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Nutritional Management of Gastrointestinal Disease

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The gastrointestinal (GI) tract is primarily responsible for acquiring and digesting food, absorbing nutrients and water, and expelling wastes from the body as feces. A proper diet and normally functioning GI tract are integral for the delivery of nutrients, prevention of nutrient deficiencies and malnutrition, repair of damaged intestinal epithelium, restoration of normal luminal bacterial populations, promotion of normal GI motility, and maintenance of normal immune functions (eg, both tolerance and protection from pathogens). The amount of food, its form, the frequency of feeding, and the composition of diet each have important effects on GI function and may be used to help ameliorate signs of GI disease. Although both nutrients and nonnutritional components of a diet are important to GI health, they also may cause or influence the development of GI pathology (eg, antibiotic responsive diarrhea, inflammatory bowel disease, dietary intolerance, or sensitivity and/or allergy). The appropriate diet may have a profound effect on intestinal recovery and successful management of chronic or severe GI disease.

A major consideration in choosing a diet to feed an animal with gastrointestinal (GI) disease is the digestibility of the nutrients. Typical maintenance pet foods have protein and carbohydrate (CHO) digestibilities ranging from 70% to 85% on a dry matter (DM) basis.[1] Pet foods formulated for dietary therapy of GI disease have CHO and protein digestibilities ≥90% (DM).[1] Therapeutic diets for GI disease also contain low levels of fat (eg, <15% DM in cats, and <10% to 15% DM in dogs), are lactose-free, and have reduced amounts of dietary fiber and other poorly digestible CHO. There are many different, highly digestible, therapeutic diets available. However, each formula is unique, and, thus, a different individual response can occur. Thus, if the animal does not respond to the diet as expected, choose another highly digestible diet with a completely different ingredient profile.

The amount of a diet fed should be calculated based on the energy needs of the individual animal. Although there is disagreement among nutritionists on the best equation for determining the energy requirements of sick animals, at the very least, the resting (or basal) energy requirements should be met.[2] In general, the equation recommended most commonly for this purpose is 70 x (body weight in kg)$^{0.75}$. However, if you need to use a linear equation in a pinch, the equation 30 x (body weight in kg) + 70 will approximate the values for the aforementioned exponential equation, as long the animal weighs more than 2 kg and less than 45 kg. Once resting energy requirements are determined, increase the kcal requirement by multiplying that number by an illness factor of 1.25-1.50 for cats, or 1.5-2.0 for dogs to account for the animals increased energy needs.

The next aspect to consider concerning the diet is meal size, frequency, and consistency. Generally, small meals (eg, <1/3 stomach capacity) are fed several times per day (eg, 3 to 6 meals). The feline stomach has a smaller capacity (approximately 60 mL/kg) and is less distensible than the stomach of a dog (capacity near 80 to 90 mL/kg), which is designed for more storage.[3] Feeding small meals more frequently reduces gastric distension, decreases gastric acid secretion, and may reduce nausea, vomiting, and gastroesophageal reflux.[1,4] Furthermore, the larger the volume of food ingested, the less that can be effectively assimilated. In general, liquid diets empty faster from the stomach than canned foods, and canned foods empty faster than dry.[1] Thus, if liquid diets are fed too fast or in large volumes, diarrhea will occur. In veterinary medicine, liquid diets are primarily used in specialized circumstances (eg, nasoesophageal or jejunostomy tube feeding) or with certain GI conditions, such as esophageal stricture, selected cases of achalasia, or gastric outflow disturbances, to reduce regurgitation or vomiting.

Nutrient Composition

Although a variety of nutritional and nonnutritional diseases affect the GI tract, the treatment of most GI diseases is enhanced by appropriate diet selection. Numerous therapeutic diets are available for the treatment of GI disease, including highly digestible diets, novel antigen or hypoallergenic diets, hydrolyzed (protein) diets, and diets with added concentrations of dietary fiber. Each of these diets may be used for the treatment of various GI disturbances. However, recognizing and understanding the differences in the nutrient composition of these diets is necessary to select the most appropriate diet. Finally, in special circumstances, homemade diets may be required for the successful dietary treatment of severe GI disease, when the available commercial products are either unacceptable or ineffective.

Protein

The effects of protein on the GI tract are subtle and often less clinically obvious than that of fat or CHO, but they are crucially important to disease treatment because the amino acid glutamine is the primary source of respiratory fuel for enterocytes.[5] The presence of a protein meal in the GI tract increases lower esophageal sphincter pressure, is a potent stimulus for secretion of GI hormones, including gastrin and pancreatic hormones, and increases gastric emptying and intestinal tran-

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sit.[1,4] Despite this, protein malassimilation is not a major stimulus for diarrhea (ie, dogs with protein-losing enteropathies (PLE) often have normal stools, unless they have concurrent fat or CHO malabsorption). However, intact protein reaching the distal small intestine and colon will increase bacterial ammonia production, alter bacterial numbers and species, and may contribute to colitis or colonic hypersensitivity.[6] Furthermore, protein antigens in food are responsible for the development of most food hypersensitivity reactions in dogs and cats.

Food allergies are caused by one or multiple food proteins, which make potent antigens when they are exposed to GI mucosa. Animals with food allergies may have an immediate hypersensititivity reaction to the protein, or may have a delayed (ie, type IV) response, thus the signs may be quite variable.[7,8] Nevertheless, a combination of intestinal and/or dermatologic signs, including vomiting, diarrhea, weight loss, pruritus, hair loss, or otitis externa, often develop in animals with food sensitivity.[9] In animals with GI diseases causing severe mucosal disruption (eg, inflammatory bowel disease [IBD], lymphoma), intact proteins may cross the mucosa–exposing the immune cells of the lamina propria to these antigens, and potentially predisposing to the development of hypersensitivity to that protein. For that reason, feeding a "sacrificial" diet during the initial stages of therapy of severe GI disease until the inflammation is controlled is sometimes recommended.[10] Once the inflammatory disease is suppressed with steroid or other immune suppressive drugs, a new highly digestible or novel antigen diet is introduced and fed as long-term therapy. One alternative to the sacrificial diet is to feed a hydrolyzed diet (eg, a highly digestible, low fat diet that contains no intact proteins, only peptides <15,000 Daltons (D), which in theory are not large enough to serve as antigens) (Fig 1). However, there are no data available to support or refute this claim. Although feeding hydrolyzed diets may not completely eliminate the possibility of immune stimulation, anecdotal evidence suggests that feeding these diets may be beneficial for some animals with severe small intestinal disease or food sensitivity.

Ultimately, the key to the successful treatment of dietary sensitivity and its associated clinical disease is to feed the affected animal a novel protein source that does not stimulate an immune response in that animal. Obviously, this may be easier said than done, requiring an appropriate diet that meets the pet's nutritional needs for maintenance, while also serving as a therapeutic diet for correction of the GI and allergic signs. As a reminder, when a food trial is used to diagnose a food allergy, a minimum of 6 to 8 weeks on the new (novel) diet is required to determine its effectiveness: 2 to 4 weeks for the inflammatory response initiated by the old proteins to subside and another 3 to 4 weeks with the new protein "on board" to see if the clinical signs resolve.[11] It is also worth repeating that it is essential to have a good dietary history to determine which protein sources have been previously fed, and it is equally important to be sure the owner understands the importance of not feeding any other foods, treats, or even flavors to the pet during the trial period.

Fats and Fatty Acids

Generally, the higher the density of nutrients, the slower the food empties from the stomach. This result is primarily because most nutrient dense foods are higher in fat, which slows gastric emptying in dogs and humans, but not cats.[1,3] In contrast to the effects of protein, increased levels of dietary fat decrease the tone of the lower esophageal sphincter and may lead to an increased risk of gastroesophageal reflux or vomiting.[1,4] Because digestion and absorption of fat is a complex process, malassimilation of fat in animals with GI disease is common. Undigested fats or fatty acids reaching the distal ileum or colon may be fermented by bacteria (especially non-beneficial species), resulting in the formation of pro-inflammatory and prosecretory hydroxy fatty acids. These hydroxy fatty acids may be injurious to the mucosa or can be a cause of osmotic diarrhea.[1,12] Nevertheless, the complete absence of fat from the diet is undesirable and may lead to a deficiency of essential fatty acids. Essential fatty acids must be supplied to provide the phospholipid and cholesterol building blocks for cellular growth and repair, which are especially important in the GI tract for the synthesis of new mucosal epithelial cells. Dietary fat enhances the palatability and acceptance of food, and is an essential energy source in sick or injured animals that cannot effectively use CHO during periods of stressed starvation.[2]

Recently, attention has been focused on the addition of omega 3 fatty acids (ie, fish oils) to diets for the anti-inflammatory therapy of GI disease. In humans with ulcerative colitis, addition of fish oils to the diet resulted in a reduction in inflammatory mediators, improved mucosal function and fluidity, and the reduced usage of anti-inflammatory drugs to maintain control of the disease.[13] The clinical benefit of fish oils for the treatment of inflammatory skin disease in dogs is well recognized. However, studies specifically assessing a reduction of intestinal inflammation in dogs and cats fed omega 3 fatty acids are lacking. Nevertheless, several pet food manufacturers have added omega 3 fatty acids to their therapeutic diet formulations to provide anti-inflammatory potential to their highly digestible and hypoallergenic diets.

CHO

There are no requirements for CHO in the diet of dogs or cats. The CHO present in pet foods are primarily plant starches such as rice, potato, corn, wheat, barley, and oats.[14] Another type of CHO present in some diets are the α-linked polysaccharides, which include the dietary fibers (eg, complex CHO not readily broken down by mammalian enzymes). CHO digestibility is determined by its origin and the degree of cooking (eg, rice and wheat are generally highly digestible, while uncooked maize or potato starch are less digestible).[14] Malassimilation of CHO results in the development of osmotic diarrhea, production of increased intestinal gas (flatus), loss of water and electrolytes, increased bacterial fermentation in both the small intestine and colon, overgrowth of pathogenic bacteria, and acidification of the colonic luminal environment promoting formation of hydroxy fatty acids and other potentially toxic intermediates.[4,14] White rice is the best CHO source for most dogs with GI disease and is found in most commercial, therapeutic highly digestible diets because it is gluten free (some dogs, especially Irish setters and soft coated Wheaten terriers, are sensitive to gluten or a sensitivity to gluten may develop, which is present in wheat, oats, and barley), readily digestible, and nonantigenic.[1,4,14,15] Other gluten-free CHO sources include potato, corn, and tapioca. Potato and tapioca are less digestible, and corn may be antigenic to animals prone to developing food sensitivity.

Dietary fibers are a large, complex group of CHO that include starch and nonstarch polysaccharides found in plants. They are readily digested by bacterial enzymes but less well digested by

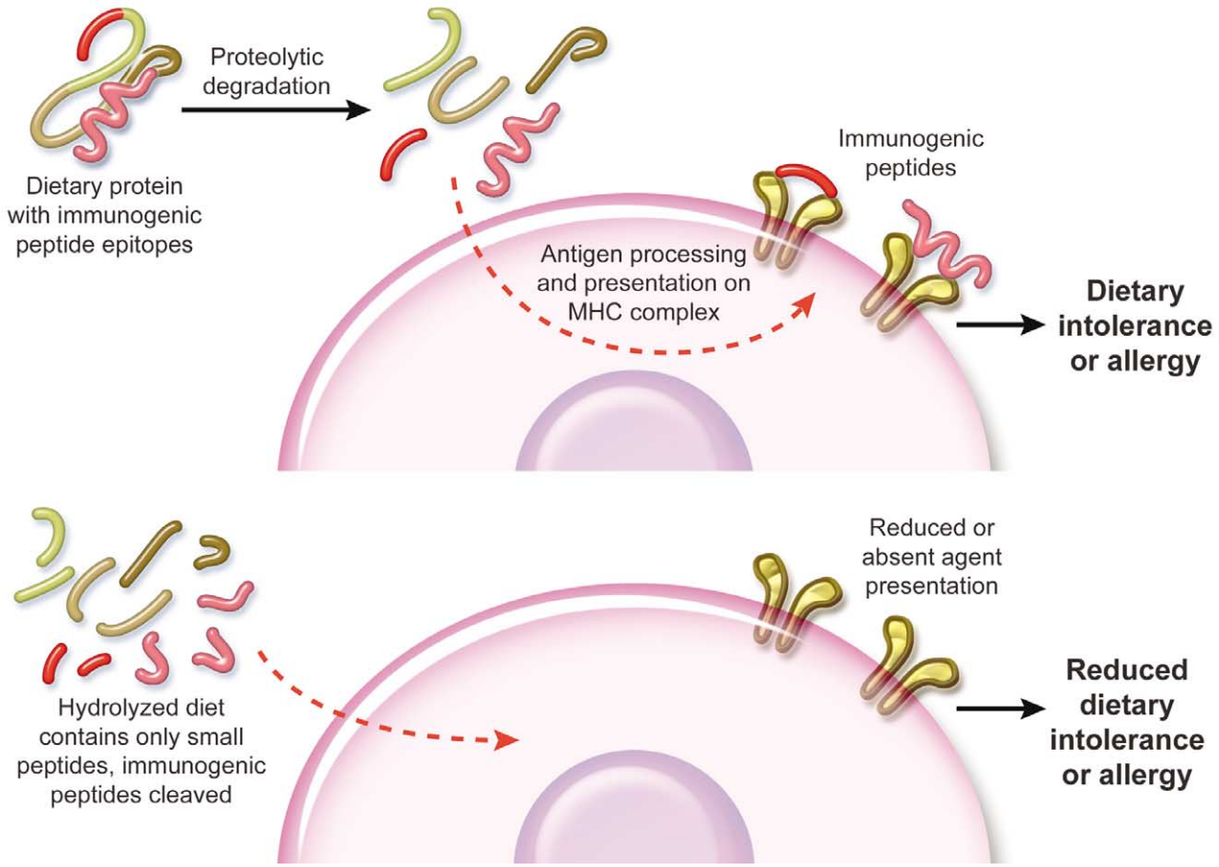

Fig 1. Theoretical mechanism of action for hydrolyzed diets. Normal dietary protein (upper panel) is degraded into peptides, some of which are immunogenic. These immunogenic peptides are taken up by antigen processing cells in the gastrointestinal mucosa and presented on the major histocompatibility complex (MHC), leading to immune reactions, dietary intolerances, or allergy. In the partially hydrolyzed diets (lower panel), the dietary protein has been partially degraded before consumption, with the proteins broken down into small peptides that are not processed and presented as effectively, thus reducing immune response against the diet.

mammalian digestive enzymes.[14] Traditionally, fibers were classified as soluble (highly fermentable) or insoluble (poorly or nonfermentable) fibers based on their digestion by amylase.[16] However, a physiologically relevant classification, based on their activity in the GI tract, is currently recommended. Fibers are soluble if they form gels in solution (thus attracting water), delay gastric emptying and slow intestinal transit, inhibit the absorption of cholesterol and some other nutrients, are poor bulking agents, are highly fermentable in the colon (eg, increase numbers of bacteria and increase short chain fatty acids, especially butyrate, an essential colonic fuel source), acidify the lumen, and stimulate colonic cellular proliferation.[14,16,17] Examples of soluble fibers include fructooligosaccharides (FOS), pectins, psyllium, oats, barley, guar gum, fruits, and some legumes.[14] Alternatively, insoluble fibers do not form gels, have no effect on gastric emptying, increase or "normalize" intestinal transit, have no effect on nutrient absorption, are good bulking agents (eg, dilute colonic content, and, thus, bind noxious agents in the colon), are fermented less, so produce fewer short chain fatty acids, and increase fecal weight.[1,14,16] Typical examples of insoluble fibers are cellulose, wheat and rye fibers (most cereal fibers), and the woody parts of plants (eg, lignins).[14]

FOS are present in a variety of fruits, vegetables and grains, and behave in the GI tract like soluble fibers. These sugars have generated considerable interest in human and veterinary medicine because they are preferentially fermented by beneficial bacterial species (eg, *Lactobacillus, Bifidobacterium*) and prevent the growth of pathogenic species.[18] In studies of humans with IBD or colitis, adding FOS to the diet highly improved the response to therapy, clinical disease was reduced, and relapses were fewer.[19] There have been only a few studies in dogs, and even fewer in cats, evaluating the role of FOS in the dietary therapy of GI disease. However, preliminary evidence supports the finding in humans that FOS increases the numbers of beneficial bacteria in the colon of dogs and cats, and may prove beneficial in controlling bacterial overgrowth, antibiotic responsive diarrhea, or other inflammatory diseases suspected to have a bacterial origin (eg, IBD).[17,20]

Specific Aspects of Nutritional Therapy of GI Disease

There is a diverse and increasingly complex array of commercial diets available for the treatment of GI disease. These diet types range from highly digestible (low residue or bland) diets used for all manner of GI upset, to hypoallergenic (novel, limited antigen, or single source protein) diets used for dietary intolerance or allergy, to hydrolyzed diets used for many GI disturbances and intolerances, and finally to fiber diets, which may contain increased concentrations of insoluble or mixed dietary fibers. Although some data support the premise that

specific diet types are beneficial for particular GI disturbances (eg, low fat diets for lymphangiectasia), the majority of dietary recommendations are based on clinical experience, anecdotal evidence, and pathophysiologic theory. Thus, these recommendations must be implemented with caution, used along with continued and careful assessment of the patient, and based on an understanding of the nutritional needs of the individual patient.

The Role of Anorexia and Food Aversion

Anorexia or inappetence is the absence of intake due to prolonged or inappropriate satiety.[1] There are many causes of anorexia, including behavioral (eg, threatening environments), inflammatory, infectious or neoplastic diseases, eating is associated with pain (eg, oropharyngeal or esophageal diseases such as dental disease or esophagitis) or severe nausea, which may be peripheral or central in origin, or secondary to vestibular disease. Although anorexia is the major reason for decreased food intake, dysphagia, poor dietary acceptability, and food aversion are also important considerations in animals that do not consume their expected intake.

Poor food palatability will often result in poor acceptance of a food, especially in cats. The properties of a food that influence palatability are (1) smell; (2) texture; (3) temperature; (4) pH; and, to some degree, (5) form.[4] Cats are especially sensitive to these characteristics, with the highest influence on palatability being the fat and protein content of the food, as well as the temperature (eg, food served at body temperature is preferred). In dogs and cats, animal digests, monosodium glutamate, and nucleotides appear to enhance the palatability of foods.[21] Food aversion may develop when ingestion of a food is associated with nausea or vomiting, and, in cats, this phenomenon can occur following force feeding. In dogs, food aversion is typically associated with a reduced or finicky appetite, while, in cats, food aversion often results in complete refusal to eat.

The treatment of anorexia or inappetence obviously begins with identification and, if possible, correction of the primary cause. Symptomatic therapy (eg, fluid therapy, correction of acid base or electrolyte imbalances, and control of vomiting) is used to control other factors that may influence food intake and response to treatment. For example, in dogs or cats refusing to eat postoperatively, administration of appropriate analgesics may remove the cause of inappetence (eg, pain) and result in the return of appetite. For animals with anorexia caused by delayed gastric emptying or ileus, the use of prokinetic drugs, such as metoclopramide or ranitidine, may be beneficial. Where feasible, both reductions in environmental stressors as well as providing a highly palatable dietary source are important to provide the optimal situation for improving intake. In summary, food acceptability can be improved by adding flavor enhancers (eg, beef or chicken broth), increasing the fat or protein content, if possible, varying the texture or presentation of the food, and warming the food offered to body temperature.

In animals that are able but still refuse to eat, appetite stimulants can be tried. Benzodiazepine derivatives (eg, diazepam 0.1 mg/kg intravenously and oxazepam 0.5 mg/kg orally once daily) will often produce a short lived increase in appetite in most cats and some dogs.[1] Cyproheptadine (0.2 to 0.5 mg/kg orally), an antiserotonergic, antihistamine drug, is the preferred appetite stimulant drug for cats. However, if the nutritional support is likely to be required for more than 3 to 4 days, or if these efforts fail to restore appropriate intake, enteral feeding via a feeding tube (ie, placement of an esophageal or gastric tube), or intravenous feeding, should be instituted.

Diets for Gastritis and Small Bowel Disease

For acute gastroenteritis in dogs and cats, the standard dietary recommendations for many years have included fasting for 24 to 48 hours, followed by feeding small amounts of a "bland" or highly digestible diet 3 to 4 times a day. This approach is well accepted, but the idea of complete fasting has come into question in recent years, especially for acute diarrheal disease. The idea of fasting was to provide "bowel rest." However, this may not be optimum for all causes of acute gastroenteritis. It is well known in humans that providing small amounts of bland food often reduces nausea during acute gastroenteritis episodes and improves diarrhea in many cases of acute diarrheal diseases. Thus, complete restriction of food may be reasonable for a short period, but an early return to appropriate oral intake is strongly recommended, unless the problem specifically requires nothing per os to prevent further disease exacerbation (eg, pancreatitis).

An ideal diet formulated for gastritis or small bowel disease should be highly digestible, which, as mentioned previously, contains readily available protein and CHO sources, is lower in fat than regular maintenance diets, contains fewer or no ingredients known to be associated with food intolerance (eg, lactose, gluten), is hypoallergenic, and contains generous amounts of electrolytes (eg, potassium, magnesium) and vitamins (both water soluble and fat soluble). The goal of feeding highly digestible diets is to reduce the likelihood of CHO, fat, and protein malabsorption (and the associated diarrhea, gaseousness, increased colonic ammonia production), and to make fewer nutrients available to the bacteria that populate the distal small intestine and colon, which can lead to bacterial derangements or overgrowth. There are a wide variety of these controlled, highly digestible diets available commercially, and each is formulated with slightly different protein and CHO sources, different levels of fat, and the presence or absence of other added nutraceuticals, such as FOS or omega 3 fatty acids. The true effectiveness of these diets and their individual additives has not been rigorously investigated. But, as with the use of short-term fasting, this dietary approach has been very effective in reducing or halting the clinical signs in many animals with acute or chronic GI distress.

There is very little information available on the best diet for ulcerative or inflammatory gastric disease in dogs or cats. In general, frequent small meals, liquidizing the diet, and reducing the fat content of the food have all been recommended to minimize gastric acid secretion and hasten gastric emptying.[1] Although high fat meals will decrease gastric emptying, lipid in foods, such as cottage cheese, provides an intragastric lipid emulsion that improves the hydrophobicity of the gastric mucosal barrier.[22] In general, the commercially available, highly digestible canned diets are acceptable for most pets with gastritis or gastric ulcer disease because they have modest levels of protein, low concentrations of fat, and empty from the stomach more readily than dry kibbled foods. It is important to remember that liquid, CHO based diets have a very rapid emptying rate from the stomach and, as such, may cause "dumping" syndrome, which results from the accelerated presence of hyperosmolar chyme emptied into the duodenum

The therapy for acute small bowel diarrhea is as for acute gastroenteritis, with the principles of providing a short-term fast (ie, 24-48 hours) followed by feeding a highly digestible diet for 3 to 5 days being the long accepted, and generally effective, dietary approach. However, "feeding through diarrhea" using oral, food based (eg, rice) rehydration solutions has frequently been used in humans, because they reduce stool volume and shorten the course of diarrhea compared with glucose based solutions or treatment with nothing per os.[23] However, one important difference is that in many humans with diarrhea, the diarrhea is secretory, caused by cholera or other toxicogenic organisms that activate cyclic adenosine monophosphate or cyclic guanosine monophosphate mechanisms. In humans with severe cases of diarrhea or in diarrhea due to viral diseases (eg, rotavirus), which cause an osmotic diarrhea, this form of therapy is less effective.[24] Both osmotic diarrhea (eg, due to parvovirus, coronavirus) and diarrhea due to dietary indiscretion are more common in dogs and cats than secretory diarrheas. Thus, oral rehydration solutions or "feeding through the diarrhea" may not be the best choice in these cases. Nevertheless, it is still important, even in cases of osmotic diarrhea, to not withhold food more than 3-5 days because the development of GI mucosal atrophy and intestinal ileus are both major contributors to ongoing morbidity in animals.[25]

In dogs or cats with chronic small bowel diarrhea, the same principles of using highly digestible diets for dietary intervention that are recommended for pets with acute diarrhea apply to feeding patients with chronic diarrhea. However, adjustments in the diet chosen may have to be made based on the specific problem, the severity of the disease, or ability to find a diet that the animal will eat. If possible, choosing a protein source not commonly included in the animal's usual diet may be advantageous because it reduces the possibility of feeding a protein to which the animal is allergic.[11] Protein sources that are often acceptable for dogs include cottage cheese, tofu, eggs, turkey, venison, or rabbit.[1,11] Cats are less likely to consume cottage cheese and should not eat tofu long term (plant based protein does not contain the necessary amino acids for cats) but will often do well on turkey, fish, venison, rabbit, or liver in small amounts and not as whole meat source.[1,11]

There are many commercial diets that provide single source, novel protein in their formulations, are highly digestible, and often are very acceptable choices for the long-term treatment of chronic inflammatory or infectious GI diseases. The key is to choose these diets based on a diet that represents a novel protein source for that animal, and results in resolution of the clinical signs. In most patients with chronic small bowel diarrhea, fat malabsorption is a significant problem. Thus, the most effective diets are low in fat. Most commercial diets have formulated their foods to reduce the concentrations of fat for the treatment of GI disease. However, the level of fat in these diets varies widely. (Consult the commercial product guide to determine and compare the amounts of fat in each diet.) In those patients who have fat malabsorption as a significant problem, it is essential to choose diets with the lowest levels of fat (eg, preferably $<$10% fat DM) or to provide ultra low fat homemade diets (eg, turkey or lean venison and rice). If a homemade diet is chosen, the diet must be supplemented with a small amount of vegetable oil and a human multivitamin to prevent the development of nutritional deficiencies.

Protein-losing enteropathies (PLE) are a group of severe intestinal diseases that occur as idiopathic (eg, lymphangiectasia), familial (eg, soft coated Wheaton terriers, Basenjis, Irish setters), or secondary enteropathies (infectious, neoplastic, or inflammatory diseases that infiltrate the GI mucosa).[1,4,15,26] The end result is a loss of mucosal function, including malassimilation of nutrients, especially fat, intestinal protein loss due to leakage across the abnormal barrier, and the loss of mucosal transport functions, and development of concurrent motility disturbances.[27] Regardless of the cause, nutritional therapy is an essential aspect of the successful treatment of PLE. In mild forms of PLE, feeding a highly digestible, low fat diet, in addition to specific therapy for the primary disease, may be sufficient. However, when severe intestinal disease results in the significant loss of serum proteins (serum albumin $<$1.25 g/dL), the subsequent development of mucosal edema causes further nutrient malassimilation. In these patients, hydrolyzed diets or elemental diets (eg, diets containing no intact nutrient sources) may be required for any nutrient absorption to occur.

In animals with severe PLE, a combination of parenteral and enteral nutrition may be required to replace lost oncotic proteins, resolve gut edema, and to correct protein-calorie malnutrition. Once serum albumin levels are more stable (ie, $>$1.75 g/dL), ultra-low fat diets containing intact proteins may be tolerated. However, some dogs with PLE may require the feeding of ultra-low fat homemade diets (eg, nonfat cottage cheese, egg whites, rice, cooked potatoes), or combinations of hydrolyzed, ultra-low fat, or elemental diets indefinitely.[10] It has previously been recommended to feed the commercial ultra-low fat/high fiber diets designed for weight loss to GI patients that need ultra-low fat diets. However, these diets have 2 major disadvantages that limit their usefulness for the treatment of pets with these severe GI diseases: (1) they are too energy restricted, especially in patients with PLE or severe IBD that need to regain weight or need increased protein to make up for protein losses; and (2) the increased concentrations of insoluble fiber present in these diets limit the availability of these nutrients for digestion and decreases the digestibility of the nutrients in an animal that already has issues requiring maximal nutrient availability.

Colitis, Constipation, and Other Disorders of the Large Intestine

In general, for diseases of the large intestine in dogs, addition of dietary fiber has been the mainstay of dietary management due to the wide variety of factors that are influenced by these complex CHO.[1,16] The amount and type of dietary fiber that is best for promotion of bowel health, maintenance of normal bowel motility, and determination of appropriate fecal characteristics are sources of considerable debate. This is best illustrated by the array of commercial diets available for the treatment of colitis in dogs. Some are based strictly on the presence of increased concentrations of insoluble fiber (eg, Hill's Prescription Diet w/d [Topeka, KS] or Purina OM [Ralston Purina Co, St. Louis, MO]), others are based on mixed fiber sources (Purina DCO), and yet others have primarily soluble fiber sources (Iams Low Residue [The Iams Co, Dayton, OH]). Nevertheless, there is general agreement that dietary fibers enhance normal colonic function in dogs, primarily by providing fuel sources through fermentation of the more soluble fibers (eg, short chain fatty acids such as butyrate), and by increasing fecal bulk with the insoluble fibers, which promotes normalized colonic motor functions and defecation. In a recent study of dogs with colitis,

the addition of dietary fiber to treatment of the disease resulted in a significant clinical improvement over dogs fed diets without added fiber.[28]

There is much less data concerning the use of fiber and importance of dietary fiber in colonic inflammatory disease in cats. Most of the published information is based on extrapolation of data from dogs, and, because of the anatomic, physiologic, and nutritional differences of cats, some of these extrapolations may be quite in error. Witness the observation that many cats have increased episodes of constipation when placed on high insoluble fiber diets for the long-term treatment of obesity. This is especially true in cats that tend to be minimal water drinkers or those that are marginally dehydrated due to polyuria (secondary to renal disease or diabetes). Thus, some caution is advised in this area as it relates to using high insoluble fiber diets for the long-term treatment of colonic disease in cats.

A good general rule to remember is that fecal incontinence and disorders of the anorectum are often best treated with low fiber diets, as increased fiber only adds bulk in a colon that already cannot deal with the presence of feces–so the best approach is to minimize the amount of fecal material by feeding a highly digestible diet. Alternatively, high fiber diets can be used to prevent constipation, as long as the colon is functional (eg, not in cats with idiopathic megacolon or colonic atony due to electrolyte imbalance), there is no obstructive disease (eg, pelvic fractures, neoplasia, or strictures), and the patient is well hydrated. High insoluble fiber diets cannot be used to treat constipation. They will simply increase the fecal mass and hardness, primarily due to the colon's impressive ability to remove water from the feces. Finally, in animals with permanent functional or structural disorders of the colon, feeding highly digestible diets, which minimize the fecal mass and maintain a softer fecal stream that is easier to pass, will provide the highest opportunity for the successful dietary treatment of the condition.

Borgorygmus and Flatulence

Intestinal gas is produced from a variety of sources without and within the GI tract, including from ingestion of air, production of gases in the bloodstream that cross the mucosal barrier into the lumen, and, finally and most importantly, from production of gases by bacterial degradation of unabsorbed nutrients. In most cases, excessive gas production occurs due to dietary indiscretion, malassimilation of the normal diet (either due to GI disease or from maldigestion from lactose intolerance), ingestion of spoiled foods, or foods high in protein or fat. In most cases, foods high in insoluble fiber do not cause an increase in intestinal gas because they are not highly fermented, but diets with mixed fiber sources or higher concentrations of soluble fibers can cause higher gas production because the bacteria ferment the fiber. The best dietary approach to reduce the formation of intestinal gas is to reduce the substrate available to the bacteria in the colon. Feed highly digestible diets that result in a reduction of available substrate for the bacteria to act on. If this approach is not helpful, the patient should be evaluated carefully for other causes of malassimilation.

Intensive Nutritional Support for Severe GI Disease

For the majority of patients with GI disease, oral enteral nutrition using one of the major types of dietary approaches will be sufficient to provide appropriate nutritional support for the patient, while not overburdening the GI tract. However, there will be occasions when more aggressive nutritional therapy, using either the placement of feeding tubes or by intravenous feeding methods, will be necessary to provide nutrition for the patient. The most common circumstances requiring intervention are animals with persistent vomiting or other obstructive processes that preclude the use of the GI tract until the problem can be solved. Examples include GI lymphoma, gastric outflow obstruction caused by, for instance, fungal or neoplastic disease, or animals with severe pancreatitis requiring upper bowel rest. The general rule of thumb for decision making in these animals is that under circumstances of stress starvation, the pet should not be allowed to go without food for longer than 3 to 5 days.[29] If this circumstance is occurring, then aggressive intervention is not only warranted for nutritional management, it is also essential for survival. Because it is beyond the scope of this article to review the approach to nutrition of these critically ill patients, the reader is referred to several recent reviews on the subject for additional information.[29,30] However, for those interested in maximizing their patient care, a few simple rules are offered:

1. Use the most cephalad part of the GI tract that is functioning when selecting the route for enteral nutrition (eg, if the jaw is broken, place an esophageal tube, if the dog has megaesophagus, place a gastrostomy tube).
2. If the gut is not working or the patient is not stable enough for the placement of tubes for enteral support, provide intravenous nutritional support at the level of your ability–at the very least, peripheral parenteral nutrition should be possible in most small animal practices.[30]
3. Calculate caloric needs, and select diets based on the patient's primary problem. Do not guess calories and then feed a high calorie recovery diet (eg, Hill's a/dor Iams Maximum Calorie) because the GI tract may not be able to tolerate the high fat content.
4. Start slowly, feed small meals frequently, and work up to the full caloric needs of that animal over several days.
5. If necessary, combine enteral and parenteral nutrition together if the GI tract is unable to tolerate (eg, diarrhea, vomiting, or discomfort develops) full enteral feeding.

Nutritional Deficiencies Resulting from GI Disease

Nutritional deficiencies commonly occur as a consequence of GI disease.[31] Protein and calorie malnutrition is the most common nutritional deficiency in severe or chronic GI disease. Not surprisingly, deficiencies of electrolytes (eg, sodium, potassium, chloride, and bicarbonate) and divalent cations (eg, magnesium, zinc, and calcium) are also common, and should be corrected.[31] A variety of vitamin deficiencies may also occur as a result of severe intestinal disease. Deficiencies of B vitamins, especially cobalamin, and some of the fat soluble vitamins (E and K) are the most common and clinically important micronutrient deficiencies recognized in dogs and cats.[1,4,31] Cobalamin deficiency alone, especially in cats, can lead to the development of GI dysfunction and diarrhea, thus, the assessment of cobalamin status in cats with GI disease is very important.[32] Furthermore, deficiencies of B vitamins can occur very quickly in cats that are anorectic or in those with severe GI disease

because they have a 4-times higher need for thiamin, niacin, pantothenic acid, and pyridoxine than do dogs.[3] Thus, parenteral supplementation in cats with GI disease or anorexia is very important to prevent these deficiencies. Little is known about deficiencies of microminerals. However, it is reasonable to assume that copper, selenium, zinc, and others may be affected, and thus corrected by nutritional support.

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