Anti-inflammatory effects of bacterial components tested on RAW 264.7, J774.A1 macrophage cell lines and on macrophages isolated from the peritoneum of BALB/c mice.

Inflammatory bowel diseases (IBD) including Crohn's disease and ulcerative colitis results from a dysregulated inflammatory response of the host to intestinal microbes in genetically predisposed individuals. Despite intensively proceeding research the mechanism of this action remains unclear. In our previous studies we confirmed that some bacterial lysates isolated from *Lactobacillus casei* DN 11400, *Bacteroides distasonis* and mycobacterial heat shock proteins (HSP) mitigate the severity of experimental colitis in mice.

The aim of our study was to investigate whether these bacterial components have an influence on macrophages which play an important role in mediating chronic inflammation. We tested the effect of bacterial components on macrophage cell lines RAW 264.7 and J774.A1 and on macrophages isolated from the peritoneum of BALB/c mice. Viability of the macrophages we tested by flow cytometry. Qualitative and quantitative determination of cytokines in supernatants was evaluated by protein microarrays and by ELISA. Another aim was to provide evidence of changes in the activation of the NFκB signaling pathway.

We observed that the bacterial components do not change the viability of cells and proved that the bacterial components downregulate TNF-α and NO production in LPS activated macrophage cell lines. We did find this inhibition in macrophages isolated from the peritoneum. Bacterial components without LPS did not give rise to any production of TNF-α and NO. We found higher production of IL-10 in HSP stimulated macrophages in comparison to other bacterial lysates.

These results indicate, that one of the possible protective mechanisms of action of orally applied bacterial components could be their ability to change the cytokine milieu in the gut, favoring tolerance induction.

Key words: mucosal immunity, macrophages, bacterial components, HSP, TNF-α, NFκB,inflammatory bowel disease, probiotics