THE CAPACITY OF PROBIOTIC *ESCHERICHIA COLI* O83:K24:H31 TO NORMALIZE THE ALTERED FUNCTION OF NEUTROPHILS AFTER ANTIBIOTIC TREATMENT

Authors: Eliška Krčmářová, Eliška Miková, Jiří Hrdý

Affiliations: Institute of Immunology and Microbiology, First Faculty of Medicine, and General University Hospital in Prague, Czech Republic

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Objectives

The fine-tuned interactions between the gut microbiota and the host immune system are critical for the maintenance of homeostasis on the mucosa surfaces. The disruption of this balance induced by antibiotic treatment, can lead to dysbiosis, changes in mutual homeostatic interactions between microbiota and host immune system, and cause metabolic and intestinal disorders. Probiotics appear to be a promising therapeutic approach to restore the mutual interactions between the microbiota and the host immune system to renew intestinal homeostasis. Neutrophils, as a substantial part of the innate immune system, play an important role in immune regulation at the intestinal barrier. In recent years, it has become apparent that neutrophils manifest divergent phenotypes and functions (ranging from immunosuppressive to proinflammatory) under specific conditions. In our study, we aimed to characterize the effect of antibiotic-induced dysbiosis on the proportional characteristics of particular neutrophil subpopulations and their function in different mouse tissue (e.g. bone marrow, blood, spleen). The condition and cytokine environment of the intestinal barrier was assessed as well.

Methods

The ability of the probiotic strain *Escherichia coli* O83:K24:H31 to normalize the abundance of neutrophil subpopulations in mice with induced dysbiosis was tested *in vivo*. Based on the combination of cell surface markers, such as Ly6G, Ly6C, CD11b, CXCR2, and CD62L, the phenotype of neutrophils was characterized by multiparametric flow cytometry. The expression of tight junction proteins (Cldc1, Cldn5, Ocld, Zonulin) reflecting the condition of the intestinal barrier was measured by qPCR. The cytokine production was determined by ELISA and qPCR.

Results

Antibiotic-induced dysbiosis led to the expansion of the neutrophil population with an activated phenotype ($CD11b^{+}CD62L^{low}$), while the administration of probiotic normalized their levels. Similarly, the expression of tight junction proteins was normalized after the probiotic treatment compared to the dysbiotic group.

Conclusion

Administration of *Escherichia coli* O83:K24:H31 could promote restoration of mutual homeostatic interactions between microbiota and host immune system together with normalization of barrier function and proportional characteristics of particular neutrophil subpopulations altered by antibiotic use.

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