to beta-lactams have been studied by PCR with electrophoretic detection with specific primers to beta-lactamases encoding genes.

39.3% of isolated strains were resistant to 1 and more classes of antimicrobials, while 16.6% of isolates were characterized by multiple resistance (resistant to 3 or more classes of antibiotics). Resistant and multiresistant strains were isolated equally often from children of all age groups. 29.5% of the strains were not susceptible to ampicillin, while 11.2% were insusceptible to cephalosporins. It was established that the resistance mechanism to ampicillin is associated with the production of beta-lactamase molecular classes of TEM, SHV and OXA; to cephalosporins — CTX-M, TEM, SHV and AmpC. The most common genes are beta-lactamases of molecular class TEM (22.7%) and CTX-M (9.6%). Simultaneous production of several beta-lactamases was found in 8.4% of strains. *E. coli* strains producing beta-lactamases, unlike strains that do not produce them, are statistically significantly more often resistant to other groups of antibiotics (quinolones, aminoglycosides, chloramphenicol, tetracycline).

The results indicate that colonization of an intestine by resistant strains of *Enterobacteriacae* starts in early childhood. In the context of the widespread use of antibiotics in medicine, veterinary, agriculture and food industry, such strains persist for a long time in the microbiota of children and adults, making them potential sources of resistance determinants for enteropathogens causing the acute intestinal and septic infections.

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VIRULENCE GENES AND PHYLOGENETIC GROUPS OF COMMENSAL STRAINS OF *ESCHERICHIA COLI*

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The species *Escherichia coli (E. coli)* is globally distributed. Representatives of the species live in the distal intestine of almost every person on the planet, as well as in the intestines of mammals, amphibians and birds, participating in the biotransformation of nutrients and the synthesis of biologically active molecules. Among the non-pathogenic members of the species various pathotypes are found, which cause diseases of intestinal tract (enteropathogenic *E. coli* (EPEC), enterohemorrhagic *E. coli* (EHEC), enterotoxigenic *E. coli* (ETEC), enteroinvasive *E. coli* (EIEC), enteroaggregative *E. coli* (EAggEC)) and extraintestinal infections (uropathogenic *E. coli* (UPEC), meningitis-associated *E. coli* (MNEC)). They are characterized by a variety of virulence factors. Studies of the evolution of intraspecific diversity revealed seven phylogenetic groups, four of which are the main (A, B1, B2, D).

The aim of the study was to determine the phylogenetic profile of the population of *E. coli* commensal strains, to identify genetic determinants of known virulence factors and to compare their prevalence in the genomes of *Escherichia* in different phylogenetic groups.

511 strains of *Escherichia coli* isolated in 2012–2014 were studied. Strains were isolated from children faeces in age groups from 1 month to 17 years old living in St. Petersburg, without diarrhea and urinary tract infections, and studied with the PCR using electrophoretic detection with specific primers to genes encoding virulence factors and markers of phylogenetic groups.

The studied population of *E. coli* was represented by strains of phylogroup A — 33%; B1 — 7%; B2 — 34%; D — 26%. The genome of commensal strains contains virulence genes of EPEC (2.5%), EAggEC (4.5%). Strains with EPEC virulence genes are more common in phylogenetic group B1(18.9%), whilst strains with EAggEC virulence genes are more common in phylogroup D (12.4%). Virulence genes of EHEC, ETEC, EIEC were not identified.The genome of commensal strains contains some genes of UPEC virulence (*hlyB* — 20.9%; *cnf* — 17.4%; *pap* — 29.5%; *sfa* — 19.8%; *aer* — 20.0%). Genes of toxins *(hlyB, cnf)* and adhesins *(pap, sfa)* are encountered more frequently, with statistical significance, in strains of phylogenetic group B2.

The study showed that genes encoding virulence factors for some pathotypes of *E. coli* are also found in the genomes of the commensal *E. coli* strains and the probability of their detection among representatives of different phylogroups is inconsistent.

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THE SEROPREVALENCE *H. PYLORI* INFECTION IN DIFFERENT GEOGRAPHICAL REGIONS

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H. pylori colonizes more than 50% of humans worldwide. It causes unnoticed chronic gastritis in all carriers and represents a major risk factor for peptic ulcer disease and gastric cancer. It is known that a higher prevalence of *H. pylori* infection will lead to a higher overall prevalence of upper gastrointestinal disease. However the form of such disease may be dictated by socioeconomic and environmental factors as well as the habits and traditions of the people living in different geographical regions.

The aim of our work was to study the seroprevalence infection caused by toxigenic *H. pylori* strains among residents of different geographical regions.

We examined residents of the North-West region of the Russian Federation, Central Asia, Guinea and North Vietnam. Age of the examined was from 20 to 50 years. IgG screening for *H. pylori* and *Cag A H. pylori* antibodies was performed using ELISA method with test-system produced DRG (Germany), Biohit (Finland).

In the inhabitants of the North-West region of the Russian Federation, the seroprevalence of infection caused by toxigenic *H. pylori* strains was 55.59±1.2%. The lowest was the infection of *H. pylori* in North Vietnam — 43.75±8.7%. The highest percentage of *H. pylori* infection was found in Guinea and Central Asia — 85.11±5.2% and 84.00±3.7%, respectively.

An uneven prevalence of infection caused by toxigenic *H. pylori* strains was found among residents of different geographical regions. An increasing process of migration of the population can lead to the spread of infection and the exchange of *H. pylori* strains, specific for the particular regions. This suggests the necessity for further epidemiological studies of *H. pylori* infection in different geographical regions.

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INFLUENCE OF CHOLESTEROL ON THE GROWTH OF *STAPHYLOCOCCUS* spp.

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The aim of research was investigation of the effect of cholesterol (C) on the growth kinetics of *Staphylococcus aureus* and *S. epidermidis*.

We used *S. aureus* and *S. epidermidis* from ATCC collection, which were cultured in meat-peptone broth with

the addition of C at concentrations of 3, 5, 7 and 9 mmol/l. After 24 hours every hour an optical density of the broth at 580 nm was measured. The concentration of C was determined in samples before and after cultivation. The concentration of C was determined in 37 patients, which were included in 3 groups: 1 — "classical" staphylococcal infection (abscess, phlegmon, carbuncle, mastitis, hydradenitis); 2 — secondary infection of wounds with staphylococci; 3 — "not staphylococcal" infections. To determine the level of C in the culture medium or serum, an enzymatic method was used. Statistical processing of data was carried out using the paired version of Student's t-test.

It was found that C in all concentrations does not have a bactericidal effect on *Staphylococcus* spp. Before cultivation of *S. aureus* the level of C was 3.16±0.06 and after — 2.69±0.04 mmol/l ($p < 0.05$). Such decrease may be due to the fact that *S. aureus* includes in its metabolism the disrepared diphosphate necessary for the synthesis of the cell wall. Under cultivation of *S. aureus* in the presence of C the accumulation of biomass was more intense than in a medium without C. A direct relationship between the accumulation of the biomass of the microorganism and the level of C was shown. In assessing the kinetics of growth of *S. epidermidis*, a similar picture was established. A feature of *S. epidermidis* was an increase in the biomass of cells in a stationary growth phase in the presence of 7 mmol/l of C.

In patients of the 1st group the level of C was 4.6±0.3; 2nd — 3.28±0.26; 3rd — 4.10±0.37 mmol/l. In general, the level of C in patients of the compared groups corresponds to the age norm. However, in patients of the 2nd group concentration of C significantly differs from the values of the 1st group. We assume that in a secondarily infected wound the processes metabolism of microorganisms proceed more intensively, as a result of which C can be utilized more by staphylococci, which leads to decrease in its concentration.

Thus, staphylococci are able to include in their metabolism human C, which may be necessary for them for plastic purposes.

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SENSITIVITY OF BIOFILM CULTURES *KLEBSIELLA* spp. TO CIPROFLOXACIN

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In the study researched the effect of 10-, 100-, 1000-fold values of the minimum inhibitory concentration (MIC_{90} = 2 μg/ml, literature data), of the antimicrobial preparation ciprofloxacin on *Klebsiella* spp. autostrains isolated from coprological probes of kids under 5 years old. The experiment included 47 biofilm-forming *Klebsiella* spp. cultures (28 strains of *K. pneumoniae* and 19 isolates of *K. oxytoca*). A study of the ability of clinical strains to form a biofilm, as well as the influence of a number of concentrations of antibiotic on mature (48-hour) biofilm was carried out in sterile polystyrene plates in a microvolume. Mature biofilm cultures were incubated with ciprofloxacin during the 12 hours under standard conditions with a preliminary purification from plankton cells. The results were considered by optical density of the dye-1% crystal-violet bound to the film on a spectrophotometer at a wavelength of 492 nm. The biofilm formation coefficient was calculated as the ratio of the average value of the optical density of the sample to the average value of the optical density of the negative control. The value of the coefficient ≥ 2.1 was taken as positive.

Biofilms formed by *K. oxytoca* autostems when exposed to ciproflocacin at concentrations exceeding 100- and 1000-fold the MIC_{90} were completely destroyed. When exposed of 10-fold the MIC_{90}, the cells adhering to the surface of the wells formed biofilms that were preserved in 30% of *K. oxytoca* isolates. Among biofilms formed by strains of *K. pneumoniae* 48.3% were insensitive to a 10-fold concentration of ciproflocacin. 35.7% out of this insensitive isolates were insensitive to a 100-fold concentration of the antibacterial drug. In addition, a strain of *K. pneumoniae* was detected, which biofilm was not destroyed by a 1000-fold concentration (2000 μg/ml) of ciprofloxacin. The zone of inhibition of growth of this strain to ciprofloxacin, which investigated by the disco-diffusion method was absent; the strain was characterized as resistant.

Mature biofilms of strains of *K. pneumoniae* were significantly less damaged by exposure to selected concentrations of the antimicrobial drug, ciprofloxacin, compared to *K. oxytoca* isolates.

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THE CORRELATION BETWEEN BIOFILM-FORMATION ABILITY OF *KLEBSIELLA* spp. AUTOSTRAINS AND ANTIBIOTIC SENSITIVITY OF PLANCTONIC CELLS

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The former study is related to planktonic cells *Klebsiella* spp.(n = 117) isolated from coprological probes of kids with disbiotic disorder. These cells were isolated using disc-diffusion method to examine the correlation between their sensivity to 11 antibiotics and ability to form firm biofilms in the wells of polystirol microplate.

The study revealed that isolates of *K. pneumoniae* had more autostrains able to form biofilms that *K. oxytoca* (n = 84, 72.6% and n = 33, 60.6% respectively). More frequently strains were resistent to amoxicillin (*K. oxytoca* — 9%, *K. pneumoniae* — 26%). The insufficient share of biofilm structures can be explained by vast spread of antimicrobial medication.

All studied autostrains of *K. oxytoca* did not reveal resistence to the majority of antimicrobial medication like imipenem, ertapenem, meropenem, cefepimum, ciprofloxacin, levofloxacin. Strains of *K. oxytoca* which do not form biofilms were completely sensitive to tetracycline, chloramphenicolum, moxifloxacin, doxycycline. In this, intestinal isolates of *K. oxytoca* which form biofilms lowered their sensivity up to 5% (tetracycline, chloramphenicolum, moxifloxacin) and 10% (doxycycline).

K. pneumoniae strains did not reveal resistance to imipenem, ertapenem. Isolates of *K. pneumonia* which do not form biofilms were completely sensitive to moxifloxacin, chloramphenicolum and meropenem. Biofilm-forming strains had lesser sensitivity up to 8.2; 3.6; 3.3; 1.6% respectively. Sensitivity of *K. pneumoniae* was 95.7% to levofloxacin. Sensitivity of *K. pneumoniae* autostrains was 95.7% and for biofilm-forming strains was lowered up to 10.4%. Sensitivity of non-biofilm isolates of *K. pneumoniae* to doxycycline and ciprofloxacin was 91.3%, and for non-biofilm — 84.4; 73.8% respectively.

The study revealed that planktone cells *Klebsiella* spp. are able to form biofilm what makes them resistent to most common antibiotics.