluke disease in more than 70 species of domestic and wild mammalian animals caused by flukes, parasitic in the bile ducts and gallbladder?

A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis

20. What disease is widespread everywhere, but its widespread occurrence is noted in the desert-pasture zone, where there are intermediate and additional hosts (land mollusks and ants)?

A.Dicroceliosis

B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
21. In which disease are the main pathoanatomic changes observed in the abomasum and duodenum 12, the mucous membrane of which is catarrhally hemorrhagically inflamed, mesenteric lymph nodes enlarged?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
22. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark gray eggs in the faeces, oval shape, size 0.12-0.17x0.07-0.1 mm, yolk cells are not completely filled with egg cavities?
A.Paramphistomatosis
B.Dicroceliosis
C.Fascioliasis
D.Orientobilkhartsioz
23.In which disease animals are dewormed with one of the following drugs: 4-x carbon chloride, bithionol, resorantel, gilomite and kerosene, which is used in aviation?
A. Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D. Fasciolosis
24. To prevent what trematodous disease of animals, planned deworming should be carried out, and in case of an outbreak of invasion, 3-4 weeks after pasture, preimaninal deworming of young animals is carried out twice at intervals of 10 days, biothermic treatment of manure and destruction of intermediate hosts?
A.Paramphistomatosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
25. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, a land mollusk from the genus Eulotalantzi as an intermediate, and grasshoppers from the genus Conocephalus and crickets – Oecanthus as an additional host?
A.Eurythremes
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
26.In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark brown eggs in the feces, oval shape, size 0,044-13-0 ,048x0,032-0,0036 mm?

A.Eurythrematosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
27. Which fluke disease is chronic with pronounced clinical signs of pancreatitis?
A.Eurythrematosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
28. In which disease animals are dewormed with one of the following drugs: bithionol, hexychol, hexychol C, tafen, panacur (fenbendazole)?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
29.In which disease are the main pathoanatomic changes observed in the ducts of the pancreas, the glandular part of the liver parenchyma atrophied, mesenteric lymph nodes enlarged?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
30. What fluke disease of poultry occurs chronically with pronounced 3-stage clinical signs (thinned eggshell, egg wash and duck gait?
A.Prostogonimosis
B.Ascariasis
C.Heterakidosis
D.Davenioz
31.What kind of disease occurs chronically, in which a depressed state, an increase in body temperature, impaired digestive function, panoses, the appearance of edema, severe emaciation and death?
A.Orientobilkhartsioz
B.Dicroceliosis
C.Paramphistomatosis
D.Fasciolosis
32. Which of the following trematodes are bisexual parasites, the size of males exceeds the size of females, males have 78-80 testes, and the female has one egg in the uterus, inside of which there are formed miracidia?
A.Orientobilhartius
B.Dicrocelium
C.Paramphistomats
D. Fascioles

33. In the diagnosis of which disease, the parasite's eggs are first cultured in a thermostat, after which the Berman-Orlov method examines for the detection of parasite larvae?
A.Orientobilcharciosis
B. Paramphistomatosis
C. Dicroceliosis
D. Fasciolosis
34. In which disease animals are dewormed with one of the following drugs: fuadin at a dose of 0.3 ml / kg, albilgar at a dose of 0.02-sh,03 g / kg, droncite at a dose of 0.05 g / kg, azinox at a dose of 25 mg / kg of animal weight?
A.Orientobilcharciosis
B. Paramphistomatosis
C.Dicroceliosis
D. Fasciolosis
35. To prevent which chronically occurring trematode disease of animals should carry out planned deworming, biothermic treatment of manure, destruction of intermediate hosts, which are ear-shaped mollusks of the genus Lymnea and feeding animals according to the diet?
A.Orientobilkhartsioz
B.Paramphistomatosis
C.Fascioliasis
D.Dicroceliosis
36. What is the name of the disease of cattle caused by cestodes, parasitizing in the intermuscular connective tissue, skeletal muscles, heart, tongue?
A.Cysticercosis
B. Echinococcosis
C. Cenurosis
D. Monesiosis
37. In which disease of cattle, the degree of infection is on average 10% and about 1% of meat, forgotten animals are disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
38. What kind of cestode does the larval (vesicular) stage of which has a rounded-oval shape, consisting of three shells, the length of the bubble is 5-15 mm, with a width of 3-8 mm, there is one unarmed scolex inside the bubble?
A.Cysticercus bovis
B. Cysticercus cellulosae
C. Coenurus cerebralis
D.Echinococcus granulosus
39. What kind of cestode does the larval (vesicular) stage of which has an ellipsoid shape, consisting of three shells, the length of the bladder is 10-20 mm, with a width of 5-10 mm, there is one armed scolex inside the bladder?

A.Cysticercus cellulosae
B. Cysticercus bovis
C. Coenurus cerebralis
D. Echinococcus granulosus
40. What kind of cestode does it belong to, having a ribbon-like shape, a length of more than 10 m, a width of 12-14 mm, whose strobila consists of dozens, hundreds and even thousands of segments?
A.Taeniarynchus saginatus
B.Taenia solium
C.Multiceps multiceps
D.Echinococcus granulosus
41. Which cestode in its biological development cycle is used as a definitive host of humans, and as an intermediate host – cattle, buffaloes, yak, zebu and sometimes reindeer?
A.Cysticercus bovis
B.Cysticercus cellulosae
C.Coenurus cerebralis
D.Echinococcus granulosus
42. Which cestode develops with the participation of the main one, which is only a human, and intermediate hosts, which are pigs, wild boars, hares, rabbits, bears, camels as well as humans?
A.Cysticercus cellulosae
B. Cysticercus bovis
C.Coenurus cerebralis
D.Echinococcus granulosus
43. What kind of cestodosis disease will be widespread if the livestock farm does not comply with zoohygienic requirements, the absence of a closed toilet and sanitary and educational work is not carried out among the general population, especially stockmen?
A.Cysticercosis of cattle
B.Echinococcosis
C.Monesiosis
D.Cenuroz
44. When preventing what kind of cestodose disease, it is necessary to comply with the rules of veterinary sanitation, animals should be slaughtered in special slaughter points and it is mandatory to conduct a vet.san.examination of meat and meat products of slaughtered animals?
A.Cysticercosis of cattle
B. Echinococcosis
C.Monesiosis
D.Cenuroz
45. In which disease should the VSE be carried out meat and offal, specially forgotten for meat or forcibly forgotten animals, and when parasites are detected on

an area of 40 cm2 of the muscles of the head and heart and on one incision of the muscles of the carcass, the whole carcass is disposed of?
A.Cysticercosis
B.Echinococcosis
V.Cenurosis
G.Monesiosis
46. Which pathogen of anthropozoonous, cestodose, asymptomatic mammalian animal disease is parasitized in parenchymal organs, mainly in the liver and lungs?
A.Echinococcosis
B.Cysticercosis
C.Cenuroz
D.Moniesiosis
47. Which cestode develops with the participation of the main one, which are dogs and other carnivores, and intermediate hosts, which are mammalian animals?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
48. To prevent which chronically occurring cestodosis disease of animals should be carried out planned deworming, biothermic treatment of manure of the main hosts, disinfection of infected parenchymal organs of intermediate hosts?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
49. What kind of cestode does it belong to, having a ribbon-shaped, 40-100 cm long, 5-6 mm wide, the strobila of which consists of hundreds of segments, in one mature segment there are up to 50 thousand eggs?
A. Multiceps multiceps
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
50. How many parts does the body of the cestode consist of?
A.of three
B.of two
C.of five
D.the body of the cestode is not divided into parts
51. Which part of the body of the cestode is the growth zone?
A.neck
B.head (scolex)
C.hermafortid segment
D.mature segment
52. Where are the hermaphordite segments of the cestode?
A. in the anterior part of the body

B. the middle part of the body
C. the back part of the body
D.About all parts of the body
53. In the posterior part of the body of the cestode, which genitals are preserved?
A.uterus
B.ovary
C.testis
D.ovary
54. Cestodes develop with the participation of how many hosts?
A.2 and 3 hosts
B.two hosts
C.three hosts
D.one host
55. Which cestode's scolexes are armed with suckers and chitinized hooks?
A.Echinococcus and multiceps
B.moniezium and avitellins
C.tizaniezium and multiceps
D.stylezium and echinococcus
56. In skoleks, what kind of cestodes do not have chitinized hooks?
A.monieziy
B.multiceps
C.echinococcus
D.teniarinhus
57. Uterus which cestode is open type, develop with the participation of three hosts, larvae in the form of worms?
A.Diphyllobothrium latum
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
58. Uterus which cestode is a closed type, develop with the participation of two hosts, larvae in the form of a bubble that is filled with fluids?
A.Taeniarynchus saginatus
B.Ligula intestinalis
C.Moniezia expansa
D. Diphyllobothrium latum
59. The form of which trematodes in the form of threads and is bisexual?
A. Orientobilchartsii
B.Dicrocelium
C.Paramphistomatids
D. Fascioles
60. The development cycle of the causative agent of which trematode disease occurs only in endogenous conditions?
A.Dicrocelium
B. Orientobilcharcium

B.Paramphistomatids
G.Fascioles
61. What trematodous diseases are also found in humans?
A.Fascioliasis, dicroceliosis
B.Paramphistomatosis and dicrocelium
C.Orientobilcharcosis and paramphistomatosis
D.Fascioles and echinococcosis
62. In vertebrates, what types of trematodous diseases are found?
A.in all vertebrates
B.in fish
C.in birds
D.in the experiencing
63. Which method is acceptable to combat trematodous diseases?
A.Devastation
B.Deworming
C.Disinvasion
D.Fighting shellfish
64. Why do the ribbon forms of cestodes live in the small intestine?
A. They do not have a digestive system
B.Their bodies are very long
C. They have scolexes (head)
D.They have a poorly developed neural system
65. In what kind of trematode disease is percutaneous infection of animals observed?
A.In orientobilcharciosis
B.With fascioliasis
C.With paramphistomatosis
D.With eurythrematosis
66. Fasciolahepatica in how many days does it reach sexual maturity in the body of a definitive host?
A.75-80 days
B.80-90 days
C.80-130 days
D.90-150 days
67. Fasciolagigantica in how many days does it reach sexual maturity in the body of a definitive host?
A.90-120 days
B. 80-90 days
C.80-130 days
D.75-80 days
68. In which zones is fascioliasis most common?
A. in the irrigation and foothill-mountain zones
B. in the foothill zone
C.in the desert pasture zone
D.in all zones

69. How is the diagnosis made in case of vital fascioliasis?
A.by the method of sequential washing of feces
B. method: Berman- Orlov
C.by the Vidamethod
D. by the Fulleborn method
70. Where does the parthenogenetic cycle of fasciole development occur?
A.in the body of an intermediate host- a mollusk
B. in the body of a definitive host
C.in water
D.in the body of a parasite
71. Which trematode disease is acute, chronic and mixed forms?
A.Fascioliasis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
72. What does the term "Devastation" mean?
A.destruction of parasites, helminths in all stages of development with all methods of control
B.liberation of the animal organism from parasites
C.destruction of intermediate hosts of helminths
D.destruction of invasive larvae of helminths
73. Who is the author of the term "Devastation"?
A.K.I.Scriabin
B. N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
74. Show the scientist who for the first time thoroughly studied orientobil-khartsioz in the conditions of our republic?
A.D.A.Azimov
B. N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
75. Who is the founder of helminthological science in Uzbekistan?
A.N.V.Badanin
B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
76. The main (definitive) host of which anthropozoonous disease is a person?
A.Cysticercosis of cattle
B. Teniosis
C. Echinococcosis
D. Cenurosis
77. Who is the author of the albendozol-copper vitriol salt mixture?
A.A.O.Oripov

B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
78. Which of the following scientists has thoroughly studied the biology of the causative agent of dicroceliosis?
A.B.S. Salimov
B. D.A.Azimov
C. A.O.Oripov
D. V.S. Ershov
79. Which of the following scientists is the first doctor of Sciences in the specialty "Helminthology"?
A.I.H.Irgashev
B.D.A.Azimov
C. A.O.Oripov
D. B.S. Salimov
80. Which cestdosis disease is mainly widespread among 1.5-8-month-old lambs, goats and calves?
A.shell mites of the genus Scheloribates
B. freshwater mollusks
C. land mollusks
D. ants
81. What is the intermediate host of the causative agent of moniesiosis?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
82. In the treatment of which disease is it recommended to use phenasal, phenalidone, 1-2% aqueous solution of copper sulfate, panacur, rintal and drugs from the albendazole group?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
83. For the prevention of what disease should animals be fed salt-phenothiazine-copper sulfate top dressing, the composition of which consists of 1% copper sulfate, 10% phenothiazine and 89% table salt, from October to June from a calculation of 1.0 per head?
A.Moniesiosis
B. Echinococcosis
C. Cenurosis
D. Cysticercosis
84. The sexually mature form of the causative agent of which nematode disease of ungulates parasitizes in the small intestine, and the larvae migrate hepatopulmonally?
A.Parascaris equorum

B. Paranaplocephala mamillana
C. Anaplocephala magna
D. Anaplocephala perfoliata
85. In the treatment of which nematode disease of single-hoofed animals are used piperazine salts, phenothiazine, panacur (fenbendazole), rintal (febantel), 4-carbon chloride, albendazole and its analogues?
A.Paraskaridosis
B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
86. To prevent which nematode disease of ungulates should preventive deworming of suckling foals be carried out for the first time in August, the second time after weaning, and adult animals for the first time in March-April, the second time in October-November?
A.Paraskaridosis
B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
87. Which nematode disease of sheep proceeds chronically, the causative agent of which parasitizes in the bronchi and trachea, as a result of which the infected animal has a violation of the function of the respiratory organs?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
88. Which nematode is large, filamentous, milky-white, bisexual, males 3-8 cm long, females 5-15 cm, spicule dark brown, boot-shaped, 0.4-0.6 mm long?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
89. Which nematode is a geohelminth, in the external environment the larva reaches the invasive stage in 6-7 days, the prepatent period of development is 3-4 months, the patent period is 1.5-2 years?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
90. With which nematode disease of sheep there is a weak, then increasing cough during movement, after the previous rest, at night, bronchitis, serous-purulent discharge from the nose, which dry out and form crusts?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis

D. Estrosis
91. When deworming which nematode disease use 1 or 2% solutions of copper sulfate, phenothiazine, naphthamone, panacur (fenbendazole), rintal (febantel), albendazole and its analogues?
A.Hemonchosis
B. Fasciolosis
C. Paramphistomatosis
D. Dicroceliosis
92. In which nematode disease do chickens of 2-10 months of age mainly become infected, and adult birds are parasitic carriers?
A.Ascariasis
B. Prostogonimosis
C. Daveniosis
D. Rayetinoz
93. In which nematode disease, the causative agent of which is parasitized in the conjunctival sac, under the third eyelid, in the ducts of the lacrimal gland and the lacrimal-nasal canal, as a result of which animals are observed lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids turning into keratitis, ulcers on the cornea, a thorn?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
94. In which helminthic disease are the characteristic clinical signs of lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids, turning into keratitis, ulcers of the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
95. In the treatment of which nematode is used 3% boric acid, Lugol solution, ditrazine citrate, iodine, lysol and others?
A.Telyaziosis
B. Setariosis
C. Onchocerciasis
D. Teileriosis
96. What chronically occurring disease of horses is characterized by skin lesions at the root of the tail, severe itching, eczema, dermatitis and severe emaciation of the animal?
A.Oxyurosis
B. Paraskaridosis
C. Anaplocephalosis
D. Pyroplasmosis

97. Which nematode of ungulates develops in a direct way, that is, without the participation of an intermediate host, the prepatent period of development is equal to 3-4 weeks, and the patent period is 6-8 months?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis
98. In which nematode disease of horses is sick mainly young animals up to one year old and old horses?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis
99. When deworming which disease is used 4-chlorinated carbon, piperazine and its salts, phenothiazine, panacur (fenbendazole), rintal (febantel), tetramizole and mebenvet in the form of granules?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Anoplocephalosis
100. For the prevention of which disease should horses undergo planned dehelminthization every 3 months, biothermic treatment of manure and full feeding of animals according to the diet?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Nuttaliosis
101. In which case of nematode diseases of birds, the pathogenic effect of the pathogen consists of mechanical damage to the mucous membrane and glands of the intestine, toxic effects and opens the gates to secondary infections?
A.Ascariasis
B. Knemidocoptosis
C. Histomonosis
D. Borreliosis
102. In the treatment of which nematode disease of birds piperazine and its salts, phenothiazine, nilverm, benacil, fenbendazole (panacur), rintal (febantel) are used?
A.Ascariosis
B. Prostogonimosis
C. Daveniosis
D. Histomonosis
103. What is the relationship of organisms, in which one organism benefits, and the other neither benefits nor harm?
A.Sinoikia
B.Mutualism

C.Parasitism
D.Predation
104. What is the name of the relationship between two organisms, in which one of the organisms feeds on the remains of the food of the other organism, without harming it?
A.Commensalism
B.Mutualism
V.Sinoikia
G.Parasitism
105. What is the name of the relationship between two organisms, in which one organism is stronger than its prey, which it immediately kills and eats?
A.Predation
B.Parasitism
C.Mutualism
D.Kommensalism
106. What is the name of the relationship between two organisms, in which one organism, as a result of evolutionary development, has adapted to its owners as much as possible and only harms it?
A.Parasitism
B.Commensalism
V.Mutualism
G.Sinoikia
107. In the diagnosis of which disease, animal feces are examined and when three, quadrangular eggs are found, the size of which varies between 0.05-0.09 mm?
A.Moniesiosis
B.Cysticercosis
B.Echinococcosis
G.Cenuroz
108. In the diagnosis of which nematode disease in birds, feces is examined by the Fulleborn or Darling method and when eggs are found, oval in shape, 0.070-0.086 mm thick, 0.047-0.051 mm wide?
A.Ascariasis
B.Davenioz
B.Prostogonimosis
G.Knemidocoptosis
109. When diagnosing which nematode disease of sheep, fecal samples should be examined by the Berman-Orlov method for the detection of parasite larvae?
A.Dictyocaulesis
B.Fascioliasis
C.Psoroptosis
D.Estroz
110. What kind of trematode is a leaf-shaped, 2-3 cm wide, 09.8-1.2 cm wide, and some have a ribbon-shaped, 4-7.5 cm wide, and 0.6-1.2 cm wide?
A.Fasciola hepatica, F.gigantica

B.Paramphistomum cervi
C.Dicrocoelium lanceatum
D.Euritrema pancreaticum
111. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, and a freshwater mollusk from the genus Lymnea as an intermediate?
A.Fascioles
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
112. What kind of disease occurs if animals graze in spring and autumn in low-lying pastures, where freshwater mollusks from the genus Lymnea are widespread?
A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Prostogonimoz
113. In which disease is the pathogenic effect of the parasite caused by mechanical damage to the liver parenchyma, inoculation of a chronic infection and intoxication of the body by the products of the parasite?
A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
114. What kind of disease is acute and chronic, with an acute course there is pallor of the mucous membranes, an increase in body temperature, impaired digestive function, diarrhea, and with a chronic one – a depressed state, standing from the herd, the appearance of edema, severe emaciation and death?
A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
115. In what disease are the corpses of fallen animals exhausted, fluid with an admixture of blood has accumulated in the abdominal cavity, lymph nodes and liver are enlarged, bile ducts are dilated, the gallbladder is overflowing with thick bile, and parasites are found in them?
A.Fasciolosis
B.Dicroceliosis
C.Orientobilkhartsioz
D.Paramphistomatosis
116. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of golden yellow eggs in the faeces, oval shape, size 0.13-0.15 x0.07-0.09 mm?
A.Fasciolosis
B.Dicroceliosis

C.Paramphistomatosis
D.Orientobilkhartsioz
117. In which disease animals are dewormed with one of the following drugs: hexachlorparaxylene, hexychol, 4-x carbon chloride, ursovermid, filixan, rolenol, closantel, combitrem, facocide?
A.Fasciolosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Paramphistomatosis
118. For the prevention of what disease of animals should be carried out planned deworming, biothermic treatment of manure and destruction of intermediate hosts by mechanical, physical, chemical and biological means?
A.Fasciolosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Dicroceliosis
119. Which disease is a widespread fluke disease in more than 70 species of domestic and wild mammalian animals caused by flukes, parasitic in the bile ducts and gallbladder?
A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
120. What disease is widespread everywhere, but its widespread occurrence is noted in the desert-pasture zone, where there are intermediate and additional hosts (land mollusks and ants)?
A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
121. In which disease are the main pathoanatomic changes observed in the abomasum and duodenum 12, the mucous membrane of which is catarrhally hemorrhagically inflamed, mesenteric lymph nodes enlarged?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
122. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark gray eggs in the faeces, oval shape, size 0.12-0.17x0.07-0.1 mm, yolk cells are not completely filled with egg cavities?
A. Paramphistomatosis
B.Dicroceliosis
C.Fascioliasis

D.Orientobilkhartsioz
123. In which disease animals are dewormed with one of the following drugs: 4-carbon chloride, bithionol, resorantel, gilomite and kerosene, which is used in aviation?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D. Fasciolosis
124. To prevent what trematode disease of animals, planned deworming should be carried out, and in case of an outbreak of invasion, 3-4 weeks after pasture, preimaninal deworming of young animals is carried out twice at intervals of 10 days, biothermic treatment of manure and destruction of intermediate hosts?
A.Paramphistomatosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
125. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, a land mollusk from the genus Eulotalantzi as an intermediate, and grasshoppers from the genus Conocephalus and crickets – Oecanthus as an additional host?
A.Eurythremes
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
126. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark brown eggs in the feces, oval shape, size 0,044-13-0 ,048x0,032-0,0036 mm?
A.Eurythrematosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
127. Which trematode disease is chronic with pronounced clinical signs of pancreatitis?
A.Eurythrematosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
128. In which disease animals are dewormed with one of the following drugs: bithionol, hexychol, hexychol C, tafen, panacur (fenbendazole)?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis

129. In which disease are the main pathoanatomic changes observed in the ducts of the pancreas, the glandular part of the liver parenchyma atrophied, mesenteric lymph nodes enlarged?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
130. What fluke disease of poultry occurs chronically with pronounced 3-stage clinical signs (thinned eggshell, egg wash and duck gait?
A.Prostogonimosis
B.Ascariasis
C.Heterakidosis
D.Davenioz
131. What kind of disease occurs chronically, in which a depressed state, an increase in body temperature, a violation of the digestive function, panoses, the appearance of edema, severe emaciation and death?
A.Orientobilkhartsioz
B.Dicroceliosis
C.Paramphistomatosis
D.Fasciolosis
132. Which of the following trematodes are bisexual parasites, the size of males exceeds the size of females, males have 78-80 testes, and the female has one egg in the uterus, inside of which there are formed miracidia?
A.Orientobilhartius
B.Dicrocelium
C.Paramphistomats
D. Fascioles
133. In the diagnosis of which disease, the parasite's eggs are first cultured in a thermostat, after which the Berman-Orlov method examines for the detection of parasite larvae?
A.Orientobilcharciosis
B. Paramphistomatosis
C. Dicroceliosis
D. Fasciolosis
134. In which disease animals are dewormed with one of the following drugs: fuadin at a dose of 0.3 ml / kg, albilgar at a dose of 0.02-sh,03 g / kg, droncite at a dose of 0.05 g / kg, azinox at a dose of 25 mg / kg of animal weight?
A.Orientobilcharciosis
B. Paramphistomatosis
C.Dicroceliosis
D. Fasciolosis
135. In order to prevent what chronically occurring trematode disease of animals should be carried out planned deworming, biothermic treatment of manure,

destruction of intermediate hosts, which are ear-shaped mollusks of the genus Lymnea and feeding animals according to the diet?
A.Orientobilharciosis
B.Paramphistomatosis
C.Fascioliasis
D.Dicroceliosis
136. What is the name of the disease of cattle caused by cestodes, parasitizing in the intermuscular connective tissue, skeletal muscles, heart, tongue?
A.Cysticercosis
B. Echinococcosis
C. Cenurosis
D. Monesiosis
137.In which disease of cattle, the degree of infection is on average 10% and about 1% of meat, forgotten animals are disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
138. What kind of cestode does the larval (vesicular) stage of which has a rounded-oval shape, consisting of three shells, the length of the bubble is 5-15 mm, with a width of 3-8 mm, there is one unarmed scolex inside the bubble?
A.Cysticercus bovis
B. Cysticercus cellulosae
C. Coenurus cerebralis
D.Echinococcus granulosus
139. What kind of cestode does the larval (vesicular) stage of which has an ellipsoid shape, consisting of three shells, the length of the bladder is 10-20 mm, with a width of 5-10 mm, there is one armed scolex inside the bladder?
A.Cysticercus cellulosae
B. Cysticercus bovis
C. Coenurus cerebralis
D. Echinococcus granulosus
140. What kind of cestode does it belong to, having a ribbon-like shape, a length of more than 10 m, a width of 12-14 mm, whose strobila consists of dozens, hundreds and even thousands of segments?
A.Taeniarynchus saginatus
B.Taenia solium
C.Multiceps multiceps
D.Echinococcus granulosus
141. Which cestode in its biological development cycle is used as a definitive host of humans, and as an intermediate host – cattle, buffaloes, yak, zebu and sometimes reindeer?
A.Cysticercus bovis
B.Cysticercus cellulosae

C.Coenurus cerebralis
D.Echinococcus granulosus
142. Which cestode develops with the participation of the main one, which is only human, and intermediate hosts, which are pigs, wild boars, hares, rabbits, bears, camels as well as humans?
A.Cysticercus cellulosae
B. Cysticercus bovis
C.Coenurus cerebralis
D.Echinococcus granulosus
143. What kind of cestodosis disease will be widespread if the livestock farm does not comply with zoohygienic requirements, the absence of a closed toilet and sanitary and educational work is not carried out among the general population, especially cattle breeders?
A.Cysticercosis of cattle
B.Echinococcosis
C.Monesiosis
D.Cenuroz
144. When preventing what kind of cestodose disease, it is necessary to comply with the rules of veterinary sanitation, animals should be slaughtered in special slaughterhouses and it is mandatory to carry out a vet.san.examination of meat and meat products of slaughtered animals?
A.Cysticercosis of cattle
B. Echinococcosis
C.Monesiosis
D.Cenuroz
145. In which disease should the VSE be carried out of meat and offal, specially forgotten for meat or forcibly forgotten animals, and when parasites are detected on an area of 40 cm2 of the muscles of the head and heart and on one incision of the muscles of the carcass, the entire carcass is disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
146. Which pathogen of anthropozoonous, cestodose, asymptomatic mammalian animal disease is parasitized in parenchymal organs, mainly in the liver and lungs?
A.Echinococcosis
B.Cysticercosis
C.Cenuroz
D.Moniesiosis
147. Which cestode develops with the participation of the main one, which are dogs and other carnivores, and intermediate hosts, which are mammalian animals?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis

D.Cysticercus cellulosae
148. To prevent which chronically occurring cestodosis disease of animals should be carried out planned deworming, biothermic treatment of manure of the main hosts, disinfection of infected parenchymal organs of intermediate hosts?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
149. What kind of cestode does it belong to, having a ribbon-shaped, 40-100 cm long, 5-6 mm wide, whose strobila consists of hundreds of segments, in one mature segment there are up to 50 thousand eggs?
A. Multiceps multiceps
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
150. How many parts does the body of the cestode consist of?
A. of three
B.of two
C.of five
D.the body of the cestode is not divided into parts
151. Which part of the body of the cestode is the growth zone?
A.neck
B.head (scolex)
C.hermafortid segment
D.mature segment
152. Where are the hermaphordite segments of the cestode?
A. in the anterior part of the body
B. the middle part of the body
C. the back part of the body
D.About all parts of the body
153. In the posterior part of the body of the cestode, which genitals are preserved?
A.uterus
B.ovary
C.testis
D.ovary
154. Cestodes develop with the participation of how many hosts?
A.2 and 3 hosts
B.two hosts
C.three hosts
D. One owner
155. Which cestode's scolexes are armed with suckers and chitinized hooks?
A.Echinococcus and multiceps
B.moniezium and avitellins
C.tizaniezium and multiceps

D.stylezium and echinococcus
156. In skoleks, what kind of cestodes do not have chitinized hooks?
A.monieziy
B.multiceps
C.echinococcus
D.teniarinhus
157. Uterus which cestode is open type, develop with the participation of three hosts, larvae in the form of worms?
A.Diphyllobothrium latum
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
158. The uterus of which cestode is a closed type, develop with the participation of two hosts, larvae in the form of a bubble that is filled with fluids?
A.Taeniarynchus saginatus
B.Ligula intestinalis
C.Moniezia expansa
D. Diphyllobothrium latum
159. The form of which trematodes in the form of threads is bisexual?
A. Orientobilchartsii
B.Dicrocelium
C.Paramphistomatids
D. Fascioles
160. The development cycle of the causative agent of which trematode disease occurs only in endogenous conditions?
A.Dicrocelium
B. Orientobilcharcium
C.Paramphistomatids
D.Fascioles
161. What trematodous diseases are also found in humans?
A.Fascioliasis, dicroceliosis
B.Paramphistomatosis and dicrocelium
C.Orientobilcharcosis and paramphistomatosis
D.Fascioles and echinococcosis
162. In vertebrates, what types of trematodous diseases are found?
A.in all vertebrates
B.in fish
C.in birds
D.in testers
163.What method is acceptable to combat trematodous diseases?
A.Devastation
B.Deworming
C.Disinvasion
D.Fighting shellfish

164.Why do the ribbon forms of cestodes live in the small intestine?
A. They do not have a digestive system
B.Their bodies are very long
C. They have scolexes (head)
D.They have a poorly developed neural system
165.In what kind of trematode disease is percutaneous infection of animals observed?
A.At orientobilkhartsioz
B.With fascioliasis
C.With paramphistomatosis
D.With eurythrematosis
166. Fasciolahepatica in how many days does it reach sexual maturity in the body of a definitive host?
A.75-80 days
B.80-90 days
C.80-130 days
D.90-150 days
167. Fasciolagigantica in how many days does it reach sexual maturity in the body of a definitive host?
A.90-120 days
B. 80-90 days
C.80-130 days
D.75-80 days
168. In which zones is fascioliasis most common?
A. in the irrigation and foothill-mountain zones
B. in the foothill zone
C. in the desert pasture zone
D.in all zones
169. How is the diagnosis made in case of vital fascioliasis?
A.by the method of sequential washing of feces
B. Berman-Orlovmethod
C.Vida method
D. Fulleborn method
170. Where does the parthenogenetic cycle of fasciole development occur?
A. in the body of an intermediate host- a mollusk
B. in the body of a definitive host
C. in water
D. in the body of a parasite
171. Which trematode disease is acute, chronic and mixed forms?
A.Fascioliasis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
172. What does the term "Devastation" mean?

A.destruction of parasites, helminths in all stages of development with all methods of control
B.liberation of the animal organism from parasites
C.destruction of intermediate hosts of helminths
D.destruction of invasive larvae of helminths
173. Who is the author of the term "Devastation"?
A.K.I.Scriabin
B.N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
174. Show the scientist who for the first time thoroughly studied orientobil-khartsioz in the conditions of our republic?
A.D.A.Azimov
B. N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
175. Who is the founder of helminthological science in Uzbekistan?
A.N.V.Badanin
B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
176. The main (definitive) host of which anthropozoonous disease is a person?
A.Cysticercosis of cattle
B. Teniosis
C. Echinococcosis
D. Cenurosis
177. Who is the author of the albendozol-copper vitriol salt mixture?
A.A.O.Oripov
B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
178. Which of the following scientists has thoroughly studied the biology of the causative agent of dicroceliosis?
A.B.S. Salimov
B. D.A.Azimov
C. A.O.Oripov
D. V.S. Ershov
179. Which of the following scientists is the first doctor of Sciences in the specialty "Helminthology"?
A.I.H.Irgashev
B. D.A.Asimov
C.A.O.Oripov
D. B.S. Salimov

180. Which cestdosis disease is mainly widespread among 1.5-8-month-old lambs, goats and calves?
A.shell mites of the genus Scheloribates
B. freshwater mollusks
C. land mollusks
D. ants
181. What is the intermediate host of the causative agent of moniesiosis?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
182. In the treatment of which disease is it recommended to use phenasal, phenalidone, 1-2% aqueous solution of copper sulfate, panacur, rintal and drugs from the albendazole group?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
183. For the prevention of which disease should animals be fed salt-phenothiazine-copper sulfate top dressing, the composition of which consists of 1% copper sulfate, 10% phenothiazine and 89% table salt, from October to June from a calculation of 1.0 per head?
A.Moniesiosis
B. Echinococcosis
C. Cenurosis
D. Cysticercosis
184. The sexually mature form of the causative agent of which nematode disease of ungulates parasitizes in the small intestine, and the larvae migrate hepatopulmonally?
A.Parascaris equorum
Б. Paranaplocephala mamillana
В. Anaplocephala magna
Г. Anaplocephala perfoliata
185. In the treatment of which nematode disease of single-hoofed animals are used piperazine salts, phenothiazine, panacur (fenbendazole), rintal (febantel), 4-x carbon chloride, albendazole and its analogues?
A.Paraskaridosis
B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
186. To prevent which nematode disease of ungulates should preventive deworming of suckling foals be carried out for the first time in August, the second time after weaning, and adult animals for the first time in March-April, the second time in October-November?
A.Paraskaridosis

B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
187. Which nematode disease of sheep proceeds chronically, the causative agent of which parasitizes in the bronchi and trachea, as a result of which the infected animal has a violation of the function of the respiratory organs?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
188. Which nematode is large, filamentous, milky white, bisexual, males 3-8 cm long, females 5-15 cm, spicule dark brown, boot-shaped, 0.4-0.6 mm long?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
189. Which nematode is a geohelminth, in the external environment the larva reaches the invasive stage in 6-7 days, the prepatent period of development is 3-4 months, the patent period is 1.5-2 years?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
190. In which nematode disease of sheep is there a weak, then increasing cough during movement, after a previous rest, at night, bronchitis, serous-purulent discharge from the nose, which dry out and form crusts?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
191. When deworming which nematode disease use 1 or 2% solutions of copper sulfate, phenothiazine, naphthamone, panacur (fenbendazole), rintal (febantel), albendazole and its analogues?
A.Hemonchosis
B. Fasciolosis
C. Paramphistomatosis
D. Dicroceliosis
192. Which nematode disease mainly infects chickens 2-10 months of age, and adult birds are parasitic carriers?
A.Ascariasis
B. Prostogonimosis
C. Daveniosis
D. Rayetinoz

193. In which nematode disease, the causative agent of which is parasitized in the conjunctival sac, under the third eyelid, in the ducts of the lacrimal gland and the lacrimal-nasal canal, as a result of which animals are observed lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids turning into keratitis, ulcers on the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
194. In which helminthic disease are the characteristic clinical signs of lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids, turning into keratitis, ulcers of the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
195. In the treatment of which nematode is used 3% boric acid, Lugol solution, ditrazine citrate, iodine, lysol and others?
A.Telyaziosis
B. Setariosis
C. Onchocerciasis
D. Teileriosis
196. What chronically occurring disease of horses is characterized by skin lesions at the root of the tail, severe itching, eczema, dermatitis and severe emaciation of the animal?
A.Oxyurosis
B. Paraskaridosis
C. Anaplocephalosis
D. Pyroplasmosis
197. Which nematode of ungulates develops in a direct way, that is, without the participation of an intermediate host, the prepatent period of development is equal to 3-4 weeks, and the patent period is 6-8 months?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis
198. In which nematode disease of horses is sick mainly young animals up to one year old and old horses?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis

199. When deworming which disease is used 4-chlorinated carbon, piperazine and its salts, phenothiazine, panacur (fenbendazole), rintal (febantel), tetramizole and mebenvet in the form of granules?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Anoplocephalosis
200. For the prevention of which disease should horses undergo planned dehelminthization every 3 months, biothermic treatment of manure and full feeding of animals according to the diet?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Nuttaliosis

4.8.2-INTERMEDIATE CONTROL

TEST QUESTIONS

1. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis
C. Multiceps multiceps
D. Echinococcus granulosus
2. When diagnosing which protozoal disease of horses, prepare smears-prints from the peripheral blood of animals and on the basis of this detect the pathogen?
A.Nuttalliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
3. In which disease are the main pathoanatomic changes observed in the genitals of animals, accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis
D. Teileriosis
4. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
A.Trichomoniasis
B. Pyroplasmosis
C. Fascioliasis
D. Cysticercosis
5. Which protozoal diseases of cattle are acutely and subacutely occurring and accompanied by fever, hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems?
A.Pyroplasmosis
B. Trichomoniasis
C. Eimeriosis
D. Balantidiaz
6. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is required, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?
A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis
7. What kind of protozoal diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in

lymph nodes, fever, anemia, disruption of the cardiovascular and digestive systems, exhaustion and a high percentage of mortality?
A.Teileriosis
B. Piroplasmosis
C. Beznoitiosis
D. Toxoplasmosis
8. The causative agent of what protozoal disease, once in the animal's body with tick saliva, multiplies at the beginning in the lymph nodes and forms macro- and microschizonts (garnet bodies), then macro- and micromerozoites, which are embedded in red blood cells?
A.Teileriosis
B. Benzoithiosis
C. Pyroplasmosis
D. Toxoplasmosis
9. At what disease croup. in cattle, the percentage of infection of erythrocytes with the pathogen is 80-95%?
A.Teileriosis
B. Piroplasmosis
C. Beznoitiosis
D. Toxoplasmosis
10. Which genus of ixodes mites is the carrier of the causative agent of bovine taileriosis?
А.Hyalomma
Б. Ixodes
В. Boophylus
Г. Haemaphysalis
11. Which genus of ixodes mites is the carrier of the causative agent of bovine babesiosis?
A.Ixodes
Б. Hyalomma
В. Boophylus
Г. Haemaphysalis
12. Which genus of ixodes mites is the carrier of the causative agent of piroplasmosis of cattle?
A.Boophylus calcaratus
Б. Ixodes
В. Hyalomma
Г. Haemaphysalis
13. Which genus of ixodes mites is the carrier of the causative agent of pyroplasmosis of sheep and goats?
A.Rhipicephalus bursa
Б. Ixodes
В. Hyalomma
Г. Haemaphysalis

14. When diagnosing which protozoal disease of cattle, smears prepared from lymph nodes are examined at the beginning of the disease, and during the period of clinical signs, blood smears from peripheral vessels are examined to detect the pathogen?
A. Teileriosis
B. Toxoplasmosis
C. Beznoitiosis
D. Pyroplasmosis
15. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists of All-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute are carefully used?
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Piroplasmosis
16. Which genus and species of ixodes mites are carriers of the causative agent of horse nuttaliosis?
A.Ixodes and Boophilus calcaratus
B.DermacentorandHyalommaplumbeum
C.BoophylusandRhipicephalusbursa
D.HaemaphysalisandIxodes ricinus
17. What protozoal disease of horses is acute, subacute and chronic, accompanied by fevers, jaundice, anemia, hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?
A.Nuttalliosis
B.Pyroplasmosis
C.Oxyurosis
D.Paraskaridosis
18. Which pathogen of protozoal disease has an oval, pear-shaped, barrel-shaped shape, they are located in red blood cells by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?
A.Nuttalia equi
Б.Piroplasma caballi
B.Theileria annulata
Г.Piroplasma bigeminum
19. What contagious disease of sheep is acute and chronic, characterized by itching of the skin, hair loss and exhaustion of the body, sometimes ends fatally?
A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz
20. In which case of arachnosis of sheep disease are animals with a thick coat and high humidity mainly infected during the winter periods of the year?

A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz
21. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
22. What is the disease of horses characterized by acute, subacute and chronic course, the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?
A.Pyroplasmosis
B.Paraskaridosis
C.Oxyurosis
D.Rhinestrosis
23. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
24. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis
C. Hypodermatosis
D.Siphunculator
25. In which invasive disease of cattle, the disease proceeds acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
26. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?

A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
27. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
28. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariasis
C.Trichocephalosis
D. Psoroptosis
29. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
30. For the prevention of any disease of animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
31. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
32. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages,

superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
33. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general strengthening drugs be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
34. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis
C.Estroz
D. no answer
35. The disease of rabbits, the infection of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
36. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
37. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
38. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis

D.Eimeriosis
39. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
40. In which parasite (pathogen), after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating the spinal canal of the animal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis
41. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
42. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
43. Which genus of ixodes mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis
44. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, barrel-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
Б.Piroplasma bigeminum
B.Oetrus ovis
Г.Hypoderma lineatum

45. The drugs himkoccid, sulfademizin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
46. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
47. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
48. In which disease the diagnosis is made comprehensively, and in particular for laboratory studies, the punctate is taken primarily from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis
49. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
50. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes an opening?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis

51. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
52. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
53. A widespread animal disease occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Pyroplasmosis
C.Accidental illness
D.Sarcoptosis
54. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
55. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?
A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
56. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz

57. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
58. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the oocysts of the parasite are detected?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
59. In which protozoal disease of pigs, the main pathoanatomic changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
60. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fulebern methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
61. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Leishmaniasis
B.Trichomoniasis
C.Toxoplasmosis
D.Cysticercosis
62. For the prevention of any disease, animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Balantidiosis
D.Psoroptosis

63. Transmissible protozoal disease, occurring acutely and subacutely, the causative agent is a parasite primarily in the RES, after it parasitizes inside the erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
64. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
B.Psoroptosis
G.Trichomoniasis
65. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Pyroplasmosis
C.Estroz
D.Moniesiosis
66. A disease in the treatment of which are used as specific drugs: azidine, bunarvalek, berenyl, diamidine and naganin, together with this should be used restorative drugs?
A.Accidental illness
B.Psoroptosis
C.Rhinestrosis
D.Pyroplasmosis
67. In which parasite (pathogen), after piercing the skin of an animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating into the spinal canal of an animal?
A.Hypoderma bovis
Б.Cysticercus bivis
B.Theileria annulata
Г.Oestrus ovis
68. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Estroz
B.Psoroptosis
C.Melophagosis
D.Demodekoz
69. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?

A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
70. With protozoal disease of dogs, the causative agent of which disease parasitizes inside the red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Piroplasma bigeminum
C.Theileria annulata
D.Eimeria bovis
71. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bovis
72. The drugs himkoccid, sulfadimesin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Siphunculatosis
D.Hypodermatosis
73. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
74. In which disease the diagnosis is made comprehensively, and in particular for laboratory studies, the punctate is taken primarily from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Teileriosis
B.Rayetinosis
C.Trichomoniasis
D.Psoroptosis
75. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz

B.Melophagosis
C.Psoroptosis
D.Demodekoz
76. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
77. Parasitic bird disease, occurring acutely, subacute and chronically, young animals aged 5 to 90 days are sick, the litter is liquid, whitish-green or dark brown with an admixture of blood, in the laboratory for the detection of oocysts of the parasite, litter samples are examined by the Darling method.
A. Eimeriosis
B.Ascariasis
C.Rayetinosis
D.Heterakidosis
78. Widespread animal disease, occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Accidental illness
C.Pyroplasmosis
D.Sarcoptosis
79. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Hypodermatosis
C.Psoroptosis
D.Siphunculatosis
80. The disease of rabbits, the infection of which reaches up to 70-100%, rabbits can become infected from the first days of the day, the disease occurs in cells with crowded rabbits, in a damp room.
A.Eimeriosis
B.Fascioliasis
C.Cystecercosis
D.Passaluroz
81. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis

C. Multiceps multiceps
D. Echinococcus granulosus
82. When diagnosing what protozoal disease of horses, smears are prepared from the peripheral blood of animals and on the basis of this the pathogen is detected?
A.Nuttaliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
83. In which disease are the main pathoanatomic changes observed in the genitals of animals accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis
D. Teileriosis
84. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
A.Trichomoniasis
B. Piroplasmosis
C. Fasciolosis
D. Cysticercosis
85. Which protozoal diseases of cattle are acutely and subacutely occurring and accompanied by fever, hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems?
A.Pyroplasmosis
B. Trichomoniasis
C. Eimeriosis
D. Balantidiosis
86. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is usually used, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?
A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis
87. Which protozoan diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in lymph nodes, fever, anemia, disruption of the cardiovascular and digestive system, exhaustion and a high percentage of mortality?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis

88. The causative agent of what protozoal disease, once in the animal's body with tick saliva, multiplies at the beginning in the lymph nodes and forms macro- and microschizonts (garnet bodies), then macro- and micromerozoites, which are embedded in red blood cells?
A.Teileriosis
B. Benzoithiosis
C. Pyroplasmosis
D. Toxoplasmosis
89. In which disease croup. in cattle, the percentage of infection of erythrocytes with the pathogen is 80-95%?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
90. Which genus of ixodic mites is the carrier of the causative agent of bovine taileriosis?
A.Hyalomma
B. Ixodes
C. Boophylus
D. Haemaphysalis
91. Which genus of ixodes mites is the carrier of the causative agent of bovine babesiosis?
A.Ixodes
B. Hyalomma
C. Boophylus
D. Haemaphysalis
92. Which genus of ixodes mites is the carrier of the causative agent of bovine pyroplasmosis?
A.Boophylus calcaratus
B. Ixodes
C. Hyalomma
D. Haemaphysalis
93. Which genus ixodes mites are the carrier of the pathogen Piroplasmozaovets and goats?
A.Rhipicephalus bursa
B. Ixodes
C. Hyalomma
D. Haemaphysalis
94. When diagnosing which protozoal disease of cattle, smears prepared from lymph nodes are examined at the beginning of the disease, and during the period of clinical signs, blood smears from peripheral vessels to detect the pathogen?
A. Teileriosis
B. Toxoplasmosis
C. Beznoitiosis

D. Pyroplasmosis
95. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists are carefully usedAll-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Piroplasmosis
96. Which genus and species of ixodes mites are carriers of the causative agent of horse nuttaliosis?
A.Ixodes and Boophylus calcaratus
B.Dermacentor and Hyalomma plumbeum
C.Boophylus and Rhipicephalus bursa
D.Haemaphysalis and Ixodes ricinus
97. What protozoal disease of horses is acute, subacute and chronic, accompanied by hemorrhages, jaundice, anemia, the appearance of hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?
A.Nuttaliosis
B.Pyroplasmosis
C.Oxyurosis
D.Paraskaridosis
98. Which pathogen of protozoal disease has an oval, pear-shaped, dot-shaped shape, they are located in erythrocytes by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?
A.Nuttalia equi
B.Piroplasma caballi
C.Theileria annulata
D.Piroplasma bigeminum
99. What arachnous disease of sheep is acute and chronic, characterized by itching of the skin, hair loss and exhaustion of the body, sometimes ends fatally?
A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz
100. In which arachnose disease of sheep are mainly infected with animals with a thick coat, increased humidity in the winter periods of the year?
A.Psoroptosis
B.Hypodermatosis
C.Piroplasmosis
D.Estroz
101. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice

of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
102. What disease of horses is characterized by acute, subacute and chronic course, clinical forms of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?
A.Pyroplasmosis
B.Paraskaridosis
C.Oxyurosis
D.Rhinestrosis
103. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and death in acute form?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
104. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis
C. Hypodermatosis
D.Siphunculator
105. In what invasive disease of cattle does the disease proceed acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
106. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis

107. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
108. In which protozoal disease of pigs, the main pathological changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariosis
C.Trichocephalosis
D. Psoroptosis
109. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
110. For the prevention of any disease of animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
111. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
112. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis

C.Trichomoniasis
D.Psoroptosis
113. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general restorative medicines be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
114. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis
C.Estroz
D.Pyroplasmosis
115. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
116. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
117. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
118. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
119. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis

B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
120. In which parasite (pathogen), after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating the spinal canal of the animal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis
121. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
122. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
123. Which genus of ixodes mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis
124. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, dot-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
B.Piroplasma bigeminum
C.Oetrus ovis
D.Hypoderma lineatum
125. The preparations himkoccid, sulfademizin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis

D.Siphunculatosis
126. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
127. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
128. In which disease is the diagnosis made comprehensively, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis
129. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
130. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
131. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis

D.Melophagosis
132. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
134. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
135. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?
A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
136. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
137. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
138. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the oocysts of the parasite are detected?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator

139. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
140. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fulebern methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
141. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Leishmaniasis
B.Trichomoniasis
C.Toxoplasmosis
D.Cysticercosis
142. For the prevention of any disease, animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Balantidiosis
D.Psoroptosis
143. Transmissible protozoal disease, occurring acutely and subacutely, the causative agent is a parasite primarily in the RES, after it parasitizes inside the erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
144. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Psoroptosis
D.Trichomonosis

145. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Pyroplasmosis
C.Estroz
D.Moniesiosis
146. A disease in the treatment of which specific drugs are used: azidine, bunarvalek, berenyl, diamidine and naganin, together with this, general strengthening drugs should be used?
A.Accidental illness
B.Psoroptosis
C.Rhinestrosis
D.Pyroplasmosis
147. Which parasite (pathogen), after piercing the skin of an animal and introducing it through it, will begin a pathogenic effect for the animal's body, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, when penetrating into the spinal canal, paresis and paralysis of the limbs are observed in the animal?
A.Hypoderma bovis
Б.Cysticercus bivis
В.Theileria annulata
Г.Oestrus ovis}
148. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Estroz
B.Psoroptosis
C.Melophagosis
D.Demodekoz
149. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estroz
C.Balantidiaz
D.Eimerioz
150. With protozoal disease of dogs, the causative agent of which disease parasitizes inside the red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Piroplasma bigeminum
C.Theileria annulata
D.Eimeria bovis
151. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?

A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bovis
152. The drugs himkoccid, sulfadimesin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Siphunculatosis
D.Hypodermatosis
153. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
154. In which disease is the diagnosis made comprehensively, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Teileriosis
B.Rayetinosis
C.Trichomoniasis
D.Psoroptosis
155. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estrosis
B.Melophagosis
C.Psoroptosis
D.Demodekoz
156. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
157. Parasitic disease of birds, occurring acutely, subacute and chronically, young animals aged 5 to 90 days are sick, the litter is liquid, whitish-green or dark brown with an admixture of blood, in the laboratory for the detection of oocysts of the parasite, litter samples are examined by the Darling method.

A. Eimeriosis
B.Ascariasis
C.Rayetinosis
D.Heterakidosis
158. A widespread animal disease occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Accidental illness
C.Pyroplasmosis
D.Sarcoptosis
159. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Hypodermatosis
C.Psoroptosis
D.Siphunculatosis
160. The disease of rabbits, the infection of which reaches up to 70-100%, rabbits can become infected from the first days of the day, the disease occurs in cells with crowded rabbits, in a damp room.
A.Eimeriosis
B.Fascioliasis
C.Cystecercosis
D.Passaluroz
161. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis
C. Multiceps multiceps
D. Echinococcus granulosus
162. When diagnosing what protozoal disease of horses, smears are prepared from the peripheral blood of animals and on the basis of this the pathogen is detected?
A.Nuttaliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
163. In which disease are the main pathoanatomic changes observed in the genitals of animals accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis

D. Teileriosis
164. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
A.Trichomoniasis
B. Piroplasmosis
C. Fasciolosis
D. Cysticercosis
165. Which protozoal diseases of cattle are acutely and subacutely occurring and accompanied by fever, hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems?
A.Pyroplasmosis
B. Trichomoniasis
C. Eimeriosis
D. Balantidiosis
166. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is usually used, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?
A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis
167. Which protozoan diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in lymph nodes, fever, anemia, disruption of the cardiovascular and digestive system, exhaustion and a high percentage of mortality?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
168. The causative agent of what protozoal disease, once in the animal's body with tick saliva, multiplies at the beginning in the lymph nodes and forms macro- and microschizonts (garnet bodies), then macro- and micromerozoites, which are embedded in red blood cells?
A.Teileriosis
B. Benzoithiosis
C. Pyroplasmosis
D. Toxoplasmosis
169. In which disease croup. in cattle, the percentage of infection of erythrocytes with the pathogen is 80-95%?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis

170. Which genus of ixodes mites is the carrier of the causative agent of bovine taileriosis?
A.Hyalomma
B. Ixodes
C. Boophylus
D. Haemaphysalis
171. Which genus of ixodes mites is the carrier of the causative agent of bovine babesiosis?
A.Ixodes
B. Hyalomma
C. Boophylus
D. Haemaphysalis
172. Which genus of ixodes mites is the carrier of the causative agent of bovine pyroplasmosis?
A.Boophylus calcaratus
B. Ixodes
C. Hyalomma
D. Haemaphysalis
173. Which genus of ixodes mites is the carrier of the causative agent of piroplasmosis of sheep and goats?
A.Rhipicephalus bursa
B. Ixodes
C. Hyalomma
D. Haemaphysalis
174. When diagnosing which protozoal disease of cattle, smears prepared from lymph nodes are examined at the beginning of the disease, and during the period of clinical signs, blood smears from peripheral vessels to detect the pathogen?
A. Teileriosis
B. Toxoplasmosis
C. Beznoitiosis
D. Pyroplasmosis
175. In the treatment of what protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists are carefully usedIn the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists of All-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute?
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Piroplasmosis
176. Which genus and species of ixodes mites are carriers of the causative agent of horse nuttaliosis?

А.Ixodes and Boophylus calcaratus

Б.Dermacentor and Hyalomma plumbeum

В.Boophylus and Rhipicephalus bursa

Г.Haemaphysalis and Ixodes ricinus

177. What protozoal disease of horses is acute, subacute and chronic, accompanied by hemorrhages, jaundice, anemia, the appearance of hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?

A.Nuttaliosis

B.Pyroplasmosis

C.Oxyurosis

D.Paraskaridosis

178. Which pathogen of protozoal disease has an oval, pear-shaped, dot-shaped shape, they are located in erythrocytes by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?

A.Nuttalia equi

B.Piroplasma caballi

C.Theileria annulata

D.Piroplasma bigeminum

179. What arachnous disease of sheep is acute and chronic, characterized by itching of the skin, hair loss and exhaustion of the body, sometimes ends fatally?

A.Psoroptosis

B.Hypodermatosis

C.Pyroplasmosis

D.Estroz

180. At what arachnose disease of sheep are animals with a thick coat of wool, increased humidity in the winter periods of the year mainly infected?

A.Psoroptosis

B.Hypodermatosis

C.Piroplasmosis

D.Estroz

181. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?

A.Pyroplasmosis

B.Toxocarosis

C.Teniidosis

D.Demodekoz

182. What disease of horses is characterized by acute, subacute and chronic course, clinical forms of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?

A.Pyroplasmosis

B.Paraskaridosis

C.Oxyurosis

D.Rhinestrosis
183. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and death in acute form?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
184. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis
C. Hypodermatosis
D.Siphunculator
185. In which invasive disease of cattle does the disease proceed acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
186. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
187. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
188. In which protozoal disease of pigs, the main pathoanatomic changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariosis

C.Trichocephalosis
D. Psoroptosis
189. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
190. For the prevention of which disease animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
191. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
192. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
193. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general restorative medicines be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
194. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis

C.Estroz
D.Pyroplasmosis
195. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
196. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
197. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
198. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
199. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
200. In which parasite (pathogen), after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating the spinal canal of the animal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis

4.9 FINAL CONTROL

FINAL TEST QUESTIONS

1. What is the relationship of organisms in which both sides benefit from such cohabitation?
A.Mutualism
B. Sinoikia
C.Parasitism
D. Predation
2. What is the relationship of organisms, in which each of them is completely independent of the others, but at the same time is closely connected with the life of the whole collective as a whole?
A.Indifferent
B. Symbiotic
C.Mutualistic
D. Commensalistic
3. What is the relationship of organisms, in which one organism benefits, and the other neither benefits nor harms?
A.Sinoikia
B.Mutualism
C.Parasitism
D.Predation
4. What is the name of the relationship between two organisms, in which one of the organisms feeds on the remains of the other organism's food without harming it?
A.Commensalism
B.Mutualism
C.Sinoikia
D.Parasitism
5. What is the relationship between two organisms, in which one organism is stronger than its prey, which it immediately kills and eats?
A.Predation
B.Parasitism
C.Mutualism
D.Kommensalism
6. What is the name of the relationship between two organisms, in which one organism, as a result of evolutionary development, has adapted to its hosts as much as possible and only harms it?
A. Parasitism
B.Commensalism
C.Mutualism
D.Sinoikia
7. In the diagnosis of which disease, animal feces are examined and when three, quadrangular eggs are found, the size of which varies between 0.05-0.09 mm?
A.Moniesiosis
B.Cysticercosis
C.Echinococcosis

D.Cenuroz
8. When diagnosing which nematode disease in birds, feces is examined by the Fulleborn or Darling method and when eggs are found, oval in shape, 0.070-0.086 mm thick, 0.047-0.051 mm wide?
A.Ascariasis
B.Davenioz
C.Prostogonimosis
D.Knemidocoptosis
9. When diagnosing which nematode disease of sheep, fecal samples should be examined by the Berman-Orlov method for the detection of parasite larvae?
A.Dictyoculosis
B.Fascioliasis
C.Psoroptosis
D.Estroz
10. What kind of trematode is a leaf-shaped, 2-3 cm long, 09.8-1.2 cm wide, and some have a ribbon-shaped, 4-7.5 cm long, and 0.6-1.2 cm wide?
A.Fasciola hepatica, F.gigantica
B.Paramphistomum cervi
C.Dicrocoelium lanceatum
D.Euritrema pancreaticum
11. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, and a freshwater mollusk from the genus Lymnea as an intermediate?
A.Fascioles
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
12. What kind of disease occurs if animals graze in spring and autumn in low-lying pastures where freshwater mollusks from the genus Lymnea are widespread?
A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Prostogonimoz
13. In which disease is the pathogenic effect of the parasite caused by mechanical damage to the liver parenchyma, inoculation of chronic infection and intoxication of the body by the products of the parasite?
A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
14. What kind of disease is acute and chronic, with an acute course there is pallor of the mucous membranes, an increase in body temperature, impaired digestive function, diarrhea, and with a chronic one – a depressed state, standing from the herd, the appearance of edema, severe emaciation and death?

A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
15. In what disease are the corpses of fallen animals exhausted, fluid with an admixture of blood has accumulated in the abdominal cavity, lymph nodes and liver are enlarged, bile ducts are dilated, the gallbladder is overflowing with thick bile, and parasites are found in them?
A.Fasciolesis
B.Dicroceliosis
C.Orientobilkhartsioz
D.Paramphistomatosis
16. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of golden yellow eggs in faeces, oval shape, size 0.13-0.15 x0.07-0.09 mm?
A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
17. In which disease of animals are dewormed with one of the following drugs: hexachlorparaxylene, hexychol, 4-x carbon chloride, ursovermid, filixan, rolenol, closantel, combitrem, facocide?
A.Fasciolesis
B.Orientobilcharciosis
C.Dicroceliosis
D.Paramphistomatosis
18. For the prevention of what disease of animals should be carried out planned deworming, biothermic treatment of manure and destruction of intermediate hosts by mechanical, physical, chemical and biological means?
A.Fasciolesis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Dicroceliosis
19. Which disease is a widespread fluke disease in more than 70 species of domestic and wild mammalian animals caused by flukes parasitizing in the bile ducts and gallbladder?
A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
20. What disease is widespread everywhere, but its widespread distribution is noted in the desert-pasture zone, where there are intermediate and additional hosts (land mollusks and ants)?
A.Dicroceliosis

B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
21. In which disease are the main pathoanatomic changes observed in the abomasum and duodenum 12, the mucous membrane of which is catarrhally hemorrhagically inflamed, mesenteric lymph nodes enlarged?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
22. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark gray eggs in the faeces, oval shape, size 0.12-0.17x0.07-0.1 mm, yolk cells are not completely filled with egg cavities?
A.Paramphistomatosis
B.Dicroceliosis
C.Fascioliasis
D.Orientobilkhartsioz
23. In which disease animals are dewormed with one of the following drugs: 4-carbon chloride, bithionol, resorantel, gilomite and kerosene, which is used in aviation?
A. Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D. Fasciolosis
24. To prevent what trematodous disease of animals, planned deworming should be carried out, and in case of an outbreak of invasion, 3-4 weeks after pasture, preimaninal deworming of young animals is carried out twice at intervals of 10 days, biothermic treatment of manure and destruction of intermediate hosts?
A.Paramphistomatosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
25. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, a land mollusk from the genus Eulotalantzi as an intermediate, and grasshoppers from the genus Conocephalus and crickets – Oecanthus as an additional host?

B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
26. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark brown eggs in feces, oval shape, size 0,044-13-0 ,048x0,032-0,0036 mm?

A.Eurythrematosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
27. Which trematode disease is chronic with pronounced clinical signs of pancreatitis?
A.Eurythrematosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
28. In which disease animals are dewormed with one of the following drugs: bithionol, hexychol, hexychol C, tafen, panacur (fenbendazole)?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
29. In which disease are the main pathoanatomic changes observed in the ducts of the pancreas, the glandular part of the liver parenchyma atrophied, mesenteric lymph nodes enlarged?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
30. What fluke disease of poultry occurs chronically with pronounced 3-stage clinical signs (thinned eggshell, egg wash and duck gait?
A.Prostogonimosis
B.Ascariasis
C.Heterakidosis
D.Davenioz
31. What is the disease occurring chronically, in which a depressed state, an increase in body temperature, impaired digestive function, panoses, the appearance of edema, severe emaciation and death?
A.Orientobilcharciosis
B.Dicroceliosis
C.Paramphistomatosis
D.Fasciolosis
32. Which of the non-following trematodes is the radon-winged parasites, the size of males exceeds the size of females, males have 78-80 testes, and the female has one egg in the uterus, inside of which there are formed miracidia?
A.Orientobilchartsii
B.Dicrocelium
C.Paramphistomats
D. Fascioles

33. In the diagnosis of which disease, the parasite's eggs are first cultured in a thermostat, after which the Berman-Orlov method examines for the detection of parasite larvae?
A.Orientobilcharciosis
B. Paramphistomatosis
C. Dicroceliosis
D. Fasciolosis
34. In which disease animals are dewormed with one of the following drugs: fuadin at a dose of 0.3 ml / kg, albilgar at a dose of 0.02-sh,03 g / kg, droncite at a dose of 0.05 g / kg, azinox at a dose of 25 mg / kg of animal weight?
A.Orientobilcharciosis
B. Paramphistomatosis
C.Dicroceliosis
D.
Fascioliosis 35. To prevent what chronically occurring trematode disease of animals should be carried out planned deworming, biothermic treatment of manure, destruction of intermediate hosts, which are ear-shaped mollusks of the genus Lymnea and feeding animals according to the diet?
A.Orientobilcharciosis
B.Paramphistomatosis
C.Fascioliasis
D.Dicroceliosis
36. What is the name of the disease of cattle caused by cestodes, parasitizing in the intermuscular connective tissue, skeletal muscles, heart, tongue?
A.Cysticercosis
B. Echinococcosis
C. Cenurosis
D. Monesiosis
37. In which disease of cattle, the degree of infection is on average 10% and about 1% of meat, forgotten animals are disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
38. What kind of cestode does the larval (vesicular) stage of which has a rounded-oval shape, consisting of three shells, the length of the bubble is 5-15 mm, with a width of 3-8 mm, there is one unarmed scolex inside the bubble?
A.Cysticercus bovis
B. Cysticercus cellulosae
C. Coenurus cerebralis
D.Echinococcus granulosus
39. What kind of cestode does the larval (vesicular) stage of which has an ellipsoid shape, consisting of three shells, the length of the bubble is 10-20 mm, with a width of 5-10 mm, there is one armed scolex inside the bubble?

A.Cysticercus cellulosae
B. Cysticercus bovis
C. Coenurus cerebralis
D. Echinococcus granulosus
40. What kind of cestode does it belong to, having a ribbon-like shape, a length of more than 10 m, a width of 12-14 mm, whose strobila consists of dozens, hundreds and even thousands of segments?
A.Taeniarynchus saginatus
B.Taenia solium
C.Multiceps multiceps
D.Echinococcus granulosus
41. Which cestode in its biological development cycle is used as a definitive host of humans, and as an intermediate host – cattle, buffaloes, yak, zebu and sometimes reindeer?
A.Cysticercus bovis
B.Cysticercus cellulosae
C.Coenurus cerebralis
D.Echinococcus granulosus
42. Which cestode develops with the participation of the main one, which is only human, and intermediate hosts, which are pigs, wild boars, hares, rabbits, bears, camels as well as humans?
A.Cysticercus cellulosae
B. Cysticercus bovis
C.Coenurus cerebralis
D.Echinococcus granulosus
43. What kind of cestodosis disease will be widespread if the livestock farm does not comply with zoohygienic requirements, the absence of a closed toilet and sanitary and educational work is not carried out among the general population, especially stockmen?
A.Cysticercosis of cattle
B.Echinococcosis
C.Monesiosis
D.Cenuroz
44. When preventing what kind of cestodose disease, it is necessary to comply with the rules of veterinary sanitation, animals should be slaughtered in special slaughterhouses and be sure to carry out a vet.san.examination of meat and meat products of slaughtered animals?
A.Cysticercosis of cattle
B. Echinococcosis
C.Monesiosis
D.Cenuroz
45. In which disease should the VSE be carried out meat and offal, specially forgotten for meat or forcibly forgotten animals, and when parasites are detected on

an area of 40 cm2 of the muscles of the head and heart and on one incision of the muscles of the carcass, the whole carcass is disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
46. Which pathogen of anthropozoonous, cestodose, asymptomatic mammalian animal disease is parasitized in parenchymal organs, mainly in the liver and lungs?
A.Echinococcosis
B.Cysticercosis
C.Cenuroz
D.Moniesiosis
47. Which cestode develops with the participation of the main one, which are dogs and other carnivores, and intermediate hosts, which are mammalian animals?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
48. To prevent which chronically occurring cestodosis disease of animals should be carried out planned deworming, biothermic treatment of manure of the main hosts, disinfection of infected parenchymal organs of intermediate hosts?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
49. What kind of cestode does it belong to, having a ribbon-shaped, 40-100 cm long, 5-6 mm wide, the strobila of which consists of hundreds of segments, in one mature segment there are up to 50 thousand eggs?
A. Multiceps multiceps
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
50. How many parts does the body of the cestode consist of?
A.of three
B.of two
C.of five
D.the body of the cestode is not divided into parts
51. Which part of the body of the cestode is the growth zone?
A.neck
B.head (scolex)
C.hermafortid segment
D.mature segment
52. Where are the hermaphordite segments of the cestode?
A. in the anterior part of the body

B. the middle part of the body
C. the back part of the body
D.About all parts of the body
53. In the posterior part of the body of the cestode, which genitals are preserved?
A.uterus
B.ovary
C.testis
D.ovary
54. Cestodes develop with the participation of how many hosts?
A.2 and 3 hosts
B.two hosts
C.three hosts
D.one host
55. Which cestode's scolexes are armed with suckers and chitinized hooks?
A.Echinococcus and multiceps
B.moniezium and avitellins
C.tizaniezium and multiceps
D.stylezium and echinococcus
56. In skoleks, what kind of cestodes do not have chitinized hooks?
A.monieziy
B.multiceps
C.echinococcus
D.teniarinhus
57. Uterus which cestode is open type, develop with the participation of three hosts, larvae in the form of worms?
A.Diphyllobothrium latum
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
58. Uterus which cestode is a closed type, develop with the participation of two hosts, larvae in the form of a bubble that is filled with fluids?
A.Taeniarynchus saginatus
B.Ligula intestinalis
C.Moniezia expansa
D. Diphyllobothrium latum
59. The form of which trematodes in the form of threads and is bisexual?
A. Orientobilchartsii
B.Dicrocelium
C.Paramphistomatids
D. Fascioles
60. The development cycle of the causative agent of which trematode disease occurs only in endogenous conditions?
A.Dicrocelium
B. Orientobilcharcium

C.Paramphistomatids
D.Fascioles}
61. What trematodous diseases are also found in humans?
A.Fascioliasis, dicroceliosis
B.Paramphistomatosis and dicrocelium
C.Orientobilcharcosis and paramphistomatosis
D.Fascioles and echinococcosis
62. In vertebrates, what types of trematodous diseases are found?
A.in all vertebrates
B.in fish
C.in birds
D.in the experiencing
63. Which method is acceptable to combat trematodous diseases?
A.Devastation
B.Deworming
C.Disinvasion
D.Fighting shellfish
64. Why do the ribbon forms of cestodes live in the small intestine?
A. They do not have a digestive system
B.Their bodies are very long
C. They have scolexes (head)
D.They have a poorly developed neural system
65. In what kind of trematode disease is percutaneous infection of animals observed?
A.In orientobilcharciosis
B.With fascioliasis
C.With paramphistomatosis
D.With eurythrematosis
66. Fasciolahepatica in how many days does it reach sexual maturity in the body of a definitive host?
A.75-80 days
B.80-90 days
C.80-130 days
D.90-150 days
67. Fasciolagigantica in how many days does it reach sexual maturity in the body of a definitive host?
A.90-120 days
B. 80-90 days
C.80-130 days
D.75-80 days
68. In which zones is fascioliasis most common?
A. in the irrigation and foothill-mountain zones
B. in the foothill zone
C.in the desert pasture zone
D.in all zones

69. How is the diagnosis made in case of vital fascioliasis?
A.by the method of sequential washing of feces
B. Berman-Orlov method
C.by the Vidamethod
D. by the Fulleborn method
70. Where does the parthenogenetic cycle of fasciole development occur?
A.in the body of an intermediate host- a mollusk
B. in the body of a definitive host
C.in water
D.in the body of a parasite
71. Which trematode disease is acute, chronic and mixed forms?
A.Fascioliasis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
72. What does the term "Devastation" mean?
A.destruction of parasites, helminths in all stages of development with all methods of control
B.liberation of the animal organism from parasites
C.destruction of intermediate hosts of helminths
D.destruction of invasive larvae of helminths
73. Who is the author of the term "Devastation"?
A.K.I.Scriabin
B. N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
74. Show the scientist who for the first time thoroughly studied orientobil-khartsioz in the conditions of our republic?
A.D.A.Azimov
B. N.V.Badanin
C. B.S.Salimov
D. V.S. Ershov
75. Who is the founder of helminthological science in Uzbekistan?
A.N.V.Badanin
B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
76. The main (definitive) host of which anthropozoonous disease is a person?
A.Cysticercosis of cattle
B. Teniosis
C. Echinococcosis
D. Cenurosis
77. Who is the author of the albendozol-copper vitriol salt mixture?
A.A.O.Oripov

B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
78. Which of the following scientists has thoroughly studied the biology of the causative agent of dicroceliosis?
A.B.S. Salimov
B. D.A.Azimov
C. A.O.Oripov
D. V.S. Ershov
79. Which of the following scientists is the first doctor of Sciences in the specialty "Helminthology"?
A.I.H.Irgashev
B. D.A.Asimov
C.A.O.Oripov
D. B.S. Salimov
80. Which cestdosis disease is mainly widespread among 1.5-8-month-old lambs, goats and calves?
A.shell mites of the genus Scheloribates
B. freshwater mollusks
C. land mollusks
D. ants
81. What is the intermediate host of the causative agent of moniesiosis?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
82. In the treatment of which disease is it recommended to use phenasal, phenalidone, 1-2% aqueous solution of copper sulfate, panacur, rintal and drugs from the albendazole group?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
83. For the prevention of what disease should animals be fed salt-phenothiazine-copper sulfate top dressing, the composition of which consists of 1% copper sulfate, 10% phenothiazine and 89% table salt, from October to June from a calculation of 1.0 per head?
A.Moniesiosis
B. Echinococcosis
C. Cenurosis
D. Cysticercosis
84. The sexually mature form of the causative agent of which nematode disease of ungulates parasitizes in the small intestine, and the larvae migrate hepatopulmonally?
A.Parascaris equorum

B. Paranaplocephala mamillana
C. Anaplocephala magna
D. Anaplocephala perfoliata
85. In the treatment of which nematode disease of single-hoofed animals are used piperazine salts, phenothiazine, panacur (fenbendazole), rintal (febantel), 4-x carbon chloride, albendazole and its analogues?
A.Paraskaridosis
B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
86. To prevent which nematode disease of ungulates should preventive deworming of suckling foals be carried out for the first time in August, the second time after weaning, and adult animals for the first time in March-April, the second time in October-November?
A.Paraskaridosis
B. Anaplocephalosis
B. Piroplasmosis
D. Nuttaliosis
87. Which nematode disease of sheep proceeds chronically, the causative agent of which parasitizes in the bronchi and trachea, as a result of which the infected animal has a violation of the function of the respiratory organs?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
88. Which nematode is large, filamentous, milky-white, bisexual, males 3-8 cm long, females 5-15 cm, spicule dark brown, boot-shaped, 0.4-0.6 mm long?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
89. Which nematode is a geohelminth, in the external environment the larva reaches the invasive stage in 6-7 days, the prepatent period of development is 3-4 months, the patent period is 1.5-2 years?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
90. With which nematode disease of sheep there is a weak, then increasing cough during movement, after the previous rest, at night, bronchitis, serous-purulent discharge from the nose, which dry out and form crusts?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis

D. Estrosis
91. When deworming which nematode disease use 1 or 2% solutions of copper sulfate, phenothiazine, naphthamone, panacur (fenbendazole), rintal (febantel), albendazole and its analogues?
A.Hemonchosis
B. Fasciolosis
C. Paramphistomatosis
D. Dicroceliosis
92. In which nematode disease do chickens of 2-10 months of age mainly become infected, and adult birds are parasitic carriers?
A.Ascariasis
B. Prostogonimosis
C. Daveniosis
D. Rayetinoz
93. In which nematode disease, the causative agent of which is parasitized in the conjunctival sac, under the third eyelid, in the ducts of the lacrimal gland and the lacrimal-nasal canal, as a result of which animals are observed lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids turning into keratitis, ulcers on the cornea, a thorn?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
94. In which helminthic disease are the characteristic clinical signs of lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids, turning into keratitis, ulcers of the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
95. In the treatment of which nematode is used 3% boric acid, Lugol solution, ditrazine citrate, iodine, lysol and others?
A.Telyaziosis
B. Setariosis
C. Onchocerciasis
D. Teileriosis
96. What chronically occurring disease of horses is characterized by skin lesions at the root of the tail, severe itching, eczema, dermatitis and severe emaciation of the animal?
A.Oxyurosis
B. Paraskaridosis
C. Anaplocephalosis
D. Pyroplasmosis

97. Which nematode of ungulates develops in a direct way, that is, without the participation of an intermediate host, the prepatent period of development is equal to 3-4 weeks, and the patent period is 6-8 months?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis
98. In which nematode disease of horses is sick mainly young animals up to one year old and old horses?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis}
99. When deworming which disease is used 4-chlorinated carbon, piperazine and its salts, phenothiazine, panacur (fenbendazole), rintal (febantel), tetramizole and mebenvet in the form of granules?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Anoplocephalosis
100. For the prevention of which disease should horses undergo planned dehelminthization every 3 months, biothermic treatment of manure and full feeding of animals according to the diet?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Nuttaliosis
101. In which nematode disease of birds, the pathogenic effect of the pathogen consists of mechanical damage to the mucous membrane and glands of the intestine, toxic effects and opens the gates to secondary infections?
A.Ascariasis
B. Knemidocoptosis
C. Histomonosis
D. Borreliosis
102. In the treatment of which nematode disease of birds piperazine and its salts, phenothiazine, nilverm, benacil, fenbendazole (panacur), rintal (febantel) are used?
A.Ascariosis
B. Prostogonimosis
C. Daveniosis
D. Histomonosis
103. What is the relationship between organisms, in which one organism benefits, and the other neither benefits nor harms?
A.Sinoikia
B.Mutualism

C.Parasitism
D.Predation
104. What is the name of the relationship between two organisms, in which one of the organisms feeds on the remains of the other organism's food without harming it?
A.Commensalism
B.Mutualism
C.Sinoikia
D.Parasitism
105. What is the name of the relationship between two organisms, in which one organism is stronger than its prey, which it immediately kills and eats?
A.Predation
B.Parasitism
C.Mutualism
D.Kommensalism
106. What is the name of the relationship between two organisms, in which one organism, as a result of evolutionary development, has adapted to its hosts as much as possible and only harms it?
A.Parasitism
B.Commensalism
C.Mutualism
D.Sinoikia
107.In the diagnosis of which disease, animal feces are examined and when three, quadrangular eggs are found, the size of which varies between 0.05-0.09 mm?
A.Moniesiosis
B.Cysticercosis
C.Echinococcosis
D.Cenuroz
108.In the diagnosis of which nematode disease in birds examines feces by the Fulleborn or Darling method and when eggs are found, oval in shape, 0.070-0.086 mm thick, 0.047-0.051 mm wide?
A.Ascariasis
B.Davenioz
C.Prostogonimosis
D.Knemidocoptosis
109.When diagnosing which nematode disease of sheep, fecal samples should be examined by the Berman-Orlov method for the detection of parasite larvae?
A.Dictyocaulesis
B.Fascioliasis
C.Psoroptosis
D.Estroz
110.What kind of trematode is a leaf-shaped, 2-3 cm long, 09.8-1.2 cm wide, and some have a ribbon-shaped, 4-7.5 cm long, and 0.6-1.2 cm wide?
A.Fasciola hepatica, F.gigantica
B.Paramphistomum cervi

C.Dicrocoelium lanceatum
D.Euritrema pancreaticum
111.Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, and a freshwater mollusk from the genus Lymnea as an intermediate?
A.Fascioles
B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
112. What kind of disease occurs if animals graze in spring and autumn in low-lying pastures where freshwater mollusks from the genus Lymnea are widespread?
A.Fasciolosis
B.Dicroceliosis
C.Paramphistomatosis
D.Prostogonimoz
113.In which disease is the pathogenic effect of the parasite caused by mechanical damage to the liver parenchyma, inoculation of chronic infection and intoxication of the body by the products of the parasite?
A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
114. What kind of disease is acute and chronic, with an acute course there is pallor of the mucous membranes, an increase in body temperature, impaired digestive function, diarrhea, and with a chronic one – a depressed state, standing from the herd, the appearance of edema, severe emaciation and death?
A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
115. In what disease are the corpses of fallen animals exhausted, fluid with an admixture of blood has accumulated in the abdominal cavity, lymph nodes and liver are enlarged, bile ducts are dilated, the gallbladder is overflowing with thick bile, and parasites are found in them?
A.Fasciolesis
B.Dicroceliosis
C.Orientobilkhartsioz
D.Paramphistomatosis
116.In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of golden yellow eggs in faeces, oval shape, size 0.13-0.15x0.07-0.09 mm?
A.Fasciolesis
B.Dicroceliosis
C.Paramphistomatosis

D.Orientobilkhartsioz
117.In which disease of animals are dewormed with one of the following drugs: hexachlorparaxylene, hexychol, 4-x carbon chloride, ursovermid, filixan, rolenol, closantel, combitrem, facocide?
A.Fasciolesis
B.Orientobilcharciosis
C.Dicroceliosis
D.Paramphistomatosis
118. For the prevention of what disease of animals should be carried out planned deworming, biothermic treatment of manure and destruction of intermediate hosts by mechanical, physical, chemical and biological means?
A.Fasciolesis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Dicroceliosis
119. Which disease is a widespread fluke disease in more than 70 species of domestic and wild mammals caused by flukes, parasitizing in the bile ducts and gallbladder?
A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
120.What disease is widespread everywhere, but its widespread distribution is noted in the desert-pasture zone, where there are intermediate and additional hosts (land mollusks and ants)?
A.Dicroceliosis
B.Orientobilkhartsioz
C.Paramphistomatosis
D.Fasciolosis
121.In which disease are the main pathoanatomic changes observed in the abomasum and duodenum 12, the mucous membrane of which is catarrhally hemorrhagically inflamed, mesenteric lymph nodes enlarged?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
122.In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark gray, oval-shaped eggs in feces, 0.12-0.17x0.07-0.1 mm in size, yolk cells are not completely filled with egg cavities?
A. Paramphistomatosis
B.Dicroceliosis
C.Fascioliasis
D.Orientobilkhartsioz

123.In which disease animals are dewormed with one of the following drugs: 4-carbon chloride, bithionol, resorantel, gilomite and kerosene, which is used in aviation?
A.Paramphistomatosis
B.Orientobilcharciosis
C.Dicroceliosis
D. Fasciolosis
124. In order to prevent what trematodous disease of animals, planned deworming should be carried out, and in case of an outbreak of invasion, 3-4 weeks after pasture, preimaninal deworming of young animals is carried out twice at intervals of 10 days, biothermic treatment of manure and destruction of intermediate hosts?
A.Paramphistomatosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
125. Which of the following parasites in their biological development use domestic and wild mammalian animals as a definitive host, a land mollusk from the genus Eulotalantzi as an intermediate, and grasshoppers from the genus Conocephalus and crickets – Oecanthus as an additional host?

B.Dicrocelia
C.Prostogonyms
D.Paramphistomas
126. In which disease is the diagnosis made on the basis of epizootological data, clinical signs, pathoanatomic changes and the detection of dark brown eggs in the faeces, oval shape, size 0,044-13-0 ,048x0,032-0,0036 mm?
A.Eurythrematosis
B.Dicroceliosis
C.Paramphistomatosis
D.Orientobilkhartsioz
127.What is a trematodous disease that occurs chronically with pronounced clinical signs of pancreatitis?
A.Eurythrematosis
B.Orientobilkhartsioz
C.Fascioliasis
D.Dicroceliosis
128. In which disease animals are dewormed with one of the following drugs: bithionol, hexychol, hexychol C, tafen, panacur (fenbendazole)?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis

129.In which disease are the main pathoanatomic changes observed in the ducts of the pancreas, the glandular part of the liver parenchyma atrophied, mesenteric lymph nodes enlarged?
A.Eurythrematosis
B.Orientobilcharciosis
C.Dicroceliosis
D.Fasciolosis
130.What is the trematode disease of poultry that proceeds chronically with pronounced 3-stage clinical signs (thinned eggshell, egg wash and duck gait?
A.Prostogonimosis
B.Ascariasis
C.Heterakidosis
D.Davenioz
131.What is the disease occurring chronically, in which a depressed state, an increase in body temperature, impaired digestive function, panoses, the appearance of edema, severe emaciation and death?
A.Orientobilcharciosis
B.Dicroceliosis
C.Paramphistomatosis
D.Fasciolosis
132. Which of the non-following trematodes is the radon-winged parasites, the size of males exceeds the size of females, males have 78-80 testes, and the female has one egg in the uterus, inside of which there are formed miracidia?
A.Orientobilchartsii
B.Dicrocelium
C.Paramphistomats
D. Fascioles
133. In the diagnosis of which disease, the parasite's eggs are first cultured in a thermostat, after which the Berman-Orlov method examines for the detection of parasite larvae?
A.Orientobilcharciosis
B. Paramphistomatosis
C. Dicroceliosis
D. Fasciolosis
134. In which disease animals are dewormed with one of the following drugs: fuadin at a dose of 0.3 ml / kg, albilgar at a dose of 0.02-sh,03 g / kg, droncite at a dose of 0.05 g / kg, azinox at a dose of 25 mg / kg of animal weight?
A.Orientobilcharciosis
B. Paramphistomatosis
C.Dicroceliosis
D.
Fascioliosis 135. To prevent what chronically occurring trematode disease of animals should be carried out planned deworming, biothermic treatment of manure,

destruction of intermediate hosts, which are ear-shaped mollusks of the genus Lymnea and feeding animals according to the diet?
A.Orientobilharciosis
B.Paramphistomatosis
C.Fascioliasis
D.Dicroceliosis
136. What is the name of the disease of cattle caused by cestodes, parasitizing in the intermuscular connective tissue, skeletal muscles, heart, tongue?
A.Cysticercosis
B. Echinococcosis
C. Cenurosis
D. Monesiosis
137.In which disease of cattle, the degree of infection is on average 10% and about 1% of meat, forgotten animals are disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
138. What kind of cestode does the larval (vesicular) stage of which has a rounded-oval shape, consisting of three shells, the length of the bubble is 5-15 mm, with a width of 3-8 mm, there is one unarmed scolex inside the bubble?
A.Cysticercus bovis
B. Cysticercus cellulosae
C. Coenurus cerebralis
D.Echinococcus granulosus
139. What kind of cestode does the larval (vesicular) stage of which has an ellipsoid shape, consisting of three shells, the length of the bubble is 10-20 mm, with a width of 5-10 mm, there is one armed scolex inside the bubble?
A.Cysticercus cellulosae
B. Cysticercus bovis
C. Coenurus cerebralis
D. Echinococcus granulosus
140. What kind of cestode does it belong to, having a ribbon-like shape, a length of more than 10 m, a width of 12-14 mm, whose strobila consists of dozens, hundreds and even thousands of segments?
A.Taeniarynchus saginatus
B.Taenia solium
C.Multiceps multiceps
D.Echinococcus granulosus
141. Which cestode in its biological development cycle is used as a definitive host of humans, and as an intermediate host – cattle, buffaloes, yak, zebu and sometimes reindeer?
A.Cysticercus bovis
B.Cysticercus cellulosae

C.Coenurus cerebralis
D.Echinococcus granulosus
142. Which cestode develops with the participation of the main one, which is only human, and intermediate hosts, which are pigs, wild boars, hares, rabbits, bears, camels as well as humans?
A.Cysticercus cellulosae
B. Cysticercus bovis
C.Coenurus cerebralis
D.Echinococcus granulosus
143. What kind of cestodosis disease will be widespread if the livestock farm does not comply with zoohygienic requirements, the absence of a closed toilet and sanitary and educational work is not carried out among the general population, especially cattle breeders?
A.Cysticercosis of cattle
B.Echinococcosis
C.Monesiosis
D.Cenuroz
144. When preventing what kind of cestodose disease, it is necessary to comply with the rules of veterinary sanitation, animals should be slaughtered in special slaughterhouses and it is mandatory to carry out a vet.san.examination of meat and meat products of slaughtered animals?
A.Cysticercosis of cattle
B. Echinococcosis
C.Monesiosis
D.Cenuroz
145. In which disease should the VSE be carried out of meat and offal, specially forgotten for meat or forcibly forgotten animals, and when parasites are detected on an area of 40 cm2 of the muscles of the head and heart and on one incision of the muscles of the carcass, the entire carcass is disposed of?
A.Cysticercosis
B.Echinococcosis
C.Cenurosis
D.Monesiosis
146. Which pathogen of anthropozoonous, cestodose, asymptomatic mammalian animal disease is parasitized in parenchymal organs, mainly in the liver and lungs?
A.Echinococcosis
B.Cysticercosis
C.Cenuroz
D.Moniesiosis
147. Which cestode develops with the participation of the main one, which are dogs and other carnivores, and intermediate hosts, which are mammalian animals?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis

D.Cysticercus cellulosae
148. To prevent which chronically occurring cestodosis disease of animals should be carried out planned deworming, biothermic treatment of manure of the main hosts, disinfection of infected parenchymal organs of intermediate hosts?
A.Echinococcus granulosus
B.Cysticercus bovis
C.Coenurus cerebralis
D.Cysticercus cellulosae
149. What kind of cestode does it belong to, having a ribbon-shaped, 40-100 cm long, 5-6 mm wide, whose strobila consists of hundreds of segments, in one mature segment there are up to 50 thousand eggs?
A. Multiceps multiceps
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
150. How many parts does the body of the cestode consist of?
A. of three
B.of two
C.of five
D.the body of the cestode is not divided into parts
151. Which part of the body of the cestode is the growth zone?
A.neck
B.head (scolex)
C.hermafortid segment
D.mature segment
152. Where are the hermaphordite segments of the cestode?
A. in the anterior part of the body
B. the middle part of the body
C. the back part of the body
D.About all parts of the body
153. In the posterior part of the body of the cestode, which genitals are preserved?
A.uterus
B.ovary
C.testis
D.ovary
154. Cestodes develop with the participation of how many hosts?
A.2 and 3 hosts
B.two hosts
C.three hosts
D. One owner
155. Which cestode scolexes are armed with suckers and chitinized hooks?
A.Echinococcus and multiceps
B.moniezium and avitellins
C.tizaniezium and multiceps

D.stylezium and echinococcus
156. In skoleks, what kind of cestodes do not have chitinized hooks?
A.monieziy
B.multiceps
C.echinococcus
D.teniarinhus
157. Uterus which cestode is open type, develop with the participation of three hosts, larvae in the form of worms?
A.Diphyllobothrium latum
B.Taenia solium
C.Taeniarynchus saginatus
D.Echinococcus granulosus
158. Which uterus is a closed type of cestode, develop with the participation of two hosts, larvae in the form of a bubble that is filled with fluids?
A.Taeniarynchus saginatus
B.Ligula intestinalis
C.Moniezia expansa
D. Diphyllobothrium latum
159. The form of which trematodes in the form of threads is bisexual?
A. Orientobilchartsii
B.Dicrocelium
C.Paramphistomatids
D. Fascioles
160. The development cycle of the causative agent of which trematode disease occurs only in endogenous conditions?
A.Dicrocelium
B. Orientobilcharcium
C.Paramphistomatids
D.Fascioles
161. What trematodous diseases are also found in humans?
A.Fascioliasis, dicroceliosis
B.Paramphistomatosis and dicrocelium
C.Orientobilcharcosis and paramphistomatosis
D.Fascioles and echinococcosis
162. In vertebrates, what types of trematodous diseases are found?
A.in all vertebrates
B.in fish
C.in birds
D.in testers
163.What method is acceptable to combat trematodous diseases?
A.Devastation
B.Deworming
C.Disinvasion
D.Fighting shellfish

164.Why do the ribbon forms of cestodes live in the small intestine?
A. They do not have a digestive system
B.Their bodies are very long
C. They have scolexes (head)
D.They have a poorly developed neural system
165.In what kind of trematodous disease is percutaneous infection of animals observed?
A.With orientobilcharciosis
B.With fascioliasis
C.With paramphistomatosis
D.With eurythrematosis
166. Fasciolahepatica in how many days does it reach sexual maturity in the body of a definitive host?
A.75-80 days
B.80-90 days
C.80-130 days
D.90-150 days
167. Fasciolagigantica in how many days does it reach sexual maturity in the body of a definitive host?
A.90-120 days
B. 80-90 days
C.80-130 days
D.75-80 days
168. In which zones is fascioliasis most common?
A. in the irrigation and foothill-mountain zones
B. in the foothill zone
C. in the desert pasture zone
D.in all zones
169. How is the diagnosis made in case of vital fascioliasis?
A.by the method of sequential washing of feces
B. Berman-Orlovmethod
C.Vida method
D. Fulleborn method
170. Where does the parthenogenetic cycle of fasciole development occur?
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B. in the body of a definitive host
C.in water
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A.Cysticercosis of cattle
B. Teniosis
C. Echinococcosis
D. Cenurosis
177. Who is the author of the albendozol-copper vitriol salt mixture?
A.A.O.Oripov
B. D.A.Azimov
C. B.S. Salimov
D. V.S. Ershov
178. Which of the following scientists has thoroughly studied the biology of the causative agent of dicroceliosis?
A.B.S. Salimov
B. D.A.Azimov
C. A.O.Oripov
D. V.S. Ershov
179. Which of the following scientists is the first doctor of Sciences in the specialty "Helminthology"?
A.I.H.Irgashev
B. D.A.Asimov
C.A.O.Oripov
D. B.S. Salimov

180. Which cestdosis disease is mainly widespread among 1.5-8-month-old lambs, goats and calves?
A.shell mites of the genus Scheloribates
B. freshwater mollusks
C. land mollusks
D. ants
181. What is the intermediate host of the causative agent of moniesiosis?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
182. In the treatment of which disease is it recommended to use phenasal, phenalidone, 1-2% aqueous solution of copper sulfate, panacur, rintal and drugs from the albendazole group?
A.Moniesiosis
B. Cysticercosis
C. Echinococcosis
D. Cenurosis
183. For the prevention of which disease should animals be fed salt-phenothiazine-copper sulfate top dressing, the composition of which consists of 1% copper sulfate, 10% phenothiazine and 89% table salt, from October to June from a calculation of 1.0 per head?
A.Moniesiosis
B. Echinococcosis
C. Cenurosis
D. Cysticercosis
184. The sexually mature form of the causative agent of which nematode disease of ungulates parasitizes in the small intestine, and the larvae migrate hepatopulmonally?
A.Parascaris equorum
B. Paranaplocephala mamillana
C. Anaplocephala magna
D. Anaplocephala perfoliata
185. In the treatment of which nematode disease of single-hoofed animals are used piperazine salts, phenothiazine, panacur (fenbendazole), rintal (febantel), 4-x carbon chloride, albendazole and its analogues?
A.Paraskaridosis
B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
186. To prevent which nematode disease of ungulates should preventive deworming of suckling foals be carried out for the first time in August, the second time after weaning, and adult animals for the first time in March-April, the second time in October-November?
A.Paraskaridosis

B. Anaplocephalosis
C. Piroplasmosis
D. Nuttaliosis
187. Which nematode disease of sheep proceeds chronically, the causative agent of which parasitizes in the bronchi and trachea, as a result of which the infected animal has a violation of the function of the respiratory organs?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
188. Which nematode is large, filamentous, milky-white, bisexual, males 3-8 cm long, females 5-15 cm, spicule dark brown, boot-shaped, 0.4-0.6 mm long?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
189. Which nematode is a geohelminth, in the external environment the larva reaches the invasive stage in 6-7 days, the prepatent period of development is 3-4 months, the patent period is 1.5-2 years?
A.Dictyocaulus filaria
B. Fasciola hepatica
C. Moniezia ezpansa
D. Piroplasma ovis
190. In which nematode disease of sheep is there a weak, then increasing cough during movement, after a previous rest, at night, bronchitis, serous-purulent discharge from the nose, which dry out and form crusts?
A.Dictyoculosis
B. Fasciolesis
C. Psoroptosis
D. Estrosis
191. When deworming which nematode disease use 1 or 2% solutions of copper sulfate, phenothiazine, naphthamone, panacur (fenbendazole), rintal (febantel), albendazole and its analogues?
A.Hemonchosis
B. Fasciolosis
C. Paramphistomatosis
D. Dicroceliosis
192. Which nematode disease mainly infects chickens 2-10 months of age, and adult birds are parasitic carriers?
A.Ascariasis
B. Prostogonimosis
C. Daveniosis
D. Rayetinoz

193. In which nematode disease, the causative agent of which is parasitized in the conjunctival sac, under the third eyelid, in the ducts of the lacrimal gland and the lacrimal-nasal canal, as a result of which animals are observed lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids turning into keratitis, ulcers on the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
194. In which helminthic disease are the characteristic clinical signs of lacrimation, photophobia, redness and swelling of the conjunctiva, swelling of the eyelids, turning into keratitis, ulcers of the cornea, an eyesore?
A.Telyaziosis
B. Teileriosis
C. Setariosis
D. Onchocerciasis
195. In the treatment of which nematode is used 3% boric acid, Lugol solution, ditrazine citrate, iodine, lysol and others?
A.Telyaziosis
B. Setariosis
C. Onchocerciasis
D. Teileriosis
196. What chronically occurring disease of horses is characterized by skin lesions at the root of the tail, severe itching, eczema, dermatitis and severe emaciation of the animal?
A.Oxyurosis
B. Paraskaridosis
C. Anaplocephalosis
D. Pyroplasmosis
197. Which nematode of ungulates develops in a direct way, that is, without the participation of an intermediate host, the prepatent period of development is equal to 3-4 weeks, and the patent period is 6-8 months?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis
198. In which nematode disease of horses is sick mainly young animals up to one year old and old horses?
A.Oxyurosis
B. Paraskaridosis
C. Pyroplasmosis
D. Anoplocephalosis}

199. In deworming what disease is used 4-chlorinated carbon, piperazine and its salts, phenothiazine, panacur (fenbendazole), rintal (febantel), tetramizole and mebenvet in the form of granules?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Anoplocephalosis
200. For the prevention of which disease should horses undergo planned dehelminthization every 3 months, biothermic treatment of manure and full feeding of animals according to the diet?
A.Paraskaridosis
B. Piroplasmosis
C. Oxyurosis
D. Nuttaliosis
201. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis
C. Multiceps multiceps
D. Echinococcus granulosus
202. When diagnosing what protozoal disease of horses, smears are prepared from the peripheral blood of animals and on the basis of this the pathogen is detected?
A.Nuttaliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
203. In which disease are the main pathoanatomic changes observed in the genitals of animals accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis
D. Teileriosis
204. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
A.Trichomoniasis
B. Piroplasmosis
C. Fasciolosis
D. Cysticercosis
205. Which protozoal diseases of cattle are acutely and subacutely occurring and accompanied by fever, hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems?
A.Pyroplasmosis
B. Trichomoniasis

C. Eimeriosis
D. Balantidiosis
206. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is usually used, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?
A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis
207. Which protozoan diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in lymph nodes, fever, anemia, disruption of the cardiovascular and digestive system, exhaustion and a high percentage of mortality?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
208. The causative agent of what protozoal disease, once in the animal's body with tick saliva, multiplies at the beginning in the lymph nodes and forms macro- and microschizonts (garnet bodies), then macro- and micromerozoites, which are embedded in red blood cells?
A.Teileriosis
B. Benzoithiosis
C. Pyroplasmosis
D. Toxoplasmosis
209. At what disease croup. in cattle, the percentage of infection of erythrocytes with the pathogen is 80-95%?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
210. Which genus of ixodes mites is the carrier of the causative agent of bovine taileriosis?
A.Hyalomma
B. Ixodes
C. Boophylus
D. Haemaphysalis
211. Which genus of ixodes mites is the carrier of the causative agent of bovine babesiosis?
A.Ixodes
B. Hyalomma
C. Boophylus
D. Haemaphysalis

212. Which genus of ixodes mites is the carrier of the causative agent of bovine pyroplasmosis?
A.Boophylus calcaratus
B. Ixodes
C. Hyalomma
D. Haemaphysalis
213. Which genus of ixodes mites is the carrier of the causative agent of piroplasmosis of sheep and goats?
A.Rhipicephalus bursa
B. Ixodes
C. Hyalomma
D. Haemaphysalis
214. When diagnosing which protozoal disease of cattle, smears prepared from lymph nodes are examined at the beginning of the disease, and during the period of clinical signs, blood smears from peripheral vessels to detect the pathogen?
A. Teileriosis
B. Toxoplasmosis
C. Beznoitiosis
D. Pyroplasmosis
215. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists of All-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute are carefully used?
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Pyroplasmosis
216. Which genus and species of ixodes mites are carriers
of the causative agent of horse nuttaliosis?
A.Ixodes and Boophyluscalcaratus
B.Dermacentor and Hyalomma plumbeum
C.Boophylus and Rhipicephalus bursa
D.Haemaphysalis and Ixodesricinus
217. What protozoal disease of horses is acute, subacute and chronic, accompanied by hemorrhages, jaundice, anemia, the appearance of hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?
A.Nuttaliosis
B.Pyroplasmosis
C.Oxyurosis
D.Paraskaridosis
218. Which pathogen of protozoal disease has an oval, pear-shaped, dot-shaped shape, they are located in erythrocytes by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?

A.Nuttalia equi
B.Piroplasma caballi
C.Theileria annulata
D.Piroplasma bigeminum
219. What arachnous disease of sheep is acute and chronic, characterized by itching of the skin, hair loss and exhaustion of the body, sometimes ends fatally?
A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz
220. In which arachnose disease of sheep are mainly infected with animals with a thick coat, increased humidity in the winter periods of the year?
A.Psoroptosis
B.Hypodermatosis
C.Piroplasmosis
D.Estroz
221. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
222. What disease of horses is characterized by acute, subacute and chronic course, clinical forms of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?
A.Pyroplasmosis
B.Paraskaridosis
C.Oxyurosis
D.Rhinestrosis
223. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and death in acute form?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
224. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis

C. Hypodermatosis
D.Siphunculator
225. In what invasive disease of cattle does the disease proceed acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
226. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
227. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
228. In which protozoal disease of pigs, the main pathological changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariosis
C.Trichocephalosis
D. Psoroptosis
229. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis

230. For the prevention of any disease of animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
231. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
232. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
233. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general restorative medicines be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
234. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis
C.Estroz
D.Pyroplasmosis
235. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
236. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis

B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
237. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
238. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
239. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
240. In which parasite (pathogen), after piercing the skin of an animal and introducing it through it, a pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed in the animal when penetrating the spinal canal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis
241. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
242. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata

D.Cysticercus bivis
243. Which genus of ixodes mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis
244. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, dot-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
B.Piroplasma bigeminum
C.Oetrus ovis
D.Hypoderma lineatum
245. Preparations himkoccid, sulfademizin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
246. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
247. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
248. In which disease is the diagnosis made comprehensively, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis

249. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
250. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
251. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
252. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
253. A widespread animal disease occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Pyroplasmosis
C.Accidental illness
D.Sarcoptosis
254. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
255. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?

A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
256. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
257. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
258. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and fecal samples are examined in the laboratory by the Darling method, where oocysts of the parasite are detected?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
259. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
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B.Trichomoniasis
C.Leishmaniasis
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261. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.

A.Leishmaniasis
B.Trichomoniasis
C.Toxoplasmosis
D.Cysticercosis}
262. For the prevention of any disease, animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Balantidiosis
D.Psoroptosis
263. Transmissible protozoal disease, occurring acutely and subacutely, the causative agent is a parasite primarily in the RES, after it parasitizes inside the erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
264. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Psoroptosis
D.Trichomonosis
265. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Pyroplasmosis
C.Estroz
D.Moniesiosis
266. A disease in the treatment of which are used as specific drugs: azidine, bunarvalek, berenyl, diamidine and naganin, together with this should be used restorative drugs?
A.Accidental illness
B.Psoroptosis
C.Rhinestrosis
D.Pyroplasmosis
267. In which parasite (pathogen), after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating the spinal canal of the animal?
A.Hypoderma bovis

B.Cysticercus bivis
C.Theileria annulata
D.Oestrus ovis}
268. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Estroz
B.Psoroptosis
C.Melophagosis
D.Demodekoz
269. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
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B.Piroplasma bigeminum
C.Theileria annulata
D.Eimeria bovis
271. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
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B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bovis
272. The drugs himkoccid, sulfadimesin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Siphunculatosis
D.Hypodermatosis
273. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis

274. In which disease is the diagnosis made in a complex, and in particular for laboratory studies, the punctate is taken primarily from the lymph nodes, subsequently from peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
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B.Rayetinosis
C.Trichomoniasis
D.Psoroptosis
275. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estrosis
B.Melophagosis
C.Psoroptosis
D.Demodekoz
276. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
277. Parasitic disease of birds, occurring acutely, subacute and chronically, young animals aged 5 to 90 days are sick, the litter is liquid, whitish-green or dark brown with an admixture of blood, in the laboratory for the detection of oocysts of the parasite, litter samples are examined by the Darling method.
A. Eimeriosis
B.Ascariasis
C.Rayetinosis
D.Heterakidosis
278. A widespread animal disease occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Accidental illness
C.Pyroplasmosis
D.Sarcoptosis
279. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Hypodermatosis
C.Psoroptosis

D.Siphunculatosis
280. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of the day, the disease occurs in cells with crowded rabbits, in a damp room.
A.Eimeriosis
B.Fascioliasis
C.Cystecercosis
D.Passaluroz}
281. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis
C. Multiceps multiceps
D. Echinococcus granulosus
282. When diagnosing what protozoal disease of horses, smears are prepared from the peripheral blood of animals and on the basis of this the pathogen is detected?
A.Nuttaliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
283. In which disease are the main pathoanatomic changes observed in the genitals of animals accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis
D. Teileriosis
284. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
A.Trichomoniasis
B. Piroplasmosis
C. Fasciolosis
D. Cysticercosis
285. Which protozoal diseases of cattle are acutely and subacutely occurring and accompanied by fever, hemoglobinuria, disorders of the cardiovascular, digestive and nervous systems?
A.Pyroplasmosis
B. Trichomoniasis
C. Eimeriosis
D. Balantidiosis
286. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is usually used, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?

A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis
287. Which protozoan diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in lymph nodes, fever, anemia, disruption of the cardiovascular and digestive systems, exhaustion and a high percentage of mortality?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
288. The causative agent of what protozoal disease, once in the animal's body with tick saliva, multiplies at the beginning in the lymph nodes and forms macro- and microschizonts (garnet bodies), then macro- and micromerozoites, which are embedded in red blood cells?
A.Teileriosis
B. Benzoithiosis
C. Pyroplasmosis
D. Toxoplasmosis
289. At what disease croup. in cattle, the percentage of infection of erythrocytes with the pathogen is 80-95%?
A.Teileriosis
B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
290. Which genus of ixodes mites is the carrier of the causative agent of bovine taileriosis?
A.Hyalomma
B. Ixodes
C. Boophylus
D. Haemaphysalis
291. Which genus of ixodes mites is the carrier of the causative agent of bovine babesiosis?
A.Ixodes
B. Hyalomma
C. Boophylus
D. Haemaphysalis
292. Which genus of ixodes mites is the carrier of the causative agent of bovine pyroplasmosis?
A.Boophylus calcaratus
B. Ixodes
C. Hyalomma
D. Haemaphysalis

293. Which genus of ixodes mites is the carrier of the causative agent of piroplasmosis of sheep and goats?
A.Rhipicephalus bursa
B. Ixodes
C. Hyalomma
D. Haemaphysalis
294. When diagnosing which protozoal disease of cattle, smears prepared from lymph nodes are examined at the beginning of the disease, and during the period of clinical signs, blood smears from peripheral vessels to detect the pathogen?
A. Teileriosis
B. Toxoplasmosis
C. Beznoitiosis
D. Pyroplasmosis
295. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists of All-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute are carefully used?
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Pyroplasmosis
296.Which genus and species of ixodes mites are carriers of the causative agent of horse nuttaliosis?
A.Ixodes and Boophylus calcaratus
B.Dermacentor and Hyalomma plumbeum
C.Boophylus and Rhipicephalus bursa
D.Haemaphysalis and Ixodes ricinus
297. What protozoal disease of horses is acute, subacute and chronic, accompanied by hemorrhages, jaundice, anemia, the appearance of hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?
A.Nuttaliosis
B.Pyroplasmosis
C.Oxyurosis
D.Paraskaridosis
298. Which pathogen of protozoal disease has an oval, pear-shaped, dot-shaped shape, they are located in erythrocytes by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?
A.Nuttalia equi
B.Piroplasma caballi
C.Theileria annulata
D.Piroplasma bigeminum
299. What arachnous disease of sheep is acute and chronic, characterized by itching of the skin, hair loss and exhaustion of the body, sometimes ends fatally?

A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz
300. In which arachnose disease of sheep are mainly infected with animals with a thick coat, increased humidity in the winter periods of the year?
A.Psoroptosis
B.Hypodermatosis
C.Piroplasmosis
D.Estroz
301. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
302. What disease of horses is characterized by acute, subacute and chronic course, clinical forms of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?
A.Pyroplasmosis
B.Paraskaridosis
C.Oxyurosis
D.Rhinestrosis
303. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and death in acute form?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
304. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis
C. Hypodermatosis
D.Siphunculator
305. In what invasive disease of cattle does the disease proceed acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?

A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
306. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
307. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
308. In which protozoal disease of pigs, the main pathological changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariosis
C.Trichocephalosis
D. Psoroptosis
309. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
310. For the prevention of which disease animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis

311. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
312. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
313. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general restorative medicines be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
314. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis
C.Estroz
D.Pyroplasmosis
315. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
316. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
317. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis

B.Estrosis
C.Balantidiosis
D.Eimerioz
318. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
319. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
320. In which parasite (pathogen), after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating the spinal canal of the animal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis
321. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
322. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped paired?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
323. Which genus of ixodes mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis

324. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, dot-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
B.Piroplasma bigeminum
C.Oetrus ovis
D.Hypoderma lineatum
325. Preparations himkoccid, sulfademizin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
326. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
327. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
328. In which disease is the diagnosis made comprehensively, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis
329. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis

330. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
331. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
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C.Demodecosis
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A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
333. A widespread animal disease occurring in all Central Asian countries (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Pyroplasmosis
C.Accidental illness
D.Sarcoptosis
334. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
335. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?
A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
336. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice

of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
337. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
338. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and fecal samples are examined in the laboratory by the Darling method, where oocysts of the parasite are detected?
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B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
339. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
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B.Ascariasis
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341. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Leishmaniasis
B.Trichomoniasis
C.Toxoplasmosis
D.Cysticercosis}

342. For the prevention of any disease, animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Balantidiosis
D.Psoroptosis
343. Transmissible protozoal disease, occurring acutely and subacutely, the causative agent is a parasite primarily in the RES, after it parasitizes inside the erythrocytes?
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B.Cysticercus bivis
C.Theileria annulata
D.Oestrus ovis}

348. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Estroz
B.Psoroptosis
C.Melophagosis
D.Demodekoz
349. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?
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B.Estrosis
C.Balantidiosis
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C.Trichomoniasis
D.Psoroptosis
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B.Melophagosis
C.Psoroptosis
D.Demodekoz
356. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
357. Parasitic disease of birds, occurring acutely, subacute and chronically, young animals aged 5 to 90 days are sick, the litter is liquid, whitish-green or dark brown with an admixture of blood, in the laboratory for the detection of oocysts of the parasite, litter samples are examined by the Darling method.
A. Eimeriosis
B.Ascariasis
C.Rayetinosis
D.Heterakidosis
358. A widespread animal disease occurring in all countries of Central Asia (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Accidental illness
C.Pyroplasmosis
D.Sarcoptosis
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A.Eimeriosis
B.Hypodermatosis
C.Psoroptosis
D.Siphunculatosis
360. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of the day, the disease occurs in cells with crowded rabbits, in a damp room.

A.Eimeriosis
B.Fascioliasis
C.Cystecercosis
D.Passaluroz
361. The causative agent of which protozoal disease of dogs is carried by ixodic ticks from the genus Dermacentor and Rhipicephalus?
A.Piroplasma canis
B. Toxocara canis
C. Multiceps multiceps
D. Echinococcus granulosus
362. When diagnosing what protozoal disease of horses, smears are prepared from the peripheral blood of animals and on the basis of this the pathogen is detected?
A.Nuttaliosis
B. Oxyurosis
C. Paraskaridosis
D. Anoplocephalosis
363. In which disease are the main pathoanatomic changes observed in the genitals of animals accompanied by catarrhal-purulent vaginitis, vestibulitis, cervicitis and pyometritis?
A.Trichomoniasis
B. Echinococcosis
C. Pyroplasmosis
D. Teileriosis
364. In which protozoal disease is an accurate diagnosis made on the basis of sowing on nutrient media, flushing from the mucous membranes of the genitals of patients?
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C. Fasciolosis
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B. Trichomoniasis
C. Eimeriosis
D. Balantidiosis
366. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific treatment is required, as what are azidine, berenyl, norotrip, babenil, diamidine, sulfantrol, DAT, polyamidine and other drugs used?
A.Pyroplasmosis
B. Trichomoniasis
C. Psoroptosis
D. Hypodermatosis

367. Which protozoan diseases of cattle is an acute and subacute vector-borne disease caused by non-pigmented protozoa, accompanied by a unilateral increase in lymph nodes, fever, anemia, disruption of the cardiovascular and digestive system, exhaustion and a high percentage of mortality?
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B. Pyroplasmosis
C. Benznoitiosis
D. Toxoplasmosis
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B. Ixodes
C. Boophylus
D. Haemaphysalis
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372. Which genus of ixodes mites is the carrier of the causative agent of bovine pyroplasmosis?
A.Boophylus calcaratus
B. Ixodes
C. Hyalomma
D. Haemaphysalis
373. Which genus of ixodes mites is the carrier of the causative agent of piroplasmosis of sheep and goats?
A.Rhipicephalus bursa
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C. Hyalomma
D. Haemaphysalis
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C. Beznoitiosis
D. Pyroplasmosis
375. In the treatment of which protozoal disease of cattle, in addition to symptomatic and pathogenetic methods, specific drugs and treatment methods recommended by scientists of All-Russian Institute of Experimental Veterinary Medicine, Uzbekistan Veterinary Research Institute, Tajikistan Veterinary Research Institute, Kazakhstan Veterinary Research Institute are carefully used?
A.Teileriosis
B. Beznoitiosis
C.Toxoplasmosis
D. Pyroplasmosis
376.Which genus and species of ixodes mites are carriers of the causative agent of horse nuttaliosis?
A.Ixodes and Boophylus calcaratus
B.Dermacentor and Hyalomma plumbeum
C.Boophylus and Rhipicephalus bursa
D.Haemaphysalis and Ixodes ricinus
377. What protozoal disease of horses is acute, subacute and chronic, accompanied by hemorrhages, jaundice, anemia, the appearance of hemorrhages, disorders of the nervous, cardiovascular and digestive systems of the body?
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B.Pyroplasmosis
C.Oxyurosis
D.Paraskaridosis
378. Which pathogen of protozoal disease has an oval, pear-shaped, dot-shaped shape, they are located in erythrocytes by four parasites in the form of a Maltese cross, the size of which ranges from 1 to 4 microns?
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B.Piroplasma caballi
C.Theileria annulata
D.Piroplasma bigeminum
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A.Psoroptosis
B.Hypodermatosis
C.Pyroplasmosis
D.Estroz

380. At what arachnose disease of sheep do animals with thick wool cover, increased humidity in the winter periods of the year mainly become infected?
A.Psoroptosis
B.Hypodermatosis
C.Piroplasmosis
D.Estroz
381. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
382. What disease of horses is characterized by acute, subacute and chronic course, clinical forms of the disease, accompanied by an increase in body temperature, anemia and jaundice of visible mucous membranes?
A.Pyroplasmosis
B.Paraskaridosis
C.Oxyurosis
D.Rhinestrosis
383. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and death in acute form?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
384. What is the protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the parasite oocysts are detected?
A.Eimeriosis
B.Psoroptosis
C. Hypodermatosis
D.Siphunculator
385. In what invasive disease of cattle does the disease proceed acutely and chronically, where the symptoms of the disease are characterized by impaired digestive function, severe diarrhea (sometimes with an admixture of blood), emaciation and in acute form ends with the death of the animal?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator

386. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fuleborn methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
387. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
388. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B. Ascariosis
C.Trichocephalosis
D. Psoroptosis
389. In which dog disease clinical symptoms appear depending on the type of pathogen and are divided into 2 types: cutaneous and visceral form. With the skin form, nodules appear on the back of the nose, lips, eyebrows, on the edges of the ears, fingers, which subsequently ulcerate. The visceral form is an increase in temperature, an increase in the spleen, liver, lymph nodes, conjunctivitis develops, blepharitis, baldness of the head and back?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
390. For the prevention of which disease animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
391. Transmissible protozoal disease, occurring acutely and subacutely, the pathogen parasitizes primarily in RES, after - inside erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis

D.Psoroptosis
392. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
393. A disease in the treatment of which azidine, bunarvalek, berenyl, diamidine and naganin are used as specific drugs, should general restorative medicines be used together with this?
A.Accidental illness
B.Pyroplasmosis
C. Rhinestrosis
D.Psoroptosis
394. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Monesiosis
C.Estroz
D.Pyroplasmosis
395. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of life. The disease occurs in cells with a crowded content of rabbits, in a damp room.
A.Passalurosis
B.Eimeriosis
C.Cystecercosis
D.Psoroptosis
396. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Estrosis
C.Pyroplasmosis
D.Moniesiosis
397. What is the causative agent of a disease in cattle in whose body all stages of development are completed within: in males for 14-16 days, and in females for 18-20 days?
A.Psoroptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
398. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?

A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
399. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercusbivis
400. Which parasite (pathogen) after piercing the skin of the animal and introducing it through it, the pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, when penetrating the spinal canal, paresis and paralysis of the extremities are observed in the animal?
A.Hypoderma bovis
B.Oestrus ovis
C.Theileria annulata
D.Cysticercus bivis
441. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
442. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear–shaped?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
443. Which genus of ixodic mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis
444. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, dot-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
B.Piroplasma bigeminum
C.Oetrus ovis

D.Hypoderma lineatum
445. Preparations chemcoccide, sulfademizine, phthalazole, coccidiovite, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Aimerioz
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
446. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
447. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
448. In which disease is the diagnosis made in a complex, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis
449. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
450. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are found in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis

451. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
452. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
453. A widespread animal disease occurring in all countries of Central Asia (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Pyroplasmosis
C.Accidental illness
D.Sarcoptosis
454. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
455. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?
A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
456. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz

457. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
458. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the oocysts of the parasite are detected?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
459. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
460. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fulebern methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis
461. A disease where treatment is carried out depending on the form of the disease, in the cutaneous form, treatment is carried out by using the drugs akrikhin, monomycin, solusurmin, and in the visceral form of the disease, treatment has not been developed.
A.Leishmaniasis
B.Trichomoniasis
C.Toxoplasmosis
D.Cysticercosis}
462. For the prevention of any disease, animals that have entered the farm are subject to the strictest supervision, artificial insemination is carried out in strict compliance with vet rules.sanitation?
A.Trichomonosis
B.Ascariasis
C.Balantidiosis
D.Psoroptosis

463. Transmissible protozoal disease, occurring acutely and subacutely, the causative agent is a parasite primarily in the RES, after it parasitizes inside the erythrocytes?
A.Teileriosis
B.Pyroplasmosis
C.Trichomoniasis
D.Psoroptosis
464. In which disease pathoanatomic changes are observed in the form of: exhaustion of the corpse, pale mucous membranes with a jaundice tinge, hemorrhages, superficial lymph nodes are enlarged. Characteristic changes in rennet – ulcers with pink edges of 2-10 mm in size?
A.Teileriosis
B.Pyroplasmosis
C.Psoroptosis
D.Trichomonosis
465. In what disease is there severe itching of the skin, damage to the skin, hair loss, skin compaction?
A.Psoroptosis
B.Pyroplasmosis
C.Estroz
D.Moniesiosis
466. A disease in the treatment of which specific drugs are used: azidine, bunarvalek, berenyl, diamidine and naganin, together with this, general strengthening drugs should be used?
A.Accidental illness
B.Psoroptosis
C.Rhinestrosis
D.Pyroplasmosis
467. In which parasite (pathogen), after piercing the skin of an animal and introducing it through it, a pathogenic effect for the animal's body begins, the parasite damages connective tissues, causes mechanical and toxic effects, and in some cases, paresis and paralysis of the extremities are observed when penetrating into the spinal canal of an animal?
A.Hypoderma bovis
B.Cysticercus bivis
C.Theileria annulata
D.Oestrus ovis}
468. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Estroz
B.Psoroptosis
C.Melophagosis
D.Demodekoz
469. The causative agent of which disease, in pigs in the body, reaches all stages of development within 15-19 days?

A.Sarcoptosis
B.Estrosis
C.Balantidiosis
D.Eimerioz
470. With protozoal disease of dogs, the causative agent of which disease parasitizes inside the red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than the radius of the erythrocyte, the characteristic shape is pear-shaped paired?
A.Piroplasma canis
B.Piroplasma bigeminum
C.Theileria annulata
D.Eimeria bovis
471. Which parasite's body consists of 3 parts, imago blackish-yellow or grayish-yellow, length 10-12 mm, width 3 mm?
A.Oestrus ovis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bovis
472. The preparations himkoccid, sulfadimesin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Siphunculatosis
D.Hypodermatosis
473. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis
474. In which disease the diagnosis is made comprehensively, and in particular for laboratory studies, the punctate is taken primarily from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Teileriosis
B.Rayetinosis
C.Trichomoniasis
D.Psoroptosis
475. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz

B.Melophagosis
C.Psoroptosis
D.Demodekoz
476. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
477. Parasitic disease of birds, occurring acutely, subacute and chronically, young animals aged 5 to 90 days are sick, the litter is liquid, whitish-green or dark brown with an admixture of blood, in the laboratory for the detection of oocysts of the parasite, litter samples are examined by the Darling method.
A. Eimeriosis
B.Ascariasis
C.Rayetinosis
D.Heterakidosis
478. A widespread animal disease occurring in all countries of Central Asia (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Accidental illness
C.Pyroplasmosis
D.Sarcoptosis
479. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Hypodermatosis
C.Psoroptosis
D.Siphunculatosis
480. The disease of rabbits, the infection rate of which reaches up to 70-100%, rabbits can become infected from the first days of the day, the disease occurs in cells with crowded rabbits, in a damp room.
A.Eimeriosis
B.Fascioliasis
C.Cystecercosis
D.Passaluroz
481. Widespread sheep disease, occurring most often in summer, especially in desert and semi-desert zones?
A.Oestrus ovis
B.Hypoderma bovis

C.Theileria annulata
D.Cysticercus bivis
482. With protozoal disease of dogs, the causative agent of which disease parasitizes inside red blood cells, placing 1-2, sometimes up to 16, the size of these parasites is larger than erythrocyte radius, characteristic shape – pear-shaped paired?
A.Piroplasma canis
B.Hypoderma bovis
C.Theileria annulata
D.Cysticercus bivis
483. Which genus of ixodes mites is the carrier of the causative agent of horse nuttaliosis?
A.Dermacentor and Hyalomma
B.Ixodes
C.Hyalomma
D.Haemaphysalis
484. The causative agent of the disease parasitizing in the erythrocytes of horses: rounded, pear-shaped, dot-shaped, most often the typical form is cross-shaped (Maltese cross)?
A.Nuttalia equi
B.Piroplasma bigeminum
C.Oetrus ovis
D.Hypoderma lineatum
485. The drugs himkoccid, sulfademizin, phthalazole, coccidiovit, clopidol, biocimine for the treatment of what diseases of cattle are used?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
486. Which pathogen of the disease in the body of the intermediate host reproduces asexually, the form of endozoites in the form of a crescent or orange slices, one edge pointed and the other blunted?
A.Toxoplasmosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
487. Protozoal disease of pigs, the incubation period of which is 3-17 days. With an acute course, there is an increase in temperature, diarrhea, characteristic signs of changing the color of feces from grayish to coffee, watery with an admixture of blood and a fetid odor?
A.Balantidiosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis

488. In which disease is the diagnosis made comprehensively, and in particular for laboratory studies, the primary punctate is taken from the lymph nodes, subsequently from the peripheral blood vessels, a thin smear is prepared, painted according to Romanovsky and examined under a microscope.
A.Pyroplasmosis
B.Rayetinoz
C.Psoroptosis
D.Siphunculatosis
489. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
490. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and lower back, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Demodecosis
C.Psoroptosis
D.Sarcoptosis
491. Mechanical, toxic and pathogenic action of the causative agent of which disease causes inflammatory processes, ulceration up to purulent processes in the nasal passages and even sometimes in the brain?
A.Estroz
B.Psoroptosis
C.Demodecosis
D.Melophagosis
492. In which disease, by palpation, subcutaneous nodules of 3-4 cm in size are detected in the withers and loins, and in the middle of the nodules are hollows or sometimes a hole?
A.Hypodermatosis
B.Psoroptosis
C.Demodecosis
D.Sarcoptosis
493. A widespread animal disease occurring in all countries of Central Asia (including Uzbekistan), animals get sick in the hot season on pastures during grazing around non-flowing, sedimentary reservoirs.
A.Su-aura
B.Pyroplasmosis
C.Accidental illness
D.Sarcoptosis

494. Invasive disease of cattle, where the course of the disease is acute, subacute and chronic, with the manifestation of symptoms: - impaired digestive function, gastrointestinal tract, severe diarrhea (sometimes with an admixture of blood), emaciation and acute death?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculatosis
495. What kind of eimeria parasitizes in the bile ducts of the liver in rabbits?
A.Eimeria stidae
B.Eimeria perforans
C.Eimeria maxima
D.Eimeria bovis
496. What protozoal disease of dogs is characterized by an acute and chronic course of the disease, accompanied by an increase in body temperature, pallor and jaundice of visible mucous membranes, hemoglobinuria, as well as increased heart rate and atony of the intestinal tract?
A.Pyroplasmosis
B.Toxocarosis
C.Teniidosis
D.Demodekoz
497. What disease of horses is characterized by acute, subacute and chronic course of the clinical form of the disease, accompanied by an increase in body temperature, anemia and jaundice of the visible mucous membranes?
A.Pyroplasmosis
B.Telyaziosis
C.Anoplocephalosis
D.Gastrophyllosis
498. Protozoal disease of cattle, in which the diagnosis is made comprehensively, and in the laboratory fecal samples are examined by the Darling method, where the oocysts of the parasite are detected?
A.Eimeriosis
B.Psoroptosis
C.Hypodermatosis
D.Siphunculator
499. In which protozoal disease of pigs, the main pathoanatomical changes occur in the colon of the blind and rectum, with characteristic redness, the contents are watery with an admixture of mucus, sometimes blood, the mesentery vessels are blood-filled, lymph nodes are enlarged, dystrophic changes in the heart?
A.Balantidiosis
B.Ascariasis
C.Trichocephalosis
D.Psoroptosis

500. Anthroponotic disease, where the final diagnosis in the laboratory is made by preparing smears from parenchymal organs, where endozootics are detected, and fecal samples in cats are examined by Darling or Fulebern methods to detect oocysts?
A.Toxoplasmosis
B.Trichomoniasis
C.Leishmaniasis
D.Cysticercosis

V. EVALUATION CRITERIA

Evaluation criteria
Students' academic performance is assessed on a 5-point scale:

5 (excellent rating):

Give a conclusion and make a decision.
Application of acquired knowledge in practice;
Understanding the essence of the subject;
Know and tell;
Imagine;

4 (good rating):

Be able to think independently;
Understanding the essence of the subject;
Know and tell;
Imagine;

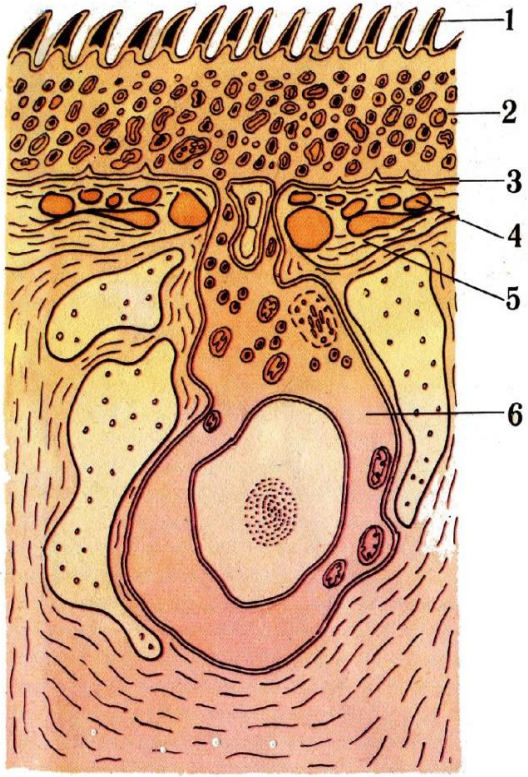
3 (satisfactory rating):

Understanding the essence of the subject;
Know and tell;
Imagine;

2 (unsatisfactory assessment):

Not mastering the program;
Ignorance of the essence of the subject:
Lack of clarity;
Inability to think independently;

VI.HANDOUTS ON THE SUBJECT



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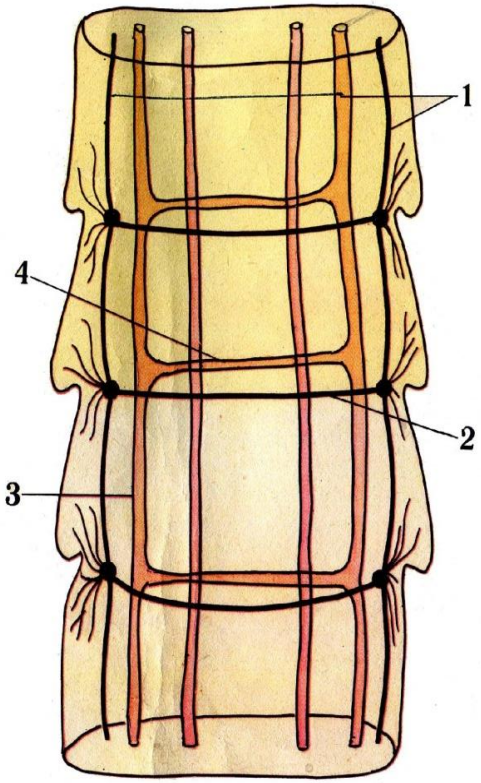

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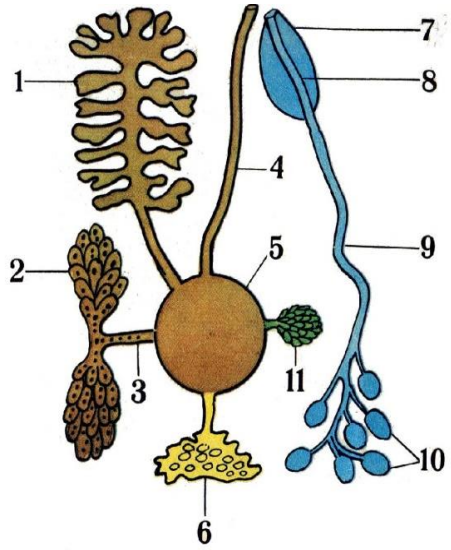

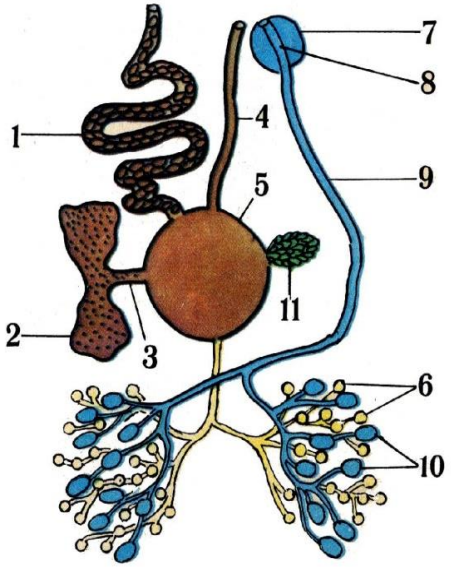

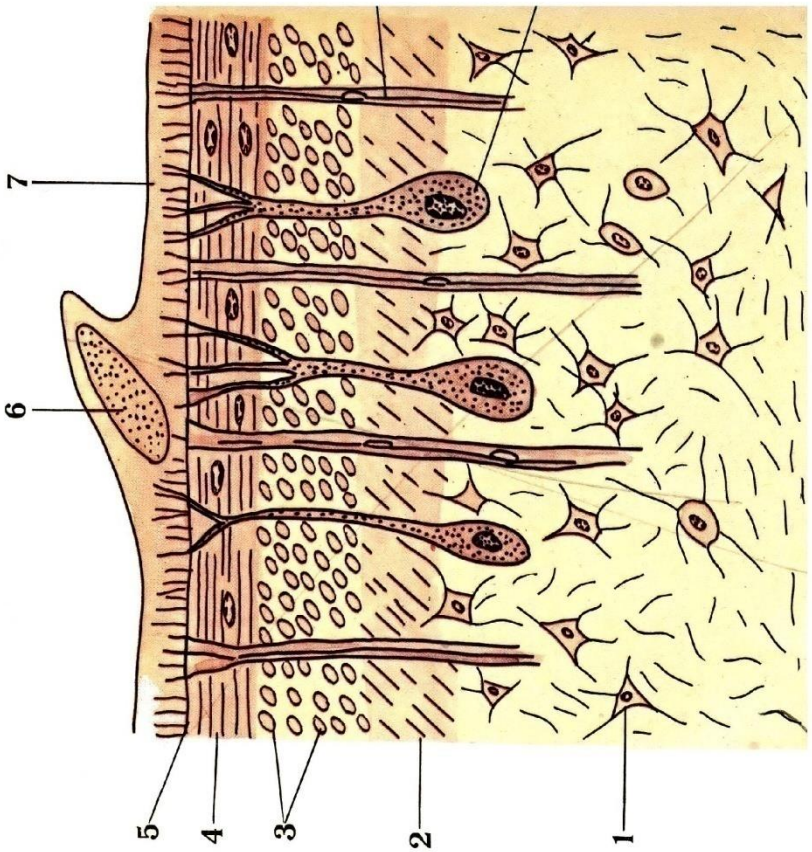
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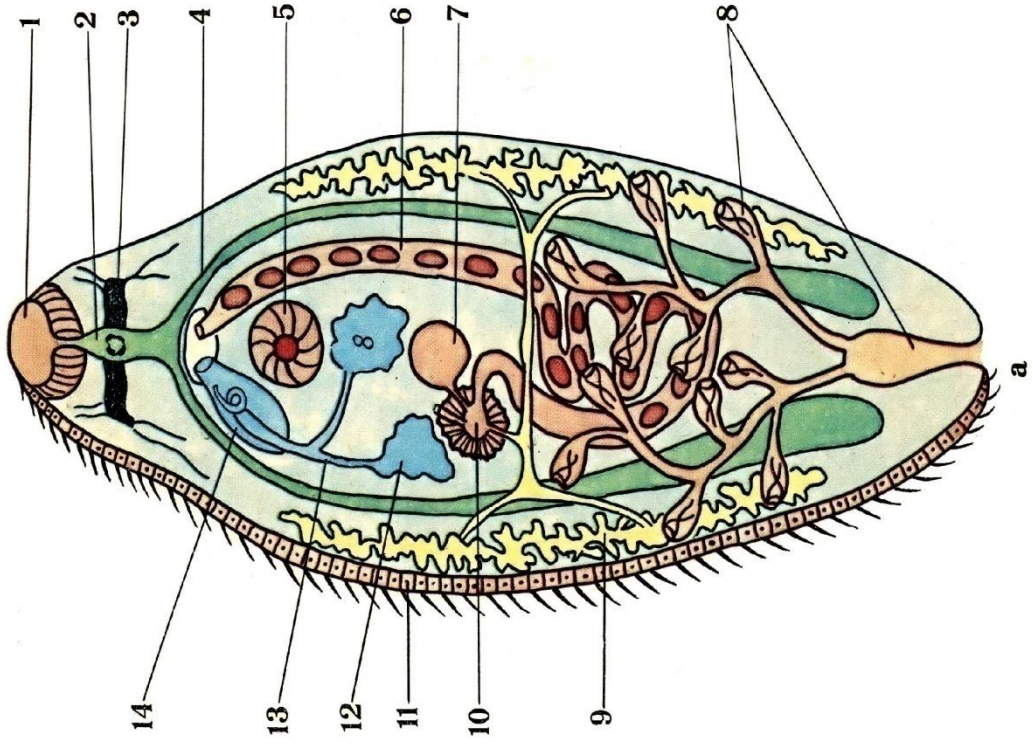
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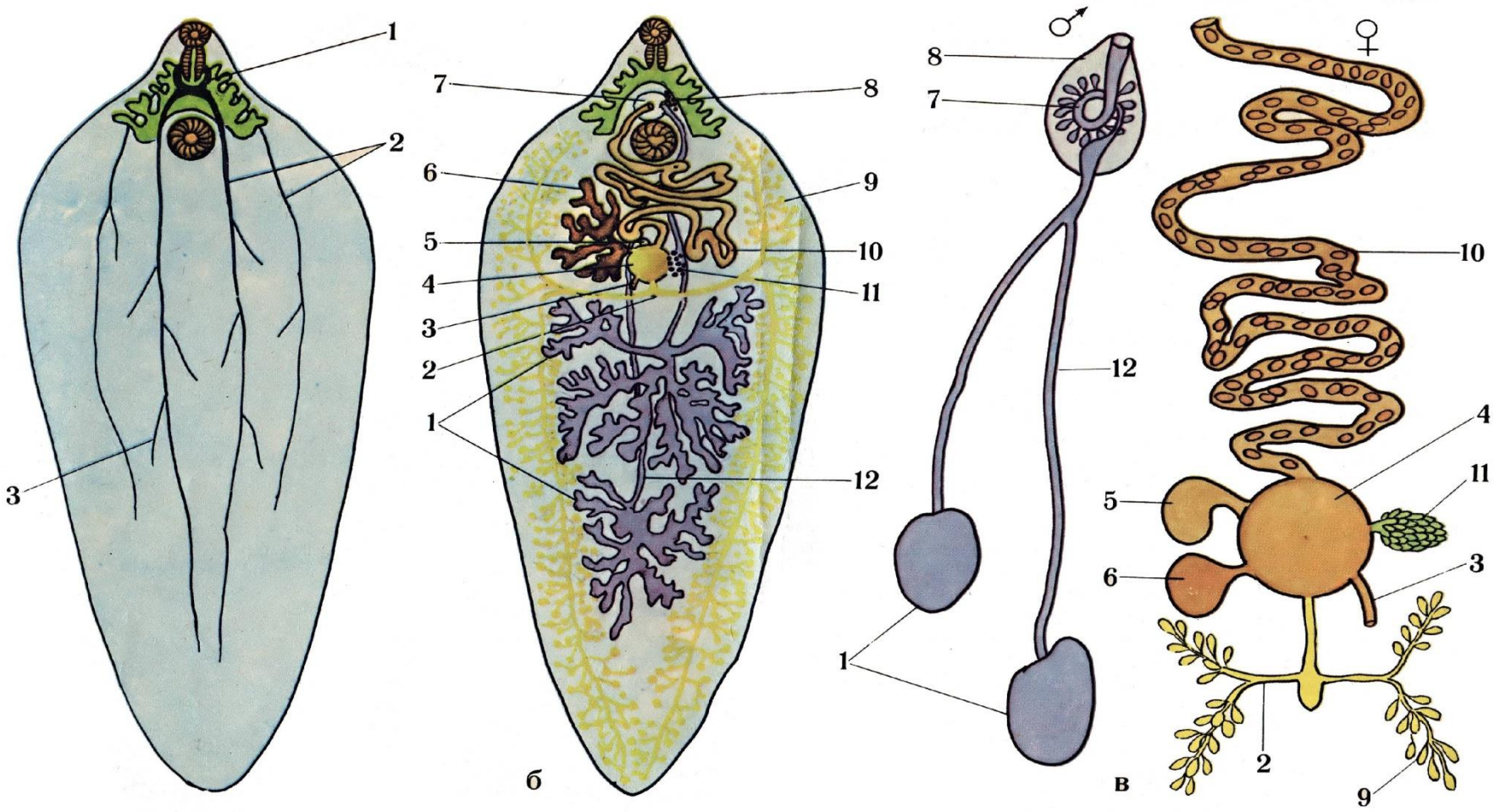
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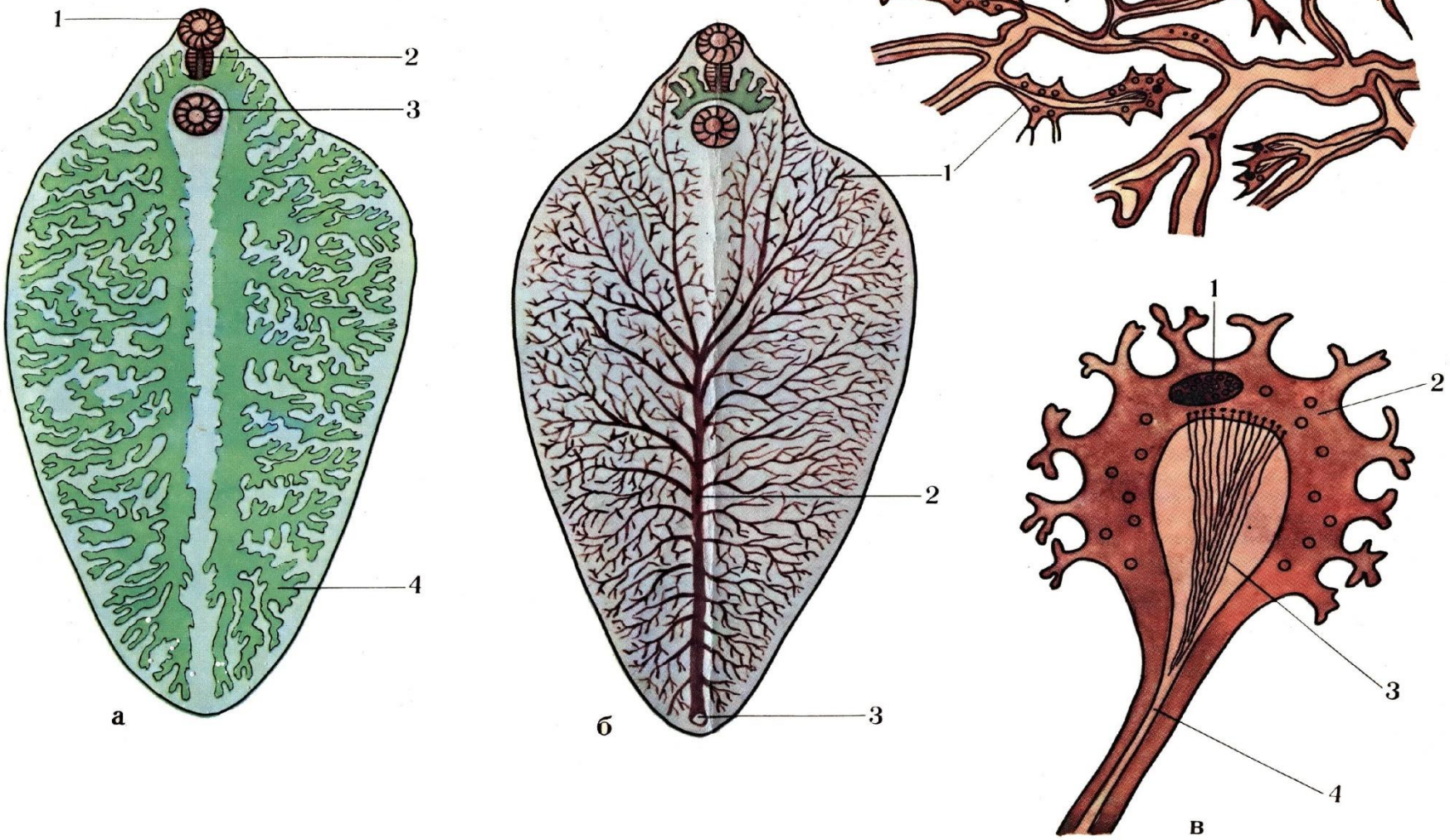
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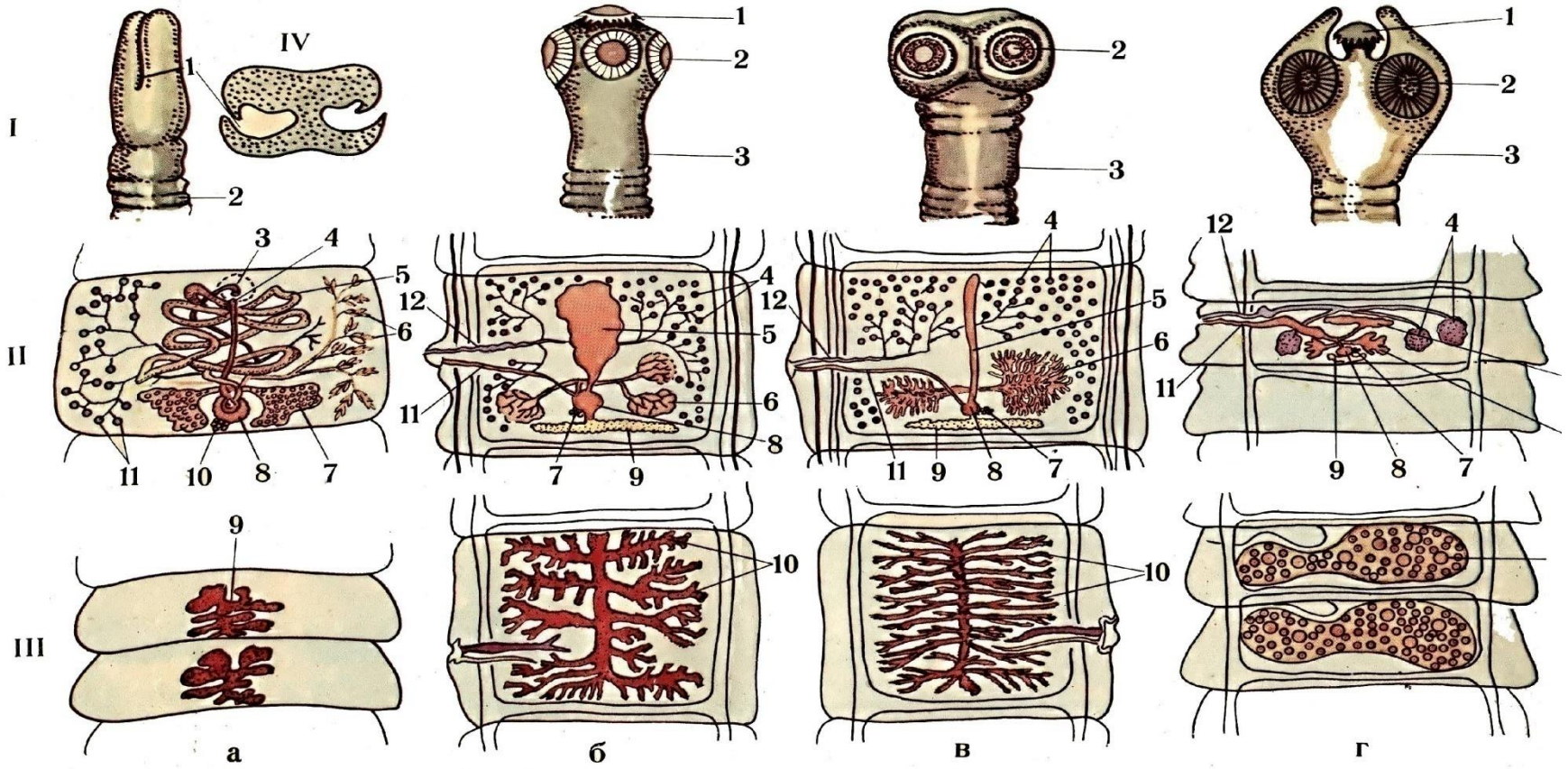
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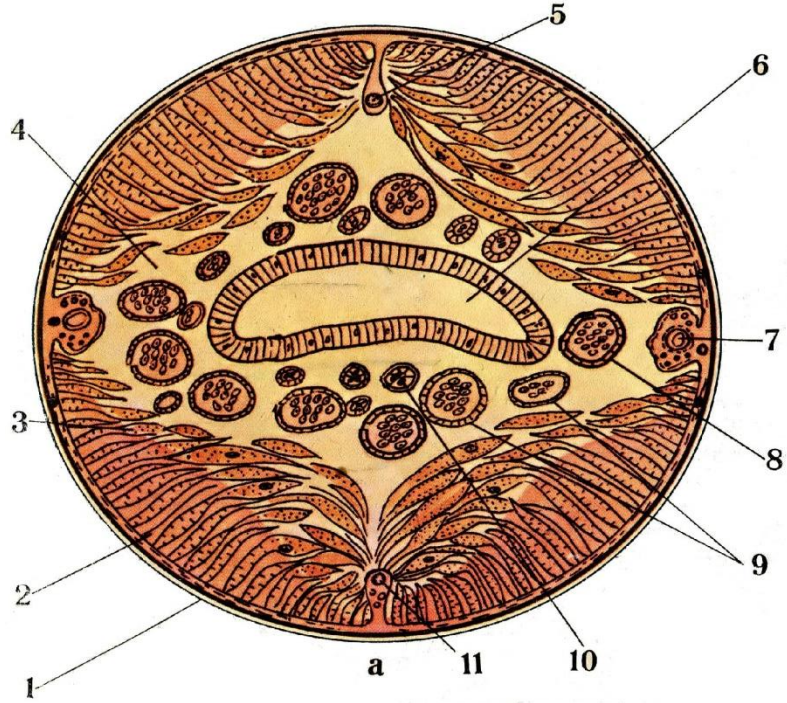

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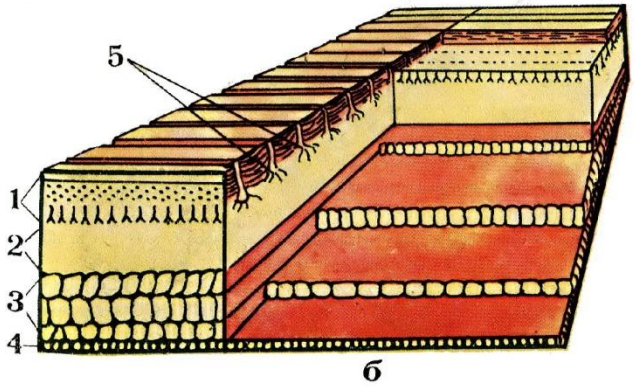

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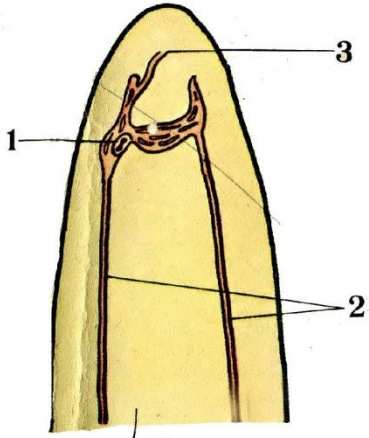

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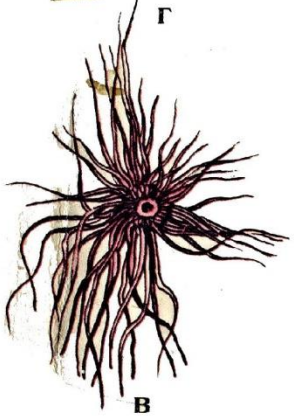

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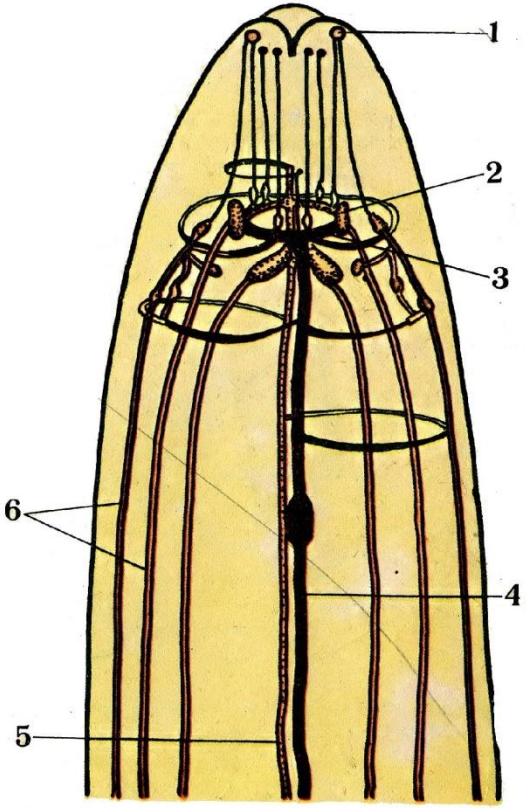

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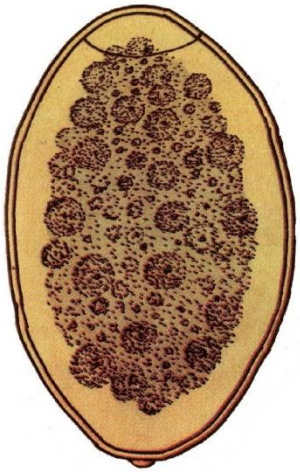

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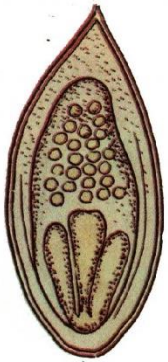

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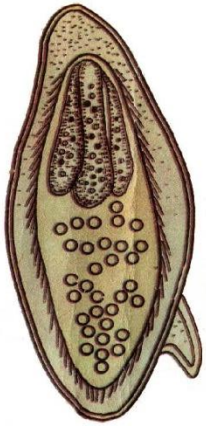

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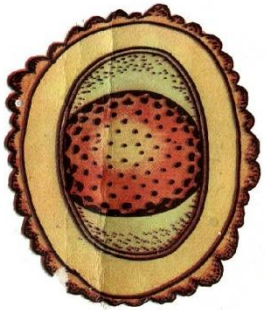

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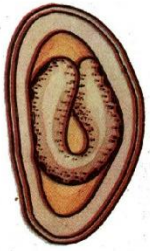

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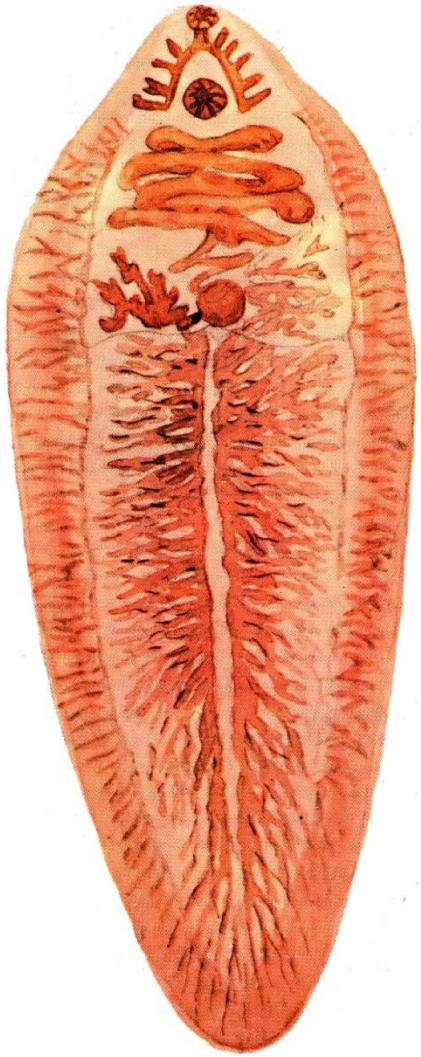

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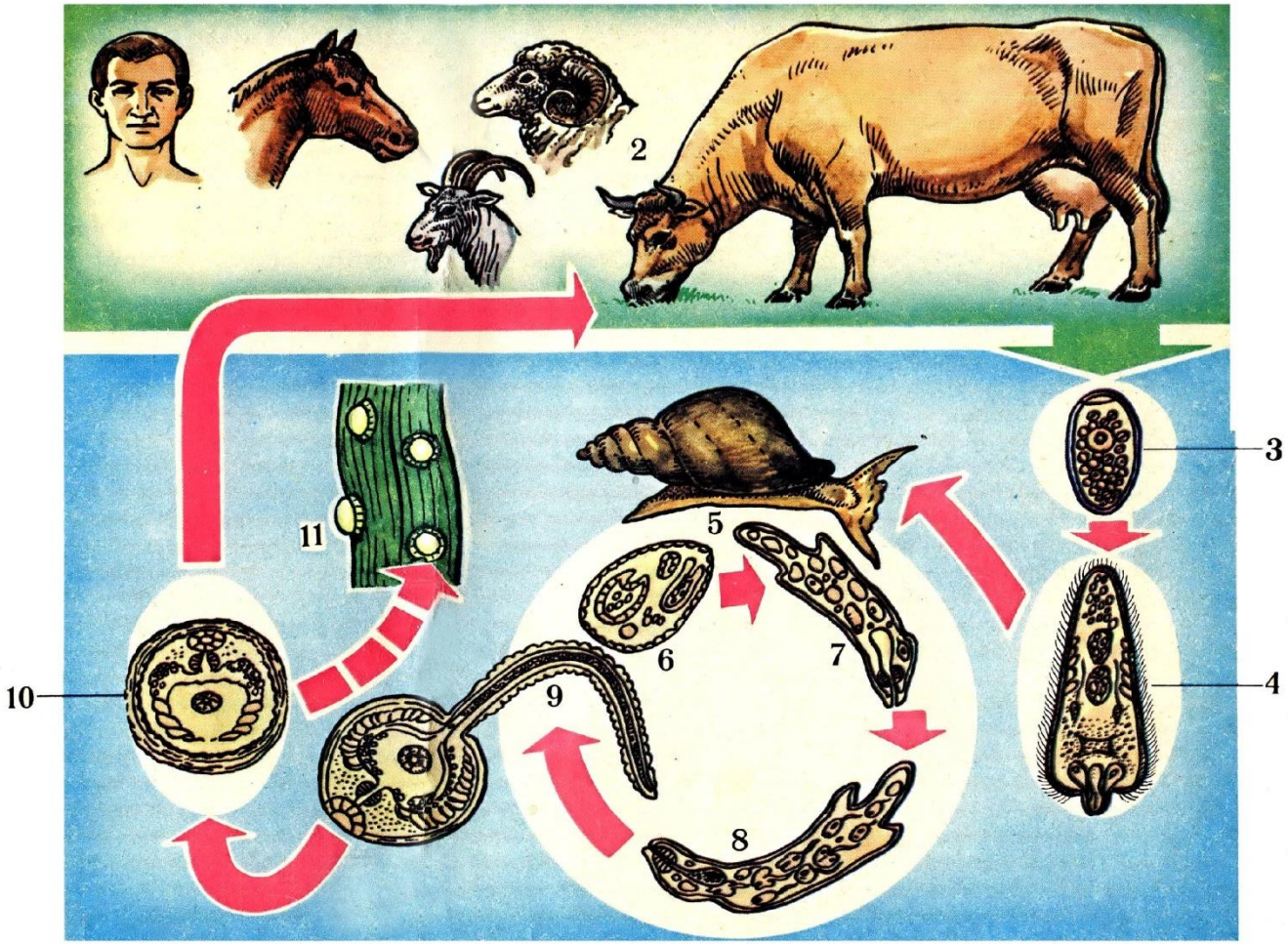


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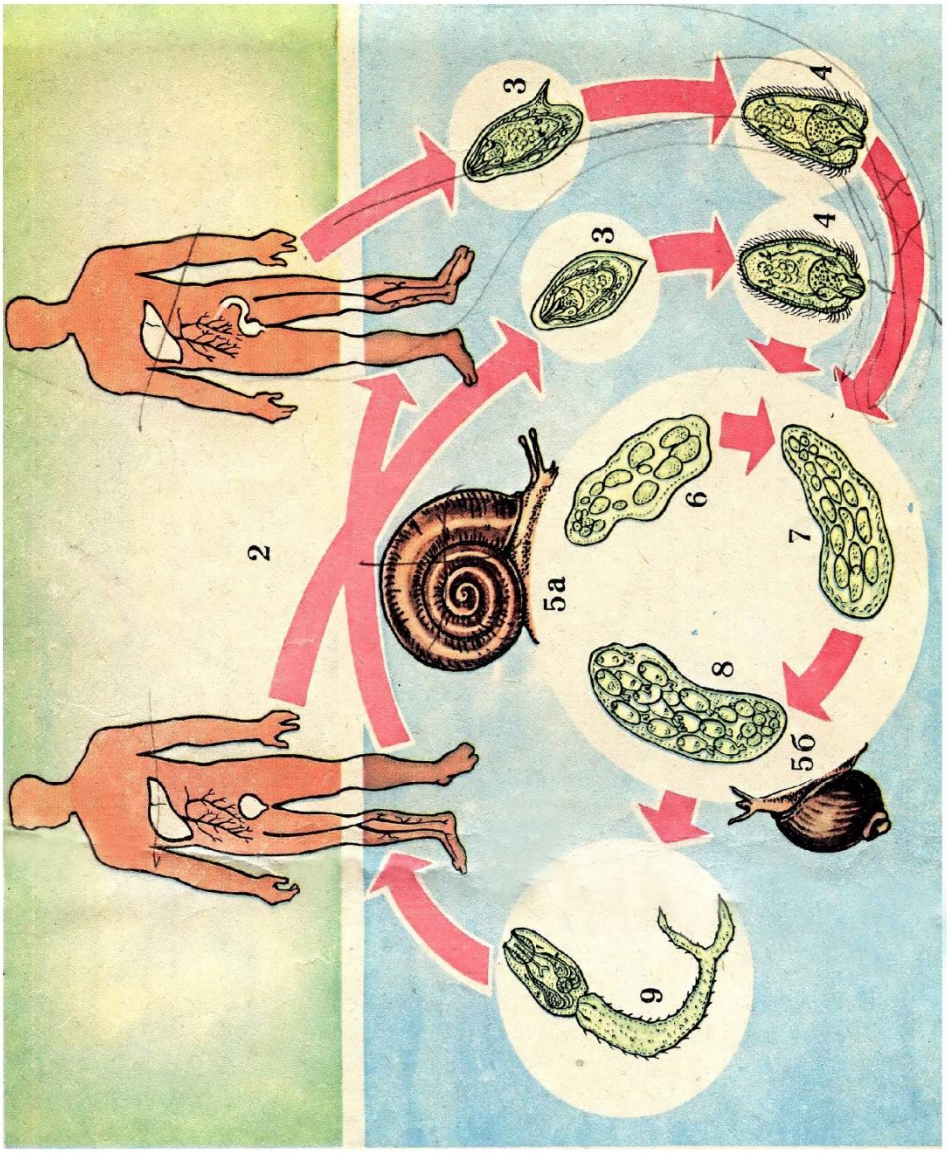
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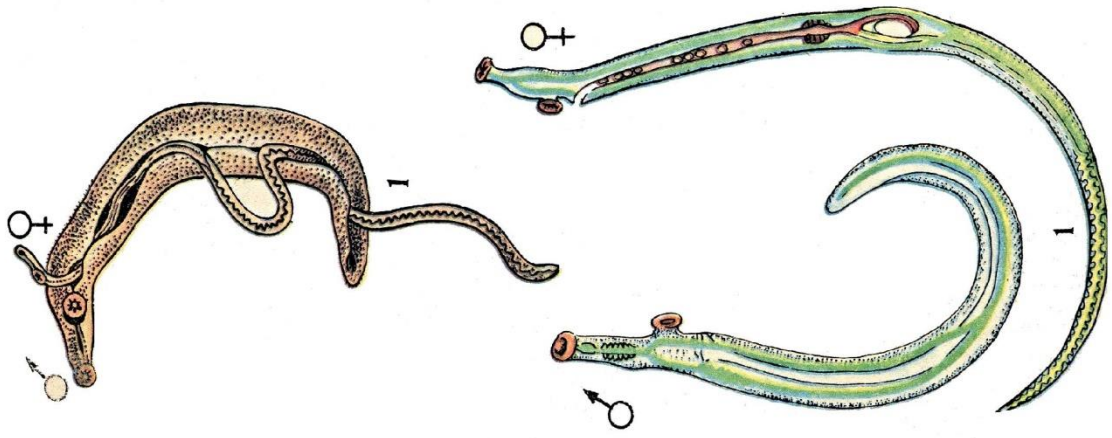
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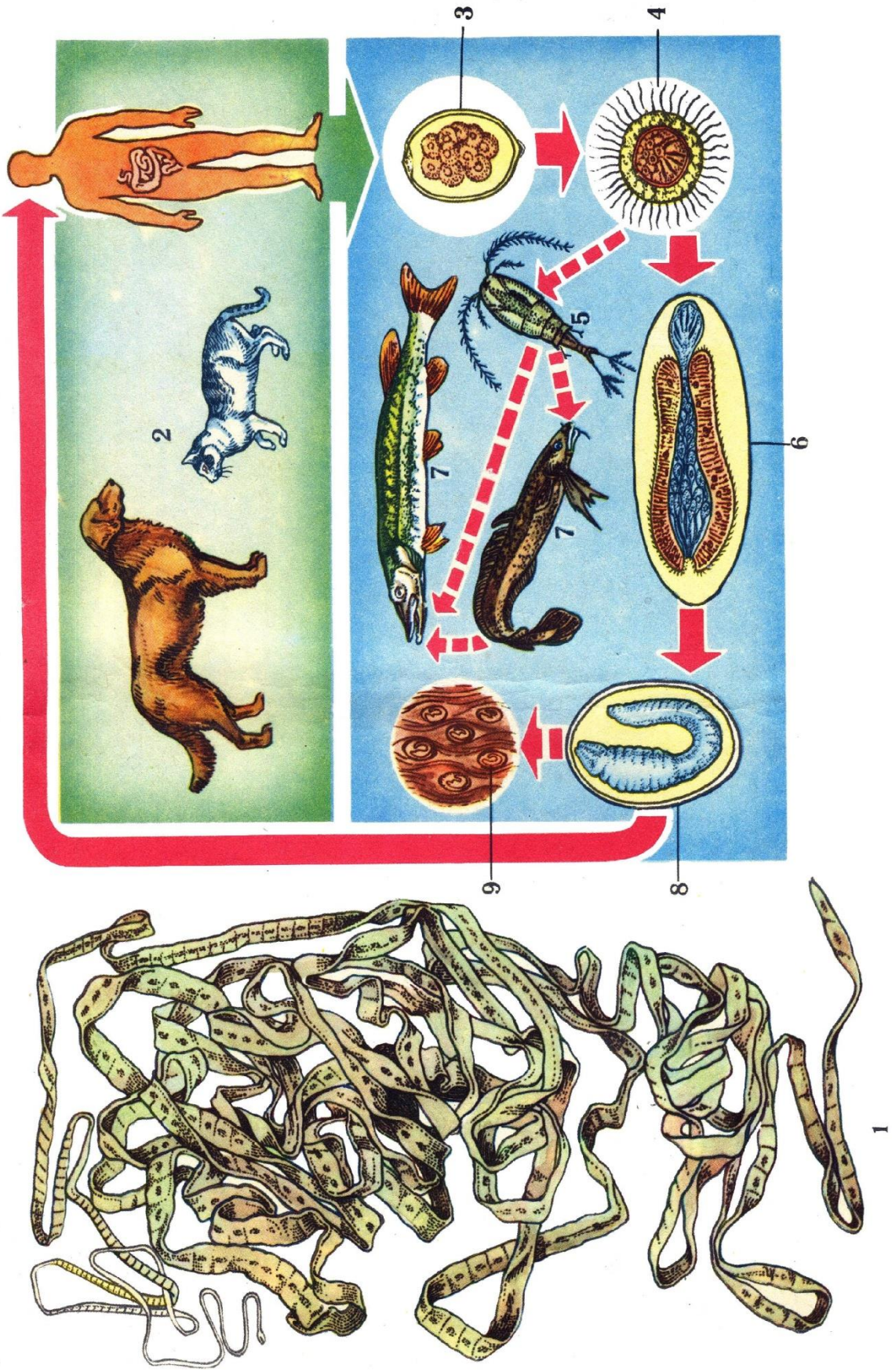
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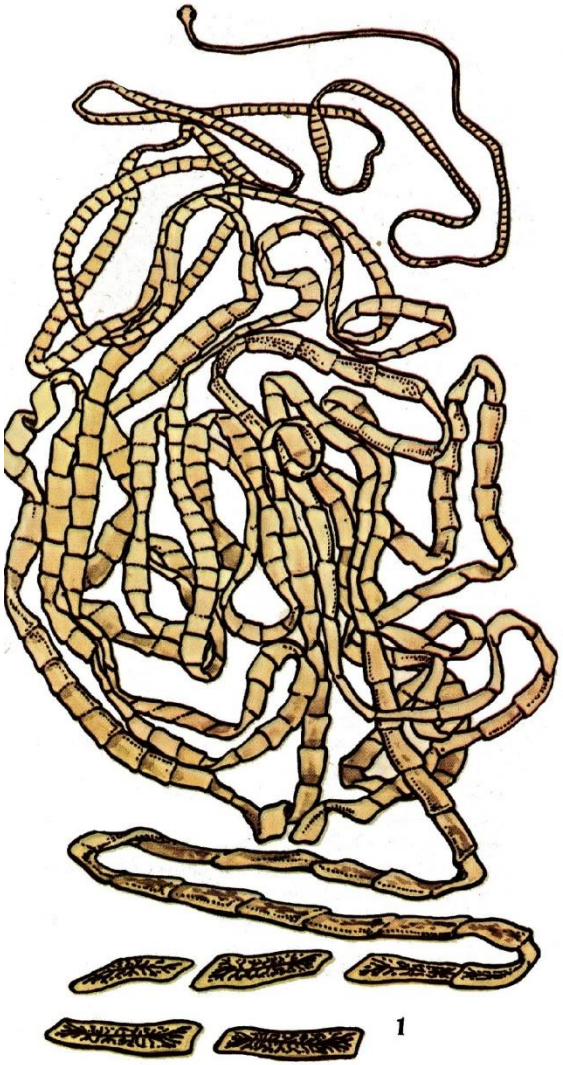
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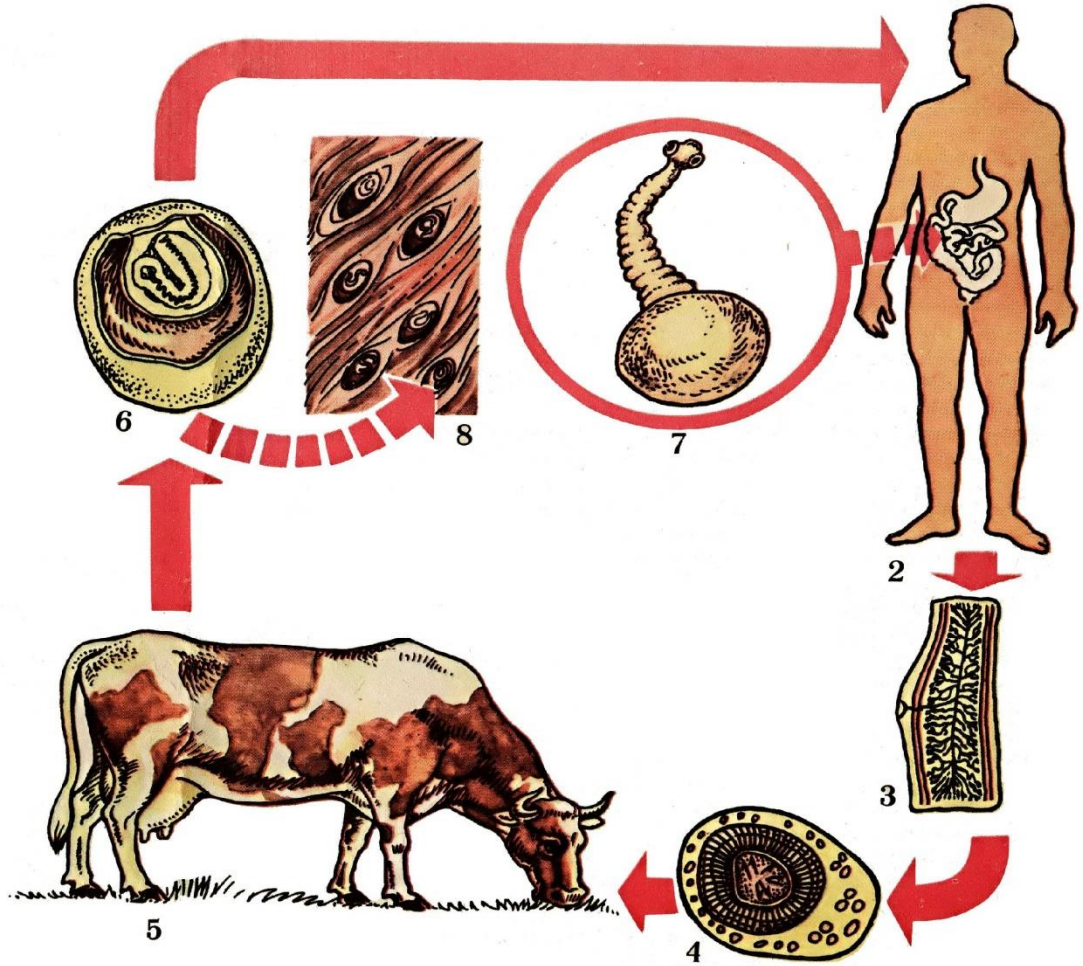
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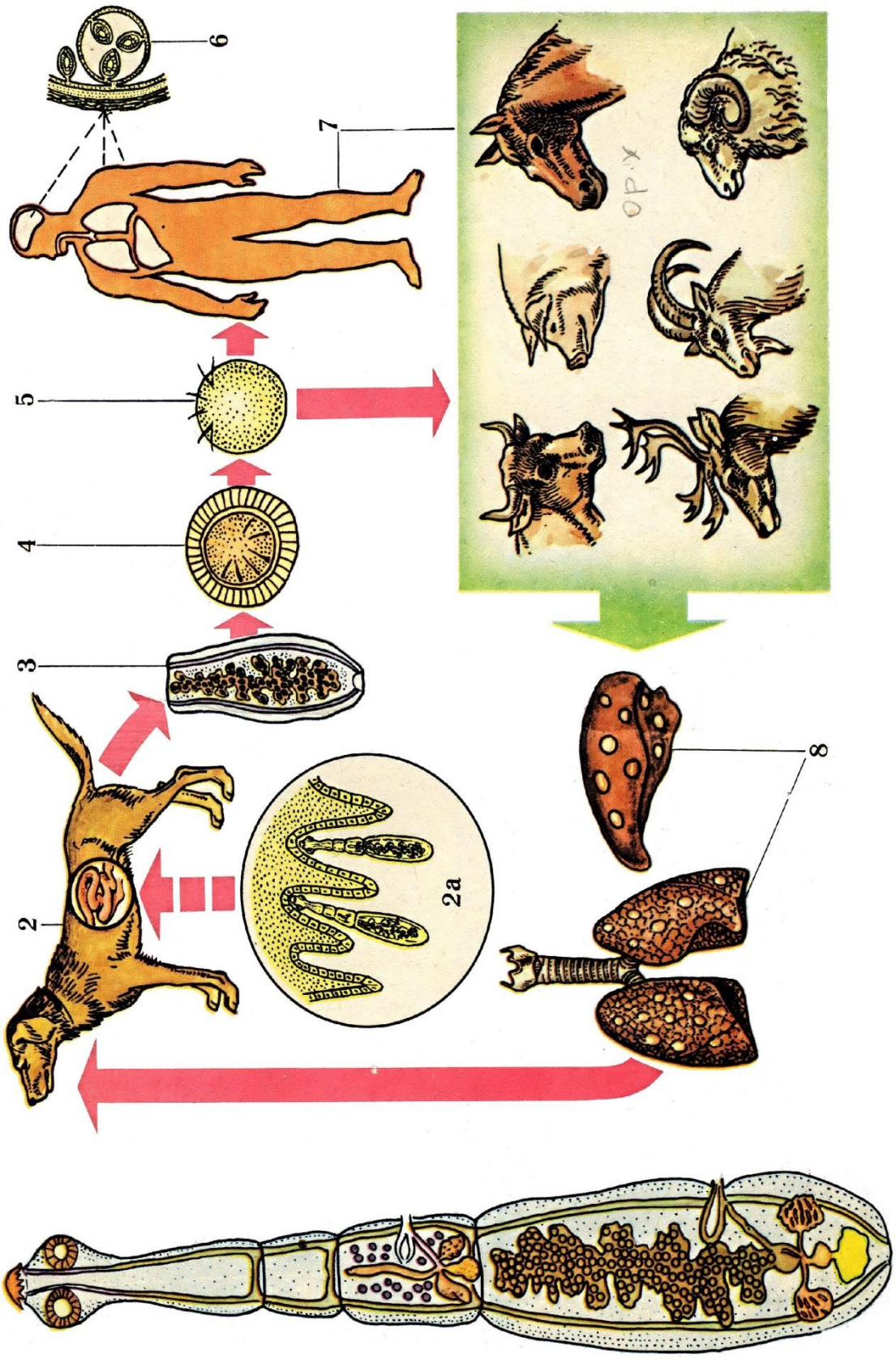
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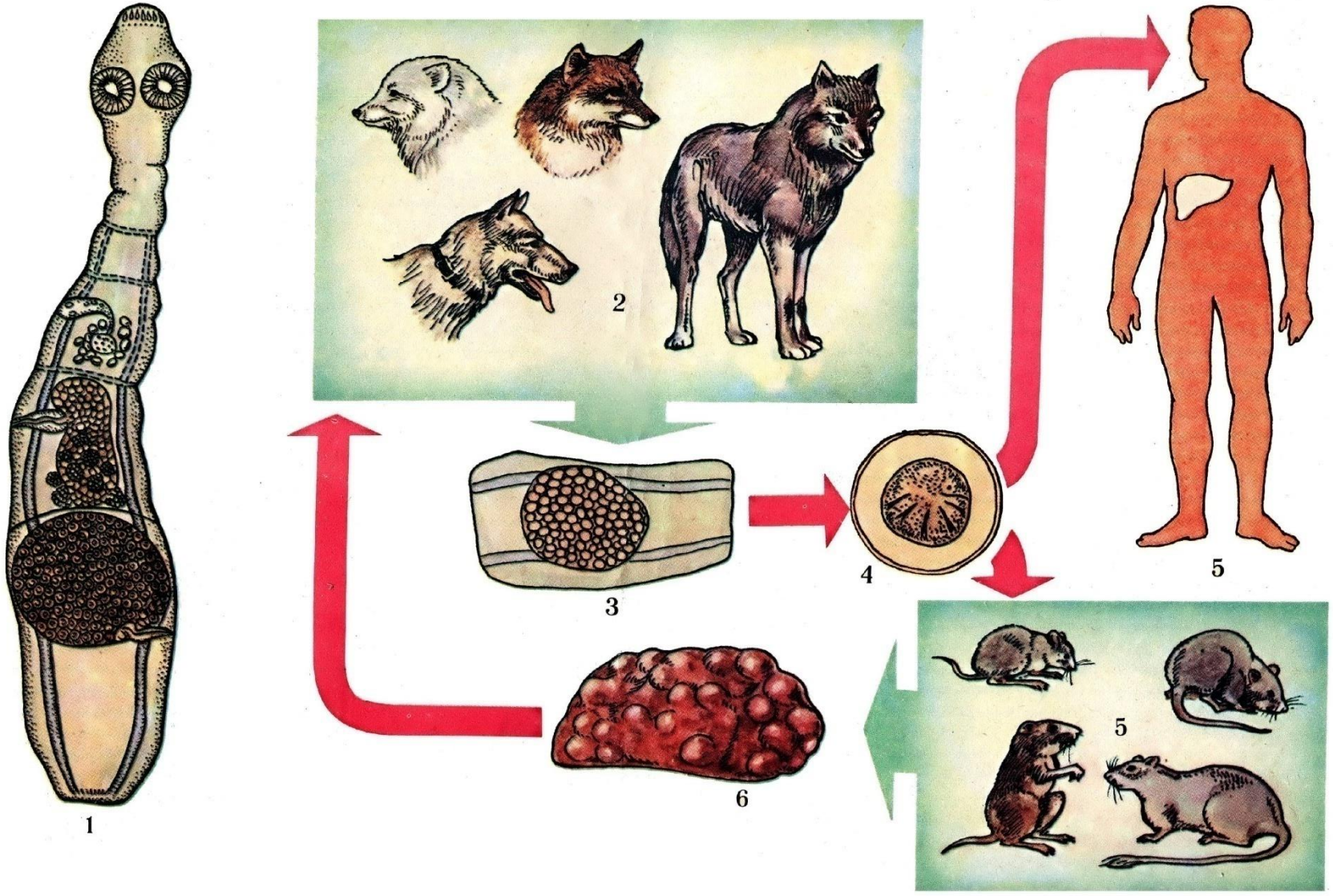
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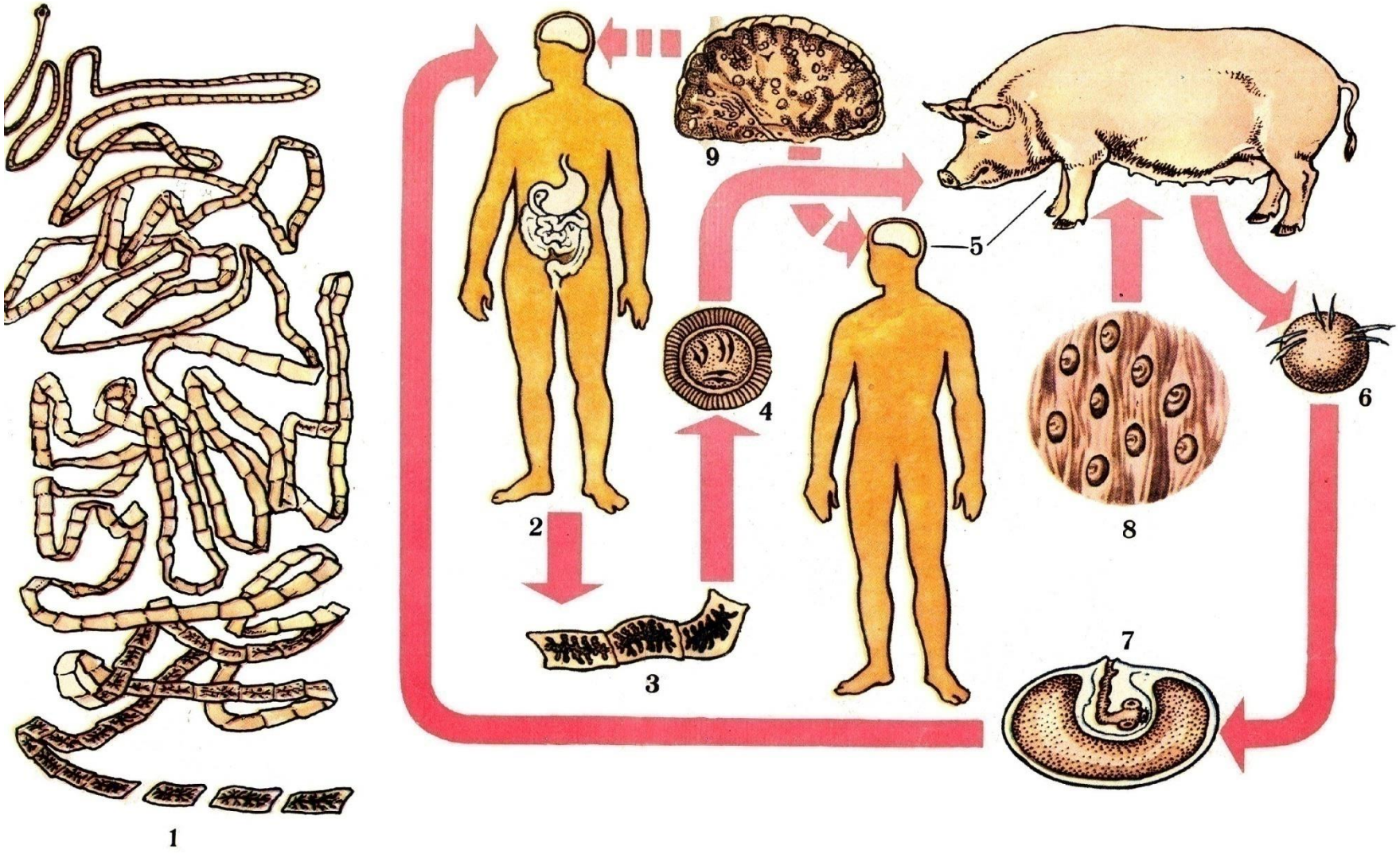
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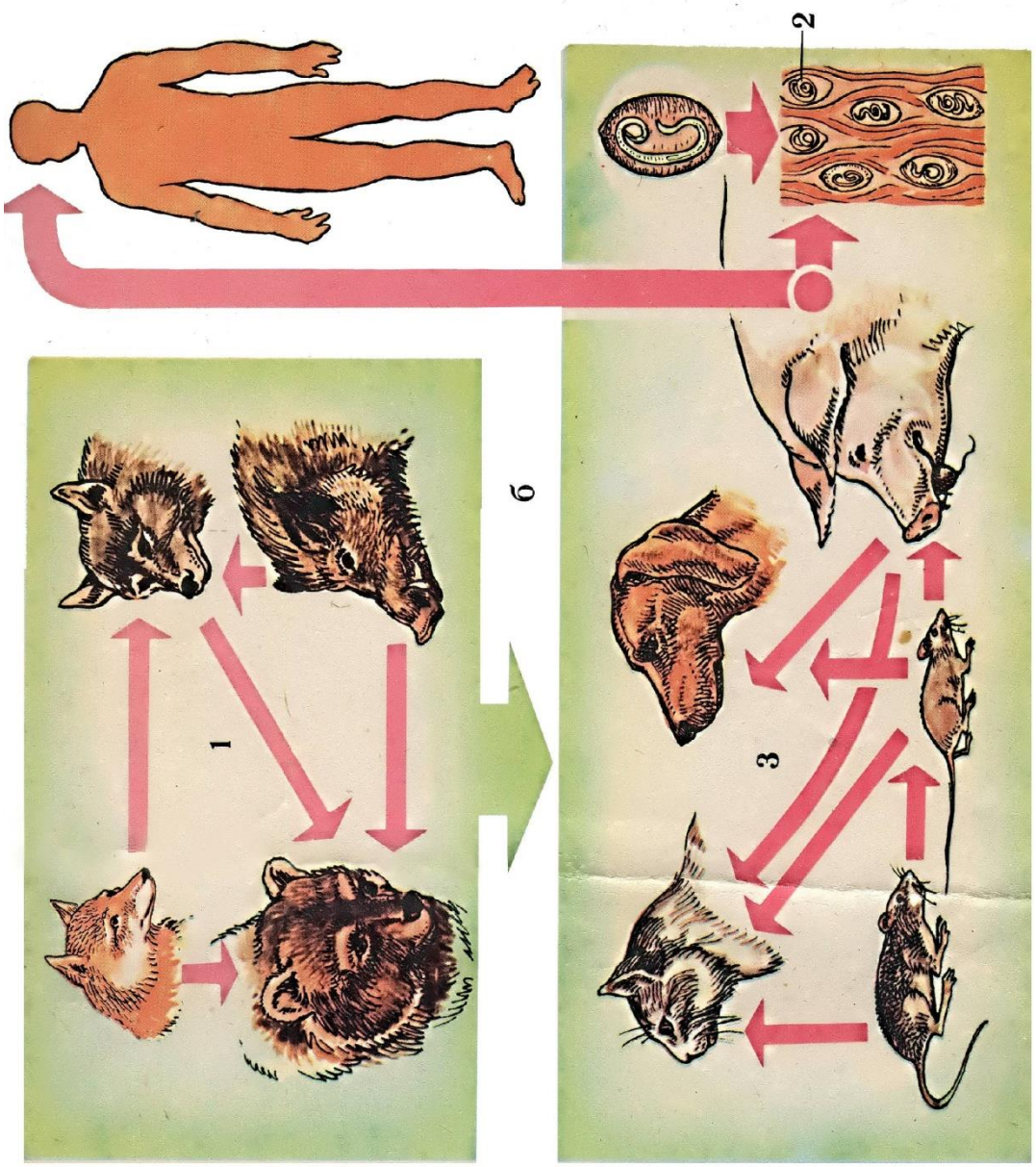
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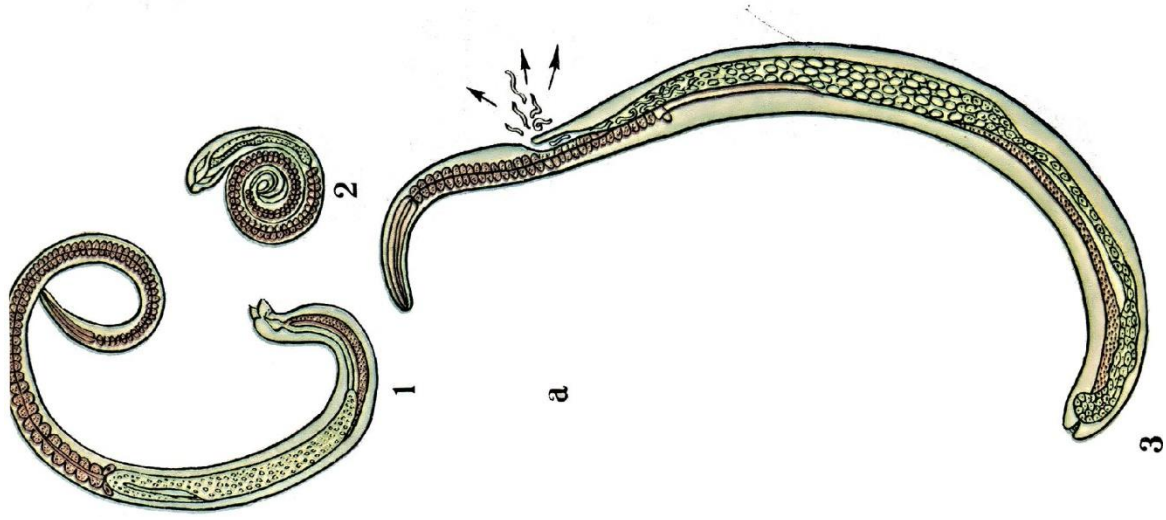
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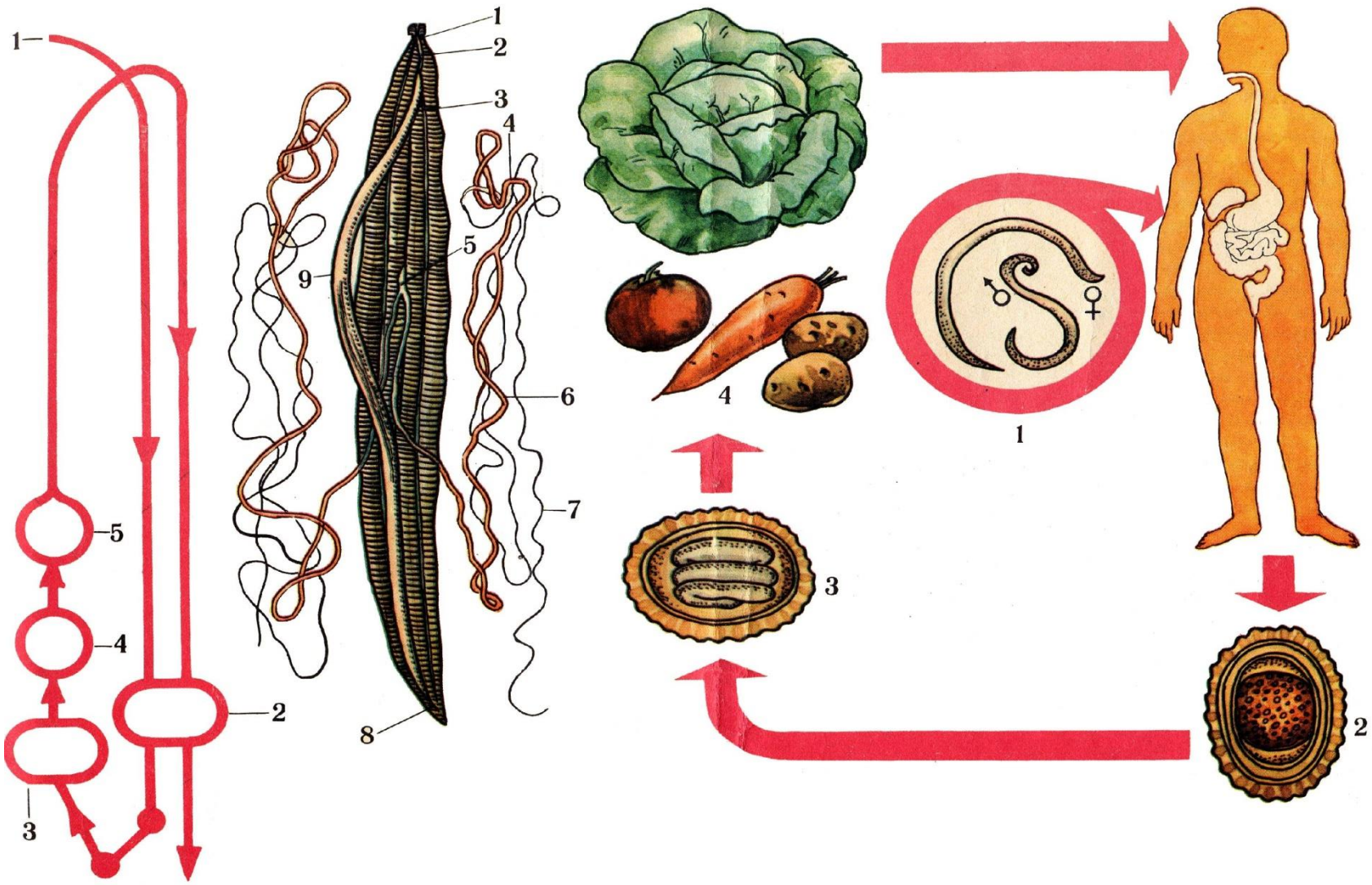
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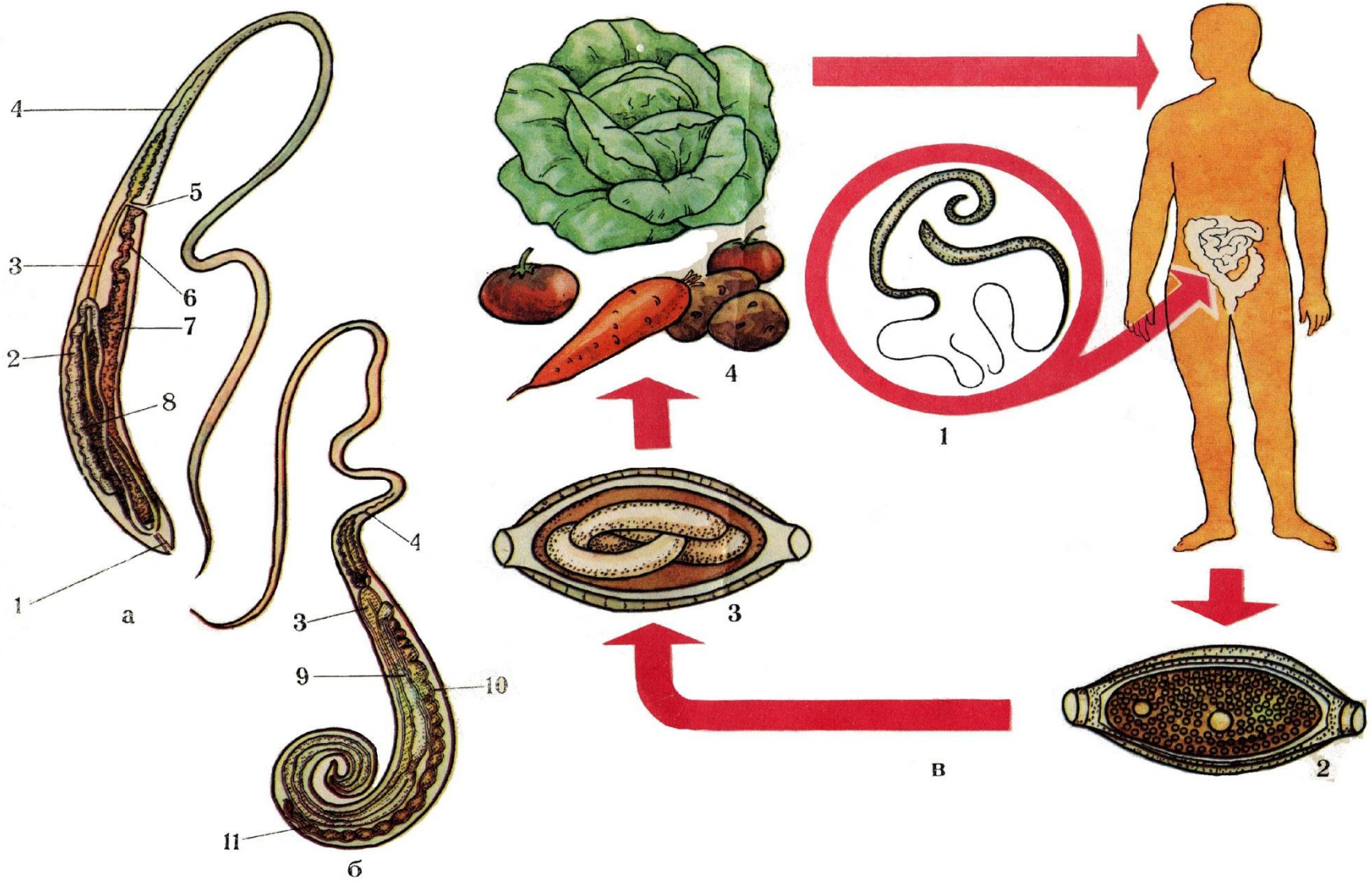
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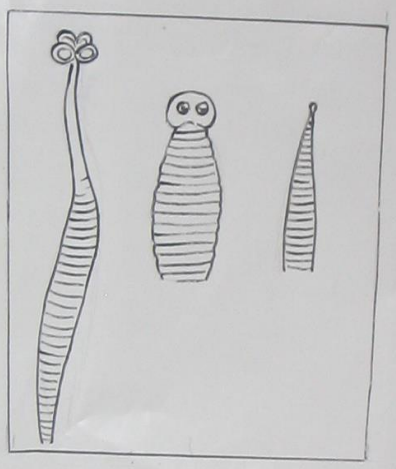

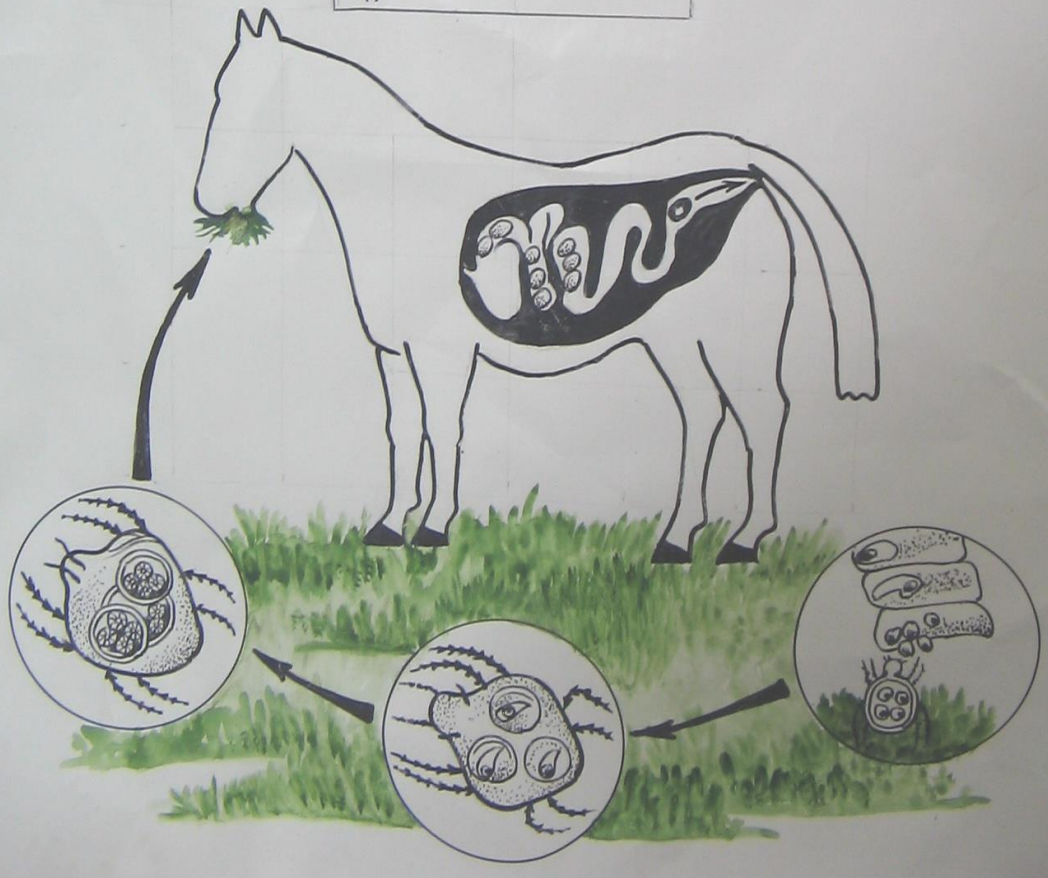

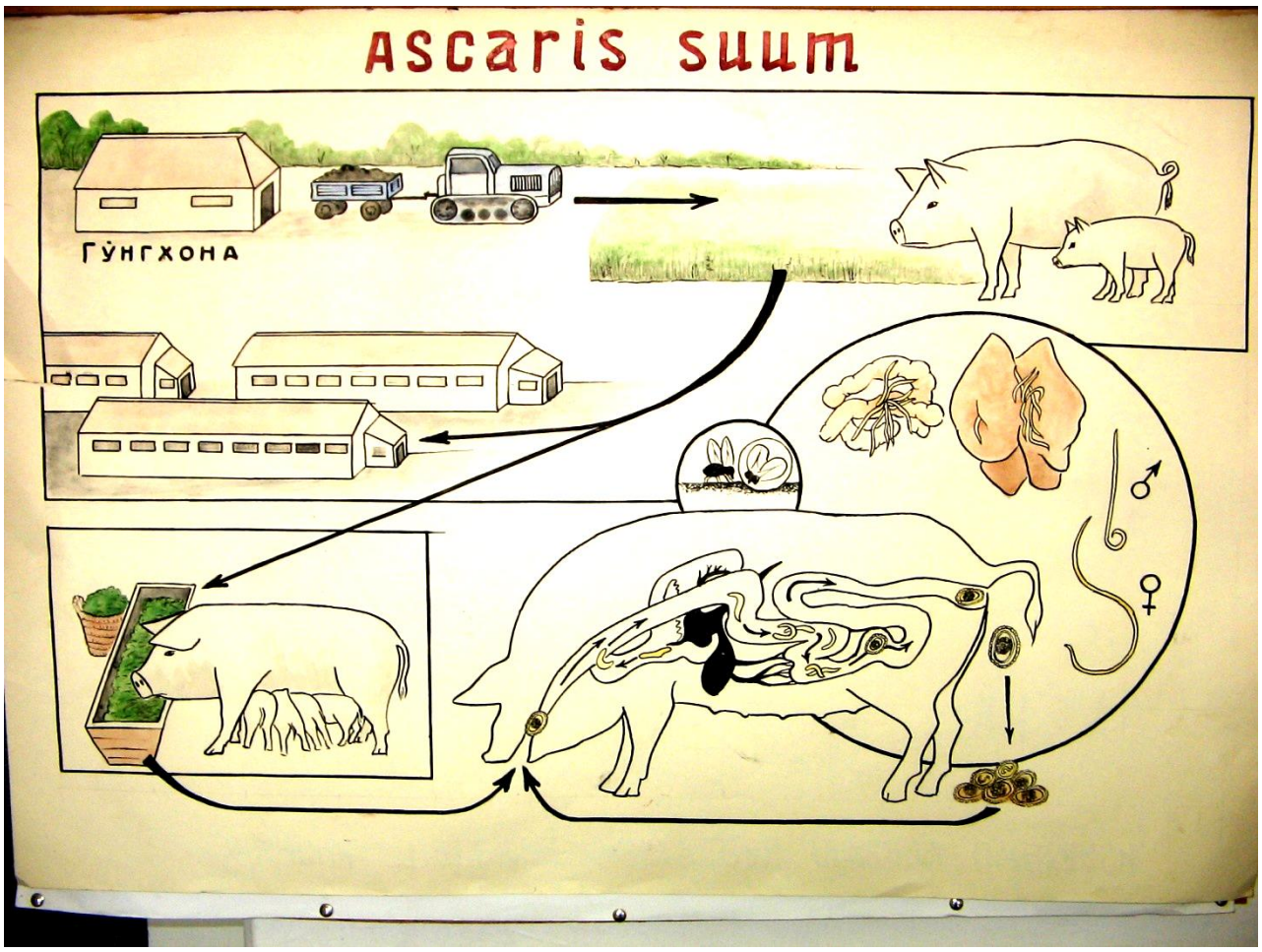
Ascaris suum
Гўнгхона
♂
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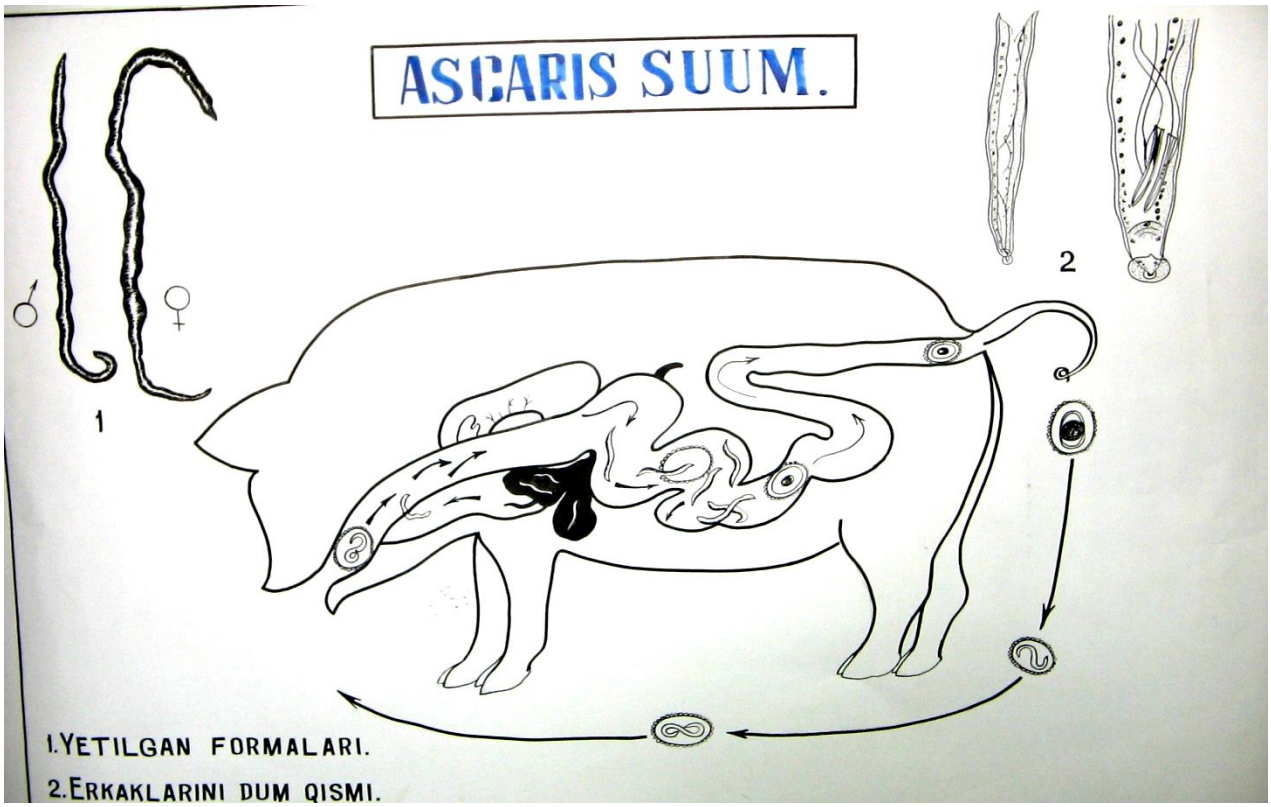
ASCARIS SUUM.
♂
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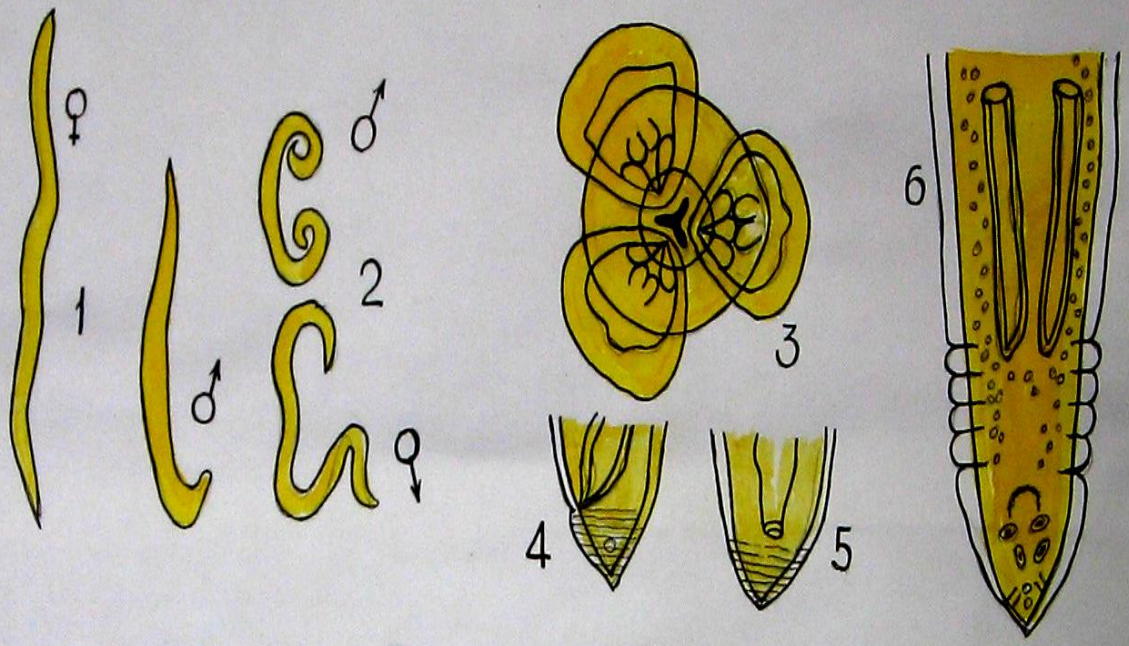
1.YETILGAN FORMALARI.
2.ERKAKLARINI DUM QISMI.

Parascaris equorum



1-yetilgan va 2-yosh shakllari; 3-Apikal oldingi qismi; 4-♀ning lateral tomondan dumi; 5-♀ning ventral tomondan dumi; 6-♂ning ventral tomondan dum qismi. (Yershov bo'yicha)

TOY ICHAGINING PARASKARIDLAR BILAN YORILISHI.

TIPI - Nemathelminthes

SINFI - Nematoda

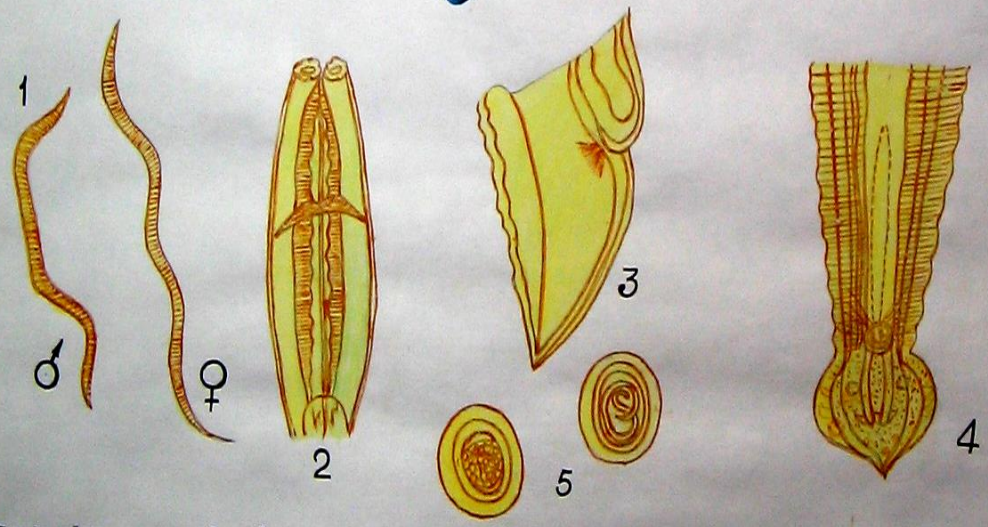
K/T - Ascaridata

OILA - Ascaridae

AVLODI - Ascaridia

TURI - A. galli ♀ ♂

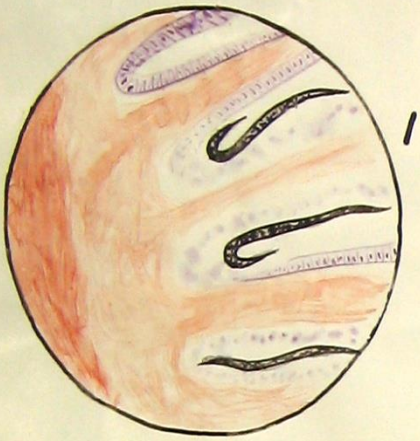
Ascaridia galli



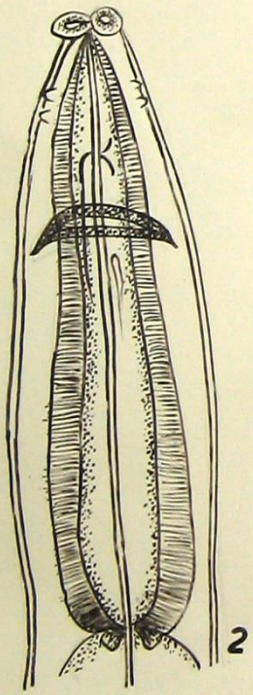
1- Erkak va urg̃ochi; 2- Paraziṭning oldingi qismi; 3- Urg̃ochining dumi; 4- Erkagining dumi; 5- Tuxum har xil rivojlanish bosqichida.

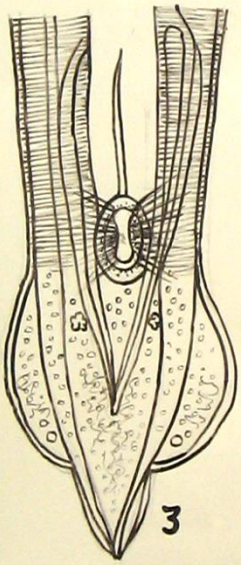
sog̃lom (A) va askaridiyalar bilan zararlangan jo'jalar.

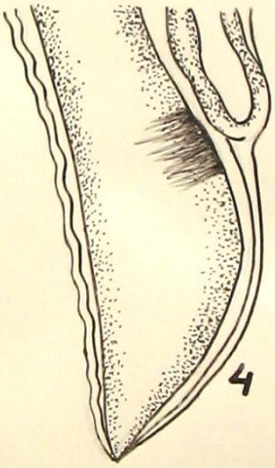
Ascaridia galli

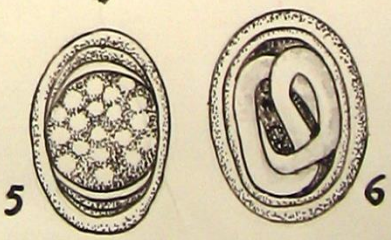


1- Zararlanishtirishdan 6 kun õtgach ascaridiy lichinkalarni Liberkyun bezlari ichidagi kõrinishi (Bez epiteliyasining deskvamasiyasi va buzilishi);

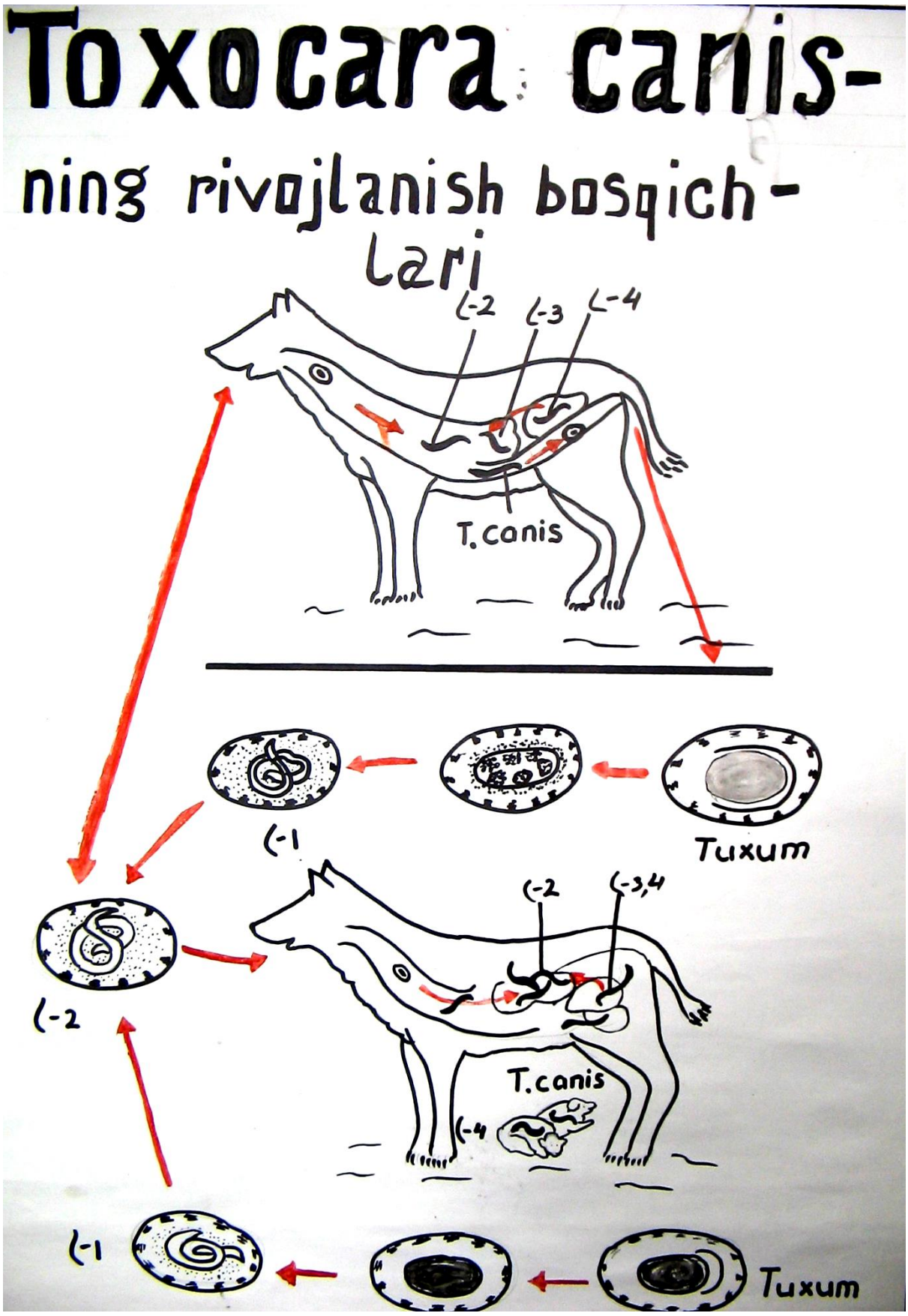








2 - Boshi; 3 - Erkagining dum qismi; 4 - Urgochisining dum qismi; 5 - Invaziyalanmagan tuxum; 6 - Invaziyalangan tuxum.


Toxocara canis-
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T. canis
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Tuxum
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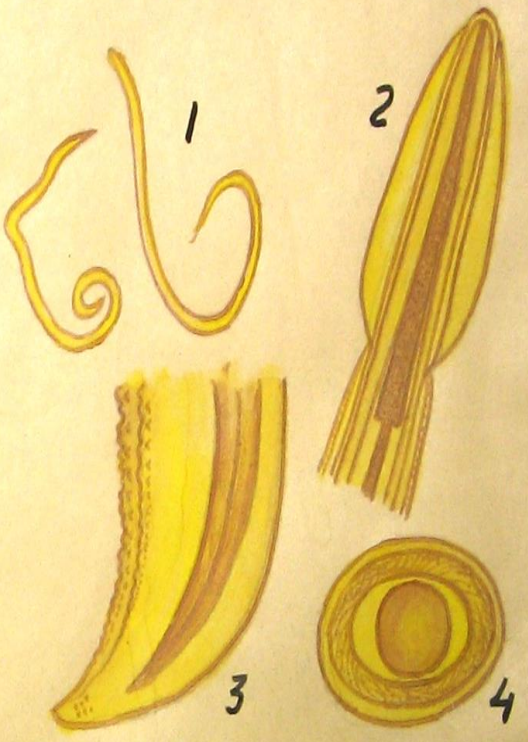
L-2
L-3,4
T. canis
L-4
L-1
Tuxum

Toxocara canis

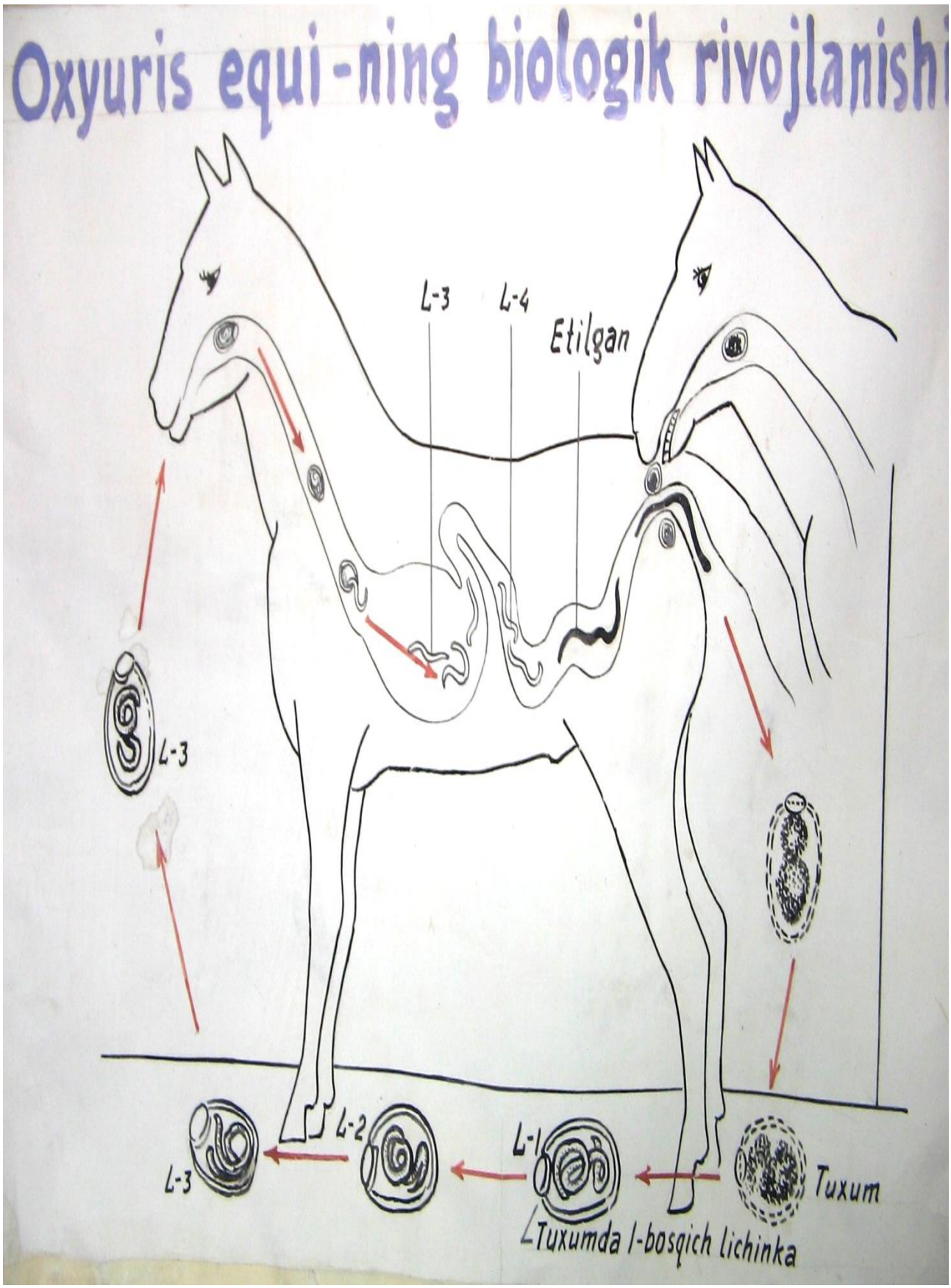
1-Лаб тузилиши; 2-Ургочининг думи; 3-Эркакнинг думи; 4-тухум.

Toxascaris leonina



1- Эркак ва ургочи;
2- Паразитнинг олдинги қисми; 3- Эркакнинг думи.

Ўн икки бармоқли ичакнинг ёрилиши


Oxyuris equi-ning biologik rivojlanishi
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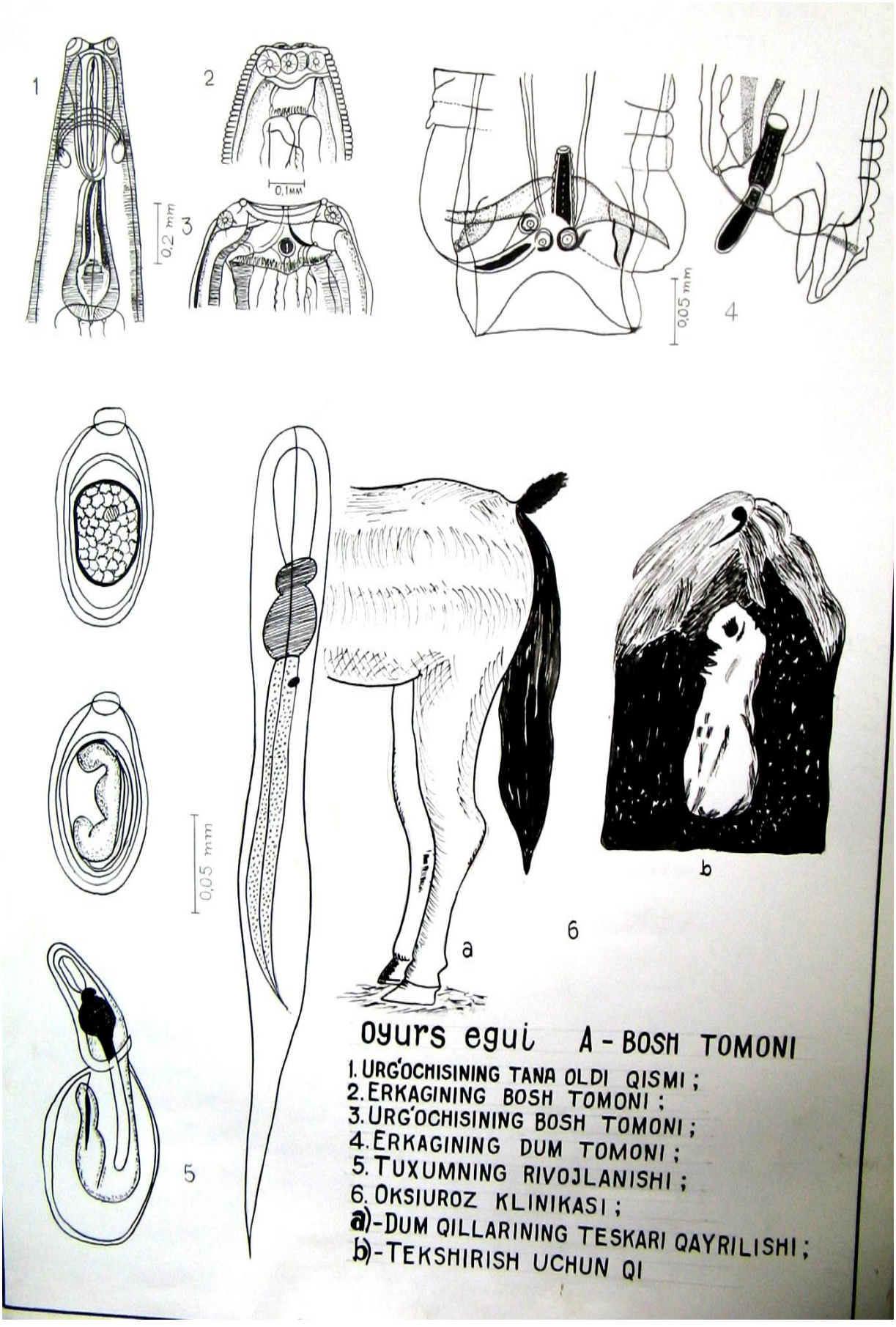
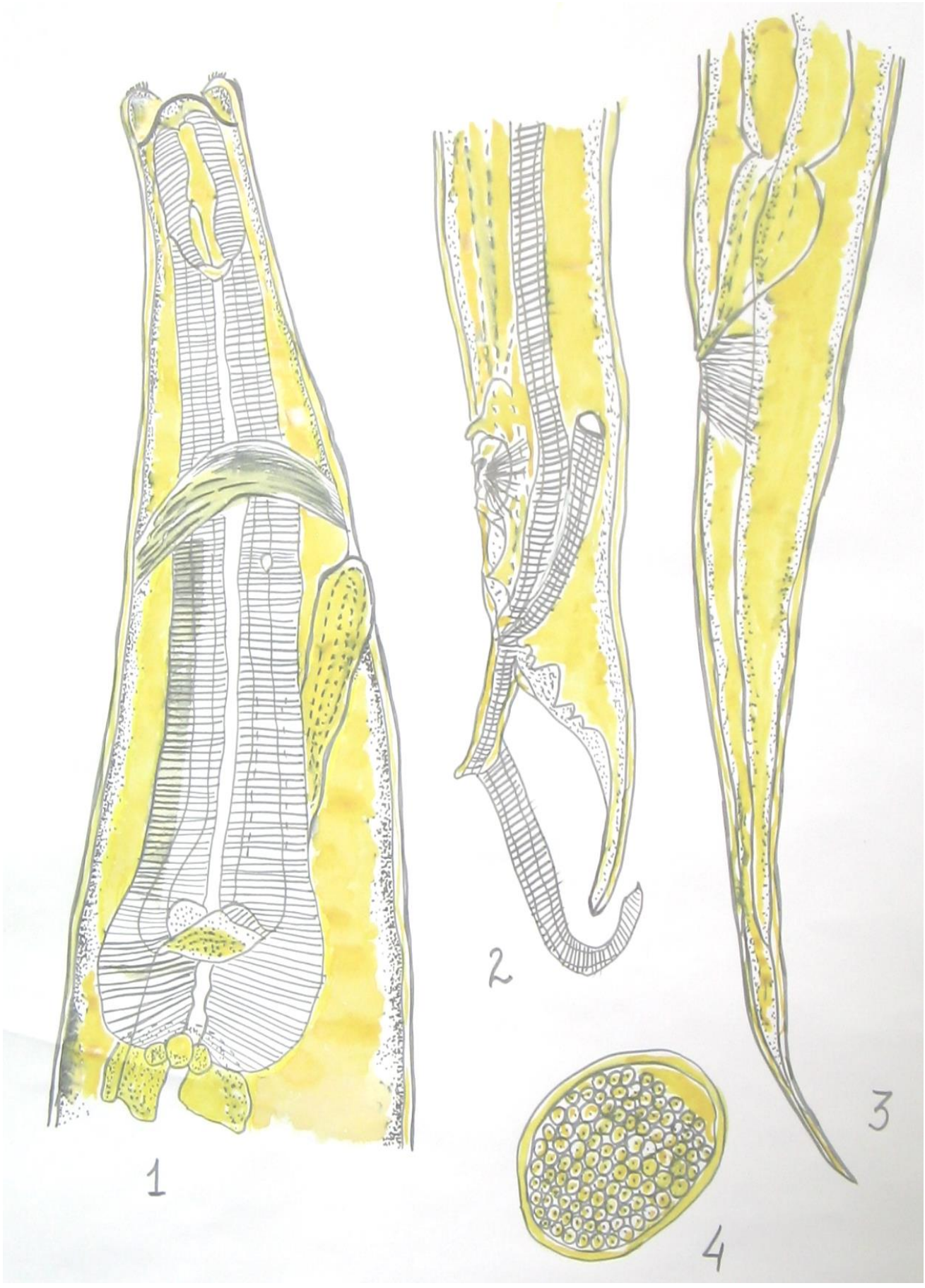
Tuxum
Tuxumda I-bosqich lichinka



Oyurs egui A - BOSH TOMONI

1. URG'OCHISINING TANA OLDI QISMI;
2. ERKAGINING BOSH TOMONI;
3. URG'OCHISINING BOSH TOMONI;
4. ERKAGINING DUM TOMONI;
5. TUXUMNING RIVOJLANISHI;
6. OKSIUROZ KLINIKASI;
a)-DUM QILLARINING TESKARI QAYRILISHI;
b)-TEKSHIRISH UCHUN QI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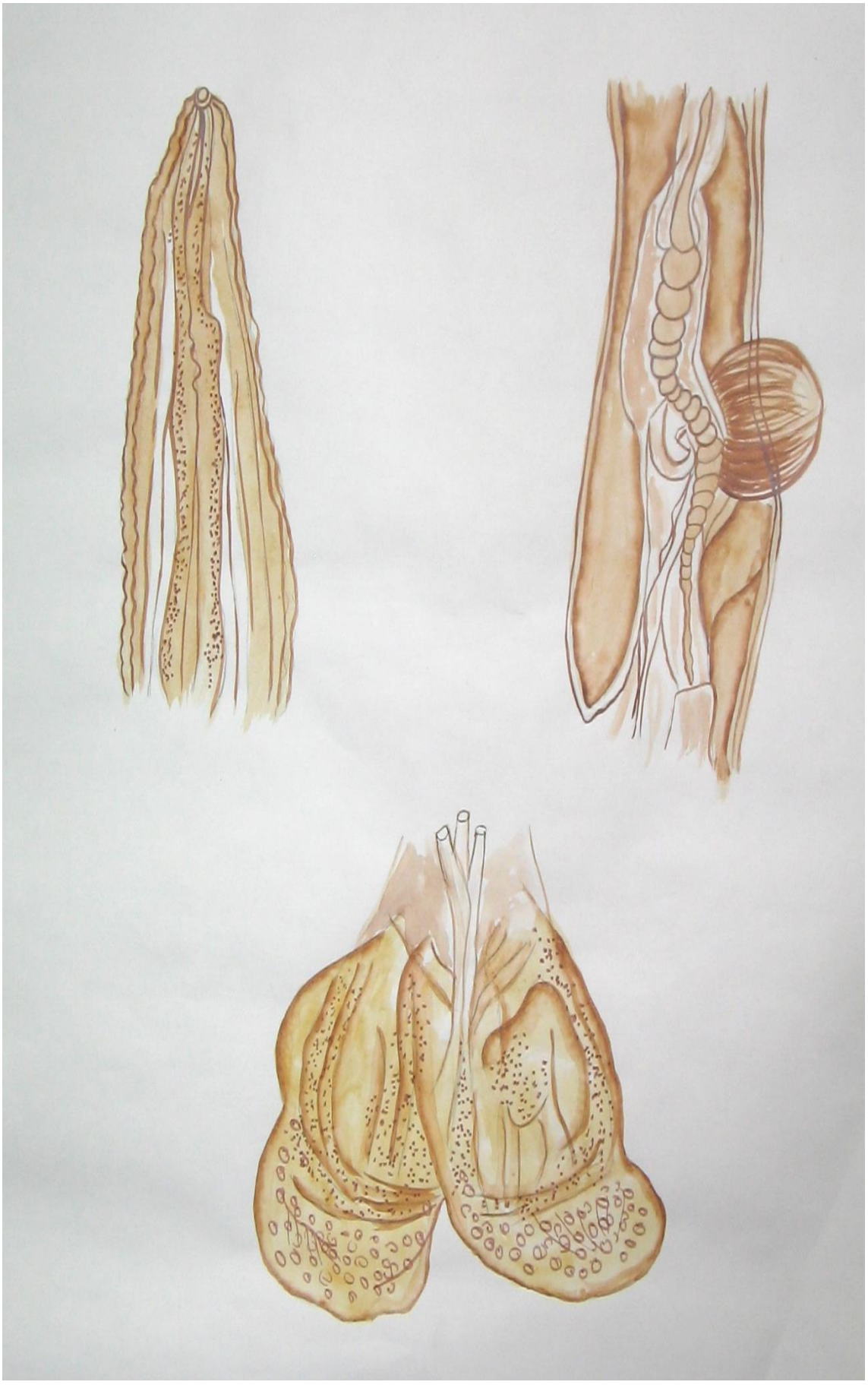
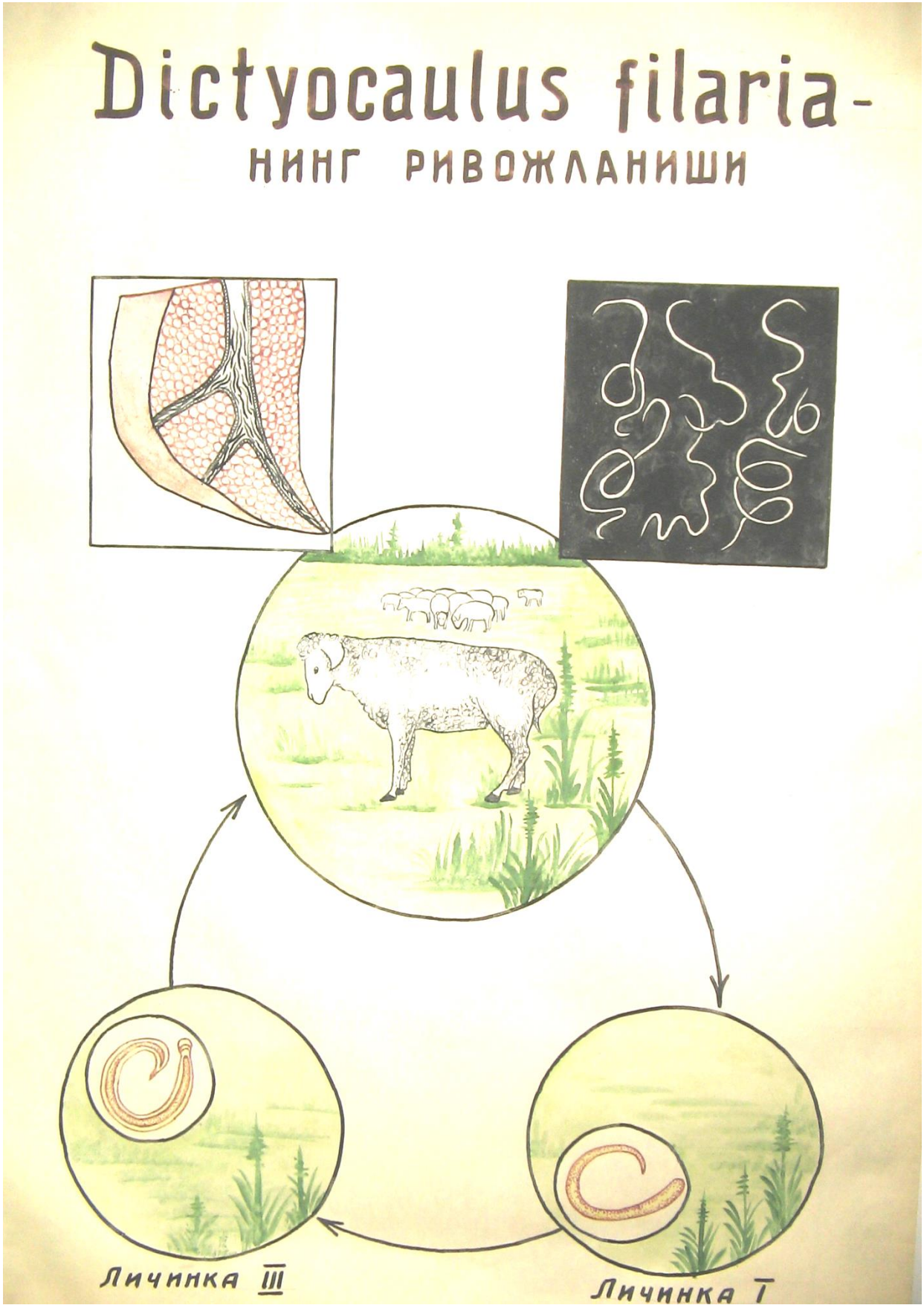
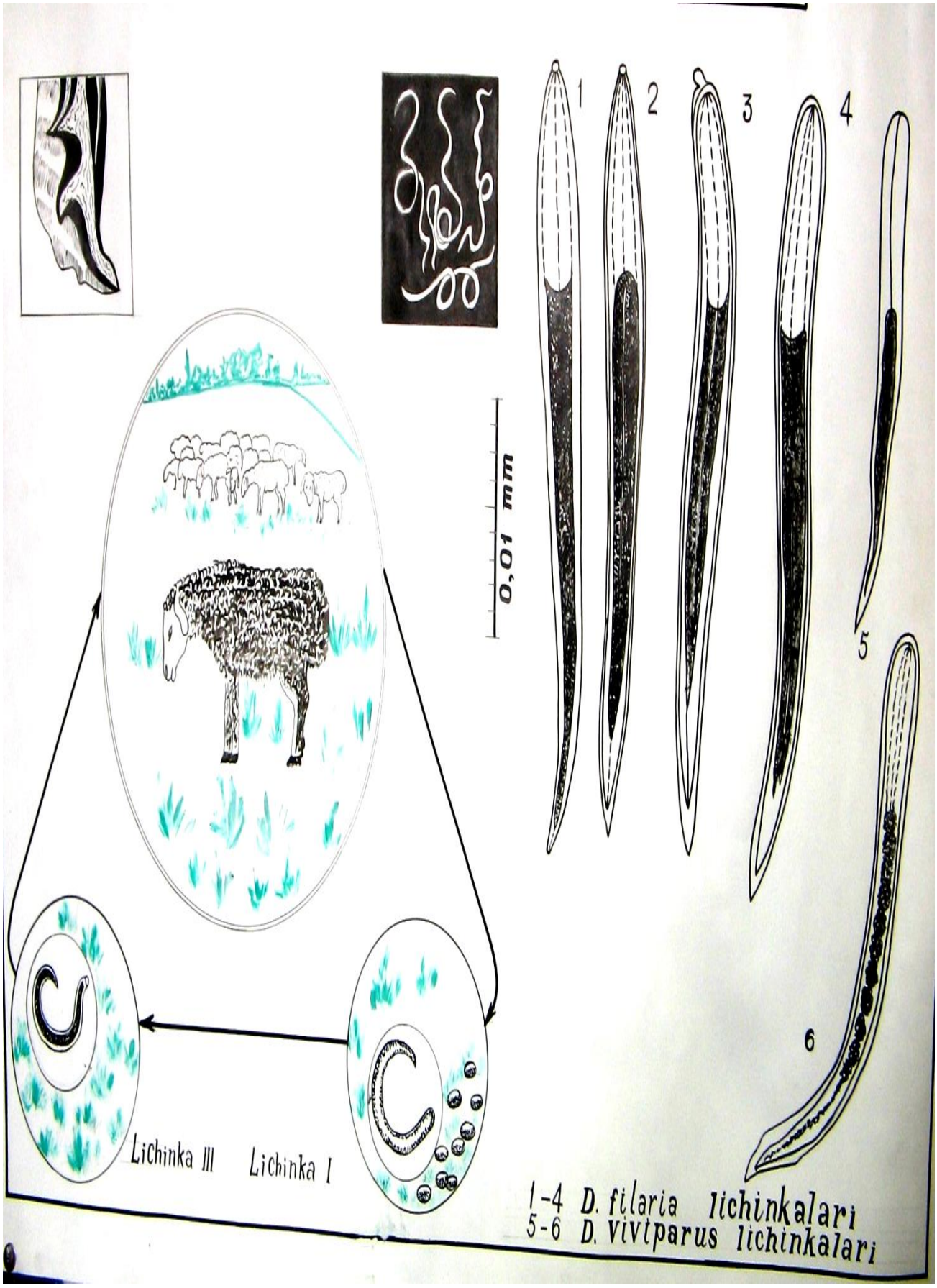
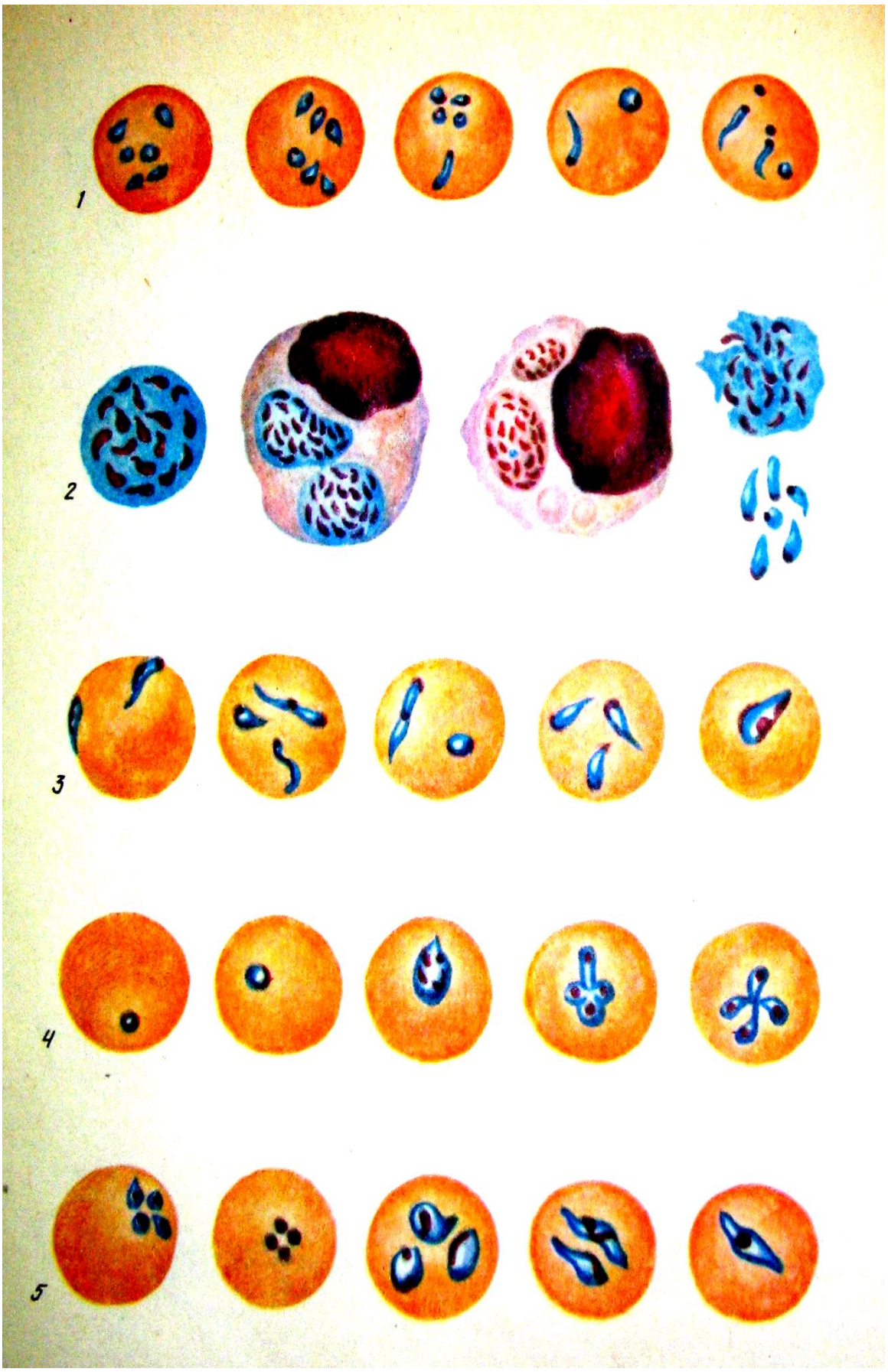
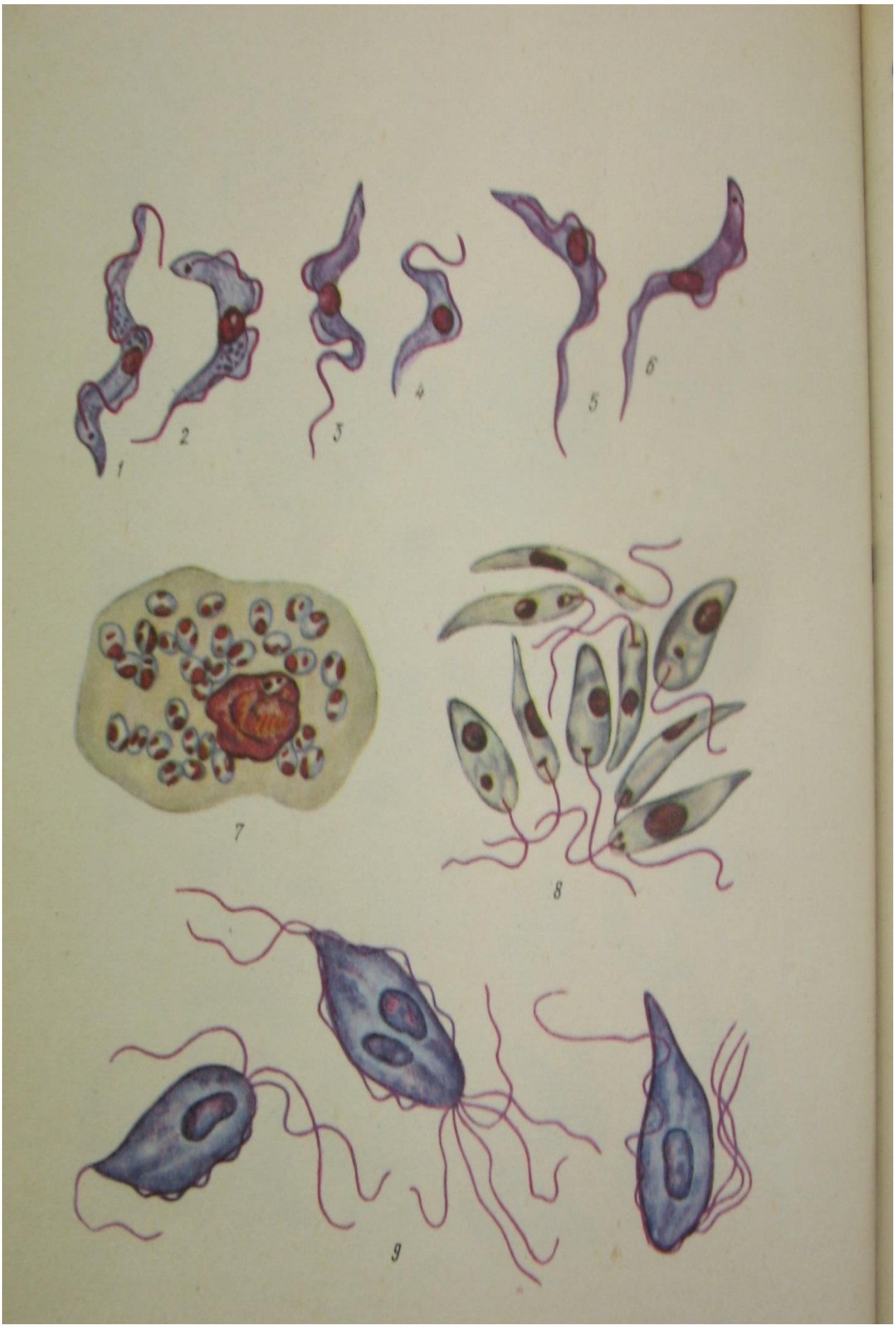



Dictyocaulus filaria-
НИНГ РИВОЖЛАНИШИ
Личинка III
Личинка I

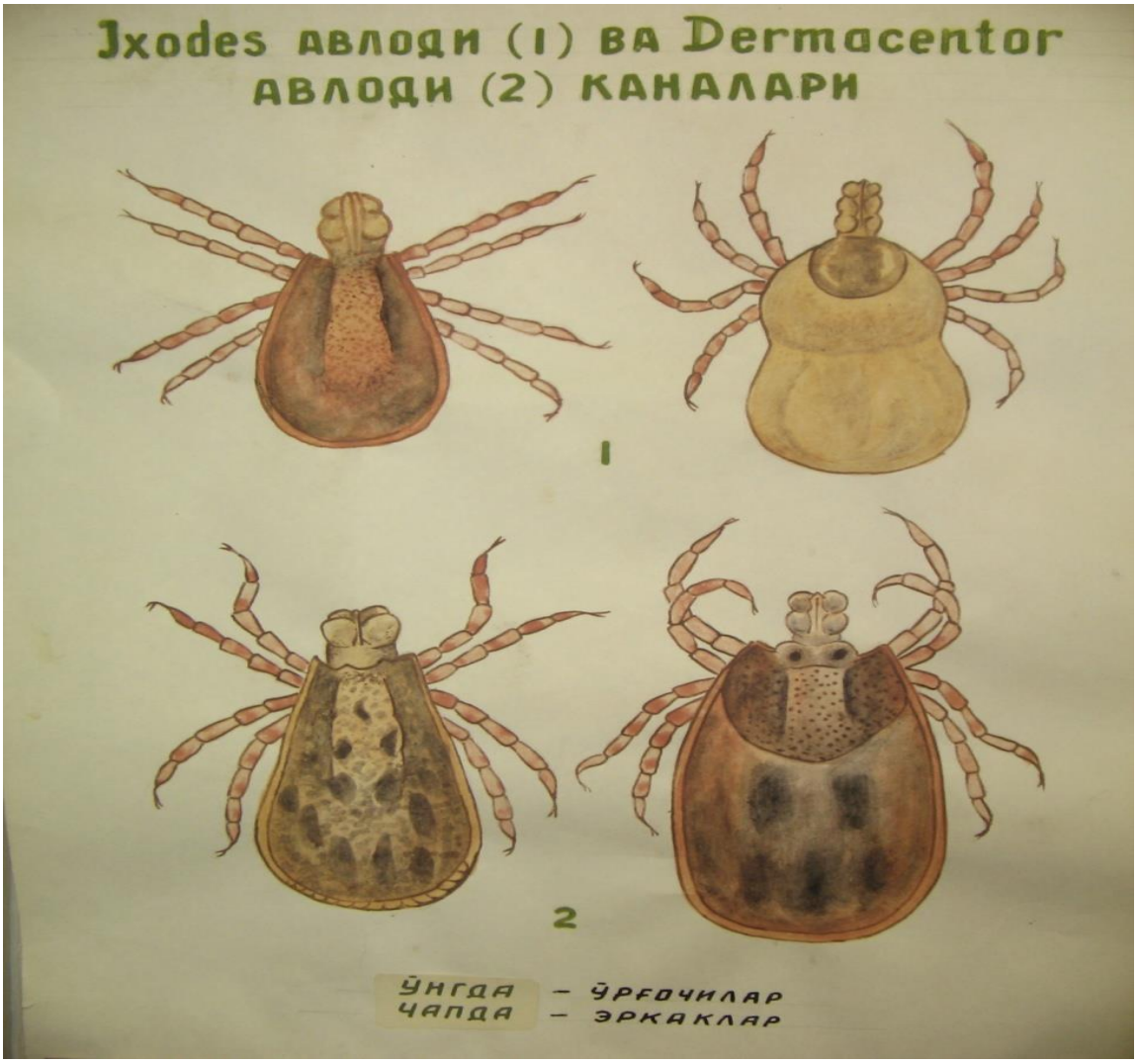

1
2
3
4
5
6
0,01 mm
Lichinka III
Lichinka I
1-4 D. filaria lichinkalari
5-6 D. vivtparus lichinkalari


TRIXOSEFALOLARNING
(QILBOSH) RIVOJLANISH
SXEMASI.
CHO'CHQA TRIXOSEFALYOZI.
55-60°


1
2
3
4
5

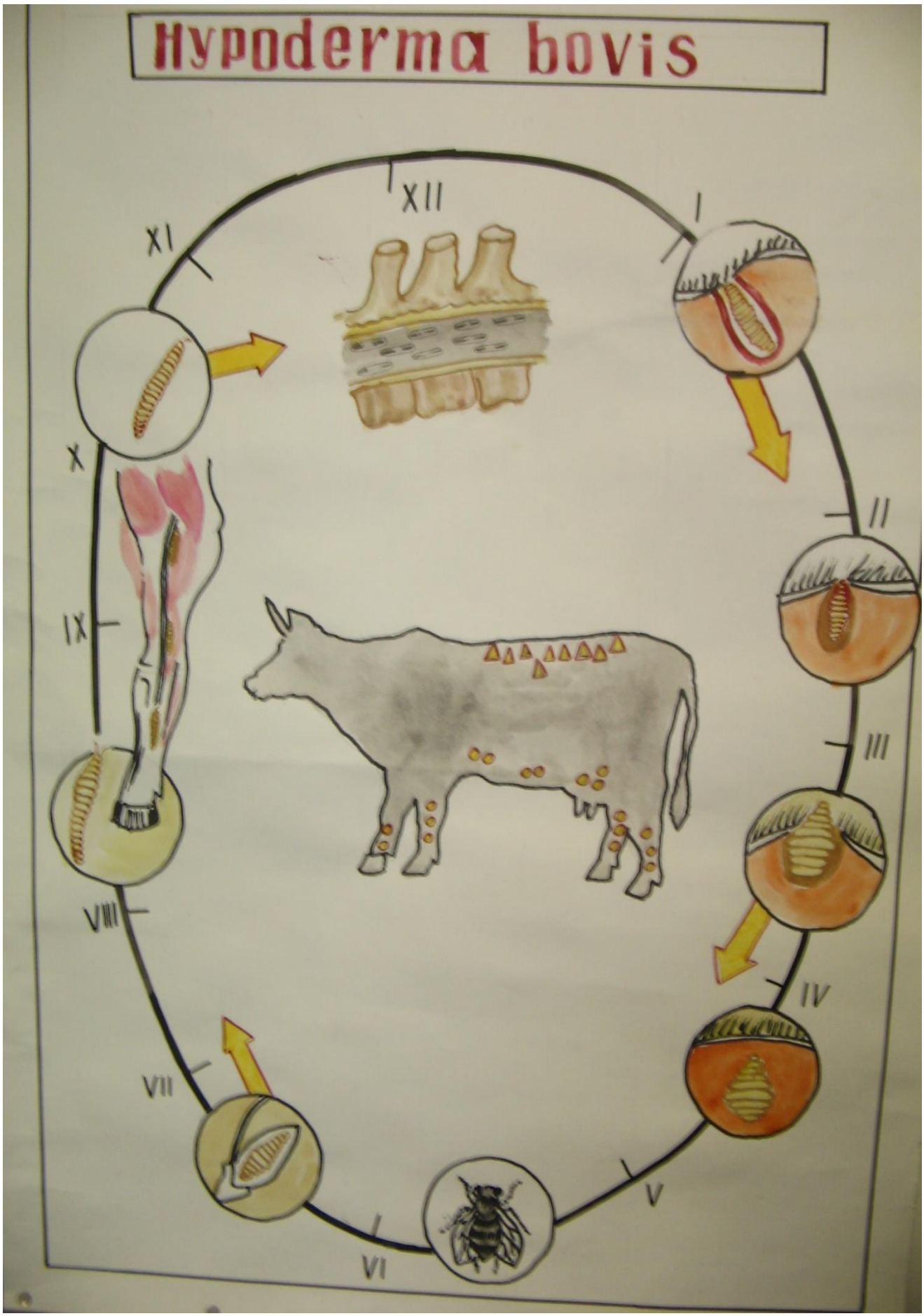

1
2
3
4
5
6
7
8
9

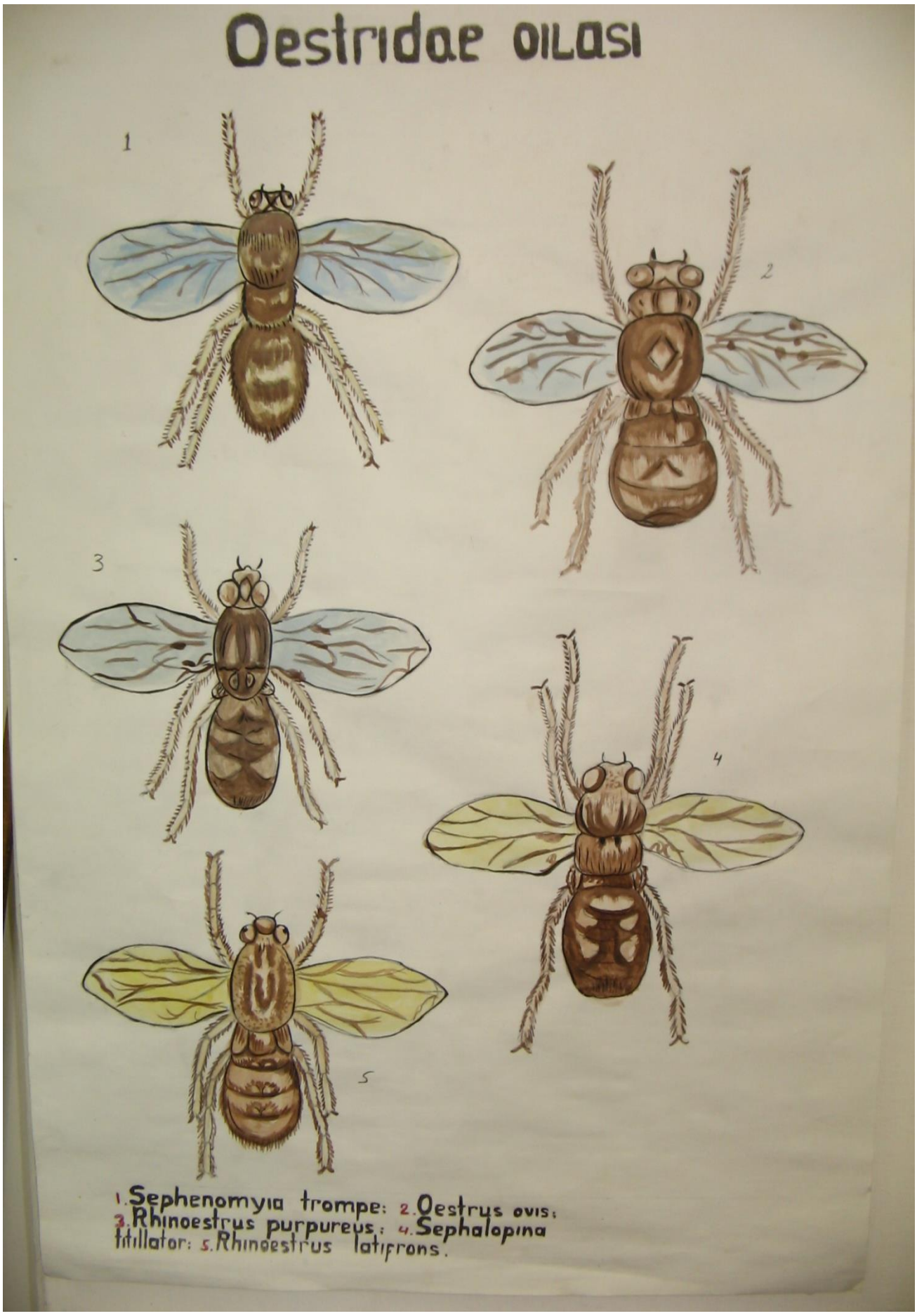

OTLARNING QOCHIRUV KASALLIGI


Ixodes АВЛОДИ (1) ВА Dermacentor
АВЛОДИ (2) КАНАЛАРИ
1
2
ЎНГДА – ЎРҒОЧИЛАР
ЧАПДА – ЭРКАКЛАР


Argas persicus-
ning rivojlanish bosqichlari
1-Tuxumlar
2-Lichinkasi
3-Nimfalar
4-Voyaga etgan kanalar


ҚЎЙ ПСОРОПТОЗИ (А.А ЦАПРУН БЎЙИЧА)


Hypoderma bovis
XII
I
II
III
IV
V
VI
VII
VIII
IX
X
XI


Oestridae oilasi
1
2
3
4
5
1.Sephenomyia trompe: 2.Oestrus ovis:
3.Rhinoestrus purpureus: 4.Sephalopina
titillator: 5.Rhinoestrus latifrons.

Rhinoestrus purpureus

1. Urg'ochi bo'kalar uchayotib tirik lichinkalarni burun teshigiga sepadi; 2-Bosh bo'shlig'larida 9-10 oy davomida lichinkalar yashaydi; 3-lichinka I; 4-lichinka III; 5-Yetilgan lichinkalarni tashqi muhitga tushishi; 6-Tuproqda lichinkalar 3-4 kunda g'umbakka aylanadi; 7-30-45 kundan so'ng g'umbakdan qanotli bo'kalar uchib chiqadi; 8-Urg'ochilarning boshi.


Gastrophilidae ОНЛАСН
1
2
3
4
1-Gastrophilus veterinus; 2-G. pecorum; 3-Gintestinalis; 4-G. haemorroidalis.

VII. Electronic version of the educational and methodical complex on the subject