presentation, clinicopathological abnormalities, treatment, and SDMA concentration pre- (PLE-T0) and post- (PLE-T1) treatment were recorded.

Results: At baseline, SDMA concentration was greater in PLE (T0 14.4 ± 3.09 μg/dL) than control (11.3 ± 3.17 μg/dL) dogs ($P < 0.001$, Hedge's G 0.98), but decreased with treatment (PLE-T1: 10.1 ± 2.73 μg/dL; T0 vs. T1: $P = 0.003$, Hedge's G 1.14). Creatinine concentration was similar in PLE (T0 68 ± 22.4 μg/dL) and control (77 ± 21.3 μg/dL) dogs at baseline ($P = 0.122$, Hedge's G 0.41). Albumin concentration was less in PLE (17.0 ± 5.56 g/L) than control (29.5 ± 5.12 g/L) dogs ($P < 0.001$, Hedge's G 2.33) before treatment, but increased with treatment (PLE-T1: 23.3 ± 6.34 g/L; T0 vs. T1: $P = 0.001$, Hedge's G 1.09), although remained less than the concentration in controls ($P = 0.001$, Hedge's G 1.14). No other clinicopathological differences were evident.

Conclusions and Clinical Importance: Similar to people with IBD, SDMA may be increased in dogs with PLE, the clinical significance of which requires further investigation.

Comparison of serum symmetric dimethylarginine (SDMA) concentrations in dogs with protein losing enteropathy before (PLE-T0) and after (PLE-T1) treatment, and control dogs.

Abstract GI25: Possible Role of Dietary N-glycolyneuraminic Acid and Dysbiosis in Canine Enteropathy Pathogenesis

Giacomo Rossi[1]; Lucia Biagini, DVM[1]; Sara Ricci[2], DVM, PhD; Daniela Oliviero[3]; Livio Galosi[1]; Sinem Gultekin[1]; Matteo Cerquetella[1]; Alessandra Gavazza[1]; Barbara Simionati[4]; Ilaria Patuzzi[4]; Fiorella Carnevali[5]

[1]University of Camerino, Camerino, Italy; [2]University of Veterinary Medicine Vienna, Vienna, Austria; [3]Biessea, Milan, Italy; [4]EUBIOME, Padova, Italia; [5]ENEA, Santa Maria di Galeria, Italy

Background: N-glycolylneuraminic acid (Neu5Gc) is synthesized from its N-acetyl precursor (Neu5Ac) by cytidine-5′-monophospho-N acetylneuraminic acid hydroxylase (CMAH). Absent in humans and ferrets, it is polymorphic in dogs. Loss of the CMAH gene generate a change in the structural profile of glycans of all tissues inducing the production of antibodies against Neu5Gc-glycans.

Hypothesis/Objectives: Prolonged uptake of Neu5Gc by negative-CMAH dog through red meat and dairy products from +CMAH mammals leads to a progressive Neu5Gc-glycans incorporation in host's tissue (xenosialization), particularly if the gut microbiota is altered in de-sialilating bacteria determining an inflammatory reaction ("Xenosialitis").

Animals: For immunohistochemistry, gastro-entero-colic biopsies from archive material belonging to European, Asian, and American breeds were analyzed (35 dogs per group). Also, the fecal microbiota of 2 cohorts (127+167) of healthy and enteropathic dogs was evaluated.

Methods: We chose a polyclonal antibody (Creative Diagnostic, DMABH-C003) for Neu5GC expression. The distribution of desializing bacteria was performed using two different sequencing techniques for different regions of the 16S rRNA gene.

Results: Neu5Gc resulted mainly expressed in colon of dogs with enteropathy ($P < 0.005$) with no relation to breed. Greater prevalence of Clostridiales and Bactroidales was observed in enteropathic dogs.

Conclusions and Clinical Importance: Dysbiosis with increase Clostridiales and Bacteroidales could predispose to xenosialization and intestinal inflammation in negative-CMAH dogs, with a possible greater desializing activity compared to healthy dogs. Increased Neu5GC could cause greater uptake of xenosialoantigens by enteropathic dogs since Bifidobacteria, known for their cross-feeding of sialic acids activity, did not differ between healthy and pathologic.

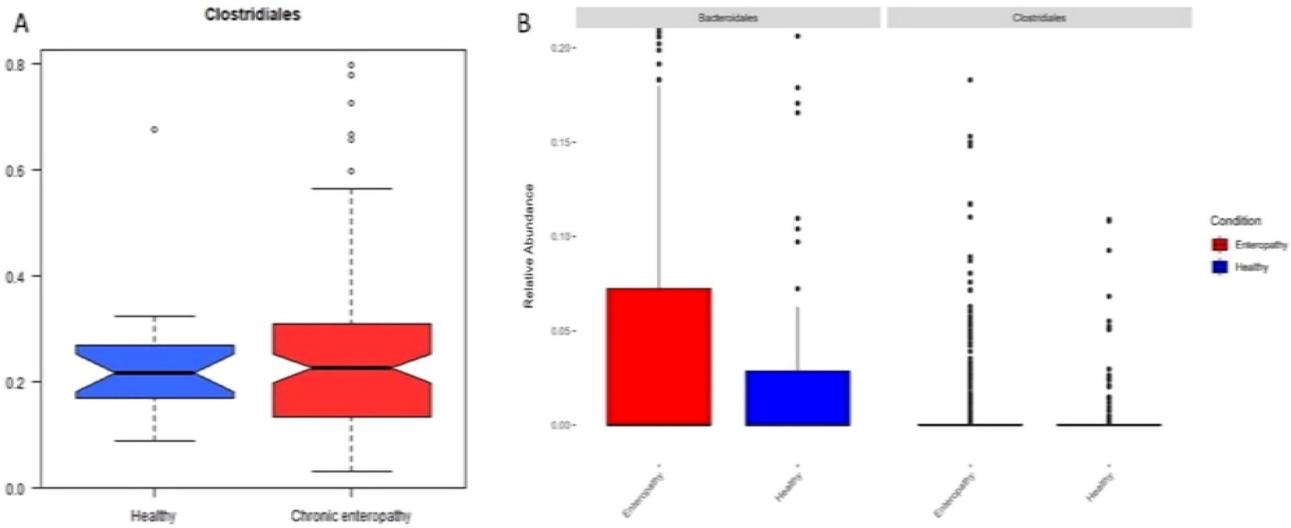

Figure 1. Fecal microbiota of dogs: (A) relative abundance of Clostridiales (sequencing of regions V3-V4); (B) relative abundance of Clostridiales and Bacteroidales (sequencing of regions V2-V9).