

Fecal microbial metabolism is altered in dogs with chronic enteropathy

Sandra Bermudez-Sanchez¹, Rachel Pilla¹, Alessandro Gramenzi², Fulvio Marsilio², Joerg M Steiner¹, Jonathan A. Lidbury¹, Jan S Suchodolski¹.

¹Gastrointestinal Laboratory, Texas A&M University, College Station, Texas, USA.

²Veterinary Medicine Sciences, Public Health and Animal Welfare, University of Teramo, Teramo, Italy.

Several studies have reported intestinal microbial dysbiosis in dogs with chronic enteropathy. Limited data is available about the microbiota gene function in this pathology in dogs. Determining the functional attributes of the microbiome is essential for understanding their role on host metabolism and disease. The aim of this study was to compare the functional roles of the fecal microbiota in healthy dogs and dogs with CE by fecal DNA shotgun sequencing.

Fecal samples were collected from 14 healthy dogs and 20 dogs with chronic enteropathy (CE). Fecal DNA was extracted using a commercial kit (PowerSoil, QIAGEN). Functional characterization of the shotgun sequence reads in the KEGG database was performed using next generation sequencing, in order to identify the relative abundance of specific metabolic pathways. A Wilcoxon test was used for comparison of the gene abundance between groups. Significance was set at $q < 0.05$.

At phylum level, low abundance of Bacteroidetes was observed in dog with CE, compared to healthy control dogs (48.5 vs 1.6%; $q = 0.0006$). Fusobacteria was also significantly increased in healthy controls (0.25 vs 0.04%; $q = 0.0111$). The pathway enrichment analysis of the bacterial metagenomes showed that 130 of 360 (36.1%) total metabolic modules were differentially abundant between studied groups. Genes for carbohydrate metabolism, biosynthesis of amino acids (lysine, threonine, histidine, isoleucine, tryptophan, leucine and serine) and vitamins (ascorbate, thiamine and riboflavin) were decreased in dogs with CE, while genes involved in transport of molecules and homeostasis maintenance during oxidative stress (glutathione biosynthesis) were increased in CE.

Our data presents, as previous reported, an intestinal microbial dysbiosis in dogs with CE. As new finding, our results show an altered microbial metabolism in dogs with CE compared to healthy dogs, characterized by reduction of amino acid biosynthesis and carbohydrate metabolism. Further studies including transcriptomic analysis are warranted to define the consequences of this microbiota dysfunction on dogs with CE.