

Vet Clin Small Anim 35 (2005) 713–742

VETERINARY CLINICS Small Animal Practice

Nutrition for Aging Cats and Dogs and the Importance of Body Condition

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The average age of pet dogs and cats continues to increase such that between one third and one half of pet dogs and cats are 7 years of age or older [1]. In the United States, there has been a nearly twofold increase in the percentage of pet cats older than 6 years of age (from 24% to 47%) over the past 10 years [2]. Likewise, in Europe the number of dogs considered to be "senior" (>7 years of age) increased by approximately 50%, whereas the number of cats older than 7 years of age increased by over 100% between 1983 and 1995 [3].

Aging brings with it physiologic changes. Some changes are obvious, such as whitening of hair, a general decline in body and coat condition, and failing senses (sight and hearing). Other changes are less obvious, however, and these include alterations in the physiology of the digestive tract, immune system, kidneys, and other organs. Of course, pets, like people, do not age consistently, and chronologic age does not always match physiologic age. Although many pets remain active and youthful well into their teens, most dogs start to slow down and may show signs of aging beginning as early as 5 or 6 years of age. The aging process is influenced by breed size, genetics, nutrition, environment, and other factors. As a general rule, dogs and cats 7 years of age or older, which is the age when many age-related diseases begin to be more frequently observed, may be considered to be "at risk" for age-related problems [3]. "Geriatric" screening should be considered as a preventive medicine service conducted to identify diseases in their early stages or to head off preventable diseases. An important part of this evaluation is a thorough nutritional assessment.

Nutritional requirements can change with age. In addition, many diseases common in older dogs and cats may be nutrient-sensitive, meaning that diet can play an important role in the management of the condition. This article

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discusses the impact of aging on nutritional requirements, reviews patient nutritional evaluation, and then addresses some common nutrition-related problems in older dogs and cats.

Effects of aging on nutritional requirements

Energy needs

Maintenance energy requirements (MERs) are the energy needs required for the normal animal to survive with minimal activity. Individual MERs can vary based on genetic potential, health status, and whether the animal is sexually intact or neutered. In addition to these factors, MERs seem to decrease with age in human beings, rodents, and dogs [4,5]. In one study involving English Setters, Miniature Schnauzers, and German Shepherd dogs, the MERs of 11-year-old dogs were approximately 25% less than those of breed-matched 3-year-old dogs [5]. Others have reported an 18% to 24% decrease in MERs of older dogs across various breeds [4]. The greatest decline seemed to occur in dogs older than 7 years of age [6].

Age-related changes in MERs in cats are more controversial. Some report no change in MERs with age when evaluated in short-term studies [4]. When MERs were evaluated over a longer period (3–12 months), however, a different picture emerged. Based on data from more than 100 cats ranging in age from 2 to 17 years, MERs decreased with age in cats through approximately 11 years of age (Fig. 1) [7]. Based on a subset of these cats for

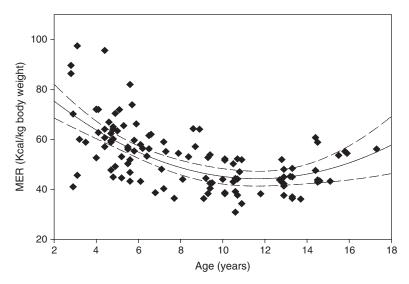


Fig. 1. Effect of age on maintenance energy requirements (MERs) of adult cats. MERs decrease until approximately 11.5 years and then increase. The lines shown represent the second-order regression with a 95% confidence interval: MERs (kcal/kg/d) = 89.576 - $[7.771 \cdot \text{Age} (\text{years}) + 0.334 \cdot \text{Age} (\text{years})^2]; r^2 = 0.34; P < 0.001.$

which repeated measures were available, MERs decreased approximately 3% per year. By approximately 12 years of age onward, however, MERs per unit of body weight actually increased. This increase was confirmed in another study involving 85 cats between 10 and 15 years of age [8]. MERs increased throughout this age group, with the greatest increases occurring after 13 years of age.

A primary driver of basal metabolic rate, hence MERs, is lean body mass (LBM). The LBM, which includes skeletal muscle, skin, and organs, contains most metabolically active cells and accounts for approximately 96% of basal energy expenditure [9]. With exercise, the contribution of muscle and LBM to energy needs increases further. Across species, including dogs and cats, LBM tends to decrease with age [10,11]. This, plus a decrease in activity, can contribute to the reduction in MERs seen in aging dogs and middle-aged cats.

If energy needs decrease in a pet and energy intake does not decrease accordingly, the animal becomes overweight. It is this last point that drives the market position of many foods for older dogs and cats. Most commercial foods for geriatric pets contain a reduced concentration of dietary fat and calories. Some have dietary fiber added to reduce the caloric density further. These products may be appropriate for the large number of pets that are overweight or likely to get that way. If energy intake is not managed appropriately, dogs and cats may become overweight and subject to associated health risks. Arthritis and diabetes, for example, which are common in older dogs and cats, are aggravated by excess body weight.

Not all older animals are overweight or less active. In fact, although "middle-aged" animals tend to be overweight, a greater proportion of dogs and cats older than 12 years of age are underweight compared with other age groups [12]. This effect is especially pronounced in cats. In addition to an increase in MERs in this age group, which may partly explain weight loss, recent research has identified that older cats may experience a reduction in digestive capabilities.

Earlier research in our laboratory indicated that older cats retain their digestive capabilities [13]. That study was done with young adult and middle-aged cats, however (>8 years of age). The only significant differences in digestive function indicated a slight increase in carbohydrate digestibility in older cats. More recently, however, an evaluation was completed to look at a broader range of ages. Consequently, it was shown that fat digestibility decreases with age in a large number of geriatric cats [11]. The prevalence of compromised fat digestibility increases with age and affects approximately one third of cats older than 12 years of age (Fig. 2) [11]. In addition, approximately 20% of cats older than 14 years of age have a reduced ability to digest protein. Reduced protein digestion or fat digestion could contribute to weight loss in aging cats.

These patients as well as others that are underweight may benefit from a more energy-dense highly digestible product to help compensate for these

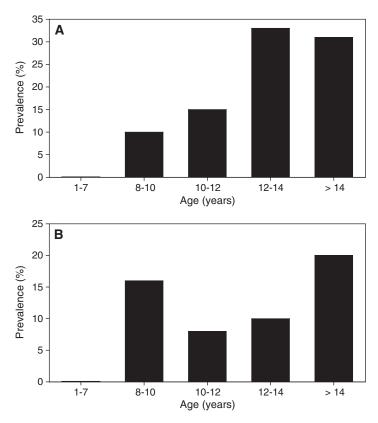


Fig. 2. Prevalence of low fat (*A*) or protein (*B*) digestive function in cats by age. Low fat digestibility was defined as less than 80% digestible compared with normal digestibility of 90% to 95%. Low protein digestibility was defined as less than 77% digestible compared with normal digestibility of 85% to 90%. (*Data from* Perez-Camargo G. Cat nutrition: what's new in the old? Compend Contin Educ Pract Vet 2004;26(Suppl 2A):5–10.)

age-related changes. A nutritional assessment should be completed on each patient to determine its individual needs rather than assuming that all older pets need reduced calorie intake.

Protein needs

Protein is another nutrient of extreme importance for aging pets. In the past, many veterinarians have recommended protein restriction for older dogs in the mistaken belief that this would help to protect kidney function [14]. More recent research has unequivocally demonstrated that protein restriction is unnecessary in healthy older dogs [15–17]. On the contrary, protein requirements sufficient to support protein turnover actually increase in older dogs [18].

In a classic study comparing the protein requirements of young and old Beagles, the older dogs required approximately 50% more protein than young adult dogs to maintain nitrogen balance and maximize protein reserves [18]. In addition, protein turnover was reduced in the older dogs, even at the highest level of protein fed.

The current standard for establishing adult protein (nitrogen) requirements is the nitrogen balance method, which compares nitrogen intake with nitrogen output. Maintenance of LBM or measures of protein turnover provide better indicators of protein adequacy compared with nitrogen balance studies, however. Protein turnover is the cycle of catabolism of endogenous protein and synthesis of new proteins needed by the body at any given time, including hormones, enzymes, immune proteins, and others. When dietary protein intake is insufficient, the body responds by decreasing catabolism and synthesis and mobilizing protein from LBM to support essential protein synthesis. Normal animals can adapt to this low-protein intake and maintain nitrogen balance yet be in a protein-depleted state. In this situation, animals may appear healthy but have a decreased ability to respond to environmental insults, including infections and toxic substances [18]. In addition to the direct effect of inadequate protein intake, aging has a detrimental effect on protein turnover. In one review, 85% of the studies found an age-related decline in endogenous protein synthesis [19]. In otherwise healthy animals, even mild protein deficiency can significantly impair immune function [20,21]. These effects may be more pronounced in the older dog because of the reduced LBM and age-related reduction in protein turnover.

In dogs, it took three times as much protein to maintain protein turnover than that needed to maintain nitrogen balance in old and young dogs, with older dogs needing more protein than young dogs [18]. Approximately 3.75 g of casein protein per kilogram of body weight per day was required for older Beagles compared with 2.5 g/kg/d for young adult Beagles. A more recent study also showed that dogs could maintain nitrogen balance on protein as low as 16% of energy but that protein turnover was maximized in young and old dogs when protein was increased to 32% of energy [22].

Actual protein needs may vary based on individual factors, such as breed, lifestyle, health, and individual metabolism. In addition, calorie intake affects dietary protein need. With lower calorie intake, the percent of calories as protein must increase to maintain the same protein intake. Older dogs tend to consume fewer calories, and thus less food, than younger dogs. Therefore, diets for older dogs should contain a higher percentage of dietary protein, or an increased protein-to-calorie ratio, to meet their needs. Diets containing at least 25% of calories from protein should meet the protein needs of most healthy senior dogs.

Similar data showing an age effect in cats are lacking; however, cats of all ages have high protein requirements. Similar to other species, cats need considerably more protein to maintain LBM than needed to maintain

nitrogen balance. Based on assessment of LBM, adult cats need more than 5 g of protein per kilogram of body weight, or approximately 34% of their dietary calories as protein, to support lean body mass and protein turnover [23].

Other nutrients

All dogs and cats have specific needs for vitamins and minerals, which are normally provided by complete and balanced diets. There is little, if any, evidence that the requirements for these nutrients differ in healthy older animals. Patients with subclinical disease associated with a mild malabsorption syndrome or polyuria may have increased losses of water-soluble nutrients, such as B vitamins, or fat-soluble nutrients, such as vitamins A and E, however. As noted previously, approximately one third of geriatric cats have a reduced ability to digest dietary fats. In these cats, there is a significant correlation between fat digestibility and the digestibility of other essential nutrients, including several B vitamins, vitamin E, potassium, and other minerals [24]. Geriatric cats with gastrointestinal disease are more likely to be deficient in cobalamin (vitamin B_{12}) compared with younger cats [25]. Thus, older cats should be carefully evaluated for possible nutrient deficiencies and may benefit from supplemental amounts of these nutrients.

Oxidative damage plays an important role in many diseases of aging, including arthritis and other inflammatory diseases, cancers, neurologic disease, cardiovascular disease, and others [26–33]. There even exists a popular theory suggesting that "aging" is induced by an imbalance between free radical production or exposure and the body's antioxidant defenses [27]. Certainly, a deficiency of antioxidant nutrients can have detrimental effects on in vivo antioxidant function, immune function, and markers of health [26,34,35]. In addition, adequate dietary protein is critical to support endogenous glutathione production, a key antioxidant for disease prevention [33].

Considerable evidence in human beings and animals suggests that dietary antioxidants may provide some protection against oxidative stress and normal aging processes [27,33,35,36]. Numerous studies on antioxidants in dogs or cats have reported beneficial effects on markers of oxidative status [37–41]. It is difficult to show clear cause-and-effect relations between the diseases and antioxidant status, however, because oxidant damage is subtle and difficult to measure and the associated diseases develop slowly over many years [28]. Given the weight of available information, it is reasonable to recommend or provide increased amounts of antioxidant nutrients for aging dogs and cats.

Geriatric nutritional evaluation

Before instituting a dietary change in any patient, especially an older dog or cat, a thorough nutritional evaluation should be completed. This should include an evaluation of the patient, the current diet, and feeding management. The goal of dietary history taking is to identify the presence and significance of factors that put patients at risk for malnutrition. Understanding how the nutritional needs of older animals may change and a thorough evaluation of the individual patient allow an appropriate dietary recommendation. Such recommendations should take into account the needs of the patient and client preferences.

Changes in feeding management should be considered part of total patient management. As with any aspect of medical management, the patient should be re-evaluated at appropriate intervals to ensure achievement of desired results.

Patient evaluation

A complete medical history should be assessed, including vaccination history, heartworm and flea preventive methods, and any prior diseases. A thorough physical examination should be conducted, including body weight and body condition score (BCS), oral examination, digital rectal examination, and evaluation of the skin and hair coat. Thin and brittle hair or dry and flaky skin can have many causes but may be a sign of nutritional deficiencies. A comprehensive geriatric evaluation may include the following blood, urine, and fecal analyses: complete blood cell count (CBC); platelet count; biochemical profile; serum bile acids analysis; and complete urinalysis, including sediment examination, urine protein/creatinine ratio, and fecal flotation. Although these tests are not sensitive nutritional indicators, abnormalities may provide evidence of clinical or subclinical problems that may benefit from dietary modification. For example, anemia, low serum albumin, low potassium, increased serum urea nitrogen, increased triglycerides or cholesterol, or increased serum glucose may indicate problems that could benefit from dietary modification as part of medical management.

Increases or decreases in body condition should trigger further evaluation. If weight loss is evident (from the physical examination or the medical history), further evaluation should determine if this is associated with increased or decreased calorie intake. A detailed dietary history and evaluation are warranted. If the patient shows an increase in or excessive BCS, it is again important to consider current diet and feeding management. Older dogs and middle-aged cats tend to have reduced energy needs. If calorie intake is not adjusted accordingly, weight gain results. Unexplained weight gain should be evaluated for predisposing causes, such as hypothyroidism. Animals that are overweight are likely to benefit from weight reduction.

Dietary evaluation

A complete dietary evaluation must include everything that is consumed. One approach to gathering this information is to have the client complete a written dietary history form (Fig. 3). The diet history should include the normal diet as well as other foods the pet has access to. Commercially prepared

Dietary Information Form	Date:
Owner's Name:	Pet's Name:
Species : Dog Cat Other	Breed: Pet's Gender: M MC F FS
Age: Body Weight*:	Body Condition Score: Activity: High Med Low Very Low
Diet History: What food(s) are currently fed - r	nain meal:
Dry Food:never occasi	onal/ small proportionabout half mostly exclusively
If fed, what brands and amounts are	fed most often:
Canned Food: o	ccasional/ small proportionabout half mostly exclusively
If fed, what brands and amounts are	fed most often:
Home prepared foodsnever	occasional/ small proportion half mostly exclusively
If fed, please provide recipes used.	
What treats and/or supplements a	re currently fed?
Commercial treats: No	Yes. What brands and amounts are fed most often:
Fresh foods or table scraps:No	oYes. What foods and amounts are fed most often:
Dietary supplements:No	_Yes. What supplements and amounts are fed most often:
Have there been recent changes in for	oods/brands fed? No Yes. If so, when and why?
How is your pet's appetite	ood Poor Any recent changes?
	ate:0-1/day2 -3 /day4 or more/day don't know
	stool? Mostly:firm/hardformed but not hard loose
	his time: IndoorsOutdoorsAbout half in and half out
	d walking, playing or running each day? : < 30 min/day 30 - 60 min/day
1 to 3 hours/day	> 3 hours/day mostly inactive but > 3 hours/day once or twice per week
Are there other pets in your househo	old? Yes No
Do you have any questions regardin	g your pet's diet?

Fig. 3. Diet history form.

foods should be identified by brand. If needed, manufacturers can be contacted to obtain product information, such as typical calorie and nutrient content, digestibility, and other details. Any changes to the diet should be identified as well as the reason for the change. Because many pet owners provide treats and human table food "samples" for their pets, these also should be identified by types and amounts. Clients may not consider nutritional supplements part of the diet, so they should be asked specifically about these.

Once the nutritional characteristics of the total diet are known, they should be compared with the individual patient's needs. In general, inactive animals or those that are somewhat overweight should be receiving lower calorie foods yet may need foods with an increased nutrient-to-calorie ratio formulated to compensate for increased needs of other nutrients. Feeding such animals a high-calorie food may require an inappropriate reduction in the volume of food, resulting in lack of satiation as well as restriction of essential nutrients. Conversely, feeding a low-calorie food to a pet with high energy needs may require excessive food intake, resulting in loss of body weight or excessive stool volume.

Feeding management evaluation

Knowing what diets are fed does not indicate whether or not they are fed appropriately and eaten acceptably. Clients should be asked how much and how often each of the foods identified previously are fed. Other important questions include the following:

Do pets in a multiple-pet household share a food bowl, or are they fed individually?

Are pets fed measured amounts of food or free choice?

How well does the pet accept the food?

Have there been any changes in how the patient is fed or how it eats?

This information is not only important in determining the adequacy of the current dietary situation but in planning a dietary recommendation that achieves good client acceptance and compliance.

Common diet-sensitive conditions in geriatric animals

Few diseases in modern pets are "diet induced." One possible exception to this is obesity, which, although many interactive factors are involved, is ultimately caused by consuming more calories than needed by the dog or cat. Many other diseases are "diet-sensitive," however, meaning that diet can play a role in managing the condition. Examples of diet-sensitive conditions common in aging dogs and cats include chronic renal disease, diabetes mellitus (DM), arthritis, and many others. Information on the management of many of these diseases can be found in other articles in this issue. The remainder of this article focuses first on weight loss and then on the most common nutritional problem in older dogs and cats, which is obesity, and some obesity-related conditions.

Weight loss

Not all older patients are overweight. In fact, a greater proportion of dogs and cats older than 12 years of age are underweight than any other age

group [12]. Weight loss is not unusual in older patients and may be associated with increased or decreased intake (Fig. 4). The implications for dietary modifications vary, depending on the specific diagnosis.

If intake is normal or increased but the client has recently been feeding a commercially available senior diet or other diet with reduced calories to a highly active dog or geriatric cat, weight loss could be a normal response. The pet may have high energy needs because of individual metabolism or lifestyle. Alternatively, malabsorption of nutrients may be involved. Approximately one in three geriatric cats experiences fat malabsorption,

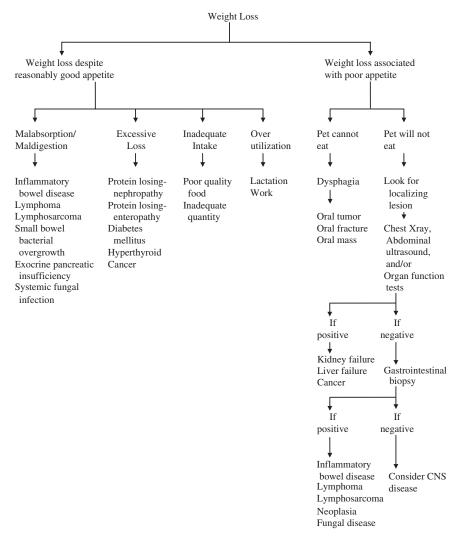


Fig. 4. Diagnostic algorithm for weight loss in geriatric patients. (Courtesy of Nestle Purina PetCare Company, St. Louis, MO.)

and one in five experiences protein malabsorption [11]. If other reasons for weight loss are excluded, the patient should be evaluated for diseases like intestinal or pancreatic diseases, renal disease, or cancer.

Body weight loss seems to be an early indicator of chronic disease in geriatric cats. When body weight data from 258 cats were evaluated retrospectively, a definite pattern emerged between body weight change and death secondary to various diseases [11]. Cats dying from cancer, renal failure, and thyroid disease began to lose weight 2.5 years before death. On average, more than 6% of the body weight was lost in the second year before death, and the average body weight loss in the last year of life was more than 10% for these cats. It is not known if tempering this disease-associated weight loss could delay mortality or reduce morbidity in aging cats, yet it seems logical to consider it.

If weight loss in dogs or cats is caused by pancreatic exocrine insufficiency, lymphangiectasia, or liver disease with fat malabsorption, a high-carbohydrate and low-fat diet may be useful. Bile acids from the liver and pancreatic lipase are important for the normal digestion and absorption of long-chain triglycerides (LCTs), the lipids found in most diets. Absorption of LCTs can drop by 50% to 70% of normal in the absence of bile acids and to near zero in the absence of pancreatic lipase. Even when steatorrhea is not apparent, fat digestion may be somewhat reduced. Hydroxylation of unabsorbed fatty acids by colonic microflora can contribute to secretory diarrhea in these patients.

In dogs that are suspected of fat malabsorption, restriction of LCTs is recommended, although adequate essential fatty acids must be provided (ie, dietary LCTs between 5% and 10% of diet dry matter). Inclusion of medium-chain triglycerides (MCTs) as part of the dietary fat in canine diets may be advantageous, because MCTs provide a concentrated source of energy and can be digested and absorbed fairly well despite a lack of pancreatic lipase or bile acids. They are mostly absorbed into the portal blood rather than lymphatic lacteals, so they are less likely than LCTs to contribute to lymphangiectasia. Because MCTs do not provide essential fatty acids, they should not constitute more than 50% of the dietary fat.

Decreased intake may occur for many reasons. In a multiple-dog household, pack relations can change with age and time. An evaluation of feeding management may indicate that an "alpha" dog has been displaced and is no longer receiving free access to a common food bowl. Poor dental health could prevent an otherwise healthy dog or cat from consuming adequate nutrition. Dry foods help to reduce the build up of plaque and tartar on teeth, but soft foods may be needed after extensive tooth loss. Systemic diseases, such as hepatic, renal, gastrointestinal, or adrenal dysfunction, or central nervous system disorders may affect appetite and should be considered if more obvious explanations are not apparent. If a specific diagnosis cannot be found, symptomatic treatment for weight loss should include consumption of a high-calorie and nutrient-dense food.

Dietary fat helps to make foods more palatable as well as providing needed calories.

If poor appetite is a problem, intake may be encouraged by selecting a palatable diet, moistening dry food with warm water, warming food to body temperature, offering fresh food frequently, and having clients pet and encourage the patient during feeding. Cats usually respond well to acidic diets with high moisture content; however, some prefer dry foods. Bowls used for feeding cats should be wide and shallow so that the sides do not touch the cat's whiskers. Minimize noise and stress during feeding periods. If a recent dietary change precipitated the anorexia, consider offering the previous diet. Nutrient modifications that are beneficial in disease management are less important than providing adequate nutrition. Ensure that the patient's nasal passages are clear, because dogs and cats rely on olfaction in selecting foods. Although uncommon, some geriatric cats do experience permanent hypogeusia.

Chemical appetite stimulants may be helpful for short-term use in overcoming anorexia. Benzodiazepine derivatives are commonly used and are effective in up to 50% of patients. Diazepam may be used in dogs or cats and is most effective when administered intravenously (0.2 mg/kg, with a maximum dose of 5 mg per patient) [42]. Fresh palatable food should be offered immediately, because feeding usually starts within 1 minute and may continue for up to 20 minutes. Oxazepam (2.5 mg per cat) results in eating within 20 minutes after oral dosing. Sedation and ataxia are common side effects to diazepam and oxazepam administration.

Recently, excellent results were reported when anorectic cats and dogs were treated with midazolam and propofol, respectively [43,44]. Anorectic cats began eating within 2 minutes after intravenous administration of midazolam (2–5 μ g/kg of body weight), with no apparent evidence of sedation or other side effects. Anorectic dogs given intravenous propofol (1–2 mg/kg of body weight) experienced a brief period of sedation, followed by a strong appetite response. No adverse effects were noted in either study. If adequate ongoing oral intake is not achieved, enteral or parenteral nutritional support should be considered.

Obesity

Approximately one of every four dogs and cats presented to veterinary practices in the United States is overweight or obese [12]. The prevalence peaks between 5 and 10 years of age, affecting nearly 50% of dogs and cats in this age group. Obesity can be defined as an excess of body fat sufficient to result in impairment of health or body function. In people, this is generally recognized as 20% to 25% greater than ideal body weight. This degree of excess body weight seems to be important in dogs as well. A lifelong study in dogs showed that even moderately overweight dogs were at greater risk for earlier morbidity and a shortened life span [45].

In that study, one group of Labrador Retrievers was fed 25% less food than their sibling-pairmates throughout life. The average adult BCSs for the lean-fed and control dogs were 4.6 ± 0.2 and 6.7 ± 0.2 , respectively, based on a nine-point BCS system [45]. Thus, the control dogs were moderately overweight and actually weighed approximately 26% more, on average, than the lean-fed group. The lean-fed dogs were well within the ideal body condition of 4 to 5 on this nine-point scale. The difference in body condition was sufficient to create significant differences between the groups in median life span, which was 13 years for the lean-fed dogs compared with only 11.2 years for the control group, a difference of approximately 15%. An impressive correlation between BCS at middle age and longevity in these dogs can be seen in Fig. 5. Dogs with a BCS of 5 or less at middle age were far more likely to live beyond 12 years of age compared with those with a higher BCS. In addition, control dogs required medication for chronic health problems or arthritis an average of 2.1 years or 3.0 years, respectively, sooner than their lean-fed siblings [45].

Obese cats also face increased health risks, including an increased risk of musculoskeletal problems (arthritis), DM, hepatic lipidosis, and early mortality [46].

Recent research has suggested a mechanism for the link between excess body weight and so many diseases. It seems that adipose tissue, once

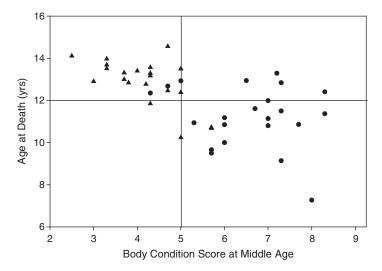


Fig. 5. Effect of body condition on longevity in dogs. The lean-fed dogs (\blacktriangle) received 25% less food than their control group littermates (•). Body condition score (BCS) was determined annually using a nine-point system. Data shown are the mean BCSs for ages 6 through 8 years for each dog as the independent variable for age of death. Dogs with a BCS of 5 or less at middle age were more likely to live beyond 12 years of age (P < 0.001) compared with dogs with a BCS higher than ideal. For additional details on the test design, see the article by Kealy et al [45]. (*Data from* Richard D. Kealy, PhD and Dennis F. Lawler, DVM, St. Louis, MO).

considered to be physiologically inert, is an active producer of various hormones, such as leptin, and cytokines. Of major concern is the production of inflammatory cytokines from adipose tissue, specifically tumor necrosis factor- α (TNF α), interleukin (IL)-1 β and IL-6, and C-reactive protein [47– 50]. The persistent low-grade inflammation secondary to obesity is thought to play a causal role in chronic diseases like osteoarthritis (OA), cardiovascular disease, and DM [49,51]. In addition, obesity is associated with increased oxidative stress, which also may contribute to obesity-related diseases [52,53].

There are many factors that play a role in creating obesity. Prevention of obesity relies on understanding contributing or associated risk factors and managing them appropriately. Important risk factors for obesity in pets include neutering and inactivity. Neutering can significantly reduce MERs as well as increase spontaneous food intake [54–56]. Controlling food intake can reduce the development of obesity in neutered pets [57].

Despite widespread concern about obesity among pet owners, most do not recognize their own overweight dog as being overweight. As noted previously, obesity is associated with significant health risks; thus, diagnosing and managing obesity is an important part of the nutritional management of aging dogs and cats.

The first step in an effective obesity management program is recognition of the problem. Perhaps the most practical methods for in-clinic assessment of obesity are a combination of body weight and BCS. There are several BCS systems. This author prefers using validated nine-point systems for dogs and cats [58–60]. With these systems, each unit increase in BCS is approximately equivalent to 10% to 15% greater than ideal body weight, so a dog or cat with a BCS of 7 weighs approximately 20% to 30% greater than the ideal weight. By recording body weight and BCS, ideal body weight can be more easily determined. Animals that are becoming obese can be recognized sooner and managed more easily. An illustrated BCS system can provide a useful tool for client education regarding obesity prevention and management.

Once the clinician and owner have recognized obesity in a pet, it is important to develop a management plan that fits the needs of the patient and owner. This must consider client ability and willingness to control calories and enhance exercise for the pet. Numerous options are available, so the keys to success are flexibility in design and regular follow-up with the client. Of utmost importance is the recognition that individual animals can differ greatly in their MERs. Thus, the degree of calorie restriction that induces significant weight loss in one dog or cat may cause weight gain in another. Adjustments in calorie allowance made on a regular basis (eg, every month) help to address these individual differences as well as the reductions in MERs that occur during weight loss.

Use of an appropriate diet for weight loss is important, and there are several criteria to consider. Although it is ultimately calorie restriction that induces weight loss, it is important to avoid excessive restriction of other essential nutrients. Therefore, a low-calorie product with an increased nutrient/calorie ratio should be considered. Further, an important goal for weight loss is to promote fat loss while minimizing loss of lean tissue, which may be influenced by dietary composition.

Fat restriction in weight-loss diets reduces calorie density, which helps to reduce calorie intake. Fat contains more than twice the calories per gram of protein or carbohydrate. In a study of obese human subjects, when carbohydrate replaced dietary fat in ad libitum–fed diets, weight loss was significantly enhanced [61]. In a canine study, dogs fed a low-fat and high-fiber diet lost more body fat compared with dogs fed a high-fat and low-fiber diet [62]. Conversely, several human studies have shown that extremely low-carbohydrate diets can facilitate increased weight loss [63–65]. In this author's experience, such diets alter the selection of foods consumed and greatly reduce intake of sugars and other highly refined carbohydrates, thus reducing calorie intake. Anecdotal reports suggest that this approach also works in overweight cats, but no data as yet support this premise. Conversely, numerous studies have shown that increasing dietary protein, often in exchange for carbohydrates, has beneficial effects for weight management [66–71].

Dietary protein is especially important in weight-loss diets. Providing low-calorie diets with an increased protein-to-calorie ratio significantly increases the percentage of fat lost and reduces the loss of LBM in dogs and cats undergoing weight loss [66,67]. Protein has a significant thermic effect, meaning that postprandial metabolic energy expenditure is increased more when protein is consumed, compared with carbohydrates or fats [72]. In addition to directly contributing to a negative energy balance in support of weight loss, the thermic effect of protein seems to contribute to a satiety effect provided by dietary proteins [73,74]. Finally, a higher protein diet helped to sustain weight maintenance after weight loss in human subjects [75]. This effect is likely to apply to dogs and cats as well.

Other nutraceuticals and herbal compounds continue to be evaluated for use in weight-loss diets. To date, published data on these have been conflicting. Carnitine seems to have received the most attention. Carnitine is produced endogenously from the amino acids lysine and methionine and facilitates β -oxidation of fatty acids. Supplementation with this compound is likely to be of greatest benefit when the intake of dietary protein or other key nutrients is insufficient to promote adequate endogenous production. In semistarved cats and rats undergoing extremely rapid weight loss, L-carnitine reduced hepatic fat accumulation in cats and enhanced lipid metabolism and reduced ketogenesis in rats [76,77]. In human subjects, severe calorie restriction resulted in reduced urinary and plasma carnitine, an effect that was attenuated by increased dietary protein during weight loss [78]. With a few exceptions, studies evaluating carnitine for weight management have shown little benefit [79–82]. In a canine study, dogs retained more LBM when fed a carnitine-supplemented diet but also lost less body weight [83]. Another study in dogs showed no significant difference in body composition changes with carnitine supplementation but implied a benefit for metabolic stimulation [84]. One study demonstrated a significant increase in the rate of weight loss in cats supplemented with carnitine compared with a control group (24% versus 20%, respectively, over an 18-week period) [85].

In addition to diet, feeding management and exercise are critically important to successful weight management. Most clients provide treats for their pets. Rather than requiring that they cease this pleasurable activity, create a "treat allowance" equal to 10% of the daily calories. Clients may be provided with a menu of low-calorie foods or commercial treats that would be appropriate.

Increasing exercise aids in weight management by expending calories. Interactive exercise provides an alternative activity for the pet and owner to enjoy together rather than food-related activities. Activity in cats may be enhanced by interactive play, such as with a toy on a string or a laser light.

Gradual weight loss in dogs, as in people, is more likely to allow longterm maintenance of the reduced body weight [86]. Weight rebound can be minimized by providing controlled food intake and adjusting the calories fed to just meet the needs of the pet for weight maintenance. Clients already accustomed to measuring food and monitoring their pet's weight should be encouraged to apply these behavior modifications to long-term weight management.

Diabetes mellitus

The most significant risk factors for feline DM are age and obesity as well as male gender [3,46,87,88]. Compared with cats less than 7 years of age, cats between 7 and 10 years of age are 8 times more likely to become diabetic and cats older than 14 years of age are 14 times more likely to become diabetic [87]. Obese cats are approximately 4 times as likely to become diabetic compared with cats with optimal body condition [46]. Obesity causes insulin resistance and impaired glucose tolerance in otherwise normal cats [89,90]. One proposed mechanism by which obesity may lead to insulin resistance is by compromising the functionality of the GLUT4 receptor [91,92]. Under normal circumstances, insulin triggers an intracellular cascade resulting in activation of the GLUT4 transport system, which then facilitates glucose entry into the cell. Obesity leads to increased triglycerides in skeletal muscle, which reduce GLUT4 expression and contribute to insulin resistance [91,92]. In addition, TNFa, which is increased in obesity, reduces the expression of GLUT4 [48]. Insulin resistance is a cardinal sign of type II diabetes, the most common form in cats [90,93].

Like cats, obese dogs develop changes in glucose tolerance and insulin resistance. Unlike the scenario in cats, however, this does not seem to progress and obesity does not seem to be a risk factor for development of diabetes in dogs [94,95]. Because obesity is a well-recognized risk factor for type II diabetes, this difference probably relates to the observation that dogs develop immune-mediated type I diabetes rather than type II diabetes. Conversely, obesity can increase the difficulty in regulating glucose in diabetic dogs; thus, obesity remains a concern for these patients.

The primary goal of therapy for DM is to maintain blood glucose concentrations as close to normal as possible through the use of exogenous insulin, diet, and other therapies, along with control or avoidance of concurrent illnesses [96]. Nutritional management is an important factor in the treatment of all diabetic patients. Type I diabetics are insulin dependent. Management is targeted at maintaining a stable and moderate blood glucose concentration through alterations in insulin. Diet serves an adjunct role because it can influence the amount of insulin required and can moderate the postprandial glycemic load [96]. Type II diabetics continue to have some capacity for insulin production. The role of dietary management in type II diabetes is to decrease the need for exogenous insulin while maintaining glycemic control. The dietary considerations for canine and feline diabetics include appropriate calorie intake to reach and maintain ideal body condition, complete and balanced nutrition to provide all essential nutrients, and nutritional modifications to address metabolic disturbances induced by DM. The specific modifications for canine and feline diabetics may differ because of differences in the underlying pathology findings of disease and species differences in normal metabolism.

Rapidly digestible carbohydrates (RDCs) provide abundant starch and sugars and promote an increase in postprandial blood glucose concentrations. In insulin-dependent diabetics, excess RDCs require more exogenous insulin to maintain glycemic control. Therefore, diets that produce less severe increases in postprandial blood glucose are preferred. Nutrient modifications that can help in this regard include an avoidance of RDCs, use of complex carbohydrates and dietary fiber, and use of protein instead of carbohydrates.

Complex carbohydrates include fiber or bran along with the starch, such as might be provided by whole grains. These are digested more slowly than the RDCs from sugars, flours, or polished grains, resulting in a delayed release of glucose into the bloodstream. Alternatively, fibrous ingredients or purified fiber sources can be incorporated into complete pet foods to provide a similar effect—a reduction in postprandial glucose [97–99]. The net effect of providing fiber in the diet is a slowing of carbohydrate absorption from the intestinal tract, a dampening of the postprandial glycemic effects of a meal, and beneficial modifications in blood lipids [100].

Several studies have evaluated fiber-enriched diets in dogs with diabetes. When dogs with well-regulated naturally occurring DM were fed canned diets supplemented with 20 g of wheat bran (a predominantly insoluble fiber source) or 20 g of guar gum (a purified soluble fiber source), reductions in postprandial hyperglycemia were noted [97]. The effect was most pronounced with the guar gum. In a similar study using dogs with alloxaninduced DM, significant reductions in the mean 24-hour blood glucose concentration and 24-hour urine glucose excretion were observed when dogs were fed diets supplemented with 15% (dry matter basis) cellulose (purified insoluble fiber) or pectin (purified soluble fiber) [98]. The cellulosesupplemented diet also caused a reduction in glycosylated hemoglobin. Total dietary fiber (TDF) in these diets was approximately 5.5 to 7.0 g per 420 kJ [101]. Kimmel et al [102] reported better results in insulin-dependent canine diabetics with an insoluble fiber diet, using TDF levels of 7.3 g and 5.6 g of TDF per 420 kJ for the insoluble and soluble fiber diets, respectively. A further study in dogs with naturally occurring DM demonstrated enhanced glycemic control when fiber from pea fiber and guar gum was included at 5.3% of a wet diet, or approximately 5.65 g of TDF per 420 kJ diet [99].

That dietary fiber can be beneficial in managing glycemic control seems to be well documented. What remains controversial is the appropriate amount of fiber needed for this purpose. Problems associated with excess fiber can include increased stool volume and undesirable calorie dilution. Little work has been done to compare a wide range of fiber levels in dogs. No differences were observed between diets when well-controlled diabetic dogs were fed 6.0 or 9.0 g of TDF per 420 kJ (J.W. Bartges, DVM, PhD, unpublished data) An evaluation of several moderate-fiber commercial diets (3.5-5.0 g of TDF per 420 kJ) showed no difference among these diets with regard to insulin or glucose measures [103], but no comparison was made with higher fiber levels. Lower levels of fiber, such as those evaluated in human diabetics, have not been investigated for use in canine diabetics. Based on the data available, diets that provide between 6 and 9 g of TDF per 420 kJ may be appropriate for overweight diabetic dogs, whereas 3 to 6 g of TDF per 420 kJ may be appropriate for diabetic dogs in ideal or thin body condition.

Fewer studies have evaluated fiber-supplemented diets for diabetic cats. Nelson et al [104] reported a significant improvement in serum glucose concentrations in insulin-treated diabetic cats fed a diet containing 12% cellulose (dry matter basis) compared with a low-fiber diet. Another study reported a decrease in insulin requirements in cats switched to a commercial high-fiber diet [105]. Conversely, cats improved significantly more when switched from a high-fiber diet to a high-protein and low-carbohydrate diet [105,106].

Increased dietary protein seems to be beneficial in patients with type II diabetes. In human beings with well-controlled type II diabetes, ingestion of higher protein and lower carbohydrate diets resulted in improved glycemic control as measured by reduced glycosylated hemoglobin or decreased

blood glucose on the higher protein diets [107–109]. Similar benefits have been observed in feline diabetics, with decreased insulin requirements or enhanced glycemic control when cats were fed a high-protein diet [105,106,110]. Further, consumption of a high-protein and low-carbohydrate diet resulted in increased insulin sensitivity in diabetic cats [105].

Most studies evaluating higher protein diets have balanced the dietary change on carbohydrates, resulting in high-protein and low-carbohydrate diets. One study maintained a constant protein level (as percentage of energy), however, and varied carbohydrates with fat [111]. Measures of glycemic control and insulin dose were unchanged with the low-carbohydrate diet, indicating that protein is the responsible beneficial nutrient in type II diabetics. This hypothesis is further supported by evidence that inadequate dietary protein has a negative impact on insulin secretion and insulin activity [112].

These findings suggest that diets high in protein may be beneficial in patients with disturbed glucose metabolism. In human patients with insulindependent type I diabetes, however, increased protein intake was associated with an increased glucose response and increased exogenous insulin requirement [113,114]. This may reflect a difference between type I and type II diabetes. Conversely, protein requirements seem to be increased in type I diabetes due to increased protein catabolism.

Not only is insulin necessary for the efficient cellular uptake of glucose, but it is required for fat and protein metabolism as well. Insulin inhibits catabolism of proteins and gluconeogenesis from amino acids and promotes the uptake of amino acids and synthesis of new proteins. Abnormalities in serum insulin concentrations result in disruptions in protein metabolism. Glucagon, which is increased when insulin levels fall, decreases cellular protein synthesis and increases protein catabolism and gluconeogenesis. Even in well-controlled diabetics, protein catabolism seems to occur at significantly higher rates, leading to protein loss [115]. To avoid depletion of LBM and protein reserves from increased skeletal muscle catabolism, it is important to ensure adequate protein intake in diabetic patients.

Osteoarthritis

OA, also called degenerative joint disease, is the most prevalent joint disorder in dogs, affecting as many as 20% of adult dogs [116]. OA is associated with inflammation and increased degradation or loss of proteoglycans from the extracellular matrix, resulting in a morphologic breakdown in articular cartilage [117]. Obesity is recognized as a risk factor for OA, and preventing obesity can help to reduce the incidence and severity of OA [45,118,119]. In a recently completed 14-year study on food restriction in dogs, those dogs fed to maintain a lean body condition throughout their lifetime exhibited a delayed need for treatment and reduced severity of OA in the hips and other joints compared with their

heavier siblings [45]. One of the most compelling findings from this study was the observation that even a mild degree of excess body weight can adversely affect joint health. This is important, because more than 25% of dogs seen by veterinarians are overweight or obese [1].

The effect of obesity on OA may be more than just physical strain caused by weight bearing. Obesity is now recognized as an inflammatory condition; adipose tissue or associated macrophages produce inflammatory cytokines [47–49]. C-reactive protein, $TNF\alpha$, IL-6, and other inflammatory mediators are elevated in the blood and adipose tissue of obese subjects and are thought to contribute to many complications associated with obesity, such as OA [47–50,120]. Obesity is also associated with an increase in oxidative stress, [52,53] another feature of OA.

Multiple studies have shown that weight loss helps to decrease lameness and pain and to increase joint mobility in patients with OA [118,121,122]. Overweight dogs with coxofemoral joint OA demonstrated decreased lameness and increased activity after weight reduction to ideal body condition.

A primary target of OA treatment is the inhibition of cyclooxygenase (COX) enzymes—especially the COX-2 enzyme—through the use of nonsteroidal anti-inflammatory drugs (NSAIDs) [123,124]. COX-2–selective inhibitors can decrease prostaglandin E_2 (PGE₂) concentrations and block inflammatory pathways involved in OA as well as reduce pain and lameness [123–126]. Blocking the COX and lipo-oxygenase (LOX) enzymes at the active sites of 5-LOX, COX-1, and COX-2 significantly reduces matrix metalloproteinases (MMPs), IL-1 β , leukotriene (LT) B₄, and PGE₂, resulting in decreased tissue damage in arthritic joints [127].

Another means of reducing PGE₂ and other inflammatory eicosanoids is through the use of dietary long-chain omega-3 (n-3) polyunsaturated fatty acids, especially eicosapentaenoic acid. The primary omega-6 (n-6) fatty acid in cell membranes is arachidonic acid, which serves as the precursor for the production of the potent inflammatory eicosanoids in OA: PGE₂, thromboxane (TX) A₂, and LTB₄. If the diet is enriched with long-chain n-3 polyunsaturated fatty acids—specifically eicosapentaenoic acid and docosahexaenoic acid-part of the arachidonic acid in cell membranes is replaced by these n-3 fatty acids [128–130]. Eicosapentaenoic acid may then be used instead of arachidonic acid for the production of eicosanoids, resulting in a different and less inflammatory set of compounds (eg, PGE₃, TXA₃, and LTB₅ instead of PGE₂, TXA₂, and LTB₄) [128,129]. Dietary n-3 polyunsaturated fatty acids also suppress the proinflammatory mediators IL-1, IL-2, and TNF in cartilage tissue [131,132]. Thus, substituting n-3 for part of the n-6 fatty acids should reduce inflammation and benefit inflammatory conditions, including OA.

A review of studies in arthritic people indicated that most showed positive results from long-chain n-3 polyunsaturated fatty acid supplementation [133]. Recent research in dogs supports many of these earlier findings

confirming the clinical benefits of dietary n-3 fatty acids in OA. Twenty-two dogs with OA of the hip were given a fatty acid supplement marketed for dogs with inflammatory skin conditions [134]. Thirteen of these dogs had noticeable improvement in their arthritic signs within 2 weeks. Another uncontrolled study evaluated dogs with naturally occurring OA of the elbow and used force-plate analysis before and after dogs were fed a diet enriched with n-3 polyunsaturated fatty acids. Improvements in vertical peak force were observed within 7 to 10 days on the diet (S.C. Budsberg, DVM, unpublished data, 2004). In yet another study, dogs fed a diet enriched with n-3 polyunsaturated fatty acids after corrective surgery for ruptured cruciate ligaments showed a significant decrease in synovial fluid PGE₂ [135]. Synovial fluid MMP-2 and MMP-9, enzymes that degrade structural proteins in cartilage, were also decreased in these dogs compared with dogs fed the control diet.

Glucosamine, an endogenously produced aminosugar, is another compound that may be beneficial in dogs with OA. A decrease in glucosamine synthesis by chondrocytes has been implicated in OA, whereas supplemental glucosamine has a stimulatory effect on chondrocytes [136]. Glucosamine is considered a chondroprotective agent and may minimize the progression of OA [136,137].

Several short- and long-term, double-blind, randomized trials evaluating glucosamine supplementation in people with OA of the knee were recently reviewed by meta-analysis [137]. These studies documented significant improvement in clinical signs of OA in patients consuming glucosamine at a dose of 1500 mg/d (approximately 21 mg/kg of ideal body weight). Two of these studies followed patients for 3 years and demonstrated that oral glucosamine inhibited the long-term progression of OA [137]. Clinical studies in dogs involving glucosamine alone are lacking.

Chondroitin sulfate, an endogenously produced polysaccharide found in the joint cartilage matrix, also has been shown to be beneficial in osteoarthritis. A number of placebo-controlled clinical trials in humans have shown a protective action of chondroitin sulfate against cartilage deterioration or a decrease in pain with supplementation [138–142].

Combinations of chondroitin sulfate and glucosamine also have been evaluated. In vitro research using bovine cartilage demonstrated a synergistic effect on glycosaminoglycan synthesis from a combination of glucosamine hydrochloride, manganese ascorbate, and chondroitin sulfate [143]. Synergistic effects also were noted for this combination in an in vivo rabbit model of arthritis [143]. Canine studies using a combination of glucosamine and chondroitin sulfate reported a benefit similar to that seen in other species [136,144].

OA is associated with an increase in oxidative stress and chondrocyteproduced reactive oxygen species and a reduction in antioxidant capacity [145–151]. The severity of arthritic lesions is increased in the face of decreased antioxidant capacity [148]. In vitro studies have shown that exposure of chondrocytes to reactive oxygen species inhibits proteoglycan and DNA synthesis and depletes intracellular ATP [149,150]. Reactive oxygen species contribute to cartilage degradation directly as well as by upregulating the genetic expression of MMPs and decreasing the production (or activity) of tissue inhibitors of MMPs [149,151]. In addition, oxidative stress induced chondrocyte senescence in vitro, with reduced glycosaminoglycan production and replicative lifespan—an effect that was reversible with antioxidant supplementation [148,152]. Physiologic concentrations of vitamin E inhibited lipid peroxidation in chondrocytes and minimized oxidation-induced cartilage degradation in vitro [151]. In a different model, vitamin C was effective at reducing premature chondrocyte senescence induced by reactive oxygen species [151].

Although limited in number, the published studies assessing in vivo benefits of antioxidants in OA support the in vitro findings. A 10-year prospective cohort study showed that intake of supplemental vitamin E (P = 0.06), vitamin C (P = 0.08), and zinc (P = 0.03) independently reduced the risk for developing rheumatoid arthritis in elderly women [153]. A 2-year clinical trial in people with existing knee OA evaluated the benefit of vitamin E supplementation on cartilage degradation [154]. No statistically significant differences were observed in cartilage loss, most likely because of the small sample size. Researchers detected directional differences, with cartilage loss reduced in the vitamin E group compared with the placebo group, however. A 1-year study in mice genetically predisposed to developing OA also showed a benefit from dietary antioxidants [155]. Glutathione peroxidase activity was significantly increased in the serum and synovium of mice fed a complete diet supplemented with pyridoxine; riboflavin; selenium; and vitamins E, C, and A, confirming an antioxidant effect. The incidence of OA in the antioxidant-supplemented mice was decreased by one third to one half [155]. Together, these various studies strongly suggest a benefit of dietary antioxidants for patients with OA.

In addition to nutrient modifications that may help in the dietary management of dogs with OA directly, dogs need appropriately balanced nutrition to support normal maintenance of joints and other tissues. Many people with OA seem to consume nutritionally imbalanced diets. Deficiencies in antioxidant nutrients, B vitamins, zinc, calcium, magnesium, and selenium are frequently reported [156,157]. Although it is not known how many of these deficiencies contribute to OA, these nutrients play a role in the normal maintenance of cartilage and other tissues. Therefore, it is important that dogs with OA receive diets that provide complete and balanced nutrition.

Summary

Before recommending a diet for a senior pet, a thorough nutritional evaluation should be completed. Although many middle-aged and older pets

are overweight, a large percentage of geriatric cats and dogs have a low BCS. Approximately one third of cats older than 12 years of age may have a decreased ability to digest fat, whereas one in five may have a compromised ability to digest protein. Thus, appropriate diets for these two age groups may differ considerably. Mature (middle-aged) cats would likely benefit from a lower calorie food, whereas geriatric cats (\geq 12 years of age) may need a highly digestible nutrient-dense diet.

More than 40% of dogs between the ages of 5 and 10 years are overweight or obese. Such dogs may benefit from diets with lower fat and calories. Senior dogs also have an increased need for dietary protein, however. Therefore, healthy older dogs may benefit from diets with an increased protein-to-calorie ratio, providing a minimum of 25% of calories from protein.

Common obesity-related conditions in dogs or cats include DM and OA. Diabetes differs between dogs and cats. Type I diabetes, common in dogs, seems to respond to fiber-enriched diets, whereas type II diabetes, common in cats, seems to benefit from high-protein and low-carbohydrate diets. OA, an inflammatory condition that occurs in approximately 20% of dogs, may benefit from weight management and nutrients that reduce the inflammatory responses, such as long-chain n-3 fatty acids.

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