

Feline Nutrition Basics from a Junk Food Junkie

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The veterinary profession is undergoing a subtle but important shift in terminology. Whereas previously a diagnosis of “Inflammatory Bowel Disease” or IBD was often given to any cat with diarrhea when time, patience, or finances precluded an actual diagnosis, we now tend to start with the term “Chronic Enteropathy”. From there we move forward diagnostically and therapeutically in an organized manner that allows us to add important qualifiers, such as Chronic Enteropathy – Food Responsive Diarrhea. Only when we have exhausted the differential list and procured histopathology do we settle for a diagnosis of IBD (NOTE: the real name is *idiopathic* inflammatory bowel disease and it is a histopathologic diagnosis). One consequence of this shift in vocabulary is a shift in our choice of trial therapies, moving away from early intervention with glucocorticoids (IBD) to a renewed appreciation for the power and importance of dietary therapy.

History

“Pharmaceutical agents are often given inappropriate precedence in the treatment of gastrointestinal tract **diseases**. Nutrients have marked influences on the gastrointestinal tract and manipulation of the diet provides clinicians with a powerful therapeutic strategy to be used alone or concurrently with drug therapy”

W. Grant Guilford, *J Nutrition*, 1994

As early as 1994 Dr. Guilford recognized that different diseases of the GI tract were likely to respond to different dietary manipulations. Simply characterizing the clinical condition was an important first step towards deciding on the best fit amongst diet choices. For example, for chronic small bowel diarrhea Dr. Guilford recommended a “highly digestible, gluten-free, hypoallergenic, isosmolar, low in fat and low in lactose” diet. That should just about cover it!

We have long recognized the cat as an obligate carnivore but we continue to debate just exactly what impact that status should have on what we actually feed this species. Bear in mind that if left to their own devices, and assuming they more closely resembled a contestant on Hunger Games as opposed to the Couch Potato so many of us are accustomed to dealing with, cats would consume a diet high in protein, with low to moderate amounts of fat and minimal carbohydrate. A cat’s obligate daily protein requirement (30% DMB) is over twice that of a dog (12%) and cats have specific requirements for particular proteins (ex. taurine) as well as a number of vitamins, arachidonic acid, carnitine, and vitamin D.

Acute gastroenteritis

Historically the first principle in the nutritional management of acute gastroenteritis has been no nutrition at all – “rest” the GI tract with a 24-48 hour fast. In addition to diarrhea, nausea and inappetence, the patient was often vomiting upon presentation, adding to the argument against putting anything (ie. food) down the pet’s throat. The potential contribution of acute pancreatic inflammation and the concern over stimulating the pancreas with food also fuels the fasting paradigm. Following the period of fasting, small quantities of a “bland” diet are gradually introduced as we hold our breath hoping the offending etiology has passed. A somewhat more scientific justification for a period of fasting would be the concern over antigen exposure in the gut during a period of inflammation, potentially creating a “food allergy” where previously there had been none. With cats this approach can be problematic. For one thing, a high protein/low carbohydrate diet does not fit the usual definition of a “bland” diet. The canine bland diet contains a small amount of highly digestible protein, a low fat content, and moderate to large amounts of highly digestible carbohydrate (ie. white rice). In addition, cats frequently can be anorectic for several days before their owner’s realize what’s (not) happening and present them to the veterinarian, and anorexia in a cat can have much more severe consequences than anorexia in a Labrador retriever. Not feeding a cat for 24 hours is still considered a viable way to “rest” the GI tract in cases of acute gastroenteritis, but the clinician must be aware of the likelihood that the clock on that 24-hour window may well have already run out by the time the patient is in your office.

Several recent pharmaceutical advances are of tremendous benefit to the cat with acute gastroenteritis, and the clinician attempting to care for that patient. Metoclopramide still may have a place as a pro-motility agent in the cat, but it has largely been replaced by cisapride (5mg per cat, two to three times daily) for that function. The pharmacology of the cat’s emetic center is simply not amenable to metoclopramide as an effective feline anti-emetic. Fortunately, ondansetron (0.5 mg/kg IV or PO once daily) and maropitant (1mg/kg daily, subcutaneously or orally – 1/4th of a 16mg tablet) appear to be very effective anti-emetics in the cat. So if needed, we can stop the cat with acute gastritis from vomiting. What about getting them to eat? Cyproheptadine (2-4mg per cat, once or twice daily) has long been used as an appetite stimulant in cats, with variable success. More recently, mirtazapine (1/4th of a 16mg tablet once daily, reduce the dose in cats with chronic kidney disease) has been shown to be an effective appetite stimulant in many cats, and may have some anti-emetic properties as well. Contrary to the original dosing information (every 3 days), research by Dr. Quimby at Colorado State University has shown that the pharmacokinetics of mirtazapine in cats would require daily administration of the drug

for full effect. It appears safe to mix and match the various anti-emetics and appetite stimulants, and the most effective combination will likely differ for different patients.

Finally, if a feline patient at CSU is approaching 48 hours without having been convinced to take on nutrition voluntarily (or with the help of pharmaceutical intervention), we will move relatively quickly towards “assisted feeding” through either a nasoesophageal feeding tube (liquid diet such as CliniCare at 1 kcal/ml, or the human product Ensure, also 1 kcal/ml), or quite frequently, an esophageal feeding tube (E-tube) with a blenderized diet, particularly if we are trying to get the cat out of the hospital.

Dietary intervention for acute gastritis in cats:

- High quality protein
- Highly digestible diet (>90%), single ingredients, no additives or flavorings
- Moderate energy density, small amounts of highly digestible carbohydrate
- High moisture content
- Fat for palatability
- 3-4 meals/day

The quality of the protein source in the diet is perhaps the single key ingredient for the successful passage and placation of an inflamed feline GI tract. Any poor-quality, undigested protein enters the colon as food for the bacterial microbiota that reside there. This may result in a change in the quantity and quality of the colonic bacterial population (“there goes the neighborhood”), stimulates the secretion of water into the GI lumen, and increases the amount of ammonia produced and thereby further damages an already diseased GI mucosa. In short, exacerbates both the feel (softer) and smell (bad) of the problem (diarrhea).

Food responsive diarrhea, a chronic enteropathy of cats

The veterinary profession (with the persistent prodding of pet food companies) is expanding the clinical definition (a bit faster than our basic understanding) of the impact diet has on gastrointestinal disease. Even the language is evolving to acknowledge the fact that diet plays a role in GI health well beyond the simple classification of allergy or intolerance. Cataloging dietary components as a cause or contributor to GI disease has evolved from “It’s the beef” to looking at the potential role of grains, gluten, preservatives and preparation. Prescribing dietary intervention as a contributor to the cure for GI disease has evolved from single-source Lamb & Rice to diets incorporating most any creature on the planet, exotic vegetables, prebiotics, probiotics, a spectrum of digestibility, combinations of fibers and various volumes of fat, essential ingredients as well as essentially eliminated ingredients.

Dr. Guilford and many others have continued to contribute strong research evidence for the impact of diet as both the cause and potential cure for GI conditions. Several key take-home points from this effort are:

- A significant percentage of cats with GI disease will respond favorably, if not completely, to dietary intervention
- A diet trial for a gastroenterologist lasts about 2-weeks, compared to the 8-12 week effort for a dermatologist
- The standard dietary intervention remains the hypoallergenic/hydrolyzed diet
- A much more diverse array of dietary options should be considered
- Sometimes it is a matter of matching a specific diet with a particular patient, especially with cats

Fiber

- Non-digestible plant carbohydrate

Soluble, fermentable fiber (ex. beet pulp) is easily broken down by GI bacteria into short-chain fatty acids (SCFA), an essential nutrient for repairing and maintaining a healthy GI mucosa. Soluble fiber will also slow down digestion, delay gastric emptying, inhibit absorption of nutrients and cholesterol, slow GI transit time, increase fecal water content, and shift the microbial balance towards “healthy” bacterial species (*Lactobacilli* and *Bifidobacter*) from unhealthy species (*Clostridium* and *E coli*).

- Oatmeal, oat cereal, lentils, apples, oranges, pears, oat bran, strawberries, nuts, flaxseeds, beans, psyllium, carrots
- Metamucil: psyllium, 1/8th – 1/4th teaspoon twice a day

Insoluble, poorly fermentable fiber (ex. cellulose) adds bulk to the stool, and may help normalize motility and act as a laxative. Colitis is the GI condition that appears to be most responsive to this action, hence the proliferation of “fiber-responsive” diets. Fiber-responsive diets high in insoluble fiber should be avoided in cats prone to constipation (chronic kidney disease) or obstipation (megacolon).

- Whole wheat, whole grains, wheat bran, seeds, nuts, barley, brown rice, zucchini, broccoli, carrots, green beans, root vegetable skins
- Canned pumpkin: 90% water, 3% fiber, 1-2 teaspoons per meal

Summary

- Dietary intervention may not be the only therapy, but it must be a part of an effective plan
- It takes 3 strikes before a cat is out; even a different version of a diet-type may hit the mark
- 2 weeks, not 12, or “Thank Heaven I’m not a Dermatologist!”, for a GI diet-trial

- Expand the definition of Dietary Intervention beyond Diets

References

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