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