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Nutritional Considerations for Animals with Pulmonary Disease

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Respiratory disease can result in malnutrition from anorexia secondary to severe dyspnea or development of a hypermetabolic state secondary to endocrine alterations and cytokine production [1,2]. Malnutrition from deficient nutrient intake may, in turn, adversely affect many factors clinically important to animals with pulmonary disease, including ventilatory drive in response to hypoxia, respiratory muscle mass and function, tissue synthesis or repair, immune competence and incidence of pneumonia, surfactant production, and drug metabolism [3–11]. Many hospitalized animals are likely to become malnourished without appropriate nutrition support, and if malnutrition occurs, it is likely to result in increased morbidity and mortality [12]. As well as having a supportive role, certain nutritional modifications may be used to modulate the underlying disease state. Provision of nutrition support incurs additional cost, however, and is not without potential detriment. Careful consideration of the individual animal and frequent reassessment are required to ensure that optimal nutrition support is maintained. This article focuses on the emerging nutritional therapies and strategies that may prove to be useful in managing small animals with pulmonary disease. Many potential avenues for future research in dogs and cats remain to be investigated.

ANIMAL SELECTION USING NUTRITIONAL ASSESSMENT

It is generally advised to attempt to stabilize the animal before considering nutrition support to minimize the risk of exacerbating existing fluid, electrolyte, and acid-base balance disturbances [2,13]. Even once the animal is deemed sufficiently stable to allow initiation of nutritional support, the clinical and metabolic response should be closely monitored to ensure that adverse sequelae do not go unrecognized. An initial nutritional assessment should be performed as soon as practical after presentation to enable the animal to be classified as malnourished, at risk of becoming malnourished, or well nourished. Nutritional assessment helps with the decision of when to initiate nutritional support

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and also gives direction on diet selection, feeding strategy selection, and monitoring guidelines [14]. The benefits and risks with refeeding, along with the overall prognosis, should be considered when formulating a nutritional strategy. Rather than using potential adverse effects as an excuse not to provide nutrition support, it should be the aim of informed veterinary clinicians to use the modality most likely to supply benefit without significant risk to the individual animal whenever possible. Some initial metabolic abnormalities may actually improve with provision of appropriate nutrition support. For example, preexisting moderate hypertriglyceridemia can resolve with nutrition support, presumably as a result of attenuated endogenous lipolysis with provision of exogenous caloric support. Additional details on performing a thorough nutritional assessment can be found in several recent articles on critical care nutrition [2,13,14]. As a minimum, the nutritional assessment should involve a subjective assessment of the animal's current nutritional status, including assignment of a body condition score and consideration of recent body weight changes, calculation of the current voluntary daily caloric intake using information from the diet history and comparison of this value with the calculated resting energy requirement (RER) for an average animal of that body weight, and consideration of the illness and expected period of anorexia [14]. Nutritional assessment of small animals is currently performed using multiple subjective parameters, because readily available, reliable, and inexpensive objective indicators remain elusive.

NUTRITIONAL GOALS FOR ANIMALS WITH PULMONARY DISEASE

Initial nutritional goals for animals with any critical illness, including those with respiratory disease, include provision of adequate calories to attenuate further breakdown of endogenous tissues (a controlled rate of weight loss can be initiated later if desired in obese animals) and provision of adequate protein to promote a positive nitrogen balance. Although weight stability serves as a surrogate marker for such factors as improved ventilatory drive, greater respiratory muscle strength, improved tissue synthesis or repair, and maintenance of immune competence, the response of these more clinically relevant parameters to nutritional intervention is currently quite difficult to assess in individual patients. Weight stability might also be difficult to assess in animals that have variable hydration status, however. Another important nutritional goal is the provision of a nutrient profile that minimizes the risk of metabolic refeeding complications, most notably hypophosphatemia, hypokalemia, hypomagnesemia, and hyperglycemia. Other considerations for animals with pulmonary disease are discussed in this article in the section on key nutritional factors.

NUTRITION PLAN FOR ANIMALS WITH PULMONARY DISEASE

Formulation of a nutrition plan requires consideration of the feeding method to be used and the type of diet to be provided. The selection of a feeding method is influenced by such factors as the current voluntary daily caloric intake,

anticipated required duration of nutritional support, gastrointestinal function and risk of aspiration pneumonia with enteral feeding, anesthetic risk, coagulation status, available vascular access, fluid tolerance, cost, and type of diet desired [14]. The selection of the type of diet to be provided is influenced by the nutrient levels desired, clinician access to particular diets, type of intravenous access available, the type and size of feeding tube available, the animal's disease condition, cost, and the type of feeding method desired [14]. Because the feeding method and the type of diet affect one another, these two factors must be considered together. Other points to consider when formulating a nutrition plan include whether to use continuous rate or intermittent feeding (continuous rate feeding may be better tolerated by some patients), the rate of introduction of nutrition support (goal daily caloric requirements are generally only achieved after 3 or 4 days of incremental increases), whether consistency of the plan is needed to allow assessment of the animal's response, and whether any treats or supplements to the base diet are desired. Some of the therapies used in animals with respiratory disease may also necessitate modifications to the diet or the feeding method (eg, surgical therapy, ventilatory support).

Enteral nutrition is the preferred route of nutrition support whenever possible because it is more physiologically normal, it assists with maintenance of gastrointestinal mucosal barrier function, it supplies some nutrients directly to the enterocytes, it is less expensive, and it requires less specialized equipment and facilities to administer than parenteral nutrition [11]. Although pulmonary aspiration is a concern in all critically ill patients, the human literature suggests that the risk is insufficient to withhold enteral nutrition support in most cases [15]. There is also some information indicating that provision of parenteral nutrition may directly impair pulmonary macrophage function [16]. Several studies have now shown that early enteral feeding (within 3 days of illness) is associated with improved outcome in human critical care patients [11]. If nasoesophageal or esophagostomy tubes are placed to allow provision of enteral nutrition support to anorexic animals, caution must be exercised to ensure that the tubes or wrapping does not adversely affect patient respiration. If megaesophagus and related aspiration pneumonia are suspected, oral and esophageal feeding should be avoided and gastrostomy feeding techniques may be preferred. When enteral feeding is not possible, such as in anesthetized animals on artificial ventilatory support, in which gastrointestinal motility may be impaired [17], parenteral nutrition support can be initiated as a bridging modality until a transition back to enteral nutrition can be achieved [18,19]. In situations in which only partial enteral nutrition is possible, a combination approach using enteral and parenteral nutrition support concurrently can be used. Drugs, such as cyproheptadine and diazepam, can be tested in an attempt to stimulate appetite, but the potential side effects should be considered. In the experience of the author, drugs have generally proven ineffective in stimulating appetite sufficiently to ensure adequate voluntary daily caloric intake. All standard critical care nutrition feeding methods can be used in animals with pulmonary

disease. Table 1 lists some of the commonly used commercially available diets suitable for critical care nutrition in dogs and cats.

KEY NUTRITIONAL FACTORS FOR ANIMALS WITH PULMONARY DISEASE

Energy

The calculated RER for the current hydrated body weight is often used as the initial estimated daily caloric requirement for critically ill animals. Although, historically, it was recommended to feed at higher caloric intakes (eg, using illness energy requirements, disease factors, or stress factors), more recent veterinary publications advocate feeding at the calculated RER (inclusive of calories from protein) initially, because this value approximates daily caloric requirements of critically ill animals determined using indirect calorimetry, is sufficient to attenuate significant weight loss in most animals, and is believed to reduce many of the other adverse effects of malnutrition [2,13,19]. The exponential formula used by many nutritionists to calculate RER (kcal/d) is $70 \cdot (\text{body weight in kilograms})^{0.75}$. To the author's knowledge, no studies have yet been performed looking at the energy requirements of dogs and cats with pulmonary disease specifically. Previous daily caloric intake (when weight is stable) is often used for dogs and cats that are not critically ill unless weight loss is desired. To determine the previous daily caloric intake accurately, a diet history with foods and amounts consumed (including all treats and supplements) must be obtained. The calculated maintenance energy requirement (MER) for the current body weight can be used if the previously daily caloric intake cannot be calculated from the available diet history (eg, because of ad lib or variable feeding). A full list of multipliers used to calculate the MER from the RER is available in veterinary nutrition texts [20]. The most commonly used MER multipliers are: 1.2 · RER for a neutered cat, 1.4 · RER for an intact cat, 1.6 · RER for a neutered dog, and 1.8 · RER for an intact dog. It should be remembered that individual daily energy requirements can vary markedly from the average values calculated using these formulas; thus, reassessment and adjustment are required if they are used. It is essential that adequate nutrient precursors for synthesis of enzyme cofactors involved in energy production be provided, along with the energy substrates. Among the most likely nutrients to be depleted in critically ill anorexic dogs and cats are the B vitamins, particularly thiamin; thus, care should be taken to ensure that these are supplied at levels greater than the known nutritional requirements [18].

PROTEIN, AMINO ACIDS, AND PRODUCTS OF AMINO ACID METABOLISM

Extended periods of protein malnutrition may result in many adverse effects relevant to animals with pulmonary disease, including reduced immune competence, respiratory muscle weakness, and inadequate tissue synthesis or repair [13]. Studies have shown that provision of adequate nutrition support can

Table 1
A selection of commercially available enteral diets formulated for critical care

Therapeutic diet	Protein (% ME)	Fat (% ME)	Carbohydrate (% ME)	Kilocalories
Hill's Prescription Diet Canine/ Feline a/d canned[a]	33.2	55.2	11.6	180 per can
Royal Canin Veterinary Diet Canine Modified Formula canned[b]	12.9	46.5	40.6	619 per can
Royal Canin Veterinary Diet Canine Modified Formula dry[b]	12.4	32.5	55.1	367 per cup
Royal Canin Veterinary Diet Feline Modified Formula canned[b]	22.8	69.1	8.1	258 or 596 per can
Royal Canin Veterinary Diet Feline Modified Formula dry[b]	22.1	42.4	35.5	432 per cup
Purina Veterinary Diets Canine CV Cardiovascular canned[c]	12.3	53.4	34.3	638 per can
Purina Veterinary Diets Feline CV Cardiovascular canned[c]	32.6	49.8	17.6	223 per can
Purina Veterinary Diets Feline DM Diabetes Management canned[c]	49.0	44.0	7.0	194 per can
Purina Veterinary Diets Feline DM Diabetes Management dry[c]	51.7	37.0	11.3	592 per cup
Eukanuba Veterinary Diets Canine/Feline Maximum-Calorie canned[d]	29.0	66.0	5.0	340 per can
Eukanuba Veterinary Diets Canine Maximum-Calorie dry[d]	31.0	54.0	15.0	634 per cup
Eukanuba Veterinary Diets Feline Maximum-Calorie dry[d]	31.0	54.0	15.0	602 per cup
Abbott Animal Health Canine/ Feline Clinicare liquid[e]	30.0	45.0	25.0	1 kcal/mL
Abbott Animal Health Feline Clinicare RF liquid[e]	22.0	57.0	21.0	1 kcal/mL
Ross Human Vital HN powder (reconstituted to liquid)[e,f]	16.7	9.5	73.8	1 kcal/mL
Abbott Laboratories Human Pulmocare liquid[e,f]	16.7	55.1	28.2	1.5 kcal/mL

Current as of September 1, 2006.
[a]Hill's Pet Nutrition, Inc., Topeka, Kansas.
[b]Royal Canin USA, Inc., St. Charles, Missouri.
[c]Société des Produits Nestlé S.A., Vevey, Switzerland.
[d]The Iams Company, Dayton, Ohio.
[e]Abbott Laboratories, Abbott Park, Illinois.
[f]Human diets are not complete and balanced for long-term feeding to dogs and cats without appropriate supplementation.

improve nitrogen balance [21,22]. Dietary protein should be adequate to minimize catabolism and maintain metabolic protein demands. Most authors recommend that protein be provided to critically ill dogs and cats at a rate of 3 to 8 g per 100 kcal depending on species and disease state [2,13], a level

that supplies approximately 10% to 32% of the total metabolizable energy (ME) as protein. Dogs and cats that have only pulmonary disease, without concurrent azotemia or hepatic encephalopathy, may safely be fed diets with protein contents at the higher end of this range, thus reducing the dependence on fat or carbohydrate to provide calories. Other authors have suggested that critically ill dogs should receive 25% to 45% of their total ME as protein and that critically ill cats should receive 30% to 50% of their total ME as protein unless the individual has uremia, hepatic encephalopathy, or excessive protein losses [12].

In addition to the total protein content of the diet, some individual amino acids and products of amino acid metabolism are worthy of specific consideration. All canine and feline enteral diets contain arginine, an essential amino acid that has been shown to improve nitrogen balance and immune function [13]. Supplementation of additional arginine greater than the known nutritional requirement to dogs and cats with pulmonary disease is of questionable benefit at this stage. Glutamine, a conditionally essential amino acid for dogs and cats, may be of benefit during periods of stress, but its expense and relative instability currently limit its use in veterinary patients [13]. The branched chain amino acids leucine, isoleucine, and valine can be metabolized directly in muscle tissue rather than in the liver and may supply an additional source of energy as well as having regulatory actions [12]. Research into the clinical utility of supplementing branched chain amino acids to critically ill animals is deficient at this time. L-carnitine, usually synthesized endogenously from lysine and methionine, is required for transport of long-chain fatty acids across the mitochondrial membrane for subsequent β-oxidation. Whether providing additional L-carnitine to animals with pulmonary disease already consuming diets with adequate levels of total protein, lysine, and methionine is of any benefit remains to be determined.

Electrolytes

Refeeding of previously anorexic patients, particularly with diets high in rapidly absorbed and metabolized carbohydrates, can result in hypophosphatemia, hypokalemia, and hypomagnesemia as part of the refeeding syndrome [18, 23–27]. These electrolyte alterations occur secondary to preexisting whole-body electrolyte depletion and intracellular movement with glucose uptake and glycolysis [18]. Refeeding syndrome can occur with parenteral, enteral, or oral feeding [18]. Hypophosphatemia is believed to result in clinical signs, including muscle weakness and respiratory insufficiency in human beings [25,26]. Hypokalemia and hypomagnesemia can also be associated with generalized muscle weakness that could exacerbate preexisting respiratory muscle dysfunction [26,28–32]. Sodium retention can occur as part of the refeeding syndrome and may exacerbate pulmonary edema if present [18]. As such, it is prudent to correct electrolyte abnormalities routinely before feeding and to monitor for electrolyte shifts with refeeding in all animals that may have experienced whole-body electrolyte depletion from extended periods of anorexia or prolonged ingestion of unbalanced diets. A recent human study indicated that

instituting a routine electrolyte replacement protocol when refeeding patients requiring nutrition support alleviated the clinical consequences of refeeding syndrome [33].

Carbohydrate

Nutritional hypercapnia is an important complication recognized in human patients who have respiratory disease [9,34–37]. Initiation of routine nutritional support in human patients has been shown to increase endogenous carbon dioxide production, which can, in turn, necessitate introduction of or adjustments to such therapies as artificial ventilation [38,39]. Alteration of the nutrient profile of the diet can affect the respiratory quotient (RQ; ratio of moles of carbon dioxide produced to moles of oxygen consumed) that results from metabolism of energy substrates in the diet. The RQ that results from carbohydrate metabolism is 1.0, indicating that similar amounts of carbon dioxide are produced as oxygen is consumed. The RQ that results from metabolism of an average animal fat is 0.7, and the RQ that results from metabolism of an average meat protein is 0.8, indicating that less carbon dioxide is produced per unit of oxygen consumed [18]. When consideration is given to the actual volume of carbon dioxide produced per unit of energy generated, however, the numbers are different. Carbohydrate and protein are relatively equivalent, at approximately 200 L and 209 L of carbon dioxide produced per 1000 kcal, respectively, whereas the amount of carbon dioxide produced per unit of energy generated when fat is metabolized is markedly lower at approximately 155 L per 1000 kcal (assuming that the carbon dioxide behaves as an ideal gas and no energy storage occurs) [40]. Because of this, the level of carbohydrate is often restricted and the level of fat is increased as the source of nonprotein calories in the diet of people who have respiratory disease. A controlled study of human patients receiving assisted ventilation showed that feeding a low-carbohydrate diet rather than a standard diet resulted in reduction of $PaCO_2$ and earlier weaning from the ventilator [34]. Provision of caloric intake beyond the animal's actual daily energy requirement may also result in undesirable alterations in the RQ, particularly if high-carbohydrate diets are used, because of increased carbon dioxide production with endogenous fat synthesis [11,13,41–47]. To the author's knowledge, studies reporting expired gas analysis of dogs and cats with pulmonary disease fed variable diet compositions and caloric loads have yet to be reported. A recent human study indicated that the RQ determined by indirect calorimetry could be used as a marker of respiratory tolerance to the nutrition support regimen, but the low sensitivity and specificity in detecting underfeeding or overfeeding of this analysis limit its use in fine-tuning the regimen [44].

Pulmocare (Abbott Laboratories, Abbott Park, Illinois) is an example of a human enteral product formulated specifically for patients who have respiratory disease. This product has a high calorie density (1.5 kcal/mL); low carbohydrate content for a human diet (28.2% carbohydrate on a ME basis); and added antioxidants, including vitamin C, vitamin E, and β-carotene. It should be noted that

although the level of carbohydrates in this product would be considered restricted for people and dogs, it would not be considered restricted for cats, because many available feline diets contain 20% to 40% carbohydrate on a ME basis. It should also be remembered that human enteral products usually do not meet the full nutritional requirements of dogs and cats; as such, they are not suitable for extended feeding periods unless supplemented appropriately. Restriction of the carbohydrate content of the diet may also reduce the risk of the animal experiencing extended periods of hyperglycemia. This is desirable, because unmanaged hyperglycemia may result in reduced immune competence and has been shown to increase mortality in human intensive care patients [18,48–52].

Fat and Fatty Acids

Consumption of high-fat diets (>40% fat on a ME basis) may have additional benefits beyond reduction in the carbohydrate load, with such factors as higher energy density (kcal per unit of weight) and palatability also important for many critically ill animals [12]. Animals rapidly deplete body glycogen stores and are reliant on protein and fat metabolism for energy after a few days of anorexia. Because some dogs and cats may exhibit fat intolerance when fed high-fat diets, however, they must be monitored for diarrhea, pancreatitis, lipemia, or hypertriglyceridemia. Consideration should be given to the lipids and calories administered concurrently in fat-containing medications, such as a propofol infusion [18]. In addition, hypertriglyceridemia can result from carbohydrate overfeeding in patients receiving parenteral nutrition [18].

It may be possible to improve lung function by providing specific fatty acids that attenuate lung inflammation. Certain polyunsaturated fatty acids (PUFAs), such as Ω-3 PUFA eicosapentaenoic acid (EPA) and Ω-6 PUFA gamma-linolenic acid (GLA), have shown promise as immunomodulatory supplements in many species and disease conditions, including people who have acute respiratory distress syndrome [53]. Recent studies have shown that administration of an enteral nutrition formula supplemented with EPA, GLA, and antioxidants reduced pulmonary inflammation, improved gas exchange and tissue oxygenation, reduced the requirement for artificial ventilatory support, reduced the time spent in the intensive care unit, and reduced the frequency of development of new organ failure [53–56]. These changes are possibly attributable to anti-inflammatory, vasodilatory, or antioxidant effects. In contrast, a recent review of the effects of Ω-3 PUFA supplementation in people who have asthma found few significant beneficial effects [57]. Incorporation of medium-chain triglycerides into the lipid solution may also be of value for patients who have pulmonary disease receiving parenteral nutrition [18,58]; however, additional studies are needed to evaluate further whether any significant clinical effects can be obtained with this modification.

Antioxidants

The antioxidant content of the diet may also require consideration in animals with pulmonary disease. The lungs are continually exposed to relatively high

oxygen concentrations and can also be subjected to a variety of environmental pollutants and irritants that generate reactive oxygen species, resulting in increased vulnerability to oxidant attack [59]. Inflammatory cells activated in the lung with infection or inflammation may also generate free radicals and oxidant injury [60]. Therapies used for respiratory conditions, such as oxygen therapy, chemotherapy, and radiation therapy, can also result in endogenous free radical generation. It has been shown that oxidized lipids in the diet of growing dogs may affect some measured parameters of antioxidant status and immune function [61]. A variety of antioxidant protective mechanisms are present in the lung but vary markedly among individuals [59]. There is increasing evidence in people that consumption of foods high in antioxidant vitamins may impart a protective effect on lung function [54–56,59,62–70]. The mechanism of this protective effect is speculated to involve maintenance of adequate antioxidant nutrient concentrations within the lung to prevent oxidant damage [54,59]. It is also possible that initial oxidative stress may initiate subsequent increases in the antioxidant protective mechanisms maintained by an individual [71].

The ideal combination and concentration of antioxidant nutrients in the diet of dogs and cats with various pulmonary conditions have yet to be determined, but a recent study in obese cats has shown that administration of a D-α-tocopherol–supplemented parenteral solution resulted in a higher concentration of red blood cell glutathione than in control cats given standard parenteral solutions [72]. It should not be forgotten that reactive oxygen species can be important for destroying microorganisms and intracellular signaling; thus, excessive suppression of their generation is not desirable [73]. Excessive supplementation of particular antioxidants may result in direct cell signaling effects or pro-oxidant effects and should be avoided until the clinical consequences of these additional effects are further understood [74,75].

PREBIOTIC AND PROBIOTIC CONTENT

Some authors are now speculating that lifelong or intermittent supplementation of diets with prebiotics or probiotics, already recognized to stimulate enterocyte and colonocyte proliferation, may support the development of normal immunologic mucosal tolerance, thus reducing the risk of allergic airway disease [76]. The clinical relevance of this modification in dogs and cats with pulmonary disease remains to be determined.

OTHER NUTRIENTS

All other nutrients should be supplied at levels sufficient to meet the known minimum requirements. Long-term administration of diets deficient in any nutrients known to be essential to dogs and cats is eventually likely to contribute to morbidity and mortality. For example, zinc deficiency may affect protein metabolism, resulting in impaired tissue synthesis or repair and reduced immune competence [12]. Copper and vitamin A deficiencies have also been reported to affect lung parenchymal tissue adversely [11]. Taurine deficiency may allow

terminal activation and release of cytotoxic mediators from lung macrophages in cats [77]. Therefore, it is desirable to feed a diet known to be complete and balanced for the species being treated. If animals are fed at daily caloric intakes lower than their calculated RER for extended periods, consideration should be given to provision of a diet with higher concentrations of the key nutrients or supplementation of these nutrients in addition to the reduced dietary intake.

WEIGHT MANAGEMENT

Weight management is an important long-term consideration in overweight or obese animals with pulmonary disease. Obesity is one of the most common forms of malnutrition in dogs and cats and can be evaluated clinically by using body condition scoring or morphometric measurements [78]. Obesity has been established to have negative effects on the health and longevity of some breeds of dogs and may be associated with cardiorespiratory disease in individual animals [79–84]. Smaller and lighter breeds of dogs generally live longer than larger and heavier breeds; thus, whether the effect of obesity on longevity is present across all dog breeds remains to be determined. Obesity was also one of the factors associated with mortality in dogs with heat stoke in a recent study [85]. Some authors have also indicated that obesity may alter drug metabolism, requiring alterations in medication dosages to achieve therapeutic levels [86,87]. Whole-body plethysmography in dogs has demonstrated that obesity increases respiration rate, increases minute volume, reduces respiratory tidal volume, reduces inspiratory time, and reduces expiratory time [88,89]. Preliminary studies suggest that obesity increases airway reactivity to histamine and impairs the positive ventilatory response to doxapram hydrochloride [88,89]. There is increasing literature to indicate that adipose tissue produces several endocrine factors that may have proinflammatory properties [90]. Therefore, there are several possible reasons why obesity management can be beneficial for animals with pulmonary disease. Obesity-hypoventilation syndrome (Pickwickian syndrome) in human beings is characterized by chronic alveolar hypoventilation (often without hypercapnia) because of increased respiration workload, dysfunction of the respiratory centers, and repeated episodes of sleep apnea [91–93]. This condition is reversible with weight loss in people, and although the syndrome has not been described specifically in veterinary medicine, it is likely relevant to small animal patients.

An effective weight loss plan should encompass an initial animal evaluation and client education process, selection of an appropriate diet and feeding strategy, implementation of an appropriate exercise program, and regular reassessment with adjustments as needed to ensure an adequate rate of weight loss until the animal can be transitioned back to a maintenance diet. Canine and feline obesity was discussed in detail in a recent edition of this journal [90]. For reader convenience, tables of the commercially available canine and feline diets suitable for active weight loss are provided with this article (Tables 2 and 3). Pet food manufacturers often make adjustments to their diets over time; thus, it would be appropriate to recheck the information contained within these

Table 2
Commercially available canine diets formulated for active weight loss

Therapeutic diet	Protein (% ME)	Fat (% ME)	Carbohydrate (% ME)	Fiber (g/1000 kcal)	Moisture (% as fed)	Density (g per can or cup)	Kilocalories
Hill's Prescription Diet Canine r/d canned[a]	29.7	24.6	45.7	71	78	404	296 kcal per 14.25-oz can
Hill's Prescription Diet Canine r/d dry[a]	29.8	24.9	45.3	78	11	82	220 kcal/cup
Purina OM Canine Formula canned[b]	51.2	23.6	25.2	77.7	82	354	189 kcal per 12.5-oz can
Purina OM Canine Formula dry[b]	34.9	17.7	47.4	34.6	12	101	276 kcal/cup
Royal Canin Canine CC HP canned[c]	42.6	53.1	4.3	6.3	84.8	360	263 kcal per 12.7-oz can
Royal Canin Canine CC 32 HP dry[c]	37.6	23.5	38.9	8.5	8.5	66	234 kcal/cup
Royal Canin Canine CC HF canned[c]	25.3	29.6	45.1	24.5	75.6	360	346 kcal per 12.7-oz can
Royal Canin Canine CC 26 HF dry[c]	34.4	28.1	37.5	56	8.5	81	232 kcal/cup
Eukanuba Canine Restricted Calorie canned[d]	31	39	30	5.4	78	397	445 kcal per 14-oz can
Eukanuba Canine Restricted Calorie dry[d]	24	17	59	5.1	10	65	238 kcal/cup
Pedigree Canine Weight Loss dry[e]	53	21	26	8.9	12	73	246 kcal/cup

Current as of September 1, 2006.
[a]Hill's Pet Nutrition, Inc., Topeka, Kansas.
[b]Société des Produits Nestlé S.A., Vevey, Switzerland.
[c]Royal Canin USA, Inc., St. Charles, Missouri.
[d]The Iams Company, Dayton, Ohio.
[e]Mars, Incorporated, Hackettstown, New Jersey.

Table 3
Commercially available feline diets formulated for active weight loss

Therapeutic diet	Protein (% ME)	Fat (% ME)	Carbohydrate (% ME)	Fiber (g/1000 kcal)	Moisture (% as fed)	Density (g per can or cup)	Kilocalories
Hill's Prescription Diet Feline r/d canned[a]	38.2	25.2	36.6	55	78	156	116 kcal per 5.5-oz can
Hill's Prescription Diet Feline r/d L&C canned[a]	41.3	24.5	34.2	50	78	156	114 kcal per 5.5-oz can
Hill's Prescription Diet Feline r/d dry[a]	40.4	24.9	34.7	41	11	88	263 kcal/cup
Hill's Prescription Diet Feline m/d canned[a]	45.7	40.7	13.6	15	78	156	156 kcal per 5.5-oz can
Hill's Prescription Diet Feline m/d dry	43.0	44.1	12.9	13	10	122	480 kcal/cup
Purina OM Feline Formula canned[b]	43.1	34.4	22.5	26	77	156	150 kcal per 5.5-oz can
Purina OM Feline Formula dry[b]	56.2	20.5	23.3	17.6	11	105	340 kcal/cup
Royal Canin Feline CC HP canned[c]	45.4	46.5	8.1	5.1	83.4	165	130 kcal per 5.8-oz can
Royal Canin Feline CC HP pouch[c]	43.9	36	20.1	7.7	82.3	85	66 kcal per 3-oz pouch
Royal Canin Feline CC 38 HP dry[c]	44.4	23.4	32.2	11.2	7	68	235 kcal/cup
Royal Canin Feline CC HF canned	28.4	44	27.6	18.7	76.6	170	164 kcal per 6-oz can
Royal Canin Feline CC 29 HF dry[c]	36.1	26.7	37.2	43	7	83	251 kcal/cup
Eukanuba Feline Restricted Calorie canned[d]	40	41	19	2.1	87	170	204 kcal per 6-oz can
Eukanuba Feline Restricted Calorie dry[d]	34	23	43	5.4	8.5	78	277 kcal/cup

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[a]Hill's Pet Nutrition, Inc., Topeka, Kansas.
[b]Société des Produits Nestlé S.A., Vevey, Switzerland.
[c]Royal Canin USA, Inc., St. Charles, Missouri.
[d]The Iams Company, Dayton, Ohio.

tables to ensure that it is still accurate before use. Dogs and cats can be started at a total daily caloric intake equivalent to 80% of their previous daily caloric intake when weight is stable. If the previous daily caloric intake cannot be calculated from the diet history because of ad lib or variable feeding, dogs can be started at a total daily caloric intake equivalent to the calculated RER using the formula RER (kcal/d) = 70 · (body weight in kilograms)$^{0.75}$, and cats can be started at a total daily caloric intake equivalent to 80% of the calculated RER using the same formula. Up to 10% of total daily calories can be provided as unbalanced treats (commercial pet treats and human foods) without risk of unbalancing the base diet. As well as reducing the total daily caloric intake, an increase in the daily energy expenditure is usually advised for animals that are able to tolerate exercise. Regardless of the initial total daily caloric intake suggested, it is critical to recheck the rate of weight loss regularly to ensure that an appropriate rate is achieved. A rate of weight loss from 0.5% to 2% of current body weight per week is considered reasonable, with slower rates suggested for animals that may be metabolically unstable. Once the desired clinical response and body condition score have been achieved, the animal may be transitioned to a low-calorie/light/lite maintenance diet designed to assist with preventing recurrent weight gain.

GASTROINTESTINAL DISEASE MANAGEMENT

Diet can also be considered a potential source of allergens or irritants manifesting as respiratory disease [94]. Recently published studies have suggested a relation between respiratory disease and gastrointestinal disease (diagnosed by endoscopy and histopathologic examination) in brachycephalic dogs [95,96]. Possible contributors to this relation suggested by the authors include pharyngeal inflammation secondary to the gastrointestinal disease or gastroesophageal reflux secondary to the respiratory disease. The detection and treatment of concurrent gastrointestinal disease have been proposed to improve the results of upper airway surgery in brachycephalic dogs with respiratory disease [95], although a study with a suitable control group would be needed to prove benefit. Although the exact mechanism behind this relation remains to be conclusively determined, the potential benefit justifies assessment for the concurrent presence of gastrointestinal disease in animals with respiratory disease, and appropriate management (dietary and medical) should be instituted.

MONITORING AND REASSESSMENT

Regular monitoring for metabolic, mechanical, and septic complications is essential to ensure that modifications are made to the nutrition plan over time to provide maximal benefit and minimal risk to the individual animal. It is also important to record the current nutrition plan and any complications in the animal's medical record, such that this information is available when making alterations to the nutrition plan. Along with the amount of food offered, it is important to record the actual amount consumed and any regurgitation or vomiting that occurs.

SUMMARY

Many dogs and cats with pulmonary disease benefit from timely nutritional assessment and provision of appropriate nutrition support. The exact nature of the support required varies depending on the severity and type of pulmonary disease. Obese animals with chronic respiratory disease may benefit from weight loss, whereas animals with acute pulmonary disease may experience malnutrition and require aggressive nutrition support interventions. Standard critical care feeding methods can be used in animals with pulmonary disease, but selection or formulation of a diet with specific modifications may reduce morbidity and mortality. Key nutritional factors to consider include energy, protein, fat, carbohydrate, specific amino acids, specific fatty acids, electrolytes, antioxidants, prebiotic and probiotic content, weight management, and gastrointestinal disease management. Regular monitoring and reassessment of the nutrition plan is needed to ensure that the animal obtains optimal support. Many avenues remain to be investigated in the field of nutrition for animals with pulmonary disease; thus, additional data to assist with selection of the feeding method and the diet are likely to become available in the future.

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