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Less Pronounced Immunopathological Responses Following Oral Butyrate Treatment of Campylobacter jejuni-Infected Mice

Inhalt

The food-borne pathogen Campylobacter jejuni causes campylobacteriosis which is the most frequently reported bacterial diarrheal disease in industrialized nations. Given that campylobacteriosis cases are rising globally and antibiotic treatment is not recommended, infected patients would substantially benefit from alternative therapies. The short-chain fatty acid butyrate is known for bactericidal and anti-inflammatory effects. This prompted us to investigate disease-alleviating properties of butyrate treatment in a preclinical murine campylobacteriosis model. Therefore, following commensal gut microbiota depletion IL-10−/− mice were challenged with 109 viable C. jejuni cells by oral gavage and treated with butyrate via the drinking water (22 g/L) starting on day 2 post-infection. As early as day 3 post-infection, butyrate reduced diarrheal severity and frequency in treated mice, whereas on day 6 post-infection, gastrointestinal C. jejuni burdens and the overall clinical outcomes were comparable in butyrate- and placebo-treated cohorts. Most importantly, butyrate treatment dampened intestinal pro-inflammatory immune responses given lower colonic numbers of apoptotic cells and neutrophils, less distinct TNF-α secretion in mesenteric lymph nodes and lower IL-6 and MCP-1 concentrations in the ileum. In conclusion, results of our preclinical intervention study provide evidence that butyrate represents a promising candidate molecule for the treatment of acute campylobacteriosis.

Keywords

Campylobacter, treatment, preclinical study, butyrate, scfa

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Professional Status of the Speaker

Professor

Junior Scientist Status

No, I am not a Junior Scientist.

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