INTRODUCTION
Cats were probably domesticated between 1600 and 1500 BC. Early Egyptians considered cats sacred and valued them for their natural hunting and predatory behavior, which helped control rodent populations. Little consideration for the nutritional needs of cats was required during the early days of domestication. As domestic cats evolved from mouse catcher to household companion, the need to understand their unique nutritional requirements also increased. Today, it is well accepted that proper nutrition and care throughout life maximizes health, longevity and quality of life. Providing proper guidance about the nutritional management of cats requires an understanding of: 1) the basic principles of nutrition (Chapters 5 and 6), 2) the foods and nutrients commonly fed to cats, 3) how to assess nutrient availability and quality of various foodstuffs and foods, 4) foods and feeding practices that may positively or negatively affect health and 5) the unique nutritional needs of cats throughout the lifecycle.

Demographics
Cats are the most popular pets in the United States, totaling approximately 77 million (APPMA, 2003). More than one-third of the households in the U.S. own cats with an average of 2.1 cats per cat-owning household. In 1996, the ratio of male to female cats was roughly equal and nearly 80% of pet cats in the U.S. were neutered (Table 19-1) (Lund et al, 1999). Table 19-2 lists the 10 countries with the largest pet cat populations. Mixed-breed cats, domestic shorthairs and longhairs, make up an estimated 95% of the world’s domestic cat population. They result from random rather than selective breeding. Domestic shorthair and longhair cats display a wide variety of sizes, and coat colors, patterns and lengths. Although most cats in the U.S. are non-pedigreed, the Cat Fanciers Association registered 41 different breeds in 2005. The four most common breeds were Persians, Maine coons, Siamese and Abyssinians (CFA, 2005).

Compared with dogs, cats make up a smaller proportion of the pets seen by veterinarians, but that proportion is increasing. Now, nearly 68% of cat owners in the U.S. regularly use veterinary services. In 2001, cats visited veterinary clinics once per year compared with 0.79 visits per year in 1987 (Center for Information Management, 2002). In 2001, cat owners spent $6.3 billion toward the health and well being of their cats. Cat food sales followed the upward trend in cat ownership and health care with almost $4.3 billion of sales in the U.S. in 1997.
The greatest growth occurred in dry cat foods and treats, whereas sales of moist foods were static, and sales of semi-moist products decreased. Pet owners are very interested in nutritional information. In one pet owner survey, interest in obtaining nutritional information equaled obtaining information about diseases. Furthermore, the pet owners' top two preferred sources of pet health information were the Internet and veterinarians (MAF, 2005).

**CATS AS CARNIVORES**

Taxonomically, cats and dogs are members of the order Carnivora and are therefore classified as carnivores (Table 12-4). From a dietary perspective, however, dogs are omnivores (Chapter 12) and domestic cats and other members of the superfamily Felidea are strict or true carnivores, along with raptors, mosquitoes and some coldwater fish. This basic difference is supported by specific behavioral, anatomic, physiologic and metabolic adaptations of cats to a strictly carnivorous diet.

**Adaptations in Feline Feeding Behavior**

Domestic cats share several feeding behaviors with their wild counterparts. Unlike most mammals, cats do not display a regular daily rhythmicity in sleep-wake cycles, activity, feeding and drinking. Cats typically eat 10 to 20 small meals throughout the day and night (Kane et al, 1981; MacDonald et al, 1984). This eating pattern probably reflects the evolutionary relationship of cats and their prey. With the exception of African lions, cats are solitary hunters. Small rodents (e.g., voles and mice) make up 40% or more of feral domestic-type cats' food source; however, young rabbits and hares may compose a large portion of their natural diet (Fitzgerald, 1988). A variety of other prey (e.g., birds, reptiles, frogs and insects) is also eaten, but in smaller amounts. The average mouse provides approximately 30 kcal (125 kJ) of metabolizable energy (ME) (Mugford, 1977). This amount is about 12 to 13% of a feral cat's daily energy requirement. Repeated cycles of hunting throughout the day and night are required to provide sufficient food for an average cat. Thus, meal feeding cats once per day is in conflict with their natural feeding behavior.

The predatory drive is so strong in cats that they will stop eating to make a kill (Adamec, 1976). This behavior may frustrate owners who confuse predatory behavior with hunger (Box 19-1). Many owners reason that a fed cat will not hunt and are disappointed when their housecat kills songbirds. Supplemental feeding may reduce hunting time, but otherwise does not alter hunting behavior (Turner and Meister, 1988).

Cats are very sensitive to the physical form, odor and taste of foods. Oral tactile sensation is important to normal feeding behavior and should be considered when feeding novel foods. Unless accustomed to foods with different textures such as dry foods, cats generally prefer solid, moist foods and reluctantly accept food with powdery, sticky and very greasy textures (NRC, 2006, 1986; Kane et al, 1981a). The flavor and texture preferences of individual cats are often influenced by early experience (Maxwell, 1998).
that can affect preferences throughout life. Cats accustomed to a specific texture or type of food (i.e., moist, dry, semi-moist) may refuse foods with different textures. This becomes an important consideration when feeding cats novel foods.

Cats find certain flavors very attractive, which seems to reflect the nutritional characteristics of their natural foods. Cats prefer the tastes of animal fat, protein hydrolysates (digests), meat extracts and certain free amino acids abundant in muscle tissue (i.e., alanine, proline, lysine, histidine and leucine). Cats search out wild prey more often when meat is not in their diet (Robertson, 1998). Even the feeding behaviors of cats in the wild reflect their preference for animal tissues. When ingesting prey, wild cats avoid consuming plant materials contained in the entrails. African lions have been observed to first empty the prey, wild cats avoid consuming plant materials contained in the entrails of prey (Mech, 1970).

Cats also respond to high-pitched sounds, which represent the range of sound frequencies emitted by typical prey (Tabor, 1983). Finally, the highly sensitive facial whiskers and tactile hairs are thought to help cats hunt in dim light and to protect their eyes.

**Limb**

The retractable claws of cats are a unique adaptation to hunting. The sharp tips of the claws with hook-like curves and needle features are ideal for capturing and securing prey, yet they are easily retracted so they do not make noise when cats stalk prey. In contrast, the claws of dogs play only a secondary role in capturing prey.

**Oral Cavity**

Cats and dogs have the same number of incisor, canine and carnassial teeth (i.e., the enlarged upper premolar and lower molar teeth specialized for shearing flesh); however, cats have fewer premolar and molar teeth, and they do not possess fissured crowns, which are a hallmark of omnivorous animals (Figure 19-2). The jaws of cats have limited lateromedial and crano-caudal movement, thereby limiting grinding ability. The scissors-like action of the carnassial teeth is ideal for delivering the cervical bite used to transect the spinal cord and immobilize or kill prey. Cats lack salivary amylase used to initiate digestion of dietary starches. This adaptation reflects the nutritional composition of the typical prey (i.e., low starch content).

**Stomach**

Because cats evolved to eat small frequent meals, the stomach is less important as a storage reservoir compared with the stomach of dogs. Thus, the stomach of domestic cats is simpler than that of dogs (i.e., relatively smaller with a smaller glandular fundus).

**Small and Large Intestine**

Intestinal length, as determined by the ratio of intestine to body length, is markedly shorter in cats than in omnivores and herbivores (Table 19-3). A greater villus height in cats increases the absorptive surface area. Overall, however, the absorptive capacity is estimated to be 10% less than that of dogs (Kendall et al,
strict carnivores, such as raptors and coldwater fish. Also, cats (drates normally make up a negligible percentage of their food systems of little value because free sugars and complex carbohy-
waste energy or protein by turning over carriers or enzyme sys-
hydrate (Buddington and Diamond, 1992). Cats do not
digestion than animal tissues.

1982; Morris and Rogers, 1989). Therefore, dogs can more effi-
ciently use a variety of foods, some of which may require more
digestion than animal tissues.

Unlike in omnivores, the sugar transport systems of the small intestine of cats are not adaptive to varying levels of dietary car-
bohydrate (Buddington and Diamond, 1992). Cats do not
waste energy or protein by turning over carriers or enzyme sys-
tems of little value because free sugars and complex carbohy-
drates normally make up a negligible percentage of their food (Table 19-4). This lack of adaptability has been noted in other strict carnivores, such as raptors and coldwater fish. Also, cats

have low activities of intestinal disaccharidases (i.e., sucrase, maltase and isomaltase) (Kienzle, 1993). This reflects adapta-
tion to foods limited in simple sugars and other carbohydrates.

In cats, pancreatic amylase production is about 5% of that in dogs (Kienzle, 1987, 1993a). Pancreatic amylase production is relatively nonadaptive in cats, as would be expected in a species unaccustomed to significant changes in dietary carbohydrate levels. Cats have higher concentrations of bacteria in their small intestine than dogs and other omnivores studied (Johnston et al, 1993, 2001; Gruffydd-Jones et al, 1998). Interestingly, the numbers typical for cats would be diagnostic for small intestin-
ANOREXIA

Although a few days of inappetence is not particularly detrimental to an otherwise healthy cat, prolonged inadequate calorie intake results in malnutrition, reduced immune function and increased risk for hepatic lipidosis. Anorexia may be caused by stress, unacceptable foods or concurrent disease. Most commonly, cats presented to veterinarians for anorexia have a concurrent disease. Cats may endure prolonged starvation rather than eat an unpalatable food. Therefore, advising owners that a cat will “eat when it gets hungry enough” can have deadly results. Anorexia of more than three days duration, even in an otherwise healthy-appearing cat, warrants investigation by a veterinarian.

A thorough history is useful for differentiating potential causes of anorexia. To determine if inadequate food acceptance is the cause, offer a small selection of highly palatable foods along with the typical food. Because improperly stored foods may develop off flavors, bacterial contamination or fungal growth, confirm that the product is fresh and wholesome. Environmental or emotional factors reported to result in stress-mediated anorexia include hospitalization, boarding, travel, introduction of new people or pets to the household, loss of a companion, overcrowding, high temperatures and excessive handling. Stress-mediated anorexia is usually diagnosed from the history and by ruling out other diseases. Providing a quiet secluded area will often allow a cat to relax sufficiently enough to begin eating. Often, increasing the food’s palatability will improve food intake. Warming food, changing the food form, adding water or choosing foods high in animal protein and fat can enhance food palatability. If cats are highly stressed or appropriate feeding sites are unavailable, mild tranquilizers or appetite stimulants (e.g., mirtazapine, oxazepam or cyproheptadine) may be beneficial (Chapter 25). Force feeding may be accepted by some cats but others find the process so stressful that any benefit is far outweighed by the additional stress.

FIXED-FOOD PREFERENCES

The food type fed by the owner during a kitten’s first six months influences the pattern of food preferences throughout life. Although uncommon, kittens exposed to a very limited number of foods may develop a food fixation, refusing to eat anything but a single food. Adult cats fed highly palatable, single-item foods have been reported to develop fixed-food preferences as well.

Cats with food fixations can be particularly troublesome if dietary modifications are necessary. Cats with strong food preferences should be transitioned to the new food over a prolonged period. Convert to the desired food by replacing 10 to 20% of the old food with an equal amount of the new food on Day 1, then gradually increase the ratio of new to old over the next 14 days. A more gradual transition may be required if food intake drops below 70% of maintenance levels. Cats should be monitored to ensure they are not selecting the preferred food from the food dish and that food intake remains adequate. Feeding kittens and cats a variety of foods (both different forms of food and different brands) and not feeding single-item foods can avoid food fixations. This approach is strongly recommended as disease management later in life often requires a dietary change.

LEARNED TASTE AVersions

Cats may develop learned aversions to certain foods when feeding is paired with a negative GI experience. The negative experience can be physical, emotional or physiologic. Typically, aversions occur when cats consume a food immediately before an episode of nausea or vomiting. Foods that were readily consumed before the negative incident will be avoided subsequently. Clinically, aversions may develop when GI upset is induced by various diseases, drugs or treatment protocols. Foods with high salience (i.e., strong odors or high protein levels) are more likely to become aversive and should not be fed within 24 hours of anticipated GI upsets. Aversions have been documented to last up to 40 days in cats. Learned aversions are considered an adaptive response. By avoiding foods that previously caused gastric distress, cats will avoid eating foods likely to be spoiled or tainted. From a clinical perspective, consideration of food aversions often equates to delaying introduction of a therapeutic food, such as a diet for chronic kidney disease, until the cat’s GI signs have been controlled with other medical management.

POLYPHAGIA

Various diseases, drugs and psychological stresses can mediate excessive food consumption. Rarely, polyphagia (hyperphagia) may occur with diseases involving the central nervous system, particularly with lesions of the ventromedial hypothalamus. Presence of weight loss or gain is of key diagnostic importance. Polyphagia with weight loss is almost always associated with an underlying disease process or simple underfeeding. Caloric intake should always be calculated because underfeeding can result in a ravenous appetite that may be misinterpreted as abnormal. Nutritional management of polyphagia requires an accurate diagnosis because treatment is aimed at the primary disease.

The Bibliography for Box 19-1 can be found at www.markmorris.org.
More important to cats than previously thought.

**Feline Nutrient Requirements and Metabolic Adaptations**

### Energy Metabolism

The liver of most animals has two enzyme systems for converting glucose to glucose-6-phosphate: hexokinase and glucokinase. This conversion is necessary before the liver can use glucose. The glucokinase system operates only when the liver receives a large amount of glucose from the portal vein. Because the typical food source of wild cats is primarily animal not plant tissue, it contains only small amounts of digestible (soluble) carbohydrate and the portal system delivers very little absorbed glucose to the liver. Thus, adult cats have very low hepatic glucokinase activity and a limited ability to metabolize large amounts of simple carbohydrates. Omnivores (e.g., people, dogs and rats) have higher hepatic glucokinase activity

**Protein Metabolism**

Protein metabolism is unique in cats and is manifested by an unusually high maintenance requirement for protein as compared with canine requirements (Table 19-5) and a special need for four amino acids: arginine, taurine, methionine and cystine. The protein requirement for growth in kittens is only 50% higher than that of puppies, whereas the protein requirement for feline maintenance is twice that of adult dogs. The higher protein requirement of cats is not due to an exceptionally high maintenance requirement for any specific amino acid (Table 19-6); instead, it is caused by a high activity of hepatic enzymes (i.e., transaminases and deaminases) that remove amino groups from amino acids so the resulting ketoacids can be used for energy or gluconeogenesis. Unlike omnivores and herbivores, cats have a limited ability to decrease the activity of these enzymes when fed low-protein foods. The cat’s strict adherence to a diet of animal tissue likely resulted in a lack of evolutionary pressure to accommodate lower protein food sources. Hepatic enzyme systems are constantly active; therefore, a fixed amount of dietary protein is always catabolized for energy (MacDonald et al, 1984). The gluconeogenic enzymes in feline liver appear to be continuously active, unlike the situation in most other species, including dogs (MacDonald et al, 1984). In addition, an alternate hepatic gluconeogenic pathway common in flesh-eating animals is active in cats (Beliveau and Freedland, 1982). This pathway uses serine as a glucose precursor. Serine is a nonessential amino acid found in large amounts in muscle, milk and egg.

ARGinine

Arginine deficiency in cats causes one of the most dramatic responses of any nutrient deficiency. Cats cannot synthesize sufficient ornithine or citrulline for conversion to arginine, which is needed for the urea cycle. After a cat eats a meal, the highly active protein catabolic enzymes in its liver produce ammonia, which is absorbed from the colon.

Without arginine, the urea cycle cannot convert ammonia to urea and ammonia toxicity occurs (MacDonald et al, 1984). Eating a single meal devoid of arginine may result in hyperammonemia in less than one hour. Affected cats exhibit severe signs of ammonia toxicity (i.e., vocalization, emesis, ptyalism, hyper-
activity, hyperesthesia, ataxia, tetanic spasms, extended limbs with exposed claws, apnea and cyanosis) and may die within two to five hours (MacDonald et al, 1984). Because the diet of wild cats is high in animal protein (that contains arginine), cats have apparently lost the flexibility in protein metabolism seen in other animal species that eat foods with more limited amino acid composition. Arginine deficiency, however, has only been reported to occur in cats fed experimental foods specifically formulated to be arginine deficient or in cats fed certain casein-based human enteral products (Diehl and Wheeler, 1992). Although not necessarily supporting the argument that cats are carnivores, excess dietary lysine does not cause arginine antagonism in cats, as it can in dogs (Fascetti et al, 2004).

TAURINE
Taurine is a β-amino sulfonic acid, abundant as a free amino acid in the natural food of cats, such as small rodents, birds and fish. Taurine is found at lower concentrations in large animal species such as cattle. In cats, dietary taurine is essential and clinical disease results if insufficient amounts are present. Many species can use either glycine or taurine to conjugate bile acids into bile salts before they are secreted into bile. Cats can only conjugate bile acids with taurine. The loss of taurine in bile coupled with a low rate of taurine synthesis contributes to the obligatory taurine requirement of cats (Box 19-2).

METHIONINE AND CYSTINE
The sulfur-containing amino acids methionine and cystine are required in higher amounts by cats than by most other species, especially during growth. Cystine is the amino acid formed when a pair of cysteine molecules are joined by a disulfide bond. Methionine and cystine are considered together because cystine can replace up to half of the methionine requirement of cats (NRC, 2006). Methionine serves as a precursor to cysteine; therefore, cysteine is not an essential amino acid. Cysteine cannot be converted to methionine; however, a minimal requirement for methionine must be met with methionine. Although these amino acids are present in high amounts in animal flesh, methionine tends to be the first limiting amino acid in many food ingredients. Nutritional deficiencies are possible, especially in cats fed home-prepared or vegetable-based foods. Clinical signs of methionine deficiency include poor growth and a crusty dermatitis at the mucocutaneous junctions of the mouth and nose. Approximately 19% of a food must be composed of animal protein to meet the methionine requirement of kittens (MacDonald et al, 1984). Foods high in plant proteins require additional methionine, which can be supplied as DL-methionine, a crystalline form of the amino acid. Cats appear to prefer foods with added methionine compared to foods deficient in methionine (Rogers et al, 2004).

Numerous theories have been advanced to explain the high methionine and cystine requirement of cats. Methionine needs may be increased because of an increased S-adenosyl methionine requirement, cysteine synthesis, taurine synthesis or because of a high rate of methionine catabolism. Additional cystine may be required for the synthesis of the antioxidant glutathione and the amino acid felinine. Felinine is a branched-chain, sulfur-containing α-amino acid found in the urine of domestic cats. Its biologic function has not been fully elucidated. The most widely accepted possible role for felinine, or its breakdown product in urine, is as a pheromone, which is of importance in territorial marking. Sexually immature kittens have been reported not to excrete felinine and adult males excrete more

<table>
<thead>
<tr>
<th>Classification</th>
<th>Species</th>
<th>Growth (%)</th>
<th>Maintenance (%)</th>
<th>G:M ratio**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Omnivore**</td>
<td>Dog</td>
<td>12</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Omnivore†</td>
<td>Dog</td>
<td>18</td>
<td>8</td>
<td>2.25</td>
</tr>
<tr>
<td>Carnivore***</td>
<td>Rat</td>
<td>12</td>
<td>4.2</td>
<td>2.9</td>
</tr>
<tr>
<td>Carnivore†</td>
<td>Cat</td>
<td>18</td>
<td>16</td>
<td>1.1</td>
</tr>
<tr>
<td>Carnivore***</td>
<td>Cat</td>
<td>29</td>
<td>19</td>
<td>1.5</td>
</tr>
<tr>
<td>Carnivore†</td>
<td>Mink</td>
<td>31</td>
<td>20</td>
<td>1.6</td>
</tr>
<tr>
<td>Carnivore†</td>
<td>Fox</td>
<td>24</td>
<td>16</td>
<td>1.5</td>
</tr>
</tbody>
</table>

*Percent of diet (dry matter basis).
**G:M ratio = ratio of growth to maintenance requirements.
***Ideal protein (i.e., meets all known essential amino acid requirements).


<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Recommended allowance for kittens**</th>
<th>Recommended allowance for puppies***</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crude protein</td>
<td>22.5 % DM</td>
<td>17.5 % DM</td>
</tr>
<tr>
<td>EAA</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Arginine</strong></td>
<td>0.96</td>
<td>0.66</td>
</tr>
<tr>
<td><strong>Histidine</strong></td>
<td>0.33</td>
<td>0.25</td>
</tr>
<tr>
<td><strong>Isoleucine</strong></td>
<td>0.54</td>
<td>0.50</td>
</tr>
<tr>
<td><strong>Leucine</strong></td>
<td>1.28</td>
<td>0.82</td>
</tr>
<tr>
<td><strong>Lysine</strong></td>
<td>0.85</td>
<td>0.70</td>
</tr>
<tr>
<td><strong>Methionine (met + cys)</strong></td>
<td>0.44 (0.88)</td>
<td>0.26 (0.53)</td>
</tr>
<tr>
<td><strong>Phenylalanine (phe + tyr)</strong></td>
<td>0.5 (1.91)</td>
<td>0.50 (1.00)</td>
</tr>
<tr>
<td><strong>Threonine</strong></td>
<td>0.65</td>
<td>0.63</td>
</tr>
<tr>
<td><strong>Tryptophan</strong></td>
<td>0.16</td>
<td>0.18</td>
</tr>
<tr>
<td><strong>Valine</strong></td>
<td>0.64</td>
<td>0.56</td>
</tr>
<tr>
<td><strong>Taurine (extruded)</strong></td>
<td>0.1</td>
<td>-</td>
</tr>
<tr>
<td><strong>Taurine (canned)</strong></td>
<td>0.17</td>
<td>-</td>
</tr>
</tbody>
</table>

Key: EAA = essential amino acids, DM = dry matter.
*Adapted from Rogers QR, Morris JG. Optimizing protein and amino acid nutrition for cats and dogs. In: Proceedings, Roche Technical Symposium and 1997 Petfood Institute Conference and Trade Show, Chicago, IL: 19-32.
***Arginine requirement increases in kittens with increased dietary protein; approximately 2 g/kg should be added for each 10% increase in crude protein above the minimum allowance (22.5%).

### Table 19-6. Comparison of minimal protein and amino acid requirements for growth in kittens and puppies.*
As a β-amino acid, taurine is neither incorporated into proteins nor degraded by mammalian tissues. However, taurine has important functions in virtually all body systems. In addition to its importance in normal bile salt function, taurine is essential for normal retinal, cardiac, neurologic, reproductive, immune and platelet function. Taurine is needed for normal fetal development and may function as an antioxidant, osmolyte and neuromodulator. Most animal tissues, particularly skeletal muscle, heart, viscera and brain, contain high levels of taurine; plants contain none. Taurine is essential in foods for cats because of two factors:

- The feline liver has a limited capacity to synthesize taurine. The rate-limiting enzymes responsible for conversion of methionine and cysteine to taurine (i.e., cysteine dioxygenase and cysteine sulfenic acid decarboxylase) are minimally active.
- Cats have an obligate loss of taurine via the enterohepatic circulation of bile acids. Taurine is important in the conjugation and secretion of bile acids. Many animals conserve taurine by switching to glycine conjugation when dietary taurine becomes scarce. Feline hepatic enzymes do not use glycine, but conjugate bile acids mostly to taurine. Most bile salts secreted into the intestinal lumen are returned to the liver after intestinal uptake. However, once deconjugated, taurine is available for intestinal uptake, fecal excretion or degradation by intestinal microbes. Microbial degradation appears to account for deconjugation and substantial wasting of free taurine. This process also results in an obligate taurine loss.

The requirement for taurine is influenced by dietary factors and the metabolic needs of cats. The protein source, commercial processing, sulfur-containing amino acid content and dietary fiber levels all influence taurine requirements. In general, taurine is abundant in animal tissues and absent in plants; thus, homemade vegetarian diets and cereal-based dog foods have long been known to cause taurine deficiency when fed to cats. However, in 1987, taurine deficiency was reported to occur in cats fed commercial foods containing the National Research Council’s recommended levels of taurine (400 mg taurine/kg food). This finding underscored the food-dependent nature of the taurine requirement and prompted an increase in taurine recommendations by the Association of American Feed Control Officials to 1,000 mg/kg and 2,000 mg/kg (ppm) food in commercial dry and moist foods, respectively. However, taurine levels of 2,500 ppm are often recommended for moist products. Taurine adequacy is best established through feeding trials.

Because taurine functions throughout the body, signs of deficiency have been demonstrated in virtually all body systems. Three syndromes of taurine deficiency in cats have been well established: 1) feline central retinal degeneration (FCRD), 2) reproductive failure and impaired fetal development and 3) feline dilated cardiomyopathy (DCM). Hearing loss, platelet hyperaggregation and impaired immune function have also been demonstrated although specific clinical disorders have not been recognized.

**CLINICAL SIGNS**

Clinical signs of taurine deficiency occur only after prolonged periods of depletion (i.e., five months to two years). Typically in non-reproducing adults, taurine deficiency may manifest as FCRD, DCM or both, with only about 40% of taurine-deficient cats exhibiting clinical signs.

Clinical signs of FCRD are inapparent until significant visual impairment has occurred. Then, owners may notice their cat bumping into objects or “miscalculating” jumps. Early disease may be detected during ophthalmic examination. Changes in retinal function can be demonstrated by electroretinograms before retinal lesions appear. The development of FCRD apparently requires three or more months of taurine depletion. Initially, lesions appear as dark granular focal defects in the area centralis, slightly temporal to the optic disk. As degeneration progresses, the lesion becomes hyperreflective and extends in a band across the tapetum. Complete blindness ensues with full degeneration of the retina and attenuation of retinal vessels. Structural changes within the retina are permanent. Therefore, a diagnosis of FCRD does not reflect the current taurine status of a cat, but indicates a period of prolonged taurine deficiency has occurred.

Cats with DCM may be clinically normal or present acutely with signs of heart failure. Clinical signs may include lethargy, anorexia and dyspnea. Physical findings may include pleural effusion, pulmonary edema, gallop heart rhythms, systolic murmurs and ventricular dysrhythmias. Cats in severe heart failure are hypothermic, have pale mucous membranes, poor pulse quality and are often too weak to stand. Only about one-third of cats with DCM have concurrent FCRD. DCM is confirmed by echocardiography. Findings most often include dilatation of the left atrium and ventricle and decreased left ventricle contractility.

**REPRODUCTION AND FETAL DEVELOPMENT**

Reproduction and fetal development are severely impaired in taurine-deficient queens. Conception and implantation appear normal; however, fetal death is frequently observed near 25 days of gestation, followed by abortion or resorption. In a group of taurine-deficient queens, only 33% of 33 matings resulted in term deliveries, with an average of 2.1 live births.

Developmental abnormalities reported to occur in kittens born to taurine-deficient queens include poor survival, cerebellar dysgenesis, abnormal hind-limb development and thoracic kyphosis, which appears as a dorsoventral flattening of the thoracic cavity. Severe hydrocephalus and anencephaly may be present in aborted fetuses. Surviving kittens are often small and weak. Growth is depressed up to 40% in the immediate postnatal period.

**DIAGNOSIS**

The diagnosis of taurine deficiency is based on clinical signs and low plasma and whole blood taurine concentrations. Care must be used when evaluating plasma taurine concentrations because levels may be altered by sample handling errors and feeding. Fasting may reduce plasma taurine concentrations, whereas poor handling may allow taurine contamination from platelets and white blood cells. Although plasma taurine concentrations reflect the labile taurine pool, whole blood taurine concentrations better reflect tissue taurine status. Normal plasma taurine levels in cats may vary up to fivefold (50 to 250 nmol/ml). Plasma taurine values less than 40 nmol/ml may suggest taurine deficiency. Cats with clinical signs of deficien-
cy typically have plasma taurine values less than 10 nmol/ml. Therefore, taurine deficiency is best evaluated using whole blood taurine concentrations. Whole blood taurine levels are normally greater than 300 nmol/ml, and values less than 160 nmol/ml are considered deficient, whereas values less than 50 nmol/ml are common. Samples should be collected and submitted according to protocols recommended by the individual clinical laboratory. Care should be used when collecting blood and plasma for taurine analysis. Falsely elevated plasma taurine levels may result from even small amounts of clotting, hemolysis or inclusion of platelets and white cells in the plasma sample. Use of serum for determining taurine status is not recommended because the quantity of taurine in serum varies with the procedure and the time allotted for coagulation and retraction of the clot.

**PATHOPHYSIOLOGY**

A central uniform mechanism of taurine action has not been determined, and may not exist. FCRD represents a disruption and loss of the photoreceptor outer segment. Within the retina, taurine may stabilize cell membranes, possibly acting as an antioxidant. In DCM, taurine is thought to regulate myocardial calcium flux through ionic channels, thereby regulating myocardial contractility and/or mitochondrial energy production. Taurine may act as a neuromodulator or neurotransmitter in fetal development. Finally, taurine appears to influence reproduction at the level of the uterus and placenta by unknown mechanisms.

**TREATMENT**

Cats with taurine deficiency should be supplemented with 250 to 500 mg of taurine twice daily to rapidly replete tissue stores. Cats with DCM may show clinical improvement within one to three weeks, whereas FCRD and developmental defects are irreversible.

**PREVENTION**

The taurine requirement of cats depends on diet composition. Poor-quality protein, Maillard reaction products or other factors that enhance bacterial overgrowth in the intestinal tract may increase the requirement two- to sixfold. Cats require approximately 50 mg of available taurine per day, which can be supplied by high-quality animal tissues or as a crystalline amino acid supplement. Commercial foods are typically supplemented with taurine in addition to the taurine provided naturally by ingredients. Animal feeding trials are essential to ensure dietary taurine adequacy.

**ENDNOTES**

a. Pion PD. University of California, Davis, USA. Personal communication. 1994.

b. Rogers QR. University of California, Davis, USA. Personal communication. 1990.

The Bibliography for Box 19-2 can be found at www.markmorris.org.

than adult females (NRC, 2006). Felinine excretion rates of 95 mg/day have been recorded in intact male cats and may significantly increase the daily sulfur amino acid requirement (Hendriks et al, 1995).

**Fat Metabolism**

Cats have the ability to digest and use high levels of dietary fat (as is present in animal tissue). Like other true carnivores, cats have a special need for arachidonic acid (AA) (20:4n6) because they have a limited ability to synthesize it from linoleic acid (18:2n6), unlike dogs and other omnivores (MacDonald et al, 1984, 1984a). An exogenous source of AA is especially important for more demanding lifestages, such as gestation, lactation and growth. The basis for this additional requirement is the low hepatic delta-6 desaturase activity in cats (Sinclair et al, 1979). Delta-6 desaturase is the rate-limiting factor in the conversion of linoleic acid to γ-linolenic acid, which is further elongated and desaturated to form AA. AA is abundant in animal tissues, particularly in organ meats and neural tissues, but absent in plants. Thus, the dietary requirement for AA has little consequence if cats consume animal tissues (MacDonald et al, 1984).

**Vitamin Metabolism**

The vitamin needs of cats differ from those of dogs in several ways. Cats do not convert sufficient amounts of tryptophan to niacin (DaSilva et al, 1952). An animal tissue-based diet is well supplied with niacin from NAD and NADP (nicotinamide-adenine dinucleotide phosphate) coenzymes; therefore, cats don't need to produce niacin from tryptophan. Although cats possess all the enzymes needed for niacin synthesis, the high activity of enzymes in the catabolic pathway (picolinic carboxylase) far exceeds the rate of niacin synthesis (Morris and Rogers, 1983). As a result, the niacin requirement of cats is 2.4 times higher than that of dogs (NRC, 2006).

The prosthetic group of all transaminases is pyridoxine (vitamin B6) (Stryer, 1975). Cats have high transaminase activities, consistent with consuming a diet from which considerable energy is derived from dietary protein. Therefore, it is logical to expect that their pyridoxine turnover and requirement would be higher than that of omnivores. The pyridoxine requirement of cats is estimated to be 1.7 times higher than that of dogs (NRC, 2006).

Vitamin A occurs naturally only in animal tissue. Plants synthesize vitamin A precursors (e.g., β-carotene). Omnivorous and herbivorous animals can convert β-carotene to vitamin A; cats cannot because they lack intestinal dioxygenase that cleaves β-carotene to retinol. In addition, cats have insufficient 7-dehydrocholesterol in the skin to meet the metabolic need for vitamin D photosynthesis; therefore, they require a dietary source of vitamin D (How et al, 1994, 1994a; Morris, 1996). Vitamin D is relatively abundant in animal liver; therefore, the need for dermal production is minimal and alternate pathways rapidly metabolize 7-dehydrocholesterol. Vitamin D is fairly ubiquitous in animal fats and primary vitamin D deficiency has been identified only in cats fed experimental diets.
**Water**

Water needs of cats also differ from those of dogs, not because of feline feeding behaviors (i.e., carnivorous vs. omnivorous) but because of their ancestors’ adaptation to environmental extremes. Domestic cats are thought to have descended from the African wildcat (*Felis silvestris libyca*), a desert dweller. Several unique features of water balance in cats may be explained by adaptation to a dry environment. Cats are able to survive on less water than dogs and may fail to increase water intake at minor levels of dehydration, up to 4% of body weight (Anderson, 1982). Cats compensate for reduced water intake, in part, by forming highly concentrated urine. Unfortunately, this strong concentrating ability coupled with a weak thirst drive may result in highly saturated urine, increasing the risk of crystalluria or urolithiasis, both components of the feline lower urinary tract disease complex.

Cats consume 1.5 to 2 ml of water/g of dry matter (DM). This 2:1 ratio of water to DM is similar to that of typical prey. This ratio represents approximately 0.5 ml water/kcal ME intake. Practical recommendations for water provision are somewhat higher at 1 ml water/kcal ME. Water ingested from moist foods containing 78 to 82% moisture will result in diuresis.

### LIFESTAGE NUTRITION

Lifestage nutrition is the practice of feeding foods designed to meet an animal’s optimal nutritional needs at a specific age or physiologic state (e.g., maintenance, reproduction or growth). The concept of lifestage nutrition recognizes that feeding either below or above an optimal nutrient level can negatively affect biologic performance and health (Figure 5-2). This concept differs markedly from feeding a single product for all lifestages (i.e., all-purpose foods), whereby nutrients are added at levels to meet the highest potential need (i.e., usually growth and reproduction). For maximum benefit, risk assessment and preventive plans should begin well before the onset of disease. The value of lifestage nutrition is greater if risk factor management is also incorporated into the feeding practice. A narrower range of nutrient recommendations often emerges when age and physiologic needs are reviewed in conjunction with reducing nutritional risk factors for disease.

### Box 19-3. Commercial Treats and Table Foods.

An estimated 41 to 60% of cats are regularly fed table foods and 34% of cats are fed commercial treats. Feeding treats and table food allows more social interaction with the owner, increases diet variety and provides additional caloric intake. Some commercial treats claim dental benefits either by mechanical cleansing or through use of an active ingredient (Chapter 47). When fed in excess, treats and table foods may negatively affect a well-balanced food. Because most commercial cat foods contain vitamins and minerals well above the nutritional needs of cats, table foods and treats fed at less than 10% of the total daily intake should be safe. Providing high-calorie treats or table foods can also contribute to obesity and must be considered in the calculation of total calories for a cat.

### MILK

One of the most common human foods offered to cats is milk. Milk is highly palatable and small quantities are well tolerated by most healthy cats. However, after weaning, intestinal lactase activity declines unless milk is a regular part of the diet. Undigested lactose is subject to bacterial fermentation and promotes osmotic diarrhea. Feeding milk to cats unaccustomed to receiving it may overwhelm digestive capacity resulting in diarrhea, flatulence or gastrointestinal distress. Although commercial lactase supplements may alleviate signs of lactose intolerance, lactose avoidance is more prudent for affected cats.

### NUTRITIONAL SUPPLEMENTS

Although many supplements are legitimate sources of essential nutrients, others represent food facts that reflect current trends in human nutrition. Poor-quality foods are rarely “fixed” by adding a supplement. Changing to a higher quality food is a more appropriate recommendation and often less expensive.
Nearly all commercial cat foods meet or exceed the minimum nutrient requirements; however, certain nutrients may still be outside of the desired nutrient range for optimal health. For cats fed commercial foods, these nutrients require particular consideration and, thus, are referred to as nutrients of concern. Specific food factors (e.g., digestibility, texture and effect on urinary pH) can also affect health and risk of disease (Chapter 1). Together, nutrients of concern and specific food factors are called key nutritional factors. Cats eating homemade foods are at greater risk for nutrient deficiencies (e.g., calcium) and excesses (e.g., phosphorus) than those eating commercial foods. Therefore, these cats have a longer list of key nutritional factors, which are discussed in Chapter 10. Box 19-3 includes information about a variety of popular topics regarding foods for cats including commercial treats, table foods, vegetarian foods and dog foods. The key nutritional factors for different lifestages of healthy cats will be discussed in the following chapters, including those factors associated with reducing the risk of specific diseases and those involved with optimizing performance during different physiologic states. In sequence, these chapters cover Feeding Young Adult Cats: Before Middle Age, Feeding Mature Adult Cats: Middle Aged and Older, Feeding Reproducing Cats, Feeding Nursing and Orphaned Kittens from Birth to Weaning and Feeding Growing Kittens: Postweaning to Adulthood. The chapter about feeding young adult cats is presented first because most pet cats are adults and the nutrient needs of adults serve as a good basis for comparing nutrient needs for reproduction, lactation and growth.

**ACKNOWLEDGMENT**

The authors and editors acknowledge the contributions of Dr. Claudia A. Kirk in the previous edition of Small Animal Clinical Nutrition.

**REFERENCES**

The references for Chapter 19 can be found at www.markmorris.org.
Minerals
Providing adequate calcium is a concern in any homemade food. A variety of calcium supplements are available from health food stores and pharmacies. Many plant ingredients contain components (e.g., fibers or phytates) that severely compromise the availability of certain trace elements. The availability of iron, zinc and copper is of particular concern in high-phytate and high-fiber foods (Chapter 5). These minerals should be provided as a highly available source.

Fat
Of the nutrients required by cats, arachidonic acid is the one not commercially available. To provide arachidonate directly, cats must be given animal fat or tissue as a nutritional source. However, cats can convert γ-linolenic acid (18:3n6) to arachidonic acid (20:4n6) via delta 5-desaturase. γ-linolenic acid is available from plant oils (e.g., borage and evening primrose oils). Prolonged feeding and reproductive trials using γ-linolenic acid have not been reported; thus, the suitability of these oils as long-term arachidonic acid supplements is unknown. Because cats fed foods high in polyunsaturated fatty acids may develop steatitis, cats fed vegetarian foods with large quantities of plant oils should be protected with added vitamin E.

DOG FOOD
Most dog foods are not nutritionally adequate for the maintenance, growth and reproduction of cats. Nutrients most likely to be deficient are protein, taurine, niacin, vitamin B6, methionine and choline. Clinical signs of deficiency depend on which nutrients are deficient and to what degree.

FOOD TOXINS
Food toxicities are relatively infrequent in cats. Most notable is hemolytic anemia caused by onion toxicity. Certain disulfides found in onions promote oxidative damage to cat hemoglobin, resulting in Heinz body production and red cell removal. The toxic compound appears to be highly stable, because it remains active in cooked onion-based broth and onion powder. Hemolytic anemia has been described in a cat fed commercial baby food containing onion powder. Onion toxicity was not proved but the anemia resolved with a diet change. Subsequent studies have demonstrated toxic effects at levels of 2.5% dry matter. Therefore, it is prudent to avoid feeding food or seasonings containing onion powder or onions. Chapter 11 provides more information about foodborne toxins.

Theobromine
Theobromine is a toxic methylxanthine found in chocolate. The clinical signs of toxicity include vomiting, diarrhea, vascular collapse and death. The oral LD50 of theobromine is 200 mg/kg body weight. Approximately 40 to 50 g of cocoa would need to be consumed to provide this dose of theobromine, which is undoubtedly why clinical reports of chocolate toxicoses in cats are rare.

Histamine
Histamine is a primary amine arising from the decarboxylation of histidine. Histamine toxicosis has been reported to occur in cats after ingestion of certain species of spoiled fish. Affected cats developed salivation, vomiting and diarrhea about 30 minutes after eating uncooked anchovies. Myosis, lacrimation, tachypnea and tachycardia were evident upon physical examination. A survey detailing the histamine content of North American cat foods found foods were well below the 500 mg/g (wet/weight) level considered hazardous in people. Thus, histamine toxicosis is most likely to occur in cats fed improperly handled fish that has undergone spoilage.

ENDNOTE

The Bibliography for Box 19-3 can be found at www.markmorris.org.