The GI Lab

VETERINARY MEDICINE & BIOMEDICAL SCIENCES
TEXAS A&M UNIVERSITY

Promoting Gastrointestinal Health in Companion Animals

Newsletter - 2017

Editors: JM Steiner, JA Lidbury & JS Suchodolski

## What's inside:



## News From the Gastrointestinal Laboratory at Texas A&M University

Every year when I sit down to write this column my first thought is – it simply is not possible that it has been a year since I wrote this column last and then my second thought is, what could I possibly write about as nothing has happened. Of course then my mind quickly floods with all that has happened and I ultimately run out of space to write about it.

I believe the two biggest events for the GI Lab in 2016 were the establishment of the Dr. Mark Morris Chair in Small Animal Gastroenterology and Nutrition and the remodeling of our main research lab.

For the last 10 years we worked hard to try to establish a Chair in the GI Lab and with the support of Hills Pet Care we were finally able to do so. There are several benefits of having a Chair within our program. Most importantly, it gives recognition to our program within the Texas A&M University System and within the Veterinary College. Also, the endowment associated with such a Chair provides a constant stream of funding that can be used as start-up funds for new projects before we have time to find a sponsor. The remodeling of our main research lab has long been awaited. As you know, one of the main foci of our lab is the development of new diagnostic assays. Most of these new assays are immunoassays, the development of which requires us to purify a protein from tissue, characterize this protein, generate an antiserum, set up an assay, and validate said assay. All of this work has essentially been accomplished on 4 short work-benches over the last 20 years. About 3 years ago we were assigned some additional space – 2 small labs with offices, adjacent to our lab. Instead of adding 4 more short benches we decided to overhaul the entire area and create a big open space with 5 long and 2 short work benches that will allow us to perform our work much more efficiently. It has taken us three long years from the first planning until the completion of this space, but we were finally able to move into our new space in the second week in January 2017 (Figure 1).

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**Figure 1. Our newly renovated main research laboratory.**

# News From the GI Laboratory at Texas A&M University (continued)

The Lab also has added two post-docs this last year. Dr. Yasushi Minamoto completed his PhD with Dr. Suchodolski in 2015 evaluating the role of certain pathogens in dogs with chronic gastrointestinal disease. After completing an internship in emergency and critical care at our teaching hospital, Dr. Minamoto returned to the GI Lab as a post-doc in order to continue his work in this area. Also, Dr. Mohammad Khattab first joined us in late 2015 as a visiting researcher from Egypt. Dr. Khattab has had an interesting background as a veterinarian, having run his own company and also serving as a senior member of the Egyptian government, before returning to his alma mater at the Veterinary School in Cairo as a faculty member. Dr. Khattab has been working on studying the gut microbiome interaction with the host immune system. We also have had two new PhD students join our ranks. Dr. Punyamanee Yamkate graduated from Mahidol veterinary school in Bangkok, Thailand. She has been awarded a King's Scholarship to complete her PhD here at the GI Lab. Her main topic of interest will be the normal feline pancreas as there are many open questions regarding this very important organ. Dr. Fatima Sarwar comes to us with a scholarship from Pakistan to complete her PhD in the GI Lab. She will be working on the GI metabolome and microbiome in dogs.

As you probably know, we are always involved in a large number of research studies, most of which are run by our graduate students or our research staff. We often identify potential candidates for a study from results in our database. We apologize in advance for disturbing your work-flow, but on occasion a member of our team will need to contact you to see whether you and your client are interested in enrolling one of your patients in one of our studies. Sometimes we just need patient material and sometimes we are conducting a treatment trial where your patient may benefit from a treatment that may not otherwise be available. We would greatly appreciate your continued support by enrolling eligible patients into our studies. Without your case material our studies are not feasible. Currently, one of our most prominent examples is a study that compares the traditional parenteral administration of cobalamin to oral administration. Several European studies suggest that the efficacy of these modes of administration are the same, so we wanted to conduct a large-scale study in the US to evaluate that hypothesis. Please contact us if you have a patient who is cobalamin deficient before you start any type of cobalamin supplementation. Please contact Dr. Chee-Hoon Chang at cchang@cvm.tamu.edu. So, thanks for helping out and thanks for your continued patronage! (Jörg M. Steiner)

**Things You May Not Know About Us**

The very fact that you are reading this newsletter means that you have a relationship with the GI Lab at Texas A&M University. When you think of the GI Lab at Texas A&M most of you will think of our diagnostic and research work in the area of small animal gastroenterology. But of course the tools we have at our disposal aren't just useful in small animal gastroenterology. So on occasion we get involved in things you may not think of and I have decided to start this column to give you a quick glance at some of those things.

About 3 years ago, I was visiting a major human hospital in Munich, Germany for a sabbatical. The main goal was for me to step out of my comfort zone and expose myself to work with genetically-modified mice, but on the first day I was told that we would need to kill a large portion of the valuable mice because one of the racks had been diagnosed with *Tritrichomonas muris* infestation. I quickly learned that *Tritrichomonas muris* infestations are quite common in research facilities that house large numbers of mice and while it is believed that this infection has little to no impact on the research projects being conducted, this has not been conclusively shown. Since there is no known treatment for this parasite in mice, many facilities will cull the rack in which *T. muris* has been identified. So of course we quickly planned a project to evaluate the efficacy of ronidazole treatment and limited culling in *T. muris* infected mouse colonies. We were able to show that, just as in cats, treatment of tritrichomoniasis can be successfully accomplished in mice. This work has since been published and may save colonies of valuable research mice from being culled in the future.[1] (Jörg M. Steiner)

1. Steiner JM, Schwamberger S, Pantchev N, Balzer H-J, Globokar Vrhovec M, Lesina M, Algül H. Use of ronidazole and limited culling to eliminate *Tritrichomonas muris* from laboratory mice. Journal of the American Association for Laboratory Animal Science, 55: 1-4, 2016.

# NetF toxin of C. perfringens in canine acute hemorrhagic diarrhea syndrome

*Clostridium perfringens* is a gram-positive anaerobic bacterium that has been associated with various diseases in many veterinary species. The virulence of *C. perfringens* is attributed to various toxins. The organism is classified into five types (A-E), based on the presence of the major toxins: alpha, beta, epsilon, and iota. In addition to these major toxins, *C. perfringens* can also harbor other toxins such as enterotoxin and a recently identified pore-forming toxin (*netF*). While the role of enterotoxin in canine and feline intestinal disease remains under debate, the newly described *netF* toxin may be of diagnostic importance, as it has recently been strongly associated with acute hemorrhagic diarrhea syndrome (AHDS) in dogs.[1]

***C. perfringens* as part of the intestinal microbiota**

The intestinal microbiota is highly complex and harbors many *Clostridium* species that are part of the normal intestinal ecosystem. However, *C. perfringens* itself is a relatively minor constituent of the total normal microbiota. Thus, analysis of fecal smears reveals mostly spore-forming bacteria other than *C. perfringens*. Similarly, anaerobic fecal culture cannot be used for the diagnosis of *C. perfringens*-associated diarrhea, because this organism, while playing only a minor role, is a normal constituent of the intestinal microbiota of healthy dogs and cats. PCR based assays are available that detect various toxins of *C. perfringens*. The PCR assay targeting *C. perfringens* toxin A measures the number of *C. perfringens* organisms that carry the gene for toxin A. This reflects the total number of *C. perfringens* organisms in a sample, since virtually all *C. perfringens* of dogs and cats will harbor this toxin. A PCR assay targeting the *C. perfringens* enterotoxin will quantify the number of enterotoxigenic organisms (i.e., those that harbor the gene for enterotoxin).

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# NetF toxin of C. perfringens in canine acute hemorrhagic diarrhea syndrome

However, it is important to note, that detection of enterotoxigenic strains by PCR does not demonstrate whether the enterotoxin was expressed. Therefore, immunoassays that measure the presence of the toxin itself are required to verify whether the toxin is actually being expressed. A new PCR assay is now available targeting specifically the gene encoding *netF* toxin, which has been associated with AHDS.

**Role of *C. perfringens* in disease**

In humans, enterotoxin is responsible for several forms of GI diseases including *C. perfringens* type A food poisoning and sporadic diarrheal disease. Enterotoxin induces toxicity by interacting with intestinal tight junctions, leading to alterations in epithelial permeability. In contrast to humans, the role of *C. perfringens* enterotoxin as a cause of diarrhea remains controversial in cats and dogs.[2] The organism is a normal inhabitant of the canine and feline intestinal tract, with prevalence rates reported up to 100% of healthy dogs and cats, and it has been reported that the organism can be detected at similar frequencies between healthy and diarrheic dogs. Also, the prevalence of enterotoxigenic *C. perfringens* (i.e., those that possess the gene for enterotoxin) is approximately 40% in healthy as well as diarrheic dogs.[3]

A recent study evaluated the severity of clinical signs and presence of *C. perfringens* enterotoxin (CPE) in the feces of 54 dogs with AHDS.[4] While the prevalence of enterotoxigenic *C. perfringens* was higher in affected dogs, there was no significant association between the presence of enterotoxin and important clinical parameters (i.e., severity of clinical signs, duration of hospitalization, mortality rate). Therefore, the conclusion of this study was that the *C. perfringens* enterotoxin does not play a role in AHDS.[4] It has also been suggested that increases in enterotoxigenic *C. perfringens* may be part of the overall microbiota dysbiosis observed in acute diarrhea rather than be causative.

Recently, Unterer et al. characterized the histopathological changes and the presence of bacteria in duodenal biopsies from dogs with AHDS, and observed an increased abundance of mucosa-adherent *Clostridium* spp (Figure 1).[5] All isolated *C. perfringens* strains were positive for the gene that encodes *netF*, a recently discovered pore-forming toxin of *C. perfringens*.[1] However, dogs in the study by Unterer recovered rapidly without the need for antibiotic treatment.[5] This is in line with another recent study by the same group that demonstrated that dogs with AHDS without evidence of sepsis recovered at a similar rate irregardless of whether they had received antibiotic therapy.[6] In our own studies we have observed the presence of the gene encoding *netF* toxin in 19/45 (42%) dogs with AHDS, 0/36 (0%) of dogs with chronic diarrhea, and 4/104 (4%) of healthy dogs (unpublished data). Therefore, it appears that the *netF* toxin gene is strongly associated with AHDS in dogs.

We now offer a PCR test for the detection of the gene encoding *netF* toxin. The test can be ordered separately or as part of our enteropathogen panel. This test can be used to suggest *netF* toxin forming *C. perfringens* as the cause of AHDS in dogs. However, testing is time-sensitive as the organism gets rapidly cleared in 3-7 days. (Jan S. Suchodolski)

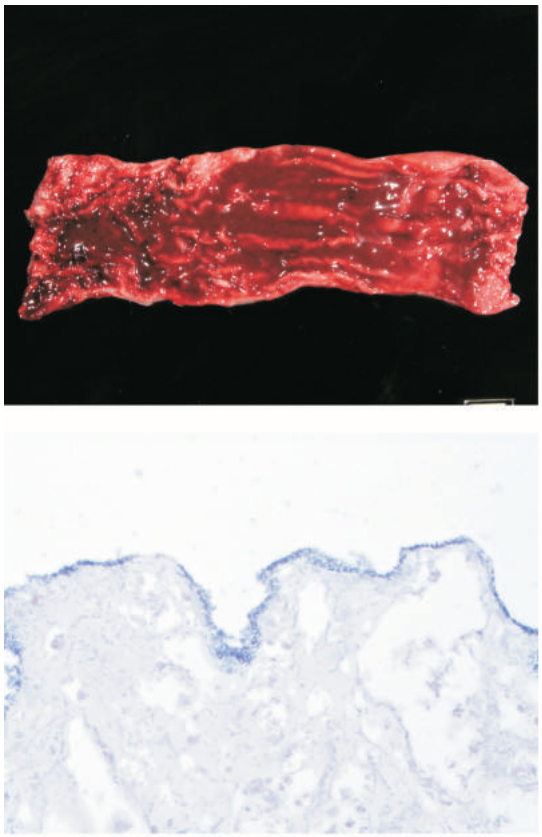

**Figure 1. Jejunum of a dog with AHDS.** There is an increased abundance of mucosa-adherent *Clostridium* spp.

(Pictures courtesy of Drs. Arenas, Porter, and Rodrigues Hoffmann; Texas A&M University)

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2. Cave NJ, Marks SL, Kass PH, et al. Evaluation of a routine diagnostic fecal panel for dogs with diarrhea. JAVMA 2002;221:52-59.

3. Minamoto Y, Dhanani N, Markel ME, et al. Prevalence of Clostridium perfringens, Clostridium perfringens enterotoxin and dysbiosis in fecal samples of dogs with diarrhea. Vet Microbiol 2014;174:463-473.

4. Busch K, Suchodolski JS, Kuhner KA, et al. Clostridium perfringens enterotoxin and Clostridium difficile toxin A/B do not play a role in acute haemorrhagic diarrhoea syndrome in dogs. Vet Rec 2014 7;176(10):253.

5. Unterer S, Busch K, Leipig M, et al. Endoscopically visualized lesions, histologic findings, and bacterial invasion in the gastrointestinal mucosa of dogs with acute hemorrhagic diarrhea syndrome. J Vet Intern Med 2014;28:52-58.

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# An update on Fecal Microbiota Transplantation

The normal intestinal microbiota plays an important role in intestinal health, as it provides nutritional benefits, stimulates the immune system, and protects from enteropathogens. Patients with chronic enteropathies often have an intestinal dysbiosis and the normalization of the microbiota is considered desirable. Normalization of the GI microbiota can be achieved to some extend by the administration of prebiotics or probiotics. Over the last decade fecal microbiota transplantation (FMT) has become increasingly popular in both human and veterinary patients. FMT is the infusion of normal microbiota from a healthy individual donor into the GI tract of a diseased individual, with the aim to restoring the dysbiotic microbiota of the recipient.

Fecal microbial transplantation (FMT) has been used successfully in humans with recurring *C. difficile* infections. The procedure is generally considered to be safe and leads to rapid resolution of clinical signs in approx. 90% of patients with *C. difficile* infection. FMT has also been used in humans with inflammatory bowel disease (IBD), but the success rate is substantially lower than in humans with *C. difficile* infection. This is likely due to differences in pathophysiology of these disorders. While *C. difficile* infection is a classical dysbiosis, the cause for IBD is more complex and multi-factorial, and intestinal dysbiosis is just one component of the disease process.

Through our recent work we have identified bacterial groups that contribute to the differences seen in the microbiota between normal and dysbiotic patients and we have developed a new diagnostic test for intestinal dysbiosis. The Dysbiosis Index (DI) is a rapid PCR panel that quantifies the abundances of major bacterial groups within the canine microbiota and summarizes them in one single number. A DI below 0 indicates a normal fecal microbiota, while a DI of 0 or above indicates intestinal dysbiosis. An increase in the DI is observed mainly in dogs with chronic enteropathies and exocrine pancreatic insufficiency. Also, antibiotic use can lead to lasting dysbiosis in some patients. The DI can be used to screen potential donor dogs for a normal microbiota and we recommend combining this test with a panel for enteropathogens to exclude subclinical infections in donor dogs. The DI is also useful to evaluate the changes in the intestinal microbiota before and after FMT (Figure 1 and 2), as an increase in the DI three to four weeks after FMT may suggest a need for a repeat procedure.

In veterinary medicine, anecdotal reports have recently emerged suggesting that FMT can lead to improvement in clinical signs in some patients with chronic enteropathies, but no comprehensive studies are available. A recent small case series evaluated 3 dogs with a history of chronic enteropathy (CE), that received FMT via nasoduodenal tube, and the fecal microbiota was evaluated using the dysbiosis index.[1] While all three dogs showed an immediate reduction of the dysbiosis index (Figure 1), after 3 weeks, dysbiosis recurred in one dog. This dog also did not show any improvement in clinical signs. In the remaining two dogs, both having tylosin-responsive diarrhea, a partial improvement in clinical signs was observed, and their dysbiosis index stayed below 0 for the follow-up period of 4 weeks. This initial study suggests a potentially beneficial effect of FMT in patients with chronic enteropathies, but larger studies are needed to elucidate the clinical use of FMT. This study also showed that the intestinal microbiota can be monitored using the dysbiosis index, and shows the potential need for repeated FMT in some dogs (see case report and Figure 2).

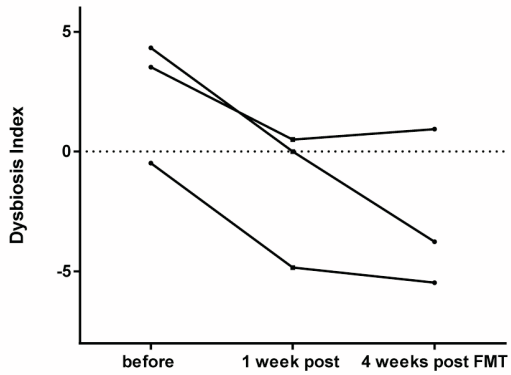


**Figure 1.** Changes in the dysbiosis index before and after FMT. A dysbiosis index below 0 indicates normal microbiota.

**Dr Jennifer Chaitman, VMD, Diplomate ACVIM** from Veterinary Internal Medicine and Allergy Specialists in New York, NY has been performing FMT for several years now (see Box 1 for her recommended protocol). She has performed over one hundred fecal transplants in dogs with various GI disorders. In her experience, FMT has a very high success rate, leading to improvement in stool quality in patients with disorders such as chronic intestinal infections that do not resolve with conventional therapy or in those with chronic non-specific diarrhea.[2] However, patients with confirmed IBD will often have improved stool quality for only a few days to a week after FMT, but then may show recurrence of clinical signs, suggesting that the dysbiosis returns due to the underlying inflammatory disease process (Figure 1). It may be useful to repeat the FMT every 3-4 weeks in these patients. While most clients have some initial resistance to the procedure, they are more receptive after being educated about the use of FMT in the human literature and after experiencing improvement in their animal's stool quality.

While there are many more studies needed to understand which patients would benefit most from a FMT, our recently developed dysbiosis index may provide a better understanding of the microbiota changes before and after FMT. Requesting a dysbiosis index is simple. We only require approximately 0.5 gram of feces (1 gram if you are also requesting our enteropathogen panel). Samples need to be shipped overnight either cooled or frozen. Samples can not be shipped without ice packs. Results will be reported within 2-3 days. (Jan S. Suchodolski)

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2. Murphy T, Chaitman, J. & Han, E. Use of fecal transplant in eight dogs with refractory Clostridium perfringens associated diarrhea. Journal of Veterinary Internal Medicine 2014;28:976.

# An update on Fecal Microbiota Transplantation (continued)

**Box 1. FMT protocol (Dr. Jennifer Chaitman, VMD, Diplomate ACVIM at Veterinary Internal Medicine and Allergy Specialists)**

- Screen donor stool for parasites, enteropathogens (via fecal flotations and PCR diarrhea panel), and for the normal microbiota (using PCR test for dysbiosis index)

- Optionally, at our clinic we place the donor dog on a limited ingredient hypoallergenic diet for six weeks prior to collecting stool for donation

- Use fresh or frozen stool. The recipient will receive 2.5 to 5 grams of donor stool per kg BW blended with 60 ml of saline (blender setting on high). For very large dogs a larger volume of saline may be needed to obtain a sufficiently liquefied stool

- Draw up the mixed stool/saline material into a 60 ml catheter tip syringe, and attach a 12 French red rubber catheter

- Push some of the material into the catheter until it comes out the tip so that no air will be introduced into the colon of the recipient

- Advance the catheter all the way in to the colon, then administer enema. The recipient dog does not need to be sedated

- If possible, do not feed the patient for 4-6 hours. Also, restrict the recipient dog's activity for 4-6 hours after the transplant to lessen the chance for a premature bowel movement

**Case report -** Henry, a five month old male Italian Greyhound was presented for a second opinion for chronic diarrhea associated with *Giardia* infection. The dog had been treated over a three-month period with rounds of drugs such as sulfadimethoxine and fenbendazole, metronidazole/fenbendazole/probiotic and most recently a course of azithromycin and praziquantal/pyrantel pamoate/febantel for 5 days. Dietary change to an intestinal prescription diet improved stool consistency but the stool was still unformed. The dog was normal on physical examination. A fecal flotation for ova and parasites showed no organisms and a PCR diarrhea panel was positive for circovirus. A fecal microbial transplant (FMT) was suggested to see if it would improve the diarrhea in case dysbiosis had been antibiotic induced. Ten days after the initial appointment the dog presented for a fecal transplant. Stool was again collected for examination for parasites and enteropathogens (fecal flotation and PCR panel) and for microbiota analysis (dysbiosis index) right before, 1 week after, and 4 weeks after the transplant. An FMT was given as an enema. The dog's stool quality greatly improved following the transplant. However, 15 days after the transplant the patient developed vomiting and severe diarrhea with mucus and blood in the feces. A stool sample from 15 days after transplant was positive for *Giardia* and circovirus. The patient was started on metoclopramide (0.2 mg/kg PO q12hrs) for vomiting and metronidazole (15 mg/kg PO q12hrs) for hemorrhagic diarrhea. The dog recovered and the *Giardia* infection resolved within one week of metronidazole administration. Later, the dog developed shedding of distemper virus. While this resolved without treatment, the dog still had loose stool. He was then placed on a hypoallergenic diet (Natural Balance venison and potato) and given another FMT and his stool became normal. A Martingale collar instead of a harness was placed on the dog to keep his head off the ground and lessen the chances of future infections. **(Dr. Jennifer Chaitman)**

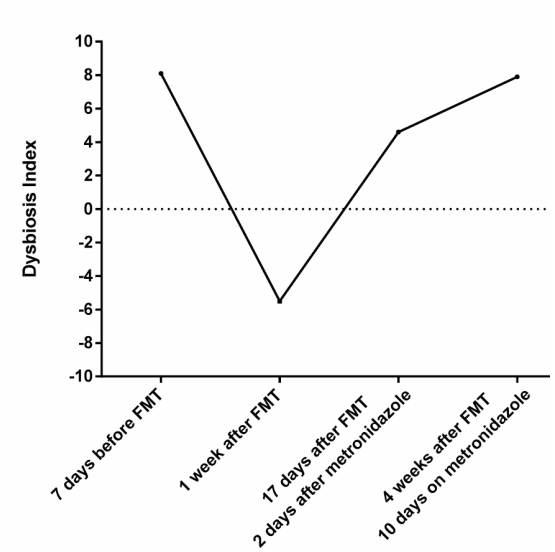


**Figure 2. Dysbiosis index before and after FMT.** A dysbiosis index below zero is indicative of normobiosis, while a value above 0 is indicative of dysbiosis. The patient's dysbiosis normalized one week after FMT. The dog then developed AHDS and the dysbiosis index increased again, likely from the use of metronidazole. This example displays how the dysbiosis index can be used to monitor the changes in the intestinal microbiota.

# Consultant's Corner

Welcome to the 2017 edition of Consultants Corner! As many of you know the GI lab offers a complimentary consultation service to the veterinarians that use our laboratory. I would like to take this opportunity to give you a little bit of background information about the service and some tips to help you get the most out of it:

Our team of board-certified internists is comprised of myself (Jonathan Lidbury), Jörg Steiner, Yuri Lawrence, and Sina Marsillio who are all based at Texas A&M University in College Station and David Williams who is based at the University of Illinois. We are happy to discuss the selection and interpretation of the test that we offer but are also happy to address more general questions about the management of gastrointestinal disease in dogs and cats. To set up a phone consult please call (979) 862-2861. Please provide our office staff your contact information, your patient's name, and the GI Lab accession number for existing cases. Please let them know if you have previous results that you would also like us to evaluate. If possible tell them the specific question that you would like us to answer. You can also email your consultations to gilab@cvm.tamu.edu. Please provide the information listed above, a case synopsis, and whether we should email or phone you back. The team performs these consultations in addition to our other clinical, teaching, and research commitments and so we may not be able to come to the phone right away. If this is the case we will endeavor to call you back within the next business day. It is helpful to tell our office staff the times that you are available to talk to us, because we know you are busy too! It is also helpful if you let your reception staff know that you are expecting this call. You are also welcome to leave a cell phone or evening phone number.

We are always happy to talk to you but I would like to answer some questions that we get frequently get asked to possibly save you a phone call.

**C-reactive protein or protein C: which test do I need?**
It is crucial to recognize that C-reactive protein and protein C are two different molecules and that the measurement of each provides very different clinical information. C-reactive protein (CRP) is a positive acute phase reactant protein that is produced by the liver. CRP is a biomarker of systemic inflammation in dogs that may be useful for monitoring response to treatment in dogs with inflammatory bowel disease. Protein C is an anticoagulant protein that is also produced in the liver. Measurement of serum protein C activity may help distinguish between dogs with congenital portosystemic shunts and dogs with microvascular dysplasia. The GI Lab does offer measurement of serum CRP concentration in dogs. However, we do not currently offer measurement of protein C. One of the laboratories that currently offer the measurement of protein C activity in canine plasma is the Comparative Coagulation Laboratory at Cornell University.

**Can I measure TLI concentrations in a dog or cat that is receiving pancreatic enzyme supplementation?**
Yes, as both the canine and feline TLI assays are immunoassays that use species-specific antibodies. These antibodies do not cross react with the trypsinogen in pancreatic enzyme supplements and so the assays are unaffected by the pancreatic enzyme supplement. However, it in dogs and cats being treated for EPI, repeated testing of TLI is not generally helpful as it is expected to remain very low. Clinical findings and frequently repeated measurement of serum cobalamin and folate concentration are used to monitor response to treatment

**I have a patient with gastrointestinal disease and a low serum cobalamin concentration; will cobalamin supplementation help them?**
Cobalamin is a water-soluble vitamin that plays an essential role in several important metabolic reactions. There is considerable evidence that some dogs and cats with gastrointestinal disease have cellular cobalamin deficiency that affects their metabolism. Cobalamin supplementation was associated with weight gain as well as a reduction in vomiting and diarrhea in a group of cats with gastrointestinal signs that had undetectable serum cobalamin concentrations. In addition to being inexpensive and safe, this vitamin can also act as an appetite stimulant. Therefore, we recommend supplementation in dogs and cats with serum cobalamin concentrations below or in the lower part of their respective reference intervals Please see our website for supplementation protocols. However, it is important to realize that cobalamin deficiency usually occurs as a consequence of gastrointestinal disease (EPI, dysbiosis, or diffuse disease of the ileum) and that is its essential to diagnose and treat this.

**I have recently heard that you can supplement cobalamin orally, how can I do this?**
In the past we only recommended parenteral cobalamin supplementation for dogs and cats with gastrointestinal disease as if they have cobalamin deficiency it is due to decreased intestinal absorption. However, recent research has shown that it is possible to effectively supplement cobalamin orally. Dosing is empiric but we recommended a dose of 250 µg PO once a day for cats and dogs <20 lbs BW, 500 µg for dogs 20-40 lbs BW, and 1,000 µg for dogs >40 lbs BW. After three months of daily treatment the supplement is discontinued for a week before serum cobalamin concentrations are rechecked. Both subcutaneous and oral supplementation of cobalamin are reasonable options and the decision on which to use can be made based on client preference.

**Do you recommend folic acid supplementation for dogs and cats with low serum folate concentrations?**
Folic acid is a water-soluble vitamin that is required for many metabolic processes. Therefore, patients with a subnormal serum folate concentration may benefit from oral supplementation. However, the benefits of folate supplementation are not as clear as they are for cobalamin and I may choose to not supplement a patients that only has mildly decreased serum folate concentrations, especially if they are difficult to pill. Dosing is empiric but we currently suggest supplementing cats and most dogs with 200 µg orally PO once a day and larger dogs with 400 µg once a day for one month. Folic acid tablets are available from most health food or grocery stores. Over-supplementation is not generally a concern as excess folate is excreted in the urine.

If you have any questions or concerns about our consultation service please feel free to contact us.
(Jonathan Lidbury)

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## Ongoing studies

**Dogs with Primary Hyperlipidemia** - Prescription diet naïve dogs newly diagnosed with primary hyperlipidemia are eligible to be enrolled in a dietary trial. Contact Dr. Lawrence at ylawrence@cvm.tamu.edu for more information.

**Dogs with Chronic Pancreatitis** - Dogs with chronic pancreatitis (cPLI >400 µg/L) and hypertriglyceridemia (>300 mg/dl) are eligible to be enrolled in a dietary trial. Contact Dr. Lawrence at ylawrence@cvm.tamu.edu

**Chronic enteropathies in dogs** - Please fill out this brief form http://tinyurl.com/ibd-enroll to see if your patient qualifies.

**Cobalamin Supplementation Study** - Dogs and cats with cobalamin deficiency with normal PLI, and either normal or low (consistent with EPI) TLI to compare the efficacy of oral vs parenteral cobalamin supplementation. Contact Dr. Chang at chchang@cvm.tamu.edu for further information.

**Chronic enteropathies/ intestinal lymphoma in cats** - Cats with signs of chronic GI disease >3 weeks may qualify for our study. Please contact Dr. Marsilio at smarsilio@cvm.tamu.edu

**Acute and chronic diarrhea in cats** - The GI lab is looking for feces from cats with any form of diarrhea. Please contact smarsilio@cvm.tamu.edu.