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КАФЕДРА ЭПИДЕМИОЛОГИИ

**И. В. Федорова, Ю. В. Митряйкина**

**ЭПИДЕМИОЛОГИЧЕСКАЯ ХАРАКТЕРИСТИКА  
И ПРОФИЛАКТИКА КИШЕЧНЫХ ИНФЕКЦИЙ**

**EPIDEMIOLOGICAL CHARACTERISTICS  
AND PREVENTION OF INTESTINAL INFECTIONS**

Учебно-методическое пособие



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Ф33

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Описываются особенности этиологии, факторы, механизм развития и проявления эпидемического процесса кишечных инфекций. Рассматриваются основные направления профилактики и противозидемические мероприятия, проводимые при выявлении лиц, инфицированных возбудителями кишечных инфекций вирусной и бактериальной этиологии.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке.

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## MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

**Total time of classes:** 4 hours.

Intestinal infections are infections that affect the digestive system, primarily caused by various bacteria, viruses, and parasites. While many intestinal infections are relatively mild and self-limiting, they can have significant consequences for human health, especially among vulnerable populations such as the very young, the elderly, and individuals with weakened immune systems.

From a public health perspective, intestinal infections pose several challenges. They can easily spread from person to person through contaminated food, water, or contact with infected individuals, leading to outbreaks in communities or even across regions. These outbreaks can strain healthcare systems and result in economic losses due to illness-related absenteeism and medical expenses. Public health measures, such as hygiene education, food safety regulations, and outbreak monitoring, are crucial in preventing the transmission of intestinal infections and safeguarding the health of populations.

The assimilation by students of information on the epidemiological characteristics of pathogens of dysentery, salmonellosis, viral hepatitis A and rotavirus infection, the study of the mechanism of development and manifestations of the epidemic process of these infections will allow them to purposefully search for the source of infection, transmission factors, determine the boundaries of the epidemic focus and the time of its existence, navigate the activity of the epidemic process, plan anti-epidemic measures in a specific epidemic situation taking into account the activity of the transmission mechanism and the susceptibility of the contingents, those who were in communication with the identified source of infection.

**The aim of the lesson:** to learn the scientific and organizational foundations of epidemiological surveillance of intestinal infections in accordance with their epidemiological characteristics, the potential effectiveness of preventive measures and antiepidemic measures in the focus of infection, the results of epidemiological diagnostics.

**The tasks of the lesson:**

**1. To study:**

- epidemiological characteristics of pathogens of dysentery, salmonellosis, viral hepatitis A and rotavirus infection;
- factors, mechanism of development and manifestations of the epidemic process of dysentery, salmonellosis, viral hepatitis A and rotavirus infection;
- directions for the prevention of intestinal infections;
- the content and potential effectiveness of antiepidemic measures in foci of intestinal infections.

**2. Familiarize** with the requirements of regulatory documents regulating the procedure for anti-epidemic measures in the foci of intestinal infections in the Republic of Belarus.

3. **To learn** how to apply theoretical knowledge to draw up a plan of anti-epidemic measures in foci, the selection of effective preventive measures against intestinal infections.

**Requirements for the initial level of knowledge.** To successfully master the topic, the student needs to know from the course of microbiology, virology and immunology: morphology and characteristics of pathogens of viral hepatitis A, dysentery, salmonellosis, and rotavirus infection, their resistance in the external environment, their effect on the human body, postinfectious immunity, laboratory research methods.

**Control questions from related disciplines:**

1. Describe morphology and characteristics of pathogens of viral hepatitis A, dysentery, salmonellosis, and rotavirus infection.
2. Describe the resistance of these pathogens to environmental factors.
3. List the methods of laboratory diagnosis of bacterial and viral intestinal infections.
4. Define the notion of «immunity». Describe the notion of «postinfectious immunity».

**Control questions on the topic of the lesson:**

1. Give a general epidemiological description of intestinal infections, name their role in human infectious pathology.
2. Describe the fecal-oral transmission mechanism and transmission routes of intestinal infections.
3. Formulate the definition of the disease for each nosological form (dysentery, salmonellosis, viral hepatitis A and rotavirus infection).
4. Describe the etiology and epidemiological features of the causative agents of dysentery, salmonellosis, viral hepatitis A, rotavirus infection.
5. Name the sources of infection in dysentery, salmonellosis, viral hepatitis A, rotavirus infection, describe their epidemiological features.
6. Describe the leading factors and ways of transmission of pathogens of dysentery salmonellosis, viral hepatitis A, rotavirus infection
7. Describe the susceptibility of children and adults to these infections, as well as post-infectious immunity.
8. Describe the main clinical manifestations for each nosological form (dysentery, salmonellosis, viral hepatitis A and rotavirus infection).
9. Specify the methods used for laboratory diagnosis of these infections.
10. Name the manifestations of the epidemic process for dysentery, salmonellosis, viral hepatitis A and rotavirus infection.
11. List the risk groups for these intestinal infections.
12. Describe the nonspecific prevention of intestinal infections.
13. Describe the specific prevention of viral hepatitis A.
14. Describe the specific prevention of rotavirus infection.

15. Name and characterize anti-epidemic measures in the focus of intestinal infection aimed at the source of infection. Specify their features for dysentery, salmonellosis, viral hepatitis A and rotavirus infection.

16. Name and characterize antiepidemic measures in the focus of intestinal infection aimed at the mechanism of transmission of the pathogen.

17. Name and describe antiepidemic measures in the focus of intestinal infection aimed at people who have communicated with the source of infection. Specify their features for dysentery, salmonellosis, viral hepatitis A and rotavirus infection.

18. Describe the disinfection and sanitary measures in the home (apartment) center of intestinal infection.

19. Name the regime-restrictive measures in the center of intestinal infection in a children's educational institution.

## **EPIDEMIOLOGICAL CHARACTERISTICS OF INTESTINAL INFECTIONS**

Intestinal infections are a group of infectious diseases characterized by the localization of the pathogen in the human intestine, the fecal-oral mechanism of transmission of the pathogen and the leading acute diarrheal syndrome. In accordance with World Health Organization (WHO) recommendations, the term «acute intestinal infections» combines more than 40 diseases of bacterial, viral or protozoal etiology, the main symptom of which is acute diarrhea. However, intestinal infections are not always accompanied by diarrhea. The epidemiological approach to the classification of infectious diseases is based on the location of the primary localization of the pathogen and the fecal-oral transmission mechanism.

The fecal-oral transmission mechanism is characteristic of pathogens with intestinal localization. The isolation of the pathogen from an infected organism (stage 1) is associated with the act of defecation. During stage 2, the pathogen is located in the external environment on various factors, the combination of which forms the pathways of transmission of the pathogen. There are usually three types of transmission routes: food, water, and household contact. The pathogen enters the body (stage 3) through the mouth.

The food route. Infection of food products with pathogens can occur under the following conditions:

1. The primary *in vivo* penetration of the pathogen into the organs and tissues of animals (meat, milk, eggs, as well as fish and other aquatic organisms). At the same time, primary infection of meat and milk is observed in the case of generalization of salmonellosis, staphylococcosis, escherichiosis, clostridiosis

in animals. Primary (transovarial) contamination of chicken and duck eggs is observed in salmonellosis;

2. Secondary infection of food products in the process of their receipt, processing, transportation, storage and sale. At the same time, the penetration of pathogens of most intestinal infections into food is possible with secretions of animals, birds and rodents. It is possible that microorganisms may enter meat products from the intestines of farm animals in violation of the rules of their slaughter and butchering. Exogenous infection of eggs can occur due to the penetration of microflora (for example, salmonella) through an intact shell. Infection of food with pathogens of intestinal infections from people (patients, carriers). Infection of food products is not excluded by arthropods (flies, cockroaches).

Pathogens can enter food products through the contaminated hands of the source of infection (it is possible to infect products when using contaminated water for washing dishes, in which the products are then placed, or for washing the products themselves). It is dangerous to infect the source of infection with the hands of a product after heat treatment (dairy, meat, fish products, confectionery) or a product that is not subjected to heat treatment (vegetables, fruits).

Local food outbreaks, which are the result of the accumulation of the pathogen in the product just before its consumption, develop, as a rule, acutely: a sharp rise in morbidity, a sharp decline (if the product is completely consumed after a single consumption), then a sluggish course of the epidemic process (the so-called contact tail) is possible for some time.

Food outbreaks are monoethiological, all infected people have the same culture of the pathogen. Diseases are most often severe, because the consumption of an infected product is accompanied by the receipt of large doses of the pathogen. The incubation period is usually minimal, so the previous episode of infection should be searched within this incubation.

The water route. Water is often polluted by faecal matter entering the soil (sewer system). This process of water contamination can be both short-term and long-term.

Aquatic intestinal outbreaks are recorded much less frequently than food outbreaks. At the same time, rotaviruses, enteroviruses, Flexner's shigella and cholera vibrios are more often used as an etiological agent.

The following types of aquatic outbreaks of intestinal infections are distinguished:

1. When using water from a centralized household and drinking water supply system, as a result of violations of its purification and disinfection regimes at sewage treatment plants, as well as due to the ingress of pathogens into the water after its purification at head facilities and in the distribution network.

2. When using household drinking water pipes without purification by groundwater;

3. When using well water infected by the penetration of feces into the well from closely located toilets or with stormwater and flood waters;
4. When using the water of open reservoirs polluted by surface waters or as a result of the discharge of fecal waters;
5. When using water from small containers (barrels, cisterns, tanks, etc.);
6. When using water from technical water pipes.

Water epidemics and outbreaks can be acute or chronic. Acute water epidemics are very indicative of a combined accident of centralized water supply and sewerage systems. Large water epidemics (outbreaks) are usually polyethological, and there is a consistent development of epidemics (outbreaks) of various intestinal diseases.

Chronic water epidemics are more common than acute ones. They have a wide territorial distribution, many more people get sick due to chronic water epidemics than due to acute ones. Chronic water epidemics, like acute ones, are polyethological.

Contact and household transmission route — infection due to contaminated household items (toys, dishes) is realized primarily in children's institutions where the necessary sanitary and hygienic regime is not observed.

When contact-household transmission is active, there must necessarily be foci (the probability of infection by contact-household means depends on the closeness of communication), as well as the slow development of the epidemic process. The slower the sources of infection are removed from the focus and the worse the sanitary and hygienic conditions in the team (or sometimes in the family), the more likely the development of morbidity due to the contact and household transmission route.

## **VIRAL HEPATITIS A**

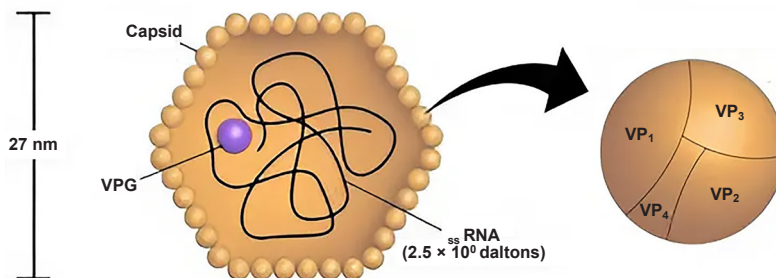
Viral hepatitis A is an acute anthroponous infectious disease characterized by predominant liver damage and, in clinically pronounced cases, general toxic symptoms and jaundice.

Viral hepatitis A remains one of the most common viral hepatitis in the world. According to WHO, about 1.5 million people get hepatitis A every year in the world. At the same time, these figures do not reflect the true picture of the spread of hepatitis A, which often occurs in jaundice-free or latent (subclinical, inapparent) forms, which, as a rule, are not diagnosed. Viral hepatitis A is characterized by pronounced socio-economic significance. According to the standards of management of this disease, the period of temporary disability is on average 35 days. In moderate to severe forms of the disease, mandatory hospitalization of the patient and a set of antiepidemic measures are required to prevent the occurrence of foci of infection.

## EPIDEMIOLOGICAL FEATURES OF THE CAUSATIVE AGENT OF VIRAL HEPATITIS A

The causative agent is the hepatitis A virus (HAV). The virus belongs to the Picornaviridae family, the genus Hepatovirus. Morphologically, the HAV looks like a small spherical particle with a size of 27–30 nm (Fig. 1).

### Hepatitis A Virus — Picornavirus



*Fig. 1. The structure of the hepatitis A virus*

The virus genome is represented by a single-stranded RNA consisting of approximately 7,500 nucleotides. The RNA is surrounded by a protein capsid, there is no outer glycoprotein envelope.

In 1992, R. Jansen and V. Robertson established the presence of 7 genotypes of the HA virus. Genotypes I, II, III, VII were isolated from human patients with GA, and IV, V and VI — from monkeys with hepatitis clinic, genotype III was also isolated from monkeys. Genotypes I and III were divided into 2 subtypes: A and B. Subsequently, when studying 86 isolates from around the world, researchers were able to establish that the differences between genotypes II and VII turned out to be insignificant, based on this they began to be determined by subtypes of the same genotype: IIA and IIB.

Genotype I has become widespread geographically, accounting for 80 % of HA cases in the world. Subtype IA was identified in the territory of the post-Soviet space, in the USA, South America, Europe, the Middle East, Central Asia, the Russian Federation, and Japan. Subtype IA is widespread in the Mediterranean countries, in Germany, in Africa, in Brazil, on the Australian continent.

Genotype II is confined to African countries. Subtype IIA is found in West African countries and in France, where it was introduced from African countries. Recently, subtype IA has been detected among residents of France who have not previously left the country, which indicates the expansion of the boundaries of



the distribution of this subtype. Subtype IIB was established only in patients from Sierra Leone.

Genotype III has a fairly wide territorial distribution. Subtype IA dominates in Malaysia, India, Sri Lanka, and post-Soviet Asian countries. Circulation of IIIA is also established in Europe, the USA, Japan, and certain regions of Russia (Yakutia, Tyva). According to the literature, subtype IIIB is found in isolated cases in Japan and European countries (Fig. 2).

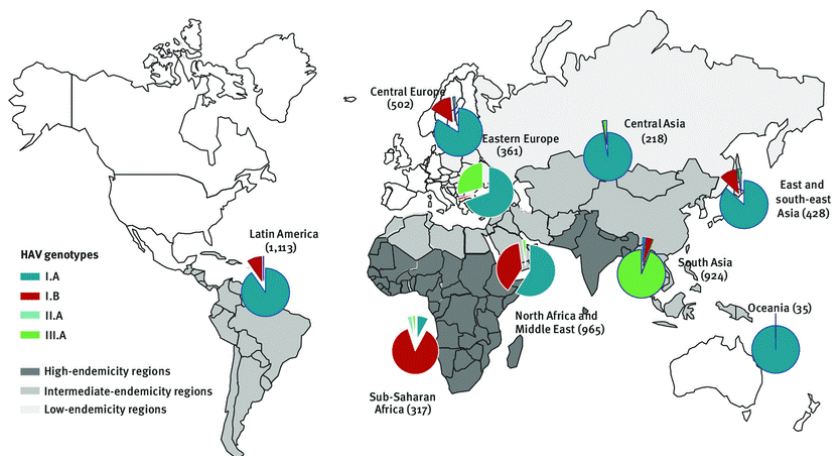


Fig. 2. Cartogram of the spread of hepatitis A virus genotypes

Genotyping and phylogenetic analysis of circulating virus variants form the basis of molecular genetic monitoring in epidemiological surveillance of HA at the national and regional levels in many countries of the world, including the Republic of Belarus. The results of molecular epidemiological monitoring of the HA virus population in Minsk during 2006, 2008–2012 showed the circulation of subtypes IA, IB and IIIA. Subtype IA was mainly imported from Kyrgyzstan, subtype IB was imported from Egypt, subtype IIIA was imported from Tajikistan.

HAV contains a unique protein pX, the main function of which is to interact with proteins of the endosomal transport complex during the formation of quasi-shell particles. The formation of quasi-enveloped forms of the virus occurs as follows: after the assembly and packaging of viral RNA into the protein envelope, viral particles move to the surface of the endosomes through the interaction of VP2 and VP1pX proteins with the components of the endosomal sorting complex. Budding of capsids inside endosomes leads to the formation of multivesicular bodies. Multivesicular corpuscles are transported to the cell membrane, after which the membranes merge and quasi-sheathed (“wrapped in a membrane”) virions are

released into the extracellular space. Quasi-enveloped virions are formed only in infected cells and are contained in the blood. There is also a non-enveloped form of HAV, which is formed when the virus passes through the membrane of hepatocytes into the lumen of the bile capillaries, where the maximum concentration of bile acid salts is. It is under the influence of bile acid salts that the bilipid layer is lost and the virus acquires stability, which determines its long-term preservation in the environment. Non-enveloped virions are present in bile, faeces and in the external environment. The ability of virions to lose the bilipid layer determines their stability in the environment.

The hepatitis A virus can persist at room temperature for several weeks, at +40 °C for months, at -20 °C for several years. At a temperature of 60 °C, HAV persists for 60 minutes, partially inactivated in 10–12 hours. Boiling for 5 minutes leads to the complete destruction of virions. HAV is resistant to ether, chloroform, and freon. HAV is inactivated by formalin at a concentration of 3 % for 5 minutes at a temperature of +25 °C, at a concentration of 8 % — for 1 minute at the same temperature. Chloramine at a concentration of 2.0–2.5 mg/l inactivates the virus for 15 minutes; sodium hypochlorite (3–10 mg/l) at a temperature of +20 °C — for 5–15 minutes; iodine-containing disinfectants (3 mg/l) — for 5 minutes; potassium permanganate (30 mg /l) — for 5 minutes.

### **THE MECHANISM OF DEVELOPMENT OF THE EPIDEMIC PROCESS**

**The source of the infection.** HAV is an anthroponotic infection. The source of infection is patients with any forms of acute infectious process — manifest (jaundice, jaundice-free) and latent (subclinical and inapparent). Hepatitis A virus antigen in faeces was identified 11 days before the appearance of jaundice, while the maximum excretion of the virus is noted 6–7 days before the development of jaundice, with the appearance of jaundice, the massiveness of the pathogen release decreases sharply, with in this case, the antigen is detected in the stool in 20 % of cases.

In general, the period of infection is 14–21 days and at the 3rd week of the disease, the HAV antigen is detected in no more than 5 % of cases. The period of contagiousness in non-jaundice and subclinical forms generally corresponds to the period of contagiousness in jaundice forms. In the early days of the disease, HAV is detected in infected individuals in the blood. Virology is short-lived and has no great practical significance.

In the structure of the sources of infection, non-jaundice and latent forms account for about  $\frac{2}{3}$  of the diseases. Taking into account the fact that with non-jaundice and latent forms of HAV, the duration of the period of infection is also 2–3 weeks, it is these clinical forms that are the leading categories of sources of infection in this disease. The prevalence in the structure of infection sources in patients with latent and jaundice-free forms is especially characteristic in

childhood. Among adults, the ratio of manifest and latent forms is 5 : 1. In this regard, young children with asymptomatic forms of HAV are the most important source of infection for other children and adults who are not immune to this disease.

At the age of 3 to 10 years, the HAV infection index reaches 0.6–0.8, which means that 60–80 children are infected with HAV per 100 children who have been in contact. In people aged 15 years and older, the index of contagiousness decreases to 0.2.

**The transmission mechanism.** The hepatitis A virus is excreted in the stool, less often in the urine, in exceptional cases — with secretions from the nasopharynx. Taking into account the primary localization of the pathogen in the intestine and its excretion with feces, HAV is transmitted by the *fecal-oral transmission* mechanism. In the process of implementing this mechanism, the virus spreads through transmission factors (food, water and household items).

Through household items, HAV is transmitted in kindergartens, schools, boarding schools, summer health facilities, organized groups of adults, especially those in unfavorable sanitary and hygienic conditions. The spread of infection is facilitated by: non-compliance with the isolation of groups in children's institutions, the formation of "prefabricated" round-the-clock and extended-day groups, violation of the anti-epidemic regime, late detection and isolation of patients. The risk of infection increases during the formation of new children's groups. At the same time, the leading transmission factors are dishes, towels, linen, toys, personal hygiene items, etc. Infection is also possible through direct contact with the patient. For example, children attending nursery and kindergarten groups of children's educational institutions, employees of preschool institutions caring for children, service personnel in institutions for the mentally retarded are infected in this way. Household items occupy a leading place in the transmission of HAV in areas with low incidence of this infection.

**The water transfer factor** is implemented mainly in areas with a low degree of communal amenities. Well-established centralized water supply and wastewater disposal systems prevent the spread of HAV through the water factor. However, even in these conditions, there is a constant danger of transmission of pathogens in this way in case of accidents or defects in the purification of drinking water. At the same time, it should be borne in mind the high resistance of HAV in the aquatic environment and its low sensitivity to chlorine-containing disinfectants used for disinfection of drinking water. In accordance with regulatory documents, the content of residual active chlorine in tap water is 0.3–0.5 mg/l, which is not enough for complete inactivation of HAV. The spread of viruses mainly by water causes the active involvement of a large number of susceptible individuals in the epidemic process, which is manifested by high levels of morbidity. The different activity of the HAV transmission waterway in different administrative territories makes it possible to distinguish areas acting as «epidemic donors» (suppliers of infection)

and areas that are mainly «epidemic recipients» (consumers of infection) from other territories where an independent epidemic process is being implemented. In conditions of low intensity of the epidemic process, the activity of the waterway is estimated as low, nevertheless, it is assigned the role of a «trigger» factor in the development of the epidemic process of the HAV.

When HAV is transmitted through the **food factor**, outbreaks develop. Usually, food outbreaks of HAV present great difficulties for epidemiological diagnosis, due to the complexity of the indication of the pathogen in food and the long incubation period. The literature provides data on food outbreaks of HAV, during which pathogens were spread through products that were not sufficiently heat-treated, or seeded ready meals (salads, cold snacks, vinaigrette, juices, dried melon, milk, ice cream, etc.). Food outbreaks of HAV associated with the use of freshly frozen strawberries and other berries, the cultivation of which was carried out using human feces as fertilizer, as well as seeded shellfish and crustaceans, raw oysters, which are capable of accumulating the virus in contaminated water are described.

**Contact-household transmission** of the virus is associated with a sufficient degree of contagiousness of this disease. The hepatitis A virus persists for a long time on household items. Transmission can occur through direct hand contact from person to person, during sexual contacts. Based on data on the high (more than 30 % of the surveyed) infection rate of homosexuals, the sexual route of transmission of HAV is allowed during oral-genital and, especially, oral-anal contacts. Household items contaminated with the virus are a transmission factor.

There are various opinions about the role of flies in the spread of HAV, but apparently flies are of limited importance in the contamination of potential transmission factors of pathogens of this infection.

During the circulation of the virus in the blood, it is possible to implement a **parenteral mechanism** of infection — through the blood (posttransfusion infection of patients with hemophilia is described, high infection of HAV addicts who inject drugs intravenously is indicated).

**Susceptibility and immunity.** If the child was born from a seronegative mother, that he is highly susceptible to HAV, starting from the first days of life. Newborns from seropositive mothers receive antibodies against HAV from them and retain immunity during the first year of life, after which they become highly susceptible to this infection. In the future, fluctuations in susceptibility to HAV in different age groups depend on the activity of transmission mechanisms that determine the intensity of the pathogen circulation, the likelihood of infection and subsequent formation of immunity. In modern conditions, there are significant changes in the parameters of the epidemiology of HAV, which determine a different type of endemicity.

Hyperendemic territories are characterized by a high incidence rate. Children are mainly involved in the epidemic process, who are seropositive in 90 % of cases

by the age of 10. The type of endemicity in the country is determined based on data on the incidence and proportion of seropositive individuals in the age groups of the population (Table 1).

As a result of the disease, a stable natural immunity is formed (possibly lifelong), manifested by the presence of IgG anti-HAV antibodies in the blood. IgM anti-HAV antibodies appear in the blood on the 10–15th day after infection and can persist for up to 4–6 months.

*Table 1*

**Proportion of seroprevalent persons in territories with different levels of endemicity**

<b>The level of endemicity</b>	<b>Percentage of seropositive</b>
High level	≥ 90 % by the age of 10
The average level	≥ 50 % by the age of 15 < 90 % by the age of 10
Low level	≥ 50 % by the age of 30 < 50 % by the age of 15
Very low level	< 50 % by the age of 30

## **THE MAIN CLINICAL MANIFESTATIONS OF VIRAL HEPATITIS A**

The incubation period ranges from 7 to 50 days, in most cases it does not exceed 35 days. The disease begins with an increase in body temperature. Muscle pain and pain are characteristic. Weakness, lethargy, headaches, sleep disorders are noted. From the first days of the disease, appetite worsens. There is a feeling of bitterness in the mouth, unpleasant sensations, sometimes pain in the epigastric region, nausea, vomiting. Less often, intestinal function is impaired — a delay or, conversely, a relaxation of the stool. A similar condition in typical cases of the disease lasts 4–5 days, and then the most typical symptom for viral hepatitis A occurs: jaundice staining of the skin and mucous membranes. With the appearance of jaundice, the well-being of patients improves. Gastrointestinal disorders decrease, and appetite improves rapidly. Patients are less concerned about nausea, vomiting stops.

## **LABORATORY DIAGNOSTICS**

Reliable confirmation of the diagnosis of viral hepatitis A is achieved by the detection of specific antibodies (anti-HAV) belonging to class M immunoglobulins (anti-HAV JgM) in the blood serum of the subject.

In hepatitis A, antibodies related to JgM are detected in the blood during the incubation period, 5–10 days before the first symptoms of the disease appear. By the time of the initial treatment of the sick patient to the doctor, the level of anti-HAV JgM

manages to reach high levels in order to be detected by the method of immunoassay. Anti-HAV JgM in patients appear at the beginning of clinical manifestations of the disease and persist up to 6 months after infection. The definition of anti-HAV JgM is the main test for the specific diagnosis of hepatitis A.

Antibodies of the JgG body to hepatitis A virus (anti-HAV JgG) appear in the blood during the clinical manifestations of viral hepatitis or during the convalescence phase, therefore they cannot serve as a criterion for early diagnosis of hepatitis A. However, the absence of anti-HAV JgG during the height of hepatitis makes it possible to exclude its association with hepatitis A virus. Quantitative determination of anti-HAV JgG It allows you to observe the dynamics of the increase or decrease in the level of specific antibodies. Detection of anti-HAVJgG in healthy people indicates previous infection and immunity (retrospective dynamics).

The hepatitis A virus is normally absent in the blood. PCR makes it possible to qualitatively determine the hepatitis A virus RNA in the blood. The determined fragment is a conservative section of the hepatitis A virus genome.

Qualitative determination of the hepatitis A virus by PCR in the blood makes it possible to confirm the presence of the virus in the patient's body and thereby establishes the etiology of the disease.

## **MANIFESTATIONS OF THE EPIDEMIC PROCESS**

Hepatitis A is a disease that is widespread, although the incidence rates vary widely in different regions of the world. The tense situation regarding this disease persists in countries with a high intensity of the epidemic process, where the incidence rates exceed 100 cases per 100,000 populations. First of all, these are the countries of Africa, Central Asia, South Asia and Southeast Asia, the countries of South America and the Caribbean. The migration factor in the epidemic process of HAV plays an important role in hypo-endemic territories, where imported cases are increasingly being registered. Hepatitis A is among the most common vaccine-preventable infections acquired during travel. Cases of travel-related hepatitis A can occur in travelers to developed and developing countries and who have standard tourist accommodations, eating behaviors, and itineraries.

Risk is greatest for those who live in or visit rural areas, trek in backcountry areas, or frequently eat or drink in settings with poor sanitation. Common-source food exposures are increasingly recognized as a risk for hepatitis A, and sporadic outbreaks have been reported in Australia, Europe, North America, and other regions with low levels of endemic transmission.

Multinational HAV outbreaks among men who have sex with men (MSM) have been described, including, since 2016, among MSM who travel to areas in European Union countries with ongoing HAV transmission among MSM.

Hepatitis A is common in areas with inadequate sanitation and limited access to clean water. In highly endemic areas (parts of Africa and Asia), a large proportion of adults in the population are infected as children, are immune to HAV, and epidemics are uncommon. In areas of intermediate endemicity (Central and South America, eastern Europe, parts of Asia), childhood transmission is less frequent, more adolescents and adults are susceptible to infection, and outbreaks are more likely. In areas of low endemicity (western Europe, the United States), infection is less common, but disease occurs among people in high-risk groups and as communitywide outbreaks. Determining HAV endemicity globally is complex, however, and limited data are available on subpopulation variation of HAV antibody seroprevalence within regions (Fig. 3).

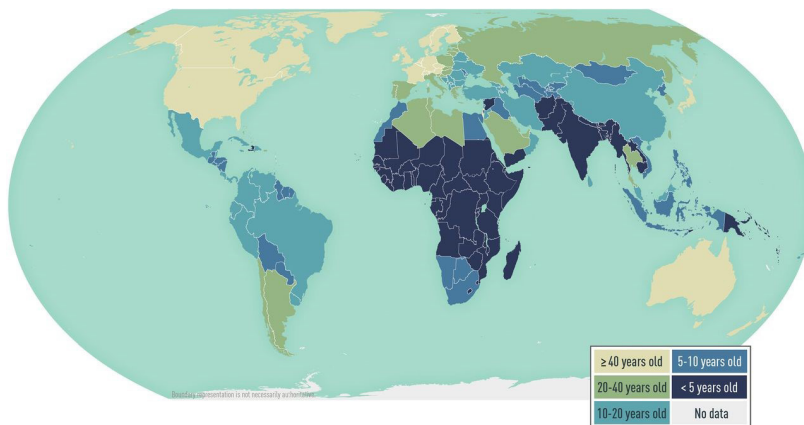


Fig. 3. Estimated age at midpoint of population immunity to hepatitis A, by country

The Republic of Belarus belongs to the territories with a low incidence of hepatitis A. The incidence rate in the country is 0.2–0.4 cases per 100,000 population. The largest number of diseases occurs in the autumn-winter period. Most of the cases are adults aged 18–39 years (the proportion of children does not exceed 20 % of cases).

## PREVENTION

Non-specific prevention. Based on the generally accepted grouping of anti-epidemic measures according to the direction of their action, when developing preventive action plans, it should be borne in mind that measures aimed at the source of infection in HAV have weak potential effectiveness. This is due to the fact that the vast majority of cases of HAV disease occur in an asymptomatic

form and remain clinically unrecognized or recognized late (with the appearance of jaundice), when the massiveness of the pathogen is significantly reduced. The effectiveness of measures for early detection of patients can be increased in the event of the occurrence of CAA diseases in organized groups, when medical supervision is established for persons who have communicated with the patient and they are subjected to laboratory examination.

**Measures aimed at breaking the transmission mechanism are of great importance for the prevention of HAV. At the same time, the most important measures are:**

- providing the population with high-quality food products and epidemiologically safe drinking water;
- rational solution of issues of communal well-being of settlements;
- introduction of new advanced technologies into the practice of water treatment and wastewater treatment, purification and disinfection of drinking water, sewage and industrial waste;
- compliance with sanitary rules and standards of operation of food industry and catering enterprises;
- observance of sanitary and hygienic and anti-epidemic regime in preschool institutions, schools and organized groups;
- laboratory monitoring of the state of environmental objects in terms of viral contamination: the presence of HAV antigen (ELISA), coliphages and enteroviruses;
- monitoring compliance with the rules of personal hygiene by the staff of food, preschool and equivalent institutions;
- sanitary and educational work among the population.

**Specific prevention.** Vaccination against HAV is currently considered as the most important preventive measure in countries with both widespread and low incidence of this disease. Currently, the following types of HAV vaccines are used in the world: formaldehyde inactivated, live attenuated, and virosomal.

Modern live attenuated vaccines against HAV, produced on the basis of the H2 virus strain and the L-A-1 strain, were licensed in China in 2008 for subcutaneous administration to children aged 1 year and older. Vaccine strains of HAV were attenuated by multiple virus passaging in monkey cell culture followed by reproduction in human diploid embryonic pulmonary fibroblasts.

The live attenuated vaccine is administered once subcutaneously. Live vaccines against HAV have not been widely used in the world due to their reactogenicity and a significant list of contraindications.

Based on the accumulated global experience in the process of using several hundred million doses, it was found that formaldehyde-inactivated HAV vaccines have excellent safety characteristics, regardless of the vaccination calendar and manufacturer. The following vaccines against HAV have been registered and



approved for use in the Republic of Belarus: Avaxime (Sanofi Pasteur, France), Havrix 720 and 1440 (GlaxoSmithKline, Belgium).

**Indications for vaccination.** In regions with high endemicity, vaccination against HAV is recommended for all susceptible populations. In regions with low or moderate endemicity, immunization is especially recommended for people with an increased risk of infection, as well as for those who may have a severe course of

HAV or for people whose hepatitis A disease, due to their professional affiliation, can lead to outbreaks (Fig. 4). These include:

- travelers: persons traveling to hyperendemic regions or to regions where some outbreaks of HAV are registered;
- military personnel, employees of military units;
- junior medical personnel, especially in infectious diseases, gastroenterology and pediatric medical institutions;
- employees of sewerage systems and water treatment plants;
- employees of catering and food industry enterprises, food warehouses;
- personnel of closed institutions, social security and medical institutions;
- persons living or staying in social centers;
- persons with a behavioral risk of HAV infection: homosexuals; persons who lead a promiscuous sex life;
- drug addicts who inject drugs;
- patients suffering from hemophilia;

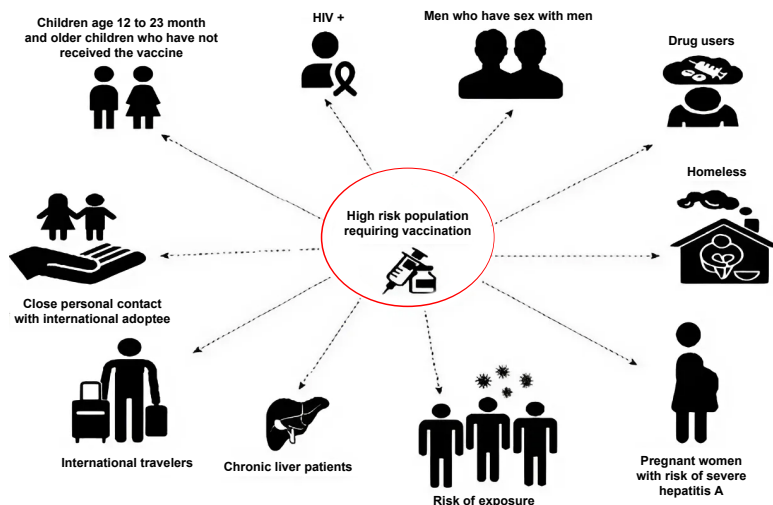


Fig. 4. The Centers for Disease Control and Prevention (CDC) recommend hepatitis A vaccination for different groups of populations

**Vaccination schemes and method of application.** The full course of vaccination consists of two doses. The interval between the first and second management is 6–12 months. For patients on hemodialysis with immune defects, an additional dose is recommended 1 month after the first one. The age at which the HAV vaccine can be administered is 1–2 years old. Up to 16–18 years of age, a children's dosage of the vaccine is used, containing a half dose of the vaccine (0.5 ml) used for adults (1 ml). All inactivated vaccines are administered intramuscularly: to children under 18 months of age in the anterolateral region of the thigh, to children over 18 months of age, to adolescents and adults — in the deltoid muscle. In special cases, in patients with thrombocytopenia or suffering from bleeding, the vaccine can be administered subcutaneously.

Immunogenicity and duration of circulation of postvaccinal antibodies. The protective effect of vaccination against HAV is manifested already from the end of the first week, therefore, the use of vaccines in foci is effective.

Clinical studies of the Havrix vaccine have shown that 99 % of vaccinated patients achieve seroprotection 30 days after the first dose ( $> 20$  mMU/ml). The data obtained with a double injection of the vaccine at an interval of 6–12 months allow us to conclude that 97 % of those vaccinated 25 years after the course will have a protective antibody titer ( $> 20$  mMU/ ml). In addition, it is possible to protect the human body, previously vaccinated against HAV and lost anti-HAV (below the level of their detection using modern methods) due to cellular immunity. Therefore, persons with unchanged immune status do not need to undergo additional revaccination after a course of vaccination consisting of two doses.

#### **Contraindications to vaccination:**

Hypersensitivity to any component of the vaccine; symptoms of hypersensitivity to previous administration of the vaccine. Acute infectious and non-communicable diseases, exacerbation of chronic diseases are temporary contraindications for vaccinations; with mild acute respiratory viral infections, acute intestinal diseases, vaccinations are carried out immediately after normalization of temperature.

**Side effects.** HAV vaccines are usually well tolerated. Local reactions may develop: short-term soreness at the injection site; redness and swelling.

General reactions are usually mild. They can manifest as headache, malaise, vomiting, fever, nausea and loss of appetite, muscle or joint pain. All these undesirable phenomena go away without consequences. In rare cases, there was a slight reversible increase in the activity of liver enzymes (transaminases).

### **ANTI-EPIDEMIC MEASURES**

Patients with viral hepatitis A are subject to hospitalization for clinical and epidemic indications.

Clinical indications for hospitalization are: the disease of children under the age of 2 years; severe and moderate clinical course; the need for etiological differentiation of viral hepatitis; the presence of other diseases in patients.

According to epidemic indications, patients with viral hepatitis A are hospitalized in cases where it is impossible to ensure compliance with the anti-epidemic regime at their place of residence, as well as if there are preschool children in the apartment center who have not previously had this infection.

Separate placement of patients with viral hepatitis A and other viral hepatitis is necessary in hospitals, and the antiepidemic regime must be strictly observed in them.

The discharge of convalescents and their admission to work (including employees of food enterprises and persons equated to them), as well as the discharge and admission of children to preschool institutions are carried out depending on the state of health. Dispensary monitoring of patients is carried out no later than 1 month after discharge by the attending physician of the hospital. In the absence of any clinical and biochemical abnormalities in the convalescents, they can be removed from the register. Reconvalescents with residual effects are registered in the KEYES after 3 months, where they are re-examined.

Persons who have been in contact with a patient with viral hepatitis A are subject to medical supervision at the place of residence, work or visit of an organized team for 35 days. The observation is carried out without separation from work or an organized team. Adults, as well as students of schools and boarding schools, are interviewed weekly, examined, thermometry, etc.

Children attending preschool institutions are monitored daily. According to the testimony of persons who have communicated in epidemic foci, vaccination is carried out. The necessity and frequency of laboratory examination of those who communicated (activity of liver enzymes in the blood, specific markers of hepatitis A virus) is determined by a pediatrician (infectious disease specialist) and an epidemiologist.

If the communicating children are preschool children, then in the group where a patient with viral hepatitis was identified, regime-restrictive measures are carried out during the observation period: separation from other groups; it is prohibited to transfer children from the quarantine group and accept new children; the self-service system is canceled in the quarantine group; The quarantine group does not participate in mass events held in rooms shared with other groups. The current and final disinfection is carried out by boiling and using chemicals.

## **ROTAVIRUS INFECTION**

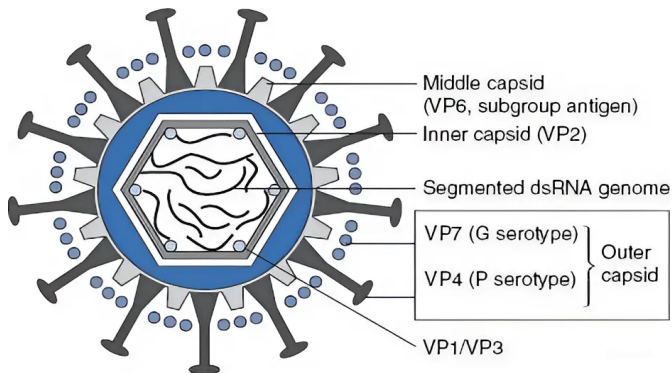
Rotavirus infection is an acute infectious disease characterized by damage to the gastrointestinal tract and manifested by symptoms of intoxication, diarrhea, vomiting and the development of dehydration.

Rotavirus is the most common cause of severe diarrhoeal disease in infants and young children globally. It is transmitted through the oral-faecal route, directly

from person to person, or indirectly through contaminated objects. People that are infected experience an abrupt onset of fever and vomiting followed by explosive, watery diarrhoea. Rotavirus diarrhoea is profuse, often leading to dehydration which can be severe, requiring hospitalization. The cornerstones of treatment of severe rotavirus diarrhoea are fluid replacement and zinc supplementation.

### EPIDEMIOLOGICAL FEATURES OF THE CAUSATIVE AGENT OF ROTAVIRUS INFECTION

The causative agent is a virus that belongs to the genus Rotavirus, family Reoviridae. Viral particles with a diameter of 65–75 nm have a two-layer capsid shell with a clearly defined edge, giving them the appearance of a wheel (Latin rota). The virus contains RNA. According to the group-specific antigen (structural protein of the internal capsid VP6), all rotaviruses are divided into 7 serogroups — A, B, C, D, E, F, G. A schematic representation of the structure of a mature rotavirus particle is shown in the figure (Fig. 5). The three concentric capsid protein layers are colored such that red represents VP4 spikes, yellow is the VP7 layer forming the outer layer, blue is the VP6 layer, and green is the VP2 layer. The dsRNA segments (brown) are packed inside the core associated to the RNA polymerase complex (VP1 and VP3, red balls).



*Fig. 5. Schematic rotavirus structure*

Up to 98 % of rotavirus diarrhea in children is caused by rotaviruses of serogroup A, in other cases, rotaviruses of serogroups B and C are isolated from patients. Within group A, subgroups are distinguished (by the structure of the structural protein VP7) and serovars (by the structure of the structural protein VP4). There is a constant change in the antigenic structure of viruses between seasons and between different regions. The most common cause of rotavirus

infection in developed countries are G1, G2, G3, G4 serovars, G9 serovars are found in developed and developing countries. Different variants of rotaviruses are often circulating among children of different age groups.

Human rotavirus infection can be experimentally reproduced in monkeys, piglets, and calves, but the possibility of interspecific infection *in vivo* has not been established.

Rotaviruses do not have a supracapsid shell, so they are relatively stable in the environment. The absence of lipids causes resistance to ether, chloroform, detergents. They are resistant to ultrasound.

Rotaviruses remain viable for up to one month in various environmental objects, and up to 7 months in faeces. In tap water at 20–40° they are stored for more than 2 months, on vegetables at 4 °C — 25–30 days. Proteolytic enzymes (pancreatin, trypsin, elastase, etc.) enhance the infectious activity of the virus. Rotaviruses are rapidly inactivated by exposure to most disinfectants and boiling.

### **THE MECHANISM OF DEVELOPMENT OF THE EPIDEMIC PROCESS**

**The source of the infection.** The source of infection is patients who secrete the pathogen with feces in very large quantities — 1010–1011 viral particles per 1 gram of feces. The period of infection is on average 7–8 days from the onset of clinical manifestations, and may increase in some cases up to 3 weeks. Persons carrying a mild infection, as well as virus carriers, are of particular epidemic importance. Persons without clinical manifestations of the disease can secrete the pathogen for up to several months. In the foci of rotavirus infection, asymptomatic carriers of rotaviruses are more often detected among adults, whereas the main group of patients are children. Asymptomatic carriers of the virus (as sources of infection) are of great importance, especially among children of the first year of life, most often infected from their mothers. Adults and older children become infected from sick children attending preschool institutions. Children with impaired cellular immunity slowly recover from rotavirus infection, the disease acquires a chronic course with prolonged release of rotaviruses.

**The transmission mechanism.** The localization of the pathogen in the intestine determines the fecal-oral transmission mechanism.

Rotaviruses are highly contagious. The infectious dose is low and the virus is shed in large quantities, as much as 1011 particles per gram of stool, both before the onset of symptoms and for several weeks afterwards. Furthermore, the virus survives on dry surfaces for as long as 10 days and on human hands for up to 4 hours.

The main factors of transmission are household items, most often children's toys, nipples, hands contaminated with the pathogen of adults and children. The implementation of the fecal-oral transmission mechanism is facilitated by the high concentration of rotaviruses in faeces, their relative resistance to environmental

factors and the long-term persistence of infectivity in the external environment. In addition to household items, rotaviruses are spread by water and food factors.

The possibility of transmission of rotaviruses by an aerosol transmission mechanism is not excluded. The basis for such an assertion is the detection of the pathogen in nasopharyngeal mucus, as well as the ease of spread of rotavirus infection with crowded placement of young children. It has been shown that in children's infectious diseases hospitals, a significant part of children in the first month of life with acute intestinal infections become infected with rotavirus infection in the first days of hospital stay.

**Susceptibility and immunity.** The human population is heterogeneous in its susceptibility to rotaviruses. Newborns receive antibodies to rotaviruses from mothers through the placenta, and in the first months of life — together with breast milk. The most susceptible are children aged 6 months to 2 years, as well as the elderly. Susceptibility to rotavirus infection increases against the background of unfavorable premorbid conditions, as well as in children who are on artificial feeding. Antibodies to rotaviruses are detected in almost 90 % of children aged 3–4 years and in almost all adults, which indicates infections with this pathogen in the past. The presence of antibodies in the blood serum does not prevent the development of the disease. Repeated cases of rotavirus infection are possible, which is associated with the loss of acquired immunity or infection with another serovar of the pathogen.

Individuals can experience multiple rotavirus infections throughout their life. However, cumulative immunity following each infection provides increasing protection against subsequent infection and illness. A longitudinal birth cohort study in Mexican infants found that the degree of protection was greatest against severe disease. The initial natural rotavirus infection provided 38 % protection against asymptomatic infection, 73 % protection against mild diarrhea, and 87 % protection against moderate to severe disease. After a second episode the degree of protection increased to 60 % for asymptomatic infection, and 83 % for mild rotavirus diarrheal illness, and complete protection was provided against infection resulting in moderate to severe symptoms. A third infection was needed, however, for complete protection against mild disease.

## THE MAIN CLINICAL MANIFESTATIONS

The incubation period is from 12–24 hours to 4–7 days, most often 12–48 hours. Initially, the body temperature rises, about half of the patients have a moderate increase in body temperature. In some cases, it reaches 40 °C. Abdominal cramps and pains, nausea and vomiting are noted. Diarrhea, which lasts 5–8 days, is most characteristic of rotavirus infection. The stool is mushy or liquid, watery, abundant, without impurities, 5–10 times a day, depending on the severity of

the disease. Due to persistent diarrhea and continuous vomiting, the patient's body is dehydrated. In severe forms, it is possible to develop disorders of water-salt metabolism with circulatory insufficiency, oliguria and even anuria, an increase in the content of nitrogenous substances in the blood.

A characteristic feature of rotavirus infection is the simultaneous development of clinical manifestations from the upper respiratory tract in the form of pharyngitis, rhinopharyngitis or rhinitis. The total duration of the disease ranges from 2 to 14 days. In adults, rotavirus infection usually occurs subclinically. Manifest forms can be observed in parents of sick children, in people who have visited developing countries, with immunodeficiency, including the elderly.

Rotavirus infection can also be asymptomatic, such cases are often found in newborns. This course further protects children from severe forms of rotavirus infection during the first 3 years of life.

### **LABORATORY DIAGNOSTICS**

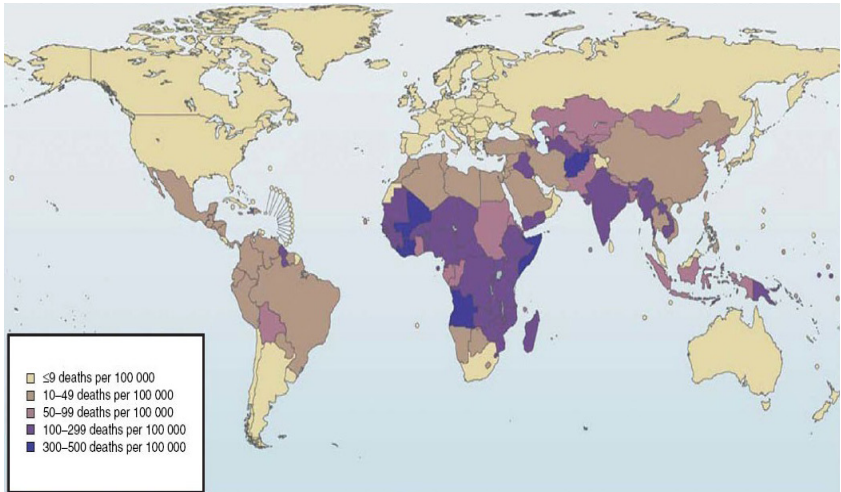
Laboratory diagnostics is based on the detection of the virus in the stool in the first week of the disease (electron and immunoelectronic microscopy, as well as the method of infecting cell cultures). The method of enzyme immunoassay is used to diagnose rotavirus infection, which allows to determine the antigens of the rotavirus capsid. This rapid diagnostic method allows you to quickly identify the pathogen variant. A more sensitive method of diagnosing rotavirus infection is the polymerase chain reaction method, which makes it possible to determine not only the presence of the virus, but also its serovar.

Methods for detecting antibodies (latex agglutination reactions, immunochromatography, hemagglutination inhibition reaction, complement binding reaction) are retrospective in nature, since the confirmation of the diagnosis is considered to be at least a fourfold increase in antibody titers in paired sera taken in the first days of the disease and after 2 weeks. It should be noted that antibodies in the patient's blood appear quite late, so this type of study has no diagnostic significance in the initial period of the disease in adults and children under 3 years of age, and in young children it does not matter at all.

### **MANIFESTATIONS OF THE EPIDEMIC PROCESS**

Rotavirus infection is widespread in many countries of the world. Rotaviruses account for 30–50 % of all cases of diarrhea requiring hospitalization and rehydration therapy. Rotavirus infection accounts for about 25 % of cases of «traveler's diarrhea». Approximately 99 % of rotavirus deaths occur in low and middle income countries (Fig. 6), and more than half of these are from just six countries: India, Nigeria, Congo, Ethiopia, China, and Pakistan. Most deaths occur

in malnourished infants living in socioeconomically disadvantaged rural regions in the low income countries of Africa and Asia, where access to healthcare is poor and where by 5 years of age more than one in 240 children will die from a rotavirus infection.



*Fig. 6. Rotavirus mortality rates*

In India alone, rotaviruses cause more than 120 000 deaths annually, 450 000 hospitalizations, 5 million clinic attendances, and 25 million diarrheal episodes in children < 5 years of age. The Asian Rotavirus Surveillance Network, which encompasses low, middle and high income countries, estimates that 45 % of diarrhea-related hospital admissions within its region are due to rotaviruses. The disease burden is similarly high in sub-Saharan Africa, where an estimated 300 000 children aged < 5 years die each year from rotavirus gastroenteritis. As noted elsewhere, the proportion of gastroenteritis cases due to rotaviruses in young African children increases with disease severity, ranging from 4 % in community-based studies, to 23 % in those attending outpatient clinics, and to 34 % in those requiring admission to hospital.

In contrast to the low income countries of Africa and Asia, the emerging middle income nations of Latin America have lower rotavirus mortality rates. Nevertheless, each year in Latin America, rotaviruses cause 15 000 deaths, and result in 75 000 hospitalizations, 2 million outpatient clinic visits, and an estimated 10 million episodes of gastroenteritis. Recent rotavirus sentinel surveillance results from Latin America show that rotaviruses were detected in 42 % of children admitted to hospital with severe diarrhea.



Diseases are registered mainly among children of the first years of life, children aged 7–12 months are most often ill. Adults get sick more often in families where there are sick children. Elderly people with weakened immune systems are often found among the sick. Children under 2 years of age and the elderly are more likely to have a severe course of the disease.

In temperate countries, rotavirus infections are more often reported in winter. In tropical countries, rotavirus infection occurs all year round with a slight increase in incidence during the cool rainy season.

Rotavirus infection in maternity hospitals and children's hospitals is observed as an inpatient infection. At the same time, children who are artificially fed, have immunodeficiency, and suffer from chronic diseases are involved in the epidemic process.

While deaths from rotavirus are rare in the high income countries of North America, Europe, East Asia, and Australasia, the incidence of disease in young children is similar to that of low and middle income countries, imposing a considerable burden upon their health systems and economies. In the pre-rotavirus vaccine era, rotavirus gastroenteritis resulted in 220 000 annual hospital admissions, 1.8 million healthcare visits, and 7.1 million episodes of diarrhea among children living in high income countries. By 5 years of age, approximately one in 50 children from these wealthy countries will have been hospitalized following a rotavirus infection. Prior to the introduction of rotavirus vaccines in the United States of America, it was estimated that rotavirus led to more than 2.7 million cases of gastroenteritis, 780 000 clinic visits, 164 000 emergency department attendances, as many as 117 000 hospitalizations, and almost 40 deaths each year. Meanwhile, in European countries, rotaviruses are responsible every year for 230 deaths, nearly 90 000 hospital admissions, 700 000 outpatient consultations, and 2.8 million diarrheal illnesses.

For every child admitted to hospital because of rotavirus gastroenteritis in a high income country, approximately ten will be seen in primary care and between 30 and 40 will be managed at home without seeking medical advice. While 20–30 % of children < 5 years of age presenting to their family practitioner with acute diarrhea will have rotavirus in their stools, this proportion increases to 40–60 % for those more severely ill and managed in an emergency department or hospital setting.

Rotaviruses are an important cause of **healthcare-associated infections** in infants and young children. They are responsible for 31–87 % of all healthcare-associated episodes of gastroenteritis, about one-third of which are judged as being severe, and these episodes account for 7.5–32 % of all children with rotavirus diarrhea in hospital. The rotavirus strains in hospital appear to be introduced from the community, possibly by children with prolonged excretion of rotaviruses following severe diarrhea. Asymptomatic healthcare staff may also play a role, since many cases of hospital-associated rotavirus infections occur in young children who have

been in hospital for more than a week and are nursed in areas of the hospital that do not normally care for children with community-acquired gastroenteritis.

## PREVENTION

**Non-specific prevention.** The complex of preventive measures for rotavirus infection includes: careful observance of all hygienic rules (as well as for the prevention of other intestinal infections); prolonged breastfeeding of children; the use of cow's milk and colostrum, which contain antibodies that have a protective effect against human rotaviruses; constant hand cleanliness by parents caring for children; presentation of quality food products intended for children of particularly high requirements; at the slightest suspicion of deterioration in their quality, exclude them from the diet; use only boiled water for drinking, especially in childhood.

Prevention of the spread of rotavirus infection among children attending preschool institutions is achieved by strict observance of sanitary norms and rules of operation of these institutions. In the prevention of nosocomial infections with rotavirus infection, rational hand treatment, the availability of individual patient care items, and compliance with the disinfection regime are important.

**Specific prevention.** The production of neutralizing antibodies can be induced by the surface proteins of rotavirus VP7 (12 G-type antigens are isolated) and VP4 (15 P-type antigens are isolated); about 90 % of cases of rotavirus infection are caused by five main serotypes (G1P8, G2P4, G3P8, G4P8 and G9P8). The protection is not serotype-specific, and the production of neutralizing antibodies to one of the serotypes provides cross-protection to the rest.

Type of vaccine: live attenuated. Inactivated or VLPs-based vaccines are in development, as well as combined vaccines against rotavirus and noravirus infections.

WHO recommends the inclusion of the rotavirus vaccine in the NPI of all countries, considering it a priority, especially in countries with a high mortality rate from rotavirus gastroenteritis. According to the manufacturer's instructions, the vaccination course is carried out in children during the age period from 6 to 32 weeks.

Vaccination schedule: the first dose should be administered to children as soon as possible after the age of 6 weeks (RV5 — from 6 to 12 weeks, RV1 — from 6 to 15 weeks), the maximum age for the last dose is 32 weeks. The interval between doses should be  $\geq 4$  weeks (4–10 weeks for RV5). Usually, the rotavirus vaccine is prescribed simultaneously with the administration of the DPT vaccine or combined CDD-containing vaccines: RV1 with the first and second doses, RV5 with the first, second and third doses.

Premature infants  $\geq 27$  weeks old should be vaccinated according to chronological age according to the usual scheme with the introduction of the first dose at the age of  $\geq 6$  weeks.

Route of administration: for oral administration only; parenteral use is prohibited. The rotavirus vaccine can be used regardless of food intake or any liquid, including breast milk. It should not be mixed with other solutions and vaccines in the same container.

**Contraindications to vaccination:**

- acute infectious diseases accompanied by fever;
- immunodeficiency conditions;
- malformations of the intestinal tract predisposing to invagination of the wall or intestine in the anamnesis;
- diarrhea or vomiting;
- previously occurring allergic reactions to the administration of this vaccine;
- violations of the enzyme complex: insufficiency of sucrose and (or) isomaltase enzymes, fructose intolerance, impaired absorption of the glucose-galactose complex.

### **ANTI-EPIDEMIC MEASURES**

A patient with rotavirus infection should be isolated. Hospitalization is carried out according to clinical and epidemic indications. Clinical indications are: age under one year, severe and moderate clinical course, presence of concomitant diseases. Hospitalization for epidemic indications is resorted to if it is impossible to comply with the anti-epidemic regime at the place of residence, as well as if the work of the sick person is related to food.

Discharge from the hospital is performed after clinical recovery and release from the pathogen. Before discharge, it is advisable to examine the bowel movements for the presence of rotaviruses. Persons who have communicated with a patient with rotavirus infection are subjected to a medical examination, collection of an epidemiological history, and medical supervision is established for them for 7 days without separation from work and visits to organized groups. If necessary, a single laboratory examination is carried out for the presence of rotaviruses in the stool.

The current and final disinfection is carried out using a physical (boiling) and chemical method.

### **SHIGELLOSIS (DYSENTERY)**

Dysentery is an infectious human disease characterized by a predominant lesion of the large intestine. It is manifested by intoxication, frequent and painful defecation, loose stools, in some cases with pathological impurities — mucus and blood. Dysentery is characterized by an acute course, in rare cases the disease acquires a prolonged and chronic course.

## EPIDEMIOLOGICAL FEATURES OF THE CAUSATIVE AGENT OF DYSENTERY

The causative agents of dysentery belong to the genus *Shigella* of the Enterobacteriaceae family. *Shigella* are gram-negative bacteria 2–4 microns long, 0.5–0.8 microns wide, immobile, do not form spores and capsules.

In accordance with the international classification, *Shigella* are divided into 4 subgroups — A, B, C, D, which correspond to 4 species: *S.dysenteriae*, *S.flexneri*, *S.boydii*, *S.sonnei*. In turn, there are 16 serological variants in the *S.dysenteriae* population (1–16). The population of *S.flexneri* consists of 8 serovars (1–5, 6, X, Y variants), while the first 5 serovars are divided into subserovars (1a, 1b, 2a, 2b, 3a, 3b, 4a, 4b, 5a, 5b). The population of *S.boydii* is differentiated into 18 serovars (1–18). *S.sonnei* does not have serovars, but she can be divided into a number of types according to her biochemical properties, relation to typical phages, ability to produce colicins, and antibiotic resistance.

The pathogenic properties of *Shigella* are due to the formation of exotoxins, lipopolysaccharide complex (endotoxin), cytotoxins and neurotoxins. One of the most important indicators of bacterial virulence is invading proteins, which determine the ability of pathogens to intracellular parasitism in colonocytes, enterocytes (to a lesser extent) and macrophages. The causative agents of the main etiological forms of dysentery are characterized by unequal virulence. The greatest entero-, neuro-, cytotoxic activity, as well as hemolytic properties are shown by toxins of pathogens of the *S.dysenteriae* subgroup, especially *Shigella* Grigorieva-Shiga (*S.dysenteriae* 1).

The virulence of *S.flexneri* are also quite high. *S.sonnei*, being less virulent in comparison with other species of *Shigella*, has a number of properties that compensate for the lack of virulence. *S.sonnei* has higher resistance in the external environment, increased antagonistic activity (more often produce colicins) and greater resistance to antibiotics. In the process of reproduction of *S.sonnei*, thermally stable toxic substances accumulate in milk more intensively, in comparison with *S.flexneri*.

*Shigella* (*S.sonnei*, *S.flexneri*) are relatively stable in the external environment and remain viable in tap water for up to one month, in wastewater for 1.5 months, in moist soil for 3 months, and on food for several weeks. *S.dysenteriae* is characterized by less stability. The causative agents of dysentery at a temperature of 60 °C die within 10 minutes, when boiled — instantly. Solutions of disinfectants in normal operating concentrations have a detrimental effect on these pathogens.

## THE MECHANISM OF DEVELOPMENT OF THE EPIDEMIC PROCESS

**The source of the infection.** Dysentery is an anthroponotic infection. The sources of infection are patients with an acute form of the disease, convalescents, as well as patients with prolonged forms and bacterial carriers.

**The transmission mechanism.** *Shigella* is excreted from the human body with feces. The patient is contagious throughout the entire period of the disease, especially in the first 5 days of the disease, when the release of pathogens is carried out most intensively. In the vast majority of patients with acute dysentery, as a result of the initiated treatment, the release of pathogens stops in the first week and only occasionally lasts for 2–3 weeks. Convalescents secrete pathogens before the end of the restoration of the mucous membrane of the large intestine. In some cases (up to 3 % of cases), the carrier may last for several months. The tendency to a prolonged course is more characteristic of Flexner's dysentery and less so for Sonne dysentery.

Dysentery is characterized by a fecal-oral transmission mechanism. The **transmission factors** are food, water, household items.

**Transmission routes:** foodborne, waterborne, household contact. Flies and cockroaches (mechanical carriers) have a certain importance in the transmission of dysentery pathogens (Fig. 7).

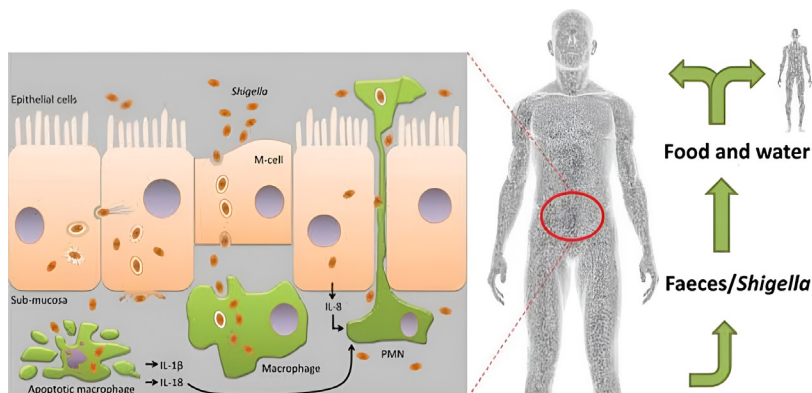


Fig. 7. *Shigella* spread via fecal-oral and person-to-person transmission

A certain relationship between transmission factors and etiological forms of dysentery has been established. In Grigoriev–Shiga dysentery, household items are the leading factors of *Shigella* transmission. This is due to the high virulence and, consequently, the low infecting dose of *Shigella* of this variant, as well as their relatively low resistance in the external environment.

*S. flexneri* are transmitted mainly through the water factor. Sufficiently high virulence and resistance in the external environment greatly contributed to the adaptation of this pathogen to the aquatic factor.

The food factor plays a major role in the spread of *S. sonnei*. The low virulence of *Shigella* of this species determines the high infectious dose. Therefore, pre-accumulation of *S. sonnei* is required for human infection, which occurs on food products. Despite the variety of foods that act as factors of *S. sonnei* transmission, milk and dairy products occupy the main place.

Flies and cockroaches mechanically transfer pathogens from one transmission factor to another, enhance the circulation of *Shigella*, create conditions for more frequent human infections and, thus, contribute to the formation of epidemic variants of pathogens.

**Susceptibility and immunity.** People's susceptibility to dysentery is determined by factors of general and local immunity. The factors of general immunity include serum antibodies of the IgA, IgM, and IgG classes. Local immunity is associated with the production of class A secretory immunoglobulins (IgA) and plays a major role in protecting against infection. Local immunity is relatively short-term and after the disease it provides immunity to repeated infections for 2–3 months. At the same time, there is evidence that after Flexner's dysentery, postinfectious immunity is formed, capable of protecting against recurrent disease for several years.

The human population is characterized by heterogeneity in susceptibility to dysentery, which is associated with the general resistance of the human body, the frequency of infection with *Shigella*, age and other factors.

### THE MAIN CLINICAL MANIFESTATIONS OF DYSENTERY

The incubation period is usually 1–3 days, but can be up to 7 days. An acute onset of the disease is typical. As a result of inflammation of the mucous membrane of the large intestine, abdominal pain, severe intestinal contractions, frequent stools with mucus, sometimes with an admixture of blood. In severe patients, ulcers form on the mucous membrane of the large intestine, and then mucus, pus and blood are found in the stool. The waste products of *Shigella*, as well as substances that are formed as a result of the destruction of these microorganisms, entering the blood, cause intoxication. The patient's body temperature rises, lethargy, weakness appear, and appetite decreases. In severe cases, vomiting begins, loss of consciousness, convulsions, and disruption of the cardiovascular system are possible.

If dysentery develops acutely in most adults, then in young children the disease begins suddenly, manifesting itself with erased signs that sometimes resemble the symptoms of ordinary dyspepsia. But later there are characteristic signs of dysentery — frequent stools with mucus, without blood. A seriously ill young child

has frequent vomiting and pale skin. The child screams before excretion, pulls his legs up to his tummy, his face turns red. There is little excretion — only a small spot of mucus remains on the diaper.

### **LABORATORY DIAGNOSTICS**

Reliable confirmation of the diagnosis is carried out using a bacteriological method — the isolation of *Shigella* from feces and vomit, and in case of Grigoriev–Shiga dysentery — from the blood. Pathogens are present in the stool from the beginning of the disease, throughout the disease, as well as during the period of convalescence. The newly isolated stool, vomit and gastric lavage are used as material for the study. The species of pathogens is determined, as well as serological and enzymatic variants of *Shigella*. Detection of *Shigella* DNA using PCR, as well as cultural research, is informative in the acute phase of the disease.

The use of serological methods of laboratory diagnostics (agglutination reaction, indirect hemagglutination reaction, enzyme immunoassay) is often limited by a slow increase in titers of specific antibodies, which gives only a retrospective result.

In recent years, rapid diagnostic methods have been widely introduced into practice, detecting *Shigella* antigens in feces, saliva and urine (an indirect hemagglutination test with antibody diagnostics, ELISA), as well as complement fixation test and the aggregate-hemagglutination reaction.

### **MANIFESTATIONS OF THE EPIDEMIC PROCESS**

Dysentery is widespread. An estimated 200 million people worldwide suffer from shigellosis, with more than 650,000 associated deaths annually. High morbidity rates are typical for hot countries, which is associated with a low degree of communal amenities in settlements and difficulties in meeting sanitary and hygienic rules by the population. Dysentery is now much less common in temperate countries.

The incidence of Sonnei dysentery in the population of Belarus in the 70s of the twentieth century was hundreds of cases per 100,000 population. In the 80s and 90s the activity of the epidemic process has become significantly lower — dozens of cases per 100,000 population. In recent years, very low incidence rates have been characteristic — less than one case per 100,000 population. All dysentery diseases are represented by isolated cases. Outbreaks of dysentery currently pose only a potential threat.

Dysentery diseases occur throughout the year. At the same time, the pronounced prevalence of dysentery in the warm season is due to an increase in the number of transmission factors, favorable temperature conditions for the

reproduction of *Shigella* in food, more frequent violations of food storage rules, a decrease in the barrier function of gastric juice and a number of other reasons.

The incidence of dysentery in the urban population is 2–3 times higher than in the rural population. The main reasons for the higher incidence of dysentery in cities are: high population density; centralization of public catering and water supply; a developed network of preschool institutions, etc.

Risk groups: children younger than 5 years old, travellers to developing countries, homeless people, men who have sex with men. People who have weakened immune systems may develop a more serious illness.

The highest incidence of dysentery is observed in children from one year to 6 years old. The proportion of dysentery associated with this group is almost half of the total incidence of this infection. Among children of this age, children who attend organized groups are most often ill with dysentery. The reasons for the relatively high incidence of dysentery in preschool children: higher sensitivity of children to dysentery (a lower dose of *Shigella* is required for the development of the disease); insufficiently formed hygienic skills (infections occur more often); close contacts of children in kindergartens create favorable conditions for the implementation of the mechanism of transmission of infection by fecal-oral route.

## PREVENTION

**Non-specific prevention.** Measures aimed at the source of infection in dysentery do not have potential effectiveness. This is due to the fact that the vast majority of cases of dysentery occur in mild form and remain clinically unrecognized or recognized late when the massiveness of *Shigella* secretion is significantly reduced. Secondary cases are rarely observed in home foci of dysentery, which indicates a low risk of patients with this infection to others.

Prevention of dysentery caused by *Shigella* relies primarily on measures that prevent spread of the organism within the community and from person to person (measures aimed at breaking the transmission mechanism). These include:

- hand washing with soap,
- ensuring the availability of safe drinking water,
- safely disposing of human waste,
- breastfeeding of infants and young children,
- safe handling and processing of food,
- disinsection measures aimed at the destruction of flies and cockroaches.

These measures will not only reduce the incidence of shigellosis, but of other diarrhoeal diseases as well. In all cases, health education.

**Water supply.** *Shigella* can pollute water at all stages of its distribution, from the source to the place of consumption. Therefore, measures to ensure safe drinking water, including safe transportation and storage, are important to prevent the spread



of the microorganism. The development of water supply systems or protected water supply sources should be a priority. It is not recommended to use surface water for drinking, for example, water from a river, pond or an open well. If it is necessary to use surface water, it should be disinfected by chlorination or boiling before use. The water source must be protected from contamination by humans and animals. Defecation should not be allowed within a radius of 10 meters from the water source, as well as on the hillside or downstream from it; drainage ditches should be created to prevent storm water and other surface waters from entering the water source. Other sources of water should be used for bathing, washing and other general purposes.

**Food safety.** Food can be contaminated by *Shigella* at all stages of production and preparation, including: during the growing period (by use of human fertilizers), in public places such as markets, during preparation at home or in restaurants, and when kept without refrigeration after being prepared. Every country should have food safety legislation that defines appropriate measures for safe handling and processing of food. Environmental health workers should monitor food-handling practices, including methods used for fly control, and be given the authority to stop street sales or close restaurants when their inspections reveal unsanitary practices.

Individual food safety practices should also be emphasized. Health education for the general population should stress the following key messages concerning the preparation and consumption of food:

- to wash hands thoroughly with soap after defecation and before preparing or eating food;
- to do not eat raw food, except undamaged fruits and vegetables that are peeled and eaten immediately;
- to eat food while it is still hot, or reheat it thoroughly before eating;
- to wash and thoroughly dry all cooking and serving utensils after use;
- to keep cooked food and clean utensils separately from uncooked food and potentially contaminated utensils; and
- to protect food from flies by means of fly screens.

Specific prevention. There is no widely available vaccine for shigellosis. A vaccine against *S.sonnei*, a dysentery bacteriophage, has been developed, however, in conditions of low morbidity, their use is impractical. Several vaccines are currently being developed, mainly against *S.flexneri*.

## **ANTI-EPIDEMIC MEASURES**

The duration of sanitary and anti-epidemic measures in the epidemic focus of dysentery is established from the date of separation from the last patient with dysentery for a period of 7 calendar days.

Patients with dysentery are subject to isolation at home or hospitalization in an infectious diseases hospital or an infectious diseases department of a hospital

organization for clinical and epidemiological indications. Clinical indications for hospitalization are severe dysentery, as well as the detection of the disease in children under the age of one year (with any severity) and in patients over 60 years of age with moderate clinical forms and in patients with chronic diseases, that can complicate the course of dysentery. Epidemic indications for hospitalization of patients with dysentery are: disease for persons staying in children's homes, children's boarding institutions; disease in a patient hospitalized in hospital organizations of a non-infectious profile; disease in a patient from a dysentery focus in the absence of conditions for sanitary and antiepidemic measures and (or) the threat of spread infections.

Patients who have suffered from dysentery are discharged after clinical recovery. Discharge of dysentery patients belonging to epidemiologically significant contingents; children in preschool institutions and institutions with round-the-clock stay; persons in boarding schools is carried out after receiving a single negative result of a control bacteriological examination of bowel movements, conducted no earlier than a day after the withdrawal of antibiotics.

Medical supervision in the focus of dysentery is established in relation to the following contact persons:

- epidemiologically significant contingents (employees of the food industry, employees of water supply organizations, etc.);
- children attending kindergartens and schools;
- children from a children's home, children's boarding schools;
- persons staying in social service institutions providing inpatient social services;
- persons staying in sanatorium-resort and wellness organizations;
- patients of hospital organizations.

Medical supervision of the contact persons includes a survey for the presence of symptoms of the disease, a medical examination, laboratory examination, thermometry and is carried out within 7 days.

Disinsection measures aimed at combating flies and cockroaches are organized at epidemiologically significant facilities for preventive purposes.

In the home (apartment) focus of dysentery, the current and final disinfection is carried out by the patient himself or by persons caring for him using physical methods of disinfection, as well as the use of detergents and disinfectants.

Sanitary and hygienic measures are of great importance. The patient should be isolated in a separate room. The patient's room is wet cleaned and ventilated 2–3 times a day. Contact with children is excluded, the number of objects that the patient can come into contact with is limited, and personal hygiene rules are followed. The patient is provided with a separate bed, towels, care items, dishes for eating and drinking. Dishes and patient care items are stored separately from the dishes of family members. In summer, a systematic fly control is carried out.

## SALMONELLOSIS

Salmonellosis is an acute infectious disease of zoonotic nature characterized by predominant damage to the gastrointestinal tract, intoxication and water-electrolyte disorders. Disease is manifested by fever, symptoms of gastroenteritis, enterocolitis, in some cases — typhoid-like or septicopyemic course.

### EPIDEMIOLOGICAL FEATURES OF THE CAUSATIVE AGENT OF SALMONELLOSIS

The causative agents of salmonellosis belong to the bacteria of the genus *Salmonella* of the family Enterobacteriaceae. *Salmonella* have the appearance of small gram-negative rods 1–3 microns long, 0.5–0.8 microns wide, and have mobility due to the presence of flagella. Spores and capsules do not form (Fig. 8).

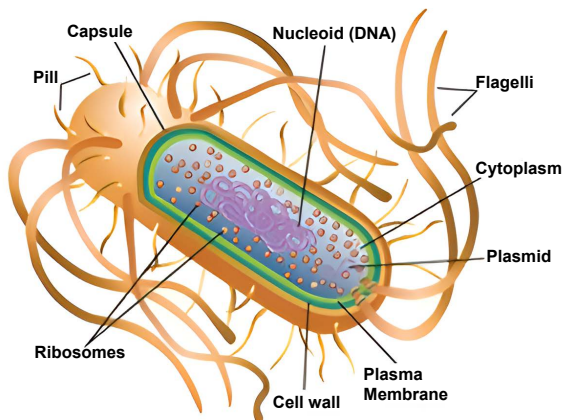


Fig. 8. The structure of the *Salmonella* bacteria

*Salmonella* is divided into 2 types: *S. enterica*; *S. bongori*. *S. enterica* serotypes can be divided into two main groups — typhoid and non-typhoid. Typhoid serotypes include *Salmonella Typhi* and *Salmonella Paratyphi A*, which are adapted to humans and are not found in other animals. Non-typhoid serotypes are more common and usually cause gastrointestinal diseases (salmonellosis). They can infect a range of animals, and are zoonotic, but can also be transmitted from person to person (Fig. 9).

The species *Salmonella enterica* includes 6 subspecies: *S. enterica*, *S. salama*, *S. arizona*, *S. diarizonae*, *S. houtenae*, *S. indica*, each of which has many serotypes. *Salmonella* species *S. bongori* are not pathogenic to humans. The division into

subspecies has a certain epidemiological significance, since warm-blooded animals serve as the natural reservoir of *S. enterica*, and for the rest of the subspecies — cold-blooded animals and the environment.

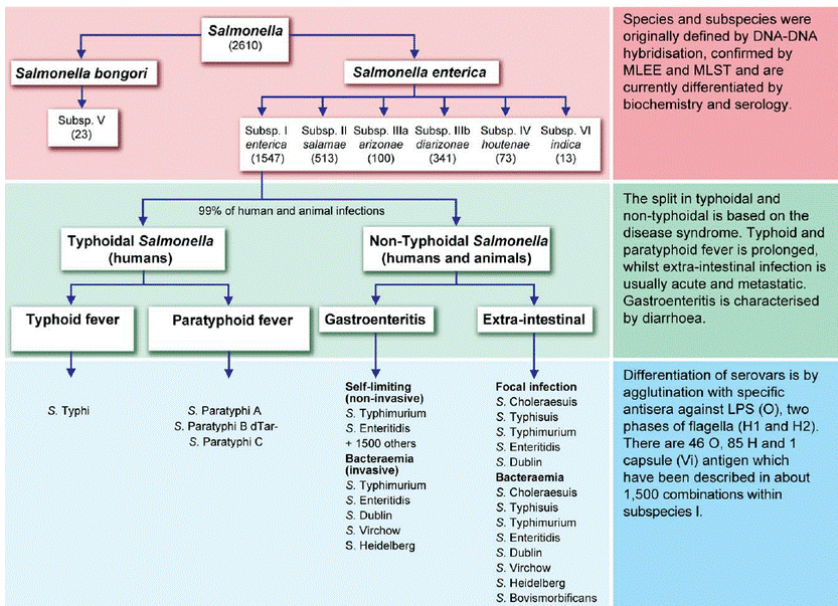


Fig. 9. General overview of the current classification of *S. enterica*

More than 95 % of all salmonella species belong to the subspecies *S. enterica*. *Salmonella* contains somatic thermostable O-antigen and flagellar thermolabile H-antigen. Based on differences in somatic and flagellar antigens, salmonella is divided into serological groups and serological variants (serovars). Currently, about 2,500 salmonella serovars have been identified worldwide. The spread of more than 120 salmonella serovars has been registered in the Republic of Belarus. Individual salmonella serovars are divided into phages.

Despite the wide typical composition of salmonella, the vast majority of diseases (up to 90 %) are caused by 10–12 serovars, which are defined as dominant — *S. typhimurium*, *S. enteritidis*, *S. infantis*, *S. newport*, *S. london*, *S. anatum*, *S. derby*, *S. oranienburg*, *S. choleraesuis*, *S. panama*.

The salmonella population is heterogeneous in antibiotic sensitivity. Antibiotic-resistant variants of salmonella can be formed both in medical institutions and in farm animal populations when antibiotics are used as prophylactic agents, growth factors, etc.

Salmonella is relatively resistant in the external environment and remains viable in water for up to 5–6 months, in frozen meat for 6 months, in egg powder for 3–9 months, in soil for up to 18 months. At a temperature of 70 °C, salmonella dies within 5–10 minutes. In the process of cooking eggs, salmonella remains viable for 4 minutes. Disinfectants in working concentrations have a detrimental effect on salmonella.

### **THE MECHANISM OF DEVELOPMENT OF THE EPIDEMIC PROCESS**

**The source of the infection.** In natural conditions, the main sources of infection for humans are cattle, pigs, sheep, chickens, ducks, geese, dogs, cats, rodents, etc. Salmonellosis in animals occurs in the form of clinically pronounced forms and carriage. The frequency of salmonella transmission in pets varies from 1–5 % to 25–50 %. The release of salmonella in animals continues throughout the entire period of acute disease (one to two weeks), carrier animals can secrete pathogens for several months.

With salmonellosis, a person can also act as a source of infection. The greatest danger of a person as a source of infection is for children of the first year of life. In some territories, in the structure of sources of salmonella infections, sick people account for up to 12 %. The duration of the infectious period in people with salmonellosis ranges from several days to 3 weeks. Convalescent carriage in humans can sometimes last up to 1 year.

**The transmission mechanism.** The localization of the pathogen in the intestine determines the fecal-oral transmission mechanism (Fig. 10). Human infection with salmonella occurs orally. The leading factors of salmonella transmission are food products of animal origin, primarily meat and meat products obtained from cattle. There are two possible ways to infect meat with salmonella: endogenous (during life) and exogenous (postmortem). The endogenous pathway is realized when receiving meat from animals with salmonellosis. Exogenously, meat becomes infected with salmonella when cutting carcasses on infected equipment, as well as at the stages of transportation, storage and sale.

In recent years, the importance of poultry meat (primarily chicken), eggs and egg products as factors of salmonella transmission has increased. Eggs are infected with salmonella endogenously (salmonella penetrates into the eggs laid during life) and exogenously (microorganisms penetrate through the shell contaminated with bird secretions). It has been experimentally established that with prolonged (more than 1 month) storage of chicken eggs in the refrigerator, *S. enterica* can penetrate into eggs through an intact shell and multiply in the yolk.

Salmonella multiplies well in food products. The rate of reproduction is most influenced by the temperature conditions and pH of the food product environment. In thermally processed minced meat and fish, boiled milk, the amount of salmonella reaches an infecting dose at a temperature of 37 °C after 4.5–6.5 hours, at a temperature of 20–22 °C — after 11.5–13.5 hours.

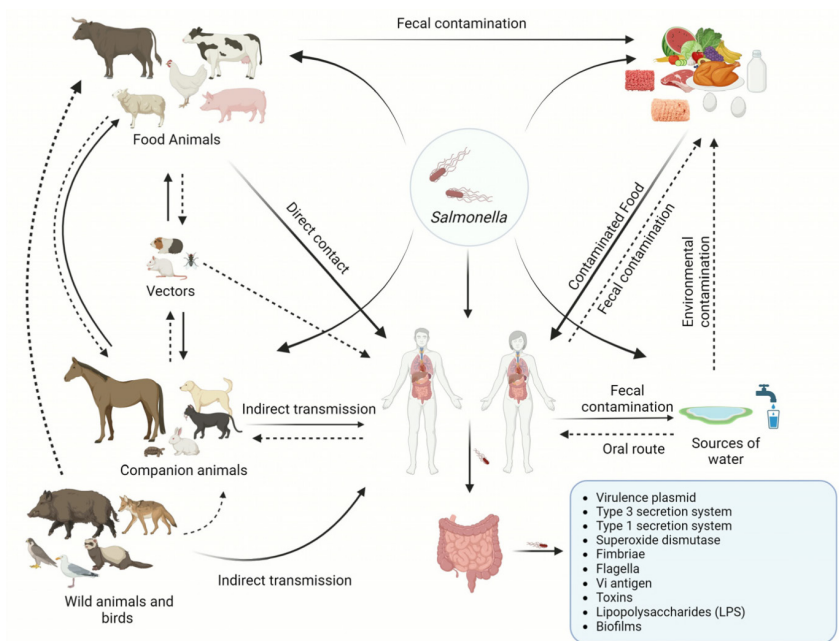


Fig. 10. Transmission cycle of *Salmonella* between animals and humans

*Salmonella* is found relatively often in the water of open reservoirs (8–28 % of the samples studied), but aquatic outbreaks of these infections are very rare. The small role of the water factor in the epidemiology of salmonellosis is explained by the fact that a significant part of *salmonella* dies very quickly in water, and the remaining viable microorganisms are not enough to cause diseases in humans.

In conditions of pediatric somatic hospitals, when the source of infection is a person, *salmonella* among children under the age of one year can be spread through household items. *Salmonella* is most often found in flushes from cots, bedside tables, baby feeding bottles, heating batteries, bedding.

**Susceptibility and immunity.** The outcome of *salmonella* infection largely depends on the dose of microorganisms, their serovars affiliation and other biological properties, as well as on the individual characteristics of the human body, immunity and other factors. The average infectious dose of *salmonella* is 106–107 microbial cells. Children of the first year of life and the elderly, weakened by chronic diseases, have the greatest sensitivity to *salmonella* infections.

The transferred disease of salmonellosis leaves behind an unstressed type-specific immunity lasting less than one year.

## **THE MAIN CLINICAL MANIFESTATIONS**

The incubation period ranges from 6 hours to 7 days, on average 2–3 days. The disease begins acutely. The body temperature rises to 39 °C and above, nausea appears, repeated vomiting, which sometimes becomes indomitable, there are pains in the epigastric region, headache. Then diarrhea joins these signs of the disease. The stool is frequent, watery, sometimes with an admixture of mucus, rarely blood. Seizures are possible.

In young children, the disease also develops acutely. Body temperature rises to 38–39 °C, appetite decreases, regurgitation and vomiting appear. The tongue is dry, overlaid. The stomach is swollen. The stool is frequent, thin, with dark green mucus. There are signs of intoxication and dehydration of the body. The disease sometimes drags on, proceeds with exacerbations (more often in weakened children).

Salmonella is isolated from sick people within 1–3 weeks, from convalescent carriers within 1–2 months. In some cases, people who have had salmonellosis develop chronic bacterial carriage and pathogens are allocated for more than one year.

## **LABORATORY DIAGNOSTICS**

Bacteriological and serological methods are used. The material for the study is feces, vomit, gastric lavage, urine, bile, and in generalized forms, the patient's blood.

Specific antibodies in the blood with salmonellosis usually appear by 6–8 days from the onset of the disease, then their titer increases. An agglutination reaction, an indirect hemagglutination reaction, and a complement binding reaction are used. Reactions are evaluated by increasing the titer of antibodies in dynamics.

## **MANIFESTATIONS OF THE EPIDEMIC PROCESS**

Salmonellosis is widespread and is 1 of the 4 key global causes of diarrhoeal diseases. Annually, around 200 million to 1 billion cases of Salmonella infections are recorded worldwide, with 93 million cases of gastroenteritis and 155,000 deaths; among them, approximately 85 % of the cases are associated with the consumption of contaminated food. The incidence of salmonellosis in the population of Belarus is mainly sporadic.

In recent years, there has been a trend towards an increase in morbidity in many countries. The increase in the spread of salmonellosis on a global scale is due to a number of reasons, the main of which are: intensification of livestock farming on an industrial basis; centralization of food production and the changed ways of their implementation; increase in the production of semi-finished products; urbanization processes; activation of migration processes; expansion of exports and imports of food and feed, etc.



Unlike most intestinal infections, salmonellosis is most widespread in large well-maintained cities, in countries characterized by a high level of economic development, which allows them to be classified as a group of diseases of civilization. In rural areas, outbreaks of salmonella infections often develop after family celebrations, for which many meat dishes were prepared in advance, stored without cold.

Cases of salmonellosis are recorded throughout the year, but their frequency increases in the summer and autumn period. This is due to the more favorable conditions that are created in the warm season for the reproduction of salmonella in food products.

All age groups of the population are involved in the epidemic process of salmonellosis. Risk groups include young children who are very sensitive to salmonella, as well as people who, by their occupation, are associated with the production, processing and sale of food products of animal origin. Weakened individuals and those suffering from digestive disorders are more susceptible to the disease than healthy people.

In the early 70s of the twentieth century, a new feature appeared in the epidemiology of salmonellosis — the nosocomial nature of the incidence. The vast majority of nosocomial outbreaks of salmonellosis were caused by salmonella from one serovar, *S.typhimurium*. Salmonella of this serovar isolated in hospital conditions were characterized by multiple antibiotic resistance. In some cases, nosocomial outbreaks were caused by *S.infantis*, *S.haifa*, *S.wien*. As a rule, nosocomial outbreaks of salmonellosis have been observed in large pediatric somatic hospitals. The epidemic process mainly involved children under the age of one who were hospitalized for pneumonia and other diseases of the respiratory system. Most outbreaks were observed during the cold season and were chronic. The sources of nosocomial salmonellosis were children hospitalized with unrecognized salmonellosis, medical workers and mothers admitted to child care who had mild salmonellosis. Salmonella was spread by the fecal-oral transmission mechanism, and various objects of the hospital environment acted as the main transmission factors.

## **PREVENTION**

Prevention of salmonellosis requires control measures at all stages of the food chain, from agricultural production, to processing, manufacturing and preparation of foods in both commercial establishments and at home.

The most important direction in the prevention of salmonellosis is veterinary and sanitary measures aimed at preventing diseases among farm animals and birds, especially in livestock and poultry complexes, as well as ensuring an appropriate sanitary and technological regime in slaughterhouses, meat processing and meat-dairy enterprises.



Continuous laboratory monitoring of the quality of bone and fish meal used for animal fattening should be carried out. Disinfection and deratization measures must be carried out at meat processing plants, food warehouses, and cold storage rooms. Wastewater from livestock complexes and meat processing enterprises is allowed to be discharged into reservoirs only after disinfection.

Preventive measures also include ensuring appropriate hygienic and technological conditions for the processing, storage, transportation and sale of food products at enterprises of the food industry, catering and food trade.

Special attention should be paid to meat products (minced meat), which are a favorable environment for the reproduction of salmonella. It is necessary that the temperature inside the meat piece reaches at least 80° when held for 10–15 minutes. It should be borne in mind that under favorable temperature conditions, salmonella multiplies faster on boiled products than on raw ones, therefore meat or fish previously subjected to heat treatment should not be cut on tables or boards on which raw products were processed.

The contact between infants/young children and pet animals that may be carriage Salmonella (such as cats, dogs, and turtles) needs careful supervision.

Prevention of nosocomial outbreaks of salmonellosis consists in preventing the introduction of salmonellosis into somatic hospitals and in preventing infection in a hospital setting. To prevent the introduction of salmonellosis into the hospital, medical workers who send sick children for hospitalization should indicate the presence (absence) of clinical symptoms characteristic of salmonella infections, as well as the presence of salmonellosis in the last 7 days at the place of residence or kindergarten. In the emergency room, regardless of the diagnosis indicated in the referral for hospitalization, the presence of clinical manifestations of salmonellosis should also be detected. If there are indications in the emergency room, a bacteriological examination of children admitted to the hospital can be carried out. It is advisable to place such children in diagnostic wards for the period until the diagnosis of salmonellosis is excluded.

National and regional surveillance systems on foodborne diseases are important means to know and follow the situation of these diseases and also to detect and respond to salmonellosis and other enteric infections in early stages, and thus to prevent them from further spreading.

### **ANTI-EPIDEMIC MEASURES**

The duration of sanitary and anti-epidemic measures in the focus of salmonellosis is established from the date of separation from the last person with salmonellosis (a person with suspected disease, carrier of pathogens) for a period of 7 days.

Patients with salmonellosis are subject to isolation at home or hospitalization in an infectious diseases hospital or an infectious diseases department of a hospital

organization. Hospitalization is carried out according to clinical and epidemic indications (as with dysentery).

Discharge from the hospital and admission to work (in organized groups) are carried out after clinical recovery and a negative result of bacteriological examination of bowel movements (in patients belonging to epidemiologically significant contingents). Persons from among the epidemiologically significant contingents who are carriers of salmonellosis pathogens are suspended from work for the duration of treatment and until laboratory confirmation of the cessation of isolation of the pathogen in accordance with labor legislation. Persons who have had salmonellosis are subject to medical supervision for one month if they belong to epidemiologically significant contingents or are members of certain organized groups.

Medical supervision of the contact persons is carried out for 7 days and includes examination for the presence of symptoms of the disease, medical examination, laboratory examination and thermometry.

In kindergarten, at school, in the group where a salmonellosis patient was identified, regime-restrictive measures are carried out during the observation period: separation from other groups; it is forbidden to transfer children from this group and accept new children; the self-service system in this group is canceled; the quarantine group does not participate in mass events held in premises, which are used in conjunction with other groups.

The current and final disinfection with the use of disinfectants is carried out in the focus of salmonellosis.

## **SITUATIONAL TASKS FOR PRACTICING PRACTICAL SKILLS**

### **TASK 1**

On December 29, a general practitioner examined patient K. and found icteric sclera and oral mucosa, enlarged and painful liver on palpation. The patient K. complained of weakness, nausea, vomiting and darkening of urine, the first symptoms appeared on December 26. Based on the clinical examination, the patient's complaints and epidemiological history, the doctor diagnosed Acute hepatitis A. The patient was admitted to an infectious diseases hospital on December 29.

The woman works as an assistant teacher in the middle kindergarten group. The kindergarten is designed for 8 groups. The principle of group isolation in the institution is respected. In the middle group of the kindergarten where the patient works, on November 20 and December 10, 2 cases of acute hepatitis A were registered in two children. It is known that both children had clinical signs of

hepatitis A in the morning, but they were removed from the group only after lunch. No cases of acute hepatitis A have been established in the remaining groups. The last time she was ill at work was on December 26. She lives in a comfortable two-room apartment with her husband. My husband works as a mechanic in an auto repair shop.

*1. Indicate the possible source of infection, pathways and transmission factors.*

*2. Draw up a plan of sanitary and anti-epidemic measures in an epidemic outbreak.*

*3. To characterize the manifestations of the epidemic process of hepatitis A in the Republic of Belarus.*

## **TASK 2**

On October 21, the epidemiological department of the district Center for Hygiene and Epidemiology received an emergency notification about a case of acute hepatitis A in a 3-year-old child, a pupil of the Ryabinka Nursery School. During the epidemiological examination, the epidemiologist found that the sick child had an elevated body temperature of 38.0 °C on October 20 during a morning examination by an assistant teacher. On October 21, a local pediatrician discovered mild jaundice of the sclera during an examination and made a preliminary diagnosis of “Acute hepatitis?”. The child was admitted to the infectious diseases hospital on the same day, where, after a laboratory examination, the final diagnosis was made “Acute hepatitis A, jaundice form.”

The child lives with his parents and older sister in a separate apartment. His father, 32 years old, is an engineer at an industrial enterprise, his mother, 30 years old, is a bank employee, is a donor, his sister, 5 years old, attends a preschool educational institution. In a preschool where a sick child has been identified, groups of children are completely isolated. There are 18 children in the younger group, which the patient attended, according to the list. 16 children have been vaccinated against viral hepatitis A, two children have not been vaccinated against this infection. One unvaccinated child was absent from the preschool on the day of the epidemiological examination for an unknown reason. Since the beginning of September, the city has seen an increase in the incidence of hepatitis A.

*1. Indicate the possible source of infection, pathways and transmission factors.*

*2. Draw up a plan of sanitary and anti-epidemic measures in an epidemic outbreak.*

*3. To give an epidemiological characterization of the sources of infection in viral hepatitis A.*

### TASK 3

When examining the patient at home on September 15, the district doctor, based on the clinical picture, diagnosed “Acute viral hepatitis, unspecified” and gave a referral for hospitalization. On the same day, an emergency notification was sent to the CGE with a diagnosis of “Acute viral hepatitis, unspecified.”

When collecting an epidemiological history, it was found that the patient A., a 33-year-old man, works as a locksmith at a machine-building plant, traveled to Uzbekistan from August 12 to 23.

Family composition: the wife is a kindergarten teacher, the child is 5 years old and attends a kindergarten group. The child has been vaccinated twice against hepatitis A.

No similar diseases were detected among family members at the place of residence. In the team at the place of work (workshop of a machine-building enterprise), such diseases have not been registered over the past 6 months.

*1. What laboratory research methods can confirm the diagnosis of acute hepatitis A?*

*2. List the activities that need to be organized by the CGE epidemiologist upon receipt of an emergency notification in a similar situation.*

*3. In what cases and at what time is the examination of residential foci of viral hepatitis A carried out?*

### TASK 4

In a family consisting of two adults and three children, the youngest child of 4 years old fell ill with rotavirus infection on January 18. During the epidemiological examination, it was found that the mother of the patient works as a primary school teacher, the father is an engineer at a machine-building plant, the sister is a first-grade student for 7 years, the brother has been attending kindergarten for 5 years. The family lives in a 2-room comfortable apartment.

*1. Indicate the possible source of infection, pathways and transmission factors.*

*2. Determine the boundaries of the epidemic outbreak.*

*3. Draw up a plan of sanitary and anti-epidemic measures in an epidemic outbreak.*

### TASK 5

In the city of N. over the past three years, there has been an increase in the incidence of rotavirus infection. The main contingent that is involved in the epidemic process are children under the age of 2 years. Among the children with rotavirus infection, the majority were children who often and long-term suffer from acute respiratory infections. Two years ago, the regional Center for Hygiene and

Epidemiology developed a plan for the prevention of intestinal infections. To date, most of the activities included in the plan have been completed, but this has not led to a decrease in the incidence of rotavirus infection.

*1. Formulate a hypothesis that can answer the question: "If, despite the ongoing preventive measures, the incidence of rotavirus infection does not decrease, then in what type of activity have the measures proved insufficient?"*

*2. Formulate a hypothesis that can answer the question: "If, despite the ongoing preventive measures, the incidence of rotavirus infection does not decrease, then in what kind of activity are mistakes made?"*

*3. Make suggestions on improving preventive measures.*

## **TASK 6**

The epidemiological effectiveness of the rotavirus vaccine was studied in the city of Moscow with a population of 210,000 people. In September, 1,920 children aged 1–2 years who had no contraindications to vaccination and had not previously had rotavirus infection were vaccinated against rotavirus infection. The control group consisted of 2,312 children aged 1–2 years who also had not previously had rotavirus infection and had not received a vaccine against rotavirus infection as part of the study of the effectiveness of vaccination. The follow-up period for both groups was one year. During the follow-up period, 3 cases of rotavirus infection were registered among children vaccinated with rotavirus vaccine. 45 cases of rotavirus infection were detected in the control group.

*1. Calculate the coefficient of effectiveness of vaccination against rotavirus infection.*

*2. Specify and explain the typical seasonal manifestations of rotavirus infection.*

*3. To characterize a group of infections, the pathogens of which are transmitted by the fecal-oral transmission mechanism, from the standpoint of the theory of the transmission mechanism.*

## **TASK 7**

On May 11, when visiting patient N. at the age of 25, the polyclinic therapist made a preliminary diagnosis of «Diarrhea and gastroenteritis of presumably infectious etiology». Anamnesis data: fell ill on May 10, when cramping abdominal pains appeared, loose stools 12 times a day and the temperature rose to 38.0 °C. The patient suffers from diabetes mellitus.

During the survey, it was found that at the place of work in the bank's office, the patient communicated with an employee who had similar clinical manifestations of the disease last week.

After hospitalization of the patient and laboratory diagnosis, the final diagnosis of «Shigellosis caused by *S.flexneri*» was made. The patient lives in a comfortable apartment with his wife, a 4-year-old child and his father. The wife of the patient is a dairy worker, the father is a truck driver, a 4-year-old child attends a kindergarten group.

*1. Indicate the possible source of infection, ways and factors of transmission of infection.*

*2. Give the epidemiological characteristic of the causative agents of dysentery.*

*3. Make a plan of anti-epidemic measures in the epidemic focus.*

## **TASK 8**

On August 15, patient M. went to see a polyclinic doctor complaining of cramping abdominal pain, stool with mucus 10–12 times a day. Ill for the 2nd day. The disease began with an increase in temperature to 38.5 °C, by the end of day 1, vomiting and diarrhea appeared. The doctor diagnosed «Bacterial food poisoning, unspecified». It became known from the epidemiological history that the patient was outside the Republic of Belarus a week before the first symptoms of the disease appeared. He lived in the village of Akyn in the Almaty region of Kazakhstan in a private house with relatives. There was no centralized water supply and sewerage in the house. The residents of the house took water for drinking and household needs from the mine well, which is flooded during floods. Over the past 3 years, cases of Flexner dysentery have been reported in this area in the summer and autumn period among all age groups of the population.

The patient lives in a comfortable three-room apartment. The wife of citizen M. works as a barmaid at a large industrial enterprise, a 2-year-old child attends kindergarten No. 25.

*1. Specify the possible source of infection, pathways and transmission factors.*

*2. Determine the boundaries of the epidemic focus.*

*3. Make a plan of anti-epidemic measures in the epidemic focus.*

## **TASK 9**

A 42-year-old woman P. on March 25, on the third day of her illness, went to the local doctor complaining of deterioration in her well-being. The disease began with chills, severe general weakness, an increase in body temperature to 38.9 °C, headache, cramping abdominal pain, nausea, vomiting, and then a liquid copious watery stool joined. On the 2nd and 3rd days of the disease, vomiting did not recur, the stool became liquid with streaks of mucus and blood. The frequency of stool is 15–20 times a day. It is known from the epidemiological history that on the eve

of the disease, the patient consumed homemade sour cream, which she purchased at the local market. The patient was given a preliminary diagnosis of «unspecified shigellosis».

The patient works as a teacher at the state educational Institution, the last time she went to work is March 23. The woman lives in a comfortable two-room apartment. Family members: husband, 45 years old, chef of the restaurant «Summer Evening», son, 17 years old, student of the professional construction lyceum No. 3.

*1. Indicate the possible source of infection, pathways and transmission factors.*

*2. Make a plan of anti-epidemic measures in the epidemic focus.*

*3. List the measures for the prevention of dysentery.*

## **TASK 10**

The preschool educational institution, designed for 240 places, is located in a separate two-storey building. Water supply and sewerage are centralized, children of all groups receive food from one food hall. There are 10 isolated groups in the institution. Groups of 20–25 people.

In the period from October 11 to October 14, 11 children of the younger group No. 1 fell ill with an acute intestinal infection. The disease in most children began acutely, with headaches, abdominal pain, frequent loose stools with mucus. All the children were admitted to an infectious diseases hospital, where they were diagnosed with salmonella enteritis, and *Salmonella enteritidis* was isolated.

Children and staff who communicated with patients in the group underwent laboratory examination, two children were found to have *Salmonella enteritidis*. In the period from 8.10 to 14.10, three children diagnosed with acute laryngotracheitis were absent in group No. 1, two more children were transferred from group No. 1 to group No. 2 on October 9 (the reason for the transfer is that the number of children in group No. 1 is significantly higher than in group No. 2).

*1. Identify the conditions that contributed to the outbreak of salmonellosis.*

*2. Evaluate the possibility of occurrence of cases of salmonellosis in children in other groups of this kindergarten.*

*3. Determine the boundaries of the outbreak and make a plan of anti-epidemic measures in the epidemic focus.*

## **TASK 11**

Patient N., 24 years old, a student of the pedagogical University, living in a dormitory, sought medical help on January 25 (on the third day of the disease) with complaints of deteriorating health. She became acutely ill, chills, general weakness, headache, dizziness, muscle aches and joint pain, and an increase

in body temperature to 38.4 °C. After 4–5 hours, nausea and repeated profuse vomiting joined. The stool was initially decorated, and then became liquid and abundant with a fetid odor, foamy, dark green in color with an admixture of mucus, up to 12–14 times a day. Body temperature all days was 39–39.7 °C.

Epidemiological history: 12–14 hours before the disease, she ate fried potatoes and a hot dog bought at a fast food station. The diagnosis of «Salmonella gastroenteritis» is suspected.

Together with the patient, his wife of 23 years, a student of the pedagogical university, a child of 2 years old, attends kindergarten, lives in the room.

- 1. Indicate the possible source of infection, pathways and transmission factors.*
- 2. Make a plan of anti-epidemic measures in the epidemic focus.*
- 3. List the main measures for the prevention of salmonellosis.*

## **TASK 12**

On January 28, the doctor was called to patient A., 37 years old. The patient complained of cramping abdominal pain, nausea, and vomiting, loose stools. He became ill the night before, when chills, weakness, abdominal pain, nausea appeared. Vomiting appeared at night, accompanied by loose stools, up to 10 times a night, temperature 38.5 °C. The day before, the patient and his wife were visiting friends, where they ate a salad of chicken and vegetables. The wife also complained of malaise and upset stool. The stool is abundant, fetid, dark green in color. A preliminary diagnosis was made — Salmonella gastroenteritis.

The patient lives in a three-room comfortable apartment. Family composition: wife, 36 years old, works as a kindergarten teacher, daughter, 5 years old, attends kindergarten, grandmother, 62 years old, retired, and does not work.

- 1. Specify the possible source of infection, pathways and transmission factors.*
- 2. Determine the boundaries of the outbreak site and draw up a plan of anti-epidemic measures in the epidemic focus.*
- 3. Give the epidemiological characteristic of the causative agents of salmonellosis.*



## LITERATURE

### *Basic*

1. *Mamchits, L. P.* Epidemiology / L. P. Mamchits. – Minsk : High School, 2021. – 191 p.

### *Additional*

2. *Shylava, M. A.* Basics of general epidemiology. Anti-epidemic measures and means. Epidemic control measures in health care organizations / M. A. Shylava, G. N. Chistenko. – Minsk : BSMU, 2018. – 28 p.

3. *Fletcher, S.* Clinical Epidemiology : The Essentials / S. Fletcher. – 6th edition, Kindle Edition Grant. – Wolters Kluwer Health, 2019. – 288 p.

4. *Shylava, M. A.* Basics of infectious diseases immunoprophylaxis / M. A. Shylava, G. N. Chistenko. – Minsk : BSMU, 2020. – 60 p.

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