serum samples. Only 4 of 17 strains tested (3 from CD patients and 1 from healthy donor) were significantly more reactive to IgG of patients sera than those of controls. Although these high-IgG-binding strains are more immunogenic they possess the same virulent potential as the other ones [4]. It is possible that they have specific surface antigens recognized by immune system in CD patients but not in controls. The lack of difference in IgG binding level among the other strains may be accounted for tolerance of the immune system for endogenous fecal bacteria without certain epitopes. It is interesting that one CD patient showed high IgG reactivity with all *E. coli* strains, regardless of their origin. Perhaps, in this individual antibody response was caused by common *E. coli* antigen which could lead to more severe manifestations of the disease.

Conclusion: The testing of *E. coli* strains reveal that some bacteria are more prone to bind to CD serum IgG than those from healthy donors. It can be assumed that the development of the disease, in addition to imbalance in bacterial community, is associated with altered interaction between the intestinal bacteria and human immune system. So further studies on identification of IgG-binding proteins may shed light on CD pathogenesis and reveal new candidates for therapeutic targets.

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TRANSCRIPTOMIC INSIGHT INTO IN VITRO INTERACTION OF *ESCHERICHIA COLI* WITH IMMUNOGLOBULIN A AS A MODEL OF THE INFLAMMATORY BOWEL DISEASE PATHOGENESIS

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Introduction: Immunoglobulin A (IgA) is secreted by the intestinal mucosa to prevent invasion of pathogenic and commensal bacteria and inflammation of the epithelium. Fecal samples of patients with inflammatory bowel diseases (IBD) contain a greater proportion of bacteria coated with IgA compared with healthy ones [1]. IgA-coated microbes increase susceptibility to intestine inflammation after transferring into mice [2]. IBD is often accompanied by intestinal dysbiosis, in particular an increased abundance of *Escherichia coli* [3]. The previous study revealed that the pathogenicity and virulence potential of *E. coli* strains isolated from feces of IBD patients does not differ from strains of healthy subjects [4].

Aims & Methods: The aim of the study was to characterize the shifts of *E. coli* Nissle 1917 transcriptome profile in response to interaction with secretory IgA.

The incubation of *E. coli* Nissle 1917 with and without (control) monoclonal IgA (55 mcg/ml) for 1 hour at 25°C in phosphate buffered saline (PBS) was performed. The experiment was carried out in two biological replications. Total RNA was extracted after cell washing in PBS and libraries were prepared for sequencing on NextSeq 500 platform (Illumina, USA). Resulting reads were aligned with Bowtie2, assigned to genes and counted with featureCounts. Differential expression analysis was performed with DeSeq2 package.

Results: Incubation of *E. coli* with secretory IgA led to a statistically significant change in the expression of 68 genes (p adjusted < 0.05). Only two genes were downregulated in the experiment with antibodies compared to control. These genes encode carbamate kinase (log2FoldChange (log2FC) = -1.10) and alternative ribosome-rescue factor A (log2FC = -1.22) of *E. coli*. The expression of the remaining 66 genes was significantly increased in the experimental group. A number of genes for the bacterial stress adaptation increased their expression in IgA condition compared to control. RNA polymerase sigma factor RpoS was upregulated (log2FC = 0.98). This factor is the main regulator of transcription of genes involved in the stress response. An increase in the expression of genes for nucleic acid reparation, fatty acid beta oxidation, glycolysis, citric acid cycle, and transport of metabolites, carbohydrates, and fatty acids has been found. Porin genes ompA, ompC, ompD (log2FC = 1.09, 1.04, 0.84, respectively) were upregulated in bacteria after incubation with IgA. Porins are known to play a role in adhesion of bacteria to the epithelium. For *E. coli* incubated with antibodies, an increase in the expression of fimbriae genes fimA (log2FC = 0.93), fimC (log2FC = 0.99) was observed. Using these fimbriae, bacteria are able to colonize human intestinal epithelium and form biofilms. The intestinal mucosa in IBD patients produces more IgA than in controls, which can stimulate commensal *E. coli* to synthesize more adhesion structures. This shift can lead to an enhancement in the ability of *E. coli* to adhere to intestinal epithelium, which can provoke inflammation.

Conclusion: Interaction of *E. coli* with large amounts of IgA produced by intestinal mucosa of IBD patients may lead to increase of bacterial adhesion, causing epithelial inflammation. Porins and fimbriae of *E. coli* may become a potential therapeutic target for IBD treatment after verification in further research.

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