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|---|---|---|---|
| 11:15 am | GI-8 | JB Honneffer | Alterations in Fecal Metabolite Profiles of Dogs with Chronic Enteropathy |
| 11:30 am | GI-9 | Rosana Lopes | Targeted Serum Amino Acid Analysis of Hypocobalaminemic Dogs with Decreased or Increased Serum Folate Concentrations |
| 11:45 am | GI-10 | Rosana Lopes | Serum Amino Acid Analysis in Hypocobalaminemic Dogs with Exocrine Pancreatic Insufficiency |
| 12:00 pm | GI-11 | Sergi Segarra | Effects of an Oral Supplement Based on Chondroitin Sulfate, Resistant Starch, and Prebiotics in Dogs with IBD |
| 12:15 pm | GI-12 | Jacqueline Whittemore | Gastrointestinal Endoscopic Mucosal Lesion Scores in Healthy Dogs Receiving Prednisone, Aspirin, and Omeprazole |
| *BREAK* | | | |
| **2:15 pm** | **GI-13** | **Cecily Bonadio** | **Characterization of Canine Dysphagia in a Veterinary University Hospital** |
| **2:30 pm** | **GI-14** | **Talia Guttin** | **Ability of Ultrasound to Predict the Presence and Location of Histologic Lesions in the Small Intestine of Cats** |
| 2:45 pm | GI-15 | Paola Gianella | Prognostic Factors for Short- and Long-Term Survival in Dogs with Protein-Losing Enteropathy |
| 3:00 pm | GI-16 | Jennifer Sinclair | Evaluating Quality and Adequacy of Gastrointestinal Samples with Reusable and Disposable Forceps |
| 3:15 pm | GI-17 | Kelli Bogard | Double-Blind, Placebo-Controlled Ultrasonographic Evaluation of the Effects of Antiemetic Drugs on Antral Motility and Gastric Emptying in Healthy Dogs |
| 3:30 pm | GI-18 | Erin Olson | Evaluation of the Effects of a 2-Week Treatment with Metronidazole on the Fecal Microbiome of Healthy Dogs |
| 3:45 pm | GI-19 | Mellora Sharman | Gastrokine mRNA Expression Is Up-Regulated in Gastric Tissue From Dogs with *Helicobacter* Colinization but without Inflammatory Change |
| *BREAK* | | | |
| 4:30 pm | GI-20 | Yasushi Minamoto | Fecal Short-Chain Fatty Acid Concentrations in Dogs with Chronic Enteropathy |
| 4:45 pm | GI-21 | Tomomi Minamoto | Assessment of Serum Lipoprotein Profiles in Dogs with Chronic Enteropathy and Healthy Control Dogs |
| 5:00 pm | GI-22 | Niels Grützner | Serum Homocysteine Concentrations in Greyhounds |
| 5:15 pm | GI-23 | Stanley Marks | Effect of the Probiotic *Enterococcus faecium* SF 68 on Presence of Diarrhea in Weanling Kittens |
| 5:30 pm | GI-24 | Joseph Parambeth | Fecal N-Methylhistamine Concentrations in Dogs with Exocrine Pancreatic Insufficiency |
| 5:45 pm | GI-25 | Giacomo Rossi | The Effect of the Probiotic Sivoy™ on Clinical and Histopathological Parameters in Cats with Chronic Idiopatic Constipation and Megacolon |
| 6:00 pm | GI-26 | Tim Kretzschmar | Effects of a Synthetic Serine Protease Inhibitor, Camostat Mesilate (FOY-305), on Markers of Pancreatic Acinar Cell Damage, Inflammation, and Fibrosis in Dogs with Suspected Naturally Occuring Chronic Pancreatitis |

**SMALL ANIMAL – IMMUNOLOGY**

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|---|---|---|---|
| **9:00 am** | **IM-1** | **Megan Grobman** | **Thymidine Kinase, C-Reactive Protein and Vitamin D Concentrations in Dogs with Immune-Mediated Thrombocytopenia and Immune-Mediated Hemolytic Anemia** |
| 9:15 am | IM-2 | Lara Barron | Comparison of Proliferative and Immunomodulatory Potential of Adipose-Derived Mesenchymal Stem Cells From Young and Old Cats |

**GI24**

**FECAL N-METHYLHISTAMINE CONCENTRATIONS IN DOGS WITH EXOCRINE PANCREATIC INSUFFICIENCY.** Joseph Cyrus Parambeth[1], Jan Suchodolski[1], Joerg Steiner[1]. [1]Gastrointestinal Laboratory, Texas A&M University, College Station, Texas, USA

Exocrine pancreatic insufficiency (EPI) is a syndrome characterized by inadequate synthesis and secretion of pancreatic digestive enzymes, resulting in maldigestion. Mast cells within the gastrointestinal tract contribute to the pathogenesis of canine chronic enteropathies because of their ability to release inflammatory mediators such as histamine. N-methylhistamine (NMH) is a major metabolite of histamine, and increased fecal concentrations of N-methylhistamine have been reported in dogs with chronic gastrointestinal disease. As EPI in dogs can be complicated by chronic enteropathies, quantification of fecal NMH may serve as a potential prognostic marker. The aim of this study was to evaluate fecal N-methylhistamine concentrations in dogs with EPI.

Surplus fecal samples from 21 dogs enrolled in an unrelated clinical trial at the Gastrointestinal Laboratory were utilized. To be included into the study, the dogs had to be at least 1 year of age, have clinical signs of EPI (i.e., polyphagia, weight loss, steatorrhea, and/or loose, voluminous and/or malodorous stools), have a serum cTLI concentration ≤2.5 μg/L, not be pregnant or lactating, and be free from any clinically apparent disease other than EPI. Three naturally voided fecal samples collected over three consecutive days were immediately frozen after collection and were used for the study. Fecal N-methylhistamine concentrations were measured using a previously validated in-house assay using stable isotope dilution gas chromatography/mass spectrometry (GC–MS). A liquid extract of each fecal sample (1:5 dilution) was used for NMH analysis and fecal NMH concentrations were back-calculated for the wet weight of the fecal samples and expressed in ng/g feces. Results were compared with the established reference interval for healthy dogs and a mean three-day fecal NMH >191 ng/g feces or a maximum fecal NMH of one individual sample of >334 ng/g feces was considered abnormal.

Forty three percent (9/21) of the EPI dogs had increased fecal NMH concentrations (median [range]: 97.1 μg/g feces [0 – 9,973]). German Shepherds and German Shepherd cross breed dogs made up 43% of dogs in the study (9/21), but 78% (7/9) of these dogs had increased fecal NMH concentrations with an odds ratio of 17.5 (95% confidence interval (CI): 2 to 15.6; $P = 0.0102$) when compared to other breeds.

This study showed that fecal N-methylhistamine concentrations may be increased in dogs with EPI, especially when German Shepherds or German Shepherd mixes. Further studies are necessary to elucidate the mechanisms that are responsible for this finding and if increased fecal NMH concentrations are associated with an altered clinical outcome.

**GI25**

**THE EFFECT OF THE PROBIOTIC SIVOY™ ON CLINICAL AND HISTOPATHOLOGICAL PARAMETERS IN CATS WITH CHRONIC IDIOPATIC CONSTIPATION AND MEGACOLON.** Giacomo Rossi[1], Albert Jergens[2], Matteo Cerquetella[1], Sara Berardi[1], Graziano Pengo[3], Jan Suchodolski[4]. [1]University of Camerino, Camerino Italy Marche, Italy, [2]Iowa State University, Ames, USA, [3]Private Clinic S. Antonio, Madignano Cremona, Italy, [4]Texas A&M University, College Station, USA

The pathogenesis of chronic constipation (CC) and idiopathic megacolon (IMC) are poorly understood in humans and animals. In particular, it is unknown whether there are abnormalities involving the extrinsic nerves, the enteric nerve plexuses, interstitial cells of Cajal (ICC) or the intestinal smooth muscle. Abnormalities of any of these components could lead to gut dilatation and impaired motility. Chronic constipation and megacolon occurs more often in the cat than the dog. In humans, probiotics have been increasingly investigated in the management of these colonic motility dysfunctions, particularly their effect on gut transit time, stool output, and constipation relief. While probiotics are used frequently in small animal practice, there are no published studies regarding their clinical efficacy in cats with CC and IMC. The aim of the study was to investigate the clinical and histological effects of a commercial multi-strain probiotic (SIVOY™), containing 200 billion lactic acid bacteria comprised of the following strains: *L. acidophilus* DSM24735, *L. plantarum* DSM24730, *L. paracasei* DSM 24733, *L. delbrueckii* subsp. *bulgaricus* DSM24734, *L. brevis* CD2 #11988, *Streptococcus thermophilus* DSM 24731, *B. longum* DSM 24736, and *B. infantis* DSM 24737.

Ten pet cats of different breeds and ages with a diagnosis of chronic constipation, non-responsive to medical management were selected on the basis of recurrence of clinical signs and absence of any antibiotic treatment for a month. Three of these 10 cats suffered from IMC and full thickness biopsies were sampled for histology. In all animals enrolled in the study, the colon was found to be dilated and impacted with faeces, with the most severe dilatation occurring in the transverse and descending colon, as observed via imaging. CC (n = 7) and IMC (n = 3) cats received orally 200 billion lyophilized bacteria daily for 90 days. Cats were assessed clinically, endoscopically, and histologically at baseline (T0) and after the end of treatment (T1). Histological samples were scored, then evaluated for the immunohistochemical (IHC) expression of CD117 + ICC, enteric neurons, glial cells, and gangliar cell apoptosis (these three latter parameters were evaluated only in the full thickness biopsy obtained at T0 from cats with IMC). Concerning CD117 IHC for ICC evaluation, mast cells, which are known to express the same antigen, were differentiated by staining with Alcian blue, and numbered. Data from constipated cats were compared before and after therapy, and with those obtained from healthy control tissues (archived material from five healthy cats) using paired t-tests or Wilcoxon matched pairs tests, where appropriate. Statistical significance was set at $P < 0.05$.

Constipated cats displayed a significant decrease in ICC, and cats with IMC had significantly more apoptotic enteric neurons than controls. After treatment with SIVOY™, significant decreases were observed for FCEAI clinical index ($P = 0.006$), and histology scores ($P < 0.001$). In contrast, a significant increase of CD117 + ICC was observed ($P < 0.05$) after probiotic therapy.

Cats with CC and IMC showed significant clinical improvement after SIVOY™ treatment, and histological parameters suggest a potential anti-inflammatory effect of SIVOY™, associated with a reduction of mucosal infiltration, and restoration of the number of ICC. The evaluation of microbiota composition after probiotic treatment is in progress to further understand the effects of probiotic therapy on chronic constipation in cats.

**GI26**

**EFFECTS OF A SYNTHETIC SERINE PROTEASE INHIBITOR, CAMOSTAT MESILATE (FOY-305), ON MARKERS OF PANCREATIC ACINAR CELL DAMAGE, INFLAMMATION, AND FIBROSIS IN DOGS WITH SUSPECTED NATURALLY OCCURING CHRONIC PANCREATITIS.** Tim Kretzschmar[1], Jan Suchodolski[1], Joerg Steiner[1]. [1]Texas A&M, College Station, TX, USA

Chronic pancreatitis (CP) in dogs is a clinically underdiagnosed condition characterized by acinar cell atrophy, fibrosis, and loss of exocrine function. Currently, there is no approved pharmaceutical treatment for chronic pancreatitis in dogs, thus management relies heavily on supportive care. Camostat mesilate (CM; FOY-305) is a synthetic protease inhibitor, which has been shown to have inhibitory effects on trypsin, the kinin system, as well as inflammatory and fibrotic cascades. CM has been approved for the treatment of chronic pancreatitis in humans in Japan for over 20 years. However, data on the efficacy of CM in dogs with chronic pancreatitis are lacking. Thus, the purpose of this study was to evaluate the effects of oral CM therapy on markers of acinar cell damage, inflammation, and fibrosis in dogs with suspected CP.

Thirty one privately owned dogs with suspected chronic pancreatitis were enrolled into the study. Dogs were included based on two consecutive canine pancreatic lipase immunoreactivity