
Patient name:
Weight:
Diagnosis
SHIGELLOSIS

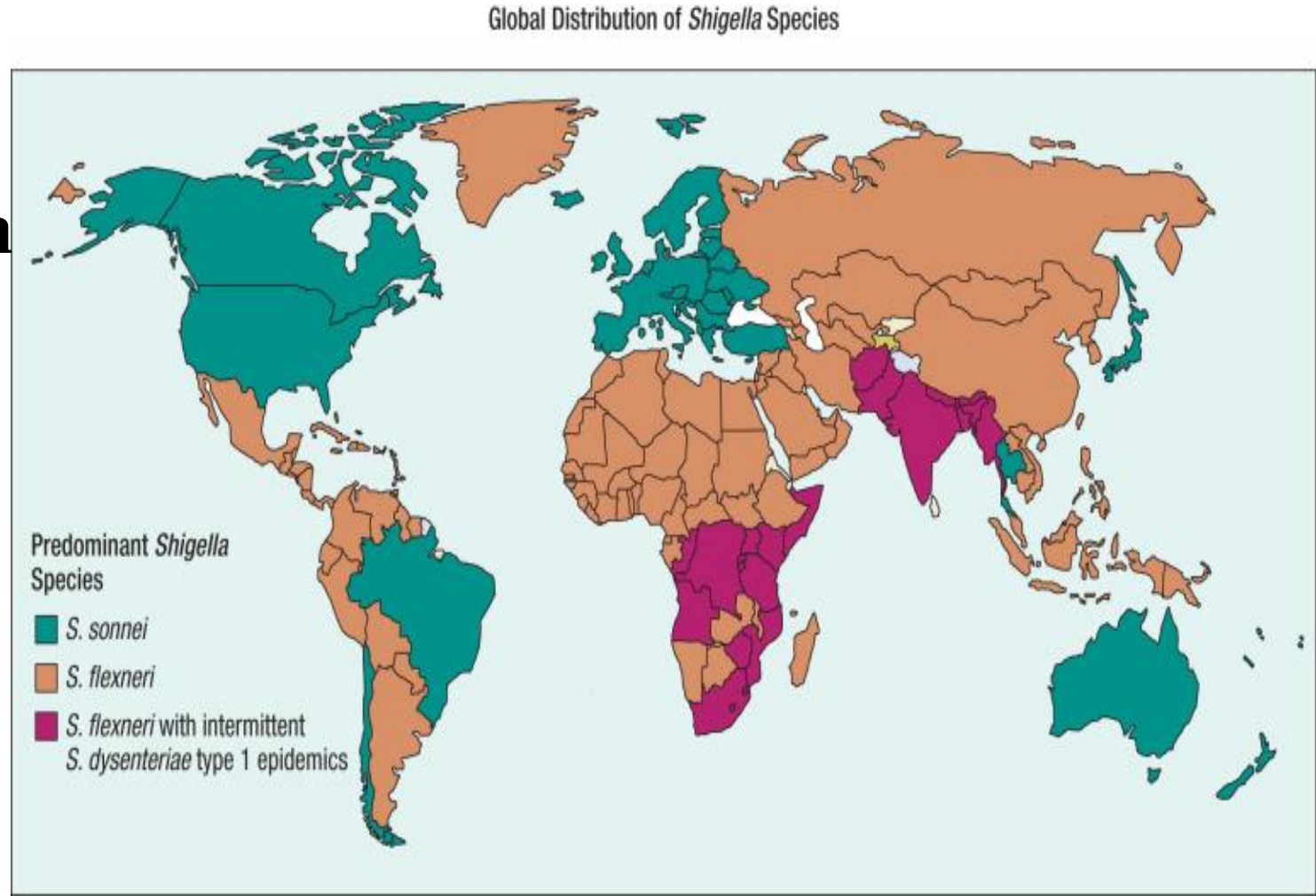
Shigellosis -

anthroponotic acute infectious disease with fecal-oral mechanism of transmission. Characterized by

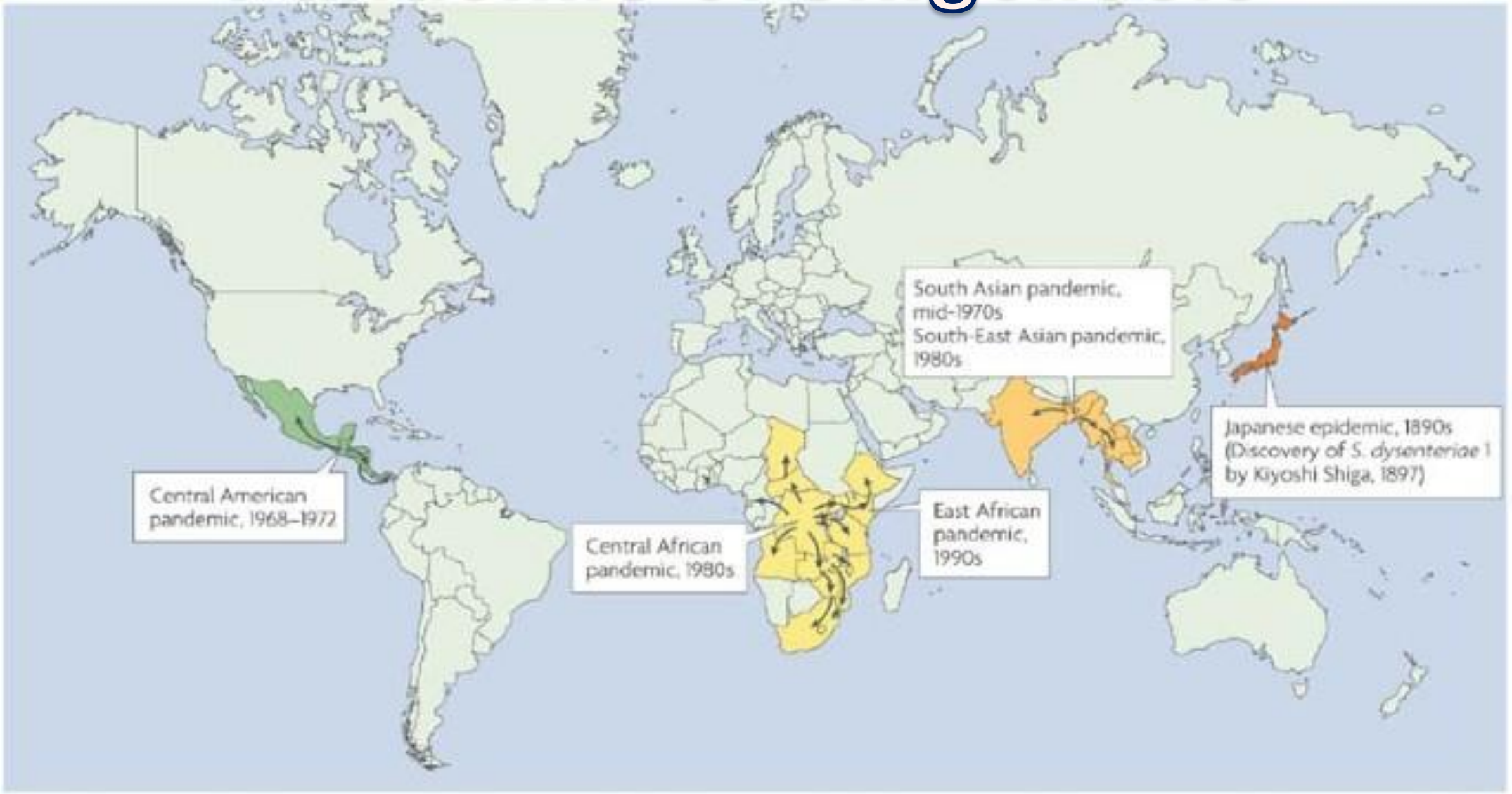
- **general intoxication and preferential mucosal lesion of the distal colon,**
- **cramping abdominal pain,**
- **loose stools with mucus and blood,**
- **tenesmus.**

Shigella **is one of the leading bacterial causes of diarrhea worldwide, causing an estimated 80–165 million cases. The number of deaths it causes each year is estimated at between 74,000 and 600,000.**

It is one of the top four pathogens that cause moderate-to-severe diarrhea in African and South Asian children.



Pandemic of shigellosis



The Discovery and Naming of *Shigella*

The several types of *Shigella* bacteria have been named after the lead workers who discovered each one.
The first bacterium to be discovered, *Shigella dysentariae*, was named after Kiyoshi Shiga, a Japanese scientist who discovered it in 1896 while investigating a large epidemic of dysentery in Japan.

In subsequent years, other microbes that cause dysentery were discovered:

1906 — Flexner,

1915 - Sonne,

1916 - Stuttzer-Schmidt,

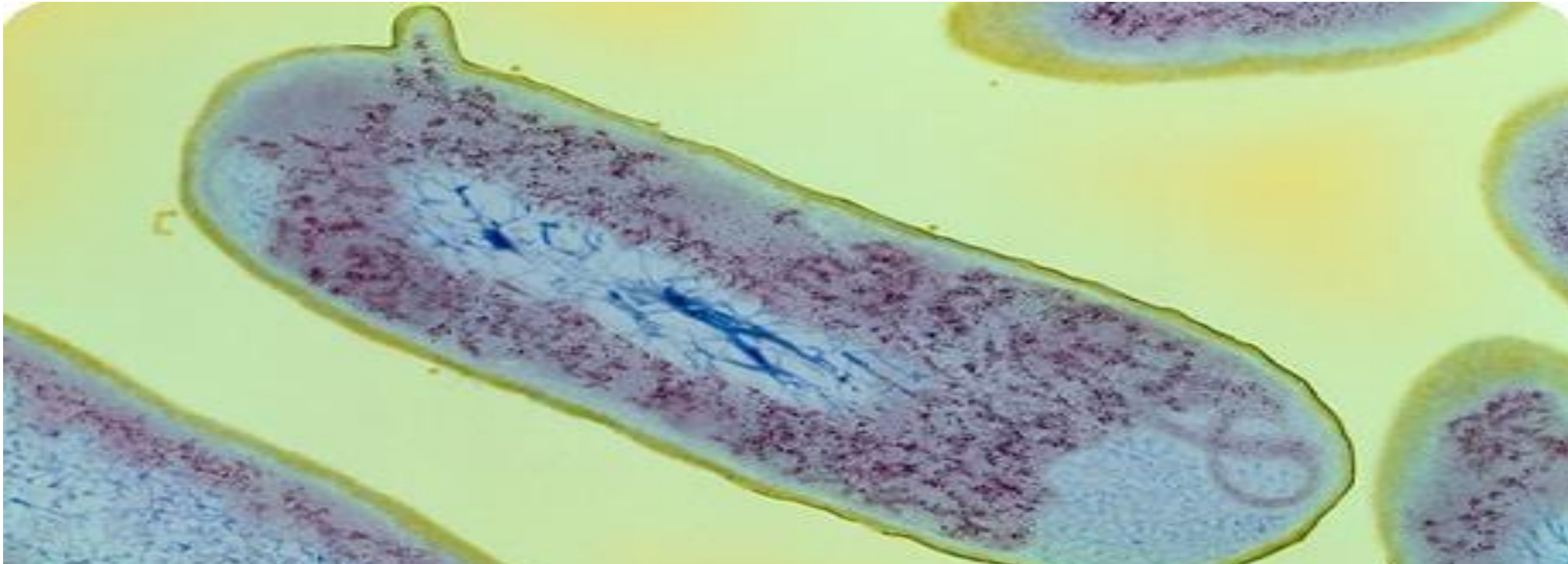
1929 - Newcastle,

1936 - Boyd,

1943 - Large-Sachs bacteria

Etiology - bacteria of the genus Shigella still family Enterobacteriaceae.

Currently, there are over 50 varieties of serological dysentery bacteria. They do not differ morphologically



Etiology

SHIGELLA

- they look like sticks in size (0.3-0.6) x (1.0-3.0) microns with rounded ends.
- Gram-negative
- Non motile
- spores and capsules do not form
- hemoorganotrofy
- oxidase negative
- catalase positive
- grow well on ordinary nutrient media
- facultative anaerobes

Antigenic structure.

All Shigella contain thermostable somatic O antigen which include group and type antigens.

According to the International Classification, Shigella is divided into four species, denoted by Latin capital letters

A, B, C, D.

.

According to the current classification (1980), *there are 4 major groups of* Shigella

A	B	C	D
S.dysenteriae, 15 serotypes (1st serotype produces Shiga toxin))	S.flexneri, 19 serotypes	S. boydii, 20 serotypes	S. sonnei. 1 serotypes

Every group is divided into serologic types and subtypes.
There are about 50 serovars Shigella

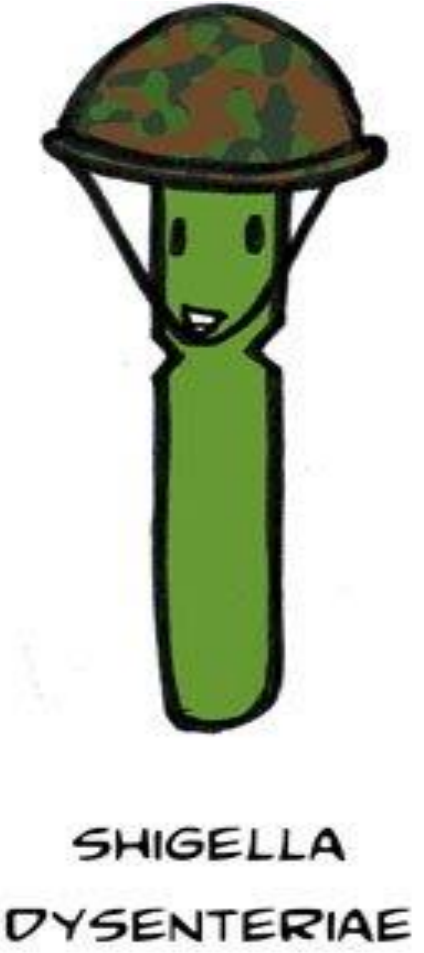
S. dysenteriae distinguish among 15 independent serotypes, including *Grigoriev-Shigi* (S.dysenteriae 1), *Schmitz-p* connection (S. dysenteriae 2) and *Lardzha-Sachs* disease (S. dysenteriae 3-7).

S.flexneri including *Newcastle* (S.flexneri 6).

S. boydii includes 20 serovar

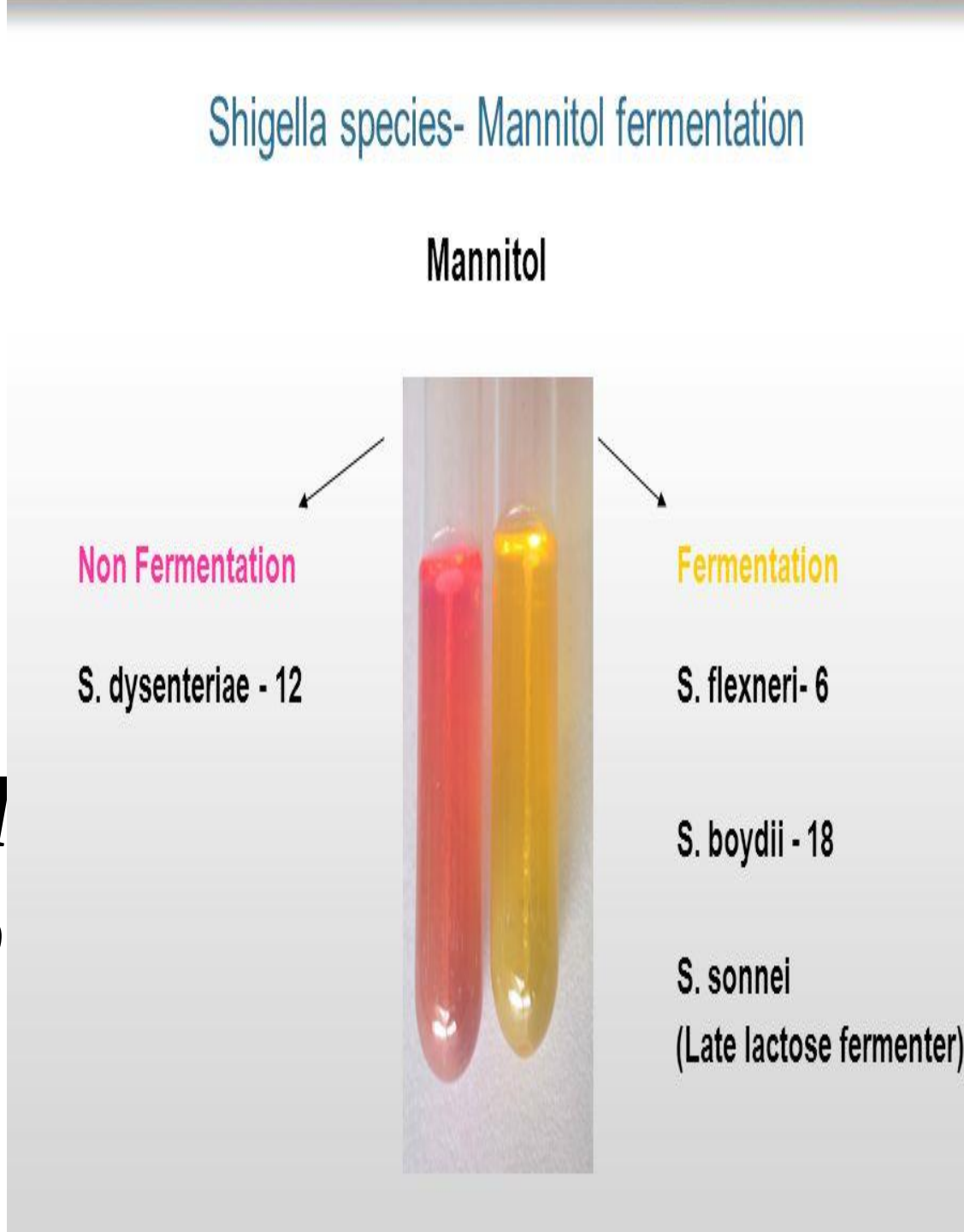
S. sonnei serologically not differentiate.

Groups *A–C* are physiologically similar;
***S. sonnei* (group *D*) can be differentiated on the basis of biochemical metabolism assays.**
Three *Shigella* groups are the major disease-causing species: *S. flexneri* is the most frequently isolated species worldwide, and accounts for 60% of cases in the developing world; *S. sonnei* causes 77% of cases in the developed world, compared to only 15% of cases in the developing world; and *S. dysenteriae* is usually the cause of epidemics of dysentery, particularly in confined populations such as refugee camps.
Each of the *Shigella* genomes includes a virulence plasmid that encodes conserved primary virulence determinants.



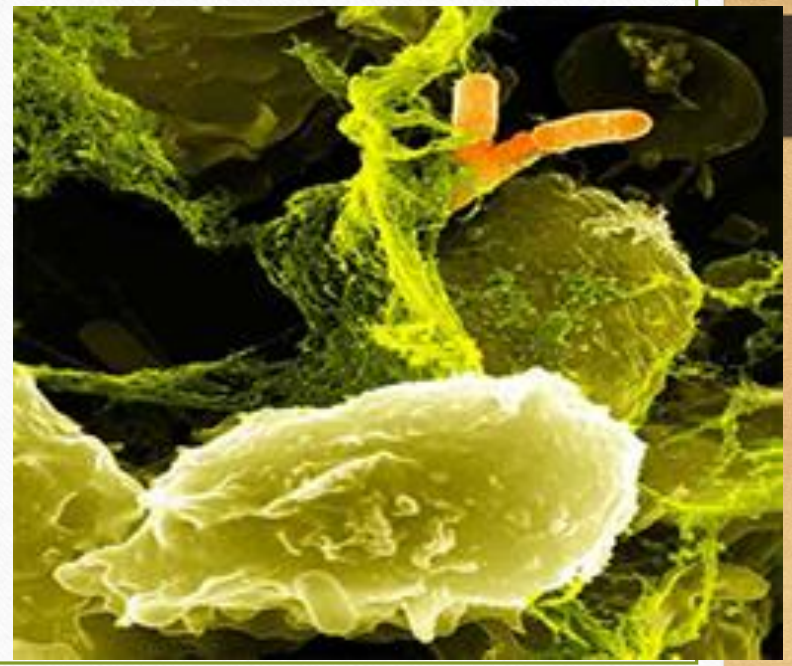
These subgroups and serotypes are differentiated from one another by their biochemical traits (ability to ferment D-mannitol) and antigenic properties.

The most recently recognized serotype belongs to subgroup C (S. boydii).



The causative agent of bacillary dysentery distinguished by **enzymatic activity, pathogenicity and virulence.**

All Shigella grow well in differential diagnostic media; optimum temperature 37°C, the bacteria sonnei can multiply at 10-15°C.



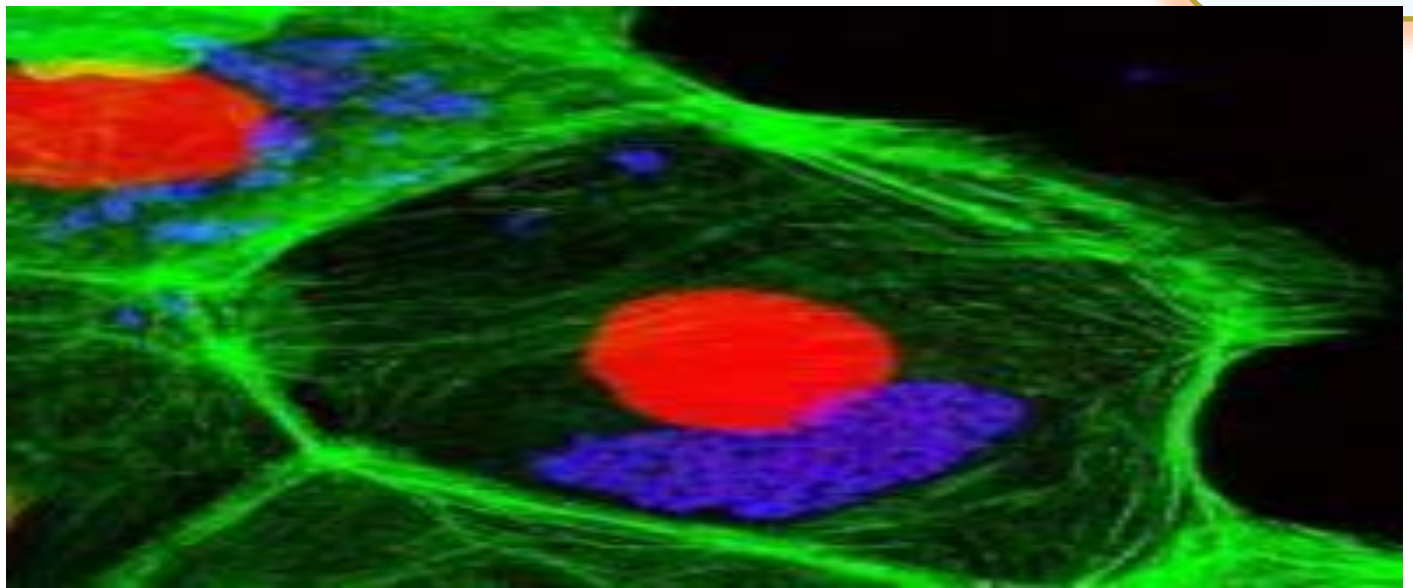
Virulence.

The virulence of Shigella is determined by three main factors -

the ability to adhere to epithelial cell membranes,

are invasive

and toxin production

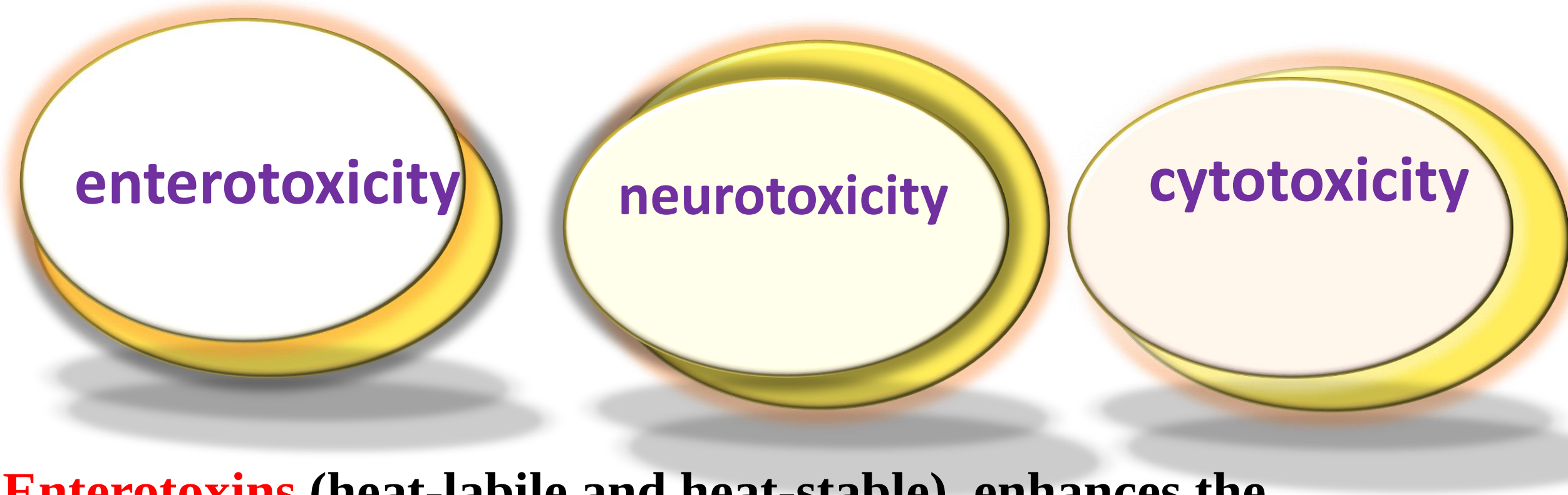


Toxin

With the destruction of the antigen is released endotoxin, which is largely due to the development of intoxication syndrome.

Shigella are capable of producing exotoxins.

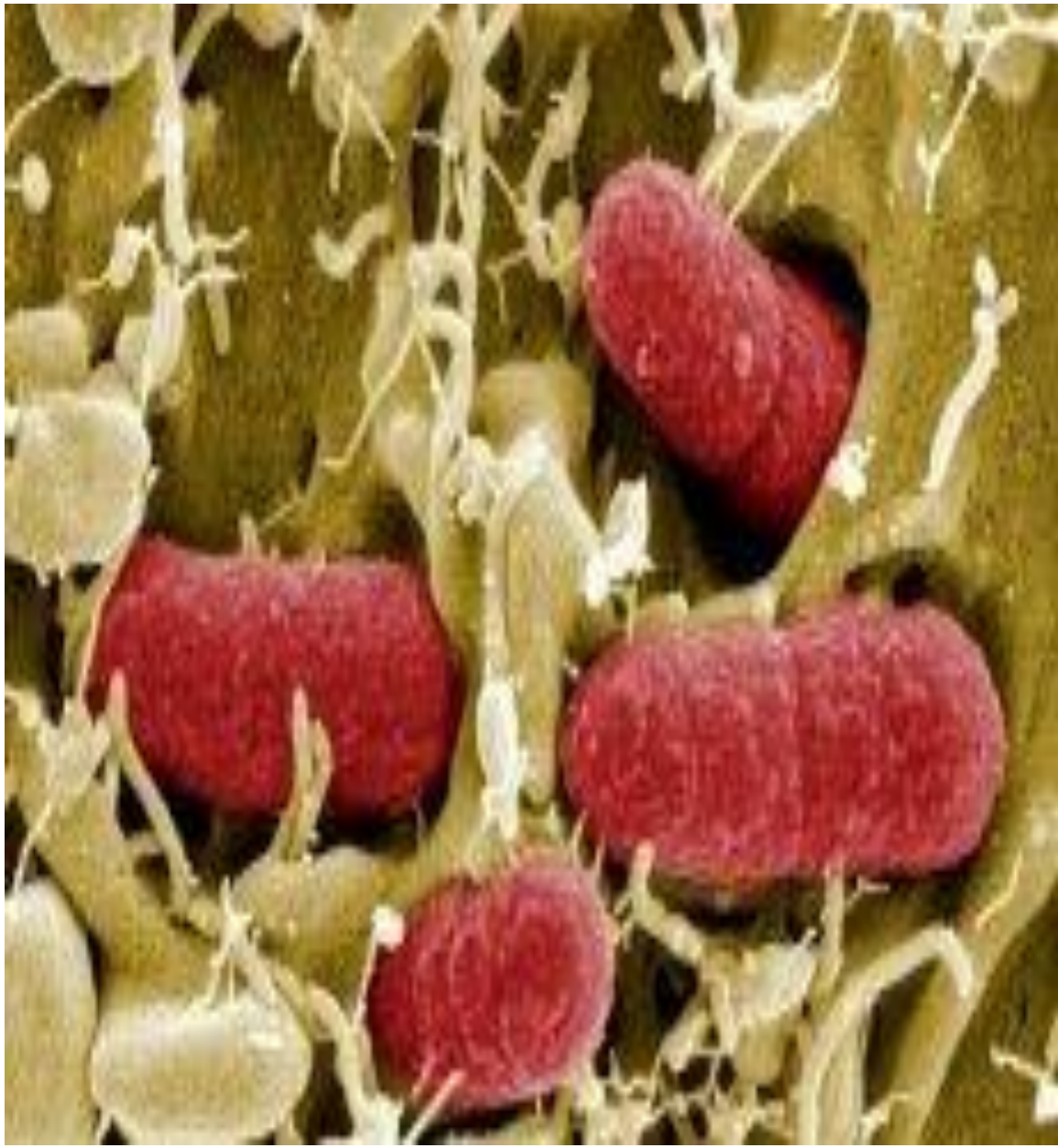
All kinds of Shigella produce endotoxin, which has 3 types of activity:



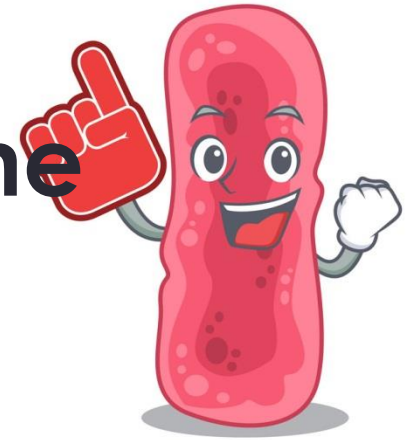
Enterotoxins (heat-labile and heat-stable), enhances the secretion of fluids and salts in the bowel lumen,
Cytotoxin damaging membranes of epithelial cells.
Bacteria Grigorieva - Shigi produce a potent neurotoxin.

Pathogenicity.

Different types of Shigella are characterized by unequal pathogenicity.
It is extremely high in Shigella Grigorieva - Shigi.
Pathogenicity of other species of bacteria is much lower dysentery.



The lack of virulence in Shigella Sonnei fully compensates for their high biochemical activity and the rate of reproduction in the infected substrate.
It takes 8 to 24 hours for a dose of S. sonnei to accumulate in milk at room temperature.
In the hot season, these periods are minimal: it takes only 1-3 hours for the bacteria dose to accumulate.



Shigella quickly dies when heated: at 60 ° C - 10 minutes, during boiling - instantly.
S.flexneri is the least stable.
In recent years, often isolated thermotolerant (capable of surviving at 59°C) strains of Shigella Sonne and Flexner.
Disinfectants in the usual concentrations are detrimental to Shigella.

The stability in the external environment.

Depending on temperature, humidity, pH, type of dysentery bacteria, the lifespan of microorganisms varies from several days to months. Food is a favorable environment for bacteria. Shigella sonnei in milk and fermented milk products are not only able to exist for a long time, but also multiply.

Disinfectants (hypochlorites, chloramines, lysol, etc.) in normal concentrations kill dysentery bacteria within a few minutes.

Dysentery is prevalent throughout the year

The incidence recorded in July – September is usually half of all cases of the disease per year.

Immunity.

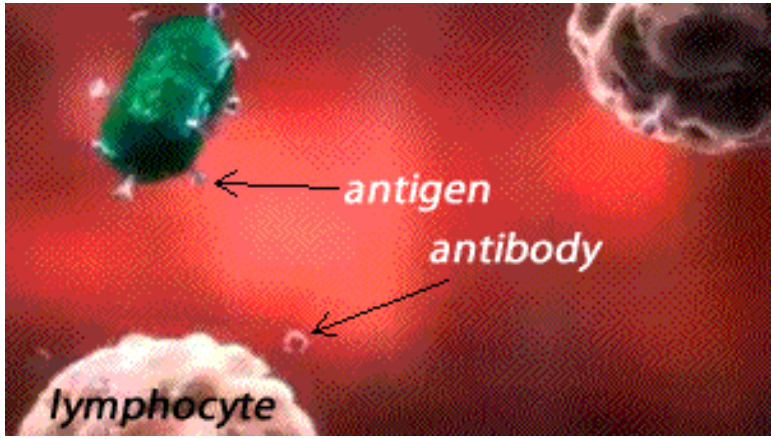
The natural susceptibility of people is high.

Immunity after the disease is *specific* and *antimicrobial*.

During the infectious process, the amount of antibodies to agglutinin, precipitin and hemoglobin in the blood increases.

However, the titer of specific antibodies does not increase and decreases in a short time, almost completely disappearing 5-12 months after the onset of the disease.

Only Sh. Flexner's immunity can last for several years.



Epidemiology

The source of infection are patients of acute or chronic dysentery, convalescents and those with subclinical infection (bacterial excretion).

The greatest epidemiological risk are patients with acute dysentery, releasing at the peak of the disease in the environment a huge number of pathogens.

For infection and development of the disease are need not less than 100 microbial Shigella cells.

Epidemiology

Reservoir and source of infection is the person (patient acute or chronic form of dysentery).

The most dangerous are those with mild and erased form of the of dysentery, especially persons of certain professions (working in the food industry and related persons).

The duration of excretion is from 7 to 10 days, plus the recovery period (on average 2-3 weeks). Sometimes bacteria shedding takes several weeks or months. The tendency to chronic infection is most characteristic of Flexner's dysentery, the smallest is Sonne's dysentery.

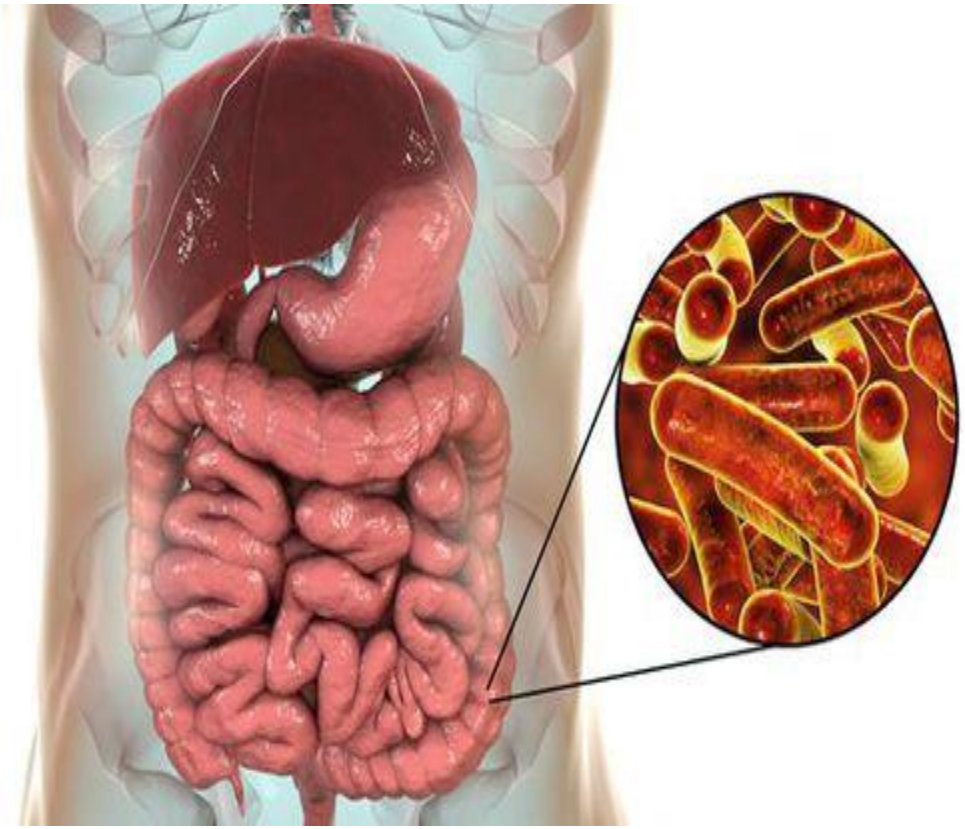
In the acute phase of the disease, each gram of feces contains an average of 10^7-10^8 microbial bodies.

The highest contagion belongs to the A-subgroup of Shigellosis (minimum infectious dose – 10 microbial bodies in 1 g of biomaterial)

and Sh.Fleksner (min. dose - 100 microbial bodies in 1 g of biomaterial).

In Sh. Zonne, the infectious dose is 10^7-10^8 microbial bodies in 1 g of biomaterial.

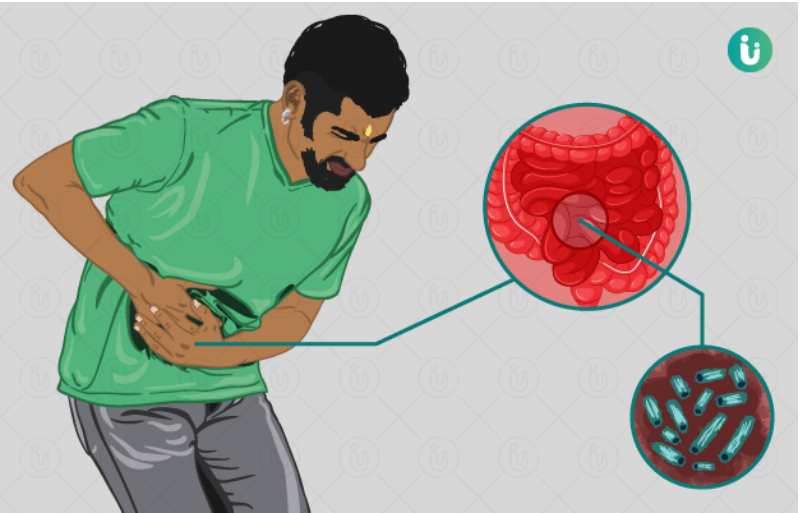
The end of the infectious period of patients can only be confirmed by laboratory microbiological examination of feces.
In 80% of patients, the excretion of bacteria stops 7-15 days after the onset of the disease,
and only in a small percent of cases, the excretion of Shigella continues for 30 days or more.

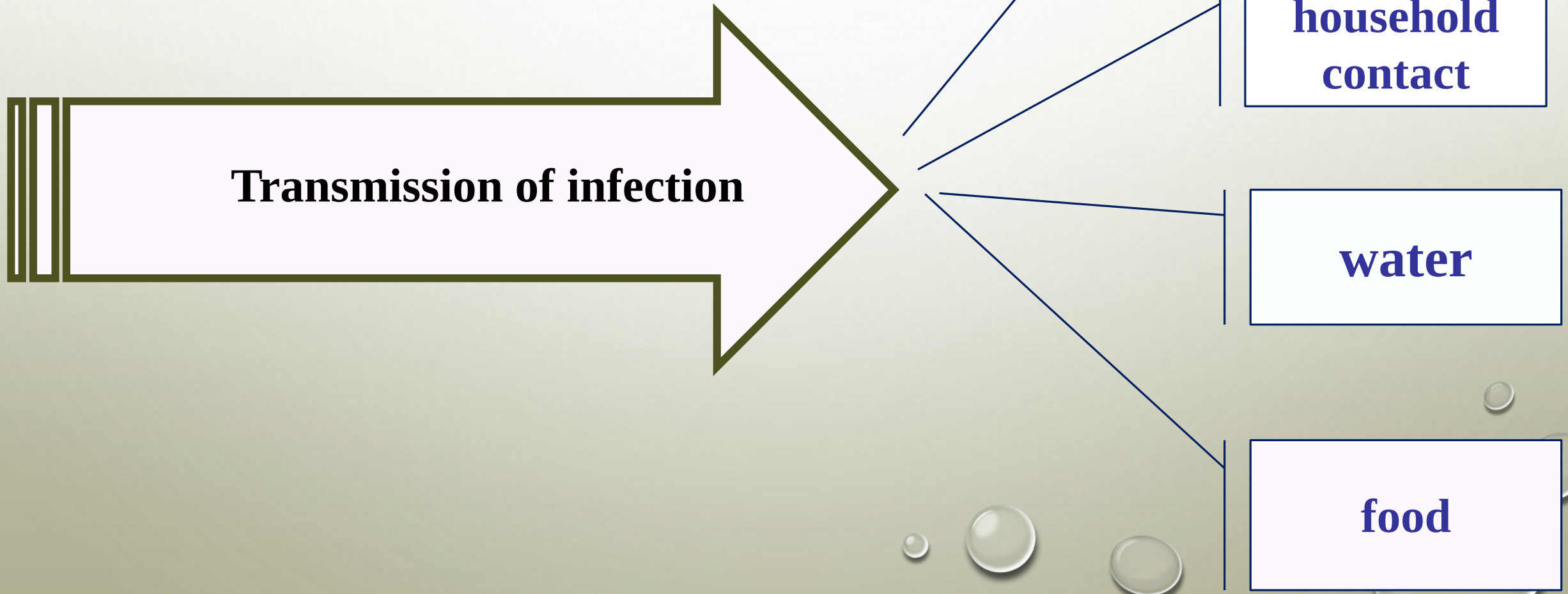


Patients with

- an erased,
- subclinical,
- inapparatus form of the disease,
- convalescents,
- as well as patients with prolonged and
- chronic dysentery

play an important role in the spread of infection.




Transmission of infection
fecal-oral
household contact
water
food

Infection through household contact is most common among preschool children (servants' coats and clothes, dishes, furniture, toys, water taps, door handles, etc.).

Infection through food are more common in adults.

With the contact-household route, the disease is often sporadic and, in rare cases, occurs in groups.

Waterborne infections take the form of epidemics. It occurs mainly when using dirty pools or using contaminated water.

The mechanism of transmission -

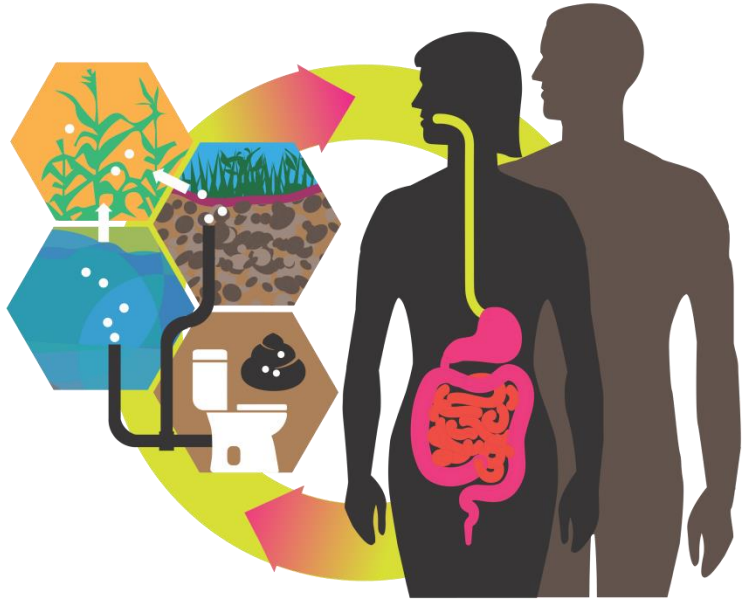
the fecal-oral transmission routes -
- **water,**
- **food and**
- **contact-household.**

In *dysentery Grigorieva-Shigi* the main route of transmission is contact-household, providing the transmission of highly virulent pathogens.

When *Flexner dysentery* main route of transmission is - water,

***dysentery Sonne* - food.**

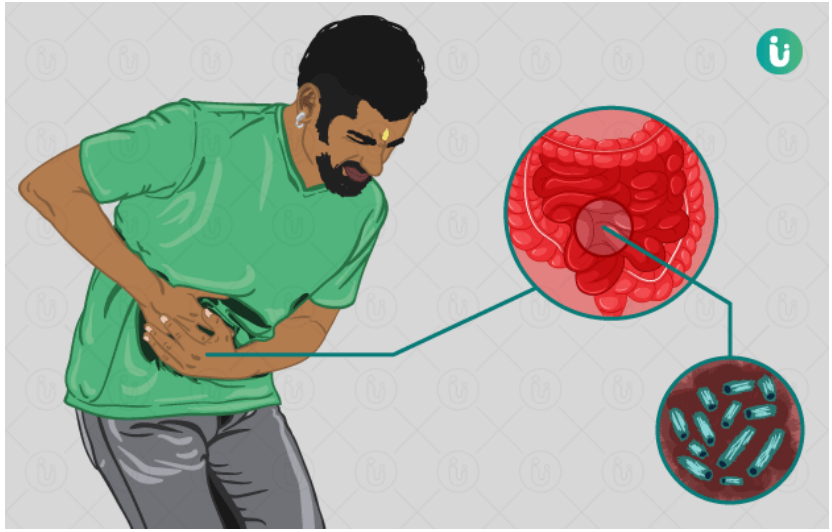
Sonne bacteria possess a biological advantage over other types of Shigella.



Foodborne infection are mainly transmitted through milk and dairy products, meat products, vegetables and fruits, as well as through non-cooked foods (ham, cottage cheese, cold foods, pate, salads, sausages, etc.).

The reason for the growth of foodborne infections in recent years is the violation of sanitary-epidemiological and hygienic standards during the processing, storage and sale of food products

Flies are an active vector of infection.
The role of flies in the transmission of diseases through food is great.
A fly that lands on food can shed 30,000 dysentery bacteria at a time.
Dirty hands also play an important role in the spread of infection.



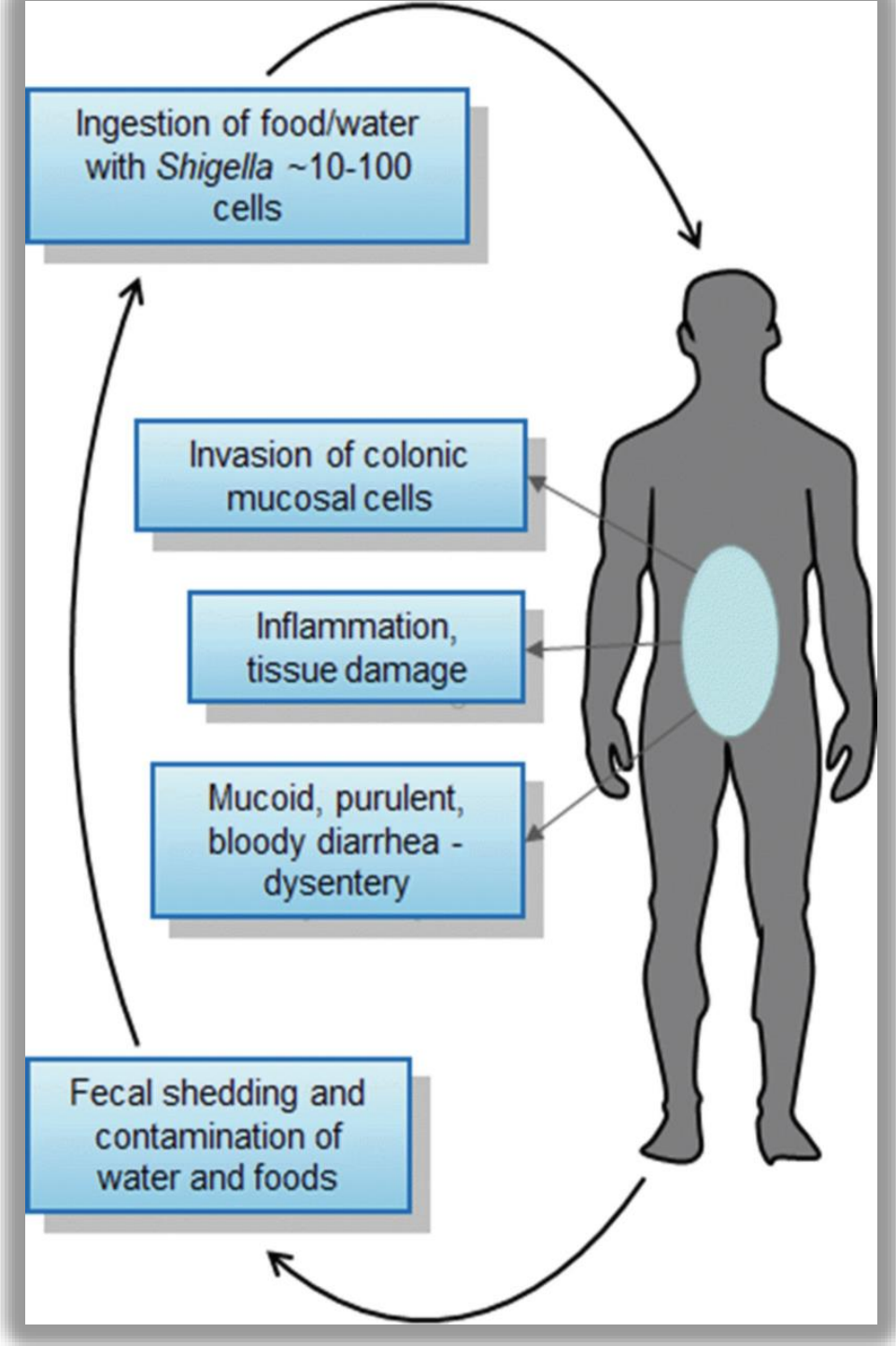
Children are more susceptible to infection than adults to the development of the disease requires a much smaller dose of the pathogen.

Pathogenesis

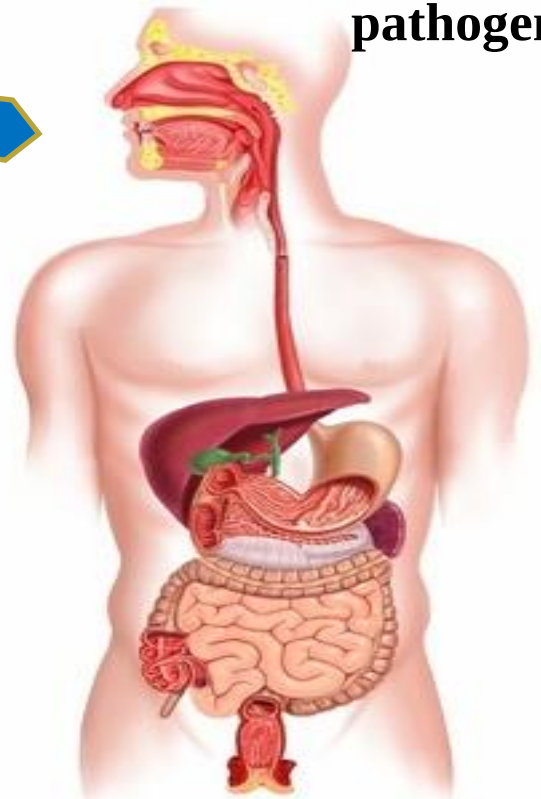
The causative agent of shigellosis enters the body through the gastrointestinal tract.

The duration of the incubation period and the severity of the disease depend on the type and dose of microbes entering the body.

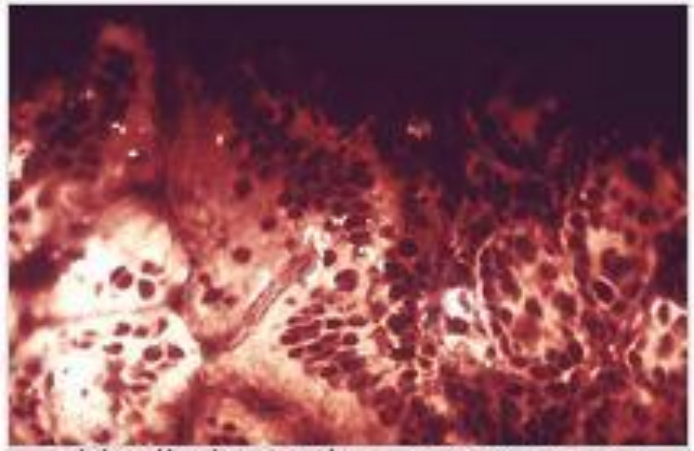
The condition of the macro-organism, gastrointestinal tract, including the level of local and general immunity is of great importance in the pathogenesis of shigellosis.



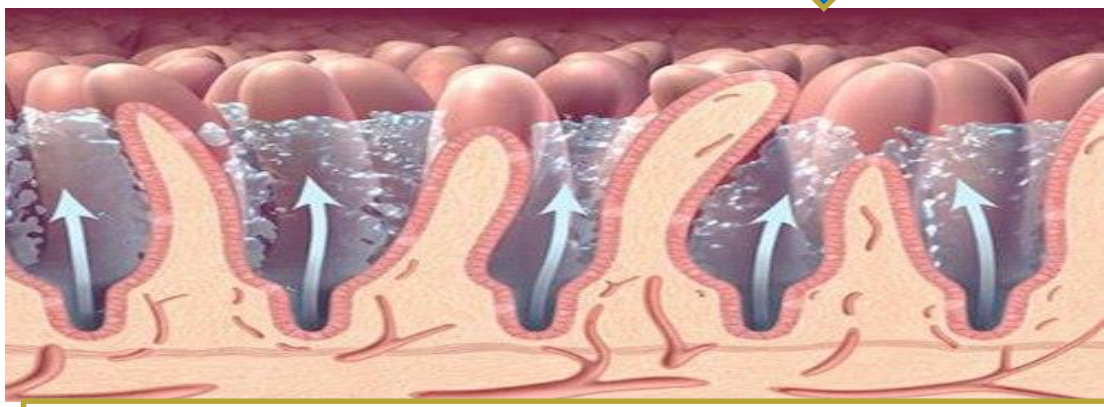
The causative agent of shigellosis enters the gastrointestinal tract only through the mouth.



Under the influence of enzymes in the stomach and along the digestive tract, the pathogens are destroyed.



Shigella bacteria may penetrate the intestinal mucosa.
(photo: CDC/Sam Formal/WRAIR)



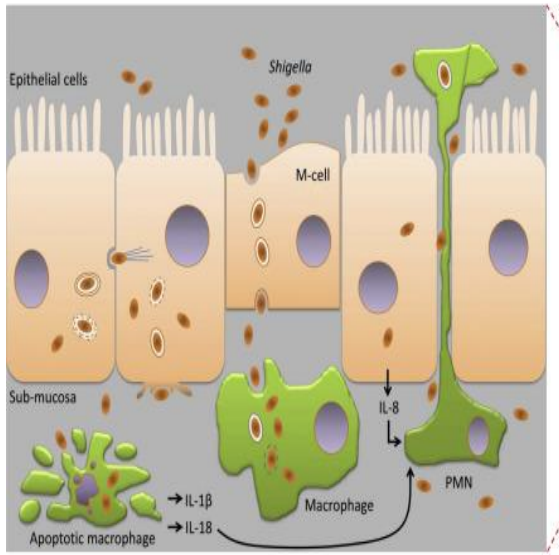
and released endotoxins

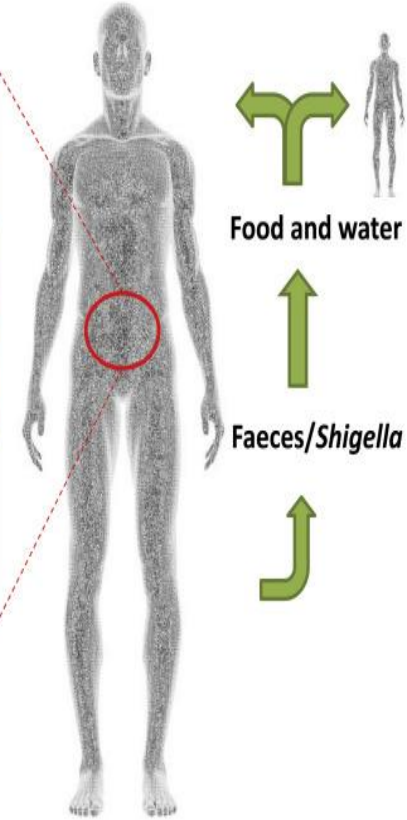
causes

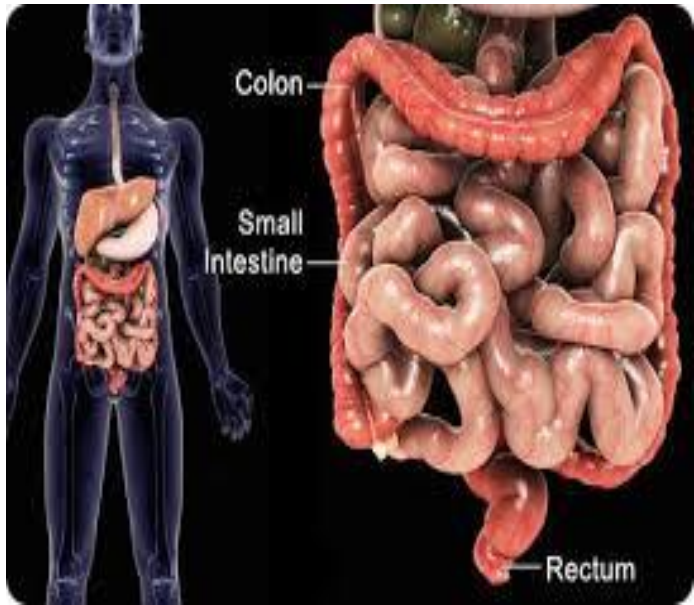
both *local* and *general* toxic syndrome, in cases of severe invasiveness - *endotoxinemia* and *neurotoxicosis*, and even *endotoxic shock*

The mechanism of development of the pathological process in bacterial dysentery is complex. Infection with dysentery occurs only through the mouth. Pathogens enter the stomach from the oral cavity, where they can stay for a day (sometimes longer). Some of them die under the action of hydrochloric acid, digestive enzymes, and lysozyme, releasing endotoxin. Surviving bacteria enter the small intestine, where they also partially die under the influence of bile. The remaining bacteria can linger in the small intestine for up to several days and even multiply. Further, Shigella enter the distal intestines, where they actively reproduce.

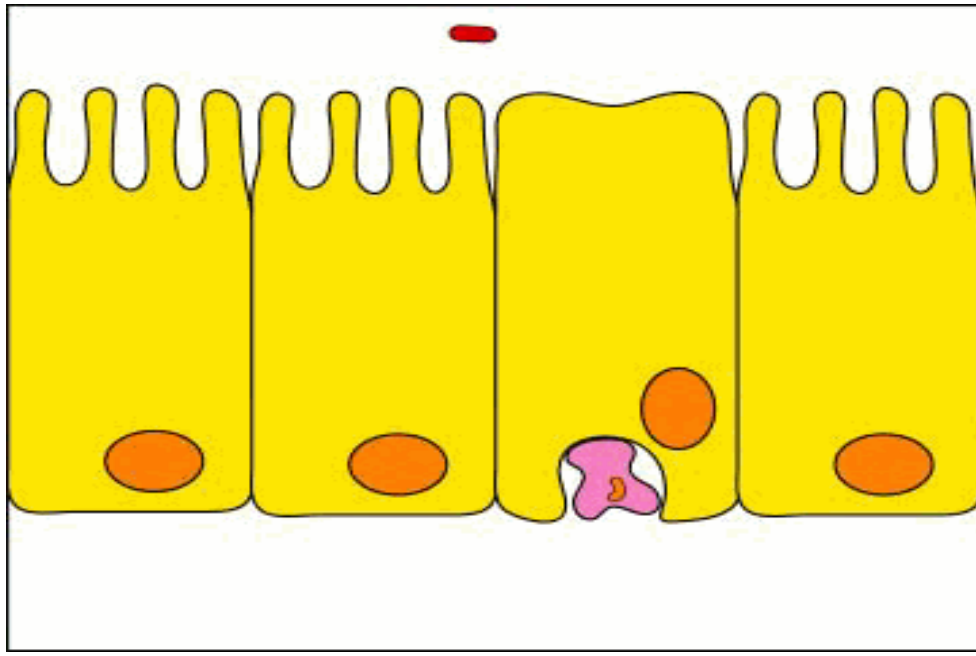
The defeat of the large intestine determines the main symptom complex that characterizes the colitis variant of acute dysentery.

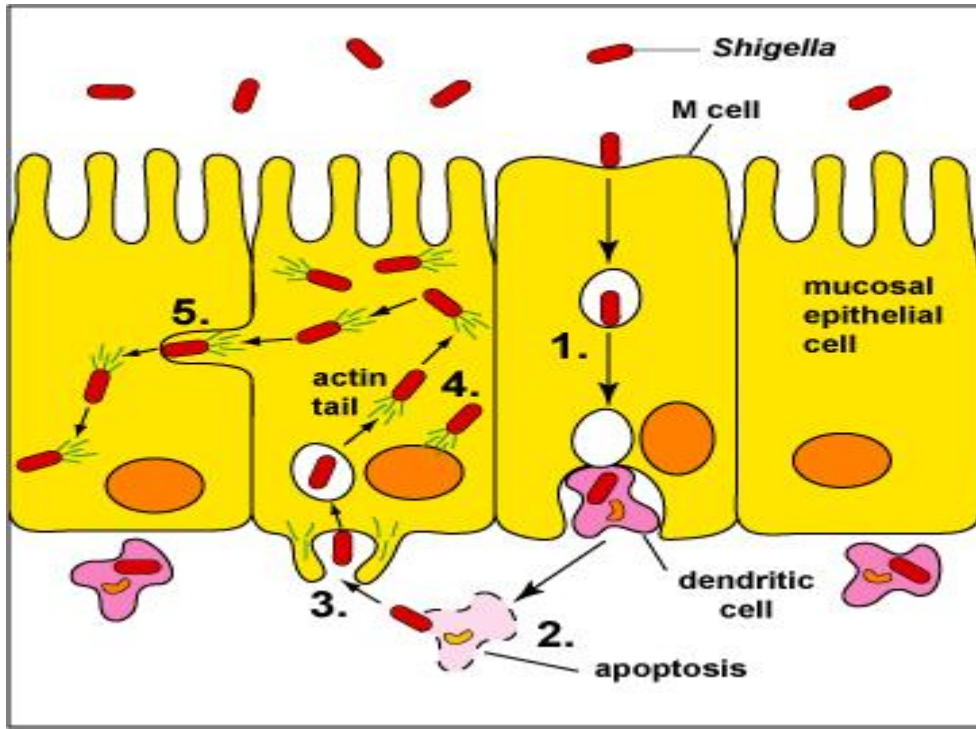




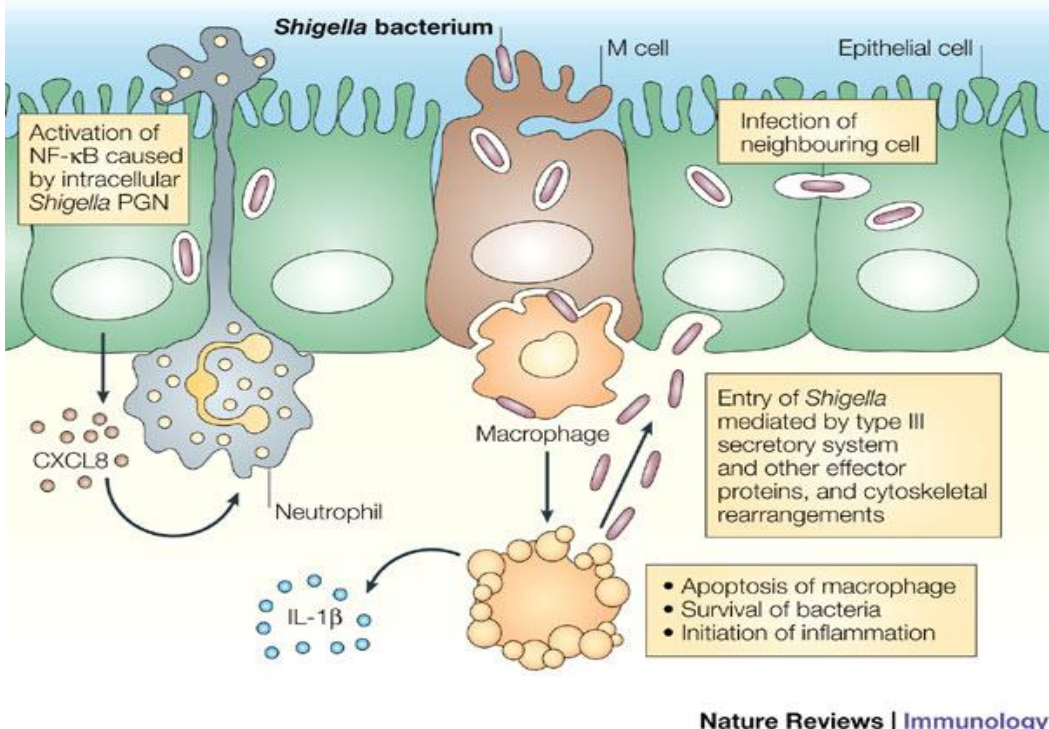


When bacteria die, a number of toxic products are released that cause various morphological changes in the mucous membrane of the colon and systemic lesions. The defining moment in the development of the infectious process in dysentery is the ability of Shigella to intracellular invasion. The interaction of Shigella with the intestinal mucosa begins with the attachment of microorganisms to the epithelial cells, which is caused first by adsorption, then by adhesion. This process depends on the number of pathogens in the space adjacent to the mucous membrane. Adhesion begins with the interaction of Shigella with unchanged, functionally active colonocytes. In this case, the cells of the mucous membrane actively capture microorganisms. Adhesion is accompanied by swelling and rejection of microvilli at the sites of attachment of the pathogen, followed by the development of inflammation.





The penetration of Shigella into colonocytes is closely related to the presence of specific proteins of the outer membrane that can interact with receptors of the plasma membrane of colonocytes. The ability to adhere is genetically determined by the presence of a large-molecular plasmid in Shigella, which controls the invasiveness of the microorganism. Shigella penetrated into intestinal cells actively multiply, releasing hemolysin, which destroys phagocytic vacuoles, which ensures the spread of the pathogen into the intestinal tissue and aggravates the inflammatory process.
Allergic component in the pathogenesis of Shigellosis plays a big role
(pre-existing specific and non-specific sensitization accelerates inflammation of the mucous membrane and the formation of erosions and superficial ulcers there).

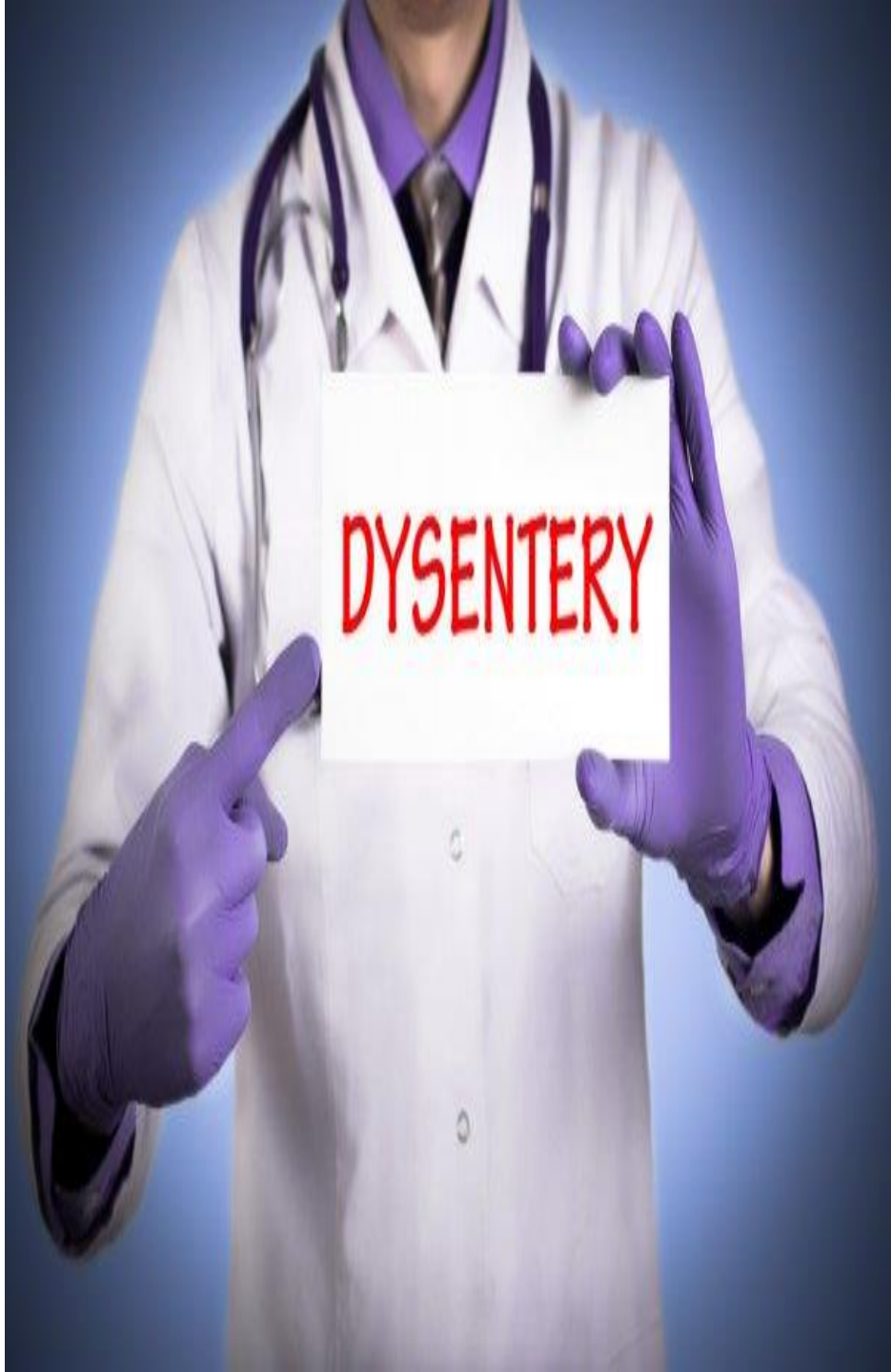


The clinical symptoms of dysentery

The duration of the incubation period ranges from *1 to 7 days (usually 2-3 days).*

There are acute and chronic dysentery.

- **Acute dysentery occurs in several variants (colitis, and gastroenterokolitic, gastroenteritic), each of which may be presented in mild, moderate and severe forms.**
- **Chronic dysentery has recurrent or continuous course and also may be mild, moderate and severe.**
- **There is also bacteriocarrier shigellosis (bacterial excretion), which is considered as a subclinical infection.**



The clinical manifestations of Shigellosis are varied. However, in typical cases, the disease begins

with signs of general intoxication

or signs of neurotoxicosis

and with colitis syndrome

- **abdominal pain,**
- **tenesmus,**
- **spasm and tenderness on palpation of the S-shaped intestine (sigmoid colon),**
- **signs of sphincteritis,**
- **softness or openness of the anus,**
- **frequent, greenish loose stools, with a slight admixture of mucus and blood**

Dysentery is characterized by a cyclical course. Shigellosis typically evolves through four phases:

incubation,
watery diarrhea,
dysentery, and
the postinfectious phase.

The clinical picture of a colitis variant (typical shigellosis) are two major syndrome –

intoxication and a colitis.

The disease almost always begins abruptly, the body temperature rises to 38-39^{0}C and more, not more than 3-5 days. In most cases, vomiting occurs 1-2 times or more on the first day of the disease, but usually does not recur in subsequent days. Shigellosis is not characteristic of vomiting that lasts for 3 or more days.

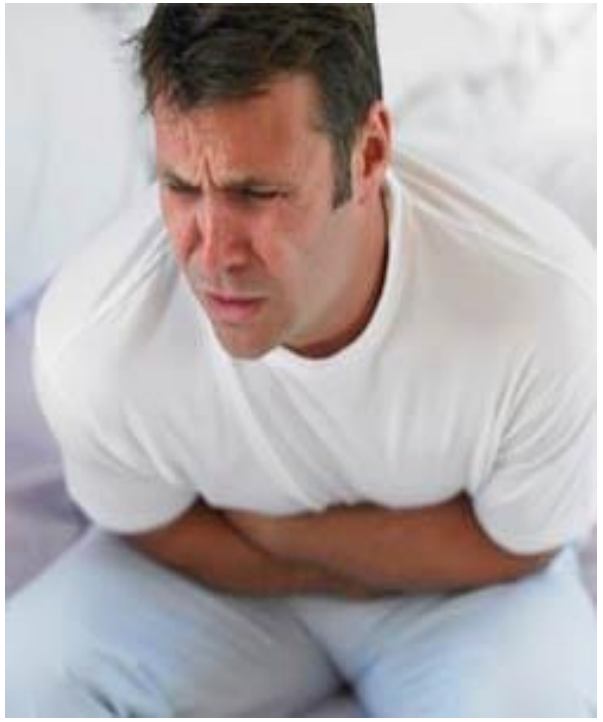
The patient is restless, sleeps poorly, complains of pain in the head and abdomen.

In typical classic cases, colitis is the leading symptom of shigellosis.

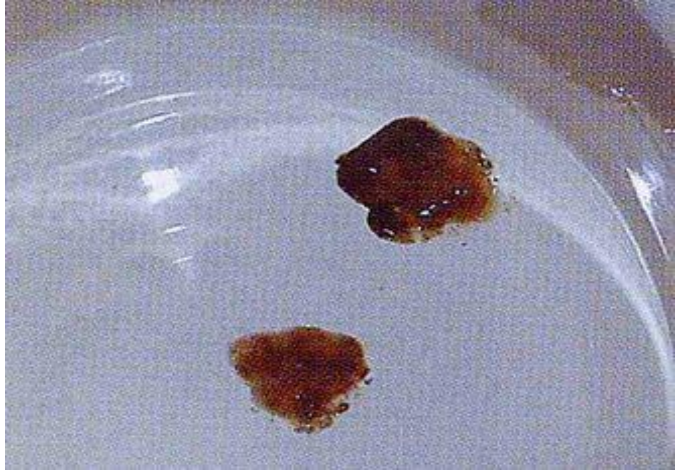
Patients complain of cutting, cramping pains in the abdomen, localized in the left inguinal region, the intensity and duration of which depend on the type and severity of the disease. Abdominal pain usually precedes and overlaps with each bowel movement. Desires are often unsuccessful, accompanied by painful pulling pains in the rectum - *tenesmus.*

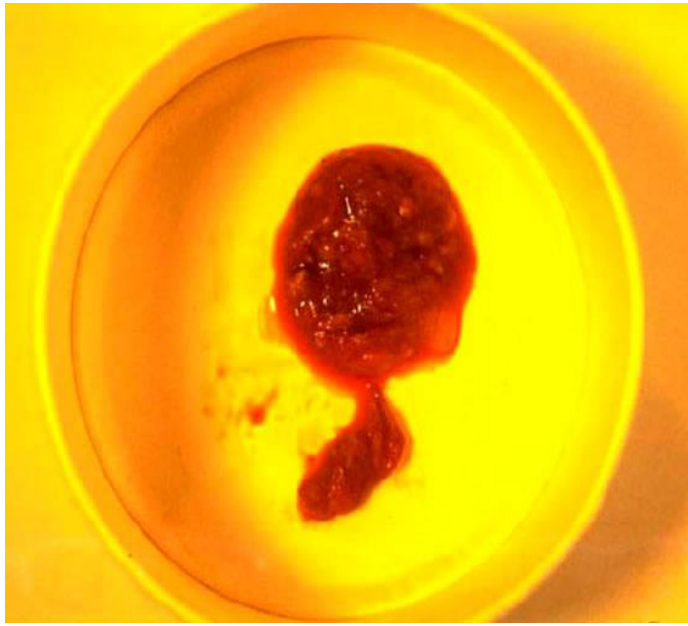
Palpation of the abdominal cavity reveals a spasm, tonic tension of the colon, in milder cases, only its distal part - the sigmoid colon. The latter is palpable as a dense, infiltrated, inactive, sharply painful cord. Palpation often increases the spasm of the intestinal muscles and provokes the urge to defecate.

The nervous system is affected very early.
In most patients, from the beginning, weakness, fatigue, lethargy, depressed mood, headache are observed, which reach the greatest extent with an increase in temperature.
Determined by the lability of the heart rhythm, sometimes a violation of the heart rhythm, a decrease in arterial and venous pressure.
The heart sounds are muffled, a systolic murmur is heard at the apex. In the most severe cases, toxic shock syndrome may develop as a manifestation of toxicity.

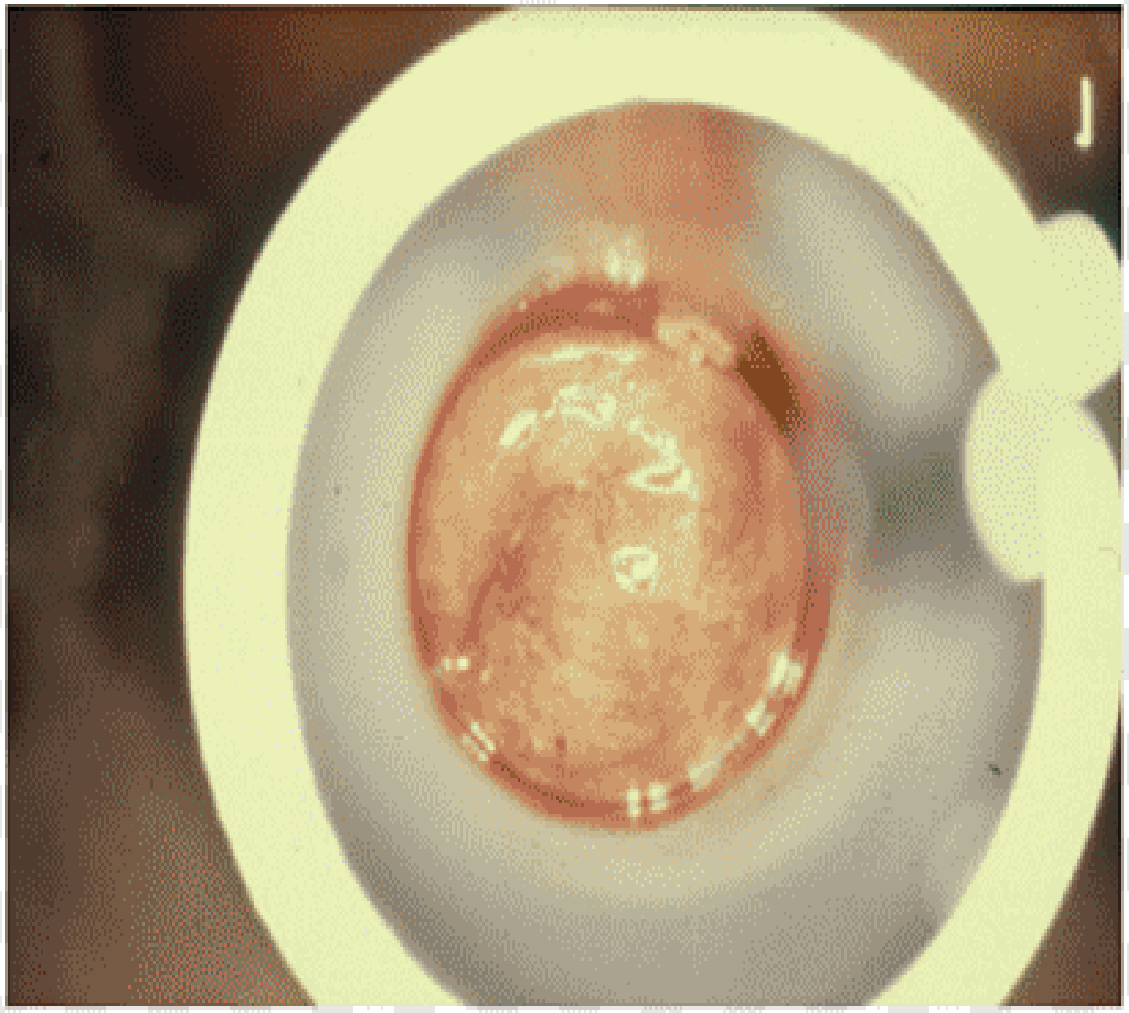


With manifest forms of shigellosis, there is an increase in stool up to 20-30 times a day or more.
Defecation usually does not bring relief.
Despite the multiplicity of stools in the typical course of dysentery in colitis, the amount of stool allocated to patients per day is small, rarely exceeding 0.5-1 liters.
In the first hours, the stool is quite abundant, frequent, thinning, cloudy, mucous, green, with blood, sometimes "hemocolitic", pathological mixtures appear in the stool.
In the early stages of the disease, the fecal matter retains its character. However, at the end of the first day, the stool often loses its character within 2-3 days of the illness, becoming cloudy or mucous-bloody (often purulent) ("rectal spitting").

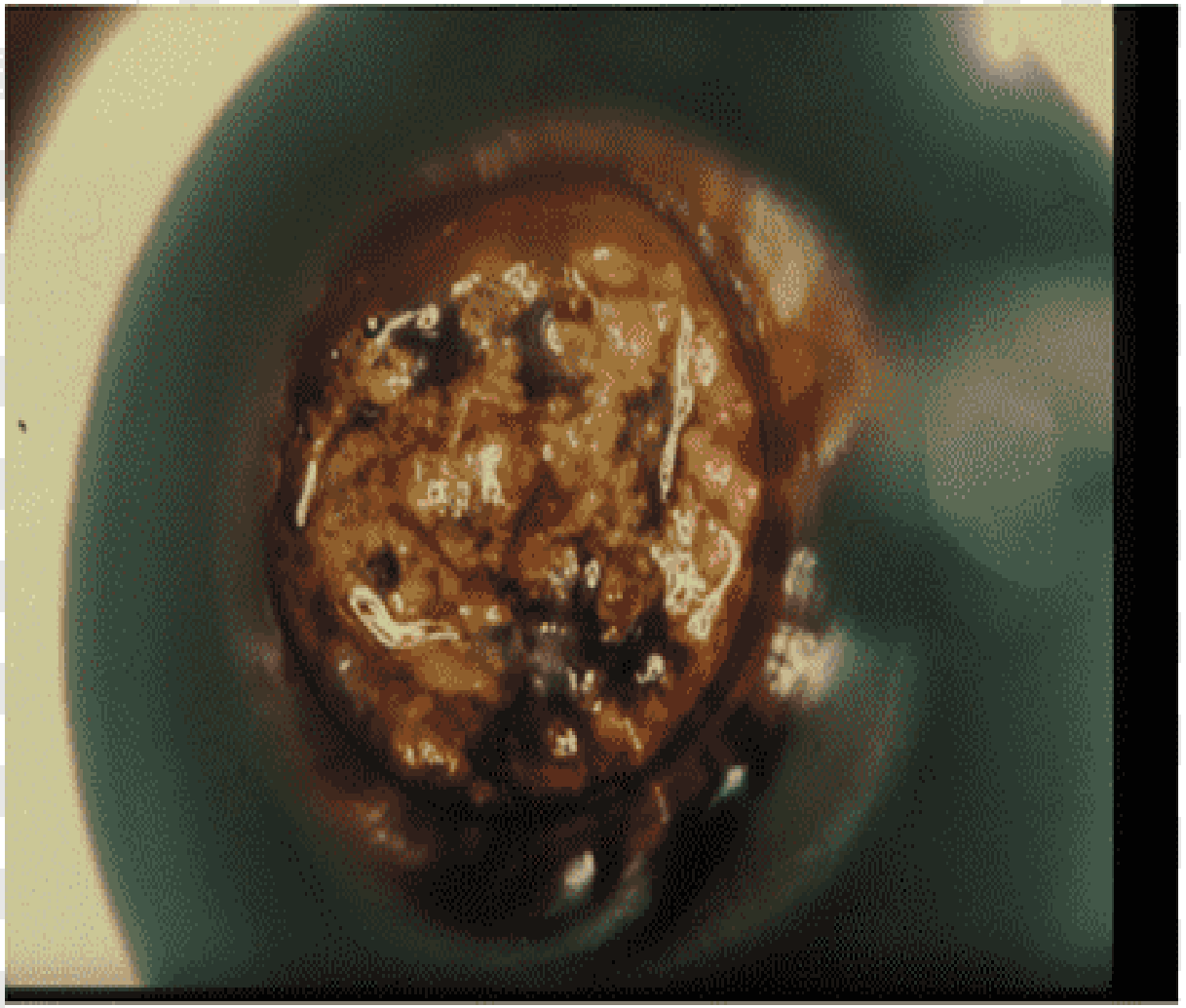




Mild dysentery



Severe dysentery



Hematological changes at the height of the disease are characterized by a slight increase in ESR, moderate leukocytosis, a shift in the leukocyte count to the left, and monocytosis. The duration of the crisis period of the disease ranges from 1-2 to 8-9 days.

As the symptoms disappear, the symptoms of intoxication and colitis subside.

The period of convalescence **is a complete restoration of the impaired functions of organs and body systems and the release of the pathogen. However, as shown by intravital morphological studies, the anatomical "healing" lags behind the clinical one by 2-3 weeks. Late hospitalization, inadequate therapy, unfavorable premorbid background can lead to the transition of the disease into a chronic form, and often to the development of so-called post-shigelliosis states. They show functional disorders of secretion, resorption and peristalsis of the gastrointestinal tract, asthenia. Depending on the severity and nature of the course of dysentery, the clinical picture may be different.**

Forecast

With dysentery, the prognosis depends on the age of the patient, the severity of the disease, concomitant diseases, complications and early treatment.

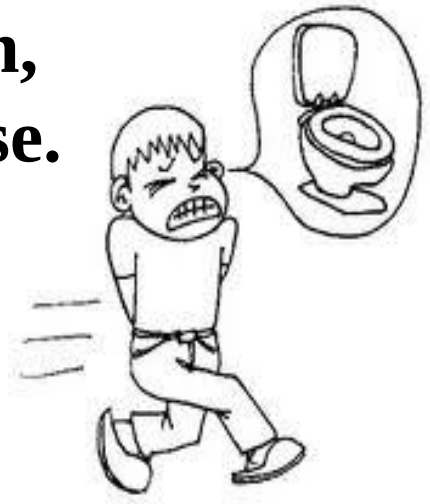
In general, this can be considered favorable for Sonne dysentery, a more severe prognosis for Flexner's dysentery should be considered, especially for Grigoriev-Shiga dysentery.

Mild forms of Shigellosis

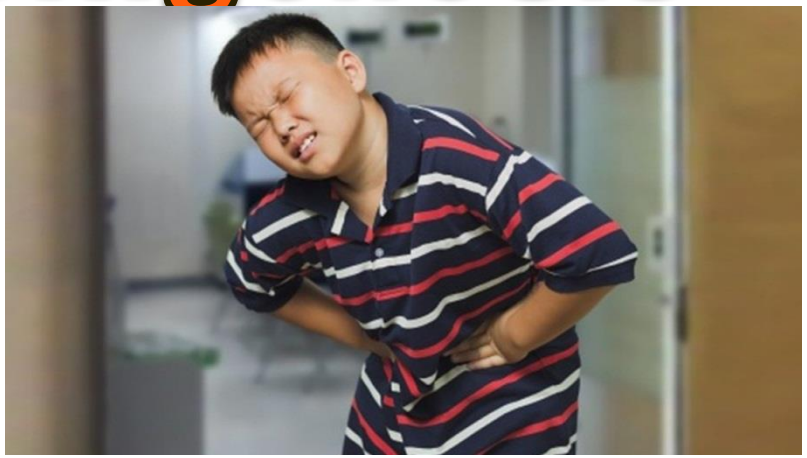
Mild forms of Shigellosis occur in 50-60% of cases.

In the mild form of Shigellosis:

- **symptoms of intoxication are weak,**
- **stool frequency is up to 10 times a day,**
- **faeces do not lose their character, sometimes less mucus, in some cases there is blood.**
- **false urge to defecate are rare.**
- **On palpation, the sigmoid colon is hard and weakly painful occurs.**
- **There is no pain in the abdomen, or defecation time pains will arise.**

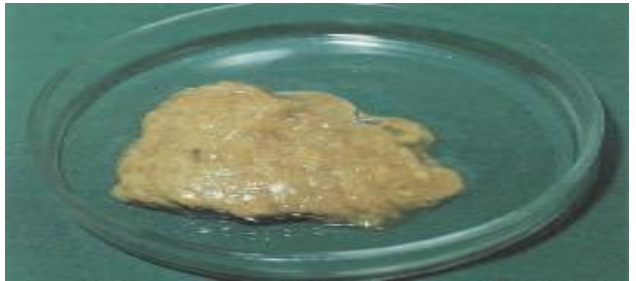


The moderate form of Shigellosis



The moderate form of Shigellosis accounts for 40% of the total structure of acute intestinal infections;

- **signs of intoxication and**
- **severity of colitis syndrome are more pronounced**
- **body temperature rises to 38-40^{0}C,**
- **repeated vomiting is noted,**
- **stools occur 10-15 to 25 times a day**
- **stool losses its fecal characteristic**
- **the smaller volume of feces, stool is greenish with mucus and bloody ("hemokolit") occurs.- in 70-75% of cases**
- **In the abdomen occurs cramps, the anus becomes elastic or gaping**

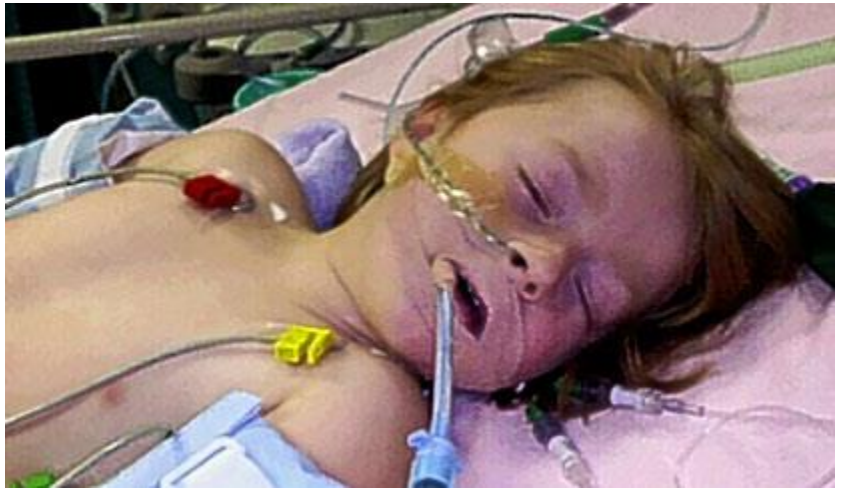


The severe form of shieelliosis

It starts sharply and grows rapidly.

In some cases, the severity of the disease is manifested by infectious toxicosis:

body temperature rises to 39.5-40^{0}C and much higher, there will be repeated vomiting.

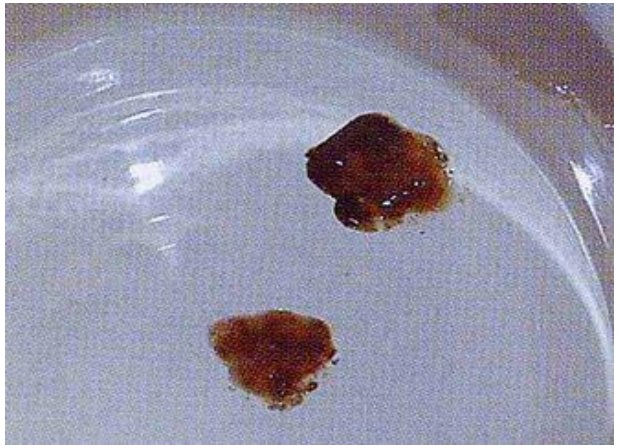


There is pain in the abdomen accompanied by very sharp tenesmus. Defecation can be 20-50 times or more.

Feces consist of mucus and blood

- **loss of consciousness,**
- **delusion,**
- **hallucinations,**
- **convulsions,**

that is, primary neurotoxicosis or signs of infectious-toxic shock appear.



In these cases, the symptoms of colitis appear only a few hours later, or at the end of the first day, which makes it difficult to diagnose the severe form of dysentery.

Complications

Shigella infection usually clears up without complications, although it may take weeks or months before bowel habits return to normal.
Complications may include:

- **Dehydration**
- **Rectal prolapse**
- **Hemolytic uremic syndrome**
- **Seizures**
- **Toxic megacolon**
- **Reactive arthritis** (Reiter's syndrome

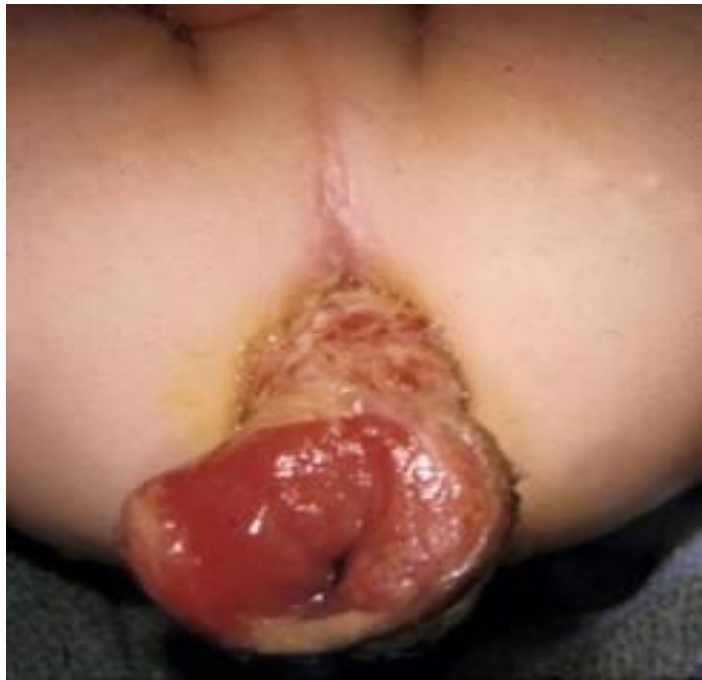
Complications

Dehydration. Persistent diarrhea can cause dehydration. Dehydration is particularly dangerous in children, older adults, and people with weakened immune systems

Symptoms include light-headedness, dizziness, lack of tears in children, sunken eyes and dry diapers. Severe dehydration can lead to shock and death.

Seizures. Some children who run high fevers with a shigella infection have seizures.

Rectal prolapse. In this condition, straining during bowel movements may cause the mucous membrane or lining of the rectum to move out through the anus.

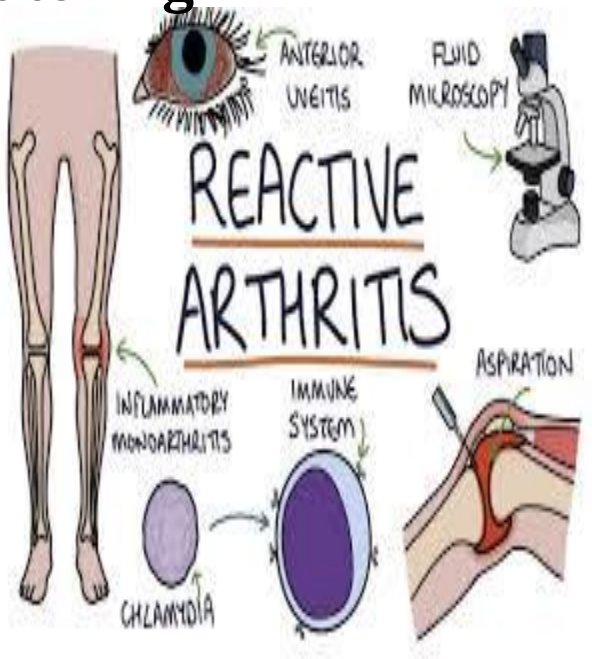


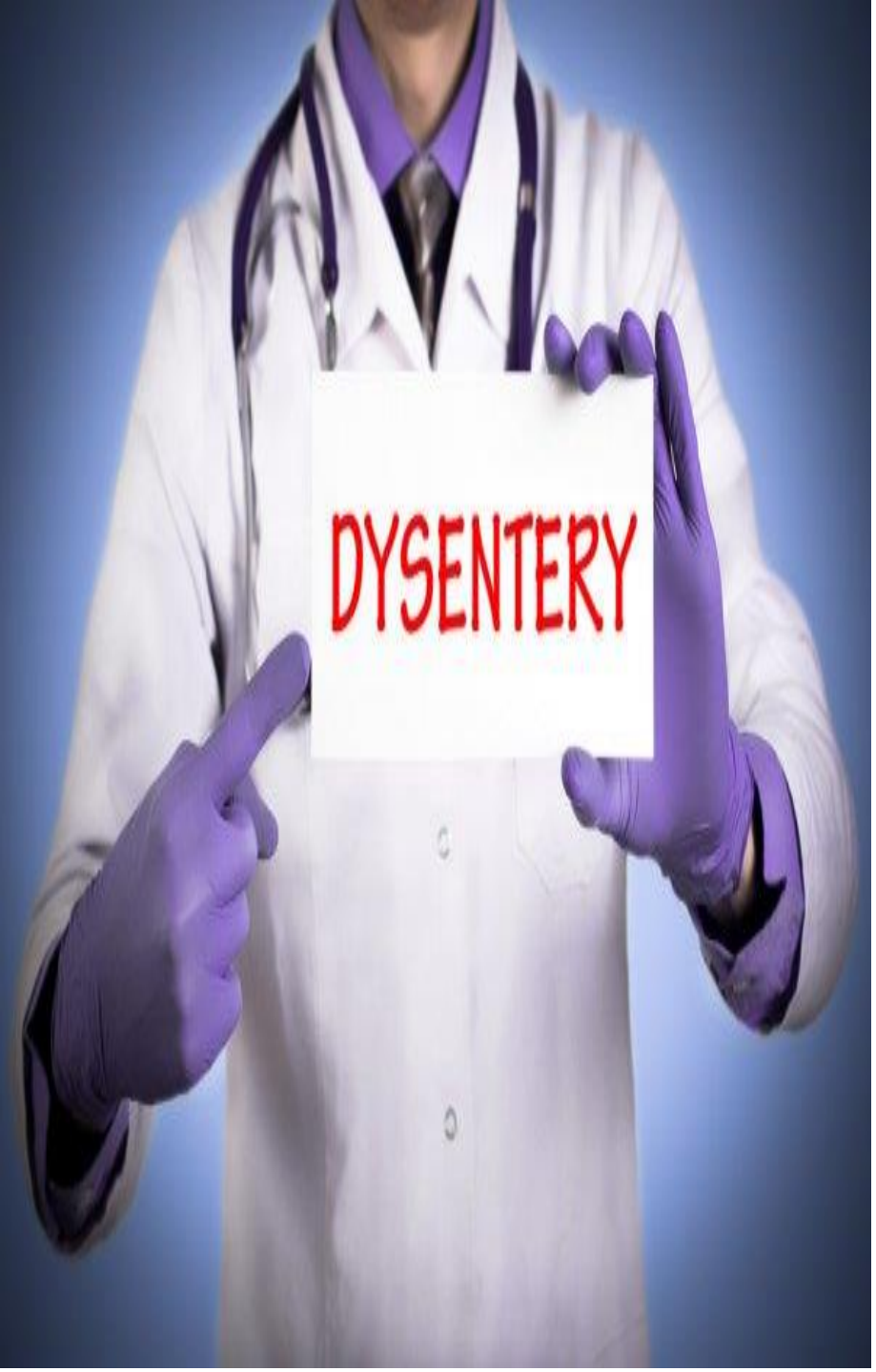
Complications

Hemolytic uremic syndrome. This rare complication of shigella, can lead to a low red blood cell count (hemolytic anemia), low platelet count (thrombocytopenia) and acute kidney failure.

Toxic megacolon. This rare complication occurs when colon becomes paralyzed, preventing from having a bowel movement or passing gas. Signs and symptoms include abdominal pain and swelling, fever, and weakness. If don't receive treatment for toxic megacolon, colon may break open (rupture), causing peritonitis, a life-threatening infection requiring emergency surgery.

Reactive arthritis. Reactive arthritis develops in response to an infection. Signs and symptoms include joint pain and inflammation, usually in the ankles, knees, feet and hips; redness, itching and discharge in one or both eyes (conjunctivitis); and painful urination (urethritis).





Diagnosis

Typically, the diagnostics does not cause difficulties dysentery except atypical disease progression.

The diagnosis is established on the basis of

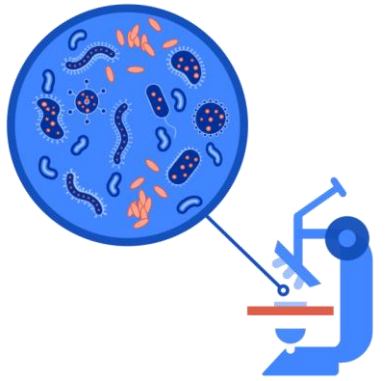
- **epidemiological history,**
- **clinical course of dysentery,**
- **instrumental and**
- **laboratory research.**

The “gold standard” for the diagnosis of *Shigella* infection remains the isolation and identification of the pathogen from fecal material.

However, inoculation of pathogens varies from 22 to 80% and to a large extent on the method, the period and frequency of sampling material, select the environment, etc.

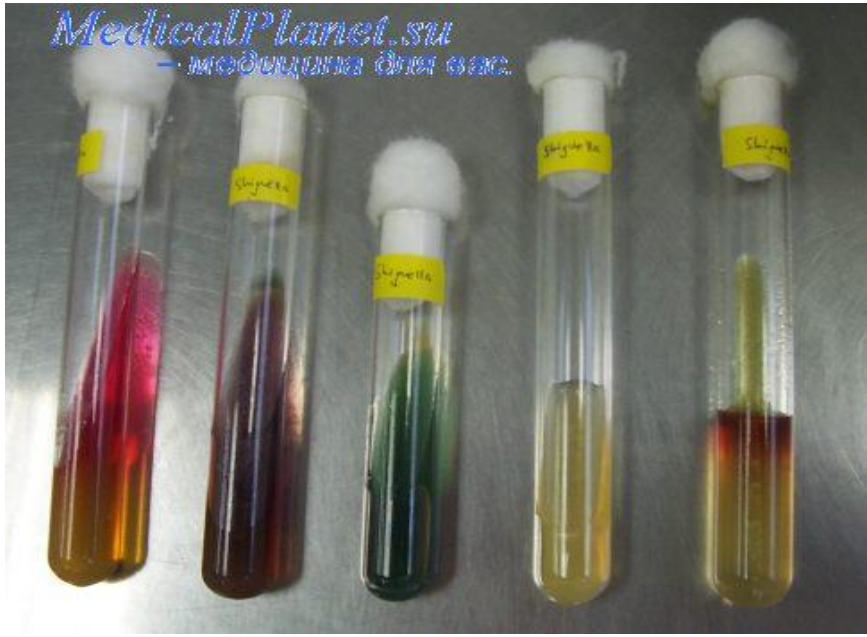
Shigella infection is diagnosed through testing of a stool sample. Because the symptoms of a *Shigella* infection are consistent with a fairly large number of potential illnesses, including most foodborne infections, a diagnosis must be confirmed by a laboratory test. First a stool sample must be obtained from the potentially infected person, and then the sample is placed on a medium to encourage the growth of bacteria. If and when there is growth, the bacteria are identified, usually by looking at the growth under a microscope.

• Shigellosis can be correctly diagnosed in most patients on the basis of fresh blood in the stool. Neutrophils in fecal smears is also a strongly suggestive sign.

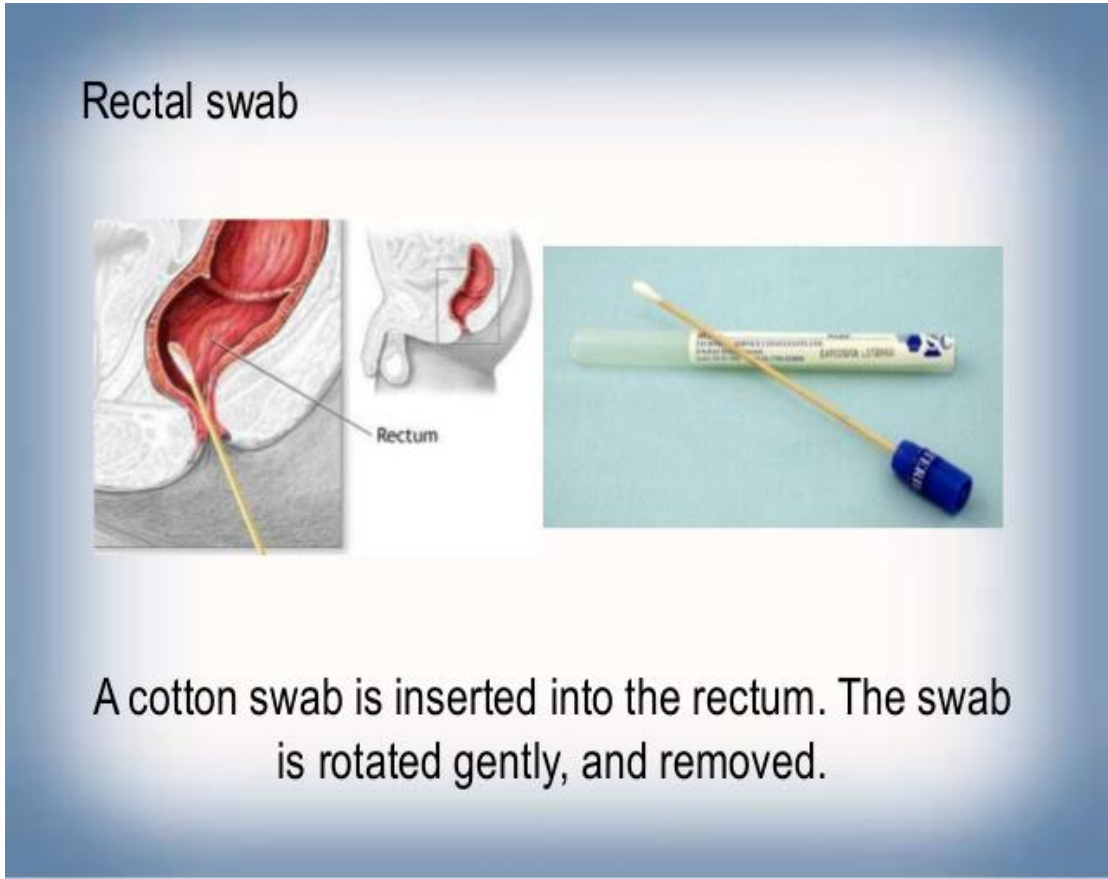


The laboratory can also do special tests to tell which species of *Shigella* the person has, and which antibiotics would be best to treat the infection.
Antibiotic-sensitivity tests are important because *Shigella* is often resistant to multiple antibiotics.
In recent years, developed serological methods for the detection of antigens of Shigella, supplementing, but not replacing the bacteriological diagnosis of Shigella.
As the rapid diagnosis of epidemic outbreaks used immunofluorescence antibody method of dysentery (IAF), RIHA with immunoglobulin (antibody), enzyme-linked immunosorbent assay (ELISA), and others.

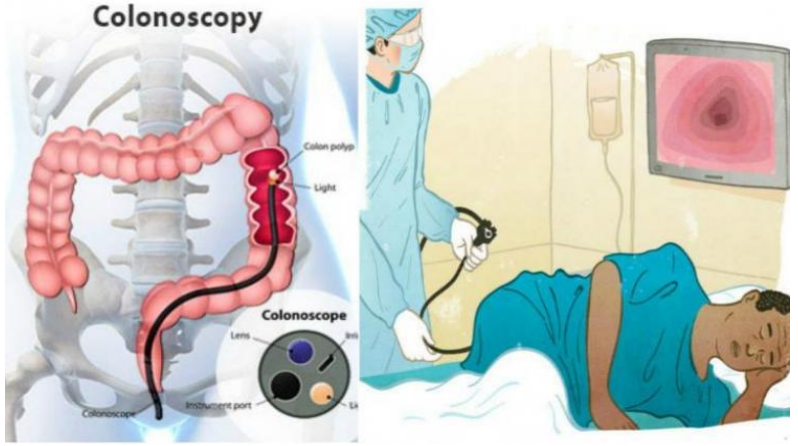
In addition to bacteriological examination to diagnose dysentery used serological method - the reaction of indirect hemagglutination (IHA) with erythrocyte diagnosticum (Phragmites). Positive responses IHA can be prepared by the 5th day of illness.
At the 2 week antibody titers increase, and 4-5 weeks of their tendency to decrease.
Minimum diagnostic antibodies in IHA - titer of 1: 200.



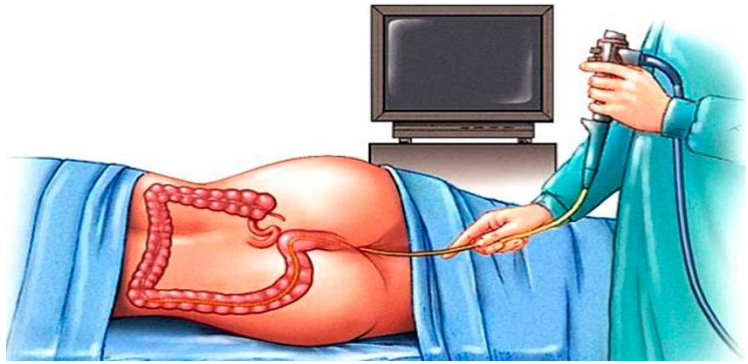
Processing of Rectal Swabs•
Rectal swabs may also be used to culture Shigella if the specimen is processed rapidly or is deposited in a buffered glycerol saline holding solution. Isolation of Shigella in the clinical laboratory typically involves an initial streaking for isolation on differential/selective media with aerobic incubation to inhibit the growth of the anaerobic normal flora.



Sigmoidoscopy is a valuable tool that extends the diagnostic capabilities and allows the physician to monitor the progress of recovery.



Allergic methods (skin allergy test by Zuverkalov) belongs to a purely supporting role in the diagnosis of dysentery.



Differential diagnosis

Dysentery must be differentiated from

salmonellosis,
esherichia Coli gastrointestinal infections,
food poisoning,
cholera,
amebiasis,
balantidiasis,
giardiasis,
trichomoniasis intestine,
some helminths,
candidiasis.

Similar dysentery symptoms may occur in cases of poisoning by fungi and salts of heavy metals, uremic colitis, intestinal tuberculosis, chronic enterocolitis, ulcerative colitis.

Often there is a need to differentiate from acute dysentery surgical diseases (acute appendicitis, mesenteric arterial thrombosis, ileus) and acute gynecological pathology (ectopic pregnancy, adnexitis, pelvioperitonit).

Well, collected medical history, epidemiological history and thorough clinical and laboratory examination of the patient allow the correct and timely recognize dysentery.

Treatment of acute dysentery

The basic principles of treatment of patients with dysentery are possible early treatment, an individual approach to therapeutic interventions for each patient, complexity of care.
Dysentery can be treated at home and in hospital.
The question of admission is decided on the basis of clinical epidemiological data.
Hospitalizations are subject to patients with moderate to severe dysentery, the person with serious underlying medical conditions, as well as patients, representing an increased epidemiological risk (food industry workers and similar contingent him).

The principle of the treatment of patients with dysentery includes

- **medical protective regime,**
- **diet,**
- **etiotropic,**
- **pathogenetic and**
- **immunoregulatory therapy**.

For public health reasons, most experts recommend treating any person whose stool culture is positive for Shigella species.

The cornerstone of the therapy is appropriate rehydration in cases of severe fluid losses.

Use of antimicrobials shortens duration of illness and shedding, decreases risk of transmission person-to-person.

If unknown antibiotic susceptibility:

ciprofloxacin 500 mg or norfloxacin 400 mg or ofloxacin 200 mg all per os twice daily × 5 days. Alternatives (usual duration 5 days):

ceftriaxone (1 g intravenous every 24h),

azithromycin (500 mg per os day 1, then 250 mg per os days 2-4), depending on susceptibility patterns.

In southeast Asia, growing resistance seen to fluoroquinolones, azithromycin may be preferred.

Prevention of dysentery

The successful fight against dysentery provides for a complex of

- **therapeutic and**
- **preventive sanitary and**
- **hygienic and**
- **anti-epidemic measures.**

Measures aimed at identifying the source of infection include early detection, mandatory registration of all patients with acute intestinal infections and their treatment.

Of particular importance is the timely detection of erased, subclinical forms of dysentery.

The search for the source of infection is carried out in foci of dysentery, with scheduled and unscheduled inspections established by professional groups and children's teams.

In the event of an outbreak of dysentery, ongoing disinfection is carried out, and after the patient is hospitalized, final disinfection is carried out.

Following measures can help prevent the dissemination of shigellosis: use of , chlorination of unreliable water sources, strict handwashing, refrigeration and proper preparation and cooking of food. Food handlers must be treated with antibiotics and should not be involved in food preparation as long as stool cultures are positive for Shigella