Formulating an initial treatment plan for gastrointestinal disease

A nutritional plan must be formulated for nearly all patients with primary gastrointestinal (GI) disease. Location of the major clinical problem (i.e. esophageal, gastric, small bowel, large bowel) and chronicity can help a clinician determine the most appropriate diet selection. A dietary plan will vary based on the primary problem however the underlying condition may not always be identified. Therapeutic GI diets can be divided into three main categories: highly digestible, novel/hydrolyzed protein, and high fiber. Further, some of these diets may be low in fat which may also benefit patients with GI disease. In general, first line therapy in patients with upper GI diseases (i.e. esophageal to small intestinal) will involve a highly digestible diet and with chronicity may move to a novel/hydrolyzed protein diet. In patients with lower GI disease (i.e. large bowel), a high fiber diet may be the first line therapy followed by novel/hydrolyzed protein diet.

Improvement of GI signs is usually seen rapidly in patients on an optimal diet for their GI disease. If clinical signs persist for > 2 weeks or worsen during this time, an alternative dietary strategy should be considered. GI disease is considered chronic when clinical signs persist for more than 3 weeks or are intermittently present for more than 3 weeks.1 The most common types of chronic enteropathies can be classified based on their response to therapy: food-responsive (including both food intolerance and hypersensitivity), antibiotic responsive, or steroid responsive. Approximately 50% of chronic enteropathy cases will be food responsive.2 In a more recent retrospective study, 64% of dogs with chronic enteropathy were food responsive, 33% antibiotic responsive, and 39% steroid responsive.3 Dietary options, particularly in patients with inflammatory bowel disease (IBD), can include the following strategies: global modification (switching to a different diet all together), optimizing assimilation (feeding a reduced fat, reduced fiber, highly digestible diet), antigenic modification (hydrolyzed/novel protein diets), or immunomodulation (i.e. omega-3 fatty acids, prebiotic fibers).2 Further, hypocobalaminemia has been reported in 36% of dogs and cats with chronic GI disease and should be supplemented in these cases or empirically.4,5

Highly digestible diets

Digestibility is defined as the percentage of a food stuff taken into the digestive tract that is absorbed into the body.6 The goal of a highly digestible diet is to maximize nutrient absorption and minimize stool production. Additionally, a highly digestible diet may decrease the volume of food required to meet energy requirements. The digestibility of a pet food can be determined using specific protocols found in the Association of American Feed Control Official (AAFCO) Official Publication. The terminology “highly digestible” is not defined by AAFCO, however products with a protein digestibility of ≥ 87% and fat and carbohydrate digestibility ≥ 90% may be considered highly digestible.7

Several factors can influence the digestibility of a diet including ingredient selection, heating and processing methods, and animal related factors. As the fiber content of a pet food increases, typically the protein and fat digestibility will decrease. The digestibility of starchy carbohydrates will improve with heating. Animal related factors include both breed and size for dogs. Large breed dogs tended to have higher fecal water contents, decreased stool quality and increased number of defecations than small breed dogs while differences were noted in certain breeds (i.e. Irish wolfhounds had lower fecal water content than Labrador retrievers).5 Age has also been shown to be a significant factor in macronutrient digestibility with increasing digestibility from 11 – 60 weeks of age in dogs.7 In this same study, large breed dogs had increased fecal moisture content and lower fecal scores than small breed dogs, but their nutrient digestibility was significantly higher demonstrating that decreased fecal quality was not related to lower nutrient utilization in these otherwise healthy dogs. Reduced protein digestibility is reported to occur in some geriatric cats10 Cats can efficiently digest and absorb carbohydrates. Seventy one percent of cats with chronic diarrhea responded with improvements in fecal scores to both a highly digestible, moderate carbohydrate and highly digestible, high-protein, low-carbohydrate diet with no difference between groups.11 Highly digestible diets are typically a first line therapy for dogs and cats with acute gastroenteritis and may also be useful in patients with GI diseases including chronic small bowel diarrhea, short bowel syndrome, megaesophagus, and post-operative GI surgery.

Low fat diets

Low fat diets can be found in all categories of GI diets, however most therapeutic GI low fat diets are highly digestible. While there is no standard cut off for “low fat” in cat or dog diets, typically the author regards diets containing <25%ME fat for dogs and < 35%ME fat for cats as low fat. Diets containing <20% ME fat are considered ultra-low fat for dogs. AAFCO defines “low fat” on an as fed basis using crude fat for both cats and dogs for dry, semi-moist, and canned diets. Fat reduction may also be relative to what the animal is currently consuming. Diets with a high fat content may be less ideal for some patients with GI disease as they can reduce the tone of the lower esophageal sphincter through gastrin inhibition and increased secretin and cholecystokinin (CCK) release, decrease gastric emptying through increased CCK, and may contribute to steatorrhea if excess fat passes into the colon.12-15 Low fat diets can
be important for the management of dogs with chronic vomiting, regurgitation, or protein losing enteropathies. Dietary fat restriction is efficacious in treating dogs with intestinal lymphangiectasia, particularly those unresponsive to prednisone treatment or with recurrent clinical signs and hypoaalbuminemia when prednisone is tapered.\textsuperscript{16} Low fat diets may be less important for the management of chronic GI disease in cats. Cats with chronic diarrhea had no difference in their response of fecal scores when placed on either a high fat (45.1\%ME) or a low fat (23.8\%ME) highly digestible diet with 78.2\% of all cats improving in their fecal scores and 36\% of cats developing normal stools.\textsuperscript{17}

**Novel protein/hydrolyzed protein diets**

Novel or hydrolyzed protein diets are typically used in patients with chronic GI disease to avoid food antigens that may lead to an immune response, lessen clinical signs of GI disease, and prevent relapse. A novel carbohydrate is also typically recommended for these patients as carbohydrate ingredient sources will also contain varying amounts of protein. Diagnosis of food hypersensitivity can only be determined at this time by food elimination trial with challenge. Salivary allergen-specific IgA tests are not recommended as validation studies are not available to determine their efficacy. Serum allergen-specific IgE tests are unreliable with one study reporting a 15.4\% positive predictive value.\textsuperscript{18} Twenty nine percent of cats with chronic GI signs were determined to be food sensitive through the use of a novel protein diet with dietary challenge finding 55\% of affected cats sensitive to more than 1 food ingredient.\textsuperscript{19} In dogs with food responsive chronic enteropathy, 55\% were on an elimination diet, 44\% on a hydrolyzed protein diet, and 1\% on a home cooked diet with no significant difference in outcome between dogs on an elimination diet or hydrolyzed protein diet.\textsuperscript{3} In another study evaluating long term efficacy of a hydrolyzed protein diet, 88\% of dogs with chronic small bowel enteropathy on both a hydrolyzed protein and highly digestible diet responded favorable in the first 3 months, however long term control was significantly better in the hydrolyzed protein group at 6-12 months and 3 years later.\textsuperscript{20} No prospective studies comparing the response to a novel protein diet versus a hydrolyzed protein diet are currently available in the dog or cat.

Therapeutic hydrolyzed protein diets may be advantageous for clinicians given the wide variety of ingredients available in over-the-counter (OTC) pet diets making selection of a novel protein diet difficult. Further, cross contamination of food antigens not listed on the label of OTC diets renders these diets inappropriate for food elimination trials and increases the potential number of antigen exposure for a pet. This cross contamination likely occurs at some point during the manufacturing process of the diet. Three of 4 OTC venison dry dog foods tested positive for beef or soy antigens with no beef or soy products in the ingredient lists.\textsuperscript{21} Another study found soy protein in 3 of 4 OTC diets claiming to contain no soy.\textsuperscript{22} Interestingly this same study found soy protein (>2.5ppm) in 4 of 7 therapeutic diets for food elimination trials with 2 containing a soy product on the label (soy protein hydrolysate). Soy-sensitized dogs have been shown to tolerate hydrolyzed soy protein with no clinical response after an oral challenge making hydrolyzed soy protein diets still a good option.\textsuperscript{23} When choosing a novel protein diet, one must have a thorough knowledge of dietary protein exposure and that the diet chosen only contains the protein listed. Even with home prepared diets, cross contamination can occur at the level of the butcher, supplier, or in the home of the owner preparing the food therefore careful selection of protein source, supplier, and preparation should be emphasized for these owners.

**High fiber diets**

While dogs and cats do not have a nutritional requirement for fiber, fiber can be added to the diet for maintenance and promotion of GI health. A high fiber diet is recommended as a first line therapy for cats and dogs with large bowel diarrhea or constipation. Fiber is a poor source of energy, therefore its inclusion in pet food will decrease energy density and digestibility. Fiber is expressed on pet food labels as a maximum percentage of crude fiber which reflects the percentage of cellulose (insoluble fiber) in the diet. Total dietary fiber more accurately reflects the fiber content of a pet food by including both soluble and insoluble fibers with the exception of certain types of fiber such as oligosaccharides. Fibers are classified based on their solubility (increased water-binding capacity to make a viscous solution), fermentation (ability of colonic microbes to produce short chain fatty acids), and prebiotic ability to support the growth and activity of health-promoting bacteria in the GI tract.\textsuperscript{24,25} Soluble fibers, which form a gel in the upper GI tract, will increase GI transit time, slow nutrient absorption, and increase fecal water content. Insoluble fibers increase fecal bulk, are moderately to slowly fermentable in the colon, and have no effect on transit time. Sixty three percent of dog with a history of chronic idiopathic large bowel diarrhea with limited success to other dietary strategies had an excellent response when started on a highly digestible diet with added soluble fiber (psyllium husk).\textsuperscript{26} A moderate fiber, psyllium-enriched, dry extruded diet was shown to be efficacious in cats with a history of recurrent feline constipation with 85\% improvement in all cats and a significant decrease in medical management.\textsuperscript{27} Prebiotic fibers, such as inulin, fructooligosaccharides, mannanoligosaccharides, and resistant starch, are selectively fermentated by commensal bacteria in the GI tract to produce short chain fatty acids that have beneficial effects on colonic health. Dietary fiber has also been shown to alter the microbiome of both dogs and cats.\textsuperscript{28,29} Cats and dogs that may benefit from a high fiber diet can be placed on a therapeutic commercial diet option or supplemental dietary fiber can be added to an existing diet.
References

Nutritional assessment of patients with chronic kidney disease
A complete nutritional assessment and staging of a patient’s chronic kidney disease (CKD) is required prior to making nutritional recommendations. The World Small Animal Veterinary Associations has guidelines to complete a nutrition assessment.\(^1\) Guidelines for staging and sub-staging recommendations are provided by the International Renal Interest Society (IRIS).\(^2\) When taking a diet and general history, the owner of a patient with CKD should be questioned about appetite, weight loss and time frame for weight loss, current diet, evaluation of current protein intake, treats or table foods, foods used for medication administration, and supplements. Evaluation of the patient should include body weight, body condition score (BCS), evaluation of muscle mass, and systolic blood pressure. Important laboratory parameters include creatinine, blood urea nitrogen (BUN), potassium, albumin, bicarbonate, urine specific gravity, and urine protein-to-creatinine ratio.

Adult maintenance diets versus therapeutic kidney diets
Kidney diets are amongst the oldest therapeutic diets for dogs and cats. The IRIS recommends nutritional intervention at IRIS stage 2 to control serum phosphate levels, however a diet change may be recommended for patients with IRIS stage 1 following a complete nutritional assessment. Double-blinded, randomized, controlled clinical trials have compared the efficacy of therapeutic kidney diets (TKD) to adult maintenance diets (AMD) in patients with ≥ IRIS stage 2 chronic kidney disease (CKD):

- Ross et al. fed cats with spontaneous IRIS 2 and 3 CKD a TKD (n = 22) or an AMD (n = 23) for 24 months.\(^3\) No cats in the TKD group experienced a uremic crisis and no kidney-related deaths occurred. Twenty six percent of cats in the AMD group had a uremic crisis and 21.7% of cats experienced a kidney-related death.
- Elliot et al. fed client-owned cats with spontaneous CKD a TKD (n = 29) or an AMD (n = 21).\(^4\) Cats fed a TKD had a median survival time of 633 days (1.7 years) compared to 264 days (0.7 years) for cats fed an AMD. Feeding a TKD also helped reduce plasma phosphate, blood urea nitrogen, and parathyroid hormone concentrations.
- Jacob et al. fed dogs with ≥ IRIS stage 2 CKD a TKD (n = 21) or an AMD (n = 17) for 24 months.\(^5\) Dogs fed a TKD has a median time to uremic crisis of 615 days and median survival time of 594 days. Dogs fed an AMD had a median time to uremic crisis of 252 days and median survival time of 188 days. Kidney related deaths accounted for 33% and 65% of deaths in the TKD and AMD groups, respectively.

These studies demonstrate that TKDs can be used to improve quality of life by controlling signs of uremia and increasing life span by altering disease progression.

Protein intake and chronic kidney disease
Protein restriction is commonly recommended for the management of CKD to reduce nitrogenous wastes and glomerular proteinuria. The timing and necessity of protein restriction to avoid protein malnutrition and loss of lean body mass (LBM) is controversial. Nitrogenous wastes can contribute to clinical signs associated with uremia, polyuria, polydipsia, and anemia. Loss of LBM related to chronic illness, also referred to as cachexia, occurs in patients with CKD. Cachexia is associated with altered strength, immune function, wound healing, and overall survival although more specific research in cats and dogs is needed.\(^6\) Dogs with a body condition score (BCS) of 1 – 3/9 had a reduced survival compared to dogs with a BCS of ≥ 4/9.\(^7\) The optimal protein intake for patients with CKD is likely multifactorial depending on the stage, sub-stage, and complete nutritional assessment of the patient.

Protein requirements
Protein requirements for dogs and cats have traditionally be determined through the use of nitrogen balanced studies, wherein nitrogen loss is equivalent to nitrogen intake. More recently, a study by Laflamme & Hannah evaluated the protein requirement of healthy adult male cats based on the maintenance of LBM.\(^8\) To achieve nitrogen balance, 56g protein/1000kcal of diet was needed while 95g protein/1000kcal was needed to maintain LBM. Regression analysis suggested that while 1.5g protein/kg body weight was required for nitrogen balance, 5.2 grams of protein/kg body weight is recommended to maintain LBM. This demonstrates that nitrogen balance can be achieved on a low protein diet, but it may be at the expense of LBM through the use of endogenous proteins. Suboptimal protein intake in the face of CKD can increase the production of uremic toxins through protein catabolism. It should be noted however that suboptimal protein intake can also result from hyporexia or anorexia which occurs commonly in patients with CKD.

Proteinuria and protein restriction
The American College of Veterinary Internal Medicine recommends a reduced protein intake in dogs with proteinuria secondary to glomerular disease.\(^9\) In rats and humans, protein directly injures the tubulointerstitium through release of vasoactive and inflammatory substances that trigger renal scarring and loss of function.\(^10\) Feeding a TKD may improve proteinuria.
In dogs with hereditary nephritis, feeding a protein restricted TKD reduced structural damage to glomeruli by decreasing glomerular basement splitting and delayed the progression of kidney failure.\textsuperscript{11}

Dogs with proteinuria treated with benazepril fed a TKD had a reduced urine protein to creatinine ratio (UPC) over 60 days (UPC 3.16 to 1.2) compared to dogs fed an AMD (UPC 3.62 to 2.14).\textsuperscript{12} The dose of benazepril did not differ between groups.

Proteinuria was reduced significantly in dogs with glomerulonephropathy when they were switched from a diet containing 72g protein/1000kcals to a diet containing 33g protein/1000kcals.\textsuperscript{13} A reduction in protein intake by 25 – 50\% is recommended based on the severity of proteinuria and patient assessment. This reduction may be relative to the patient’s current intake.

**Dietary protein in TKDs**

At this time, there is no definitive conclusion in the veterinary literature of which is better: a diet with unrestricted protein with the remaining features of a TKD versus a protein restricted TKD. The answer to this question is likely dependent on the species, IRIS stage and sub-stage. Studies demonstrating improved survival of dogs and cats fed a TKD compared to an AMD are limited to those with confounding dietary variables such as phosphorus restriction. The amount of protein needed to achieve protein restriction is not clearly defined.

- National Research Council (NRC) Minimum Requirement for crude protein is 40 grams and 20 grams of protein per 1000kcal for cats and dogs, respectively
- Association of American Feed Control Officials (AAFCO) minimum crude protein is 65 grams and 45 grams of protein per 1000kcal for cats and dogs, respectively

At this time, TKDs for CKD range from 25 – 55 grams protein in dogs and 58 – 82 grams protein per 1000kcal in cats. A highly digestible protein source is recommended. Reduced protein digestibility is reported to occur in some geriatric cats.\textsuperscript{14}

**Phosphorus restriction**

Phosphorus restriction, independent of other dietary factors, delays progression of CKD in both dogs and cats. Dogs with induced CKD fed a high phosphorus diet had significantly lower glomerular filtration rates and decreased survival compared to dogs fed a phosphorus restricted diet.\textsuperscript{15} Cats with induced CKD fed a normal phosphorus diet had evidence of renal mineralization, fibrosis and mononuclear cell infiltrates compared to cats fed a low phosphorus diet which has no histologic changes.\textsuperscript{16} AMDs often contain added phosphorus (usually \textgreater;1.5g per 1000kcal) to avoid phosphorus deficiency and maintain a 1:1 – 2:1 calcium to phosphorus ratio. AAFCO minimum for adult cats is 1.25g/1000kcal and adult dogs 1g/1000kcal. TKDs range from 0.4 – 1.2 g/1000kcal and 0.8 – 1.35 g/1000kcal for dogs and cats, respectively.

**Omega-3 fatty acids**

Supplementation with polyunsaturated omega-3 fatty acids (EPA and DHA) can have renoprotective effects. Dogs fed a diet supplemented with a high dose of fish oil had reduced proteinuria, creatinine, and histopathologic lesions compared to dogs fed a diet supplemented with safflower oil or beef tallow.\textsuperscript{17} A standard dose of 40mg/kg EPA + 25 mg/kg DHA once daily is recommended for both cats and dogs. Recently, a specific dose for dogs with CKD was recommended: 140 mg EPA + DHA / (kg body weight)\textsuperscript{0.75}.\textsuperscript{18} Some companies may add alpha-linoleic acid to TKDs, however this omega-3 fatty acid is insufficiently converted to EPA and DHA in both cats and dogs. Additional supplementation with EPA and DHA is recommended if the diet does not provide these nutrients.

**Dietary potassium**

Hypokalemia is common in cats with CKD and therapeutic diets may provide supplemental potassium beyond that added to a typical AMD. While hyperkalemia is typically associated with acute kidney failure, it may be a complication in some dogs with CKD. Hyperkalemia has been corrected by feeding a home prepared diet with reduced potassium (0.91 ± 0.14 g/1000kcal) in some dogs with CKD.\textsuperscript{19} Some TKDs contain potassium levels around this concentration which may be useful in these patients. Referral to a board-certified veterinary nutritionists for a home-prepared diet formulation may also be considered for a potassium restricted diet.

**Other dietary features of TKDs**

- Reduced sodium content to avoid the potential for sodium retention and contribution to systemic arterial hypertension
- Calorie dense with moderate to high levels of dietary fat to increase caloric intake and enhance palatability
- Alkalinizing to help correct metabolic acidosis
- Added antioxidants such as vitamin C and E to decrease oxidative stress
- Added soluble fiber to promote colonic bacterial growth and utilization of nitrogen and urea; beneficial for constipation
Tips for feeding patients with CKD

Introduce a TKD before clinical signs of uremia occur if possible. Diets are available in a variety of forms, flavors and textures. Provide clients with various samples to establish patient preferences. Be cautious when introducing a TKD to a hospitalized patient. This may lead to a food aversion in a diet best utilized for long-term feeding.

Feeding tubes are useful when managing patients with CKD. Liquid enteral diets containing < 1.5g phosphorus/1000kcal (both human and veterinary) are available for use with nasogastric and nasoesophageal feeding tubes. When an esophageal or gastric feeding tube is in place, a slurry of a canned TKD is recommended. Caloric density of a slurry can be improved when using a liquid enteral diet rather than water.

Home prepared diets are useful in patients with a poor or selective appetite. Referral to a board certified veterinary nutritionists is recommended. Home prepared diets found in books and on websites have numerous inadequacies and are not recommended. Before referral, updated IRIS staging including systolic blood pressure and UPC is recommended.

Overall, nutritional intervention in a cat or dog with CKD can greatly affect patient morbidity and mortality. The optimal diet for a patient with CKD relies on a variety of factors including the stage and sub-stage of disease, nutritional assessment, and patient preferences.

References
Formulating a Plan for Feeding Hospitalized Patients
Martha Cline, DVM, DACVN
Red Bank Veterinary Hospital
Tinton Falls, NJ

Nutritional assessment of a hospitalized patient
Malnutrition in hospitalized small animals can contribute to both patient morbidity and mortality. Caloric intake is positively associated with hospital discharge and outcome in small animal patients. The nutritional assessment of a hospitalized patient should take place daily. Historical information that can indicate malnutrition and impact the feeding plan includes:
- Weight loss and the time frame for weight loss
- Dietary intake and the length of anorexia/hyporexia
- Nutritional adequacy of the diet consumed (e.g., unbalanced home cooked diet versus complete and balanced commercial diet)
- Persistent gastrointestinal signs such as vomiting or diarrhea
- Other disease states that may impact metabolic needs (i.e., diabetes mellitus)

Physical exam should include evaluation the patient’s body weight, body condition score (BCS) and muscle condition. Muscle mass score, while not a validated scale, can be useful in assessing lean body mass (LBM). It is important to note the BCS does not account for LBM therefore it is possible to have an overweight/obese patient with poor muscle condition as a result of cachexia. Cachexia is the loss of LBM due to a disease state that decreases the body’s ability to preserve protein from catabolism which can directly impact strength, immune function, wound healing, and survival. Additional physical exam parameters as part of a nutritional assessment includes mentation, presence of ascites or edema, and hair coat quality. Laboratory parameters that may directly impact the nutritional plan include albumin, blood glucose, electrolyte status (potassium, magnesium, phosphorus), triglycerides, or presence of renal azotemia. A daily nutritional assessment should also assess feeding orders and previous day’s intake to determine if sufficient or if nutritional intervention is needed.

In practice, the author commonly encounters hospitalized patients with gastrointestinal dysmotility. Clinical signs include persistent vomiting or regurgitation, abdominal pain, constipation, or intolerance to enteral feeding. The primary types of gastrointestinal motility disorders in small animal patients include esophageal dysmotility, delayed gastric emptying, functional intestinal obstruction (ileus), and colonic motility abnormalities. Assessment of gastrointestinal dysmotility is limited for many clinicians to clinical signs, abdominal imaging, and gastric residual volumes (GRVs) if a nasogastric or gastric feeding tube is in place. Currently there is no clearly defined rule on how to adjust feeding when accounting for GRV. Clinical decisions regarding feeding after a GRV is recorded should take into account current pharmacological interventions and clinical signs. Dogs with nasoesophageal or nasogastric feeding tubes were randomized to receive either continuous or bolus feeding enteral nutrition. There was no difference in GRV between the continuous or bolus fed groups of dogs with nasogastric feeding tubes. Additionally the researchers were unable to find a significant correlation between the GRV and occurrence of vomiting or regurgitation. This suggests that termination of enteral feedings due to high GRV may not be warranted. If gastrointestinal dysmotility is diagnosed or suspected, therapy should be multimodal to include fluid therapy, early ambulation, early nutritional support, pharmacological intervention to stimulate motility and control pain as well as treatment of the underlying condition, metabolic derangements, and establishing normothermia.

Writing appropriate feeding orders
Feeding orders should include the following information: Resting energy requirement (RER), type(s) and amount of food to be offered, and frequency of feeding. Veterinary nurses and students were given a hypothetical voluntarily-eating canine patient with vague (frequency only), intermediate (diet and frequency), and specific (diet, amount, and frequency) feeding orders and asked to feed the patient. Significant differences in kilocalories fed per day were found between specific orders when compared to vague and intermediate, with no differences between vague and intermediate. Results of this study highlight that the amount of kilocalories fed is impacted by the manner in which the patient feeding orders are written. In a study evaluating caloric intake of hospitalized dogs, dogs were in a negative energy balance (<95% RER achieved) 73% of days evaluated (601/821 days). This was due to anorexia/hyporexia (44%), food withheld (34%), and poorly written feeding orders (22%). Evaluation of the patient’s intake over the last 24 hours during the morning assessment is also an important step before determining a patient’s nutrition plan and feeding orders for the day.

Resting energy requirements (RER)
RER is the number of calories required to maintain homeostasis at rest in a thermoneutral environment while the animal is in a fed state. While disease states can alter energy requirements, it is recommended to start with a patient’s RER as the initial goal to avoid overfeeding. Predictive equations are used to estimate energy requirements in small animal patients. While use of indirect calorimetry
Food typically offered to hospitalized patients includes therapeutic recovery type diets or therapeutic highly digestible gastrointestinal type diets. When creating an in-hospital stock of food for feeding hospitalized patients, recommend the following general considerations:

- Choose a highly digestible gastrointestinal diet approved for both growth and adult maintenance.
- Have available a low-fat (<25% metabolizable energy (ME)), highly digestible gastrointestinal diet for dogs with fat intolerance (i.e. pancreatitis).
- Recovery type diets are typically higher in protein (>25%ME) and fat (>45%ME). These diets are highly digestible and palatable, however are not appropriate for every patient especially dogs with fat intolerance. Also ensure these diets are approved for growth if fed to growing animals.
- Have both dry and canned options available for cats including canned foods with a variety of textures and flavors.
- If stocking alternative food choices like meat baby food or home prepared foods like chicken and rice, know the general nutrient profile and possible contraindications for feeding (e.g. meat baby food is typically high fat (>50%ME), 50:50 boiled boneless, skinless chicken breast and cooked white rice is highly digestible and ultra-low fat (<20%ME)).

Amount of food to be offered
The amount of food should be recorded as a standard measurements such as cup, can, or tablespoon. The use of non-specific feeding amounts such as “meatball” should be avoided without some type of quantification. If using dry extruded kibble, ensure an 8 ounce measuring cup is used. However, measuring cups are imprecise. Over 12 studies, variable accuracy when measuring dry extruded kibble ranged from an 18% under-estimate to an 80% over-estimate in portion size. Weighing both canned and dry food with a kitchen scale in grams is more precise and is preferred by the author in practice. Weighing the food in grams prior to and after feeding is an objective way to determine a patient’s caloric intake and help a clinician determine if assisted feeding is necessary. In the author’s experience, subjectively recording a patient’s intake by percentage is limited and can be further complicated if the person offering the food is different from the one recording intake. Hospitalized patients can be started on a fraction (25 – 33%) of RER initially gradually increasing in increments every 12 – 24 hours to full RER. This method useful in patients with anorexia or hyporexia to avoid food waste, and helps to minimize complications such as gastrointestinal signs or refeeding syndrome.

Frequency of feeding
The number of times per day a patient is fed should also be recorded on the treatment sheet. Typically the author advocates twice daily feeding for patients following routine procedures such as spay, neutering, dental, or orthopedic procedures or patients with an excellent appetite that are otherwise feeling well. Increased frequency of feeding should be considered in other patients including the critically ill, very young (<6 months in age), hypoglycemic, or those with gastrointestinal disease, pancreatitis, or post-operative gastrointestinal surgery.

Assisted feedings for hospitalized patients
Nutritional intervention should be considered in any patient on admission with anorexia ≥ 3 days, a patient where anorexia is anticipated for ≥ 3 days, long term hyporexia, and sooner in some patients following nutritional assessment. This includes and is not limited to overweight or obese cats, septic peritonitis, pancreatitis, or parvovirus. Nutritional support initiated <24 hours postoperatively in dogs with septic peritonitis was associated with a significantly shorter hospitalization length (1.6 days) when compared to dogs receiving nutritional support after 24 hours. The concept of “bowel rest” is no longer recommended as several studies have indicated that early enteral nutrition is well tolerated with few complications. Early enteral nutrition through an esophagostomy tube in dogs with severe acute pancreatitis was well tolerated with fewer complication that dogs receiving parenteral nutrition. Early enteral nutrition instituted at 12 hours after admission through a nasogastric tube in dogs with parvovirus resulted in earlier clinical improvement and possibly improved gut barrier function compared to dogs receiving enteral nutrition after vomiting.

The linear equation to calculate RER can be used for patients between 2 – 25kgs. The linear equation is not advised in patients with an ideal body weight (IBW) outside of these parameters as it will overestimate their energy requirements. Patients that are underweight or IBW should be fed to their RER based on their current body weight. Typically the author will adjust the RER of obese patients (≥ 8/9 BCS) to IBW or RER modified for obesity. Approximately 25 – 40% of an obese human’s excess body weight is LBM therefore modification of feeding to IBW may underestimate the energy needs for LBM. Approximately 25% of the difference between IBW and current body weight can be added back to IBW prior to calculating RER. The author typically reserves this method for ill obese patients although further research evaluating energy requirements for hospitalized obese cats and dogs is needed at this time.

Type(s) of food to be offered
Food typically offered to hospitalized patients includes therapeutic recovery type diets or therapeutic highly digestible gastrointestinal type diets. When creating an in-hospital stock of food for feeding hospitalized patients, recommend the following general considerations:

- Have available a low-fat (<25% metabolizable energy (ME)), highly digestible gastrointestinal diet for dogs with fat intolerance (i.e. pancreatitis).
- Recovery type diets are typically higher in protein (>25%ME) and fat (>45%ME). These diets are highly digestible and palatable, however are not appropriate for every patient especially dogs with fat intolerance. Also ensure these diets are approved for growth if fed to growing animals.
- Choose a highly digestible gastrointestinal diet approved for both growth and adult maintenance.
- If stocking alternative food choices like meat baby food or home prepared foods like chicken and rice, know the general nutrient profile and possible contraindications for feeding (e.g. meat baby food is typically high fat (>50%ME), 50:50 boiled boneless, skinless chicken breast and cooked white rice is highly digestible and ultra-low fat (<20%ME)).

The linear equation to calculate RER can be used for patients between 2 – 25kgs. The linear equation is not advised in patients with an ideal body weight (IBW) outside of these parameters as it will overestimate their energy requirements. Patients that are underweight or IBW should be fed to their RER based on their current body weight. Typically the author will adjust the RER of obese patients (≥ 8/9 BCS) to IBW or RER modified for obesity. Approximately 25 – 40% of an obese human’s excess body weight is LBM therefore modification of feeding to IBW may underestimate the energy needs for LBM. Approximately 25% of the difference between IBW and current body weight can be added back to IBW prior to calculating RER. The author typically reserves this method for ill obese patients although further research evaluating energy requirements for hospitalized obese cats and dogs is needed at this time.

Exponential Equation: \( RER = (\text{Body Weight}_{\text{kg}})^{0.75} \times 70 \)
Linear Equation: \( RER = 30(\text{Body Weight}_{\text{kg}}) + 70 \)

The linear equation to calculate RER can be used for patients between 2 – 25kgs. The linear equation is not advised in patients with an ideal body weight (IBW) outside of these parameters as it will overestimate their energy requirements. Patients that are underweight or IBW should be fed to their RER based on their current body weight. Typically the author will adjust the RER of obese patients (≥ 8/9 BCS) to IBW or RER modified for obesity. Approximately 25 – 40% of an obese human’s excess body weight is LBM therefore modification of feeding to IBW may underestimate the energy needs for LBM. Approximately 25% of the difference between IBW and current body weight can be added back to IBW prior to calculating RER. The author typically reserves this method for ill obese patients although further research evaluating energy requirements for hospitalized obese cats and dogs is needed at this time.

Assisted feedings for hospitalized patients
Nutritional intervention should be considered in any patient on admission with anorexia ≥ 3 days, a patient where anorexia is anticipated for ≥ 3 days, long term hyporexia, and sooner in some patients following nutritional assessment. This includes and is not limited to overweight or obese cats, septic peritonitis, pancreatitis, or parvovirus. Nutritional support initiated <24 hours postoperatively in dogs with septic peritonitis was associated with a significantly shorter hospitalization length (1.6 days) when compared to dogs receiving nutritional support after 24 hours. The concept of “bowel rest” is no longer recommended as several studies have indicated that early enteral nutrition is well tolerated with few complications. Early enteral nutrition through an esophagostomy tube in dogs with severe acute pancreatitis was well tolerated with fewer complication that dogs receiving parenteral nutrition. Early enteral nutrition instituted at 12 hours after admission through a nasogastric tube in dogs with parvovirus resulted in earlier clinical improvement and possibly improved gut barrier function compared to dogs receiving enteral nutrition after vomiting.
had ceased for 12 hours (approximately 50 hours after admission). Prior to initiating nutritional support, a patient should be cardiovascularly stable, recovered from anesthesia, and electrolyte and acid-based abnormalities addressed.

The use of nasoesophageal (NE) and nasogastric (NG) feeding tubes are utilized frequently in practice by the author. NE and NG feeding tubes are very useful for temporary feeding support (3 – 5 days). Use of an esophageal of gastric feeding tube is recommended in patients needing long term assisted feeding (>1 week). The placement of NE and NG feeding tubes does not require specialized equipment or general anesthesia. Some animals may require minimal to heavy sedation during NE or NG tube placement. A liquid enteral diet is required with nasoenteric feeding tubes to avoid clogging of the tube. Nasojejunal (NJ) feeding tube placement has been described in dogs using fluroscopy, endoscopy, and have also been placed in the author’s experience during abdominal surgery with guidance of the tube into the jejunum by the surgeon. NJ feeding tubes are indicated in patients where post pyloric feeding is ideal including acute pancreatitis, pyloric disease, or patients where gastric feeding is poorly tolerated. The use of NJ tubes typically requires the use of specialized equipment and therefore can limit their use in general practice. Complications from NE and NG tube placement can include vomiting, diarrhea, tube blockage, tube removal, rhinitis, epistaxis, and esophagitis. The most severe complication may be tracheal intubation with iatrogenic pneumothorax, abscess, tracheal perforation, fistula formation, or death. The technique of measuring and aspirating the tube at the level of the thoracic inlet may help to reduce tracheal intubation and associated complications. Retrospective review in dogs with NE or NG tube placement revealed no significant difference in recorded complications. There was also no significant difference in gastrointestinal complications or nutrition delivered between continuous versus intermittent feeding through nasoenteric feeding tubes in both cats and dogs. The use of 1/4 teaspoon pancreatic enzymes and 325 mg sodium bicarbonate in 5 mL of water has been used successfully by the author to unclog nasoenteric feeding tubes. When using esophageal or gastric feeding tubes, a slurry of the diet with a liquid enteral diet instead of water can improve caloric density. Patients requiring other types of assisted feeding such as jejunostomy feeding tube or parenteral nutrition likely required 24 hour care and referral to such a hospital is recommended.

References
Nutritional Management of Calcium Oxalate Urolithiasis
Martha Cline, DVM, DACVN
Red Bank Veterinary Hospital
Tinton Falls, NJ

In 2013, Calcium oxalate (CaOx) uroliths submitted to the Minnesota Urolith Center for analysis represented 41-42% of submissions in cats and dogs. CaOx uroliths cannot be dissolved and must be removed physically from the patient. Unfortunately, recurrence rates of up to 50% of dogs within 3 years and 33% of cats in two years of removal have been reported. Risk factors for the formation of CaOx urolithiasis in cats include males, neutering, increasing age, and breed such as Burmese, Persian, and Himalayan. Risk factors for the formation of CaOx urolithiasis in dogs include <7yr age, males, neutering, toy-small sized breeds, acidic urine pH and history of cystitis. In a recent retrospective study of 135 dogs with CaOx urolithiasis, Miniature Schnauzers were 3 x at risk for recurrence than other breeds. This study also evaluated the effect of two therapeutic CaOx preventative diets versus any other type of diet on the recurrence of CaOx uroliths finding no significant difference between groups. Prospective controlled clinical trials are needed to further evaluate the long term effects of therapeutic CaOx preventative diets given that the time to recurrence can be several years. Given the inability to medically dissolve CaOx uroliths and the relatively high recurrence rate, preventative strategies should be instituted. Urolith prevention includes the following principals regardless of stone type: increasing water intake, altering urine pH, decreasing precursors, and increasing urinary inhibitors.

CaOx form in urine oversaturated with calcium and oxalate. Relative supersaturation (RSS) is a validated method for measuring the pH and saturation of mineral ions in the urine of cats and dogs to determine their risk for urolith formation. This method is commonly used by pet food manufactures to determine the efficacy of their diet for CaOx prevention. When urine is oversaturated with mineral precursors, crystals will form which will then grow and aggregate to form a calculi. Crystals will dissolve in urine when the RSS is <1 (undersaturated). When urine has an RSS of >1, it is supersaturated. Supersaturated metastable urine will prevent spontaneous crystallization while labile or unstable supersaturation will allow crystallization to occur. Since CaOx uroliths cannot be dissolved, target RSS is metastable to prevent crystallization.

Increasing water intake
The purpose of increasing water intake is to decrease the urinary concentration of urolith precursors and increase urine volume to increase voiding frequency. Target urine specific gravity (USG) in dogs is <1.030, while a USG <1.040 is appropriate for cats. Switching to a canned diet is this simplest and most practical way to accomplish this goal. High moisture content diets increase the daily fluid intake, resulting in lower USG and lower RSS for CaOx versus diets with a lower moisture content. The use of water fountains has also been suggested as a way to increase water intake in cats. Two studies reported water intake of cats using still or flowing water sources. Neither study was able to find statically significant differences in water intake. A recent abstract by the author and colleagues investigated the differences in water intake and urinary parameters between free-falling, circulating, and still water bowls in a randomized, cross-over design with 14 laboratory cats. No significant differences were found between groups in water intake, USG, urine osmolality, and RSS for CaOx and struvite. Three individual cats in our study did have statically significant differences in water intake between bowls, but these were different for each cat suggesting that some cats will have an individual preference for water source.

Sodium is added to some therapeutic urinary diets to increase water intake. Dietary supplementation with NaCl in dogs increased urine volume and urine calcium excretion while urine oxalate concentrations and RSS for CaOx were significantly decreased. Some evidence has suggested that cats with early renal disease (IRIS Stage I) will have progression of their renal parameters on a high sodium diet. Alternatively, healthy aged cats (median age 10.4yrs) had no alterations in glomerular filtration rate, blood pressure, or routine clinical pathological parameters in a 2 year period randomized to either a high sodium (3.1g/1000kcal) versus a low sodium (1g/1000kcal) diet. The exact appropriate level of sodium for a therapeutic CaOx preventative diet in cats and dogs is unclear as commercial therapeutic diets have a wide range from sodium contents.

Altering urine pH
Acidic urine pH is a reported risk factor for the formation of CaOx urolith formation in dogs and cats by increasing urine calcium excretion. The role of urine pH in CaOx prevention unclear. A recent abstract demonstrated that while calcium excretion increased with decreasing urine pH (mean 6.37 – 5.93), CaOx RSS was not affected by urine pH. Another study demonstrated that urinary saturation of CaOx was decreased in cats consuming an alkalinizing diet with a urine pH >7.2 compared to cats consuming the same diet but acidifying with an increased in CaOx urinary saturation at a pH <6.5. Multifunction urolith preventative diets typically target urine pH slightly acidic as struvite urolith solubility is pH dependent and dissolution can be achieved. Supplementation with potassium citrate at 75mg/kg PO q12hr is recommended to alkalinize the urine. Target urine pH for CaOx urolith prevention is 6.6-7.5.
Decreasing precursors
Oxalate is primarily derived from dietary intake. High oxalate containing foods include certain vegetables (e.g. spinach), legumes, and fermentable fibers (e.g. beet pulp). The endogenous hepatic metabolism of glycine and vitamin C will also contribute to oxalate formation. Excess intake of vitamin C should be avoided as this is a precursor to oxalic acid. Cats and dogs do not have a dietary requirement for vitamin C, therefore additional supplementation is unnecessary. Cranberry supplements are also not recommended as these contain both vitamin C and oxalates and contribute to urine acidification. Experimentally induced vitamin B6 deficiency in kittens causes hyperoxaluria however vitamin B6 deficiency has not been reported with spontaneously occurring CaOx urolithiasis in dogs and cats. Supplementation may be needed in patients consuming an unbalanced home prepared diet (2-4 mg/kg q24hr) but is unnecessary in patients consuming a complete and balanced commercial diet. Decreased dietary animal protein is generally recommended for people with CaOx as this is associated with increased risk of CaOx formation. High protein intake in cats and dogs has been shown to be protective in epidemiologic studies. Additional prospective research is needed to determine the optimal protein intake in dogs and cats with CaOx urolithiasis.

One possibility for reducing dietary oxalate is to reduce enteric absorption. *Oxalobacter formigenes* is a Gram-negative anaerobic bacteria that degrades oxalate in the intestinal tract. The absence of enteric colonization of this bacteria has been reported as a risk factor for CaOx urolithiasis in dogs. The bacteria’s presence in the gastrointestinal tract is associated with reduced urinary oxalate concentrations in humans. The presence of *O. formigenes* by fecal PCR and culture was compared between dogs with CaOx uroliths, healthy age-, breed-, and gender-matched dogs, and healthy non-stone forming breed dogs. Dogs with CaOx uroliths had a prevalence of 25% versus 50% in healthy age-, breed-, and gender-matched dogs, and 75% in the healthy non-stone forming breeds. The prevalence of *O. formigenes* in the feces of healthy non-stone forming cats is reported to be 86%. Lactic acid bacteria, such as *Lactobacillus* and *Bifidobacterium*, have also been shown to have intestinal oxalate-degrading activity and decreased urinary oxalate concentration with supplementation in people. *Lactobacillus spp* were cultured from two veterinary probiotics and their in vitro oxalate-degrading capacity was measured. Oxalate concentrations were significantly reduced by *L. acidophilus*. Oxalate concentration was significantly increased by *L. plantarum* and no change in oxalate concentrations occurring with *L. casei*. In vivo studies in cats and dogs are needed to determine if enteric colonization of *L. acidophilus* or *O. formigenes* can reduce urine oxalate concentrations and reduce the risk of urolith recurrence.

Decreased dietary intake of calcium is not recommended without also decreasing dietary oxalate in normocalcemic patients with CaOx urolithiasis as this may increase their risk of formation. Dietary calcium can bind oxalate in the lumen of the gastrointestinal tract thus preventing absorption. Thirty five percent of cats (7/20) with idiopathic hypercalcemia were diagnosed with urinary or renal calculi. Two of these cats underwent cystotomy and analysis revealed 100% CaOx uroliths. Dietary and medical strategies should be aimed at treating the hypercalcemia in these cats. Lower calcium intake may be beneficial for idiopathic hypercalcemic patients. Two of 3 hypercalcemic cats with a history of CaOx urolithiasis fed a calcium oxalate-prevention diet achieved normocalcemia while the third cat had a significant reduction in its hypercalcemia. These three cats also had significant reductions in urinary calcium excretion. Hypercalcemia was reported in 33% of cats undergoing a prospective randomized clinical trial investigating the recurrence rate of CaOx urolithiasis in cats on a test diet for CaOx prevention versus a regular maintenance diet. Cats on the prevention diet had fewer episodes of hypercalcemia and the RSS for CaOx and urinary calcium concentration were lower in cats fed the prevention diet. CaOx preventative diets may be useful in these cases if reductions in hypercalcemia or normocalcemia can be achieved although additional research is needed.

Increasing urinary inhibitors
Citrate is a urinary inhibitor that forms a soluble salt with calcium to reduce CaOx precipitation. When urine pH is low, urinary concentration of citrate decreases due to increased absorption of citrate by the renal proximal tubules. Supplemental potassium citrate can also be used to alkalinize the urine which can increased renal citrate excretion. Recommended dosing is 75mg/kg PO q12hr. Large molecular weight proteins in the urine, such as Tamm-Horsfall glycoprotein, nephrocalcin, uropontin, and glycosaminoglycans, are recognized as inhibitors of CaOx formation by influencing crystal formation, aggregation, and growth. Cats with a history of CaOx urolithiasis fed a therapeutic CaOx prevention diet has significantly higher concentrations of glycosaminoglycans than when they were fed the diet they consumed prior to diagnosis. No differences in Tamm-Horsfall glycoprotein and nephrocalcin were found between diets.

Monitoring tips
Recommended monitoring includes urinalysis with evaluation of a fresh urine sediment in-house. Persistent calcium oxalate crystalluria, particularly in a predisposed breed, represents an increased risk for CaOx urolith formation. CaOx crystalluria is not a reliable indicator of CaOx urolithiasis as uroliths can be present with or without the presence of crystalluria. Follow-up urinalysis is recommended every 3-4 months until target urinary parameters are achieved. Bi-annual monitoring of urinalysis and abdominal imaging is recommended for long term monitoring.
References


548
Obesity and (Pick Your Disease!): Managing Obesity and Concurrent Disease in Pets
Martha Cline, DVM, DACVN
Red Bank Veterinary Hospital
Tinton Falls, NJ

Obesity is the most common form of malnutrition in cats and dogs. Animals are considered obese when they reach a body condition score (BCS) of 8/9 which is approximately 20% over their ideal body weight or ≥35% body fat. Owners and veterinarians must both recognize that a patient is overweight or obese while also acknowledging the health consequences of excess adiposity. Surveys of dogs and cats presenting to veterinarians in primary or referral practices indicated that only 11.4% of overweight/obese dogs and 3.6% overweight/obese cats had overweight or obesity listed as a medical problem. Further, 53% of owners in one study assigned their overweight or obese dog with an appropriate BCS, however 39% of these owners thought their dog’s weight was acceptable or normal.

Life span and quality of life
Overweight/obesity significantly impact life span, quality of life, and the development of chronic diseases in cats and dogs. Labrador retrievers fed 25% less than age-matched pairs had a median life span of 13 years while dogs fed 25% more had a median life span of 11.2 years. Dogs fed more were overweight, with a mean BCS of 6.7±0.19 compared to the feed restricted dogs with a mean BCS 4.6±0.19. Chronic treatment for osteoarthritis was delayed in the feed restricted group (13.3 vs 10.3 years) and treatment for chronic diseases in general was also delayed (12 vs 9.9 years). Radiographic evidence of osteoarthritis (OA) was also found later in the feed restricted dogs (at 2 years of age, hip OA 4% versus 42%). A similar life time study does not exist at this time in cats.

Mobility is often a factor for owners when assessing their pet’s quality of life. Modest weight loss of 6.1% significantly decreased lameness in dogs. Obese cats are 4.9 times more likely to develop lameness requiring veterinary care. Dogs completing a weight loss plan had improved quality of life (increased vitality and decreased emotional disturbance and pain) when compared to dogs that failed to achieve their ideal body weight. Cats undergoing weight loss for 8 weeks had an increase in pre-feeding behaviors such as begging, following, meowing, pacing however owner reported their cats became more affectionate post feeding.

Obesity paradox
The finding that overweight or obesity may be protective in regards to mortality in a variety of disease conditions is termed the obesity paradox. While overweight, obesity, and abdominal adiposity are associated with increased risk of heart failure in people, overweight/obesity are associated with lower all-cause and cardiovascular mortality in people with congestive heart failure (CHF). While the protective mechanism of the obesity paradox is not clear and likely multifactorial, increased reserve of lean body mass (LBM) with obesity and lack of cachexia likely plays a major role. Few studies investigate this phenomenon in veterinary medicine. In a recent abstract, cats below a median body weight of 4.2kgs at the time of diagnosis for chronic kidney disease (CKD) has a significantly shorter survival time compared to cats with a body weight ≥4.2kgs. Underweight dogs with CKD (BCS 1-3/9) had a significantly shorter median survival time (MST) compared to moderate (BCS 4-6/9) and overweight dogs (BCS 7-9/9) with no difference between moderate and overweight. A study of survival in dogs with CHF failed to demonstrate a significant association with BCS, however weight change was significantly associated with survival with dogs gaining weight surviving the longest. A similar study in cats with CHF found a U-shaped relationship between body weight and survival with reduced survival times in cats with the lowest and highest body weights. Given the available literature, maintenance of a BCS of 6-7/9 in patients with chronic diseases such as CHF or CKD is a reasonable recommendation to preserve LBM and prevent cachexia. Additional research is required to investigate this recommendation.

Chronic kidney disease
Obesity is a risk factor for the development and progression of CKD in people. Experimentally-induced obesity in dogs has been shown to increase mean arterial pressure and plasma renin activity, alter renal function (glomerular hyperfiltration), and cause histologic changes including expansion of Bowman’s capsule, increased mesangial matrix, thickening of glomerular and tubular basement membranes, and increased cell proliferation in the glomerulus. Following weight loss, dogs had evidence of improved renal function with increased urine specific gravity (USG), decreased urine protein to creatinine ratio (UPC), and decreased levels of biomarkers of renal injury (homocysteine, cystatin, and clusterin). Creatinine was also decreased after weight loss, but this may be confounded by the loss of LBM in addition to fat mass. In this same study, 8 dogs had UPC > 0.5 prior to weight loss, while only 1 dog had UPC > 0.5 after weight loss. Another study comparing UPC in dogs with BCS 4-5/9 versus ≥6/9 found no statistical difference between groups. The researchers in this study were unable to separate out overweight versus obese dogs for additional statistical analysis due to the limited sample size. Overweight/obese dogs and cats with CKD must be assessed on an individual basis...
to achieve optimal body weight. Generally the use of a therapeutic kidney diet will take precedence in dogs and cats with later stage CKD. In obese dogs and cats with early stage CKD (IRIS stage 1 ± 2), a weight loss plan may be considered only after a complete nutritional assessment and only if close monitoring is available. If progression of CKD is noted, active weight loss should be suspended and appropriate diet modifications should occur until further evaluation. Consultation with a board-certified veterinary nutritionists is recommended for obese patients with CKD.

Cancer
In people, increased body-mass index is associated with an increased risk of development and death from cancers including esophageal, thyroid, colon, kidney, endometrium, gallbladder, breast, and pancreas as well as development of leukemia, multiple myeloma, and non-Hodgkin lymphoma. Limited studies are available in veterinary medicine investigating overweight/obesity with cancer risk and outcomes. Some work suggests an increased risk in the development of mammary cancer and transitional cell carcinoma in dogs. In one study, the prevalence of overweight/obesity was slightly lower in dogs with cancer, although there was a higher prevalence of overweight/obesity in dogs with mammary cancers though this was not statically significant. Out of 100 dogs presenting to an oncology service at a veterinary teaching hospital, 26 were overweight and 29 obese based on BCS. Fewer data is available in cats with one study evaluating BCS and survival time finding feline cancer patients with a BCS <5 having a MST of 3.3 months compared to 16.7 months in cats with a BCS ≥ 5.

Before instituting a weight loss plan for an overweight/obese cancer patient, a complete nutritional assessment including cancer staging, evaluation of clinical signs, and determination of prognosis must be performed. Clinicians must decide if weight loss will provide a clinical benefit in light of the patient’s survival time. Weight loss to improve quality of life rather than achievement of ideal body weight may be appropriate for some animals such as an obese dog with osteosarcoma and OA now ambulating on 3 legs or an obese cat with lymphoma and diabetes mellitus receiving corticosteroids. Typically the author starts with a conservative caloric reduction to achieve slow rate of weight loss (0.5-1% body weight per week). Aggressive caloric restriction in a sick animal may contribute to preferential loss of LBM therefore serial assessment of muscle condition is also recommended. A high protein, high fat, and low carbohydrate diet is typically advocated in veterinary cancer patients to support LBM and protein metabolism as well as decrease the energy supply to neoplastic cells that are inefficient in oxidizing fat for energy. A diet with the above characteristics enriched in omega-3 fatty acids has been shown to prolong survival time is a subset of dogs with lymphoma compared to a similar diet unenriched in omega-3 fatty acids. Additional work is needed to investigate potential clinical benefits of high fat versus low fat diets in dogs and cats with cancer. Weight loss diets for dogs are generally low in fat to avoid increases in caloric density, but high in protein to support LBM. Some weight loss diets are formulated with higher amounts of omega-3 fatty acids which may be desired for reasons other than neoplasia including OA.

Diabetes mellitus
Diabetes mellitus (DM) in dogs is due to inadequate insulin production from immune-mediated destruction of beta cells or from pancreatitis. Feline DM occurs commonly due to peripheral insulin resistance as a result of excess adiposity and beta cell dysfunction. Although insulin resistance in dogs does exist, dogs will have an absolute requirement for insulin administration that diet alone will not correct. Insulin sensitivity in lean dogs was 58% greater than overweight dogs. Dogs with a body weight gain of approximately 43% had significantly higher basal insulinemia and insulin resistance. In contrast, cats can have insulin resistance at 10% over their lean body weight. Obese cats are up to 3.9 x as likely to have DM than cats with an ideal BCS. In dogs, the role of obesity in the development of DM is less clear although a relationship likely exists. Twenty percent of obese dogs in one study had criteria for obesity-related metabolic dysfuction. This criteria was modeled from human guidelines for diagnosis of metabolic syndrome, a risk factor in people for the development of Type II DM and cardiovascular disease. The progression of insulin resistance to the development of DM in dogs is not documented and further studies are needed to determine what these criteria in dogs mean for disease risk and outcomes.

Goals for managing an obese diabetic patient include eliminating clinical signs associated with hyperglycemia and glycosuria, avoiding hypoglycemia, improving patient body weight and condition, and maintaining the pet and owner’s quality of life. Initial recommendations for overweight and obese dogs should be weight maintenance until glycemic control is achieved. Once this is established, conservative weight management (0.5-1% body weight per week) can be instituted which may help to improve insulin sensitivity. A modest reduction in energy intake (decreasing caloric intake by 20%) with control of treat consumption (≤ 10% caloric intake) can be tried initially. Some dogs may require more or less caloric restriction for weight loss at follow-up. The use of a therapeutic weight loss diet is recommended for obese patients. These diets are typically high in protein (>30% metabolizable energy (ME)) with moderate to high amounts of total dietary fiber (>30g/1000kcal). Added soluble and insoluble fiber to a high carbohydrate diet (>50%ME) has been shown to improve glycemic control in dogs.

Weight loss is an important goal in overweight/obese cats with DM and can help achieve and maintain diabetic remission in conjunction with appropriate medical management. Every 1kg increase in body weight has been associated with a 30% decrease in
insulin sensitivity which was normalized with weight loss.\textsuperscript{36} The timing of weight management however should be delayed in an overtly ill cat until stabilized. Diabetic remission typically occurs in cats during the first 3-4 months of therapy. A recent survey of veterinarians in the Southeastern United States revealed that 97% (87/90) of vets always or usually recommended dietary management at the time of DM diagnosis and 93% of respondents recommended diets marked as low carbohydrate (LC).\textsuperscript{37} A low carbohydrate (<15% ME) diet may be advantageous for cats with DM. Cats fed a LC-low fiber diet had a significantly higher remission rate of 68% compared to cats fed a moderate carbohydrate-high fiber diet (41%).\textsuperscript{38} Cats also appeared to be better regulated on the LC diet. The author typically recommends canned, low carbohydrate diets for weight loss in diabetic cats. In obese cats that only consume dry food, a low calorie density, high fiber weight loss diet is typically preferred to improve owner compliance. Both therapeutic low carbohydrate and high fiber weight loss diets contain moderate to high amounts of protein (>35% ME) to support LBM.

References

Popularity of raw meat based diets
In the United States and Australia, raw food or bones were reported to be fed at least daily as part of the main meal in 9.6% of cats and 16.2% of dogs. Another 0.9% of cats and 7.4% of dogs received raw meat or bones as a treat or snack at least once weekly. Interestingly, these survey results were collected prior to the melamine pet food recall of 2007. It is reasonable to hypothesize that feeding practices amongst pet owners has changed over the last decade. Raw freeze-dried pet food retail sales increased 64% in 2014 from US$25 million to US$40 million while raw frozen pet food increased 32% from US$52 million to US$69 million. Given the increasing popularity of commercial raw pet food products, it is likely veterinary professionals are encountering pets fed a raw meat-based diet (RMBD) or treat commonly in practice.

Nutritional adequacy of raw meat based diets
The nutritional adequacy of commercial RMBDs can be determined based on the Association of American Feed Control Officials (AAFCO) guidelines for formulation or by feed trial. RMBDs labeled for intermittent or supplemental feeding are not nutritionally complete and balanced. The use of whole prey diets may be used by some owners feeding a RMBD. Whole 1-3 day old chicks and adult ground chicken were found to meet macronutrient requirements but were deficient in some mineral requirements, including manganese, copper, potassium and sodium compared to AAFCO requirements for adult cats. These diets were not evaluated for vitamin content. Research kittens consuming a diet solely of whole raw ground rabbit developed dilated cardiomyopathy secondary to taurine deficiency resulting in the death of one kitten. The diet of a prey animals (e.g., chicks, mice, or rabbits) sold for pet consumption may also influence their nutrient profile. Home-prepared diets, raw or cooked, can be obtained by owners through a number of sources including the Internet, pet magazines, and books written by veterinarians and non-veterinarians with varying levels of nutrition training. Several studies have evaluated the nutritional adequacy of home-prepared diets in companion animals finding numerous and significant nutritional imbalances. Evaluation of 200 published home-prepared recipes for adult maintenance in dogs written by veterinarians (64.5%) and non-veterinarians (35.5%) revealed at least one essential nutrient deficiency according to National Research Council or AAFCO guidelines in the majority of diets (95%), and 83.5% of recipes has multiple deficiencies. Analysis of 77 home prepared bone and raw food rations for dogs in Germany found that 76% had one or more nutritional imbalances. A home prepared raw food diet formulated by a now board-certified veterinary nutritionists and commercial raw food diet were determined to be nutritionally adequate based on a 10 week AAFCO feeding trial in kittens.

Animals may develop clinical signs associated with nutrient deficiency or toxicity when consuming an unbalanced diet. Nutritional secondary hyperparathyroidism has been reported in both cats and dogs consuming unbalanced home prepared diets manifesting as spontaneous fracture, muscle twitching, seizures, and limb deformities. A dog consuming an unbalanced home prepared diet with deficiencies including calcium, phosphorus, and vitamin D developed tetanic seizures and hyperthermia during evaluation of bilateral humeral osteochondritis dissecans. Other reported nutrient imbalances resulting in clinical signs include metabolic osteopathy with extensive new bone formation from hypervitaminosis A in a cat consuming raw pork liver, pansteatitis in cats secondary to vitamin E deficiency while consuming a high poly-unsaturated fat raw diet, and hyperthyroidism in dogs consuming raw beef gullet with thyroid tissue.

Potential benefits of raw meat based diets
Owners may choose to feed a RMBD to their pets due to anecdotal health benefits and to provide a more natural or ancestral diet. Many of these benefits remain unproven, although the body of scientific evidence surrounding RMBDs is limited. Cats and wolves in the wild will consume a variety of prey to support survival and reproduction, and some owners may choose to mimic this diet closely. However, this same type of diet may not be optimal for domestic animals expected to live long and healthy lives primarily indoors and reproductively altered.

Some owners may report a benefit of smaller stool volumes, less fecal odor, and improved gastrointestinal health in cats and dogs fed home-prepared or commercial RMBDs. Several studies have demonstrated a high digestibility of RMBDs fed to both exotic and domestic cats when compared to extruded diets. Digestibility of dry matter, organic matter, crude protein, and gross energy was significantly higher in a commercial and homemade raw food diet compared with a canned heat-processed diet in domestic kittens. Cats consuming whole ground rabbits had significant improvements in stool quality compared to cats consuming a commercial diet. Another study found no difference on total tract energy and macronutrient digestibility in cats fed a commercial raw beef-based diet or the same diet cooked in the microwave to at least 160°F. Significant differences have been noted in the fecal microbiota of cats fed a
raw diet consisting of 1-3 day old chicks compared to a chicken-based extruded diet although these differences could not be only attributed to the raw vs extruded nature of the diets as the nutrient composition differed.21

Heat processing of pet foods can result in Maillard reactions responsible for the browning of foods when heated. This reaction causes decreased bioavailability of lysine and formation of Millard reaction products (MRPs) and advanced glycation end-products (AGEs) which may have harmful biological effects.22 A study found that dog and cats had an average daily intake of one MRP 122 and 38 times higher respectively than the average daily intake for adult humans.23 Further studies are needed to investigate the long-term health implications of this in dogs and cats. Heat processing of meat proteins can also negatively impact the creatine concentration of the dry dog food and meat/meat and bone meal, although the benefits of a creatine-rich diet is dogs is unknown.24 Researchers suggest this may also be due to Maillard reactions.

The infectious potential of raw meat based diets

Commercial and home prepared RMBDs may be contaminated with potentially harmful pathogens. The concern for public and animal health has led organizations such as the American Veterinary Medical Association and the American Animal Hospital Association to discourage the use of RMBDs.25–28 Studies have documented the presence of bacterial contamination in both commercial and home prepared RMBDs. A study by the United States Food and Drug Administration (FDA) in 2011-2012 found that out of 196 commercial RMBDs, 7.7% were positive for Salmonella spp., and 16% positive for Listeria monocytogenes.27 A study examining 25 commercial raw canine and feline RMBDs found contamination with Clostridium perfringens (20%), Escherichia coli (64%), Salmonella spp. (20%), Clostridium difficile (4%), and Staphylococcus aureus (4%) based on culture.28 Twenty-three percent of RMBDs for dogs contained extended spectrum cephalosporin-resistant E.coli.29 Salmonella in raw meats sold for human consumption has reported rates of 44% in chicken and 4% in beef and pork, therefore it is not surprising to find contamination in raw pet food.30,31 While the presence of Salmonella is allowed on meat for human consumption by the USDA, the FDA maintains a zero-tolerance policy on Salmonella in pet foods.32 The FDA lists 26 dog and cat food recalls from 2015 (expanded recalls are counted as 1 with the original recall), with the vast majority from RMBD.33 Eleven treat products were recalled, 10 of these from Salmonella contamination. Seven of the recalled treats due to Salmonella contamination were beef products including beef gullet, tripe, trachea, jerky, and bone. Twelve of 15 complete and balanced recalled commercial diets were RMBD. Seven RMBDs were recalled for Salmonella, 1 for Listeria monocytogenes, 3 for both Salmonella and Listeria, and 1 for thiamine deficiency.

Once fed a RMBD containing Salmonella, cats and dogs have been shown to shed the bacteria in their feces.10,34,35 Kittens fed a home prepared and commercial raw food diet had significantly higher globulin levels and red blood cell microcytosis compared to kittens consuming canned heat-processed diet.10 Positive fecal Salmonella Heidelberg and Clostridium difficile toxin was noted in the raw diet groups. Lab work changes were purposed to be associated with known enteropathogenic exposure. Seven of 16 dogs fed a known Salmonella-contaminated single meal shed the bacteria in their feces within 7 days of exposure.35 None of the dogs fed the Salmonella-contaminated meal experienced clinical signs. Feeding a commercial or home prepared RMBD is also a risk factor for antimicrobial-resistant Salmonella spp. and E. coli in the feces of dogs.36 In addition to the zoonotic potential, animals may be clinically affected. Three of twelve cats fed a raw food diet of whole or ground 1-3 day old chicks developed clinical salmonellosis (anorexia and diarrhea).37 Significant differences in the fecal microbiome were noted in the symptomatic cats including the detection of other potentially pathogenic bacteria and increased proportions of other potentially pathogenic bacteria. Salmonella bacteriuria was reported in a cat with lower-urinary tract signs fed a Salmonella-contaminated uncooked granular diet by a company that manufactures RMBDs.38 Septicemic salmonellosis in two cats resulting in death after being fed a diet containing uncooked beef has also been reported.39 In one of the cases, Salmonella newport was isolated and found to be identical to the bacteria isolated in the diet.

Clinical recommendations

The potential for human and animal disease with commercial and home-prepared RMBDs is well documented. Food utensils, feeding bowls, litter boxes, the RMBD, feces and animals with bacteria present in their mouths or on their coat are all sources of potential pathogen exposure for people. The risk of pathogen contamination is particularly a concern among elderly, young, pregnant, lactating, or immunocompromised pets and people. Policies to protect veterinary staff and hospitalized animals from pathogens shed in feces are recommended when treating an animal consuming a RMBD. The FDA also has resources on safe handling tips for pet food and treats and recommendations to owners who choose to feed a RMBD.40 Consultation with a board-certified veterinary nutritionists or individual with similar training for owners wanting to home prepared any diet, cooked or raw, is recommended.

References