

Fecal microbiota are altered and concentration of volatile fatty acids decreased in dogs with inflammatory bowel disease

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Introduction: Microbial dysbiosis has been implicated in the pathogenesis of canine inflammatory bowel disease (IBD) (1). The aim of the present study was to characterize the composition (mainly focused on butyrate producing bacteria) and functionality of gut microbiota of dogs with IBD.

Animals, material and methods: Twenty-three dogs diagnosed with IBD (0.8-12.3 years) and 11 healthy control dogs (0.5-8.0 years) were included in the present study. Dogs with IBD were given a clinical score using the canine chronic enteropathy clinical activity index (CCECAI) (2). Fecal samples (n: IBD: 15; control: 11) were used for DNA extraction, denaturing gradient gel electrophoresis (DGGE), real-time PCR and volatile fatty acid (VFA) analyses. Differences between IBD and control dogs were analyzed using the T-test after data has been confirmed to be normally distributed. Within the IBD group, Pearson's correlation coefficients were calculated between CCECAI and abundance of different bacterial groups.

Results and discussion: IBD and control dogs did not show distinct fecal microbial communities based on DGGE profile cluster analysis; most probably due to the large variations in age and breed. Similarly, real-time PCR revealed no significant differences in the abundance of total Bacteria, Firmicutes, Bacteroidetes, *Enterobacteriaceae*, *Clostridium* clusters I, IV and XIVa and gene expression of butyryl-CoA: acetate-CoA transferase in IBD vs. control group, except for lower abundance of *Lactobacillus* in dogs with IBD ($P=0.029$). A reduction in the abundance of *Lactobacillus* was also found in humans with Crohn's disease and ulcerative colitis (3). Simultaneously, fecal concentrations of total VFA ($P=0.085$), acetate ($P=0.043$) and isobutyrate ($P=0.045$) were lower in IBD vs. control dogs, whereas no difference was observed in butyrate between IBD and control dogs. However, negative correlations were found between CCECAI and *Clostridium* cluster IV, ($r = -0.51$, $P = 0.050$), XIVa ($r = -0.60$, $P = 0.017$), and butyryl-CoA: acetate-CoA transferase gene ($r = -0.62$, $P = 0.014$), suggesting lower numbers or lower activity of butyrate-producing bacteria in dogs with severe IBD vs. dogs with mild IBD indexes. These results indicate microbial dysbiosis might be more noteworthy in severe canine IBD.

Conclusions: Canine IBD was associated with a declined fecal abundance of *Lactobacillus* and butyrate-producing bacteria were decreased in severe cases of IBD. Altered gut microbiota in dogs with IBD was accompanied with less pronounced VFA production.

References:

1. J. S. Suchodolski, S. E. Dowd, V. Wilke, J. M. Steiner, A. E. Jergens, *Plos One* 7, e39333 (2012).
2. K. Allenspach, B. Wieland, A. Grone, F. Gaschen, *J of Vet Int Med* 21, 700 (2007).
3. M. H. Gjaffer, C. D. Holdsworth, B. I. Duerden, *J Med Microbiol* 35, 238 (1991).