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FEDERAL STATE BUDGETARY EDUCATIONAL INSTITUTION
OF HIGHER EDUCATION
"BASHKIR STATE MEDICAL UNIVERSITY"
OF THE MINISTRY OF HEALTHCARE OF THE RUSSIAN FEDERATION
(FSBEI HE BSMU MOH Russia)

PARASITOLOGY

Textbook

Edited by professor T.V. Viktorova

Ufa – 2020

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CONTENTS

ВВЕДЕНИЕ

В учебном пособии представлена общая характеристика паразитарных инвазий как биотических факторов экологических систем, воздействующих на организм человека. Рассматриваются вопросы общей организации простейших, гельминтов и членистоногих, имеющих немаловажное значение для нормальной жизнедеятельности и здоровья человека. В краткой форме, максимально адаптированной для понимания обучающимися первого курса, изложены вопросы частной паразитологии: систематическое положение, особенности морфологии, циклы развития наиболее значимых паразитов человека. Рассматриваются некоторые аспекты паразитарных заболеваний, в частности, вопросы патогенеза, симптоматики, лабораторной диагностики и профилактики. Пособие содержит достаточный иллюстративный материал. Примерные оценочные средства (контрольные вопросы, тесты и ситуационные задачи), предназначенные для проверки полученных знаний, помогут лучше усвоить теоретический материал и сформировать следующие общепрофессиональные компетенции:

ОПК-1 – готовность решать стандартные задачи профессиональной деятельности с использованием информационных, библиографических ресурсов, медико-биологической терминологии, информационно-коммуникационных технологий и учетом основных требований информационной безопасности.

ОПК-7 – готовность к использованию основных физико-химических, математических и иных естественно-научных понятий и методов при решении профессиональных задач.

INTRODUCTION

The training manual presents a general description of parasitic infestations as biotic factors of ecological systems that affect the human body. The issues of the general organization of protozoa, helminthes and arthropods, which are of no small importance for normal life and human health, are considered. The issues of private parasitology are described in a short form, which is most adapted for understanding by first-year students: systematic position, morphological features, development cycles of the most significant human parasites. Some aspects of parasitic diseases are considered, in particular, the issues of pathogenesis, symptoms, laboratory diagnosis and prevention. The manual contains sufficient illustrative material. Sample assessment tools (control questions, tests and situational tasks) designed to test the knowledge gained will help to better understand the theoretical material and build the general professional competency of GPC-1 and GPC-7.

GPC-1: the ability to solve the standard tasks of professional activity using information, bibliographic resources, biomedical terminology, information and communication technologies and taking into account the basic requirements of information security.

GPC-7: the ability to use the basic physical, chemical, mathematical and other natural science concepts and methods in solving professional problems.

CHAPTER 1
PARASITISM AS AN ENVIRONMENTAL PHENOMENON

Parasitism is a widespread phenomenon in nature. The number of species of parasitic animals is about 7 % of the total number of animal species. The first scientific definition of the phenomenon of parasitism was given by R. Leucart in 1879. Parasitism is defined as a form of coexistence of organisms of different species, in which one organism (parasite) uses another (host) as a source of food and environment, rule, harming him, but not destroying him.

The basics of parasitism

A parasite is an organism that finds food and a dwelling in another animal, i.e. it is connected with another organism (host) by spatial and nutritional relationships (R. Leikart, 1879).

A host is an organism that provides the parasite with housing and food.

Great contribution to the development of parasitology of domestic scientists: K.I. Skryabin (opened the first department of parasitology in Russia), E.N. Pavlovsky (developed the doctrine of the natural foci of diseases), V.A. Dogel (created an ecological direction in parasitology), E.I. Martsinovsky (opened the Institute of Parasitology and Tropical Medicine), P.G. Sergiev (proposed measures to combat malaria), etc.

The main directions of development of parasitology of the XXI century. They are the development of biological methods for the prevention of parasitic diseases, the creation of specific prevention, and the development of genetically resistant animals to helminthes. In the future, the development of science will be significantly affected by its four components: biotechnology, bioinformatics, biomathematics and molecular research methods.

Parasitism as a biological phenomenon is studied by the discipline of parasitology. Its section – medical parasitology – studies the structural features and life cycles of parasites, the relationships in the "parasite-host" system, as

well as methods for diagnosing, treating and preventing parasitic diseases. Diseases caused by parasites are called invasions.

Classification of parasites. Depending on the localization in the host organism, ectoparasites (living on the outer surfaces of the host body) and endoparasites (intracellular, interstitial, inside organs and cavities) are distinguished. According to the duration of communication with the host, the parasites can be permanent and temporary, according to the stage of parasitism stages, they can be larval or imaginal (imago – adult adult parasites).

Classifications of parasite hosts. Depending on the stage of the parasite's life cycle, the *final (definitive) host* is distinguished, in the body of which the parasite reaches a mature stage and (or) reproduces sexually; *intermediate host*, in the body of which the parasite lives in the larval stage or propagates asexually; additional (second intermediate) host, in the body of which the development of subsequent larval stages occurs.

Depending on the nature of the formation of the "parasite-host" system, an *obligate (obligatory) host* is distinguished, in which the parasite is provided with the best survival, rapid growth and greatest fertility; *facultative (optional) host*, in which the parasite can inhabit, but not fully adapt, sometimes not reaching puberty; *reservoir host*, in which the parasite does not undergo a full cycle of development, but only its accumulation in the invasive stage is observed.

*The source of invasion*is the final or reservoir host.

The factors of transmission (factors of invasion) are household items, environmental elements, food contaminated with the invasive stages of parasite development.

*The stage of invasion*is the stage of the parasite's life cycle, which, when it penetrates the human body, can cause a disease.

Often the sources of invasion are carriers of parasites – humans and animals. It is important to know that sometimes the entire cycle of parasite development takes place within the same host. This phenomenon is called *autoinva-*

sion. For example, hymenolepidosis (pathogen – dwarf tapeworm), children are ill for a long time, because it is possible to enter the intestines (without the release of eggs into the environment). Auto-invasion is observed with enterobiosis (pathogen – pinworm) and hymenolepidosis, when a person re-infects himself.

Characteristics of the "parasite-host" system

The relationship of the parasite, the host and their environment is being studied by *ecological parasitology*, the foundations of which were laid in the 30s. XX century the works of V. A. Dogel, V. N. Beklemishev and E. N. Pavlovsky. The host is the connecting link between the parasite and the external environment, for the parasite it serves as the first-order habitat, and the external environment – the second-order habitat – affects the parasite indirectly through the host's body.

The "parasite-host" system includes one host individual and one or a group of parasite individuals.

The following conditions are necessary for the formation of a "parasite-host" system:

- the parasite must come into contact with the host;
- in the host organism, the parasite must find the optimal conditions for its development;
- the parasite must withstand protective responses from the host organism.

Way of invasion. The following way of invasion (i.e. penetration) of parasites into the host organism exist:

- *alimentary* – through the mouth, i.e. orally (invasion factors – dirty hands; contaminated vegetables, fruits, greens; insufficiently thermally processed foods – meat, liver, lard; contaminated water);

- *airborne droplets*– through the mucous membranes of the upper respiratory tract (invasion factors – air, sputum of a sick person or animal, dust);
- *contact* – through close contact with a sick person or animal, through toys, handshakes, underwear and household items;
- *transmissible* – through the skin, that is, percutaneously (observed with bites of specific blood-sucking vectors – arthropods, invasion factor – carrier saliva);
- *contaminative* – percutaneously (carried out during scratching and active rubbing into the skin of invasion factors – excrement or hemolymph carriers);
- *active penetration into the skin* – percutaneously (occurs with the active introduction of parasite larvae located in water or land through intact skin);
- *transplacental* – through the placenta (invasion factor – the blood of a pregnant woman);
- *sexual contact* – through the genitals (invasion factor – mucous genital);
- *transfusion* – through the blood (invasion factors – infected donated blood or transfusion fluids, non-sterile medical instruments).

In the process of evolution, a stabilization of relations is observed in the "parasite-host" system, which leads to smoothing of antagonistic reactions and the development of an equilibrium system. The evolution of the host goes along the path of improving the mechanisms of protection against parasite, the evolution of parasites is aimed at complicating the mechanisms of adaptation to the host.

Protective reactions from the host. The first reaction of the host organism is an attempt to destroy the parasite with non-specific protective agents

(free radicals and hydrolases), to neutralize the parasite toxins with the help of proteases and enzyme inhibitors.

In the absence of effect, protective reactions of the host organism are manifested at different levels: cellular, tissue, and organism. At the cellular level, a change in the shape or size of cells affected by parasites occurs. At the tissue level, the host organism is trying to isolate the parasite-affected tissue from healthy tissue, a connective tissue capsule is formed around the pathological foci, the expansion of blood vessels, the accumulation of leukocytes is noted. At the body level, the host organism's immune responses (the formation of antibodies and immune lymphocytes, phagocytosis) in response to the action of the parasite. Immune host reactions lead to a decrease in the rate of parasite proliferation and a delay in their development.

Adaptation of parasites. There are morphophysiological and biological adaptations of parasites.

Morphophysiological adaptations are associated with changes in the structure of parasites and the functioning of their organ systems. Progressive adaptations include fixation organs (suckers, hooks, claws, oral apparatus), the complex structure of the outer covers (cuticle, tegument), molecular mimicry (similar structure of proteins and enzymes of the parasite and the host), antigen enzymes excreted by intestinal parasites (protection against digestion by the digestive enzymes of the host organism), intracellular parasitism, immunosuppressive action of parasites (endoparasites secrete proteases that destroy immune complexes and host cells), etc. Regressive adaptations include reduction of the organs of motion and some systems (for example, digestive in tapeworms), simplification of the structure of the nervous system and sensory organs.

Biological adaptations are associated with the peculiarities of reproduction and life cycles of parasites. For most parasites, the following biological adaptations are characteristic:

– hermaphroditism (more common in parasites than in free-knife forms);

– good development of the reproductive system and high fertility (per day, an order of magnitude more eggs are secreted than free-living forms: pork tapeworm – 100 thousand, human roundworm – 250 thousand, free-living ciliary worms – 5–10 eggs);
– improvement of various forms of asexual reproduction (schizogony in sporozoans, budding in tapeworms, polyembryony in flukes);
– complex development cycles with the presence of several larval stages and a change of owners;
– migration of larvae throughout the host (Ascaris, pork tapeworm, trichinella).

Changes in the host organism as a result of the pathogenic action of parasites. *Toxic-allergic effects* are exerted by the products of vital activity or decay of dead parasites. An attack of malaria is associated with the release into the blood of metabolic products of plasmodia with the destruction of red blood cells. The bites of ectoparasites insects cause itching due to the toxic effect of their saliva on the nerve endings in the skin. Skin rashes, eosinophilia, headaches are a consequence of the action of metabolic products of helminthes larvae circulating in the blood.

Parasites cause *mechanical damage* to organs and tissues: migratory Ascaris larvae violate the integrity of the intestinal wall and alveoli of the lungs, Ascaris tangles can cause intestinal obstruction, parasitic cat flukes in the liver – blockage of the bile ducts. Sucker tapeworms, infringing on the intestinal mucosa, lead to tissue non-crush. The proboscis of ticks and insects damage the skin.

The *absorption of nutrients* and vitamins from the host also has a negative effect on it. Helminthiasesare usually accompanied by hypovitaminosis (A and C).

The *opening of ways for secondary infection* is due to the fact that helminthes or their larvae, violating the integrity of the skin or intestinal mucosa, contribute to the penetration of microorganisms.

Violation of all metabolic processes in the host (protein, carbohydrate, fat, etc.), a general weakening of the body, a decrease in its resistance and increased sensitivity to other diseases can also be the result of exposure to parasites.

The consequences of the relationship in the "parasite-host" system can be different:

- death of the parasite – if the response of the host organism is strong enough;
- parasitic carrier (the presence in the body of the pathogen without pronounced clinical signs of the disease) – if the relationship between the parasite and the host is relatively balanced;
- development of the disease – if the parasite has a high pathogenicity, and the host response is insufficient;
- death of the host (often the parasite also dies with the host).

From a medical point of view, such parasite characteristics as pathogenicity, virulence, and specificity are of interest. *Pathogenicity* is the ability to harm the host. The pathogenicity of the parasite is a relative concept. It depends on a number of factors: the host's genotype, its age (young organisms are most sensitive to infection), the food regime (an incomplete or excessive diet weakens the host's body, for example, an increase in human blood sugar leads to more frequent and more severe attacks of malaria), the presence of other parasites and diseases in the host. *Virulence* is the degree of manifestation of pathogenicity.

Transmissible and focal diseases

Diseases that exist for a long time in a certain territory in nature, regardless of a person, are called natural focal. The doctrine of the natural foci of parasitic diseases was developed by academic E.N. Pavlovsky in 1940.

Natural focal diseases spread in a certain area with a specific landscape where the pathogen is circulated from one animal to another, regardless of the

person, and wild animals of this ecosystem serve as the reservoir of the pathogen.

Components of the natural focus include the causative agent of the disease, pathogen sources (reservoir hosts, carriers) organisms susceptible to the causative agent, and certain environmental conditions.

Depending on the classification method, the following types of foci of parasitic diseases are distinguished:

- by origin – natural (hosts – wild animals), synanthropic (hosts – domestic animals), mixed;
- along the length of the outbreak – narrowly limited (small areas, such as a rodent's hole), diffuse (extensive territories, such as the taiga), conjugate (several pathogens circulate in the outbreak);
- in relation to the owners – *anthroponosis* (parasites circulate only among people), *zoonosis* (parasites circulate only among animals), *zooanthroponosis* (parasites circulate among people and animals).

Some natural focal diseases cause *endemically*, i.e., occur in strictly defined limited territories. A small number of natural focal diseases are found everywhere. Prevention of these diseases is very difficult.

Diseases whose pathogens are transmitted from one organism to another by means of specific blood-sucking arthropods (insects, ticks) are called *transmissible* (malaria, taiga encephalitis). Most vector-borne diseases are natural focal.

Among human parasites, protozoa, helminthes and arthropods are of the greatest medical importance, respectively, the following sections of medical parasitology are distinguished:

- *protozoology* – studies parasitic representatives of the subkingdom unicellular and protozoa diseases;
- *helminthology* – studies parasitic worms and the diseases they cause – helminthes infections;
- *arachnoentomology* – studies parasitic representatives of the Arthropod type, their role in the occurrence of invasive diseases.

CONTROL QUESTIONS

1. Types of relationships between organisms.

2. Parasitism and its ecological foundations. Classification of parasites. Classification of parasite hosts.

3. Characteristic of the host – parasite relationships. The pathogenic effect of parasites on the host organism. The response of the host organism.

4. Morphological, physiological and biological adaptation of parasites.

5. Key terms of medical parasitology: sources of infection, invasive stage, modes of infection, mode of transmission, virulence factors.

6. Transmissible diseases.

7. Natural-focal anthroponosis, zooanthroponoses, zoonosis.

CHAPTER 2
MEDICAL PROTOZOOLOGY

Medical protozoology studies the biology and ecology of parasitic representatives of the Protozoa subkingdom, as well as the issues of epidemiology, pathogenesis, diagnosis, treatment and prevention of human protozoa diseases.

Protozoa are unicellular living organisms. In morphological terms, the body of the protozoa is equivalent to one cell, but physiologically represents a whole organism. Of the 70 thousand species of protozoa, more than 10 thousand lead a parasitic lifestyle, about 50 species are human parasites. Many protozoa have adapted to parasitism inside cells, in human blood and tissue fluid.

In 1980, the International Committee on Systematics proposed dividing all unicellular into seven types. According to the classification adopted by the zoology of V.A. Dogel (1981), the protozoa that live in the human body and have medical value belong to three Phylums: Sarcomastigophora (class Sarcodina and class Mastigophora), Apicomplexa (Coccidiomorpha) and Ciliophora (class Ciliata).

Zoological Classification of Protists (Dogel V.A., 1981)

KINGDOM: ANIMALIA (ZOA)

SUBKINGDOM: PROTOZOA

Phylum: Sarcomastigophora

Class: Sarcodina
Species: Entamoeba histolytica; E. coli; E. gingivalis

Class: Mastigophora
Genus: Leishmania
Species: Leishmania donovani; L. tropica; L. braziliensis

Genus: Tripanosoma
Species: Trypanosoma brucei gambiense;
Tr. brucei rhodesiense; Tr. cruzi

Genus: Giardia (Lamblia)
Species: Lamblia intestinalis

Genus: Trichomonas
Species: Trichomonas vaginalis; Tr. hominis; Tr. tenax

Phylum: Apicomplexa

Class: Coccidiomorpha
Order: Coccidia
Species: Toxoplasma gondii; Pneumocystis carinii; Sarcocystis lindemanii

Order: Haemosporidia
Species: Plasmodium vivax; Pl. malaria; Pl. falciparum; Pl. ovale

Phylum: Ciliophora

Class: Ciliata
Species: Balantidium coli

General characteristics of Protozoa

The protozoa cell consists of the plasma membrane, cytoplasm, one or more nuclei.

The protozoan plasma membrane can increase the thickness and density due to the formation of the outer layer – the pellicle or a stronger cuticle.

The protozoan cytoplasm is divided into two layers. The outer layer (ectoplasm) is lighter and denser with respect to the inner layer (endoplasm). The endoplasm contains organelles, due to which the functional activity of protozoa (nutrition, respiration, excretion, etc.) is carried out.

Most protozoa have a single nucleus, but there are also multi-nuclear forms. In many protozoa, the nucleus contains no more than three pairs of chromosomes.

The movement of protozoa is carried out using pseudopods, flagella or cilia.

The nutrition of protozoa can be carried out in several ways: using pseudopods (pinocytosis and phagocytosis); endosmotically (absorption of dissolved nutrients by the entire surface of the cell); using the cell mouth (cytostome) and cell gullet (cytopharyngs). The digestion is carried out in the digestive vacuole.

The excretion. The contractile (pulsating) vacuole plays the role of an organelle of excretion, respiration and osmoregulation. Some representatives have an excretory organ – the anal pore.

Reproduction of protozoa are asexual and sexual. Forms of *asexual* reproduction include binary division by mitosis (for example, amoeba), multiple fision occurs in Plasmodium. In some protozoa, asexual reproduction alternates with the sexual process. During *sexual* reproduction in sporozoans, *copulation* takes place. The sexual process of Ciliata (Paramecium) is called *conjugation*.

The forms of existence of protozoa are different. Some protozoa can exist in two morphological forms: vegetative (trophozoite) and cyst. *Vegetative forms* under adverse conditions are able to encyst (i.e., become round and cover with a dense shell). *Cysts* are more resistant to adverse external factors. When the cysts get into favorable conditions, they are excised, i.e., the cyst membrane is destroyed and vegetative forms are formed, which pass into the period of active life.

The *Phylum Sarcomastigophora* includes two Classes: Sarcodina and Mastigophora.

CLASS SARCODINA

Representatives are inhabitants of the seas, reservoirs and soil. Some sarcodes (ameba) adapted to commensalism or a parasitic lifestyle. Amoeba do not have mitochondria typical of eukaryotic cells; they do not have a Golgi complex. As a result of the absence of a dense shell (pellicle), the shape of the Sarcode cell is unstable, due to which protrusions of the cytoplasm — pseudopodsare formed. Pseudopods perform a dual function – they serve to eatingby

endocytosis and to move. In adverse conditions, some Sarcodina can form cysts.

The following representatives of the Sarcode class are of medical importance:

– oral amoeba (Entamoeba gingivalis) – found in carious teeth, in raids on the tonsils, feeds on bacteria and food debris. This form is commensal;
– intestinal amoeba (Entamoeba coli) – is localized in the lumen of the large intestine, feeds on bacteria, is able to encyst (intestinal amoeba cysts contain more than 4 nuclei, usually up to 8). This form is commensal;
– dysenteric amoeba (Entamoeba histolytica) – is a human parasite. This form causes *amebiasis*.

AMEBIASIS.

Characteristic of parasitic disease:

1. The name of the parasite – *Entamoeba histolytica.*

2. Geographical distribution – warm countries.

3. Location (place of localization) – large intestine.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick people;

4.2. Invasion stage – cyst form;

4.3. The way of invasion – alimentary;

4.4. Factors of invasion – unwashed vegetables, fruits, greens, dirty water, dirty hands;

4.5. Vectors (mechanical)– flies, cockroaches.

5. Life cycle (Fig. 1).

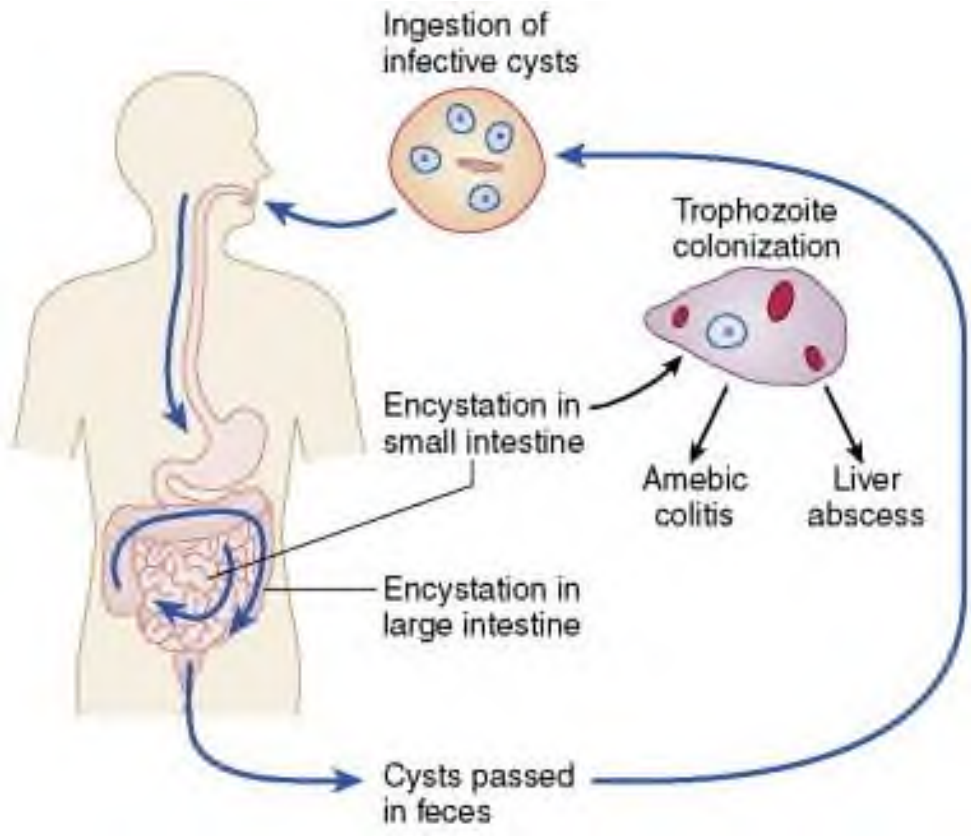


Fig. 1. Life cycle of *Entamoeba hystolitica* (https://www.sciencedirect.com)

In the life cycle of a dysenteric amoeba, a vegetative form (trophozoite) and a cyst are isolated. Trophozoites can exist in three forms: small vegetative, or luminal (forma minuta), large (forma magna) and tissue (erythroblast).

6. Pathogenic effects. There is pronounced toxic-allergic effect. When cysts are ingested in a human being, the cysts are excised in the large intestine, 8 small vegetative (luminal) forms are formed from one cyst. The luminous form is non-pathogenic. If in the host's organism it does not turn into a large vegetative form, then a person does not get sick, but becomes a parasitic carrier. Unaware of his disease, he poses a danger to others as a source of invasion. When the human body is weakened, the luminal form can turn into a large vegetative, which secretes proteolytic enzymes and causes necrosis of the mucous membrane of the colon with the formation of bleeding ulcers. The large form feeds on red blood cells, which can always be found in its cytoplasm.

Through ulcers, the parasite can enter the bloodstream and spread throughout the body. If they enter the bloodstream, amoeba can migrate to oth-

er organs (liver, brain), where they turn into a tissue form with the formation of foci of inflammation (tissue abscesses). In the external environment, vegetative forms areturns into a cyst. The cyst has a rounded shape, a dense shell. The cytoplasm of a mature cyst contains no more than four nuclei – this is an important differential diagnostic sign of dysenteric amoeba.

7. Clinical Symptoms. Fever is characteristic and allergic manifestations are possible. The most striking and painful manifestation of amebiasis is bloody diarrhea up to ten or more times a day. The proximal colon is more often affected. The stool is usually not abundant, slimy, with an admixture of blood. Patients are concerned about abdominal pain. The severity of intoxication varies greatly.

In the most severe cases, develop abscesses of the internal organs: an abscess of the liver, less often of the lungs and brain.

8. Laboratory diagnosis. Immunodiagnostics and PCP analysis are used. Laboratory diagnosis is based on a microscopic examination of feces smears. In parasitic carriers (cystocarriers), only cysts are found; in patients, along with cysts, vegetative forms are detected.

9. Preventive measures. *Personal prevention* consists in processing vegetables, fruits, herbs, boiling water, washing hands. *Public prevention* is basedon identifying and treating sick and parasitic carriers, disinfecting human feces, monitoring the sanitary condition of water sources and public catering establishments, destroying insects (flies and cockroaches) that are mechanical carriers of cysts, and educational work.

Some sarcodes (akantameba, negleria, gartmanella), leading a free lifestyle and living in soil and contaminated water, can cause severe inflammatory processes in the central nervous system (meningitis, encephalitis), which often have fulminant (very fast) course and end lethally.

CLASS CILIATA

The phylum Ciliophora includes the class Ciliata (Infusoria). Infusoria have a constant shape, the body is covered with a pellicle. Organoids of movement – cilia covering the whole body. Ciliates have two nuclei: a macronucleus that regulates metabolism, and a micronucleus that serves to exchange genetic information during sexual reproduction by conjugation. The food of the ciliates is captured using the cell mouth (cytostomy) and the pharynx (cytopharynges). Undigested food residues are thrown out through the specialized area of the cell surface. In humans, Intestinal Balantidia parasitizes, which causes *balantidiasis*.

BALANTIDIASIS.

Characteristic of parasitic disease:

1. The name of the parasite–*Balantidium coli.*

2. Geographical distribution – the disease is ubiquitous.

3. Location (place of localization) –thelarge intestine.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick person and many mammals;

4.2. Invasion stage – a cyst, less often trophozoite;

4.3. The way of invasion – alimentary;

4.4. Factors of invasion – unwashed vegetables, fruits, greens, dirty water, dirty hands;

4.5. Vectors (mechanical) – flies, cockroaches.

5. Life cycle (Fig. 2).

6. Pathogenic effect. There is pronounced toxic-allergic effect. Balantidium can live in the lumen of the large intestine of a person, without causing damage. With a decrease in immunity, for example, weakening of the body by other diseases, balantidia become more active and begin to show pathogenicity. By isolating the enzyme hyaluronidase, parasites infect the intestinal mucosa and invade it, which leads to the formation of deep bleeding ulcers. Balantidi-

um feeds on bacteria, food particles in the human intestines, sometimes red blood cells and white blood cells are found in its cytoplasm.

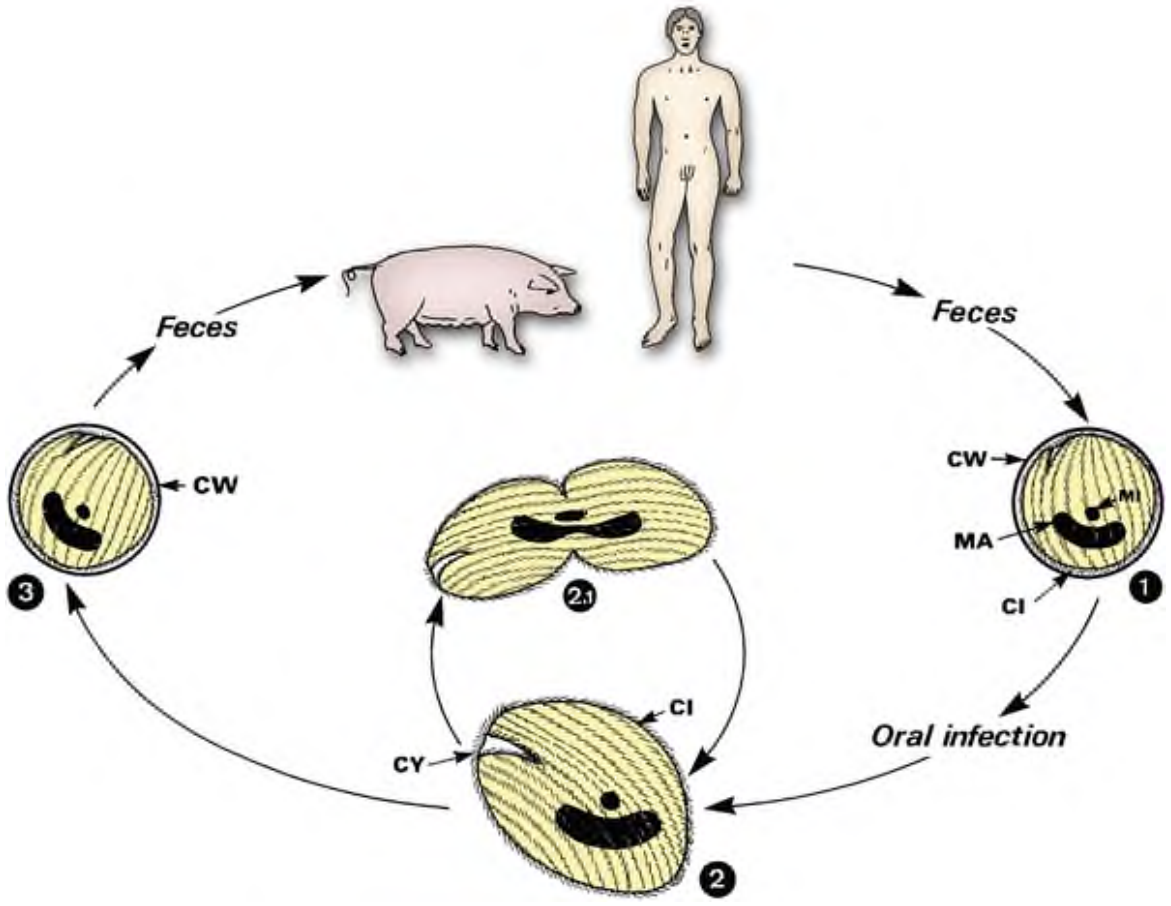


Fig. 2. Life cycle of *Balantidium coli*(https://link.springer.com)

7. Clinical symptoms. Fever is characteristic and allergic manifestations are possible. The disease is characterized by abdominal pain, prolonged diarrhea with an admixture of blood, mucus and pus. In complicated cases, perforation (perforation) of the intestinal wall with the development of peritonitis (inflammation of the peritoneum) is observed.

8. Laboratory diagnosis is based on the detection of vegetative forms and cysts of balantidia in smears of feces. Immunodiagnostics and PCP analysis are used.

9. Preventive measures: *personal*– washing food (vegetables, fruits, herbs), hands, boiling water. *Public* – identification and treatment of patients, veterinary supervision at pig farms, cattle slaughterhouses, health education.

CLASS MASTIGOPHORA

In the class of mastigophora, there are about 8 thousand species. Self-sustaining representatives live in ponds, many flagella bear a parasitic lifestyle. Their body is covered with a pellet, so the shape of the cell is constant. Some flagella have a supporting organelle – axostyle – in the form of a dense strand that runs along the cell. Movement is carried out using one or more flagella. The base of each flagellum is attached to the basal body (blepharoplast), formed from modified mitochondria. The blepharoplast function is associated with the generation of energy for the movement of the flagellum. In some forms, the flagellum runs along the body and is connected to it by a thin membrane, forming a wave-like (undulating) membrane, which provides translational movement of the simplest. Many flagella have a special education – kinetoplast (parabasal body). A distinctive feature of flagella with kinetoplast is the ability to form several morphological forms (with or without flagella) during the life cycle, depending on the conditions of existence.

Genus Giardia (Lamblia)

Giardia is a representative of one of the earliest branches of the phylogenetic tree of eukaryotes. In shape, the lamblia resemble a pear, cut in half, have two nucleus, four pairs of flagella. An axostyle runs along the midline along the entire cell, which divides the cell into two symmetrical halves. They have two symmetrically located suction discs, with the help of which they are attached to the villi of the duodenum.

Parasitization of giardia in the human body is most often asymptomatic, but in the case of weakened immunity, the parasite begins to exhibit a pathogenic effect. A disease caused by giardia is called giardiasis.

GIARDIASIS.

Characteristic of parasitic disease:

1. The name of the parasite–*Giardia lamblia, Lamblia intestinalis.*

2. Geographical distribution – diseaseis ubiquitous.

3. Location (place of localization) – small intestine and bile ducts.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick person or carrier;

4.2. Invasion stage – a cyst;

4.3. The way of invasion – alimentary;

4.4. Factors of invasion – unwashed vegetables, fruits, greens, dirty water, dirty hands;

4.5 Vectors (mechanical) – flies, cockroaches.

5. Life cycle (Fig.3).

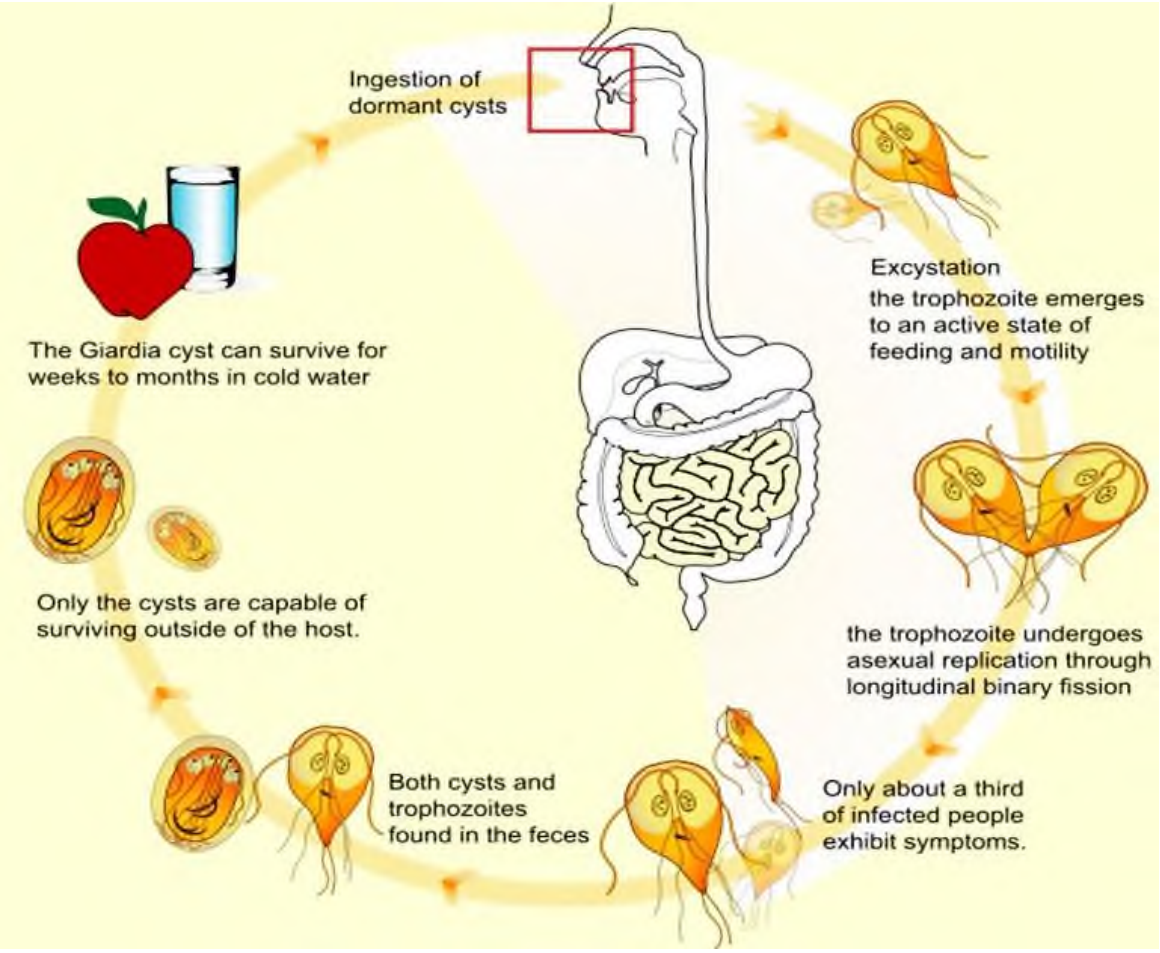


Fig. 3. Life cycle of *Giardia lamblia*(https://slideplayer.com)

6. Pathogenic effect. There is wear toxic effect, often allergic effect. Giardia is parasitized in the duodenum or gall bladder, where cysts are excised to form vegetative forms. Using suction discs, lamblia are attached to the villi of the intestine and disrupt the processes of parietal digestion and absorption. Gi-

ardia feed on substances from the surface of intestinal epithelial cells. In the lower parts of the small intestine and in the large intestine, Giardia is encysted.

7. Clinical symptoms. Fever is characteristic and allergic manifestations are possible. Sometimes allergic skin rashes are observed in children. Dyspeptic symptoms (nausea, vomiting, intolerance to fatty foods, unstable stools, etc.) are observed.

8. Laboratory diagnostics. Immunodiagnostics and PCP analysis are used. Detection of cysts in fecal smears. In the material obtained by duodenal sounding, mobile vegetative forms can be found.

9. Preventivemeasures: *personal* – washing hands, vegetables, fruits, herbs, boiling water. *Public* – the identification and treatment of patients, the fight against insects – mechanical carriers of Giardia cysts (flies, cockroaches).

Genus Trichomonas

Trichomonads have a pear – shaped body, a cellular mouth, four free flagella are located at the anterior end, one of which is connected with the undulating membrane. Axostyle extends along the entire cell, protruding at the posterior end in the form of a spike.

Intestinal Trichomonas (*Trichomonas hominis*) lives in the intestine, oral Trichomonas (*Trichomonas tenax*) – in the oral cavity. For intestinal and oral Trichomonas pathogenic action is not established, they relate to commensals.

The vaginal trichomonas (*Trichomonas vaginalis*) is a parasite, has great medical importance, causes trichomoniasis.

UROGENITAL TRICHOMONIASIS.

Characteristic of parasitic disease:

1. The name of the parasite – *Trichomonas vaginalis.*

2. Geographical distribution – diseaseis ubiquitous.

3. Location (place of localization) – genitourinary tracts.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick person (usually a woman) or a parasitic carrier (more often than a man);

4.2. Invasion stage – vegetative form (trophozoits);

4.3. The way of invasion – sexual contact;

4.4. Factors of invasion – transmitting factors may be shared towels and underwear.

5. Life cycle (Fig.4).

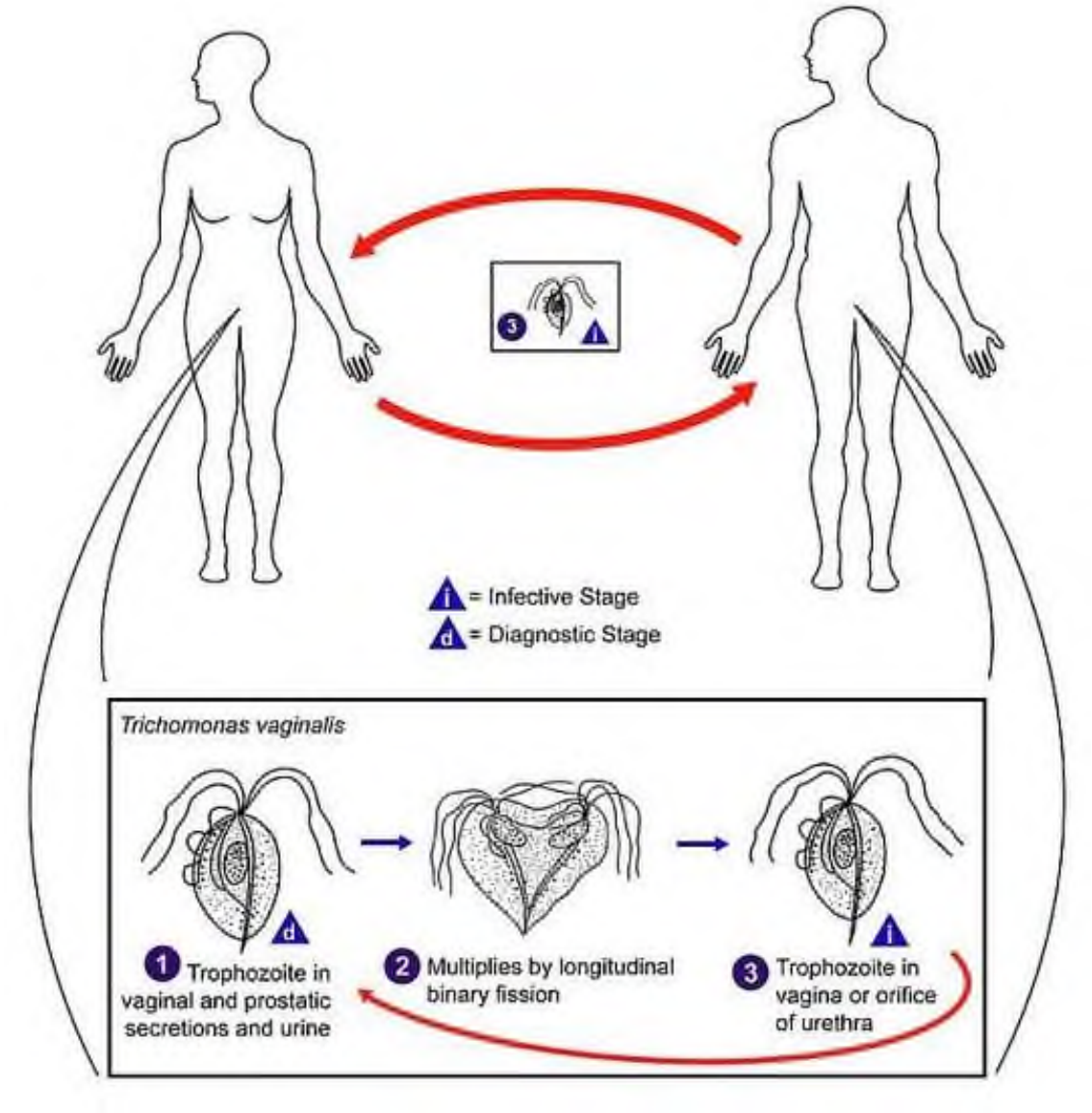


Fig. 4. Life cycle of *Trichomonas vaginalis* (https://fineartamerica.com)

6. Pathogenic effect plays a significant role in the pathology of the genitourinary system, especially in women. Parasitizes in the mucous urogenital tract, does not form cysts, quickly dies in the external environment. There is wear toxic effect.

7. Clinical symptoms. In women, the mucous membranes of the vagina and cervix are affected. The main symptoms of the disease are itching, pain, burning, profuse serous-purulent discharge (leucorrhoea) from the vagina. In men, the parasite is localized in the urethra, bladder, prostate gland. Often the disease is long and asymptomatic, so infected men are dangerous for others as a source of spread.

8. Laboratory diagnostics. Detection of Trichomonas in secretions from the genitourinary tract during microscopic examination, immunological analysis, PCR.

9. Preventive measures: *personal* – the exception of random sexual contacts, the presence of individual hygiene items. *Public* – the identification and treatment of patients (at the same time both sexual partners), monitoring the sterility of gynecological instruments in medical institutions.

Genus Trypanosome

Tripanosome genus includes single-flagellated parasites having a kinetoplast and undulating membrane. The shape of the cell is elongated. In the life cycle of trypanosomes, which are parasitic in humans, there are two obligatory (obligate) hosts: an insect – a specific carrier and a mammal.

Trypanosomes are the causative agents of African and American trypanosomosis. Trypanosomosis is a transmissible natural focal disease. The natural foci of trypanosomiasis are associated with the geographical distribution of vector insects.

AFRICAN TRYPANOSOMIASIS (SLEEPING SICKNESS).

Characteristic of parasitic disease:

1. The name of the parasite – *Trypanosoma brucei gambiense* (spreed in West Africa), and *Trypanosoma brucei rhodesiense* (spreed in East Africa).

2. Geographical distribution – Africa.

3. Location (place of localization) – blood, cerebrospinal fluid, lymph nodes.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick people, as well as animals that are natural reservoirs: pigs, dogs (with the Gambian version), antelopes, rhinos, cattle and small cattle (with the Rhodesian version);

4.2. Invasion stage – vegetative form;

4.3. The way of invasion – transmissible, with Tsetse fly bite;

4.4. Factors of invasion – insect saliva;

4.5 Vectors (specific) – Tsetse fly.

5. Life cycle (Fig. 5).

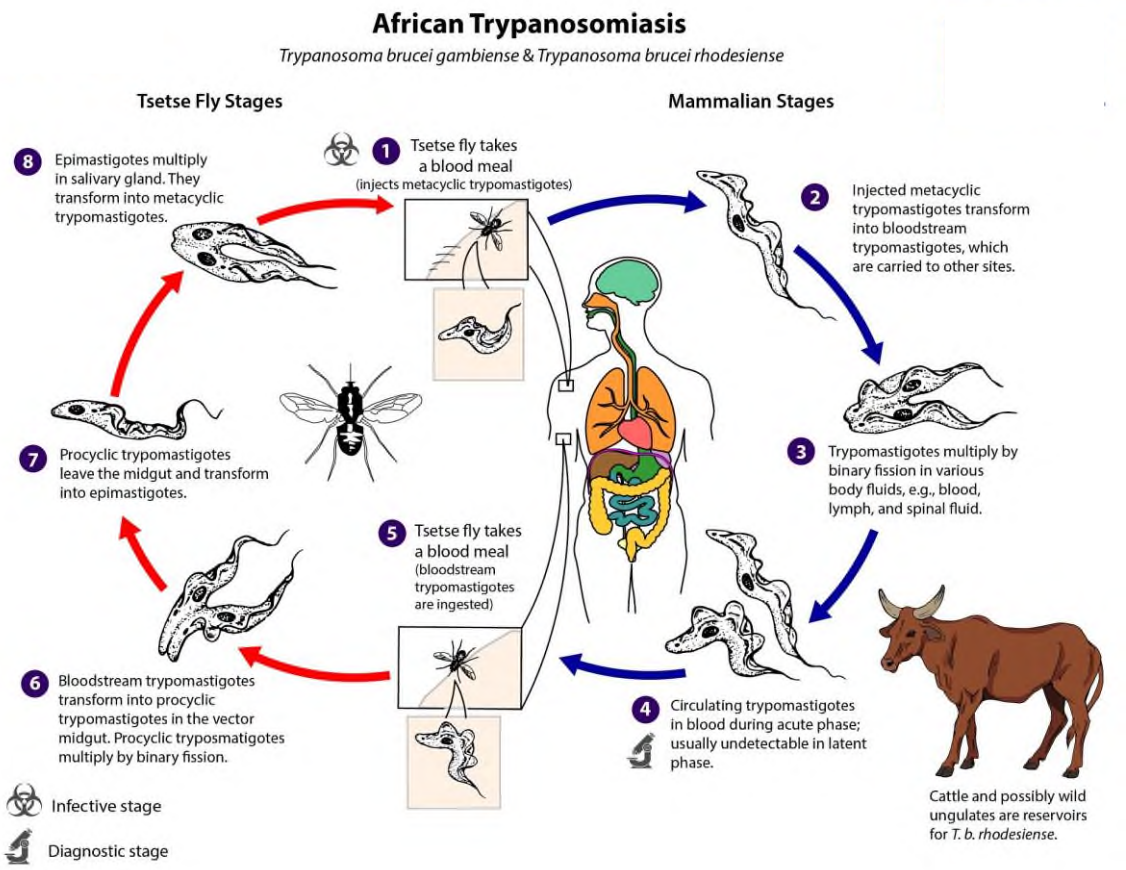


Fig. 5. Life cycle of *Trypanosoma brucei gambiense (rhodesiense)* (https://www.cdc.gov)

6. Pathogenic effects. There is pronounced toxic-allergic effect. After getting into the human body for 9–10 days, trypanosomes live in the subcutaneous tissue, where trypanosomal chancre is formed. Then the parasites migrate to the lymphatic system, actively multiply there, and through20 – 25 days enter

the bloodstream, from where they spread to all tissues and organs. The predominant localization of trypanosomes is cerebrospinal fluid, from where they settle into the brain and spinal cord. The vital products of trypanosomes have a pronounced toxic-allergic effect.

7. Clinical symptoms. Characterised by high body temperature. The disease develops gradually and lasts with the Gambian version from 6 to 10 years, with the Rhodesian (more severe) – several months. Trypanosomal skin ulcers appears at the site of the bite of the tsetse fly, then fever joins, an increase in lymph nodes, splenomegaly, weakness, and exhaustion are observed. Later, lesions of the central nervous system occur: severe drowsiness (hence the name – sleeping sickness), muscle weakness, rapidly progressing dementia. In the terminal stage, indifference to the environment appears stupor, then coma. If untreated, the disease is fatal.

8. Laboratory diagnosis of trypanosomosis is based on the detection of various morphological forms of trypanosomes in smears of peripheral blood (extracellular parasitism), as well as in punctate lymph nodes, cerebrospinal fluid. Immunodiagnostics and PCP analysis are used.

9. Preventive measures: *personal* – protection against bites of the tsetse fly. *Public* – the identification and treatment of patients, if possible – the destruction of natural reservoirs, the fight against insect vectors.

AMERICAN TRYPANOSOMIASIS (CHAGAS DISEASE).

Characteristic of parasitic disease:

1. The name of the parasite – *Trypanosoma cruzi*.

2. Geographical distribution – Latin America.

3. location (place of localisation) – blood, cerebrospinal fluid, lymph nodes.

4. The mechanism of human infection:

4.1. Sources of invasion – sick people, as well as animals that are natural reservoirs: armadillos, possums, anteaters, guinea pigs, dogs, cats and other wild and domestic animals living in Latin America;

4.2. Invasion stage – vegetative form;

4.3. The way of invasion – transmissible. In the body of the tripod nasal bug, they multiply, then enter the hind gut, where they achieve a state of invasion. Favorite places of a bite are the areas of transition of the skin into the mucous membrane, most often the lips, hence the name of the bug. The biology of the kissing bug is such that immediately after bloodsucking it defecates in the wound, from where trypanosomes penetrate the blood – this is facilitated by combing the bite;

4.4. Factors of invasion – insect feces;

4.5. Vectors (specific) – bug of the genus *Triatoma* (kiss bug).

5. Life cycle (Fig. 6).

6. Pathogenic effects – the pathogenic effect is based on toxic-allergic reactions to the decay products of trypanosomes and affected cells.

7. Clinical symptoms – the disease affects mainly young children, in which it proceeds acutely. The incubation period lasts 1 to 2 weeks. At the site of penetration of trypanosomes, a tripanosomal skin ulcers are develops. Then there is a fever, headache, swelling of the face, pain in the heart. Myocarditis, cerebral hemorrhages and meningoencephalitis are characteristic of the disease.

8. Laboratory diagnostics – detection of trypanosomes in blood smears, cerebrospinal fluid, punctured lymph nodes, spleen, bone marrow. Immunodiagnostics and PCP analysis are used.

9. Preventive measures: *personal* – individual protection from bites of bedbugs (especially young children). *Public* – the identification and treatment of patients, if possible – the destruction of natural reserves, the fight against the spread of triatomic bugs in the foci of the disease.

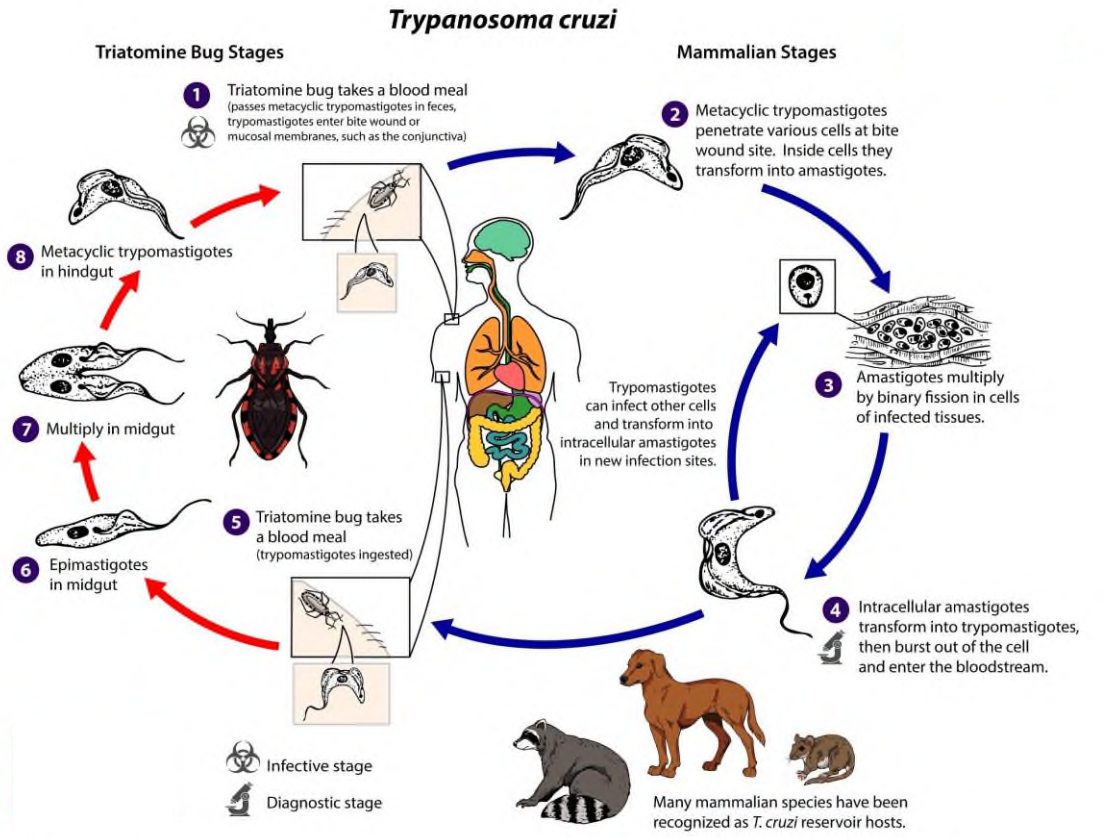


Fig. 6. Life cycle of *Trypanosoma Cruzi* (https://www.cdc.gov)

Genus Leishmania

A structural feature of Leishmania is a relatively large nucleus, occupying up to 1/4 of the cytoplasm. In the life cycle of Leishmania, two morphological forms alternate: in the body of carriers – promastigotes (flagellum form), in the body of the final hosts – amastigotes (lack of flagellum).

Leishmania are the causative agents of vector-borne natural focal diseases – Leishmaniasis. Specific blood-sucking vectors of all types of Leishmania are sand flies (genus *Phlebotomus*), in which parasites multiply in the digestive tract and accumulate in the proboscis. Human infection occurs with sand flies bites. There are cutaneous, visceral and mucocutaneous leishmaniasis.

CUTANEOUS LEISHMANIASIS.

Characteristic of parasitic disease:

1. The name of the parasite – *Leischmania tropica minor* (rural version) and *Leischmania tropica major* (urban version).

2. Geographical distribution – widespread in the countries of Southern Europe, North and West Africa, the Middle East, and Central Asia (the old name for the disease is Ashgabat).

3. Location (place of localization) – skin.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick people and natural reservoirs: in the rural type – rodents (gerbils, gophers), in the urban – only people;

4.2. Invasion stage –vegetative form (trophozoits);

4.3. The way of invasion – transmissible, with sand fly bites;

4.4. Factors of invasion – insect saliva;

4.5. Vectors (specific) – sand fly *(Phlebolomus* species).

5. Life cycle (Fig. 7).

6. Pathogenic effect – Leishmania is characterized by intracellular parasitization in the cells of the reticuloendothelial system. At the site of the bite, parasites actively multiply, which leads to the development of foci of local inflammation, then the affected areas are not crotized, tissue ulceration is observed. The occurrence of an ulcer is accompanied by edema of the surrounding tissues, inflammation and enlargement of the lymph nodes. Healing occurs with the formation of a coarse disfiguring scar.

7. Clinical symptoms – itching appears on the skin at the site of the bite, subsequently a specific granuloma (leishmanioma) is formed. First, brownish-red tubercles appear on the skin, which gradually increase in size, then ulcerate. With the rural type, the development of the ulcer progresses rapidly, with the urban type, later ulceration is observed.

8. Laboratory diagnosis – laboratory diagnosis is based on the preparation of smears from the contents of ulcers, followed by microscopy. Smearless forms of leishmania inside the cells are found in smears. Immunodiagnostics and PCP analysis are used.

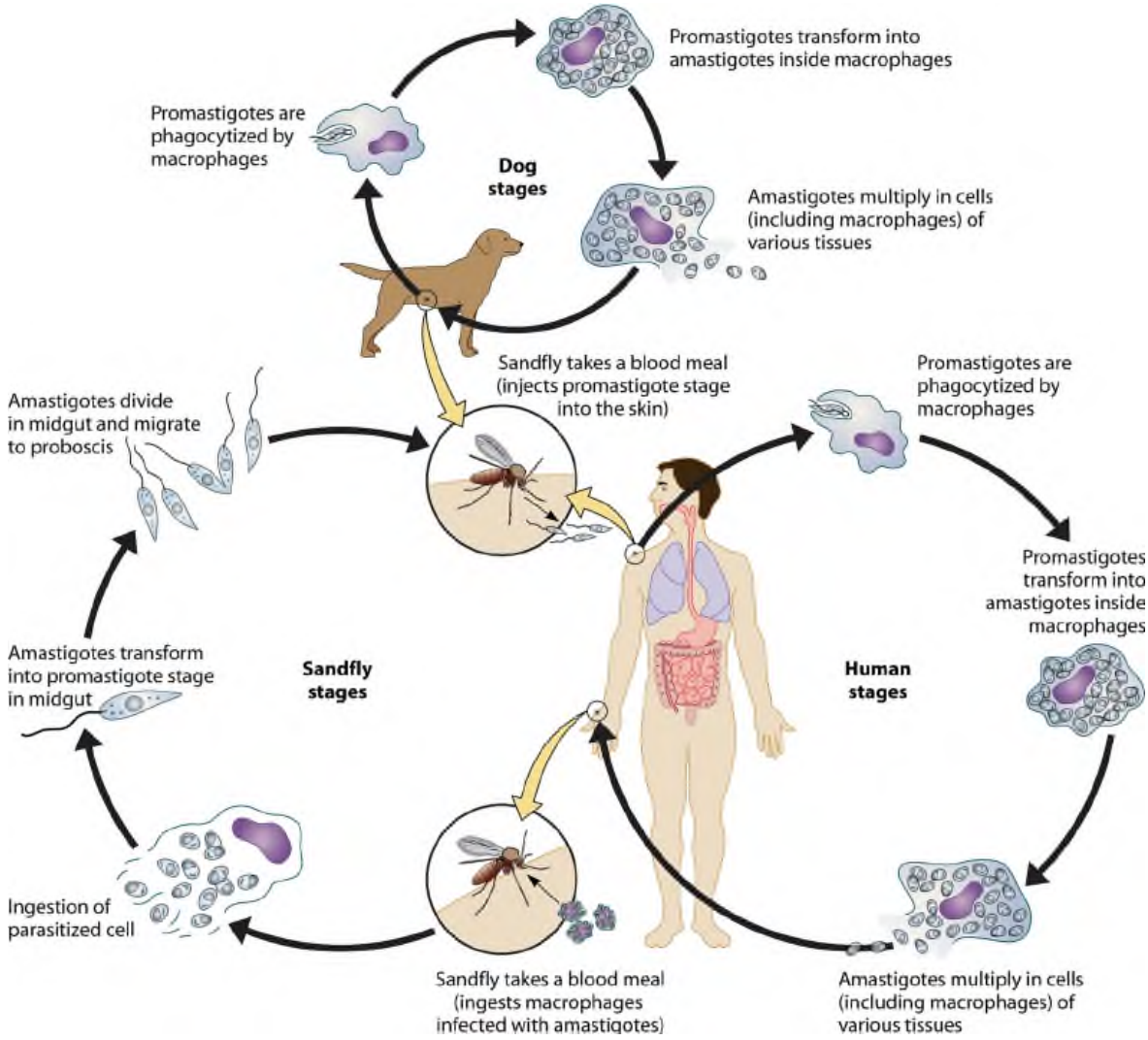


Fig. 7. Life cycle of *Leischmania* (https://www.researchgate.net)

9. Preventive measures: *personal* preventive measures come down to individual protection from sand flies bites. *Public* prophylaxis is aimed at identifying and treating patients, if possible – the destruction of natural reserves within macrophages.

VISCERAL LEISHMANIASIS (BLACK DISEASE, FEVER DOOM-DOOM, CHILDREN'S KALA-AZAR).

Characteristic of parasitic disease:

1. The name of the parasite – *Leischmania donovani* and *Leischmania infantum.*

2. Geographical distribution – widespread in the countries of the Mediterranean, Asia, Africa and South America.

3. Location (place of localization) – liver, spleen, lymph node, bone marrow.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick people and animals – natural reservoirs (jackals, dogs, rodents);

4.2. Invasion stage –vegetative form (trophozoits);

4.3. The way of invasion – transmissible, with sand fly bites;

4.4. Factors of invasion – insect saliva;

4.5 Vectors (specific) – sand flies;

5. Life cycle (see Fig.7).

6. Pathogenic effect – Leishmania that enters the human body is captured by macrophages, loses flagella and proceeds to intracellular parasitism. In the host cells, they multiply intensively. After the destruction of these cells, they enter the surrounding tissues, affect healthy cells. Pathogenesis is based on a toxic-allergic effect.

7. Clinical Symptoms – the incubation period lasts from several weeks to 6 to 8 months. The disease is characterized by the appearance of the wrong type of fever, which quickly exhausts the patient. Weakness, headache, intoxication, exhaustion increase. Pallor of the skin is observed, sometimes dark pigmentation, a rash appears, an increase in the liver, spleen and lymph nodes occurs. In the affected areas, necrotic processes develop. The red bone marrow is affected, which leads to the development of anemia. In most cases, children get sick.

8. Laboratory diagnosis – laboratory diagnosis is based on the detection of non-flagellate forms of leishmania inside macrophages in punctates of bone marrow, lymph nodes, sometimes the liver and spleen. Immunodiagnostics and PCP analysis are used.

9. Preventive measures: *personal* preventive measures come down to individual protection from sand flies bites. *Public* prophylaxis is aimed at identifying and treating patients, if possible – the destruction of natural reserves within macrophages.

MUCOCUTANEOUS LEISHMANIASIS (ESPUNDIA).

Characteristic of parasitic disease:

1. The name of the parasite – *Leischmania brasiliensis.*

2. Geographical distribution – widespread in South and Central America.

3. Location (place of localisation) – skin, mucous membranes, and cartilage are affected, most often the nose, pharynx and larynx.

4. The mechanism of human infection:

4.1. Sources of invasion – asick people and animals – natural reservoirs (rodents, monkeys, sloths);

4.2. Invasion stage –vegetative form (trophozoits);

4.3. The way of invasion – transmissible, with sand fly bites;

4.4. Factors of invasion – insect saliva;

4.5. Vectors (specific) – sand flies.

5. Life cycle (see Fig.7).

6. Pathogenic effect. With this form of the disease, parasites penetrate from the skin through the blood vessels into the nasopharynx, larynx, soft palate, genitals, where they multiply in the macrophages of the connective tissue and cause inflammatory and destructive changes.

7. Clinical Symptoms – the incubation period lasts from several weeks to 3 months. At the site of the sand fly bite, ulcers occur, at the same time tissue proliferation appears. The skin, mucous membranes, and cartilage are affected (most often the nose, pharynx and larynx). Symptoms of intoxication are clearly expressed.

8. Laboratory diagnostics – laboratory diagnostics is based on a microscopic analysis of smears from skin ulcers and the detection of flagless Leishmania forms inside macrophages there. Immunodiagnostics and PCP analysis are used.

9. Preventivemeasures: for all types of leishmaniasis, *personal* preventive measures come down to individual protection from sand flies bites. *Public* prophylaxis is aimed at identifying and treating patients, if possible – the destruction of natural reserves within macrophages.

PHYLUM APICOMPLEXA

The representatives of phylum Apicomplexa are intracellular parasites. Their life cycle is often associated with a change of owners and the alternation of sexual and asexual reproduction. Asexual reproduction is observed in the body of the intermediate host. Sexual reproduction (gametogony) occurs in the body of the final host.

Trophozoites do not have organelles of movement, digestive and contractile vacuoles. Nutrition, respiration, and excretion occur across the entire surface of the cell. Outside, the body is covered with a three-layer pellicle. Under the pellicle are fibrils and microtubules that perform supporting and contractile functions, providing the movement of the parasite.

Representatives of the class Coccidia, which belong to two orders: Coccidia and Blood spores, have medical value.

Order Coccidia

Representatives of the Coccidia order lead an intracellular lifestyle, parasitize in the cells of the reticuloendothelial system, liver, intestines, nervous system, etc. A conoid, a special organoid, is located on the front end of the trophozoite's body. Is involved in the process of penetration of the parasite into the host cell. Roptria are located next to the conoid – a kind of organoid that secretes a secretion for dissolving the host cell membrane.

Toxoplasmas, pneumocysts and sarcocysts are of medical importance.

TOXOPLASMOSIS.

Characteristic of parasitic disease:

1. The name of the parasite – *Toxoplasma gondii.*

2. Geographical distribution – diseaseis ubiquitous.

3. Location (place of localization) – cells of organism.

4.The mechanism of human infection:

4.1. Sources of invasion;

4.1.1. Invasive cats and representatives of the cat family, secreting oocysts with feces;

4.1.2. Invasive animals and birds that can secrete toxoplasma into the external environment in various ways: with feces, saliva, nasal mucus, amniotic fluid, sperm, milk, etc;

4.2. Invasion stage – oocysts, pseudocysts, cysts and trophozoites;

4.3. The way of invasion;

4.3.1 Alimentary: Infection occurs by contact with invaded cats and oocyst contamination of the hands. In addition, the Nazis can get into the soil, and then on food (vegetables, fruits, herbs, water, etc.). Mechanical carriers of oocysts can be flies and cockroaches. When eating meat from invasive animals, as well as with milk and dairy products, pseudocysts and cysts can fall into the human body;

4.3.2. Percutaneous – when caring for invasive animals, as well as when processing hides and carcasses of such animals, pseudocysts and cysts can enter the human body through the skin and mucous membranes;

4.3.3. Intrauterine – infection of the fetus from an invasive mother is most dangerous, since in this case the birth of children with multiple congenital malformations is possible, primarily the brain;

4.3.4. Transfusion and transplantation;

4.4. Factors of invasion – unwashed hands after contact with cats, undercooked meat;

4.5. Vectors (mechanical) – flies, cockroaches.

5. Life cycle (Fig.8).

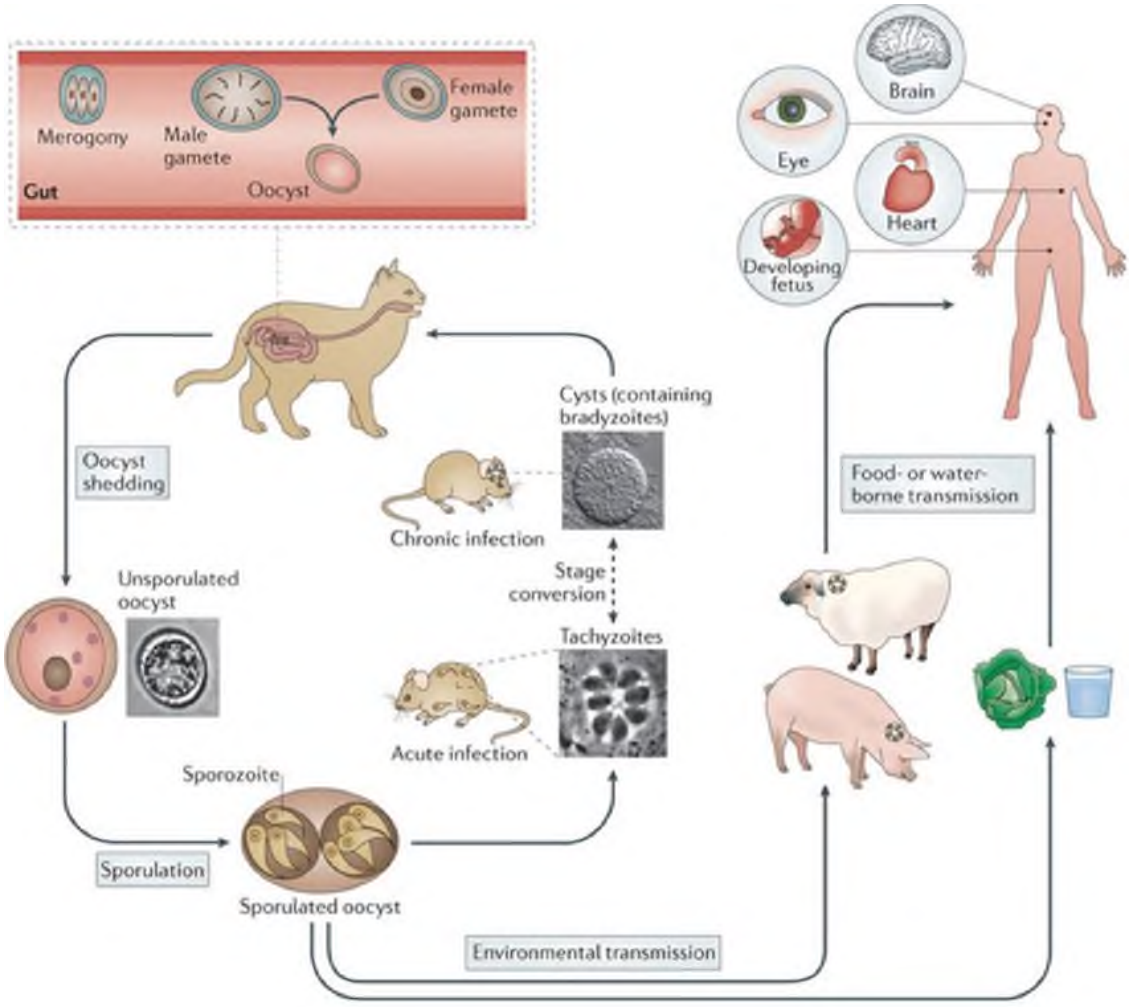


Fig. 8. Life cycle of *Toxoplasma gondii* (https://www.researchgate.net)

The life cycle of toxoplasma takes place with the participation of two hosts, with alternating stages of multiple division, gametogony and sporogony.

The definitive hosts are cats and representatives of the feline family, in which the gametogony occurs in the body, i.e., the sexual process with the formation of a zygote and oocyst. Infection of the definitive hosts occurs when the infected animals are eaten – intermediate hosts (rodents, birds), in the internal organs of which contain pseudocysts and cysts. In this case, in the digestive tract of feline trophozoites are excised and penetrate into the intestinal epithelial cells, where they multiply by endogony with the formation of merozoites. Part of the merozoites is converted into male (microgametes) and female (macrogametes) germ cells. When gametes are copied, a zygote is formed.

The zygote is coated, an oocyst is formed. Oocysts with feline feces are released into the environment. Within 1 – 5 days in oocysts division occurs by sporogony and two spores are formed with four sporozoites in each. These oocysts are invasive and can remain viable in the environment for several years.

When an oocyst is swallowed by an intermediate host (wild and domestic animals, birds, humans), its membrane dissolves in the intestine, sporozoites are released from it. Through the intestinal wall, sporozoites enter the lymphatic vessels and lymph nodes, where they grow and turn into trophozoites. Trophozoites enter the bloodstream, spread throughout all organs and tissues, penetrating the cells of the liver, spleen, nervous system, eyes, skeletal muscles, myocardium, etc. Trophozoites multiply inside cells by multiple division (endogony). In the acute period of the disease, an intense formation of pseudocysts is observed. A pseudocyst is a collection of trophozoites in a host cell. After the death of the host cell, trophozoites are released and infect other cells. As the formation of immunity that limits the reproduction of toxoplasma, the disease acquires a chronic course. At this time, cysts form. A cyst is an accumulation of trophozoites in a cell covered with a dense membrane.

6. Pathogenic effect. Sometimes observed toxic-allergic effect. Toxoplasmosis belongs to zooanthroponic pathologies, since toxoplasma affects a huge number of animal species, including birds and humans. The disease is ubiquitous, the average prevalence of the population in Russia is 20–30 %.

7. Clinical symptoms. Usually, parasites have a very low pathogenicity, therefore, in the majority of infested persons, the disease proceeds latently, i.e., without obvious clinical manifestations. However, in some conditions, an acute form of the disease may occur in 0.5–1 % of those who are invaded, which depends on the individual sensitivity of the host organism and on the way the toxoplasma enters the human body. The decay products and metabolism of toxoplasma have a toxic-allergic effect. The organs of vision are affected, as well as the nervous, reproductive and lymphatic systems. With intrauterine infection with toxoplasmosis in the first months of pregnancy, fetal death is most often

observed, which leads to spontaneous abortion or stillbirths. With later infection, a fetal brain development disorder occurs, meningoencephalitis often develops, sometimes inflammation of the membranes of the eye.

8. Laboratory diagnosis – laboratory diagnosis is based on the use of immunological methods and polymerase chain reaction (PCR). Sometimes it is possible to detect parasites in blood smears, punctured lymph nodes and cerebrospinal fluid.

9. Preventive measures: *personal* – heat treatment of food of animal origin (meat, eggs, milk), sanitary control at meat processing plants, prevention of close contacts of children with pets and stray cats. *Public* prophylaxis are detection and treatment infected people; sanitary control of food production, shops and markets; elimination of flies.

Order Hemosporidia

Blood sporozoa are developing in red blood cells of vertebrates. The representatives of the Plasmodium genus cause malaria. This disease is of greatest medical importance.

MALARIA.

Characteristic of parasitic disease:

1. The name of the parasite: *plasmodium vivax* – the causative agent of three-day malaria; *plasmodium malariae* – causative agent of four-day malaria; *plasmodium falciparum* – the causative agent of tropical malaria; *plasmodium ovale* is the causative agent of oval malaria. Malaria is vector-borne disease.

2. Geographical distribution – widespread in regions with a tropical and subtropical climate, the latter – only in tropical Africa.

3. Location (place of localization) – blood (RBs), cells of liver.

4. The mechanism of human infection:

4.1. Sources of invasion – a sick person and monkeys (natural reservoirs);

4.2. Invasion stage – sporozoite;

4.3. The way of invasion – transmissible (with mosquitoes bite), blood transfusion from a sick person to a healthy person (transfusion method), as well as transplantally from the mother to the fetus (intrauterine method);

4.4. Factors of invasion – mosquito saliva;

4.5. Vectors (specific) – female mosquitoes (genus *Anopheles*).

5. Life cycle (Fig.9). The definitive host is the female mosquito of the genus Anopheles, the intermediate is ahuman. When a mosquito bites, sporozoites penetrate the blood of a person, then they spread through the bloodstream to different organs and tissues, including into the liver cells, where they switch to intracellular parasitization: they grow, turn into trophozoites. In hepatocytes, trophozoites multiply by schizogony. The process lasts 15 days and corresponds to the incubation period of the disease.

After the destruction of the liver cells, tissue trophozoites enter the bloodstream and penetrate red blood cells.

Being inside red blood cells, trophozoites growand divided by schizogony. The erythrocyte membrane breaks down and the merozoites, as well as toxic products of their vital activity, exit into the blood plasma. This stage corresponds to an attack of malaria, which is due to the absorption of red blood cell decay products and the vital activity of parasites. Then the merozoites penetrate into new red blood cells and the cycle continues until the next erythrocyte decay. The duration of trophozoite development between two bouts of malaria varies for different types of plasmodia and is 24 hours for Pl. falciparum, 48 hours at Pl. vivax and Pl. ovale, 72 hours at Pl. malariae.

In some red blood cells, merozoites are transformed into male (micro-mammoths) and female (macro-mammoths) germ cells, the further development of which can only occur in the mosquito.

A mosquito, attacking an infected person, swallows red blood cells containing gamonts during bloodsucking. In the mosquito's stomach, gamonts turn into mature germ cells – gametes, then they merge: the mobile microgame fully

penetrates the macrogamet, resulting in the formation of a diploid zygote, which is transformed into an ookinet (mobile stage). Ookineta penetrates through the wall to the outer surface of the mosquito's stomach, here it encysts and turns into an oocyst.

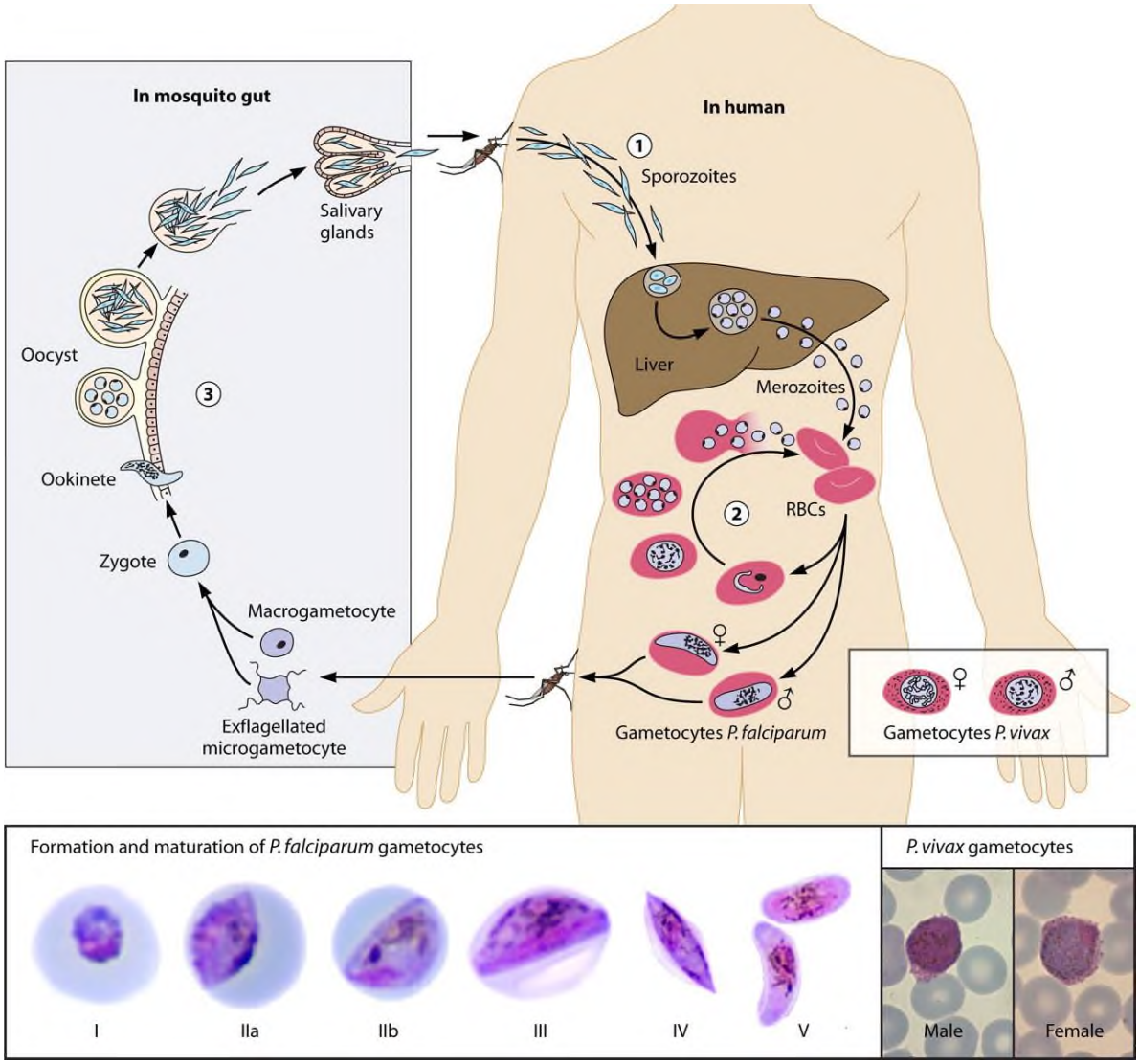


Fig. 9. Life cycle of *Plasmodium* (https://cmr.asm.org)

The oocyst grows, the plasmodium inside it is repeatedly divided by sporogony, as a result of which thousands of sporozoites are formed. The shell of the mature oocyst is torn and the sporozoites appear in the mosquito hemolymph. They migrate throughout the body of the insects and accumulate in the salivary glands. The entire process of plasmodium development in the mosquito body, depending on the external temperature, lasts from 7 to 45 days.

6. Pathogenic effect. The products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause damage of liver cells and RBCs.

7. Clinical symptoms. Malaria is characterized by a cyclical course, when after a period of relative well-being, paroxysm of malaria begins in 2–3 days. The **malaria paroxysm** comprises three successive stages. The first is a 15-to – 60 minute cold stage characterized by shivering and a feeling of cold. Next comes the 2-to – 6 hour hot stage, in which there is fever, sometimes reaching 41°C, flushed, dry skin, and often headache, nausea, and vomiting. After a few hours, a sharp drop in temperature occurs, accompanied by torrential sweat, after which patients usually fall asleep. The entire attack of malaria lasts from 6 to 12 hours, the interictal period lasts from 48 to 72 hours. As a result of complications from the central nervous system or kidneys, the patient may die. The number of attacks of malaria can reach 10–15, after which they cease due to the formation of immunity, but the parasites remain in the liver and the person becomes a parasite carrier. It poses a danger to others as a reservoir host.

8. Laboratory diagnosis. Laboratory diagnosis of malaria is based on a laboratory study of the patient's blood during an attack or immediately after it, when microscopic analysis reveals various stages of the development of malarial plasmodium in red blood cells. Immunodiagnostics and PCP analysis are used.

9. Preventive measures. The World Health Organisation (WHO) has developed a simple 4 letter ABCD tool to help travellers safely prepare for ventures in any corner of the world:

- **A**wareness of risk
- **B**ite prevention
- **C**hemoprophylaxis
- **D**iagnosis.

You can protect yourself against malaria and must do each time you visit a country that is at risk. No one has full immunity, therefore you should still protect yourself if you grew up or lived in an area that is at risk of malaria. Use the **ABCD** approach for protection:

A– AWARENESS OF RISK.

Before you travel, you need to think about whether where you are headed has a risk of Malaria. Sub-Saharan Africa, some areas of South East Asia and South America carry a particularly high risk of malaria transmission. It is important that you are clued up on the risk in the area you are travelling.

Seasonal rainfall can increase mosquito breeding and as a result, in some areas, the risk of Malaria can be highly seasonal. So make sure you think about the time of year you are travelling too.

For a full breakdown of what injections or medication you will need to check out our handy **Malaria Risk Map**.

Alternatively, you can talk to your Pharmacist, GP or any other trained health professional.

B– BITE PREVENTION.

Whether you are headed to a high or low-risk area, it is important that you try to avoid being bitten by mosquitos. In short, the fewer bites you get, the safer you are.

There are many **repellents** on the market but the UK Government Guidelines for **Malaria Prevention** recommends the use of repellents containing DEET at a concentration of 20 % or over for maximum protection. The higher the strength of DEET the less frequently you will need to apply the repellent. DEET up to a concentration of 50 % is considered suitable for infants over 2 months, breastfeeding mothers and pregnant ladies although the product packaging may differ.

Mosquitos which transmit malaria typically bite between dusk and dawn so ensuring you apply repellent to any exposed areas of skin in the evenings is crucial. You should also ensure you sleep with a **mosquito net** particularly if

you are staying in budget hostels without air conditioning. It is also worthwhile to ensure you have some form of spray repellent that can be applied such as the **Lifesystems Expedition Plus Spray** or **Jungle Formula Maximum Strength Body Spray**.

C– CHEMOPROPHYLAXIS.

In some areas where there is a risk of Malaria, it is advised that you use **antimalarial medication** to reduce your risk of contracting malaria should you get bitten.

The medication you need will vary dependent upon:

- Where you are travelling
- Your medical status
- Personal preference

Not all antimalarials are suitable for all areas. This is because the malaria parasite has developed resistance to some of the drugs used in some areas of the world. Be sure you obtain the right medication for the area you are travelling.

Some **antimalarials** will require a prescription from your doctor or another suitably qualified person such as an Independent Prescriber which can be completed online, so make sure you leave plenty of time to get it sorted out.

Remember, it is important you take the tablets exactly as prescribed and finish the course to ensure you are properly protected.

D– DIAGNOSIS.

Nothing is absolute. Even if you follow all these measures there is still a small chance of you contracting malaria.

After you return from your trip, it is essential that you report any fever, diarrhoea, vomiting or shivering occurring within a year of your return.

Prompt diagnosis of malaria ensures you get the right treatment when you need it and ultimately, improves your chances.

Follow this simple ABCD to get clued and stay protected on your travels.

To get your antimalarial tablets before you travel you can use our **online consultation service**, this means you won't have to book an appointment with your doctor!

CONTROL QUESTIONS

1. Zoological classification and general features of kingdom *Protozoa*, Subphylum *Sarcodina* and *Ciliata.*

2. Zoological classification, epidemiology (geographical distribution), peculiarities of morphology, the mechanism of human infection, life cycle, pathogenicity of ameba (*Entamoeba histolytica, Entamoeba coli, Entamoeba gingivalis*).

3. Clinical features, laboratory diagnosis, prevention and prophylaxis of amebiasis.

4. Zoological classification, epidemiology, geographical distribution, morphology, the mechanism of human infection, life cycle, pathogenicity of balantidium (*Balantidium coli*).

5. Clinical features, laboratory diagnosis, prevention and prophylaxis of balantidiasis.

6. Zoological classification and general characteristics of Class *Mastigophora*.

7. Zoological classification, morphology, geographical distribution, life cycle, mode of transmission and pathogenesis of giardia.

8. Clinical features, laboratory diagnosis, prevention and prophylaxis of lambliasis.

9. Zoological classification, morphology, geographical distribution, life cycle, mode of transmission and pathogenesis of vaginal, oral and intestinal trichomonads.

10. Clinical features, laboratory diagnosis, prevention of urogenital trichomoniasis.

11. Zoological classification, morphology, geographical distribution, life cycle, mode of transmission and pathogenesis of trypanosomes (*Trypanosoma brucei gambiense, Trypanosoma brucei rhodesiense, Trypanosoma cruzi*).

12. Clinical features, laboratory diagnosis, prevention of african trypanosomia-

sis and american trypanosomiasis.

13. Zoological classification, morphology, geographical distribution, life cycle, mode of transmission and pathogenesis of leishmania (*Leishmania tropica*, *Leishmania donovani*).

14. Clinical features, laboratory diagnosis, prevention of cutaneous, mucocutaneous and visceral leishmaniasis.

15. Zoological classification and general features of Phylum *Apicomplexa* and Class *Sporozoa*.

16. Zoological classification, morphology, geographic distribution, life cycle (Toxoplasma gondii). Modes of infection, mode of transmission, pathogenesis and clinical features, laboratory diagnosis, prevention public and personal of toxoplasmosis.

17. Zoological classification, morphology, geographic distribution, life cycle (*Pneumocystis* pneumonia.). Modes of infection, mode of transmission, pathogenesis and clinical features, laboratory diagnosis, prevention general and personal of pneumocystosis.

18. Zoological classification, morphology, geographic distribution, life cycle (*Sarcocystis*). Modes of infection, mode of transmission, pathogenesis and clinical features, laboratory diagnosis, prevention general and personal of sarcocystosis.

19. Zoological classification and morphology of malarial plasmodium (*Plasmodium vivax).*

20. Malaria is the transmissible disease: geographic distribution, life cycle of malarial plasmodium (*Plasmodium vivax).*

21. Malaria pathogenicity and clinical features depending on the life cycle pathogen. Laboratory diagnosis, public and personal prevention of Malaria. The success of the fight against malaria and the tasks of the antimalarial service at the present stage.

CHAPTER 3
MEDICAL HELMINTHOLOGY

Medical helminthology studies parasitic worms and human diseases caused by them – helminthiasis. Currently, over 250 types of helminthes are known to be parasitic in humans, 65 of which are found in Russia. In some regions of the world, helminthiasis affects up to 80–90 % of the population, mainly children. According to official WHO data, about 3 to 4 billion people in the world are affected annually by helminthiasis. Helminthiases are most widespread in countries with a hot climate (Africa, Asia, Latin America, etc.).

A great contribution to the creation of the science of helminthology was made by domestic scientists. So, academician K.I. Scriabin formulated the basic principles of the fight against helminthiases. In 1925, K.I. Skryabin put forward the principle of deworming, which includes a set of measures for the treatment of the patient and the destruction of eggs and larvae of helminthes in the external environment. Later, in 1944, he put forward the principle of devastation, which implies the complete elimination of helminthes as a species in a certain territory.

In 1929, K. I. Skriabin and R. S. Shultz proposed an epidemiological classification of helminthes, based on the peculiarities of the parasite's life cycle, according to which geohelminthes and biohelminthes are distinguished. Geohelminthes include species that develop without the participation of an intermediate host. Usually part of the life cycle of geohelminthes passes in the soil with access to oxygen. Biohelminthes pass one part of the development cycle in the human organism, the other in the body of one or more intermediate hosts. Therefore, for the distribution of biohelminthes in the environment, a specific intermediate host is required, due to which there are natural foci of these diseases.

Diseases caused by different groups of helminthes, respectively, are divided into geohelminthoses and biohelminthoses. All helminthiases are charac-

terized by a relatively slow development, a chronic course, often with prolonged compensation. More pronounced pathological changes cause larval stages of helminthes. With most intestinal helminthiases, parasitization of single individuals in the human body is often asymptomatic.

All helminthes have common organizational features: firstly, they are multicellular animals; secondly, characterized by the presence of bilateral symmetry; thirdly, they develop from three germ layers.

Helminthes of medical importance are two types of invertebrate animals: Flatworms (Plathelminthes) and Roundworms (Nemathelminthes).

Zoological classification of helmintes (Dogel V.A., 1981)

Kingdom: Animalia (Zoa)

Subkingdom: Metazoa

Phylum: Plathelminthes

Class: Trematoda

Species: *Fasciola hepatica, Opisthorchis felineus, Dicrocoelium lanceatum, Paragonimus westermani, blood flukes – Schistosoma mansoni; Sch. japonicum; Sch. Haematobium*

Class: Cestoda

Species: *Taenia solium, Taeniarhynchus saginatus, Diphyllobotrium latum, Echinococcus granulosus, Alveococcus multilocularis, Hymenolepis nana.*

Phylum: Nemathelminthes

Class: Nematoda

Species: Geohelminthes: *Ascaris lumbricoides, Ancylostoma duodenale, Necator americanus, Strongyloides stercoralis, Trichocephalus trichiurus, Enterobius vermicularis.*

Biohelminthes: *Trichinella spiralis, Dracunculus medinensis, Wuchereria bancrofti, Onchocerca volvulus, Loa-Loa, Dirofilaria repens, Toxocara canis.*

General Description of Plathelminthes.

Flatworms are characterized by the development of three germ layers (ectoderm, endoderm, mesoderm); the presence of bilateral symmetry. The body is flattened, has a leaf-like or tape-like shape.

The skin-muscle bag consists of cover tissue – the tegument, and three layers of smooth muscles. The *body cavity* is absent, the space between the organs is filled with parenchyma. The following organ systems are developed: *nervous, digestive (*only in Trematodes*), excretory and reproductive*. *The circulatory and respiratory systems are absent*, gas exchange is carried out by the entire surface of the body.

In the process of evolution, flatworms formed adaptations to a parasitic way of life, which include high fertility, the presence of fixation organs, a complex life cycle with a change of owners and alternating stages. All plathelminthesare biohelminthes.

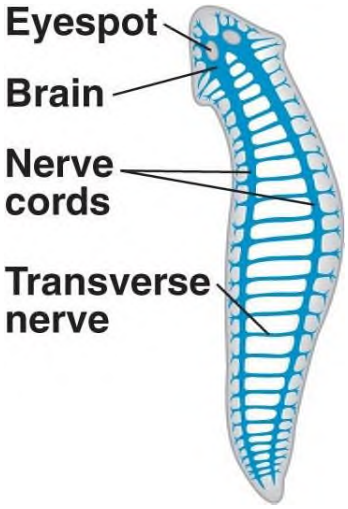
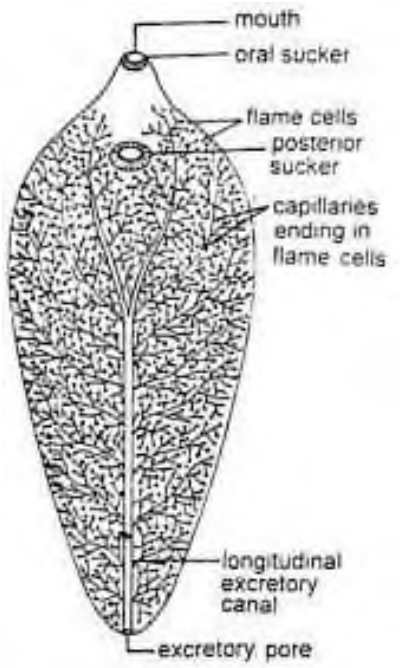
Plathelminthesof two classes are of medical importance: Trematoda and Nematoda.

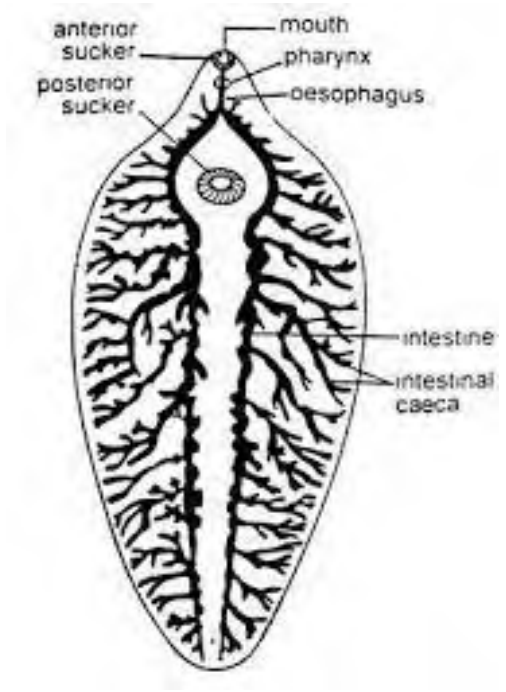
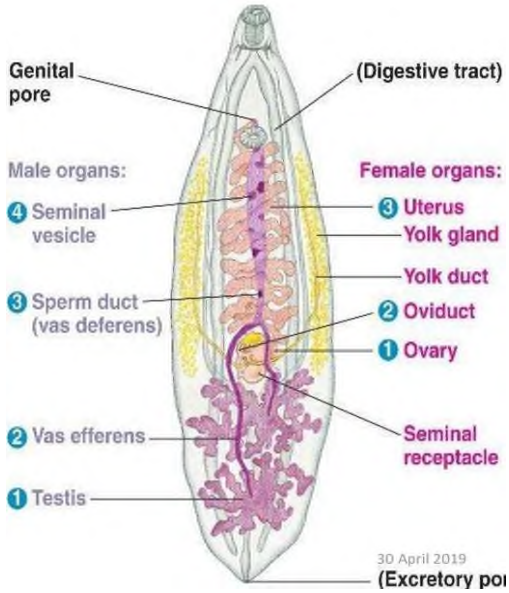
PHYLUM PLATHELMINTHES

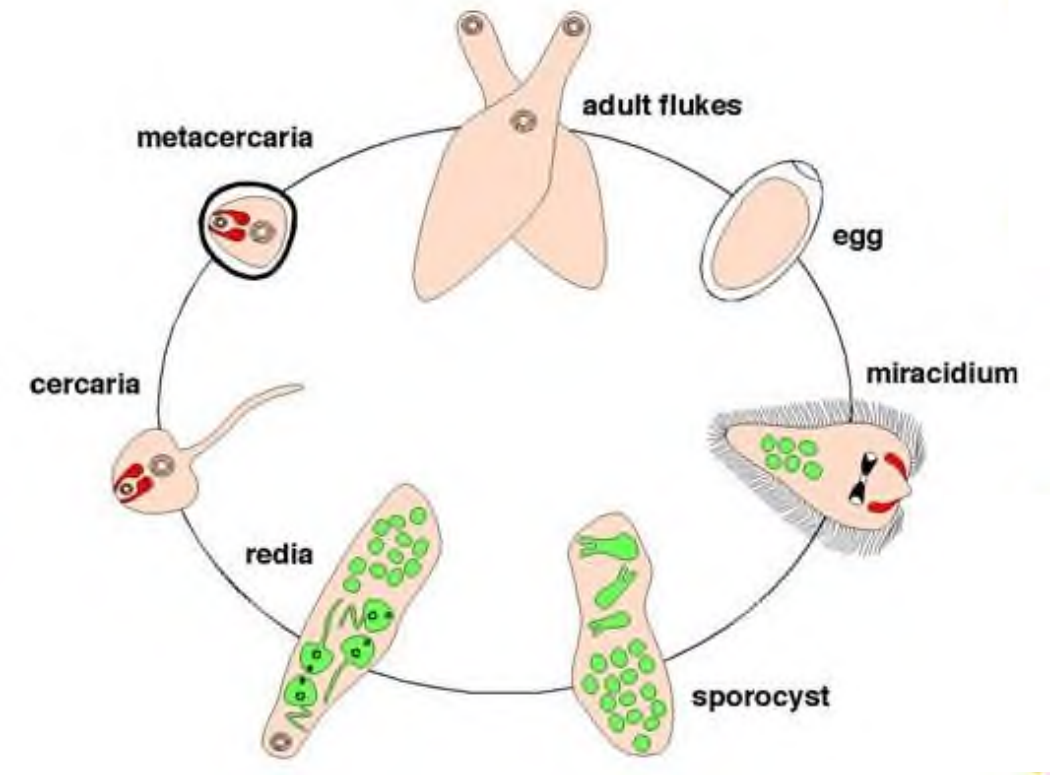
CLASS TREMATODA

Currently, about 3 thousand species of representatives of the class Trematoda are described, all of them lead a parasitic lifestyle. Trematodes are small (length from several millimeters to 7 cm), the body is flat, leaf-shaped. To adapt to a parasitic lifestyle, they have two powerful suckers (oral and abdominal), from where the old name for flukes is fluke.

Structure of organ systems of Trematoda

	***The Nervous system*** has a ladder-nodal structure similar to *orthogon*. At the front end of the body is the nerve ring form brain. Two lateral nerve cords are connected by transverse nerves (comissures).
	***The Excretory system*** has *protonephridial type*. It is represented by terminal cells and excretory tubules. Terminal cells are equipped with cilia, which ensure the movement of fluid into the cells and into the tubules. Excretory tubules are connected into the excretory canal that open outward with excretory pore. Protonephridia removes the products of dissimilation and regulates the osmotic pressure in the body cavity of the helminthes.

	***The Digestive system*** is developed only in Trematodes, in which it is blindly closed and has two sections: the anterior intestine (mouth, pharynx, esophagus) and the middle intestine. Undigested food is released through the mouth opening. Cestodes don't have digestive system.
	***The Reproductive system.*** The plathelminthes are hermaphrodites. The male reproductive system is represented by paired testes and vas deferens, the ejaculatory duct, and cirrus. The female reproductive system includes ovaries, oviducts, vitellines with ducts, the receiving seed, the body of Mellis (this gland produces ova shell fluid), ootype (chamber for fertilization and egg formation) and uterus. The ova, moving along the oviduct, first get into ootype, where fertilization and formation of the egg membranes takes place. From the ootype, eggs enter the uterus and out through the sexual pore.



The life cycle of Trematoda is characterized by a change of owners and an alternation of larval stages.

Adult worm is a parasite in the body of the definitive (final) host mainly in vertebrate herbivorous mammals (sometimes in humans). *The eggs* secreted by it for further development should fall into the water. In water, a *larva* (miracidium) emerges from an egg, which move and penetrate the body of the first intermediate host – the mollusk. In the body of the mollusk, the larvae divided (form sporocyst and redia). Then the larva emerges from the body of the mollusk, fall into the water, and turns into a cercaria. Cercaria enters the body of the second intermediate host and turns into the last larva stage – *metacercaria* – ***invasive stage***.

The life cycle includes the following stages: adult worm (sexually mature), egg, larva, cyst.

Fasciola hepatica* cause *FASCIOLOSIS.

Life cycle. *Adult worm*is localized in the bile ducts of the liver of definitive hosts (herbivorous mammals, sometimes humans). Eggs are passed in feces that contaminate river or lake water. In water, a *larva* emerges from an egg, which move and penetrate the body of the intermediate host –the fresh-water snail (mollusk). In the body of the mollusk, the larvae divided. Then the larva emerges from the body of the – mollusk, fall into the water, and turns into a *metacercaria* – it is invasion stage for human. Cyst together with the grass can be swallowed by the definitive hosts (herbivorous vertebrates, humans), in the intestines of which the membrane dissolves, the larva penetrates the bile ducts of the liver and there it turns into an adult worm (Fig. 10).

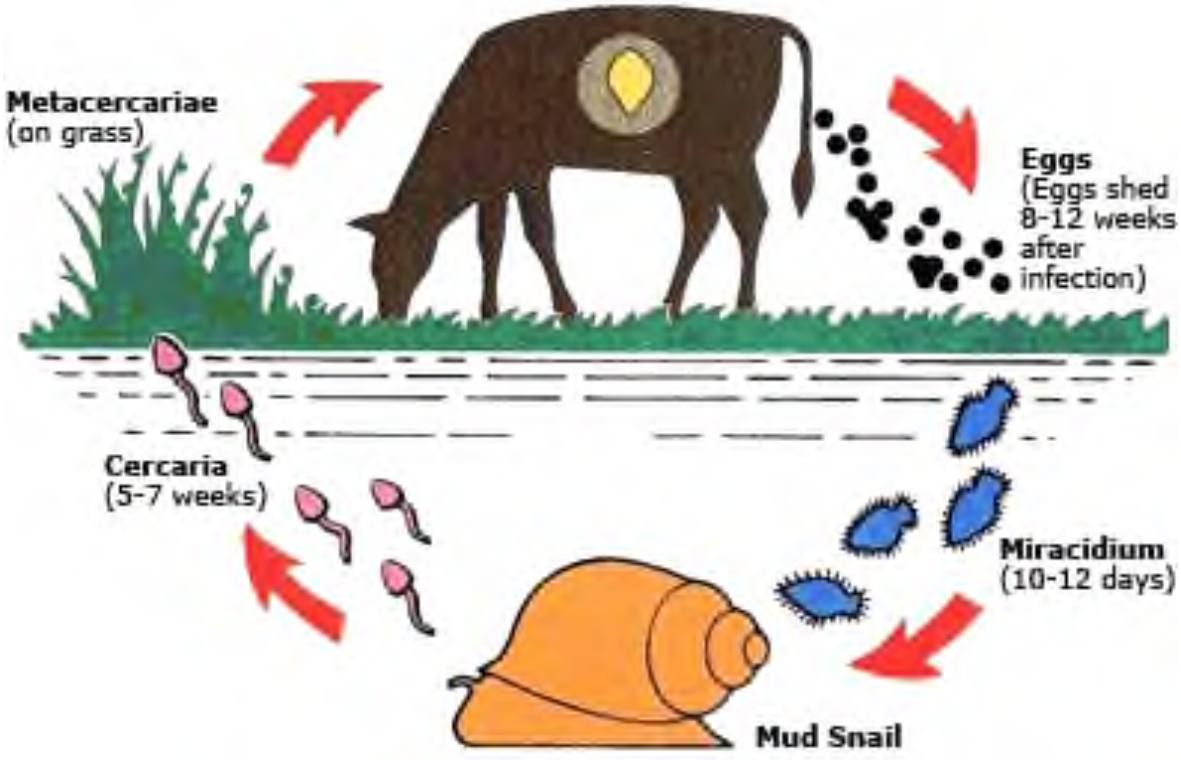


Fig.10. The life cycle of *Fasciola hepatica.*

Characteristics of *FASCIOLOSIS:*

1. Name of the parasite (in Latin) – *Fasciola hepatica*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Trematoda.

3. Structure, organ systems.

Adult worm: It is large in size, flat leaf-shaped fluke measuring 30 mm long. There are two suckers: *oral* and *abdominal*.

4. Disease name – fasciolosis.

5. Geographical distribution – the disease is common everywhere.

6. Sources of invasion – infected human and various mammals.

7. The way of penetration – alimentary.

8. Invasion stage for human – cyst.

9. Factors of invasion – water contaminated by cysts from river or lake.

10. Place (organ) of localization in the human body – liver, bile ducts.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of liver organ tissues, inflammation in the bile ducts. This can lead to the development of cholecystitis, hepatitis, cirrhosis.

12. Clinical symptoms – pain in the liver, jaundice, anemia, food intolerance, nausea, vomiting.

13. Laboratory diagnosis – immunological methods, PCR, detection of eggs in feces and in duodenal fluidby microscopy;

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – you should to comply with hygiene rules – avoid drinking waterdirectly fromlakes or river, avoid washing hands and fruits, vegetables by water from the river or lake; *public* –detection and treatment infected people; prevention of contamination of the environment and water sources with human and animal feces, personal and public health education.

Opistorchis felineus* cause *OPISTHORCHOSIS.

Life cycle. Definitive hosts are human, cats and various mammals. There are two intermediate hosts in the developmental cycle of *Opisthorchis felineus*: the first – fresh-water mollusk, the second – fish.

Adult worm is localized in the bile ducts of the liver, sometimes in the ducts of the pancreas and in the gall bladder. *Eggs* are passed in feces that con-

taminate river or lake water. In water, a *larva* emerges from an egg, which move and penetrate the body of the intermediate host –the fresh-water snail (mollusk). In the body of the mollusk, the larvae divided. Then the larva migrates from the mollusk to the second intermediate host – fish, where it turns into a *metacercaria.* Human becomes invased by eating poorly cooked fish (Fig. 11).

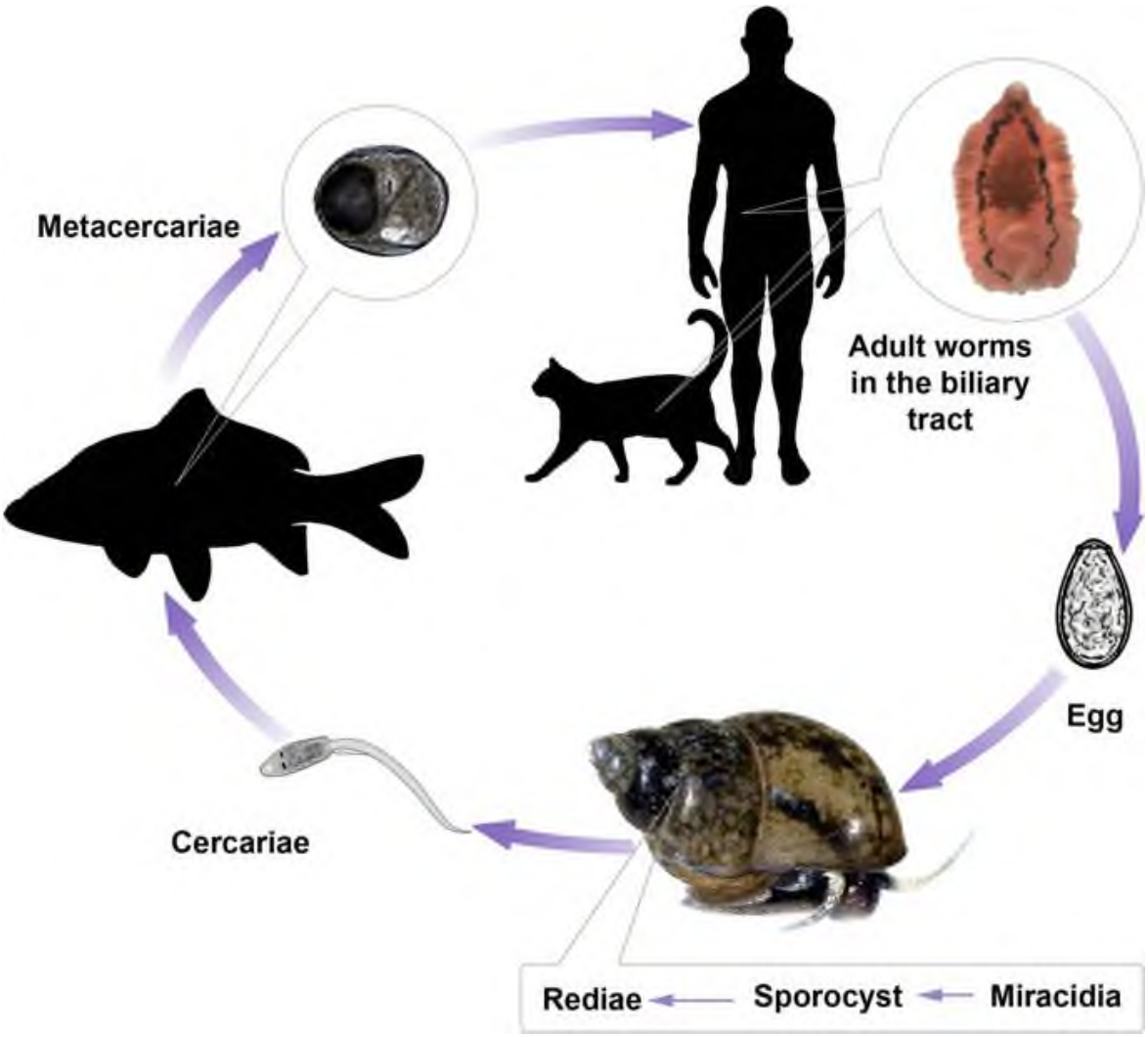


Fig. 11. The life cycle of *Opisthorchis felineus*
(https://bmcgenomics.biomedcentral.com)

Characteristics of *OPISTHORCHOSIS:*

1. Name of the parasite (in Latin) – *Opisthorchis felineus*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Trematoda.

3. Structure, organ systems. *Opisthorchis felineus* is a leaf-shaped fluke. It sizes are from 10 to 12 mm in length. The fluke is tapered at the anterior end and rounded at the posterior end. There are two suckers: *oral* and *abdominal*.

4. Disease name – opisthorchosis.

5. Geographical distribution – natural foci of opisthorchosis are located in Western Siberia, the European part of Russia along the banks of large rivers.

6. Sources of invasion – infected human and various mammals.

7. The way of penetration – alimentary.

8. Invasion stage for human – cyst.

9. Factors of invasion – undercooked fish meat infected by cyst.

10. Place (organ) of localization in the human body – liver, bile ducts.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of liver organ tissues, inflammation in the bile ducts. This can lead to the development of cholecystitis, hepatitis, cirrhosis, pancreatitis, sometimes even liver cancer.

12. Clinical symptoms – about 80 % of infected people have no symptoms. Pain in the liver, jaundice, anemia, food intolerance, nausea, vomiting, pancreas. Chronic infection may lead to carcinoma – a malignant cancer of the bile ducts.

13. Laboratory diagnosis – immunological methods, PCR, detection of eggs in feces and in duodenal fluid by microscopy.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – you should to comply with hygiene rules – avoid eating undercooked fish. *Public* –detection and treatment infected people; prevention of contamination of the environment and water sources with human and animal feces, personal and public health education.

Paragonimus westermani* cause *PARAGONIMOSIS.

Life cycle. In the development cycle of *Paragonimus westermani* there are also two intermediate hosts: the first is a freshwater mollusk, the second is crabs or crayfish. Human infection occurs when eaten poorly thermally processed crabs and crayfish containing *cyst* – it is invasion stage for human. (Fig. 12).

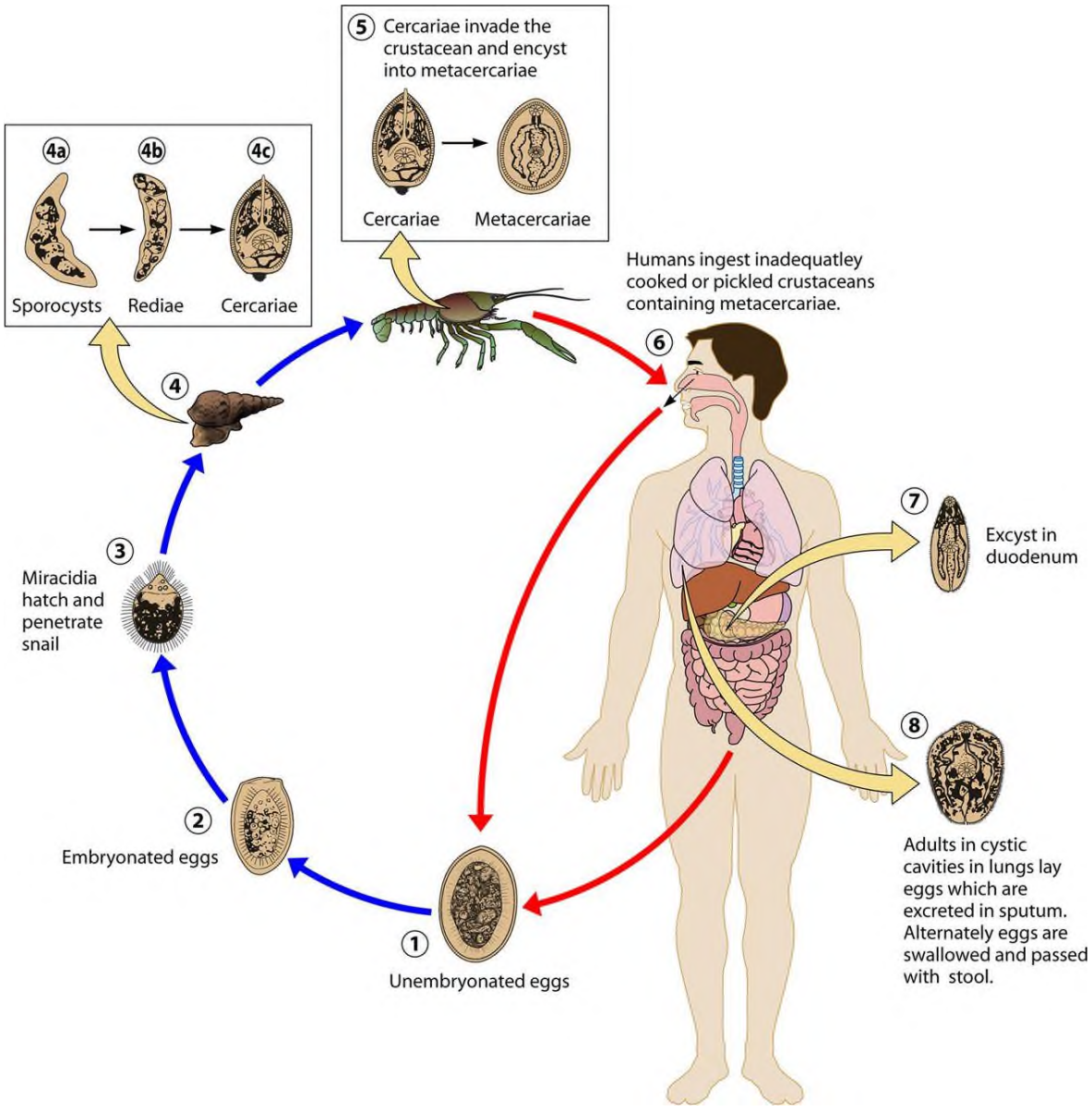


Fig. 12. The life cycle of *Paragonimus westermani* (https://cmr.asm.org)

Characteristics of *PARAGONIMOSIS:*

1. Name of the parasite (in Latin) – *Paragonimus westermani.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Trematoda.

3. Structure, organ systems. The *Paragonimus westermani*adult worm is egg-shapedabout 10 mm long. There are two suckers: *oral* and *abdominal*.

4. Disease name – Paragonimosis.

5. Geographical distribution –common in Russia (seaside), South-Eastern Asia and South Asia and South America.

6. Sources of invasion – infected human and various mammals.

7. The way of penetration – alimentary.

8. Invasion stage for human – cyst.

9. Factors of invasion – undercooked crab and crayfish meat infected by cyst.

10. Place (organ) of localization in the human body – bronchioles, lungs.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of lung organ tissues. Complications: pulmonary heart disease, brain abscesses, meningoencephalitis.

12. Clinical symptoms – disease causes pains in the chest, breathlessness, cough with purulent sputum and sometimes with blood, high temperature, headache.

13. Laboratory diagnosis – immunological methods, PCR, detection of eggs in sputum or feces by microscopy.

14.Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* –you should to comply with hygiene rules – avoid eating undercooked crab and crayfish meat. *Public* – detection and treatment infected people; prevention of contamination of the environment

and water sources with human and animal feces, personal and public health education.

Dicrocelium lanceatum* cause *DICROCOELIOSIS.

Life cycle. In the *Dicrocelium lanceatum*, unlike other flukes, the development cycle takes place on the soil, in moist grass.

Adult worm is localized in the bile ducts of the liver, sometimes in the ducts of the pancreas and in the gall bladder. *The eggs* are passed in feces of the definitive hosts (mainly herbivorous mammals) that contaminate the ground. The *larva* emerges from an egg, which move and penetrate the body of the intermediate host – the ground mollusk. In the development cycle of *Dicrocelium lanceatum*, there are also two intermediate hosts: the first is a ground mollusk, the second is ants. Further development takes place in the body of ants after swallowing *larva*, where *cyst* are formed. It is invasion stage for human. Several cysts are penetrate the antipharyngeal ganglion of the ant and, cause it to have a cataleptic state. While such ants are on plants and berries in a stationary state, they are swallowed by the definitive host (herbivores). People, usually children, are random hosts. In Russia, the disease is cosmopolitan, especially among herbivores (Fig. 13).

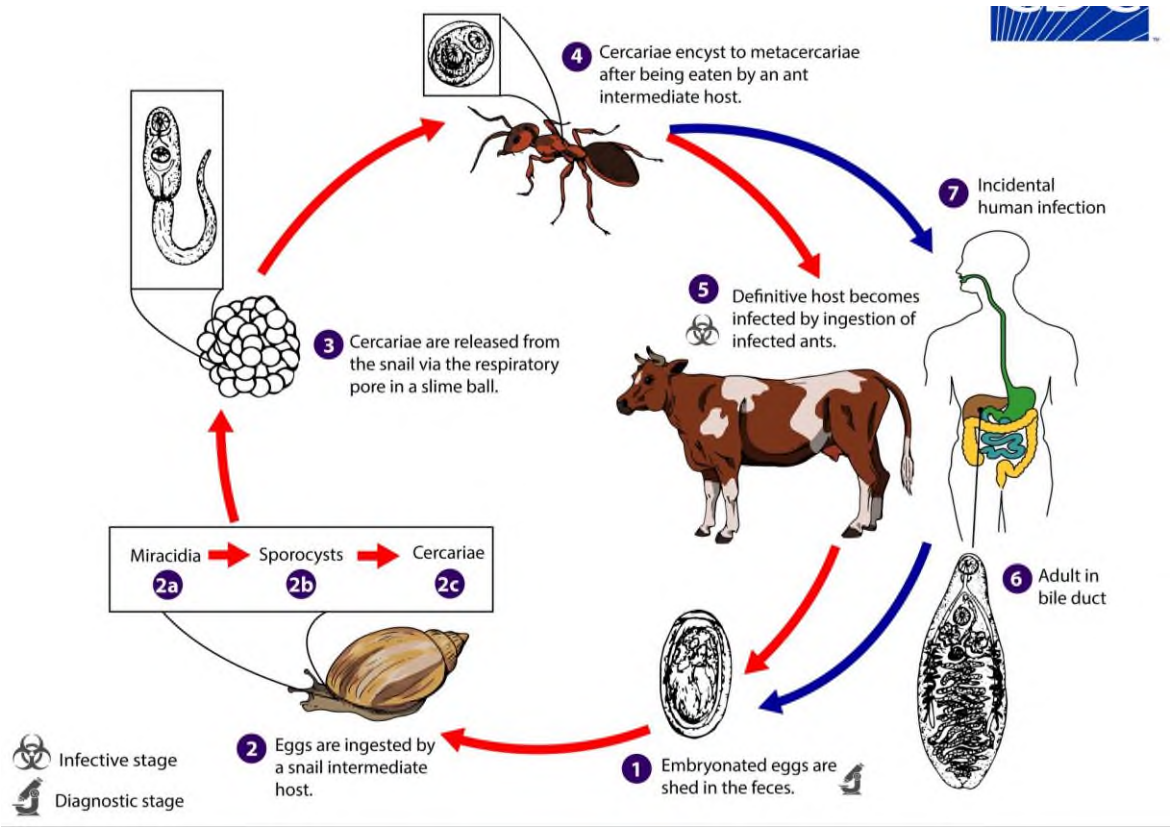


Fig. 13. The life cycle of *Dicrocelium lanceatum* (https://www.cdc.gov)

Characteristics of *DICROCOELIOSIS*:

1. Name of the parasite (in Latin) – *Dicrocelium lanceatum.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, classTrematoda.

3. Structure, organ systems (figure). *Dicrocoelium lanceatum*is a leaf-shaped fluke of a lancet-shaped form. Its sizes are from 10 to 12 mm in length. There are two suckers: *oral* and *abdominal*.

4. Disease name – Dicrocoeliosis.

5. Geographical distribution – the disease is common everywhere.

6. Sources of invasion – herbivores.

7. The way of penetration – alimentary.

8. Invasion stage for human – cyst.

9.Factors of invasion – ants infected by cyst.

10. Place (organ) of localization in the human body – liver, bile ducts.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of liver organ tissues, inflammation in the bile ducts. This can lead to the development of cholecystitis, hepatitis, cirrhosis.

12. Clinical symptoms – pain in the liver, jaundice, anemia, food intolerance, nausea, vomiting.

13. Laboratory diagnosis – immunological methods, PCR, detection of eggs in feces and in duodenal fluid by microscopy.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* –you should to comply with hygiene rules – avoid contamination of food by ants and avoid the ingestion of ants. *Public* –detection and treatment infected people; prevention of contamination of the environment with human and animal feces, personal and public health education.

Shistosoma* cause *SCHISTOSOMOSIS.

Life cycle. Marita parasitizes in blood vessels, namely the veins of the small pelvis or intestines (depending on the type of parasite). Mature females lay eggs equipped with a sharp spike, through which enzymes that dissolve the vessel wall are secreted. Due to this, eggs enter the cavity of the bladder or intestines and are secreted out with the urine or feces of the host. For further development, the eggs must fall into the water, where larva equipped with cilia form from them. Larva penetrate into the body of an intermediate host – the mollusk, where it develops. The last sage of larva have a powerful tail, and glands of penetration at the front end of the body. By actively introducing larva through the skin, they fall into the circulatory system of the final host when it is in water (Fig. 14).

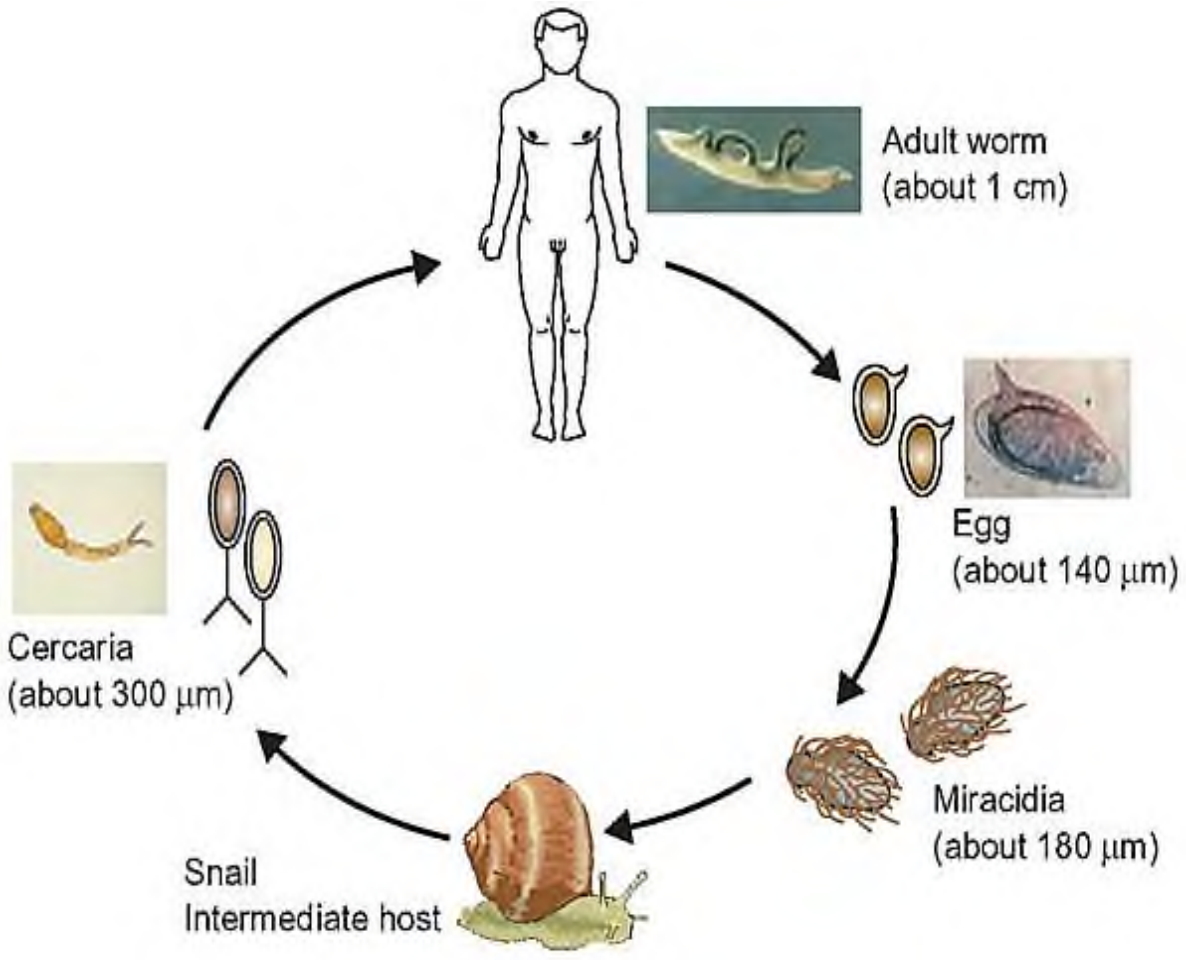


Fig. 14. The life cycle of *Shistosoma* (https://figshare.com)

Characteristics of *SCHISTOSOMOSIS*:

1. Name of the parasite (in Latin) – *Shistosoma haematobium*; *Shistosoma mansoni*; *Shistosoma japonicum*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Trematoda.

3. Structure, organ systems. Dioecious (have separate sexes). Male's body is short and broad (10–15 mm), female's body is up to 20 mm. Females are situated in the gynecophoral canals on the abdominal side of male flukes. Males have a developed abdominal sucker, which ensures a reliable fixation to the walls of blood vessels.

4. Disease name – Schistosomosis.

5. Geographical distribution – the disease is common in the warm countries with a tropical and subtropical climate (Asia, Africa and America).

6. Sources of invasion – infected human and various mammals.

7. The way of penetration – alimentary, percutaneous.

8. Invasion stage for human – larva.

9. Factors of invasion – water contaminated by larva.

10. Place (organ) of localization in the human body – veins of the abdominal cavity and urogenital system.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of organ tissues (veins of the abdominal cavity and urogenital system).

12. Clinical symptoms – dermatitis and itching at the site of cercaria invasion. During the migration of young schistosomes there are cough with mucus and spitting of blood, symptoms of bronchial asthma with general malaise, headache, weakness and loss of appetite. Symptoms of genitourinary schistosomiasis are disuria (impairment of micturition) hematuria (excretion of blood in urine), painful micturition. Characteristic signs of gastrointestinal schistosomiasis are pains in the abdomen, irregular bowel movements, blood and mucus in feces, diarrhea, oedema of lower extremities and the abdomen.

13. Laboratory diagnosis – immunological methods, PCR, detection of eggs in urine or in feces.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* –avoid prolonged contact with water while swimming or working in water in the territory where the disease spreads. *Public* –detection and treatment infected people; prevention of contamination of the water sources with human feces, personal and public health education.

CONTROL QUESTIONS

1. Helminthology and its sections.
2. The concept of the cycle life and of hosts in helminthes. The helminthes pathogenicity in humans.
3. Classification of helminthes. Epidemiological classification helminthosis.
4. Contributions of Skrjbin K.I. to medical helminthology. Definition of terms «devastation» and «dehelmintization».
5. Classification and general features of Phylum *Platheiminthes.*
6. Class *Trematoda*: general features.
7. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of liver fluke – *Fascioal hepatica*.
8. Laboratory diagnosis, general and personal prevention of fasciolosis.
9. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of liver fluke – *Opisthorchis felineus.*
10. Laboratory diagnosis, general and personal prevention of opistarchosis.
11. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of liver fluke – *Dicrocelium Lanceolatum.*
12. Modes of infection, localization and pathogenesis clinical features, laboratory diagnosis, general and personal prevention of dicroceliosis.
13. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of liver fluke – *Paragonimus westermani.*
14. Modes of infection, localization and pathogenesis clinical features, laboratory diagnosis, general and personal prevention of paragonimosis.
15. Zoological classification, morphology, geographic distribution, life cycle, mode of transmission and pathogenesis of blood flukes – *Schistosoma haematobium, Schistosoma mansoni, Schistosoma japonicum.*
16. Modes of infection, localization and pathogenesis clinical features, laboratory diagnosis, general and personal prevention of intestinal schistosomosis and urinary schistosomosis.

PHYLUM PLATHELMINTHES

CLASS CESTODA

In the class of tapeworms (Cestoda), there are more than 3 thousand species, nine of which are human parasites.

The sizes of tapeworms are very diverse, the body length varies from a few millimeters to several meters.

The body of the cestode is covered with tegument, under which the outer layer of the parenchyma and skin muscles are located. Deeper enough powerful layers of musculature, consisting of longitudinal peripheral and internal transverse muscles, lie deeper. The *body cavity is absent*, the space between the organs is filled with *parenchyma*. *The nervous system* of Cestodes (*ortogon*) consists of a central nerve node located in the head section and longitudinal nerve trunks extending from it, passing along the entire body. *Excretory system – protonephridial type* (see Trematodes).

In the process of evolution, due to its high specialization and parasitic lifestyle, cestodes have completely *lost their digestive organs*, nutrients are absorbed by the entire surface of the parasite's body. This is facilitated by microtrichia – special-purpose organelles resembling microvilli, due to the presence of which cestodes significantly increase the area of the suction surface. So, *digestive, circulatory,and respiratory organs in tapeworms are absent*.

The structure of the *reproductive system* of Cestodes is shown in Fig.15.

Initially, the beginnings of the male reproductive system (testes and excretory ducts) appear, later – the beginnings of the female reproductive system (ovaries, oviducts, yolks, testes, Melis body, ootype and uterus). The formed eggs enter the ootype, where they are fertilized, and then accumulate in the uterus.

Tapeworms are characterized by a flat ribbon-like shape, their body consists of three sections: the head (scolex), the neck and body (strobiles) (Fig. 16).

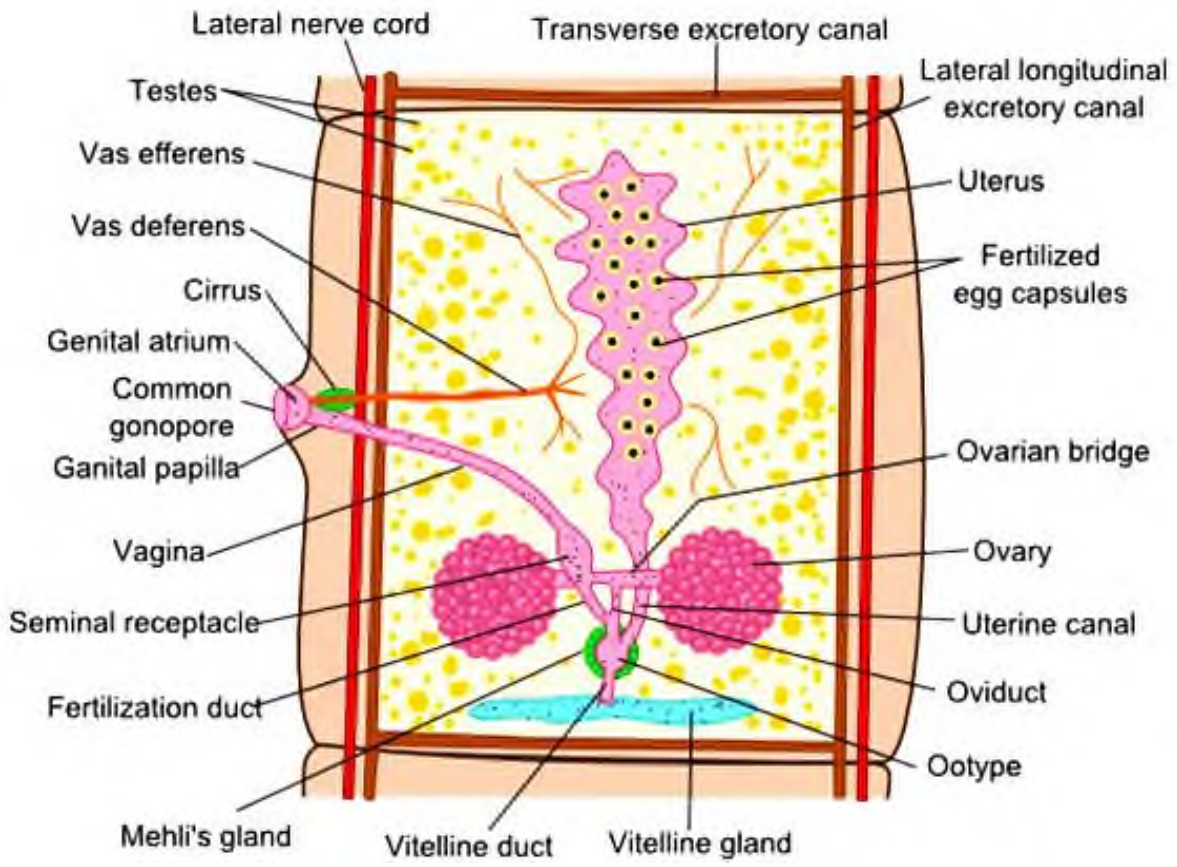


Fig. 15. Reproductive system of Cestodes (https://www.studyandscore.com)

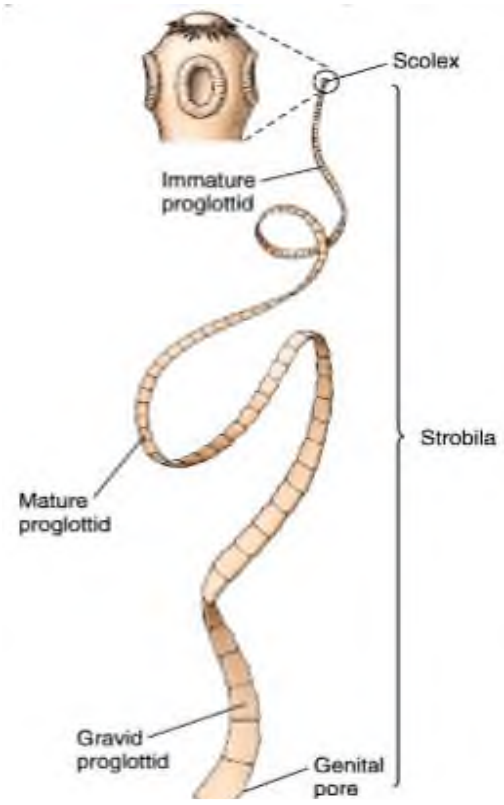


Fig. 16. Cestode structure (https://quizlet.com)

The scolex has the shape of a pinhead and serves mainly to fix on the mucous membrane of the intestines of the host.Representatives of the detachment chain, parasitizing in humans, have four suckers on the scolex, which are hemispherical hollow muscle formations that, pulling in the intestinal mucosa, infringe it with a muscular roller. In addition to suction cups, many cestodes have additional fixation organs on the scolex in the form of a proboscis with chitinous hooks.

The neck is the narrowest part of the body of the parasite. From the neck, new seg-

ments are constantly growing, which are part of the building.

*The strobila*consists of segments, or proglottids, the number of which varies significantly among representatives of different species and varies from three to four to several thousand. The segments in most cases are quadrangular in shape with different ratios of length and width. In the front of the body are young segments that have recently separated from the cervix and have an undeveloped reproductive system. Morphologically young segments are characterized by non-significant length and exceeding its width. With the growth and development of young segments, the beginnings of the reproductive system begin to appear in them, which in each segment develops completely independently.

As the joints mature, the male and then the female glands atrophy first. The mature segment, located at the very end of the body, contains only the uterus, filled with eggs. Inside the egg there is an embryo with three pairs of hooks (oncosphere), surrounded by a double-contoured cross-striated shell. In the process of helminthes growth, mature segments are constantly torn off and brought out, hermaphroditic ones become mature, and new young segments bud from the neck.

Life cycle. *The adult worm* – sexually mature stage – is localized in the intestine of the definitive (final) host. The segments secreted by the feces into the environment contain a uterus filled with *eggs*. When drying in the air, the shell of the segments and eggs collapses very quickly, the eggs freely disperse in the environment, birds and flies contribute to their distribution. Further development takes place in the body of the intermediate host, which becomes infected by swallowing the eggs, and often even the whole segment, with contaminated food, grass or water.

In the body of the intermediate host, *larva* emerges from an egg with the help of hooks are introduced into the wall of the small intestine, enter the blood or lymph vessels and are carried throughout the body, settling in different or-

gans, especially in the muscles, liver, lungs, brain and spinal cord. A *cyst (finn*) is formed from the larva – an invasive stage.

TENIASIS.

Life cycle. Human infection occurs when eating pork meat, fat or liver, invaded by the cyst (finn) (Fig. 17).

From the moment human swallows cyst to the appearance of the first mature segments, about two months pass. In the human intestine, under the influence of digestive enzymes, the scolex inside the cyst is turned out and attached to the mucous membrane in the upper part of the small intestine with the help of suction cups and hooks.

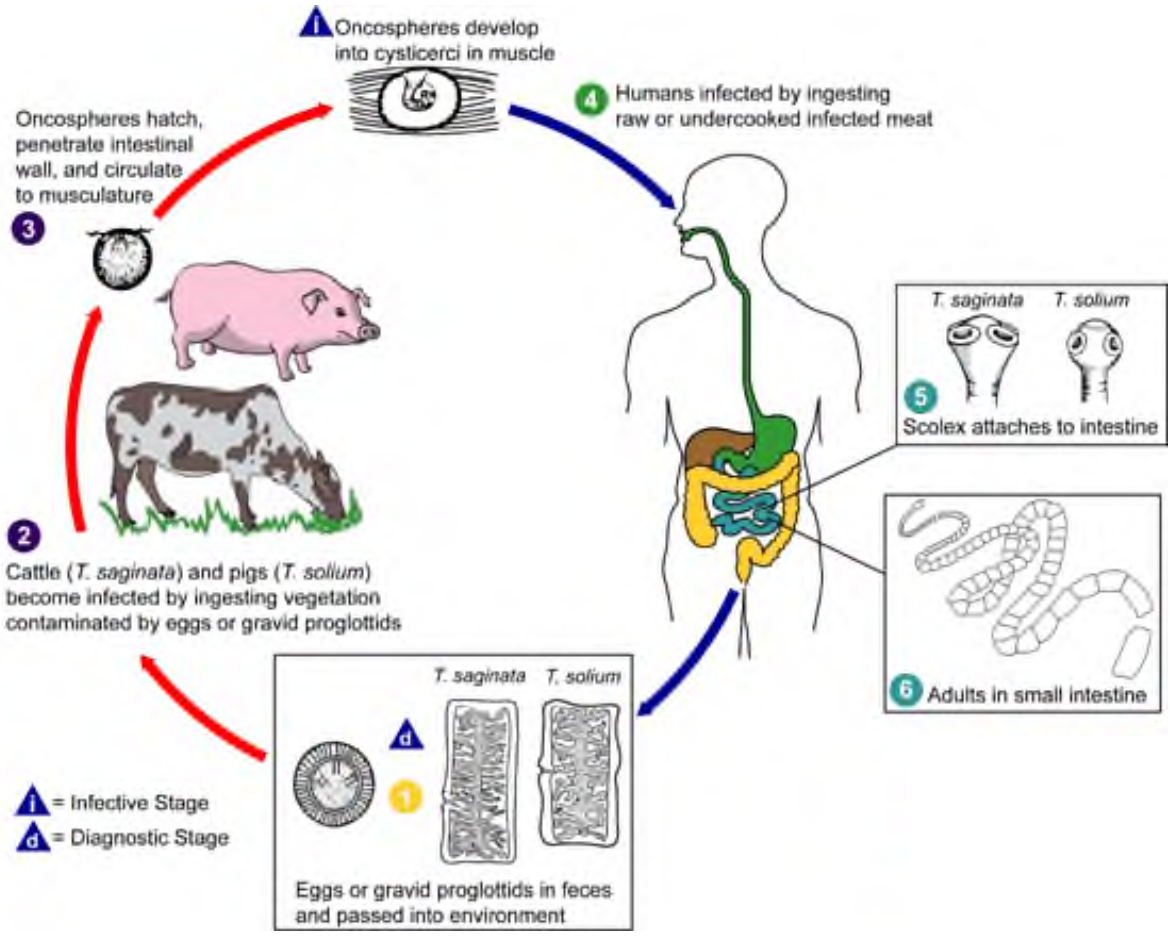


Fig. 17. The life cycle of *Taenia* (https://www.cdc.gov)

Characteristics of *TENIASIS*:

1. Name of the parasite (in Latin) – *Tenia solium, Taeniarhynchus saginata.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Cestoda.

3. Structure, organ systems. The parasites reaches a length of 2–10 m. The skolex is equipped with a proboscis, four suckers. *Tenia solium* has a nimbus of hooks.

4. Disease name – teniosis.

5. Geographical distribution – the disease is common everywhere where there is an obligate intermediate hosts – a pigs and cows, especially in regions where pork and beef dishes are popular.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – undercooked meat contaminated by cyst (finn).

9. Invasion stage for human – cyst (finn).

10. Place (organ) of localization in the human body – the small intestine.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of intestine by irritation of the intestinal mucous membrane by suckers.

12. Clinical symptoms – dyspeptic digestive disorders abdomen pains, unstable stool, weakness, loss of appetite, loss of weight, anemia.

13. Laboratory diagnosis – laboratory diagnosis of teniosis is based on the detection of mature segments and eggs in feces.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – avoid eating undercooked pork and beef meat; *public* –detection and treatment infected people; prevention of contamination of the environment by human feces, veterinary control of pork/beef meat, personal and public health education.

Sometimes a human becomes an intermediate host for the pork tapeworm – *Tenia solium*, becoming infected with the larva, which leads to the development of severe complication of teniosis -cysticercosis.

CYSTICERCOSIS.

7. The way of penetration – alimentary, then autoinfection.

10. Place (organ) of localization in the human body – the brain, the muscles.

11. Pathogenic effects – in most cases, cysticercosis is observed as a complication of teniosis, as a result of autoinvasion (self-infection). Autoinvasion is possible with vomiting, when, together with vomit, mature segments of the parasite are thrown into the stomach. Under the influence of gastric juice, the joints and membrane of the egg are destroyed, and the oncospheres from the intestinal lumen enter the blood vessels and spread throughout the body, where they turn into cysticerci (Fig. 18).

12. Clinical symptoms – localization of cysticerci in the brain can lead to death, in the eyeball – to blindness. Currently, thanks to the effective prevention of the disease, cysticercosis in humans is extremely rare.

13. Laboratory diagnosis – of cysticercosis is difficult and is based on the detection of specific antibodies in the blood by immunological test. Use PCR method.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* –avoid eating undercooked pork meat; *public* –detection and treatment infected people; prevention of contamination of the environment by human feces, veterinary control of pork meat, personal and public health education.

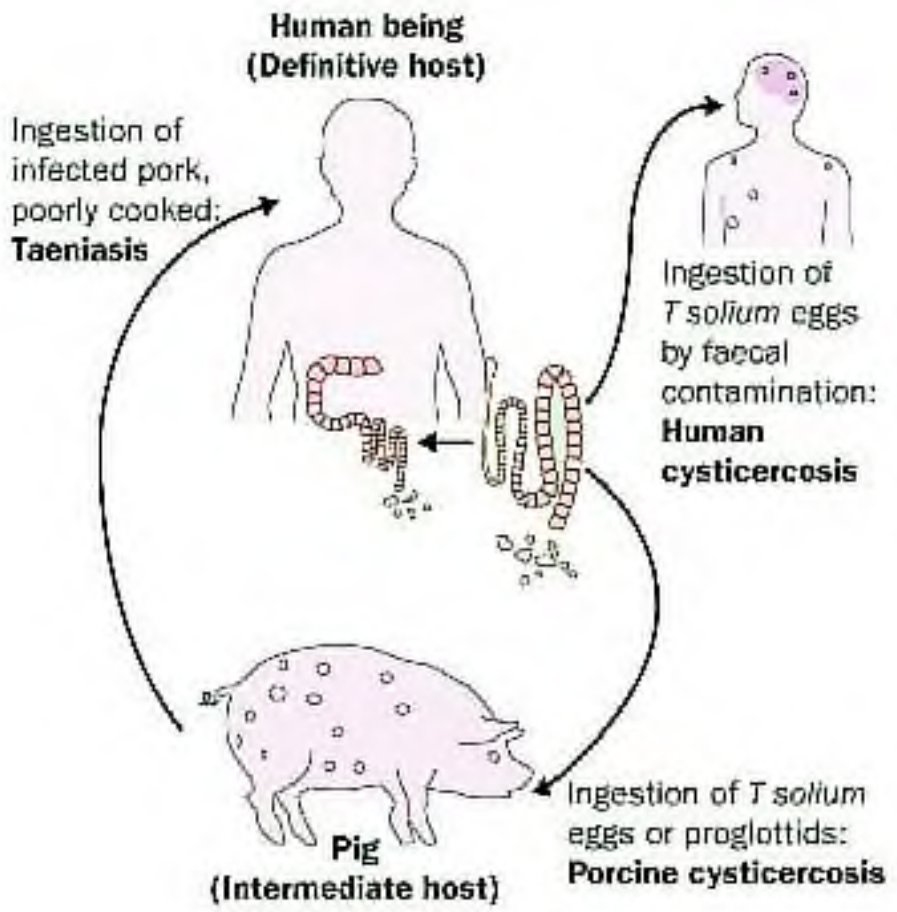


Fig. 18. Cysticercosis is a parasitic tissue infection caused by larval cysts of the tapeworm Taenia solium (https://www.thelancet.com)

Diphyllobotrium latum cause DIPHILLOBOTHRIASIS

Life cycle. The life cycle occurs with the change of three hosts. The definitive (obligatory) hosts are a human, a dog, less often a pig, a cat, some wild animals that eat raw fish. The first intermediate host is crustaceans (cyclops), the second intermediate host is freshwater fish. Mature proglottids of wide tape contain the uterus. The uterus has an sexual pore, through which eggs are still removed from it in the intestine. The eggs enter the environment with the feces of infested humans or animals.

The development of a larva in an egg occurs in water and, depending on temperature and other conditions, lasts on average 10–18 days. The larva leaves the egg. With the help of cilia, the larva move in the water, are swallowed by cyclops, where in 10–12 days they are develop. Cyclops in the composition of

plankton are swallowed by freshwater fish, in the tissues of which cyst is formed.

Characteristics of *DIPHILLOBOTHRIASIS*:

1. Name of the parasite (in Latin) – *Diphyllobotrium latum.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Cestoda.

3. Structure, organ systems (figure). The body length is 10–20 m. There are 2 sucking grooves (bothria) on the scolex. The width of proglottids is significantly more than their length. Mature proglottids contain an open rosette – shaped uterus.

4. Disease name – diphyllobothriasis.

5. Geographical distribution – the disease is common everywhere.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – undercooked or raw fish meat contaminated by larva (cyst).

9. Invasion stage for human – cyst.

10. Place (organ) of localization in the human body – the small intestine.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). The parasite upsets the digestion and absorbs nutrients and vitamins, especially B12.

12. Clinical symptoms – dyspeptic digestive disorders abdomen pains, unstable stool, weakness, loss of appetite, loss of weight, anemia (deficit of vitamin B12).Bright-red spots and fissures appear on the tongue, atrophy of nipples occurs. The skin is pale, slightly yellowish; the liver and spleen are enlarged.

13. Laboratory diagnosis – laboratory diagnosis of diphyllobothriasis is based on the detection of mature segments and eggs in feces. The uterus has a rosette-like and is located in the center.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* –avoid eating undercooked fish meat; *public* – detection and treatment infected people; prevention of contamination of the environment by human feces, veterinary control of fish meat, personal and public health education.

Human as an intermediate host for the larvae.

The human is an intermediate host for the larvae of echinococcus and alveococcus. The sources of infection are dogs, wolves, foxes which excreted mature proglottids filled with eggs with feces. The invasive stage is the egg.

The way of penetration is alimentary. Infection occurs by ingestion the eggs when eating unwashed fruits, vegetables, greens, through dirty hands after contact with dogs. Place of localization of the larvae of echinococcus and alveococcusare liver, lungs, brain, rarely – other organs.

Echinococcus cause ECHINOCOCCOSIS.

Life cycle. The life cycle of echinococcus occurs with the change of two hosts. The definitive hosts are dogs, wolves, jackals and some other predatory mammals. Intermediate hosts are wild and domestic herbivores or omnivores. An optional (optional) intermediate host is a human (Fig. 19).

Larvae are released from the eggs in the intestines of the intermediate host, which penetrate into the blood vessels, then through the portal vein into the liver (first place in the frequency of damage), as well as into the brain, lungs and other organs. There the larva transform into larvae (hydatic cysts). Hydatic cyst is a single-chamber bubble, which can reach sizes from millet grain to the head of a newborn baby and more. The wall of the echinococcal bladder is dense, consists of the cuticular (external) and germinal (internal) membranes. Germinal liningthe bubble, forms many small parietal protrusions with scolexes inside (brood capsules), the number of which can reach several thousand. In each capsule – more than a hundred scolexes. A fibrous capsule forms around the bladder in the host tissue.

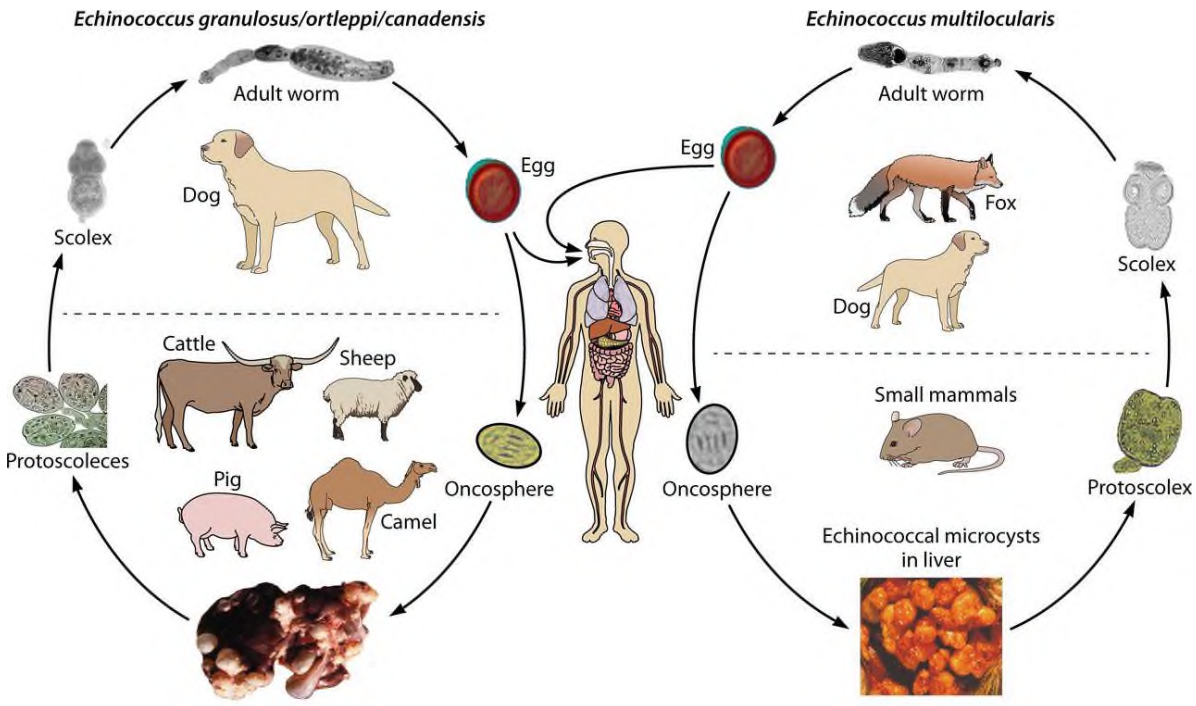


Fig. 19. The life cycle of *Echinococcus* (https://cmr.asm.org)

Characteristics of *ECHINOCOCCOSIS:*

1. Name of the parasite (in Latin) – *Echinococcus granulosus and Echinococcus multilocularis*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Cestoda.

3. Structure, organ systems (figure). The length is 3–5 mm. The scolex has suckers and hooks. The strobila consists of 3–4 proglottids (see Fig. 19). The second to the last proglottid is hermaphroditic, the last one is mature. The uterus is closed.

4. Disease name –*ECHINOCOCCOSIS*.

5. Geographical distribution – the disease is common everywhere. The most endemic are considered zones of the Mediterranean, the Black Sea, the Middle East, Asia. In Russia, echinococcosis is more common in areas, where the pathogen is circulated between dogs, domestic herbivores, and omnivores (mainly pigs and sheeps).

6. Sources of invasion – infected dogs, foxes.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions – transmitting factors are dirty hands contaminated by eggs after contact with dog or sheeps.

9. Invasion stage for human – egg.

10. Place (organ) of localization in the human body – the hydatic cystsare mainly located in the liver, lungs, rarely – the brainof intermediate hosts (human).

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Larvae of parasites cause mechanical damage of organ tissue (brain, liver, lung).

12. Clinical symptoms – in the patient, the affected organ increases, its function is impaired. A growing hydatic cyst squeezes the surrounding tissue, which leads to circulatory disorders. When localized in the liver, jaundice can develop, in the lungs – cough, shortness of breath, chest pain. Especially dangerous is the rupture of hydatic cyst, which leads to the desimination of small embryonic heads according to the body and the development of multiple echinococcosis, which often ends in the death of the patient. Patient may suffer from pains in the chest, cough, breathlessness, sometimes hemoptysis.

13. Laboratory diagnosis – laboratory diagnosis of echinococcosis is based on the X-ray method, ultrasound, tomography, radioisotope scanning. Used immunodiagnosis and PCR.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – to comply with hygiene rules -wash hands after contact with stray dogs; *public* – detection and treatment infected people; prevention of contamination of the environment by dog feces, veterinary control – deworming the dogs, compliance with hygiene rules when processing the skins of commercial animals, the prohibition of feeding rodent carcasses to dogs, personal and public health education.

Hymenolepis nana cause HYMENOLEPIASIS.

Life cycle. *Hymenolepis nana*d evelops, as a rule, without an intermediate host. Larvae are released from the swallowed eggs of the worm, which are embedded in the villi of the small intestine. Larva grow and gradually begin to squeeze the vessels that feed them. After a week, the villi are torn away from the intestinal mucosa, their integrity is violated. Released young larvae find themselves in the lumen of the intestine. They are attached to its walls by suction cups and hooks located on the proboscis, grow and turn into adult form, from which mature segments with eggs are separated. Here, in the intestines, the proglottids are destroyed and the life cycle repeats.

Thus, unlike most other cestodes, the development of *Hymenolepis nana* from an egg to adult stage is completed in the body of one host, which for a parasite is first an intermediate host (development of larva in the villus), and then final (development of a mature stage in the lumen intestines).

Autoinfection contributes to the long-term maintenance of infection (up to several years). Despite intra-intestinal autoinvfection, hymenolepidosis cannot continue indefinitely: after changing several generations of parasites, immunity is formed and self-healing occurs.

Sometimes the development cycle of *Hymenolepis nana* includes an intermediate host (Fig. 20).

In this case, the obligate definitive hosts are rodents, the optional host is a man, the intermediate hosts are many insects (for example, a pest of flour – flour grains, a caterpillar of a bread moth, cockroaches, fleas, etc.), in the tissues of which it develops larva. The definitive host accidentally ingests the intermediate hosts infected by the larva. In humans, this can happen when eating poorly baked bread. The intermediate host invased, swallowing the parasite eggs, if they are in flour and dust, where they get from the excrement scattered by rodents.

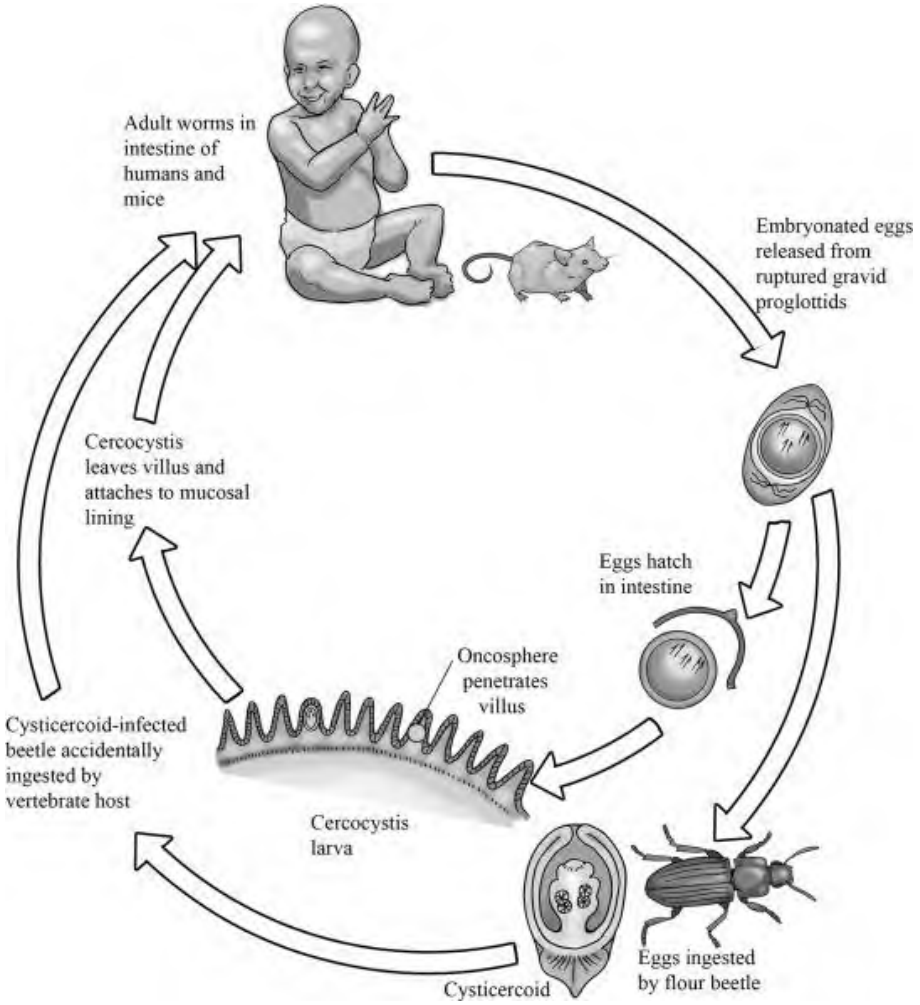


Fig. 20. The life cycle of Hymenolepis nana (https://www.sciencedirect.com)

Characteristics of *HYMENOLEPIASIS*:

1. Name of the parasite (in Latin) – *Hymenolepis nana.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Plathelminthes, Class Cestoda.

3. Structure, organ systems (figure). The length of the dwarf tapeworm is 1–5 cm, its body consists of about 200 proglottids; the scolex has 4 suckers and a rostellum with a double circlet of hooks. The uterus is closed, but a thin wall of proglottids is easily destroyed and eggs get into the intestinal lumen.

4. Disease name – hymenolepidosis.

5. Geographical distribution – the disease is common everywhere. Hymenolepidosis belongs to the group of contagious infestations of childhood, as infection occurs through close contact with infected children through dirty

hands, toys, and household items. Eggs in the environment quickly (after 5–6 hours) die.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions, transmitting factors are dirty hands, food, vegetables, fruit and water contaminated by eggs.

9. Invasion stage for human – egg. Possible autoinfection.

10. Place (organ) of localization in the human body – the small intestine.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of intestine organ tissues (destruction of villi of a small intestine, irritation of the mucous membrane by fixation organs of the parasite).

12. Clinical symptoms – disease asymptomatic or causes following symptoms itching around the anus, abdomen pains, unstable stool, weakness, loss of appetite, loss of weight, anemia.

13. Laboratory diagnosis – laboratory diagnosis of hymenolepidosisis based on the detection of eggs in feces microscopically; immunological methods and PCR are used.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – you should to comply with hygiene rules –wash body and hands, trim your nails, wash vegetables and fruits with hot water. *Public*– detection and treatment infected people; prevention of contamination of the environment with human feces, eradication of flies, cockroaches, personal and public health education.

CONTROL QUESTIONS

1. Characteristics of the Class *Cestoda* representatives.
2. Features of the *Cestoda* larval stages.
3. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of beef tapeworm (*Taeniarhynchus saginatus)*.
4. Laboratory diagnosis, general and personal prevention of beef tapeworm infection.
5. Zoological classification, morphology, geographic distribution, life cycle, pathogenesis and clinical features of pork tapeworm (*Taenia solium*).
6. Laboratory diagnosis, general and personal prevention of cysticercosis.
7. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of transmission and pathogenesis of *Diphyllobotrium latum*.
8. Laboratory diagnosis, general and personal prevention of diphyllobothriosis.
9. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of transmission and pathogenesis of *Echinococcus granulosus*)
10. Laboratory diagnosis, general and personal prevention of echinococcosis.
11. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of transmission and pathogenesis of *Alveococcus multilocularis*.
12. Laboratory diagnosis, general and personal prevention of alveococcosis.
13. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of transmission and pathogenesis of *Hymenolepis nana*.

CLASS NEMATODA

General Description of Roundworm Type

A characteristic feature of representatives of the type Round Worms is the presence of a fusiform or filiform body shape, round on a transverse section. Roundworms (nematodes) live in different environments: soil, fresh, sea water. Many lead a parasitic lifestyle.

According to the epidemiological classification, most nematodes are geohelminthes – causative agents of geohelminthiasis.

Roundworms are characterized by the development of three germ layers. They are characterized by bilateral symmetry. The body shape is elongated, fusiform. The skin-muscle bag consists of integumentary tissue – a cuticle (multilayer inextensible structure) and four ridges of longitudinal smooth muscles, under the cuticle there is a hypodermis with a syncytial structure (plasma mass with nuclei and without cell boundaries). Roundworms have a primary body cavity filled with liquid and acting as a hydroskeleton. *The circulatory and respiratory systems are absent*, gas exchange is carried out through the entire surface of the body.

The nervous system consists of a peri-pharyngeal nerve ring and four longitudinal nerve trunks located in the ventral, dorsal and lateral hypodermal ridges and connected by commissures.

The excretory system is represented by altered protonephridia, characterized by the absence of terminal cells, as well as skin glands.

The digestive system looks like a through tube and is represented by the anterior intestine (mouth, pharynx, esophagus), middle intestine, posterior intestine, and anus.

Roundworms are dioecious. Sexual dimorphism is well expressed: females are usually larger than males, in most males the posterior end of the body is spirally twisted. *The reproductive system* has a tubular structure: in females – paired ovaries, oviducts, uterus and an unpaired vagina, in males – unpaired testis, vas deferens and spicule – a copulative apparatus (Fig. 21).

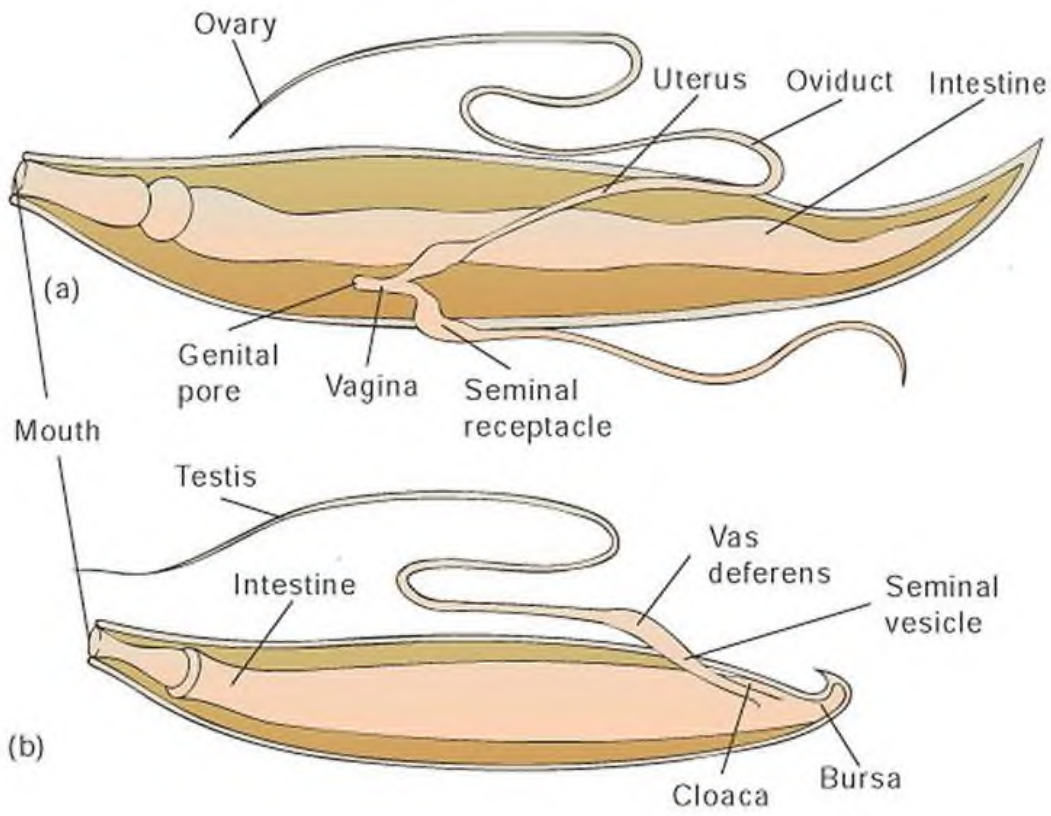


Fig. 21. Nematoda reproductive system: (a) – female; (b) – male (https://slideplayer.com)

Nematodes have aromorphoses, i.e., signs indicating a higher level of their organization compared to plathelminthes. Such aromorphoses include the primary body cavity, the posterior part of the intestine, which ends with an anal opening, and dichotomy.

Parasitic representatives of roundworms live in the organism of vertebrate and invertebrate animals, in plants. Only representatives of the class Actually roundworms (Nematoda) are of medical importance.

Nematodoses – geohelminthes.

Ascaridosis, hookworm infection, strongyloidosis, trichocephalosis are the most widespread in the world and in Russia.

The source of invasion is sick people (or animals).

Invasive stage – invasive eggs or larvae that develop in the surface layers of the soil with the access of oxygen and sufficient moisture.

The invasion method is alimentary. Ingestion of eggs or larvae occurs when contaminated foods (vegetables, fruits, greens) are consumed, through dirty hands and water. In addition, the invasive larvae of hookworms and blackheads can enter the human body percutaneously, with the active introduction into the skin.

Prevention personal – washing hands, fruits, vegetables, herbs, drinking boiled water. Public – sanitary – educational work, the fight against mechanical carriers (flies, cockroaches), disinfection of feces, the protection of soil and water from contamination by feces.

Ascaris lumbricoides cause ASCARIDIASIS.

Life cycle. The invasive stage is a mature egg with a larva. The eggs matured (into egg develop larva) in the soil, under favorable conditions (optimum temperature +25 °C, humidity, oxygen) during 20 to 25 days after excretion the feces of infected human. After swallowing the eggs, larvae are released from them, which pass a migration period of 2 weeks along the following path: veins of a large circle of blood circulation, the right half of the heart, pulmonary circulation, and lungs (Fig. 22).

In the capillaries of the lungs, larvae emerge from the bloodstream into the alveoli, and actively ascend the bronchi, trachea, and larynx. These movements cause a cough reflex in a person, which contributes to the entry of larvae into the throat and re-ingestion. Only after this, entering the small intestine, the larvae reach puberty. The lifespan of roundworm is about 1 year. In the intestine, they are mobile and can sometimes creep into the liver, stomach, esophagus, pharynx, mouth.

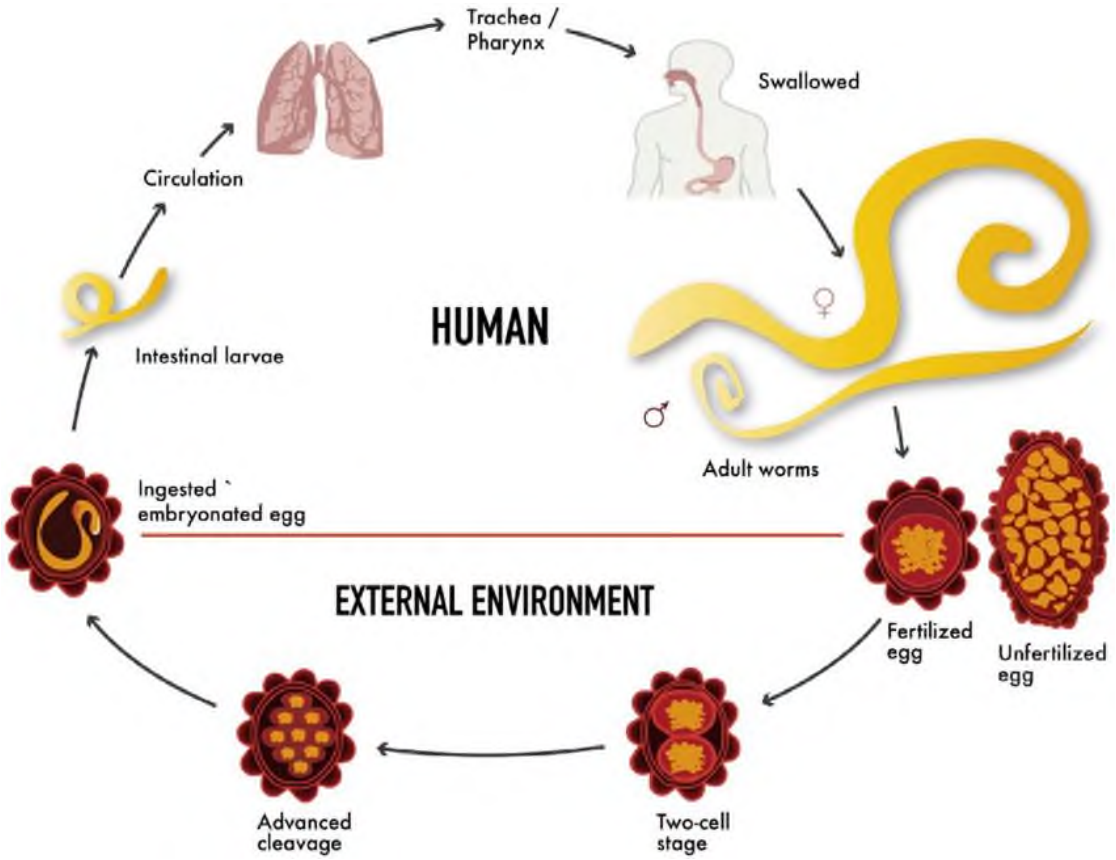


Fig. 22. The life cycle of *Ascaris lumbricoides* (https://www.researchgate.net)

Characteristics of *ASCARIDIASIS*:

1. Name of the parasite (in Latin) – *Ascaris lumbricoides.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). The dimensions of the female are up to 45 cm, the male is up to 25 cm. The mouth opening is surrounded by three lips.

4. Disease name – ascaridosis.

5. Geographical distribution – the disease is common everywhere. The disease is anthroponosis.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions, transmitting factors are dirty hands, food, unwashed vegetables, fruit and water contaminated by eggs. **Mechanic vector** of eggs are flies and cockroaches.

9. Invasion stage for human – egg with larva, which develops in an egg within 25 days.

10. Place (organ) of localization in the human body – the small intestine.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of intestine by irritation of the intestinal mucous membrane and mechanical action of the larvae during the migration period (intoxication, cough as a result of irritation of the upper respiratory tract, allergic skin rashes), as well as the influence of the parasite's vital products on intestinal function.

12. Clinical symptoms – dyspeptic digestive disorders abdomen pains, unstable stool, weakness, loss of appetite, loss of weight, anemia.

13. Laboratory diagnosis – laboratory diagnosis of ascaridosisis based on the detection of eggs in feces (not earlier than 3 months after infection) or larvae in sputum. Used immunodiagnosis and PCR.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* **–** wash hands, wash vegetables and fruits with hot water; protecting food from flies and cockroaches; *public* – detection and treatment infected people; prevention of contamination of the environment by human feces, destraction of flies and cockroaches, personal and public health education.

Hookworms *Ancylostoma duodenale* and *Necator americanus cause ANCYLOSTOMIASIS (HOOKWORM INFECTION).*

Life cycle. Hookworms are obligate human parasites. The invasive stage is a larva. The eggs enter the soil with human feces, a day later larvae are formed (Fig. 23).

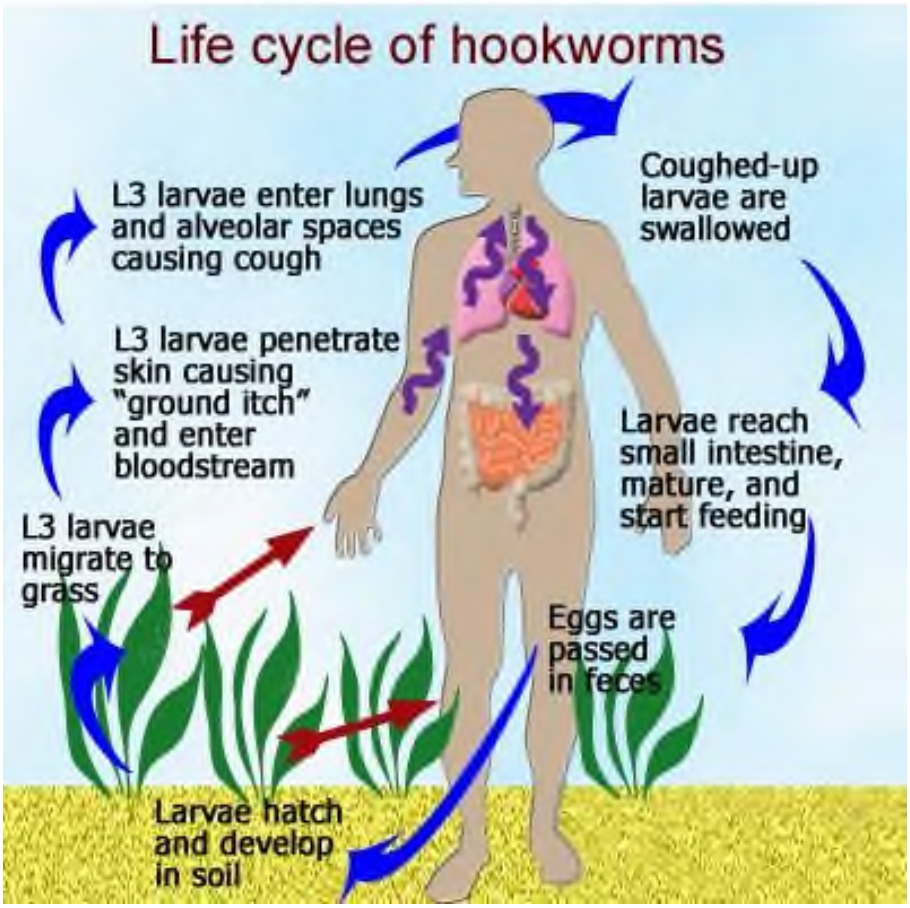


Fig. 23. Life cycle of hookworm (http://www.infectionlandscapes.org)

Larvae that have percutaneous entered the human body migrate (within 5 days) through the blood vessels through the heart to the lungs. When swallowed with sputum, they reach puberty in 1 month. The larvae that enter the human body through the mouth, when swallowed, immediately enter the duodenum, where they reach puberty after 1 month. Mature individuals are attached by the oral capsule to the intestinal villi and secrete anticoagulants into the wounds. The lifespan of hookworm is 5 to 6 years.

Characteristics of HELMINTHIASIS:

1. Name of the parasite (in Latin) – *Ancylostoma duodenale* and *Necator americanus.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems. Hookworms are small nematodes, the size of the female is up to 15 mm, the male is up to 10 mm. The oral capsule of *A.*

duodenale has four teeth, at the N. americanus – two half-moon plates (Fig. 24).

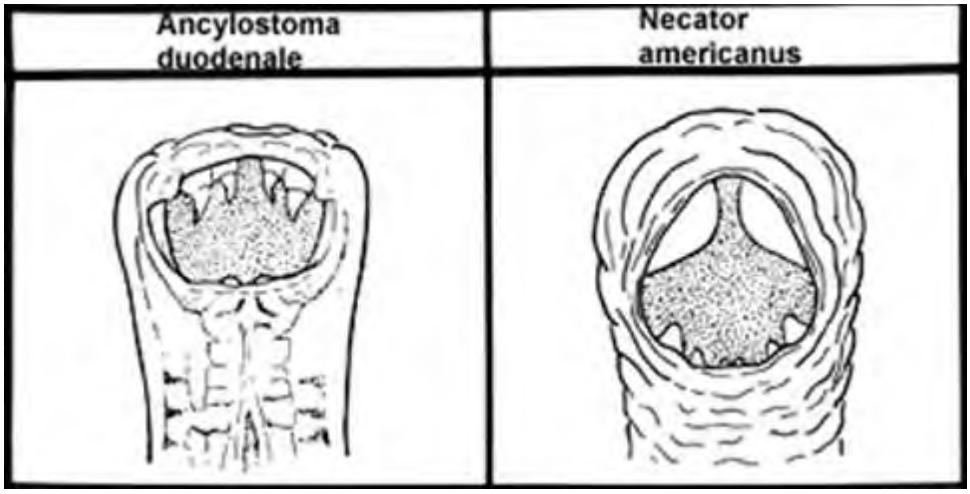


Fig. 24. Hookworm buccal capsules.

4. Disease name – ancylostomiasis (hookworm infection).

5. Geographical distribution – the diseases are widespread in areas with a tropical and subtropical climate (Southeast Asia, Central and South America).

6. Sources of invasion – infected human.

7. The way of penetration – 1-alimentary; 2 – percutaneous.

8. Factors of invasion – soil and water contaminated by larvae.

9. Invasion stage for human – larva.

10. Place (organ) of localization in the human body – the duodenum.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of duodenum by irritation of the intestinal mucous membraneand mechanical action of the larvae during the migration period (intoxication, cough as a result of irritation of the upper respiratory tract, allergic skin rashes).

12. Clinical symptoms – symptomatic hookworm infections are due to either larvae or adults. Larvae produce dermatitis (ground itch) at the site of skin penetration. Larvae migrating through the lungs can produce a mild pneu-

monitis. Disease causes abdominal pain, diarrhea and vomiting during early phase of infection.

13. Laboratory diagnosis – laboratory diagnosis of hookworm infection is based on the detection in feces eggs or larvae. Used immunodiagnosis and PCR.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – adequate footwear and gloves provide physical barriers to the entry of larvae through feet and hands respectively (to wear boots, not to lie on the ground). *Public*– detection and treatment infected people; prevention of contamination of the environment by human feces, personal and public health education.

Strongyloides stercoralis cause STRONGILOIDIASIS.

Life cycle. The life cycle is characterized by significant variability. It can take place completely in the external environment, partially or completely in the host body. The alternation of free-living and parasitic generations is more common.

Sexually mature female lay eggs, from which larvae are already emerging in the intestines. Some of them with feces are excreted into the external environment, some are converted into larvae, which leads to autoinfection. In the external environment, the fate of the larvae can be twofold: either they reach maturity in the soil, or after molting in the soil they become invasive and penetrate the human body, where they reach puberty. Adult females living in the soil lay their eggs and give rise to free-living generations. When conditions change, free-living larvae can become invasive for humans and again go on to parasitic existence. Strongyloidiasis has been characterized by a prolonged course for many years (cases of disease duration over 25 years have been described), which is explained by the possibility of autoinfection.

Characteristics of *STRONGILOIDIASIS*:

1. Name of the parasite (in Latin) – *Strongyloides stercoralis*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). Colorless thread-like nematodes 1–3 mm in size.

4. Disease name – stroingyloidosis.

5. Geographical distribution – the diseases are widespread in areas with a tropical and subtropical climate (in the South-East Asia, East and South Africa and South America).

6. Sources of invasion – infected human.

7. The way of penetration – alimentary, percutaneous.

8. Factors of invasion – soil and water contaminated by larvae.

9. Invasion stage for human – larva.

10. Place (organ) of localization in the human body – the duodenum bile and pancreatic ducts.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of duodenum by irritation of the intestinal mucous membrane and mechanical action of the larvae during the migration period (intoxication, cough as a result of irritation of the upper respiratory tract, allergic skin rashes).

12. Clinical symptoms – skin inflammation, weakness, irritancy, headache, skin itching, symptoms of bronchitis, pneumonia. Then appear signs of enteritis, gastroenteritis. Often parasites penetrate the bile ducts of the liver, causing jaundice. Complications: perforation of the intestine with peritonitis, pancreatitis.

13. Laboratory diagnosis – laboratory diagnosis of stroingyloidiasis is based on the detection of rhabditiform larvae in fresh feces, sometimes in duodenal content, sputum, vomited matter. Used immunodiagnosis and PCR.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – adequate footwear and gloves provide physical barriers to the entry of larvae through feet and hands respectively (to wear boots, not to lie on the ground). *Public* – detection and treatment infected people; prevention of contamination of the environment by human feces, personal and public health education.

Trichocephalus trichiurus cause TRICHOCEPHALIASIS.

Life cycle. The invasive stage is a mature egg with a larva. The eggs matured in the soil, under favorable conditions (optimum temperature +25 °C, humidity, oxygen) during 20 to 25 days after excretion the feces of infected human. After ingestion the mature eggs, the parasites are localized in the intestine and in a few weeks reach puberty. An adult worm sucks blood.

The lifespan of the parasite is about 5 years.

Characteristics of *TRICHOCEPHALIASIS*:

1. Name of the parasite (in Latin) – *Trichocephalus trichiurus.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). The length of the female is up to 55 mm, the male is up to 45 mm. The helminth has a very elongated hairy front end of the body and a thickened rear. Reproduction organs are unpaired.

4. Disease name – trichocephaliasis.

5. Geographical distribution – the disease is common everywhere. The disease is antroponosis.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions, transmitting factors are dirty hands, food, unwashed vegetables, fruit and water contaminated by eggs. Mechanic vector of eggs are flies and cockroaches.

9. Invasion stage for human – egg with larva, which develops in an egg within 25 days.

10. Place (organ) of localization in the human body – upper region of the large intestine (mainly in the caecum).

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of large intestine by irritation of the intestinal mucous membrane.

12. Clinical symptoms – the lack of migration of larvae through the vessels is reflected in the clinical picture of the disease, which is characterized by a more benign course compared with ascaridosis. Disease causes ache along the large intestine, irregular stool, meteorism, poor appetite, nausea, vomiting, weakness, headache. Complications: appendicitis and convulsive attacks.

13. Laboratory diagnosis – laboratory diagnosis of trichocephaliasis is based on the detection of eggs in feces (not earlier than 1 month after infection).Used immunodiagnosis and PCR.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – wash hands, wash vegetables and fruits with hot water. *Public* – detection and treatment infected people; prevention of contamination of the environment by human feces, eradication of flies and cockroaches, personal and public health education.

CONTACT HELMINTHIASIS

Enterobius vermicularis cause ENTEROBIASIS.

Life cycle. The invasive stage is a mature egg with a larva. Fertilized females crawl out through the anus and lay eggs in the perianal folds, causing severe pruritus. The eggs matured during 4 to 5 hours after laying (Fig. 25).

When combing itchy places, the eggs fall on the hands, under the nails, then on the laundry, household items and toys, therefore, with enterobiasis, auto-invasion is often possible. Pinworms have a lifespan of 1 to 2 months. Preschool and junior school children fall ill more often.

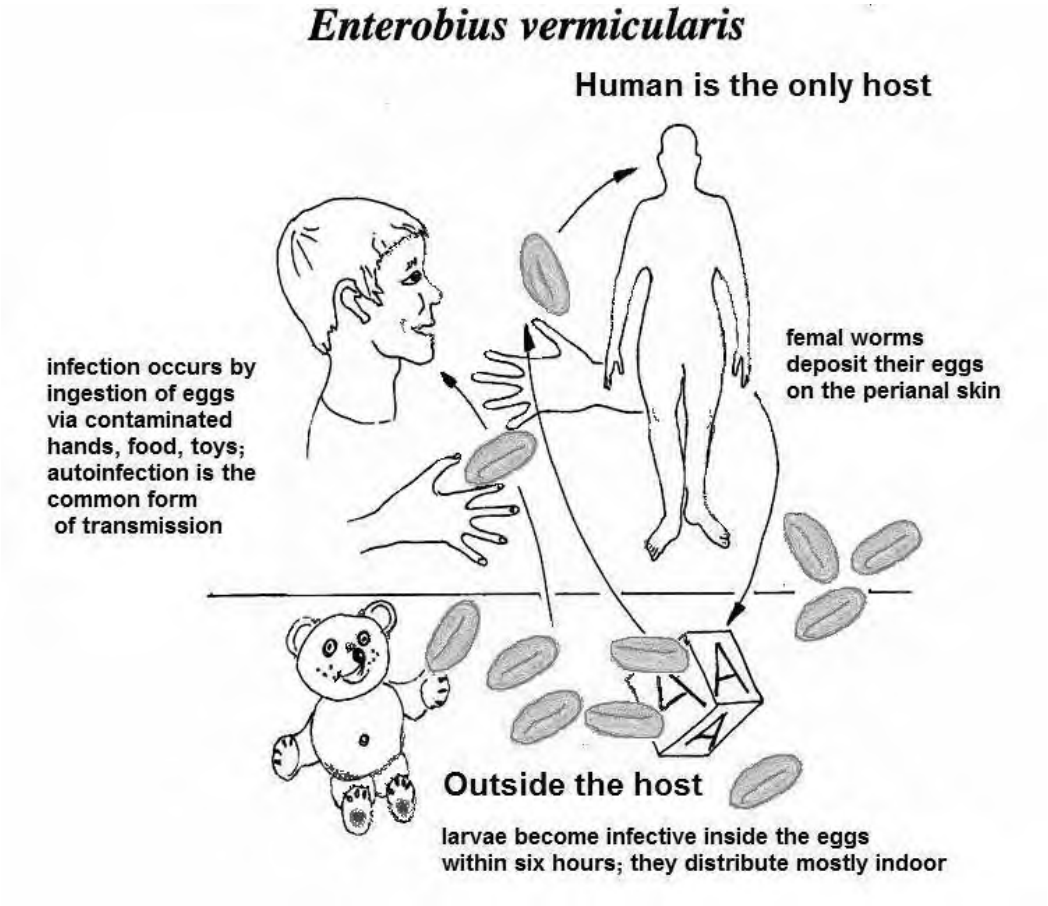


Fig. 25. Life cycle of *Enterobius vermicularis* (https://www.researchgate.net)

Characteristics of *ENTEROBIASIS*:

1. Name of the parasite (in Latin) – *Enterobius vermicularis*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure) The length of a female is about 10 mm, that of a male is 2–5 mm. There are vesicles (cuticular swellings) at the anterior part of the body. Posterior part of the esophagus has a bulb – a ball-like dilation that takes part in fixation of the parasite to intestinal walls.

4. Disease name – enterobiasis.

5. Geographical distribution – the disease is common everywhere. The disease is antroponosis.

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions, transmitting factors are dirty hands, food, toys contaminated by eggs.

9. Invasion stage for human – egg with larva, which develops in an egg within 4–6 hours.

10. Place (organ) of localization in the human body – terminal region of the small intestine.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of large intestine by irritation of the intestinal mucous membrane.

12. Clinical symptoms – disease causes itching and a burning sensation around the anus. Itching troubles patients day and night, becomes unbearable, spreads to the perineum, sex organs and abdomen. The well-being and sleep of patients become worse, there appears irritancy, diarrhea with mucus, nausea, vomiting.

13. Laboratory diagnosis – laboratory diagnosis of enterobiosis is based on the detection of eggs by an adhesive tape test (or scrapings from perianal folds). Eggs are colorless, asymmetric, one side is flattened. Eggs are rarely found in feces.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – wash hands after sleep, cut nail, wash toys with hot water. *Public* – detection and treatment infected people; prevention of contamination of the environment by human feces, sanitary conditions, personal and public health education.

Nematodoses – biohelminthiasis

Nematodes-biohelminthes include Trichinella, Dracunculus, Wuhireria, Dirofilaria, Onchocercus, and Loa-Loa. It is important to note that all representatives of this group have a threadlike body elongated in length, females are longer than males. ***Life cycle of biohelminthes have no eggs: females after fertilization give birth to live larvae.***

Biohelminthiasiss are characterized by the following general features:

The source of invasion is sick people (or animals).

The invasive stage – microfilariae.

Way of invasion – alimentary (when eating the tissues of the intermediate host) and transmissible (with the bites of insect vectors).

The pathogenic effect is due to the migration of larvae and the local influence of sexually mature individuals.

Clinical symptoms. The leading symptoms of biohelminthiasis are toxic-allergic reactions and mechanical tissue damage.

Laboratory diagnosis is often difficult, the diagnosis is made on the basis of objective examination, patient complaints, medical history. Immunological methods and PCR are used to confirm the diagnosis.

The following biohelminthoses are most widespread: trichinellosis, dracunculiosis, vuchereriosis, onchocercosis, loaosis, and dirofilariosis.

Trichinella spiralis cause TRICHINELLIASIS.

Life cycle. *Trichinella spiralis*is a biohelminth. It has life cycle with migration.One and the same organism is a definitive host at first (sexually mature forms are in the intestine) and then an intermediate host (larvae are in muscles).

Swallowed larvae enter the thin intestine, where they reach puberty after 2nd – 3rd day. Sexually mature females, localized in the thickness of the walls of the small intestine, give birth to live larvae. larva penetrates through the intestinal wall, enter the bloodstream and migrate into the muscles. Between the muscle fibers they are spirally twisted and encapsulated exerting a pronounced toxic-allergic effect (Fig. 26).

After several months in the process of developing immunity, a calcined capsule forms around the larva (cyst form), toxic-allergic manifestations subside. In this state, the larvae can remain in the muscles for years, they remain viable even after the death of the host and decomposition of the corpse. In the digestive system of carnivorous animals (beetles, soil worms, crustaceans, fish,

birds of prey, gulls), the larvae into the cyst remain viable for several days. Pigs, as the most common source of human invasion, become infected by eating rats and mice, insects, worms, fish, corpses and droppings of birds, in which viable larvae are preserved.

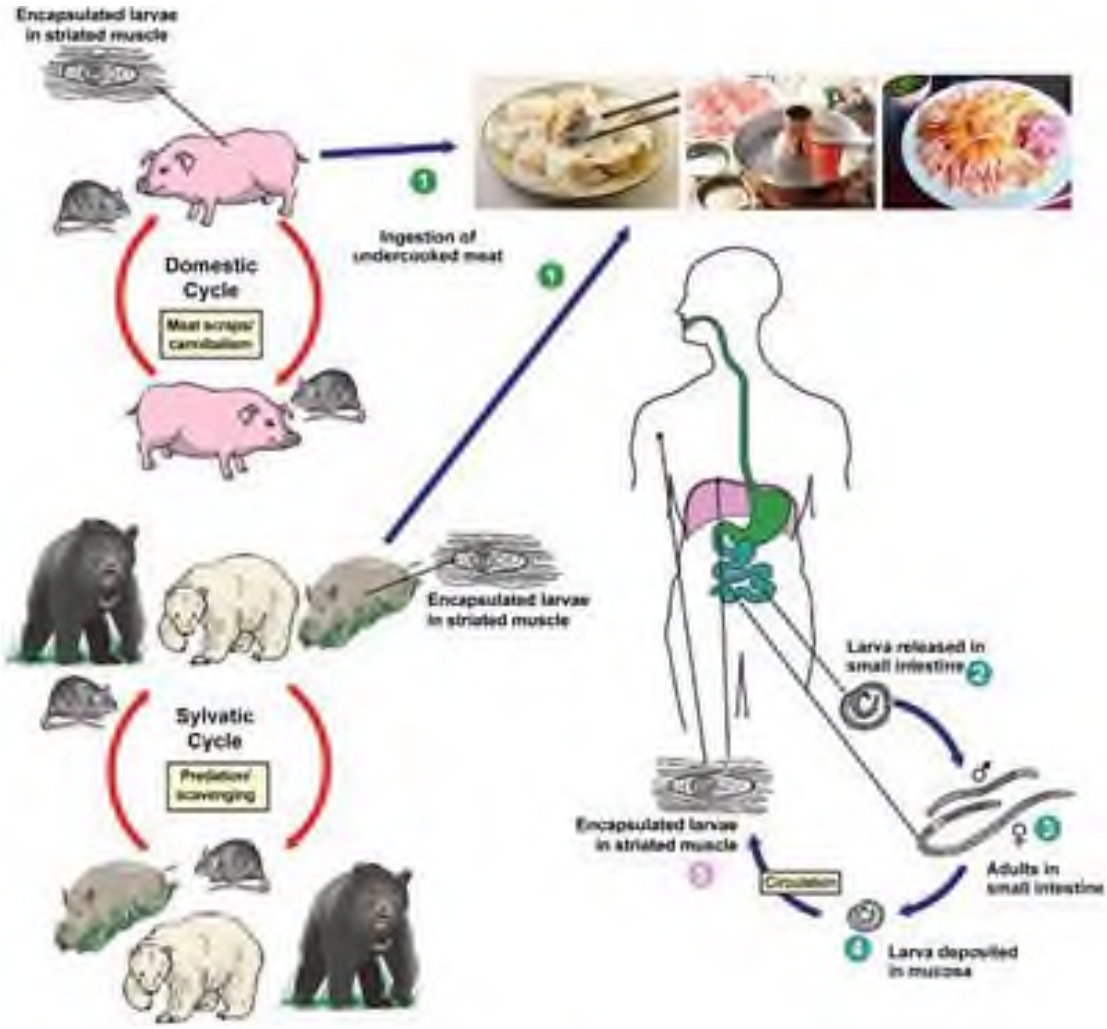


Fig. 26. Life cycle of Trichinella spiralis (https://www.frontiersin.org)

Characteristics of *TRICHINELLIASIS*:

1. Name of the parasite (in Latin) – *Trichinella spiralis.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda (biohelmint)

3. Structure, organ systems (figure). Spindle-shaped body shape. The size of sexually mature individuals varies from 2 mm (males) to 4 mm (females) (see Fig. 26).

4. Disease name – trichinellosis.

5. Geographical distribution – the disease is common everywhere.

6. Sources of invasion – infected carnivorous and omnivorous animals (pigs, wild boars, cats, dogs, mice, rats, bears, etc.).

7. The way of penetration – alimentary.

8. Factors of invasion – meat (pork, meat of wild boars, bears, etc.) contaminated by spiral larvae.

9. Invasion stage for human – cyst with larva.

10. Place (organ) of localization in the human body – small intestine, diaphragm, muscles.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products).

12. Clinical symptoms – the severity of the disease depends on the state of host immunity and on the number of trichinella larvae swallowed by a human. The disease begins acutely, about 1 week after infection, the temperature rises to 38–39 °C, there are swelling of the eyelids and face, pain in the muscles, heartache.

When larvae enters a weakened organism, the first generation of larvae are develop in the intestinal villi, after the destruction of which the larvae reappear in the lumen of the intestine, they reach puberty and actively reproduce. The next generation of larvae is already settling in the muscles. This achieves a sharp increase in the population of the parasite in the host organism, and, accordingly, a sharp aggravation of the disease until the death of the organism.

13. Laboratory diagnosis – laboratory diagnosis of trichinosis is based on the anamnesis – the consumption of meat by an unverified veterinary service of pork or wild animals, as well as on the results of muscle biopsies (2 to 5 weeks after infection). Currently, immunological methods and PCR are mainly used to confirm the diagnosis.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – do not use meat without sanitary ex-

amination (heat processing of meat does not kill larvae); *public* – detection and treatment infected people; elimination of rodents (reservoirs of the pathogen); veterinary control of meat, zoohygienic keeping of pigs (not allowing them to eat rats), personal and public health education.

Dracunculus medinensis cause DRACUNCULIASIS.

Life cycle. The life cycle of *Dracunculus medinensis* lasts 1 year and occurs with the change of hosts: the definitive hosts are human, sometimes dogs, monkeys; the intermediate host is Cyclopes (Fig.27).

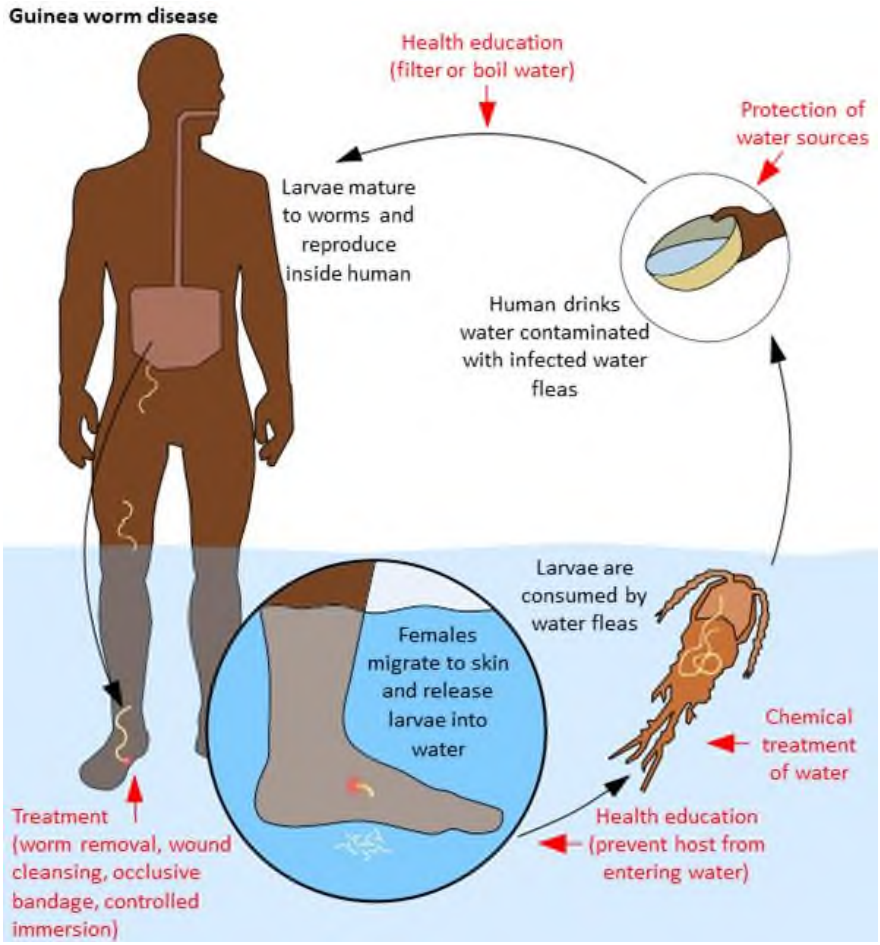


Fig. 27. Life cycle of *Dracunculus medinensis* (https://ourworldindata.org)

After swallowing the cyclops, the larvae enter the intestinal lumen and penetrate into the abdominal cavity, where they reach maturity. Fertilization of the female occurs in the abdominal cavity, after which the female penetrates into the subcutaneous tissue of the limbs. Above the anterior end of the mature female, a skin bladder is formed, filled with serous fluid. At the same time, a

human feels a severe itch that subsides upon contact with water. When the feet are immersed in water, the bubble bursts, female gives birth to microfillaria fall into the water and are swallowed by intermediate hosts – cyclopes.

Characteristics of *DRACUNCULIASIS*:

1. Name of the parasite (in Latin) – *Dracunculus medinensis.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). Female up to 150 cm long, male up to 3 cm.

4. Disease name – dracunculiasis.

5. Geographical distribution – the disease is common in areas with a tropical and subtropical climate (in Central Asia, now there are foci of endemic disease in India and Afghanistan).

6. Sources of invasion – infected human.

7. The way of penetration – alimentary.

8. Factors of invasion – water contaminated by cyclops (water flea) infected with larva (microfilaria).

9. Invasion stage for human – microfillaria within cyclops.

10. Place (organ) of localization in the human body – subcutaneous tissues, especially of the legs, arms and back.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products) and local mechanic effect such as injury of subcutaneous tissues of the legs, arms and back.

12. Clinical symptoms – leg pain, itching and burning in the area of the formed subcutaneous bladder.

13. Laboratory diagnosis – laboratory diagnosis of dracunculus is based on detection of parasites under the skin of the lower extremities (the worm can be detected visually). Immunological methods and PCR are mainly used to confirm the diagnosis.

The old method of treatment is the gradual extraction of the worm with winding of a few centimeters on a stick every day, over 3–4 weeks. The worm can also be removed surgically.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – filtering and boiling drinking water taken from river and lake; *public* – detection and treatment infected people; guarding places of water intake, prohibition to bathe in water intakes elimination of cyclopes; personal and public health education

Transmissible biohelminthiasis

Pathogens of transmissible biohelminthiasis are common in the tropics. All of them belong to the Filarioidea family and cause filariasis. Sexually mature filarias, live in the tissues of the main host, and the larvae they produce, or microfilariae, periodically enter the blood or lymph. During an attack on the host of a blood-sucking insect at this time, microfilariae in the body of this insect in several weeks mature, pass into the proboscis and then, when bitten, enter the blood of the main host. Some types of filarias can have different main hosts (human, dog, monkey, etc.), and carriers are always specific. Due to the fact that parasite larvae develop in carriers, they are also intermediate hosts. In most filarias, the larvae enter the host bloodstream at a strictly defined time of the day, corresponding to the period of maximum activity of the carriers. This feature of biology of filarias should be taken into account when making a diagnosis and blood sampling should be performed for patients to detect microfilaria at a time when their concentration in the peripheral bloodstream is maximal. In addition to a microscopic blood test, immunological methods are used to confirm the diagnosis of filariasis. Prevention personal – protection against insect bites, public – the identification and treatment of patients, vector control.

Vuchereriasis, onchocercosis,and loiasis are transmissible diseases belong to the group of biohelminthiasis.

Wuchereria Bancrofti cause VUCHERERIASIS.

Life cycle. *Wuchereria bancrofti*is a biohelminth. Wuchereriosis is transmissible natural focal disease. Definitive host is human. Intermediate hosts (vector) are a mosquito. Larvae are transmitted by the mosquito bite.

Females give birth to active larvae (microfilariae). Passing through the lymph nodes, these embryos find their way by the main lymphatic trunks into the circulating blood. If these microfilariae are not sucked up by the mosquito, they die in course of time. The lifespan of microfilaria in the human body has been found to be as long as 70 days. Infective larvae penetrate in inguinal, scrotal or abdominal lymphatic nodes and begin to grow into adult forms during 18 months. Again females give birth to larvae. A new generation of microfilariae is emitted which passes either through the thoracic duct or the right lymphatic duct, to the venous system and pulmonary capillaries and then to the peripheral circulation (capillaries of the systemic circulation), thus completing the cycle.

Characteristics of *VUCHERERIASIS*:

1. Name of the parasite (in Latin) – *Wuchereria Bancrofti.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). These are long hair-like transparent nematodes (often creamy-white in color). They are filiform in shape and both ends are tapering, the head end terminating in a slightly rounded swelling. The male measures 2 to 4 cm in length. Its tail-end is curved ventrally and contains two spicules of unequal length. The female measures 8 to 10 cm in length. Its tail-end is narrow and abruptly pointed.

Males and females remain coiled together and can only be separated with difficulty (females are usually more numerous than males and the latter are difficult to find). The lifespan of the adult worms is long, probably several years (5 to 10 years).

4. Disease name – vuchereriasis.

5. Geographical distribution – disease is common in tropical and subtropical counries.

6. Sources of invasion – infected human.

7. The way of penetration – transmissible (bite of mosquito).

8. Factors of invasion – saliva of mosquito.

9. Invasion stage for human – microfillaria.

10. Place (organ) of localization in the human body – lymph nodes, peripheral blood.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products), enlarged of lymph nodes, blockage of the lymphatic ducts, lymph congestion in the limbs, genitals.

12. Clinical symptoms – stagnation of lymph, lymphedema, elephantiasis, and toxic-allergic reactions.

13. Laboratory diagnosis – laboratory diagnosis of vuchereriosis is based on detection of microfilaria in the peripheral blood (at night), lymph and detection of adult worms in the biopsied lymph node. Immunological methods and PCR are mainly used to confirm the diagnosis.

Microfilariae are released into the blood in the evening and at night – a period of maximum activity of mosquitoes.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – prevention of mosquito bites by using the insecticides (repellents); *public* – detection and treatment infected people; elimination of mosquitoes; personal and public health education.

Onchocerca volvulus cause ONCHOCERCOSIS.

Life cycle. Life cycle is completed in two hosts. Humans are the only definitive host. Intermediate hosts are midge (day-biting female black flies of the genus *Simulium*). The vector *Simulium* species breed in «fast-flowing rivers»; and therefore, the disease is most common along the course of rivers. Hence, the name «river blindness». The female black flies are «pool feeders» and suck in blood and tissue fluids. Microfilariae from the skin and lymphatics are in-

gested and develop within the vector, becoming the infective third-stage larvae, which migrate to its mouth parts. The extrinsic incubation period is about 6 days. Infection is transmitted when an infected *Simulium* bites a human. The prepatent period in man is 3–15 months. The adult worm lives in the human host for about 15 years and the microfilariae for about 1 year.

Characteristics of *ONCHOCERCOSIS*:

1. Name of the parasite (in Latin) – *Onchocerca volvulus.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). The adult worms are whitish, opalescent, with transverse striations on the cuticle the posterior end is curved, hence the name Onchocerca, which means "curved tail". The male worm measures about 30 mm in length and the female measures 50 cm. The microfilaria is found typically in the skin and subcutaneous lymphatics. They may also be found in the conjunctiva and rarely in peripheral blood.

4. Disease name – onchocercosis.

5. Geographical distribution – disease is common in the area of the tropical and subtropical climate mainly in tropical Africa, but also in Central and South America. A small focus of infection exists in Yemen and South Arabia.

6. Sources of invasion – infected human.

7. The way of penetration – transmissible – bite ofmidge (day-biting female black flies of the genus *Simulium*).

8. Factors of invasion – saliva of midge (black flies of the genus *Simulium).*

9. Invasion stage for human – microfillaria.

10. Place (organ) of localization in the human body – the adult worms are seen in nodules in subcutaneous connective tissue of infected persons.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products) and local mechanic effect such as injury of subcutaneous tissues.

12. Clinical symptoms – the subcutaneous nodule or onchocercoma is a circumscribed, firm, non tender tumor, formed as a result of fibroblastic reaction around the worms. 1 nodules vary in size from a few mm to about 10 cm. They tend to occur over anatomical sites where the bones are superficial, such as the scalp, scapulae, ribs, elbows, iliac crest, sacrum and knees. The nodules are painless and cause no trouble except for their unsightly appearance Microfilariae cause lesions in tl1e skin and eyes.

13. Laboratory diagnosis – laboratory diagnosis of onchocercosisis based on detection of microfilariae which maybe found in conjunctival biopsies microscopically. Adult worms can be detected in the biopsy material of the biopsy material of the subcutaneous nodule; immunoassay and PCR analysis.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – midge (flies of the genius *Simulium)* bites protection by using the insecticides (repellents); *public*– detection and treatment infected people; elimination of midge (black flies of the genius *Simulium)*; personal and public health education.

Loa-Loa cause LOIASIS.

Life cycle. Definitive host is human. Intermediate host or vectors – horsefly of the genus *Chrysops* in which the microfilariae develop into the infective third-stage larvae. Infection is transmitted to man through the bite of infected *Chrysops* during their blood meal. The infective third-stage larvae enter the subcutaneous tissue, moult, and develop into mature adult worm over 6–12 months and migrate in subcutaneous tissues. Female worms produce sheathed microfilaria which have diurnal periodicity. The microfilaria is ingested by *Chrysops* during its blood meal.

They cast off their sheaths, penetrate the stomach wall and reach thoracic muscles where they develop into infective larvae. Development in *Chrysops* is completed in about 10 days.

Characteristics of *LOIASIS*:

1. Name of the parasite (in Latin) – *Loa-Loa*.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). The adult worm is thin and transparent, measuring about 30–70 mm in length and 0.3–0.5 mm in thickness. Adults live for 4–17 years. The microfilariae are sheathed with column of nuclei extending completely to the tip of the tail. They appear in peripheral circulation only during the day from 12 noon to 2 pm diurnal periodicity).

4. Disease name – loaosis.

5. Geographical distribution – disease is common in the area of the tropical and subtropical climate mainly in tropical Africa.

6. Sources of invasion – infected human.

7. The way of penetration – transmissible – bite of horsefly (day-biting fliesof the genus*Chrysops).*

8. Factors of invasion – saliva of horsefly(day-biting flies of the genus *Chrysops).*

9. Invasion stage for human – microfillaria.

10. Place (organ) of localization in the human body – the subcutaneous tissues, the subconjunctival tissue.

11. Pathogenic effects – the products of metabolism have a toxic-allergic effects (poisoning by waste products) and local mechanic effect such as injury of subcutaneous tissues, the subconjunctival tissue.

12. Clinical symptoms – the wanderings of adult worm through subcutaneous tissues set up temporary foci of inflammation, which appear as swellings, of up to 3 cm in size, usually seen on the extremities. These are the Calabar swellings or fugitive swellings, because they disappear in a few days, only

to reappear elsewhere. Ocular manifestations occur when the worm reaches the subconjunctival tissues during its wanderings. The ocularlesions include granulomata in the bulbar conjunctiva, painless edema of the eyelids and proptosis. Complications like nephropathy, encephalopathy and cardiomyopathy can occur but are rare.

13. Laboratory diagnosis–laboratory diagnosis of loaz is based on detection of microfilaria in peripheral blood microscopically or the isolation of the adult worm from the eye. Microfilariae may be shown in peripheral blood collected during the day. The adult worm can be demonstrated by removal fromthe skin or conjunctiva or from a subcutaneous biopsy specimen from a site of swelling; immunoassay and PCR analysis.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – prevention of horsefly (day-biting flies *Chrysops* bitesby using the insecticides (repellents); *public* – detection and treatment infected people; elimination of horsefly (day-biting flies *Chrysops)*, personal and public health education.

Dirofilaria repens cause DIROFILARIASIS.

Life cycle. The lifecycle of *Dirofilaria repens* consists of adult worm, microfilariae (in a vertebral host: often – dogs and cats, rarely – people, and microfilariae in specific vector – mosquito. In the first stage, mated adult female worms produce thousands of microfilariae into the circulation daily, which are ingested by mosquitoes in a blood meal. Microfilariae develop into invasive stage within the mosquito over the 10–16 days, depending on environmental conditions, before being reintroduced back into a new host. Microfilariae undergo secondary developmental changes in the insect. For the final two stages of development, microfilariae are inoculated back into a vertebral host during an act of biting. The adults of *Dirofilaria repens* reside in the subcutaneous tissues of dogs and cats, raely – hunans where they mature in 6–7 months.

Humans may become infected as random host, the worms fail to reach adulthood while infecting a human body. Dirofilariasis is zoonotic filariasis.

Characteristics of *DIROFILARIASIS*:

1. Name of the parasite (in Latin) – *Dirofilaria repens.*

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). Adult worms are 1–2 mm in diameter (females are 25–30 cm in length, the males being shorter).

4. Disease name – dirofilariosis.

5. Geographical distribution – the disease is common in the Mediterranean region, sub-Saharan Africa, and Eastern Europe, Italy, France, Greece and Spain.

6. Sources of invasion – infected dogs and wild canids.

7. The way of penetration – transmissible – bite of mosquito.

8. Factors of invasion – saliva of mosquito.

9. Invasion stage for human – microfillaria.

10. Place (organ) of localization in the human body – the subcutaneous tissues.

11. Pathogenic effects–the products of metabolism have a toxic-allergic effects (poisoning by waste products) and local mechanic effecton subcutaneous tissues.

12. Clinical symptoms – the patient has pains arising from the migration of dirofilaria.

13. Laboratory diagnosis – laboratory diagnosis of dirofilariasis is based on detection of the excised worm microscopically.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – prevention of mosquito bitesby using the insecticides (repellents); *public* – detection and treatment infected people; elimination of mosquito, personal and public health education.

Some nematodoses are common among animals, but occasionally they can enter the human body.

The group of nematodes for which the person is a facultative host includes causative agents of ascariasis in vertebrate mammals, especially domestic animals – toxocaras. In the human body, these helminthes migrate, but do not reach puberty.

Toxocara canis* cause *TOXOCARIASIS.

Life cycle. Cats, dogs and foxes can become infected with Toxocara through the ingestion of eggs or by transmission of the larvae from a mother to her offspring.

Transmission to cats and dogs can also occur by ingestion of infected accidental hosts, such as earthworms, cockroaches, rodents, rabbits, chickens, or sheep. Eggs hatch as second stage larvae in the intestines of the cat, dog or fox host (for consistency, this article will assume that second stage larvae emerge from Toxocara eggs, although there is debate as to whether larvae are truly in their second or third stage of development). Larvae enter the bloodstream and migrate to the lungs, where they are coughed up and swallowed. The larvae mature into adults within the small intestine of a cat, dog or fox, where mating and egg laying occurs. Eggs are passed in the feces and only become infective after several weeks outside of a host. During this incubation period, molting from first to second (and possibly third) stage larva takes place within the egg. In most adult dogs, cats and foxes, the full lifecycle does not occur, but instead second stage larvae encyst after a period of migration through the body.

Reactivation of the larvae is common only in pregnant or lactating cats, dogs and foxes. The full lifecycle usually only occurs in these females and their offspring.

Second stage larvae will also hatch in the small intestine of an accidental host, such as a human, after ingestion of infective eggs. The larvae will then migrate through the organs and tissues of the accidental host, most commonly the lungs, liver, eyes, and brain. Since L2 larvae cannot mature in accidental

hosts, after this period of migration, Toxocara larvae will encyst as second stage larvae.

Human infection occurs by swallowing toxocar eggs when eating unwashed vegetables, fruits, greens, through dirty hands in contact with dogs and cats.

Characteristics of *TOXOCARIASIS*:

1. Name of the parasite (in Latin)–*Toxocara canis* – ascaris of dogs, *Toxascaris leonina* – ascaris of cats, *Ascaris suum* – ascaris of pigs.

2. Systematic position (Phylum, Class) – Kingdom Animalia, Subkingdom Metotozoa, Phylum Nemathelminthes, Class Nematoda.

3. Structure, organ systems (figure). Adult *T. canis* are found only within dogs and foxes and the males are 4–6 cm in length, with a curved posterior end. The males each have spicules and one "tubular testis". Females can be as long as 15 cm, with the vulva stretching one third of their body length. The females do not curve at the posterior end.

4. Disease name – toxocarosis.

5. Geographical distribution – the disease is common everywhere (the disease is ubiquitous).

6. Sources of invasion – infected dogs, cats, pigs.

7. The way of penetration – alimentary.

8. Factors of invasion – unsanitary conditions, transmitting factors are dirty hands, food, unwashed vegetables, fruit and water contaminated by eggs. Mechanic vector of eggs are flies and cockroaches.

9. Invasion stage for human – eggs.

10. Place (organ) of localization in the human body – the small intestine.

11. Pathogenic effects – the products of worm metabolism have a toxic-allergic effects (poisoning by waste products). Parasites cause mechanical damage of intestine by irritation of the intestinal mucous membrane and mechanical action of the larvae during the migration period (intoxication, cough as a result

of irritation of the upper respiratory tract, allergic skin rashes), as well as the influence of the parasite's vital products on intestinal function. Granulomas can be found throughout the body and can be visualized using ultrasound, MRI, and CT technologies.

12. Clinical symptoms – dyspeptic digestive disorders abdomen pains, unstable stool, weakness, loss of appetite, loss of weight, anemia.

13. Laboratory diagnosis – immunological and PCR methods are used.

14. Preventive measures – prevention is based on knowledge of the development cycles of parasites. *Personal* – washing hands before eating, after contact with pets, eliminating contact with stray cats and dogs; *public* – detection and treatment infected people; deworming of domestic animals, personal and public health education.

CONTROL QUESTIONS

1. Characteristics and Classification of Phylum *Nemathelminthes.*
2. Progressive morpho – physiological and adaptive traits of *Nemathelminthes.*
3. Zoological classification and morphology of Class Nematodes. Epidemiology of nematodosis. Medical importance of species.
4. Zoological classification, geographic distribution, morphology, life cycle of ascaris (*Ascaris lumbricoides).*
5. The mechanism of human infection and localization of ascaris in the host organism. Pathogenesis and clinical features of ascariasis. Laboratory diagnosis, general and personal prevention of ascariasis.
6. Zoological classification, geographic distribution, morphology, life cycle of whipworm *(Trichochephalus trichiurus).*
7. The mechanism of human infection and localization in the host organism, pathogenesis and clinical features of whipworm. Laborotary diagnosis, general and personal prevention of trichocephalosis.

8. Zoological classification, geographic distribution, morphology, life cycle of pinworm *(Enterobius vermicularis).* The mechanism of human infection and localization in the host organism. Pathogenesis and clinical features. Laboratory diagnosis, general and personal prevention of enterobiasis.
9. Zoological classification, geographic distribution, morphology, life cycle of hookworms (*Ancylostoma duodenale* and *Necator americanus)*. The mechanism of human infection and localization in the host organism. Pathogenesis and clinical features. Laboratory diagnosis, general and personal prevention of ancylostomiasis and necotoriasis.
10. Zoological classification, geographic distribution, morphology, life cycle of threadworm *(Strongyloides stercoralis).* The mechanism of human infection and localization in the host organism. Pathogenesis and clinical features. Laboratory diagnosis, general and personal prevention of strongyloidiasis.
11. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of infection and mode of transmission, pathogenesis of trichina worms (*Trichinella spiralis*). Laboratory diagnosis, general and personal prevention oftrichinelliasis.
12. Zoological classification, morphology, geographical distribution, morphology, life cycle, mode of transmission, localization and pathogenesis of guinea worm (*Dracunculus medinensis*). Laboratory diagnosis, general and personal prevention of dracunculiasis.
13. Zoological classification, morphology, geographical distribution, morphology, life cycle of filaria (*Wuchereria bancrofti, Loa-Loa, Onchocerca volvulus, Dirofilaria repens*).

CHAPTER 4
MEDICAL ARACHNOENTOMOGY

Medical arachnoentomology is a branch of parasitology that studies the importance of arthropods as ectoparasites, intermediate hosts of parasites, pathogens and carriers of human pathogens, and poisonous animals. Arthropods include about 2 million species of animals that live on land, in water and in the air. This is the richest type of animal in terms of species diversity.

The General Description of Arthropod Type

Arthropods develop from three embryonic leaves, belong to the primary. The body is divided into segments that have a different structure, perform different functions and are grouped into three departments: head, chest and abdomen. Such a unit is called heteronomic segmentation (Fig. 28).

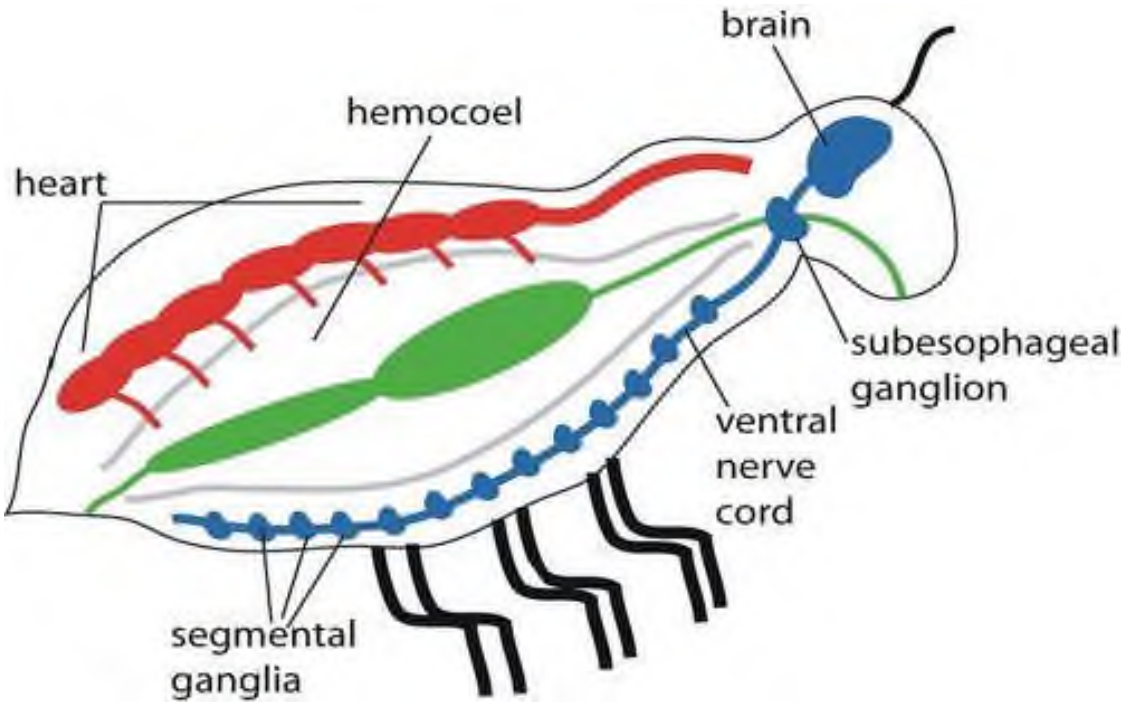


Fig. 28. Structure of Arthropods (https://www.ck12.org)

Morphology. The limbs have a joint structure (hence the name of the type), are located on the head and chest and perform different functions. The limbs localized on the head perform the functions of the senses (antennas and

antennules), are components of the oral apparatus, and participate in defense and attack. The limbs localized on the chest are designed for movement.

Arthropods are characterized by the development of striated muscles and the isolation of individual muscle groups. The outer covers are a chitinized cuticle that functions as the outer skeleton and protects.

Arthropods have a mixed body cavity – the myxocele, which is formed during embryonic development as a result of the fusion of the primary and secondary body cavities; developed all organ systems – nervous, digestive, excretory, respiratory, circulatory, and reproductive.

The nervous system is represented by the pharyngeal ganglion, which performs the function of the brain, and the abdominal nerve chain. The nerve ganglia of the head department are interconnected with the help of near-pharyngeal commissures. On the ventral side of the body there are two nerve trunks having thickenings in each segment, which are connected by commissures. Almost all senses are developed: sight, hearing, smell, touch, balance, taste.

The digestive system develops on the basis of the digestive tube and consists of three sections: anterior (ectodermal origin), middle (endodermal) and posterior (ectodermal origin). The front section begins with a mouth opening, followed by a pharynx, esophagus. The middle section is represented by the stomach and intestines, the presence of digestive glands (hepatopancreas) is characteristic. The posterior part consists of the rectum and ends with the anus.

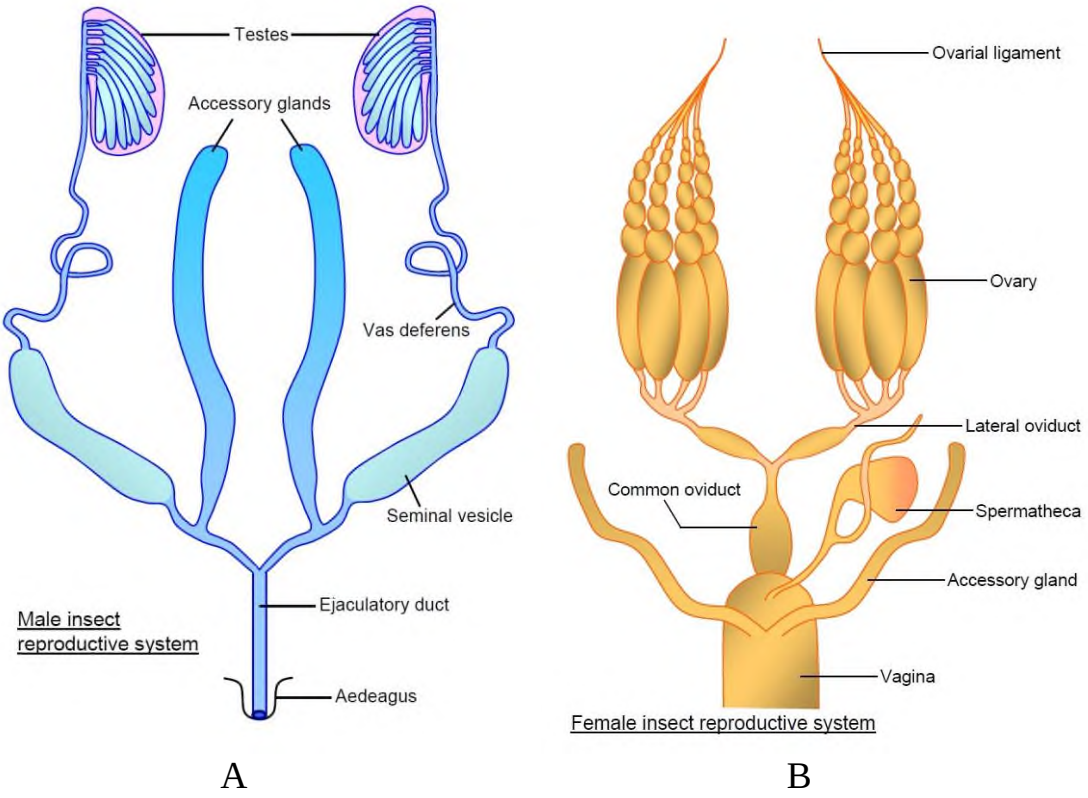
Excretory organs are represented by altered metanephridia, malpigium vessels, and coxal glands. Metanephridia begins in the body cavity with a funnel (nephrostomy). A convoluted tubule follows the funnel, which flows into the excretory duct and opens out by the excretory pore in the lateral part of the body. The funnel and duct have cilia, causing the movement of the excretory fluid from the body cavity into the lumen of the tubule.

The respiratory systems are different depending on the habitat of arthropods. Representatives of the type living in water develop gills that are able to

absorb oxygen dissolved in water. Inhabitants of land have lungs or trachea, which are adapted to the use of atmospheric oxygen.

The circulatory system is open. On the dorsal side of the body is a pulsating organ – the heart. Hemolymph circulates through the vessels.

The reproductive system (Fig. 29).



A B

Fig. 29. Reproductive system of Arthropods: A – male; B – female (https://cronodon.com)

Arthropods are dioecious animals and are characterized by pronounced sexual dimorphism. Females have paired ovaries and oviducts, adnexal glands, as well as unpaired organs – the semen, the copulative bag and the vagina. Males have paired organs – the testes, vas deferens and seminal vesicles, and unpaired – the ejaculatory canal and the copulatory organ.

Development through metamorphosis – complete or incomple (Fig. 30).

In the process of historical development, arthropods acquired the following aromorphoses; heteronomic body segmentation, external chitinous integu-

ment, jointed limbs, striated muscles, complicated structure of the nervous system, blood and respiratory systems.

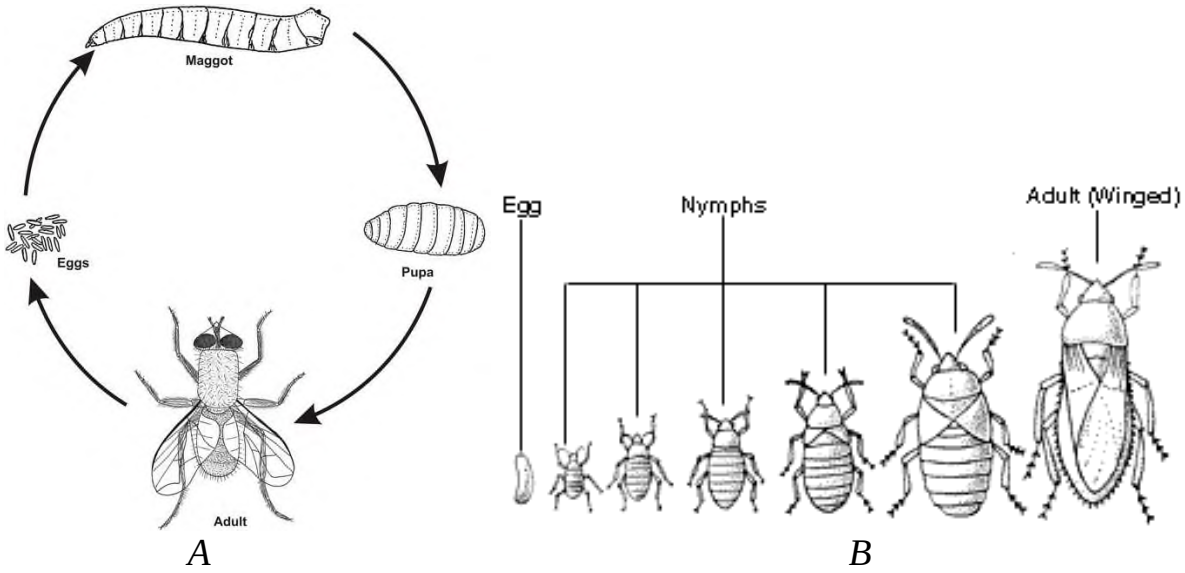


Fig. 30. Development of arthropods: A – complite metamorphosis; B – Incomplite metamorphosis (https://www.intechopen.com)

The medical importance of arthropods is diverse, and in accordance with their role in the occurrence of human diseases, the following groups can be distinguished:

– specific vectors of pathogens;
– mechanical vectors of cysts of protozoa and helminth eggs;
– pathogens;
– intermediate hosts of helminthes;
– poisonous animals.

Among the arthropods, three subphylum are of medical importance: Branchiata, Chelicerata and Tracheata.

Zoological Classification of Arthropods (by V.A. Dogel, 1981)

Kingdom Animalia (Zoa)

Subkingdom Multicellular (Metazoa)

Phylum Arthropoda

Subphylum Branchiata

Class Crustacae

Subphylum Chelicerata

Class Arachnoidae

Order Scorpionidae

Order Aranei

Order Acari

Subphylum Tracheata

Class Insecta

Order Aphaniptera

Order Hemiptera

Order Anoplura

Order Blattoidae

Order Diptera

PHYLUM ARTHROPODA

SUBPHYLUM BRANCHIATA

CLASS CRUSTACEA

Representatives of the class – the inhabitants of the aquatic environment (*cyclops, crayfish, crabs, lobsters*).

Morphology. Respiratory organs – gills. Maxillopods (*lower crayfish*) usually live in the water column and are part of plankton.

Medical importance. Cyclops are intermediate hosts of broad tape and rishta. Higher cancers are the inhabitants of marine and fresh waters. Crayfish, crabs, lobsters are consumed by humans. Freshwater crayfish and crabs are intermediate hosts for pulmonary trematode.

Many crayfish are necrophagous: eating corpses of animals, they clear water from decaying organic remains.

PHYLUM ARTHROPODA

SUBPHYLUM CHELICERATA

CLASS ARACHNIDAE

The arachnid class has about 35 thousand species. These animals are adapted to live on land, so they have air breathing organs. A distinctive feature of arachnids is the fusion of body segments with the formation of the cephalothorax and abdomen. Scorpions have segmentation only on the abdomen; in spiders, the abdomen is not segmented, and ticks have completely lost the division of the body into departments.

Morphology. Arachnids have six pairs of limbs. The first pair of modified limbs is used to capture and grind food (chelicera). The second pair of altered limbs fulfills the functions of the sensory organ (pedipalps). The first two pairs are located on the head, the remaining four pairs, designed for movement (walking legs), are on the chest segment.

The respiratory system is represented by leaf-shaped lungs or trachea. They open outward with special holes at the base of the legs – stigmas. Tracheas are a system of branched tubules that are suitable directly to all organs where tissue gas exchange occurs.

The excretory system is represented by coxal glands (altered metanephridia), which open outward at the base of the legs, and malpigium vessels (blindly closed outgrowths of the intestinal tube) located in the body cavity and opening into the posterior intestine.

All arachnids are dioecious animals characterized by sexual dimorphism.

Medical representatives – Scorpions, Spiders and Ticks.

ORDER SCORPIONIDAE (scorpions).

Geographic distribution – inhabitants of deserts and semi-deserts, are found in Europe, Asia, America, as well as in the southern regions of Russia.

Scorpions live in areas with a warm and hot climate: in the Crimea, the Caucasus, and Central Asia. Body size 5–10 cm, the last segment of the abdomen carries a sting, at the base of which lie poisonous glands.

Medical importance – poison animals. When attacked by a scorpion, bending its tail over the back, a venomous sting strikes prey. For a person, a scorpion injection is not fatal, but causes severe pain, redness, swelling, drowsiness. As a specific medicine, antitoxic anti-scorpion serum is used.

ORDER ARANEI (spiders).

Representatives. Spiders of medical importance include karakurt. Karakurt (black widow) has a velvety-black color, sometimes with bright red spots.

Geographic distribution. Karakurt lives in Central Asia, Crimea, the Caucasus, and Moldova.

Medical importance – poison animals. Karakurt bites are very painful, accompanied by severe abdominal pain, chills, shortness of breath, difficulty speaking, and can be fatal for animals and humans. For therapeutic purposes, antikarakurt serum is used.

ORDER ACARI (ticks).

Representatives have an unsegmented body.

Life cycle. The development of ticks occurs with incomplete metamorphosis: a larva hatches from an egg, followed by a stage of a nymph, which after molting turns into an adult.

Medical importance. Among ticks, permanent and temporary human parasites are found, many representatives of the order are specific carriers of vector-borne human diseases.

The following families of ticks have the greatest medical value:

Ixodidae (Ixodidae):

- genus Ixodes, representatives – canine tick (Ixodes ricinus) and taiga tick (I. persukatus);
- genus Dermacentor, representative – pasture tick (Dermacentor pictus);
- genus Hyaloma, representative – Hyaloma (Hyaloma plumbeum).

Argasaceae (Argasidae): genus Ornitodorus, representative – settlement tick (Ornitodorus papillipes).

Acariformes (Acariformes), or Sarcoptic, representative – scabies itch (Sarcoptes scabiei).

Iron (Demodicidae), representative – Acne gland (Demodex folliculorum).

Gamasidae (Gamasidae), representatives – house tick, rat tick.

Tyroglyphic (Tyroglyphidae), representatives – barn tick, flour mite, house dust mites.

Reddish (Trombiculidae).

Genus Ixodes.

Geographical distribution.

Ticks of the genus Ixodes are common in central Russia, live in forest and forest-steppe zones.

***Morphology*.** They are rather large, have a dark brown dorsal shield.

In males, the shield covers the entire dorsal part of the body, and in females, nymphs and larvae, only the front of the back.

Tick bites are painless because their saliva contains anesthetics. Females are able to suck out a large amount of blood, many times more than their own body weight. The greatest activity is manifested in the spring-summer period.

Life cycle. Ixodid ticks have tree hosts. After bloodsucking, females lay their eggs in minks, sand, and soil. Larvae having three pairs of legs hatch from eggs; there are no respiratory and genital openings. Larvae feed on small vertebrates (rodents, birds, hedgehogs), then go into the soil, molt there and turn into nymphs. Nymphs have four pairs of walking legs, there is no genital opening; they feed on chipmunks, squirrels, hares. After the next molt, the nymphs turn into adult individuals (adults), which feed on large animals (cattle, deer, moose), and can attack a person.

The medical importance of ticks lies in their transfer of more than 20 pathogens of bacterial and viral infections and in maintaining the natural foci of tick-borne spring-summer encephalitis, tularemia, Lyme disease.

Lyme disease was described in 1970. The twentieth century at present, it is one of the most common natural focal vector-borne diseases on all continents

(occurs twice as often as tick-borne encephalitis). The causative agent of the disease – spirochete borrelia – penetrates the human body with saliva or feces of ixodid ticks. The disease is characterized by seasonality, endemicity, and staging. Lyme disease can affect any organs and systems. In the early period, when the disease is easily treatable, migratory erythema appears on the skin (but erythema is not observed in some patients). At the second stage, signs of damage to various organs and tissues appear.

Genus Dermatsentor.

Mites of the genus Dermacenter differ from others by the presence of a large dorsal shield, which is covered with a white enamel pattern. Along the edges of the flap are flat eyes. Adult individuals feed on the blood of farm animals and are carriers of taiga encephalitis, tularemia, tick-borne typhus, typhoid, and brucellosis.

Genus Hialoma.

Ticks of the genus Hialoma, rather large, have a dark brown dorsal shield, along the edge of which bulging eyes are located. They are carriers of Q fever, Crimean hemorrhagic fever.

Argazovye family.

Argasic ticks are common in Central Asia. These are shelter forms that live in caves, rodent burrows, in stones, in steppe and semi-desert areas. Argas ticks do not have a dorsal shield; they are covered with a finely tuberous cover with a characteristic edging along the entire edge of the body. The oral apparatus is located ventrally.

Medical importance. A typical representative is the settlement tick, which is the carrier and reservoir of pathogens of tick-borne tick-borne typhus.

Gamazidae family.

Gamasid mites are small (up to 3 mm), the body is covered with dorsal and abdominal scutes, and there are setae on the body. They settle in rodent burrows and bird nests.

Medical importance. Pathogens of tick-borne spirochetosis, encephalitis, hemorrhagic fever with renal syndrome are transmitted to humans. Mouse, bird and rat ticks can attack humans.

Family Tyroglyphs.

Tyroglyphic (barn) mites are the smallest mites of pale yellow color. No eyes. Tyroglyphic ticks feed on organic matter. Barn mites and flour mites infect food supplies (grain, flour, bread, dry vegetables, fruits, cheeses). When using such products, catarrhal phenomena in the digestive tract and various allergic reactions can develop.

Medical importance of ticks are house dust mites that live in towels, mattresses, carpets, bedding, and upholstered furniture (according to some reports, 100 to 500 specimens are found in 1 g of house dust). They cause severe allergic reactions from subtle rashes on the body to severe forms of bronchial asthma.

Family Red ticks.

Ticks of this family are characterized by larval parasitism. Most larvae have bright red velvety integuments. Often attack during field work, when harvesting, bites are accompanied by severe itching and scratching – the so-called autumn erythema. The natural reservoir is wild rodents.

Sarcoptes scabiei cause SCABIES.

Morphology. They are characterized by very small body sizes (0.1–0.4 mm), pronounced reduction of limbs, low mobility and the passage of a complete **life cycle** on the body of one host.

Medial importance. Close species cause scabies in wild and domestic animals, but they do not possess strict specificity with respect to the owner: the itch of cats, horses, sheep, goats, camels, dogs and other animals can affect a person. Place of localization – in the thickness of the epidermis on the human body (interdigital folds of the hands, inner surface of the shoulder and forearm, elbow bends, region of the mammary glands, lower abdomen, buttocks, genitals

of men). A female tick gnaws passages up to 2–3 mm long in the thickness of the stratum corneum per day (Fig. 31).

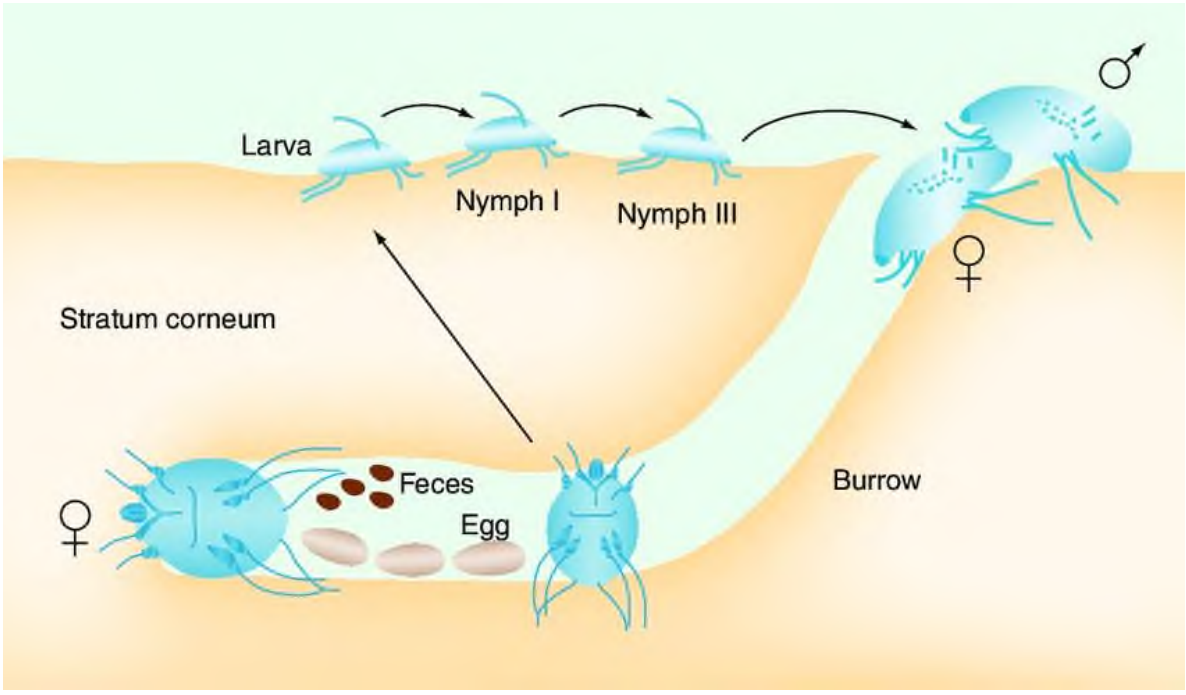


Fig. 31. Life cycle of *Sarcoptes scabiei* (http://44.coff.vitamin-joe.de)

The strokes of the scabies mite look like straight and winding thin strips of whitish color 5–8 mm long. They slightly rise above the skin and resemble an aging scratch. Along the course you can see dark dots – breathing holes. The mite's life span is about 15 days, during which time the female lays up to 50 eggs. Scabies itching feeds on host tissues, irritates nerve endings, and causes severe itching. When combing, the passages are opened and the ticks spread throughout the body. Infection of people occurs through close contact with patients, as well as through bedding and personal belongings of the patient. To confirm the diagnosis of scabies, the vesicle and the lid of the itch passage are opened with a scalpel, the material obtained is transferred to a glass slide and microscopic. Personal prophylaxis – caution when dealing with animals, preventing contact with patients and their household items (bedding, personal items); public – identification and treatment of patients, disinfection of their clothes, linen and towels.

Demodex Folliculorum cause DEMODECOSIS.

Morphology. These are small mites of a worm-shaped form with a length of not more than 0.4 mm.

Medical importance – causes demodecosis in humans. Place of localization – sebaceous glands and hair follicles of the skin of the face, conjunctiva, neck, shoulders. Often found in healthy individuals without causing any symptoms.

Demodecosis develops in weakened people, prone to allergies, iron glands begin to multiply actively, causing blockage of the ducts of the sebaceous glands. In this case, acne of pink color with purulent contents occurs. Infection occurs by contact with a sick person. In 40–60 % of the population, you can find blackhead living as a commensal. To confirm the diagnosis, a microscopic analysis of the contents of the sebaceous glands or hair follicles is performed.

Prevention. *Personal* **–** treatment of major diseases that weaken the body; *public* – identification and treatment of patients.

PHYLUM ARTHROPODA

SUBPHYLUM TRACHEATA

CLASS INSECTA

Insects are the highest invertebrate animals, which are characterized by the greatest diversity in the number of species (about 1 million).

Morphology. Their body is segmented and consists of a head, chest and abdomen.

On the head are the senses: antennae, eyes (simple or facet) and a complex oral apparatus. The oral apparatus consists of paired upper and lower jaws and the lower lip, which are modified limbs, as well as the outgrowth of the chitin membrane – the upper lip. Depending on the type of food, the oral apparatus can be licking (housefly, male mosquitoes), stinging-sucking (female mosquitoes) and gnawing (cockroaches). The thoracic section consists of three

segments, each of which carries a pair of walking legs (three pairs in total). Most free-living insects have two pairs of wings, but some groups that have switched to a parasitic lifestyle have lost them again. There are no limbs on the abdomen, it consists of 6–12 segments. Insects have an external chitin cover under which there is hypodermis. Respiratory organs of insects are represented by trachea. The remaining systems of insect organs correspond to the organization of arthropods.

Life cycle. The development of insects can occur either with incomplete metamorphosis (a larva hatches from an egg that gradually turns into an imago after several molts) or with complete metamorphosis (in the development of an individual there are stages of an egg, larva, pupa, and imago).

Medical importance. Various insects representatives are specific carriers of dangerous vector-borne diseases, often covering large number of the population, as mechanical carriers of cysts protozoa and eggs of helminthes. Insect bites cause pain and, in some cases, allergic reactions. Larvae or adults of some species can parasitize in the skin or internal organs of a person. There are poisonous insects.

Among insects of medical importance, the following groups are distinguished: temporary blood-sucking ectoparasites; constant blood-sucking parasites; tissue and cavity larval (larval) parasites; synanthropic species.

Insects – temporary blood-sucking ectoparasites.

1. Order Aphaniptera (Fleas).

Morphology. Fleas are wingless insects whose body is flattened laterally and covered with chitinous half rings. The back pair of legs is the longest and is suitable for jumping.

Life cycle. Development takes place with complete metamorphosis.

Representatives of the detachment – rat flea, groundhog flea, human flea – are specific blood-sucking carriers of plague pathogens, rat typhus. The human flea lives in floor cracks where the female lays eggs. Worm-shaped larvae develop from eggs, which pupate and then turn into adults. Medical im-

portance. Human fleas usually bite at night. Bites are painful, cause severe itching. Fleas are ubiquitous.

2. Order Hemiptera (bugs).

The representatives of the order have a flattened red-brown body. Bedbug – a wingless insect 4–7 mm in size, an inhabitant of human habitation. It attacks a person at night, bites are painful, cause itching.

Geographical distribution is everywhere.

The kiss (triatomic) bug has a pair of wings, it is a specific blood-sucking vector of pathogens of the American trypanosomiasis – Chagas disease. Geographical distribution is in Latin America.

3. Order Anoplura (lice).

Morphology. Lice are characterized by pronounced adaptations to ectoparasitism: their size is small, the limbs are equipped with a fixation device on the hair (head lice – *Pediculus humanus capitis*), skin (body lice – *Pediculus humanus humanus*), and pubic areas (pubic lice – *Phtirus pubis*). The development cycle is simplified (with incomplete metamorphosis) (Fig. 32).

Medical importance. Head lice is the causative agents of pediculosis, louse borne typhus. Body liceis carrier causative agents of epidemic and relapsing fever. Transmission of infections occurs when rubbing the excrement of lice into the skin during crushing in combing. Pubic louse causes phthyroidism and is not a carrier of pathogens.

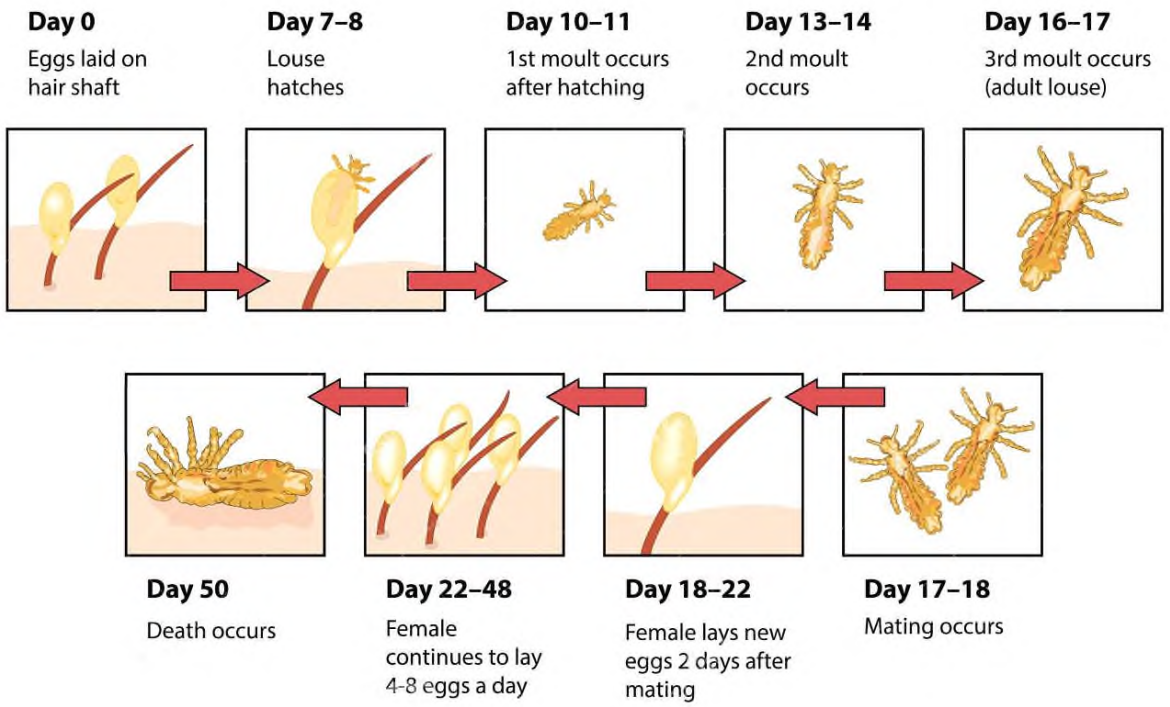


Fig. 32. Lice development (https://www.dreamstime.com)

Non-parasitic synanthropic species

4. Order Blattoptera (Cockroach).

Cockroaches are large insects. In Russia there are red (Prusak) and black cockroaches.

Morphology. They have a flattened body, well-developed vision, a gnawing mouth apparatus, two pairs of wings not intended for flight, they move quickly, are omnivores.

Medical importance. Cockroaches are mechanical vectors of protozoan cysts, helminthes eggs, pathogens of various bacterial diseases, especially gastrointestinal. In some cases, they can bite sleeping people.

The representative of the family Real flies – housefly – is a mechanical carrier of protozoan cysts, eggs of helminthes, pathogens of various bacterial diseases.

Synanthropic species also include house ants and small beetles – flour grains that live in food (flour, cereals). Flour crust is an intermediate host of dwarf tapeworm.

5. Order Diptera.

This is the most diverse insect order in the number of species.

Morphology. Dipterans are characterized by the presence of one pair of wings on the thoracic segment, while the second pair is reduced to the ground beetle. Females of most species of dipterans before laying eggs need a protein of animal origin, which they get during bloodsucking. Diptera males usually feed on nectar, with the exception of male tsetse flies and an autumn lighter that feed on blood. Dipterans develop with complete metamorphosis, most lay eggs, however, gray meat flies, tsetse flies and cavity gadfly hatch live larvae.

Dipterans are divided into two suborders: Long-billed (louse, biting midges, midges, mosquitoes) and Short-billed (horseflies, real flies, gadflies).

The Culicidae family includes three geniuses of great medical importance: Culex, Aedes and Anopheles.

Representatives of the genus Culex and genus Aedes (non-malarial mosquitoes) are carriers of the causative agents of tularemia, Japanese encephalitis, vuchereriosis, brugiosis, yellow fever and dirofilariasis; representatives of the genus Anopheles (malarial mosquitoes) are specific vectors of malarial plasmodium, as well as vuchereriosis and brugiosis. In general, mosquitoes transmit to animals and humans pathogens of more than 50 viral, bacterial and parasitic diseases.

Anophelines and Culicines mosquitoes easily differ from each other at all stages of the life cycle (Fig. 33).

The eggs of malaria mosquitoes are located separately on the water surface, each equipped with two air floats. Their larvae swim in a horizontal position under the surface of the water, and on the last segment have a pair of breathing holes. Pupae resemble commas in shape, are in water and breathe oxygen through the air horns of a funnel shape. The body of adult malaria mosquitoes sitting on objects is located at an angle to the surface with the head down (see Fig. 33). On palps located on both sides of the proboscis, there are

club-shaped thickenings. The antennae are equal in length to the proboscis. The males on the antennae have long bristles, and the females have short bristles.

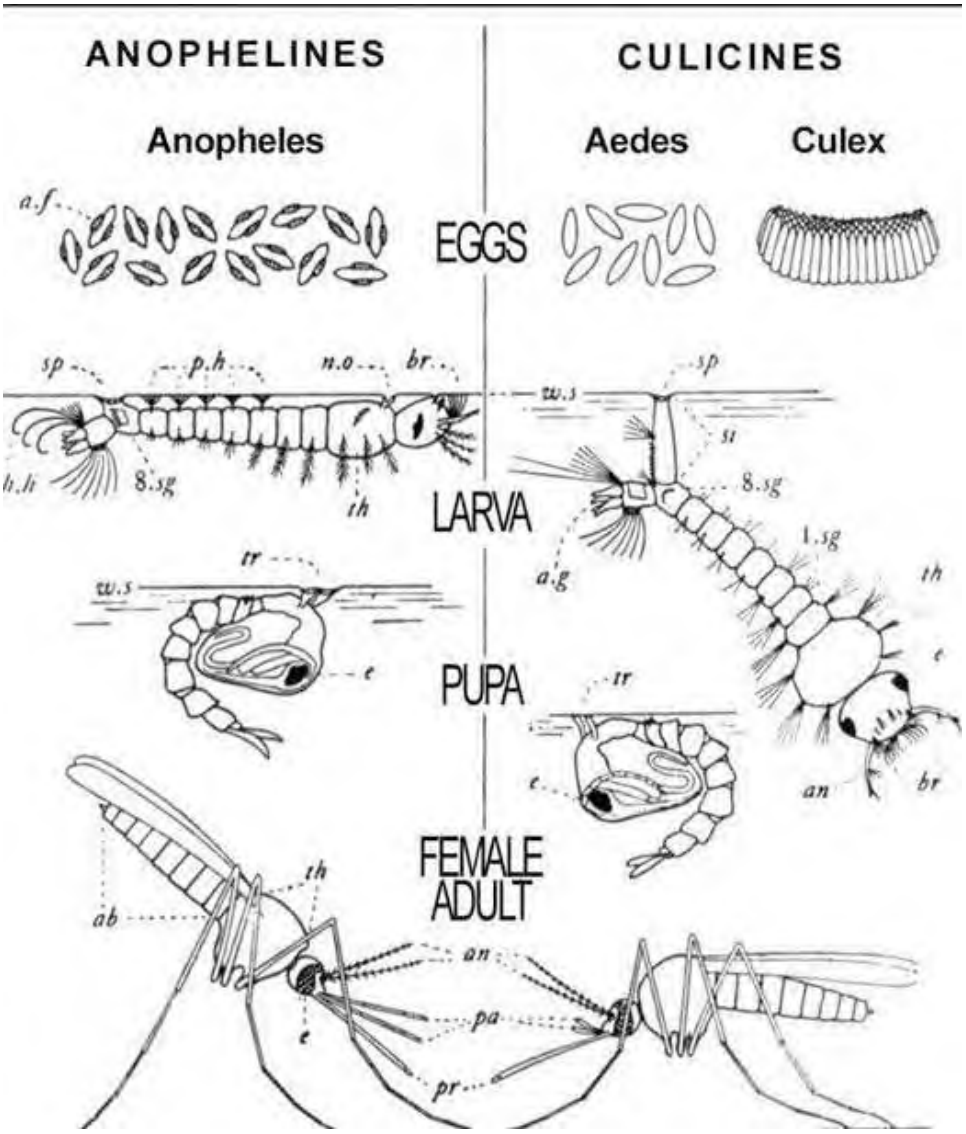


Fig. 33. Development stages of Anophelines and Culicines (https://www.kullabs.com)

Non-malarial mosquitoes lay eggs glued in groups in the form of a boat. Larvae are at an angle near the surface of the water and have a long breathing siphon on the penultimate segment of the body. Pupae have cylindrical breathing horns. When planting, adults hold the body parallel to the surface. The mandibular palps are short, not more than one third of the length of the proboscis. Males have long setae on antennae, and females have short ones.

The Phlebotomidae family includes small insects (less than 3 mm), light brown body, large black eyes, antennae, body, wings densely covered with hairs. The legs are long, thin, flying "jumping". Sandflies are common in regions with a warm climate.

Medical importance. Sandflies are specific vectors of leishmaniasis.

The Simulidae family is the most widespread and intrusive bloodsucker of the wooded valleys of the rivers of Siberia and the Far East.

Morphology. Outwardly similar to small flies of dark color, legs are short, eyes are large, wings are wide. Attack a person during the day.

Medical importance. They are the causative agents of anthrax, tularemia and onchocerciasis.

The Muscidae family are fairly large insects.

These include indoor, gray meat, house flies, tsetse flies, autumn lighters, wolfarth flies, etc. Adults have a moving head with large eyes and short antennae.

Depending on the nature of nutrition, the mouth apparatus of flies can be licking-sucking (nectarophagous), sucking (coprophagous), stinging-sucking (hematophagous) in structure.

Medical importance. Bloodsuckers include the autumn lance – the carrier of the causative agents of tularemia, plague, brucellosis and anthrax; tsetse fly (genus Glossina) is a carrier of the pathogen of African trypanosomiasis. Autumn lighter has a piercing-type mouth apparatus; in August and September it flies into houses, sheds, and actively attacks people. The tsetse fly has relatively large sizes (up to 12 mm), dark brown in color, spots on the abdomen. Tsetse flies live in East and West Africa, near human dwellings or along the banks of water bodies.

Representatives of Dipterta – tissue and cavity larval parasites

The larvae of some flies can parasitize in tissues and body cavities.

Representatives. These include a gray meat fly and a Wolfart fly. The Wolfart fly is the causative agent of myiasis, a human disease caused by distributions larvae's flies in human tissues and organs.

Ovid family (Oestridae) – large flies, adults live for several days and do not feed, hatch live larvae.

Medical importance. Larvae develop in the organs and tissues of affected animals, rarely in humans. The larvae of all gadflies in the human body can pass only the early stage of their development, and then stand out or die. Depending on the place of parasitism, gadflies are divided into gastric, intestinal, subcutaneous and abdominal. The female gastric gadfly of horses glues its eggs to the horse's coat. The larvae of the subcutaneous gadfly penetrate the skin, where within a day they make a stroke up to 3–5 cm long. In humans, ovoid larvae can invade the skin, nasal cavities, eyes, causing tissue miasis. The larva is removed surgically. Females of cavity (sheep and Russian) gadflies on the fly release a liquid containing larvae that can enter the nostrils of goats and sheep. Sometimes they infect a person in the same way, especially asleep, while the larvae can affect the nose or eyes. Intestinal miases arise when a person accidentally swallows small larvae of flies with food (most likely for children). Larvae that enter the intestines, especially in individuals with reduced acidity of gastric juice, can remain alive and even develop there. This leads to irritation and inflammation of the intestinal mucosa, the appearance of abdominal pain, vomiting, and upset stool. After 1–2 days, the larvae excrete with feces.

CONTROL QUESTIONS

1. General features and classification of Phylum *Arthropoda.* Zoological classification, aromorphosis. Medical importance and prevalence of species.
2. The structure and medical importance of Scorpions and Spiders (order *Scorpiones* and *Aranei).*
3. Zoological classification, morphology, life cycle and medical importance of the order *Acari.* Prevention measures.
4. Zoological classification, the structure, life cycle and medical importance of ticks (family *Ixodidae).*
5. Reinforcing *mite related* Prevention, precautionary measures.
6. Zoological classification, morphology, life cycle and medical importance of the family *Argasidae.* Prevention, precautionary measures.
7. Zoological classification, medical importance and geographic distribution species of the family *Trombiculidae* and superfamily *Gamasoidea.*
8. Family *Sarcoptidae*. Zoological classification, morphology, life cycles of human scabies and demodectic mange, preventive measures.
9. Zoological classification, geographic distribution, morphology, life cycle of *Sarcoptes scabiei.* Mode of infection and localization, pathogenicity. Laboratory diagnosis, general and personal prevention of scabies.
10. Zoological classification, morphology, life cycles and medical importance of cockroaches (order *Blattodea)*. Measures for eradication of cockroaches.
11. Zoological classification, morphology, life cycles and medical importance of fleas (order *Aphaniptera)*. Measures for eradication of fleas.
12. Zoological classification, morphology, life cycles and medical importance of Human lice (order *Anoplura)*. Measures for eradication of lice .
13. Zoological classification, morphology, life cycles and medical importance of chinches (order *Hemiptera).*Measures for eradication of chinches.
14. Zoological classification, morphology, life cycles and medical importance of the Order *Diptera.* Measures for eradication of insect.

15. Zoological classification, morphology, life cycles and medical importance of the family *Culicidae*. Differences between malaria and non-malaria mosquitoes at different stages of development. Measures for eradication of mosquito.

16. Zoological classification, morphology, life cycles and medical importance of the family *Muscidae.* Measures for eradication of flies.

17. Zoological classification, geographic distribution, morphology, life cycles and medical importance of the family *Phlebotomidae.*Measures for eradication of sandflies.

18. Zoological classification, general features and medical importance of the family *Simulidae*, *Ceratopogonidae*, genus *Tabanidae.* Measures for eradication of midges, biting midges, horseflies.

19. Insects as pathogens of myiasis. Zoological classification, geographic distribution, morphology, life cycles and medical importance of *Wohlfahrtia magnifica*, *Dermatobia hominis*, *Oestridae.* Prevalence in the Republic of Bashkortostan.

TESTS QUESTIONS

The teaching process is aimed at the formation the following general professional competencies: GPC-1, GPC-7.

Choose one correct answer.

MEDICAL PROTOZOOLOGY

1. THE INVASION STAGE OF *ENTAMOEBA HISTOLYTICA* FOR PEOPLE IS…

1) vegetative form (trophozoit)
2) gametes
3) pseudocysts
4) cysts

2. THE WAY OF PENETRATION OF *TRYPANOSOMA BRUCEI* INTO HUMAN ORGANISM IS

1) transmissible
2) percutaneous
3) alimentary
4) contact-domestic

3. THE TYPE OF REPRODUCTION OF *PLASMODIUM FALCIPARUM* IS…

1) binary fission by mitosis
2) schizogony
3) budding
4) conjugation

4. RESERVOIR HOSTS OF *CUTANEUS (SKIN) LEISHMANIASIS* ARE …

1) dogs
2) fishes
3) birds
4) rodents

5. CLINICAL SYMPTOMS CHARACTERISTIC OF AMOEBIASIS ARE …

1) frequency of stools is 6–8 per day
2) ulcers on the skin
3) serious disorders of the nervous system
4) vaginitis

5) periodic fever

6. FINAL HOSTS FOR *TOXOPLASMA GONDII* ARE …

1) cows
2) humans
3) cats
4) dogs

7. SINGLE FLAGELLA – ORGANS FOR MOVEMENT IN …

1) *Trichomonas vaginalis*
2) *Leishmania donovani*
3) *Toxoplasma gondii*
4) *Balantidium coli*

8. LABORATORY DIAGNOSTICS OF LEUSHMANIASIS

1) detection vegetative forms in feces
2) detection cyst in blood
3) detection cysts in feces
4) detection parasites in lymph nodes

9. MESURES OF PERSONAL PREVENTION OF LAMBLIASIS ARE …

1) protection of food from flies and cockroaches
2) personal protection from Sand Flies' bites
3) personal protection from Tsetse flies’ bites
4) personal protection from bugs’ bites
5) personal protection from mosquitoes’ attacks
6) prevented by the avoidance of casual sex contacts
7) heat treatment of meat

10. MESURES OF PUBLIC PREVENTION OF MALARIA ARE…

1) disinfection of feces
2) elimination of mosquitoes
3) treatment of patients and carriers
4) elimination of bed-bugs

MEDICAL HELMINTOLOGY

Phylum Plathelminthes

1. THE FACTORS OF INVASION OF *OPISTHORCHIS FELINEUS* ARE …
   1) water, contaminated with cyst
   2) vegetables, fruit, greens, contaminated with eggs
   3) insufficiently cooked fish
   4) insufficiently cooked crabs

2. INVASION STAGE OF *PARAGONIMUS WESTERMANI* FOR PEOPLE IS…
   1) adult parasite
   2) eggs
   3) larva
   4) cyst (metacercaria)

3. THE WAY OF PENETRATION OF *FASCIOLA HEPATICA* INTO HUMAN ORGANISM IS…
   1) alimentary
   2) transmissible
   3) contact-domestic
   4) percutaneous

4. INTERMEDIATE HOSTS OF *DICROCELIUM LANCEATUM IS*…
   1) fishes
   2) cyclops
   3) ants
   4) crabs

5. LABORATORY DIAGNOSIS OF SHISTOSOMOSIS IS…
   1) detection segments in feces
   2) detection eggs in the urine
   3) magnetic resonance imaging
   4) detection cysts in feces

6. THE FACTORS OF INVASION OF *HYMENOLEPIS NANA* ARE…
   1) dirty hands
   2) insufficiently cooked fish
   3) insufficiently cooked pork
   4) insufficiently cooked beef

7. INTERMEDIATE HOSTS OF *TAENIA SOLIUM*

1) cattle (cow)
2) pigs
3) fish
4) people

8. TAENIARHYNCHUS SAGINATUS. THE FACTORS OF INVASION

1) dirty hands
2) vegetables, fruit, greens, contaminated with eggs
3) insufficiently cooked fish
4) insufficiently cooked pork
5) insufficiently cooked beef

9. LABORATORY DIAGNOSTICS OF DIPHYLLOBOTHRIASIS…

1) detection eggs in feces
2) detection eggs in in the urine
3) magnetic resonance imaging
4) detection cysts in feces
5) detection segments in feces

10. METHODS OF PERSONAL PREVENTION OF ECHINOCOCCIASIS…

1) protection of food from flies and cockroaches
2) washing hands
3) the use of mosquito nets
4) cooking of fish and crabs

MEDICAL HELMINTOLOGY

Phylum Nemathelminthes

1. *TRICHOCHEPHALUS TRICHIURUS.* THE FACTORS OF INVASION ARE…

1) mosquito bites
2) vegetables, fruit, greens, contaminated with eggs
3) insufficiently cooked fish
4) insufficiently cooked pork
5) insufficiently cooked beef

2. *ENTEROBIUS VERMICULARIS*. THE FACTORS OF INVASION ARE…

1) dirty hands and toys
2) mosquito bites
3) insufficiently cooked fish
4) insufficiently cooked pork
5) insufficiently cooked beef

3. INVASION STAGE OF *ASCARIS LUMBRICOIDES* FOR PEOPLE IS…

1) invasive eggs
2) larva
3) cyst
4) adult parasite

4. INTERMEDIATE HOSTS OF *DRACUNCULUS MEDINENSIS*

1) mosquitoes
2) cyclops
3) predatory animals
4) people

5. THE GEOGRAPHICAL DISTRIBUTION OF *TRICHINELLA SPIRALIS*…

1) everywhere
2) in tropical countries
3) in humid regions
4) near major rivers

6. THE WAY OF PENETRATION OF *ANKYLOSTOMA DUODENALE* INTO HUMAN ORGANISM IS…

1) alimentary
2) transmissible
3) contact-domestic
4) percutaneous

7. INTERMEDIATE HOSTS OF *WUCHERERIA BANCROFTI*…

1) mosquitoes
2) cyclops
3) predatory animals
4) people

8. INTERMEDIATE HOSTS OF *TRICHINELLA SPIRALIS*…

1) mosquitoes
2) cyclops
3) carnivores animals
4) snails

9. LABORATORY DIAGNOSTICS OF ENTEROBIASIS…

1) detection eggs in feces
2) detection eggs in in the urine
3) magnetic resonance imaging
4) detection cysts in feces
5)analysis of scrapings from perianal area

10. METHOD OF PERSONAL PREVENTION OF WUCHERERIASIS – …

1) protection of food from flies and cockroaches
2) personal protection from mosquito's bites
3) washing hands
4) cooking meat and fish

MEDICAL ARACHNOENTOMOLOGY

1. THE MEDICAL IMPORTANCE OF THE CYCLOPS…

1) intermediate host of a wide tape
2) bothersome ectoparasite
3) carrier of leishmaniasis
4) intermediate host of Dracunculus medinensis

2. THE POISONOUS ARACHNIDS ARE...

1) Scorpions
2) Mites
3) Bedbugs
4) Fleas
5) Lice

3. WHEN EATING CANCERS, A PERSON CAN BECOME INFECTED...

1) opisthorchiasis
2) fascioliasis
3) paragonymiasis

4) ascaris
5) dracunculiasis

4. TICKS ARE CHARACTERIZED BY A TYPE DEVELOPMENT…
1) complete metamorphosis
2) incomplete metamorphosis

5. THE SPECIFIC VECTOR OF SPRING-SUMMER TICK-BORNE ENCEPHALITIS IS…
1) meadow tick
2) taiga tick
3) dog tick
4) village tick

6. PERSONAL PROTECTION OF PEDICULOSIS IS...
1) protection from insect bites
2) washing fruit and vegetables
3) observance of personal hygiene rules
4) washing hands

7. EXCRETORY ORGANS OF INSECT ARE...
1) malpighian tubes
2) skin glands
3) the kidneys
4) protonephridia

8. THE MEDICAL IMPORTANCE OF HUMAN FLEA…
1) mechanical vector of the causative agents of gastrointestinal disturbance
2) carrier of a malarial plasmodium.
3) carrier of plague agents
4) causative agent of leishmaniasis

9. BY COMPLETE METAMORPHOSIS DEVELOPS...
1) cockroach
2) mosquito
3) louse
4) bed bug

10. THE INSECT, WHICH PARTICIPATE IN SPREADING OF THE LEISHMANIASIS ARE…

1) blowflies
2) sandflies
3) cockroaches
4) tse-tse flies

SITUATIONAL PROBLEMS

The solution of situational problems is aimed at the formation of the GPC-7.

MEDICAL PROTOZOOLOGY

Task № 1. The patient complained about frequent liquid stool mixed with blood and pain the iliac area. He thinks he is sick after the trip to the Central Asian republics a month ago. He used unwashed fruits and vegetables while was traveling by bus. What kind of disease can we think of? What is the laboratory diagnostics?

Task№ 2. The patient complained about general weakness, lethargy, decreased performance, constant sub-febrile temperature during the last 5 months. Before his illness, he worked for two years as a part of the environmental expedition in Brazil, where he was bitten by a barbeiro bedbug. What kind of disease can we assume? What is the laboratory diagnostics?

Task № 3. After a long business trip to Iran and Iraq, a man complained about the appearance of an ulcer on the right hand and face. During examination it is established that on the right superciliary arch and on the right hand there are shallow ulcers of a round shape with ragged eggs. What kind of disease can we assume? What is the laboratory diagnostics?

Task № 4. After a long trip to one of the countries of equatorial Africa, the patient felt a sharp weakness, drowsiness, weight loss. He has been sick for several years. What laboratory analyses are necessary to diagnose the disease?

Task № 5. A man with signs of cystitis, complained about pain and burning when urinating. It turned out that he had casual sex. A laboratory study of the mucus from the urethra showed flagellates in the undulating membrane with 4 cilia and pear-shaped an unsoulling membrane. What is prevention?

MEDICAL HELMINTOLOGY

Phylum Plathelmintes

Task № 1. The patient complained about weakness, dizziness and general malaise. It is clear from the story that he has been working in the oil field for more than three years and used river fish in the form of stroganina, a sein of a fresh ambassador's pike. Repeatedly noted the discharge tape-shaped formations up to 2–3 mm. with small brown dots on the middle line. What kind of disease can we assume?

Task № 2. A hunter, the resident of Bashkiria, went to the doctor with complaints about chest and liver pain, feeling of heaviness, shortness of breath, weakness, cough with mucous sputum, subfebrile temperature. It turned out that for several years he hunted had been hunting foxes and polar foxes. He didn't always wash his hands after flaying. What kind of helminthiasis can we assume?

Task № 3. During a preventive X-ray examination of the chest in the shepherd's lungs, a tumor-like formation of a rounded shape was found. The edge of the tumor is smooth, inside of it there is an even blackout, while the "shallow breathing" of this tumor is noted. What kind of helminthiasis can we assume?

Task № 4. The patient complained of nausea, vomiting, weakness, lethargy, decreased performance, discomfort, unstable stool. A week ago he noted the elimination of tape-shaped formations. He considers himself sick for more than two months after a trip Western Belarus, where he regularly used pork chops "with blood". What kind of disease can we assume?

Task № 5. A family of three people consumed boiled bear meat, which was bought from a hunter. After 7–8 days, all three felt unwell, fever, pain in the whole body, puffiness of the face. The doctor suspected a dangerous helminthiasis and sent to a hospital. What kind of helminthiasis can be expected?

MEDICAL HELMINTOLOGY

Phylum Nemathelmintes

Task № 1. Eggs of golden brown color lemon-shaped form were found in human feces. What helminthiasis is a person infected with? In which section of the intestine does the helminth parasitize?

Task № 2. A family of three people consumed boiled bear meat, which was bought from a hunter. After 7–8 days, all three felt unwell, fever, pain in the whole body, puffiness of the face. The doctor suspected a dangerous helminthiasis and sent to a hospital. What kind of helminthiasis can be expected?

Task № 3. A month after the deworming of a person from ascaridosis in the study of feces, unfertilized ascaris eggs were found. Is there a complete cure for the disease?

Task № 4. The mother of a three years girl turned to the children's clinic. According to the mother; over the past three weeks, the daughter began to lose weight, sleeps poorly, became irritable, during sleep gives a gnashing of teeth and combes the crotch. What kind of helminthiasis can we talk about and what are the methods of laboratory diagnosis?

Task № 5. A rice field tenant in Central Asia consulted a doctor with complaints of abdominal pain, weakness, pallor of the skin, nausea, vomiting, and loose stools. A month ago, itching and swelling of the lower extremities were noted. Examination revealed anemia. What helminthiasis could he get? What is the laboratory diagnosis of the disease?

MEDICAL ARACHNOENTOMOLOGY

Task № 1. During a tourist trip to Turkmenistan, a tourist was “bitten” by a black spider with red spots on the abdomen. After 30–40 minutes, a painful swelling appeared at the site of the bite, which gradually increased. After

that there was a chill, sweatiness, shortness of breath, palpitations, respiratory rhythm disturbance, weakness, severe pain in the joints. What first aid should be provided to the suffer?

Task № 2. A group of students went to Central Asia to work in student construction teams. During the equipment of the camp, they saw karakurt spiders. In the morning one of the sleeping students woke up from a bite of some arachnid, which has the following morphological features: length 4–5 cm, the segments of the head and chest, merging, form an undivided cephalothorax. The body is separated for a number of segments. The front part of the body is as wide as the cephalothorax, and the back is narrow. The last segment of the body is equipped with a sharp sting. What kind of arachnids are we talking about? What effect does it have on the human body?

Task № 3. A 6-year-old girl brought to the infectious diseases department. Typhus was diagnosed. What type of insects is the causative agent of this disease? Explain the transmission mechanism.

Task № 4. The insect has a sizeof 1.5–5 mm. The color is light yellow. The body is laterally compressed, the back pair of legs is longer than the others. Determine which order the insect belongs to. What is the medical importance?

Task № 5. Eggs, larvae and imago of the arthropod type representative were found in the laboratory during the study under the microscope of scraping from the skin of the patient. What pathogen are we talking about?

CORRECT ANSWERS TO TEST QUESTIONS

MEDICAL PROTOZOOLOGY

1	2	3	4	5	6	7	8	9	10
4	1	2	4	1	3	2	4	1	2

MEDICAL HELMINTOLOGY
Phylum Plathelmintes. Correct answers

1	2	3	4	5	6	7	8	9	10
3	4	1	3	2	3	2	5	1	2

MEDICAL HELMINTOLOGY
Phylum Nemathelmintes

1	2	3	4	5	6	7	8	9	10
2	1	1	2	1	4	1	3	5	2

MEDICAL ARACHNOENTOMOLOGY

1	2	3	4	5	6	7	8	9	10
4	1	3	2	2	3	1	3	2	2

CORRECT ANSWERS
TO SITUATIONAL PROBLEMS

MEDICAL PROTOZOOLOGY

Task № 1. Amoebiasis, detectium of eggs in stool.

Task № 2. American trypanosomosis, detectium of sample smear of blood.

Task № 3. Leishmaniasis, detectium of sample smear of blood.

Task № 4. African trypanosomosis. Detectium of sample smear of blood and cerebrospinal fluid.

Task № 5. Urogenital trichomoniasis, excludecasual sexual relations.

MEDICAL HELMINTOLOGY

Phylum Plathelmintes

Task № 1. Diphyllobothriosis.

Task № 2. Alveococcosis.

Task № 3. Echinococcosis.

Task № 4. Teniosis.

Task № 5. Trichinellessis.

MEDICAL HELMINTOLOGY

Phylum Nemathelmintes

Task № 1. Trichocephalosis. Lower small intestine, cecum.

Task № 2. Trichinellesis

Task № 3. No, a complete cure did not come. In the body, a female ascaris parasitizes.

Task № 4. Enterobiosis. Laboratory diagnostics – analysis of scrapings from the perianal region.

Task № 5. Ankilostomidosis. Laboratory diagnostics – detection of helminth eggs in feces.

MEDICAL ARACHNOENTOMOLOGY

Task № 1. Injected anti-corticosteroid serum.

Task № 2. Scorpions. Painful bite.

Task № 3. Pediculus humanus corporis or Pediculus humanus capitis. Transmissible by contamination.

Task № 4. OrderAphaniptera. Carrier of the plague pathogen.

Task № 5. Wolfarth fly. Miasis.

RECOMMENDED LITERATURE

1. Chatterjee, K. D. Parasitology (Protozoology and Helminthology): in relation to clinical medicine / K. D. Chatterjee. – 12th ed. – Calcutta : [S. n.], 2001. – 238 p.
2. General and medical genetics [Текст]: (methodical recommendations for independent work of foreign students/ ed. G.V. Khomullo. – Tver : Publishing house "Triada", 2007. – 56 p.
3. Medical parasitology: (methodical recommendations for independent work of foreign students / ed. G.V. Khomullo. – Tver : Publishing house "Triada", 2007. – 91 p.

The electronic educational-information resources of the organization on the Internet

1. Electronic educational library http://library.bashgmu.ru/jirbis.
2. The electronic library system "Student Consultant" for the HPE www.studmedlib.ru.
3. The electronic library system "Doe" http: //elanbook.com.
4. Electronic library system eLIBRARY. Collection of Russian scientific journals in medicine and healthcare http://elibrary.ru.
5. A collection of electronic magazines on the OvidSP platform “LWW Proprietary Collection Emerging Market – w/o Perpetual Access” http://ovidsp.ovid.com.
6. Database of scientific medical 3D illustrations Visible Body Premium Package on the platform OvidSP – 3D anatomy online http://ovidsp.ovid.com.
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