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Increased microbe-receptor contact in early life – approaching immune regulation

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The crucial colonization in early life educates immune receptors and cells of the body to form the immune system that we depend on during maintenance, disease, and repair. When regulatory mechanisms fail and the system itself becomes the cause of disease, we should look to the proposed early window of opportunity, where it may be possible to affect the developing immune system towards tolerance.

We hypothesize that increased contact in early life between immune receptors and microbial-associated molecular patterns (MAMP), like TLR-4 and LPS, favors a regulatory immune environment later in life. Dextran Sulphate Sodium interrupts the barrier function of the gut wall by shaving the mucus layer. In low doses it may have the desired contact-increasing effect without inducing colitis-related disease.

Following low-dose DSS treatment in early life of BALB/c mice, we did a gene expression screening in ileum and colon together with cell counts in the spleen and mesenteric lymph nodes combined with sequencing the gut microbiota. We investigated the effect of DSS alone, and in combination with Ampicillin and LPS to elucidate the importance of bacterial ligands.

Our study shows that DSS changes the gut microbiota, and Ampicillin itself can act protective as well as activating on inflammatory markers in a time-dependent manner. It is apparent that DSS works differently in the ileum and colon for some genes. In some cases LPS as only ligand reduces inflammatory markers, but overall it is confirmed that the abundance in bacterial ligands is the most important factor for immune regulation.

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