

# NUTRITION OF THE DOMESTIC CAT, A MAMMALIAN CARNIVORE

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## INTRODUCTION

Wild or domesticated carnivores belonging to the family Felidae were kept in a captive or semidomesticated state by the Egyptians at least 3000 years ago (264). Despite this long association between humans and carnivores, precise information on the nutritional requirements of these mammals has been lacking until relatively recently. This review focuses on unique aspects of the nutrition of the domestic cat (*Felis domesticus*), the most thoroughly studied mammalian carnivore. It is our thesis that the domestic cat is a good model for studying the peculiarities in the nutrition of carnivores.

The ancestral origin of the domestic cat *F. domesticus* and the precise date of its domestication are open to dispute. Resolution of both of these questions is hampered by the fact that there were relatively few skeletal changes in the cat after its domestication. The cat may have been domesticated by the Egyptians as early as 3000 BC, but Zeuner (264) considers the evidence ambiguous. In any case, the cat was clearly domesticated and regarded as a sacred animal by 1600 BC. Cats were sacred to the Egyptian goddess Bast of Bubastis and were regarded as her incarnation (35). According to the Roman historian Diodorus (circa 100 BC), the Egyptians fed their cats on bread, milk, and slices of Nile fish, and they called their animals to their meals by special sounds (35). On the basis of mummified embalmed cat bodies recovered in recent times, the number of cats kept by the Egyptians must have been prolific. During the latter part of the nineteenth and early twentieth centuries, mummified cats were excavated at Bubastis and other places in Egypt and were spread on the land as fertilizer. They were so numerous that they were also exported; one consignment sent to England for fertilizer weighed nineteen tons (170).

The Egyptians collected and kept various recognizable species of cats including *F. chaus*, the jungle cat, and *F. lybica*, the African wild cat. However, most of the mummified skulls referred to above (170) were found on examination to be smaller than *F. chaus* but larger than *F. lybica* and the domestic cat. It has been suggested that the present domestic cat may have been derived from interbreeding of *F. silvestrus*, the European wild cat, and *F. lybica* (202).

In classifying the cat, Carl Linne named the domestic cat *F. catus*, which he described as having blotched tabby markings; apparently he neglected or was oblivious to the equally common mackerel type (202), which came to receive the designation of *F. torquata*. Thus, the specific name *F. domesticus* appears to be the most appropriate and all-inclusive name to describe the domestic cat.

The Feloidae (Viverrids, Hyaenids, and Felids) diverged from the other members of the order Carnivora relatively early in their evolutionary development, i.e., during the late Eocene to the early Pleistocene. After the Felids diverged from the Viverrid ancestry, they underwent rapid evolution to fully specialized forms, which have not changed for millions of years (48).

The closest relatives of the Feloidae, the Canoidae (which include Procyonids, Ursids, Ailurides, Canids, and Mustelids) exhibit much greater diversity than the Feloidae. The Ursids (bears) are well recognized as omnivores. The molar teeth of the Ursids are elongated, and the enamel of the crowns is fissured, a characteristic which indicates an omnivorous or herbivorous diet. The Procyonids, of which the raccoon is a typical member, are also omnivorous. The Ailurids, which probably are an offshoot of the Ursids, are represented by the lesser and greater pandas, which are strict herbivores.

In contrast to the Canoidae, all members of the Feloidae are flesh-eaters, i.e. strict carnivores, so it is clear that the order Carnivora contains mammals with widely divergent dietary patterns. The order may also provide researchers with an opportunity to determine whether adherence to a specialized diet during evolution induced or contributed to changes in metabolic pathways and nutritional requirements. One could anticipate that these changes might be expressed in the Feloidae but not in the Canoidae.

A comparison of the nutritional requirements of the cat and the dog as representative members of the Feloidae and Canoidae supports the thesis that

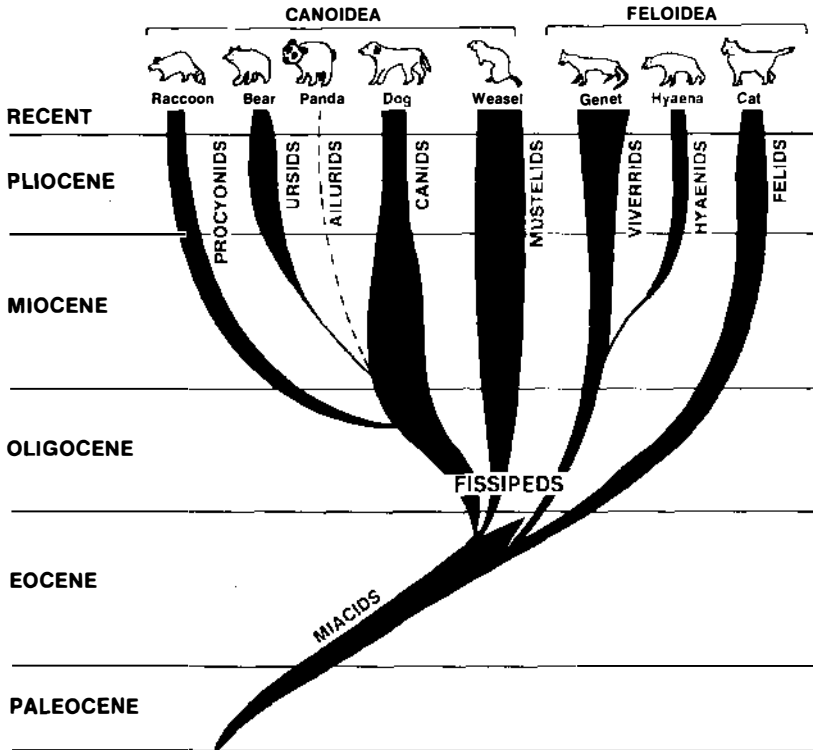


Figure 1 The relationship of the cat to other members of the order Carnivora. (After Refs. 165, 210).

specialization consistent with the evolutionary influence of a strict carnivorous diet has occurred in the cat. Examples of this specialization, which will be discussed more fully later, are: (a) The cat has limited ability to regulate catabolic enzymes of amino acid metabolism, which causes the cat to require a higher level of dietary protein for maintenance than the dog. (b) The cat does not synthesize niacin from tryptophan because of a very active alternate degradative pathway. (c) The dog (but not the cat) converts carotene to vitamin A and may therefore obtain its vitamin A requirement from plants. (d) The dog (but not the cat) can convert enough linoleic acid to arachidonic acid, and may therefore meet its arachidonic acid requirements from plant sources. (e) The dog can synthesize sufficient taurine from cysteine and can conserve taurine when limited by using glycine conjugation. The cat has a lower capacity to synthesize taurine than the dog and is unable to use the alternate glycine conjugation. Thus, unlike the cat, the dog can meet its taurine requirement from dietary sulfur amino acids supplied from plant sources. All the above nutrients that the cat cannot synthesize are found in a carnivorous diet.

## EATING AND DRINKING BEHAVIOR

### *Rhythms in the Cat*

Many biological variables that exhibit rhythmicity in most mammals (15, 160) do not appear to do so in the domestic cat (102, 189, 241). The behavior patterns that do not show daily rhythmicity include feeding and drinking (131, 171, 246), activity (102), and sleep-wake (159). Thus, if the domestic cat is allowed to eat ad libitum, it will eat many (12–20) small meals per day, and these will be approximately evenly distributed between the light and dark periods (131, 171, 246). Core body temperature is normally circadian in most mammals, but it does not appear to vary on a daily basis in the cat (102). This virtual absence of circadian rhythmicity appears to be a function of the cat's natural behavior as a predator; circadian rhythmicity is also absent in certain other felines, such as the bobcat. There is some evidence that these rhythms are also less rigid in the dog, which is a less strict carnivore than is the cat (160). The lack of many of the usual circadian rhythms does not mean, however, that cats have no biological clocks. Instead, it appears that many of their activities simply are not coupled to their biological clocks. Cats' reproductive cycles depend upon day length to trigger the female estrus cycle (19, 63). It has been shown that the most pronounced estrus in the cat is induced by a period of extended daylight (more than 12 hours) after a period of short days (119). In nature this occurs with the change of seasons, which establishes the breeding season in the late winter and early spring; however, it can be induced in the laboratory during any season by lighting manipulations (119). Also, cerebrospinal fluid (CSF) vasopressin concentration varies as a circadian rhythm of

slightly greater than 24 hours. This rhythm is characterized by high daytime values and low night values (189).

As in other mammals (190), melatonin concentrations in cat CSF exhibit a prominent daily rhythm that is characterized by high levels during the dark period and low levels during the light hours (189). However, the CSF melatonin pattern splits into several components as the time in constant light is increased. The fact that the melatonin rhythm in the cats persisted during exposure to constant light is somewhat enigmatic, because in most mammals artificial light of moderate intensity suppresses melatonin production so that no rhythm exists (190). The splitting of the CSF melatonin into at least two distinct components during constant light may represent the disassociation of multiple circadian oscillators normally involved in the generation of this hormone rhythm. More work on the circadian rhythms of cats needs to be done to establish which rhythms are intrinsically linked to feeding behavior and to establish whether they exert the driving force for the behavioral patterns that result in their natural predatory behavior.

### *Feeding Patterns and Food Intake Regulation*

The predatory behavior of the cat has been studied and described in great detail (145). The feral cat (*Felis domesticus*) eats a wide variety of prey, which varies seasonally and includes mice, rats, rabbits, birds, lizards, insects, and other animals (51, 72, 74, 154). Most wild felids (the lion is one exception) are solitary hunters that rely on craftiness and speed to catch their prey (25, 74, 219). It is easy to see that the caloric content of each of their meals may vary considerably. Mice are the most common prey for the domestic cat (51, 72), and an average mouse consists of about 30 kcal (171). Thus, the average cat would require 12 meals of one mouse each per day. It is often thought that domestic cats would naturally eat large infrequent meals, as do lions (74, 219).

Grouping all cat families together, researchers usually described the cat feeding pattern as intermittent or occasional (225). It has now been shown, however, that uncaged cats (133) or caged cats fed purified diets (131) or commercial diets (128, 131, 171, 172) will eat 10–20 times a day if they are given the food free-choice. As many of these meals will be eaten during the dark period as will be eaten during the light period (131, 172). Normally a slightly greater quantity of food and a slightly larger number of meals are eaten during the period when the animals are given fresh food, whether it is light or dark (131, 172).

It has been shown for both caged (50, 128) and uncaged (133) cats that the more work a cat must do to obtain a meal, the fewer meals and the larger the meal size it will eat. This same phenomenon occurs in other species (49, 132).

Adamec (1) showed that predatory behavior takes precedence over consummatory behavior; cats will stop eating highly palatable food to go kill a rat,

even though they will then go back to their initial food rather than eat the rat. This behavior appears to have the functional value of increasing food input by providing for multiple kills when the opportunity arises.

Kanarek (128) and Hirsch et al (112) reported that when cat diets were diluted with celluloflour (128) or kaolin (112) the cats ingested a constant quantity of food (and therefore fewer calories) rather than ingesting more food so that they would obtain a constant quantity of calories. Kanarek (128) concluded that this might be true because cats are carnivores and the caloric content of their food supply would not vary greatly in nature. She suggested that cats may therefore regulate their food intake on the basis of bulk rather than calories. Although both studies considered palatability as a possible factor, it was dismissed because total food intake did not decrease when there was increased nondigestible material in the diet. Careful examination of the results of Hirsch et al (112), however, shows that the cats' food intake decreased on the first day after they received the higher levels of kaolin more than it did on subsequent days. Also, food intake was greatly increased the first day after the cats were returned to the kaolin-free diet, which indicates that palatability certainly was one factor. Most prey animals contain at least 4.5 kcal/g dry weight; therefore, the basal diet of dry cat food which contains 3.4 kcal/g dry weight should be considered already calorically dilute for the cat, which has a short gastrointestinal tract and therefore a rapid transit time. It is possible that the total volume of diet ingested was becoming somewhat limiting before the diets were calorically diluted. Mugford & Thorne (172) showed that when cats are fed isocaloric diets, they decrease their intake of a diet that is low in palatability by decreasing both meal size and meal frequency. Castonguay (42) showed that cats compensate effectively for water dilution of the diet by increasing their intake of the calorically dilute diets. Kane et al (131) used both purified and commercial diets of good palatability but with different caloric contents and showed that cats quickly adjust the quantity of food they ingest to maintain a quite constant caloric intake. Although there is high variability in eating patterns among cats and although the cat is more sensitive to the physical form and taste of its diet than are the rat, dog, and many other animals, the cat does have mechanisms to control caloric intake. This is consistent with reports that cats have fewer problems with obesity than dogs when both are given highly palatable diets (116, 171).

Anand & Brobeck (6) showed that cats that are bilaterally lesioned in the ventromedial hypothalamus (VMH) become hyperphagic and obese, whereas those lesioned in the lateral hypothalamus (LH) become aphagic. Other workers have shown that lesions in the VMH (233, 235), periaqueductal gray matter (234), rhinencephalon (161), and dorsal periaqueductal gray matter (236) result in hyperphagia in cats. Thus, the neuropathways involved in the control of food intake in cats appear to be similar to those of other animals. One might expect,

however, that some of the metabolic cues would be different in the cat, a carnivore, than in the rat, an omnivore. Although little is known about metabolite mediation of satiety in the cat, it has been shown that low doses of 2-deoxy-D-glucose (50–100 mg/kg body weight) increased short term (1–3 hour) food intake fourfold (213), which suggests that the cat may have glucostatic, short-term controls of feeding as do other mammals. It appears, in fact, that lower doses are required to produce this effect in the cat than in other animals; administration of 2-deoxy-D-glucose at the dose levels used for the rat (200–750 mg/kg) caused vomiting (213) and produced a soporific effect (121), which resulted in a decrease in food intake.

Cats respond classically to a learned-aversion paradigm using lithium chloride (171) but appear to be generally neophilic rather than neophobic like the rat. Cats will eat a novel diet rather than a familiar diet unless they are put in a new environment or a stressful situation, in which case they will choose the familiar diet (172).

As a carnivore, the cat might be expected to have taste preferences that differ from those of omnivores and herbivores. Indeed, early studies indicated that the cat did not detect (185) or select (40) sweet solutions. Other studies indicated that cats would select sweet substances only if they were in diluted milk (82) or in saline solutions (22), because the cat has a negative response to pure water solutions (22, 82). Although it was reported that saccharin solutions were selected by cats (263), Bartoshuk et al (23) found that cats rejected both saccharin and cyclamate solutions. The work by Beauchamp et al (24) supports the idea that cats are neutral to sugars whether in water or saline. These workers also found that cats show a preference for sucrose and lactose in a diluted milk solution, but a mechanism for this phenomenon has not yet been elucidated. Beauchamp et al (24) confirmed and extended the work of Boudreau and coworkers (32, 257) by showing that the cat has a positive taste preference for protein hydrolyzates, meat extracts, and certain free amino acids such as alanine, proline, lysine, and histidine. This demonstrated the practical significance of the elegant work of Boudreau and coworkers, who showed that amino acids that elicit an increase in spike output from geniculate ganglion chemoresponsive group II units were preferred in a preference test, while amino acids that decreased group II discharge were avoided (32, 257).

Animal lipids improve the palatability of cat diets (128). Acceptance by cats of diets containing 25% fat derived from different sources was reported (129). These authors tested bleached tallow versus six alternate fats in a two-choice test. All cats preferred diets based on bleached tallow to those made with butter oil or chicken fat. There was a tendency for cats to prefer unbleached tallow and yellow grease over bleached tallow, but cats showed no preference between partially hydrogenated vegetable fat and bleached tallow. When purified diets containing 10, 25, and 50% yellow grease were presented to cats, they showed

a preference for the diets containing 25% yellow grease over either 10% or 50% yellow grease. The level of fat in purified diets has a marked effect on texture of the diet, however, so the cats may have preferred diets containing 25% fat partly because these diets have a more desirable texture than those containing 10% fat, which are powdery in nature, or those containing 50% fat, which are greasy (129). In a subsequent study, these authors found that cats consumed more of a diet made with 40% bleached tallow than of those made with either 12 or 25% bleached tallow.

Experimental diets based solely on coconut oil as a fat source have been found to be aversive to cats. Similarly, diets containing medium-chain triglycerides are aversive, and the aversion is stronger for triglycerides with C8–10 fatty acids than for those with C12 fatty acids. The aversive nature of these diets may be related to an ability of the cat to detect and avoid low levels of medium-chain fatty acids that are normally present in these fats (M. L. MacDonald, unpublished results).

### *Water Requirements and Drinking Patterns*

The cat is thought to have evolved as a desert animal, in part because it can concentrate its urine more than the dog or man can (43). This enables the cat to survive on less water, but the physiologic processes involved may also contribute to the cat's susceptibility to feline urological syndrome (FUS). Even though the cat is able to overcome a previous water deficit when given sea water to drink (262), the cat will spontaneously drink considerably less pure water under the same conditions than the dog will. A dog will accurately replenish a water deficit up to 8% of its body weight in just a few minutes; the cat will not immediately make up a water deficit of as little as 4% of its body weight (2). Although the minimum water requirement for survival is less for the cat than for the dog, the weaker thirst drive of the cat results in an ad libitum intake of water that is often too low to prevent crystallization of struvite in the urine, which makes the cat particularly susceptible to FUS. Although the actual course of FUS may differ from case to case, it is clear that increased water intake helps prevent recurrences. The only effective way to increase the cat's water intake is to feed a high-protein and/or a high-salt diet or to put water in the diet. Because a high salt level (4% or more) is needed in the diet to provide a substantial increase in water intake (13, 36, 97), the only practical way to increase water intake is to add water to the food. If fed a normal dry cat food, cats will drink about 1.5–2.0 ml of water for each gram of dry food (36, 131). This proportion (2:1) is near the proportion of water in the animals that are prey for carnivores. Thus, cats need or drink very little water if they are fed other animal tissues and probably seldom drink if they are fed canned food that is 75–80% water (131). Indeed, the most effective way to treat FUS in cats is to feed nothing but canned cat food, because this increases water intake and thereby increases urine



volume. A nutritionally complete canned food that contains 85–90% water should be even more effective in controlling FUS in susceptible cats, although no such food is currently on the market in the US.

The dog drinks only during the day unless it is dehydrated, but the cat drinks during both the light and dark periods (131). Although the number of drinking bouts are about the same as the number of meals eaten, no correlation has been found between eating and drinking patterns (131) unless the cat was eating only one or two meals a day (128).

## ENERGY REQUIREMENTS

The mature body size of members of the genus *Felis* ranges from 5 kg or less for the black-footed cat (*F. nigripes*), domestic cat (*F. domesticus*), and the African wild cat (e.g. *F. lybica*) to more than 100 kg for the puma (*F. concolor*) (57). Other members of the family Felidae, e.g. lions (*Panthera leo*) and tigers (*P. tigris*), may attain mature body weights in excess of 200 kg. To our knowledge, there have been so few estimates of energy requirements over this range of body weights, especially at the upper end, that no generalized relationship between body mass and energy requirements can be made.

Measurements from which estimates of energy requirements can be derived are available from two sources: short-term measurements of gaseous exchange and longer-term measurements of food intake and body weight changes. Theoretically, either approach should give similar values for maintenance energy requirements, but activity is extremely limited in a respiration chamber or cage and animals must be acclimated to the chamber before reliable measurements can be obtained. Further, most measurements on cats in chambers have been of fasting (basal) heat production, not of maintenance energy requirements.

Haldane (96) described a new apparatus for measuring respiratory exchange in animals and reported data on a single 2.5-kg cat of unspecified age or physiological state. When the usual thermal equivalent values are applied to the CO<sub>2</sub> production, the calculated heat production was 79 kcal/kg body weight/day.

In 1938, Benedict (30) compiled all measurements on basal heat production of cats up to that time (38, 191), which yielded a total of 30 individual measurements from five sources. The regression of basal heat production on body weight approximated linearity and gave a mean value of 57 kcal/kg body weight/day for cats with weights ranging from 1.9–3.7 kg. This mean value is similar to the heat production value we calculated from the respiratory exchange data of Carpenter (41) for cats in the postabsorptive state. The mean  $\pm$  SE was  $52.2 \pm 2.8$  kcal/kg body weight/day. A somewhat higher value of  $66.1 \pm 2.8$  kcal/kg body weight/day was found by E. Kane, J. G. Morris, and Q. R.

Rogers (unpublished data). The reason for the latter value being greater than that reported by Carpenter is not apparent.

All the measurements discussed above refer to fasting (basal) heat production, which is only one component of maintenance. If these data are to be applied in a feeding system, the other components of maintenance energy, activity and heat increment, must be included. The maintenance energy requirement is generally on the order of 1.5–2 times the basal heat production. If the approximation of 1.5 times basal heat production (58 kcal/kg body weight/day) is taken for the cat, the maintenance energy requirement is 87 kcal/kg body weight/day.

Various estimates of the cat's energy requirements based on measurement of food intake and frequently calculated energy values of foods (usually physiological fuel values) have been reported in the literature. Typical values (in kcal/kg body weight/day) are 61–67 (138), 80 (5), 66–88 (168), and 60 (85, 155) for mature cats; 76 for 13-month-old cats (252); 129 for 3.5-month-old cats (252); and 160 (5) and 250 (85, 155) for kittens. The upper estimates for mature cats are in reasonable agreement with the value of 87 kcal/kg/day calculated from basal heat production. It is difficult to reconcile the lower values, which approach basal energy expenditure.

Recently Kendall and coworkers used 143 digestibility trials to calculate the digestible energy (DE) requirements of six adult cats for maintenance (136). When energy intake for maintenance was expressed as a mass exponent of unity, the mean value was 76 kcal/day. When the mass exponents of body weight (kg) were 0.75 and 0.67, the coefficients were 104 and 114 kcal.

Although no definitive value for maintenance energy requirement can be derived from the foregoing data, a value of about 80 kcal DE/kg body weight/day appears reasonable. Compared to other mammals, the difference between the cat's maintenance and basal heat production (i.e. energy required for activity and heat increment) appears excessively small. As the cat's heat losses in the digestion, assimilation, and metabolism of food probably do not differ materially from other mammals, it appears that the activity of the cat used in the measurements must have been similar under the basal and maintenance conditions. Certainly adult cats give the impression of not engaging in unnecessary exercise, but whether this is an impression or fact remains to be investigated.

### *Carbohydrates*

The milk of the queen contains about 3–5% carbohydrate as lactose, which accounts for about 20% of the metabolizable energy of its milk (123, 134), so carbohydrate is a major component of a kitten's diet. However, the adult cat that consumes only an animal tissue diet ingests little carbohydrate. Morris et al (167) reported that adult cats efficiently utilized most common isolated carbohydrates that were added to a meat diet. Apparent digestion coefficients were

0.88 for raw corn starch, 0.87 for lactose, and more than 0.91 for glucose, sucrose, and dextrin. High concentrations of lactose produced diarrhea in some cats, presumably because they had inadequate lactase activity. Part of the disappearance of lactose may have been the result of fermentation in the lower gut, because the apparent digestibility coefficient of protein was reduced with lactose. Utilization of starch in the cereal grains, wheat and corn, was significantly greater when these grains were finely ground rather than coarsely ground; cooking also increased utilization (167). The digestibility coefficients of partially decomposed potato flour and wood cellulose were measured by deWilde & Huysentruyt (67) and found to be 0.95 and 0.31 respectively. The value for potato flour is in agreement with that previously cited for starch (167), but the wood product, because of partial hydrolysis, was digested to a greater extent than unhydrolysed cellulose, which was found to be totally indigestible.

The livers of most omnivorous animals have two enzymes, glucokinase and hexokinase, that catalyze the phosphorylation of glucose to glucose-6-phosphate. Hexokinase has a low  $K_m$  for glucose, whereas glucokinase has a high  $K_m$  and operates only when the liver receives a large load of glucose from the portal vein. The activity of glucokinase in the liver of the cat is extremely low, whereas the activity of hexokinase is in the normal range (18). Carnivores with omnivorous dietary habits, e.g. domestic dogs, possess both enzymes, so it seems likely that they have a greater capacity to handle a high glucose meal than the cat; however, it does not appear that this has been tested.

In omnivorous animals, maximal gluconeogenesis occurs in the postabsorptive state, which occurs some hours after a meal; in carnivores ingesting diets of animal tissue, maximal gluconeogenesis occurs in the absorptive phase immediately following a meal because the capacity to store protein per se is minimal. The relationships between maximal gluconeogenesis and meals are similar in carnivores and ruminants; also, gluconeogenesis is more or less permanently "switched on" in both groups of animals (146, 147, 165). There may also be differences between cats and omnivores in the relative importance of various gluconeogenic pathways. Beliveau & Freedland (27) showed that inhibition of cytosolic phosphoenolpyruvate carboxykinase depressed gluconeogenesis from serine in rat hepatocytes but not in cat hepatocytes. This finding suggests that in the cat, serine is converted to glucose by a route that does not involve pyruvate and the enzyme serine dehydratase. Low activity of serine dehydratase in the liver of the cat has been reported (209, 214), which provides support for the notion that there is an alternate pathway to pyruvate that probably involves hydroxypyruvate. Rowsell et al (214) suggested that high activity of the first enzyme of this pathway (L-serine-pyruvate aminotransferase) in mammals is associated with diets of animal flesh.

No dietary requirement for carbohydrate has been demonstrated for the cat. Growing kittens and adult cats at maintenance probably do not require dietary

carbohydrate if their diets furnish triglycerides and adequate concentrations of glucogenic amino acids. A need for dietary carbohydrate has been demonstrated in pregnant Beagle bitches given a carbohydrate-free diet containing 26% of energy from protein and 74% from fat (211). Bitches fed this diet became hypoglycemic at whelping and had depressed concentrations of lactate and alanine (glucose precursors) in plasma. Puppies from hypoglycemic bitches had a low survival rate, which may have been at least in part because of reduced mothering ability of the dams. Whether the queen requires a source of dietary glucose under comparable conditions has not been determined.

### *Lipids*

Fat in the diet has three major functions: It provides a concentrated source of energy, it supplies essential fatty acids, and it serves as a carrier for fat-soluble vitamins. Fat may also have other roles related to the acceptability of food and to the provision of sensory (textural and organoleptic) cues for cats.

Dry commercial diets intended for cats usually contain 9–15% crude fat on a dry-matter basis (129). However, experimental diets ranging from less than 1 to 64% fat in the dry matter have been fed to cats. Similarly, a wide variety of animal and vegetable fats and oils have been incorporated in experimental diets (118). Cats have the ability to digest and utilize high levels of dietary fat. Most of the measurements reported on apparent digestibility of fat by cats have been derived from measurement of crude fat. Crude fat is the material extracted with anhydrous diethyl ether; it is represented in cat foods primarily by triglycerides but can also include other lipid-soluble substances of little or no nutritional value, such as plant pigments, sterols, and waxes. In expanded or baked cat foods, not all of the glycerides may be extracted with diethyl ether (34, 113) unless ether extraction is preceded by acid hydrolysis (16). Therefore, ether extraction may not provide adequate measurement of all of the triglyceride in these diets.

Apparent digestibility coefficients of crude fat in commercial cat foods marketed in the US have been reported to range from 0.85 to 0.94 (179). These values are similar to those reported from the United Kingdom for apparent digestibilities of acid ether extract in commercial cat foods, which were 0.79 in dry foods, 0.88 in canned foods, and 0.92 in semimoist foods (137). Apparent digestibility coefficients of crude fat in experimental diets that did not involve extruding or baking generally have been higher than those reported for commercial diets. Mean values of 0.99 for beef and mutton fat (167) and 0.98 for butter, lard, unbleached and bleached tallows, chicken fat, and yellow grease have been reported (129). In one study (129), no significant differences were found between the apparent digestibility coefficients of individual fatty acids ranging from C12:0 to C18:3 for adult cats.

## PROTEIN AND AMINO ACIDS

One of the most striking aspects of protein and amino acid nutrition is that the same amino acids are essential for virtually all animal species across the whole scale of the phylogenetic tree. Our emphasis, however, is on what makes the cat different or unique in its protein and amino acid nutrition.

The essentiality of the amino acids has been determined in the growing kitten. As expected, the essential amino acids include arginine, histidine, lysine, leucine, isoleucine, valine, methionine, threonine, tryptophan, and valine (206). Dietary asparagine is required for maximal growth in the early post-weanling kitten (E. M. Kamikawa, J. G. Morris, Q. R. Rogers, unpublished results) but is not necessary after the first few weeks. Taurine is a dietary essential for the cat in order to prevent central retinal degeneration (CRD), but because this amino acid is not an  $\alpha$ -amino carboxylic acid, is not found in proteins, and is a metabolite of cysteine, it is discussed in the section on Sulphur Amino Acids below. From a qualitative point of view then, nothing appears unique about the essentiality of these amino acids. From a quantitative viewpoint, however, several interesting differences have been found.

### *Protein Requirement*

An animal's protein requirement normally reflects both its minimal nitrogen requirement and its essential amino acid requirements. If the amino acid requirements are known, the researcher can formulate the diet so that even the lowest level of protein tested meets all the essential amino acid requirements; under these conditions the minimal protein requirement becomes a measure of the nitrogen requirement of the animal. Likewise, to determine the essential amino acid requirements, the nitrogen requirement must be met in all diets so that the only limiting factor is the amino acid being examined.

The earliest experiments in determining the protein requirements of kittens and adult cats used foodstuffs (68, 92, 93) or purified components (5, 79, 122, 155) and were done without any knowledge of the amino acid requirements. Under those conditions the protein requirement was shown to be about 30% of the diet for the growing kitten and about 20% of the diet for the adult cat. Anderson et al (9) used amino acid diets in which the amino acid requirements were met and concluded that 16% of the dietary calories as protein meets the nitrogen requirement. When the data from the latter study are plotted, there are no break-points on the curves, so it is not obvious how the authors arrived at their interpretation. After ensuring that all amino acid requirements were met, Burger et al (37) estimated the adult cat protein requirement for maintenance to be about 12% of the diet; K. Smalley, Q. R. Rogers and J. G. Morris (unpublished results; see also Ref. 208) have estimated the protein requirement

of growing kittens to be about 18–20% of the diet whether the increments of nitrogen in the diet were provided by free amino acids or by casein.

All purified diets used recently for determining protein and amino acid requirements of the cat have had about 4.7 kcal/g of diet. Using the above results and the standard assumption that protein and amino acid requirements vary directly with the energy content of the diet, we find that adult cats would require about 10% of dietary energy as protein and growing kittens would require about 17% of dietary energy as protein. Because cats accept high-fat diets better than low-fat diets (especially with purified dry diets), the requirements for protein and amino acids have not yet been determined with low-fat diets. If comparisons are made of the nitrogen requirements (listed as the protein requirements in Table 1) of several domestic mammalian species, it is apparent that they fall into two groups: carnivores and omnivores. Herbivores would be part of the group of omnivores with respect to protein requirements.

Nutritionally the dog is a member of the group of omnivores even though it is a member of the order Carnivora. The cat and other true carnivores have substantially higher protein requirements than do omnivores. This higher requirement is primarily an increased requirement for maintenance rather than for growth. For example, the rat requires three times as much protein for growth as for maintenance, but the cat requires only 50% more. If one examines the increased increment for growth as an absolute increase in protein need above maintenance, the amount is quite similar for cats and rats. The metabolic reason for the high protein requirement of the cat is the high activity of the nitrogen catabolic enzymes in cat liver (209). These enzymes are nonadaptive, so the obligatory nitrogen loss is high even when cats are fed low-protein diets (207).

**Table 1** Protein requirement (% of diet) for adult and growing animals of several mammalian species<sup>a</sup>

Species	Young	Adult	Reference
Dog	12	4	14, 90, 124, 183
Rat	12	4.2	178
Man	8	5	176
Cat	29	19	68, 92, 93, 155, 177
Cat <sup>b</sup>	17	10	9, 37; K. Smalley, Q. R. Rogers & J. G. Morris, unpublished results
Mink	31	20	175
Fox	24	16	175

<sup>a</sup>Ideal protein, i.e. meeting all the known essential amino acid requirements. This may not be true for the data for the mink and fox.

<sup>b</sup>Values expressed as percentage of dietary energy.

## Amino Acid Requirements

**ARGININE** A summary of the amino acid requirements of the growing kitten is presented in Table 2. The arginine requirement is listed as 1.05% of the diet, the dietary level of arginine required to prevent an increase in orotic aciduria, which occurs when an arginine-deficient diet is fed (56).

Arginine deficiency causes the most dramatic response to any nutrient deficiency in the cat. The feeding of a single meal of an arginine-devoid diet may result in hyperammonemia in less than one hour and causes such severe hyperammonemia within two to five hours that the cats exhibit severe symptoms of ammonia intoxication, which include lethargy, emesis, vocalization, hypersalivation, hyperactivity, hyperesthesia, ataxia, emprosthotonos, extended limbs, and exposed claws. The most severely affected cats show

**Table 2** Current estimate of amino acid requirements of growing kittens<sup>a</sup>

Amino acid	Requirement	Reference
Arginine	1.05 <sup>b</sup>	7, 56
Histidine	0.30 <sup>c</sup>	10; D. Quam, J. G. Morris, Q. R. Rogers, unpublished results
Isoleucine	0.50	11; D. M. Hargrove, Q. R. Rogers, J. G. Morris, submitted for publication
Leucine	1.20	11; D. M. Hargrove, Q. R. Rogers, J. G. Morris, submitted
Lysine	0.80	7; J. A. O'Donnell, Q. R. Rogers, J. G. Morris, unpublished results
Methionine (with excess cystine)	0.40	237, 245
Methionine + cystine	0.75	217, 245
Phenylalanine (with excess tyrosine)	0.40	10; J. Williams, Q. R. Rogers, J. G. Morris, unpublished results
Phenylalanine + tyrosine	0.90	10; J. Williams, Q. R. Rogers, J. G. Morris, unpublished results
Threonine	0.70	11, 206, 247
Tryptophan	0.11	10, 99
Valine	0.60 <sup>d</sup>	11, 98

<sup>a</sup>Based on experimental diets containing about 4.7 kcal/g diet. No work has been done to determine whether it would be valid to express amino acid requirements of kittens as a percentage of a diet based on a standardized 4.0 kcal/g diet. Unless otherwise stated, each requirement is based on the minimal quantity of each amino acid required in the diet to give maximum weight gain and maximum nitrogen retention.

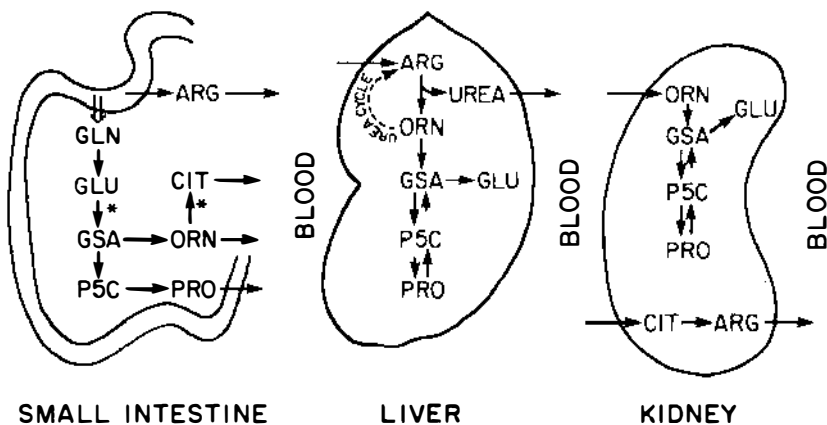
<sup>b</sup>Based on minimizing orotic acid excretion in the urine. Maximum weight gain is sustained by 0.8–0.9% arginine in the diet.

<sup>c</sup>Based on the dietary level of histidine required for the prevention of cataracts after several months. Histidine at 0.25% of the diet is sufficient to maximize nitrogen retention.

<sup>d</sup>The minimal requirement could be 0.4 or 0.5% of the diet; increments between 0.3 and 0.6% dietary valine have not been tested.

hypothermia and bradypnea cyanosis, which are followed by death in a low percentage of cats (162, 163, 166, 207). This raises the question of why the cat is so much more sensitive to arginine deficiency than are other mammals, such as rat or man. Animals are more sensitive to ammonia load when they are in the postabsorptive state (254), and the administration of arginine greatly decreases the ammonia intoxication under these conditions (95). The generally accepted explanation for the prevention of hyperammonemia by arginine is that it acts anaplerotically to stimulate urea synthesis by providing an intermediate of the urea cycle, as the intermediates are all depleted during the postabsorptive state. The reason for the high sensitivity of the cat to arginine deficiency is related to the metabolism of the urea cycle intermediates. A summary of the differences between the cat and the rat is shown in Figure 2.

There appear to be at least two reasons for the differences between the responses of the cat and those of the rat: (a) The cat apparently is less able to synthesize ornithine and (b) the cat may be less able to provide citrulline to the kidney for conversion to arginine. Several lines of evidence point to impaired ornithine synthesis in the cat. Dietary ornithine completely prevents the hyperammonemia of arginine deficiency in cats, although the animals still lose approximately the same amount of weight as they would if any other essential amino acid were omitted from the diet (163, 166, 206). Rats, in contrast, will grow without any dietary arginine, although not maximally; yet the addition of ornithine to an arginine-free diet does not improve weight gain in rats (156) or in cats. Stewart et al (242) measured ornithine, citrulline, and arginine in the livers of cats fed an arginine-free diet. They confirmed that the cats



**Figure 2** Metabolism of urea-cycle intermediates in the rat and cat. Enzymes that have been shown or are presumed to be missing in the cat are marked with an asterisk (\*). GLN= glutamine, GLU= glutamic acid, GSA= glutamic semialdehyde, P5C= pyrroline-5-carboxylate, PRO= proline, ORN= ornithine, CIT= citrulline, ARG= arginine.



showing severe hyperammonemia were the ones that were most severely depleted of liver ornithine. The concentrations of ornithine in liver were lower in cats than in rats fed arginine-free meals. However, the authors found no difference between the cat and the rat in terms of the activity or kinetic constants of ornithine aminotransferase (OAT) or in terms of N-acetylglutamate concentrations of liver mitochondria.

The *de novo* synthesis of ornithine in rats appears to occur primarily in the intestine. By measuring arterio-venous (A-V) differences across the perfused rat intestine, Windmueller & Spaeth (259, 260, 261) showed that glutamine and glutamic acid are converted to ornithine, citrulline, and proline. Herzfeld & Raper (109) showed that OAT isolated from rat intestine could be made to act in the ornithine-forming direction; OAT from rat liver could not. Furthermore, the intestine has the enzyme glutamic acid reductase, which forms glutamic acid semialdehyde that can be converted to ornithine and proline (108, 212). These authors did not find glutamate reductase in any other rat tissues examined, including the liver (108, 212). In contrast, the cat may lack glutamic acid reductase in both the intestine and the liver. Costello et al (55) have not been able to detect this enzyme in cat intestine or liver, thus verifying that the cat cannot make ornithine from glutamate (see Ref. 164).

Citrulline can completely replace arginine in the diet of the cat (166) as in the rat, but a higher dietary concentration of citrulline is needed for an equivalent growth rate (C. Johannsen, Q. R. Rogers, J. G. Morris, unpublished results). In the rat, citrulline synthesis occurs primarily in the intestine (261); citrulline released from the intestine reaches the kidney where it is converted to arginine for use by other tissues (76, 203). Since citrulline is made from ornithine, a lack of glutamic acid reductase in cat intestine would preclude citrulline synthesis from glutamate. The cat may have additional metabolic defects, such as a lack of ornithine transcarbamylase in the intestine (see Figure 2). These metabolic defects would also explain why the level of citrulline in cat plasma is normally quite low (207).

Proline is dispensable for the cat (206), as it is for the rat. In neither the rat (95) nor the cat (C. A. Buffington, Q. R. Rogers, J. G. Morris, unpublished results) does proline ameliorate hyperammonemia. Although it might be expected that proline would be converted to ornithine in the liver or kidney, the kinetic characteristics of OAT in these tissues and the substantial activity of pyrroline-5-carboxylate dehydrogenase would prevent the synthesis of ornithine and favor the conversion of pyrroline-5-carboxylate to glutamate if proline and glutamate were present at physiological levels. The difference between the cat and the rat is that proline spares at least one half of the dietary arginine requirement of the rat (204, 205); it does not spare the arginine requirement of the cat. It seems likely that in the cat, OAT is a constitutive enzyme like the urea-cycle enzymes (209), so ornithine degradation would be

obligatory in the cat and thus would provide a precursor for proline synthesis whether proline were in the diet or not. In contrast, OAT is adaptive in the rat, and ornithine catabolism is reduced when rats are fed a low-protein diet compared to when they are fed a high protein diet (220).

What is the physiological meaning of the differences in arginine metabolism in the cat? From a teleological standpoint, it is attractive to postulate that as a strict carnivore, the cat has never had a diet limiting in protein and therefore in arginine or ornithine but has always had an excess of amino acids, much of which is needed for gluconeogenesis because animal tissues are low in carbohydrate. Thus, the metabolic need is not to make ornithine when nitrogen intake is high, because arginine from dietary protein would provide the ornithine needed anaplerotically in the urea cycle as well as providing arginine for protein synthesis; rather, the need is to deplete ornithine during the post-absorptive state so that urea synthesis can be markedly reduced to prevent excessive catabolism of amino acids to urea. This phenomenon also occurs in rats, but to a lesser extent. Because the rat is an omnivore, there are times when it ingests a low-protein diet and adapts by conserving nitrogen, in part via metabolic adaptations (220); these adaptations, i.e. changes in enzyme activities as a result of dietary compositional changes, appear to be much more limited in the cat (209). Work done with the mink (144) and the ferret (66) indicates that the cat's arginine metabolism is not unique but is representative of that of strict carnivores.

**HISTIDINE, LEUCINE, ISOLEUCINE, VALINE, LYSINE, PHENYLALANINE & TRYOSINE** Nothing unique has been reported about histidine deficiency in the cat; the usual depletion of hemoglobin has been found, and after long-term histidine deficiency, cataracts have been found (D. Quam, J. G. Morris, Q. R. Rogers, unpublished results). Isoleucine and valine requirements are similar in the cat and rat, but the leucine requirement is somewhat higher for the cat (D. M. Hargrove, Q. R. Rogers, J. G. Morris, submitted for publication). These workers (unpublished results) recently found that little or no branched-chain amino acid antagonism occurs in the kitten. That is, 10% of any one of the three branched chain amino acids did not decrease the growth rate of kittens fed a low-protein diet that was somewhat limiting in each of the branched-chain amino acids.

The cat and the rat have similar lysine requirements, but as a percentage of the protein, the requirement for lysine is considerably less for the cat than the rat because the cat has a higher protein requirement. Lysine is limiting only when a cereal protein such as wheat gluten is the major dietary protein (122, 207).

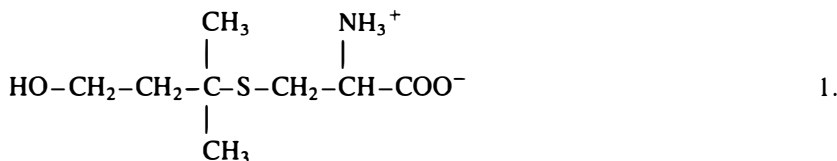
The total phenylalanine-plus-tyrosine requirement of the kitten is about 0.8–0.9% of the diet. Approximately 60% of that requirement can be met with tyrosine (Table 2), which has been shown to be a dispensable amino acid (206).

The tryptophan requirement of the cat is quantitatively similar to those of several other species (99). However, there is negligible flow of tryptophan through the quinolinic acid pathway, and the cat therefore does not synthesize a significant quantity of niacin (see section on Water-Soluble Vitamins).

**SULPHUR AMINO ACIDS** There are several unique aspects of sulphur amino acid nutrition and metabolism in the cat. First, the dietary requirement is considerably higher for the cat than for most other mammals (Table 2). Second, the cat excretes a unique, branched-chain sulphur amino acid (felinine) in the urine. Third, the cat requires a dietary supply of taurine, a  $\beta$ -amino sulphonic acid, to prevent central retinal degeneration (CRD).

The reason for the cat's high requirement for sulphur amino acids is not readily apparent, but it has been suggested that it is related to the cat's thick hair and its need to synthesize felinine. This high sulphur amino acid requirement results in these amino acids being the most limiting amino acids in most cat diets. About 19% animal protein is needed in the diet to meet the sulphur amino acid requirement of the growing kitten. This requirement would just barely be met in a meat diet containing enough protein to meet the nitrogen requirement (208). There is not an inordinate quantity of methionine converted to cysteine. Cystine spares about one half of the methionine requirement in the cat, as in other species (237, 245). Therefore, part of the higher sulphur amino acid requirement appears to be for cystine and part of it for methionine per se. Cysteine is used for felinine synthesis and for the synthesis of hair, and this may account for part of the increased requirement. Since the cat normally eats a high-fat diet, perhaps the increased need for phospholipids for absorption and transport of fat has resulted in an increase in the need for S-adenosyl methionine for methylation reactions. A high constitutive level of methionine transaminase would also result in an increased need for dietary methionine. An examination of the activities of the enzymes in the various pathways should help clarify the reason for this anomaly.

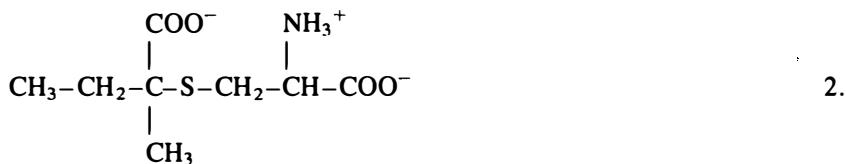
*Felinine* The structure of felinine is shown below:



This branched-chain sulphur amino acid was first reported by Datta & Harris in 1951 (62) and was isolated and characterized by Westall et al in 1953 (255). Because felinine is not available commercially, very little work has been done on the effect of diet on the biosynthesis and excretion of felinine. Roberts

(201), however, examined several aspects of feline metabolism and reported that feline levels were highest in adult male cat urine, quite low in female cat urine, and undetectable in kittens of either sex. He found an approximately fourfold variation in feline excretion among a group of 19 male cats. He also showed that giving an adult female cat testosterone for one week resulted in an increase in feline excretion to normal adult male levels and that the administration of estradiol to an adult male cat for two weeks had no effect on feline excretion. Roberts (201) and Avizonis & Wriston (17) both used isotopic incorporation methods *in vivo* and found that leucine was a better precursor for feline biosynthesis than was mevalonic acid. Using similar techniques, Roberts failed to get any  $^{35}\text{S}$  incorporated in feline from either  $^{35}\text{S}$ methionine or  $^{35}\text{S}$ cysteine. The site of feline synthesis is uncertain because Roberts (201) found some feline in both liver and kidney and a smaller amount in both skin and blood. Wang (251) also attempted to determine the source and precursors of feline. He too was largely unsuccessful in showing feline biosynthesis *in vitro*, but he did find  $[2-^{14}\text{C}]$ acetate incorporation into feline *in vivo*.

The metabolic role of feline is a matter for speculation. It may be a urinary component for territorial marking or perhaps is somehow involved in the regulation of sterol metabolism (230). It is interesting to note the similarity of feline (Equation 1, above) and isovalthine:



Isovalthine has been found in cat urine (83) and in the urine of hypercholesterolemic patients (181). Little work has been done the past 20 years in this interesting area of feline synthesis and function.

The total quantity of dietary sulphur amino acids required to provide for the synthesis of feline that is excreted in the urine of a male cat should not exceed 0.05% of the diet (50–75 mg). Therefore, regardless of the interesting aspects that are still to be learned about feline synthesis and function, it is doubtful that it alone is the reason for the high sulphur amino acid requirement of the cat, especially the kitten.

**Taurine** Taurine deficiency in the cat has been shown to cause central retinal degeneration (104, 105). Taurine is a  $\beta$ -amino sulfonic acid (2-amino ethanesulfonic acid) and as such is not present in protein. It is normally made from cysteine in the liver and some other tissues, e.g., brain of mammals. Except

for its conjugation to bile acids (100, 250) and its presence in a few peptides such as the hormone glutaric acid (gamma-L-glutamyl-*L*-taurine) (77, 84), taurine is essentially metabolically inert, i.e. taurine from either endogenous synthesis or from dietary intake is not further metabolized and is eventually excreted as such in the urine. CRD had been reported in cats a number of times (28, 29, 169, 215, 216, 227) before Hayes and coworkers (104, 105) discovered the nutritional cause of the disease. The discovery of taurine deficiency as the cause of CRD led Hayes and coworkers (106, 243) to examine its synthesis and metabolism in the cat. The effects of feline CRD and the function of taurine in general have been reviewed several times (20, 103, 106, 243), but there are no published experiments that clearly define the taurine requirement. Several laboratories have reproduced CRD under laboratory conditions (174), so it is clear that the cat needs some taurine in the diet, but the time required to produce CRD varies from 23 to 52 weeks or longer, and the incidence of CRD in cats fed taurine-free diets varies from 12 to 100% (8, 21, 104).

Part of the uncertainty about the requirement is caused by unknown dietary or environmental factors that affect the requirement. For example, CRD has been found in kittens fed commercial cat foods that contained between 200 and 300 mg of taurine/kg dry weight of diet (174), but some laboratories have been unable to produce lesions in weanling kittens after feeding purified diets containing no taurine for one year. Q. R. Rogers and J. G. Morris (unpublished results) examined the importance of type of dietary fat, level of dietary protein, presence of dietary fiber, and level of dietary sulphur amino acids on the incidence and severity of CRD in cats. After examining all these factors they have concluded that only the level of sulphur amino acids affects the outcome of CRD. When weanling kittens were fed a diet with no taurine and with a level of sulphur amino acids near the requirement (8; Q. R. Rogers, and J. G. Morris, unpublished results), all the kittens developed CRD within 5–6 months; when twice the sulphur amino acid requirement was fed, no CRD was found in 12 months. Therefore, it appears that the reason that kittens fed commercial cat food containing 200–300 mg of taurine/kg diet have developed CRD is that the total available sulphur amino acids were near the requirement, although even when kittens are fed a high level of sulphur amino acids with no dietary taurine, a low percentage of kittens develop CRD. Thus, the recommendation of the National Research Council committee (174) that the taurine requirement (when the sulphur amino acid requirement is just met) is between 250 mg and 1000 mg/kg of dry diet still appears valid. An interpolation of existing data might put the minimal requirement at approximately 350 mg/kg diet. A more accurate estimate of the requirement may not be available for some time, because studies of the lower levels probably should be extended for three to five years before they can be considered safe.

**THREONINE** Although Anderson et al (11) selected the threonine requirement at 0.8% of the diet, a close examination of their results indicates that kittens grew just as well on 0.7% threonine. Further, Titchenal et al (247) and Rogers & Morris (206) found maximal weight gain with threonine at 0.7% of the diet and somewhat less than maximal growth at a dietary level of 0.6% threonine, so the minimal threonine requirement appears to be about 0.7% of the diet. Kittens fed inadequate threonine developed neurological problems as early as the fifth day, even though the kittens were growing (247). Signs included tremor, ataxia, incoordination, dysequilibrium, and defective righting reflex. Neurological signs were progressive but completely reversible. All signs indicated cerebellar dysfunction. In addition to the neurological signs, several kittens developed thoracic and pelvic limb lameness associated with carpal deviation and stiff movement of the hind limbs. These kittens seemed reluctant to walk or run, but no visible pain or discomfort could be induced by vigorous palpation of the affected limbs and joints. The problems were completely reversed by feeding an excess of threonine.

### *Amino Acid Intolerance*

Little has been done to examine the possible effects of high dietary levels of individual amino acids on cats. It is known that cats are less tolerant to an excess of glutamic acid than are rats, chickens, and most other species (64). Although the kitten appears to be able to tolerate 6% glutamic acid in the diet, 9% or 12% free glutamic acid results in increases in free glutamate in plasma to three to ten times normal levels. These high levels of glutamic acid result in sporadic emesis and eventually cause classical thiamin deficiency in kittens fed 4.4 mg of thiamin/kg diet, a level which is normally adequate (177). The nature of the interaction between these two nutrients has not been completely clarified, but it appears that high dietary glutamic acid somewhat increases the fecal excretion of dietary thiamin (65). In relation to this, it is worth noting that most animal proteins contain only about half the content of glutamic acid as that in many plant proteins.

Fau et al (75) recently examined the effect of excess dietary methionine in weanling kittens. They found a growth depression in kittens fed 2% additional methionine. With 3% methionine, some cats completely rejected the diet. This is in contrast to recent experiments in which feeding 10% of the diet as leucine, isoleucine, or valine had no adverse effect (D. M. Hargrove, Q. R. Rogers, J. G. Morris, unpublished results). Thus, the cat appears to be less tolerant than some other mammals to excesses of some amino acids, such as glutamic acid and methionine, but more tolerant to excesses of other amino acids such as the branched-chain amino acids.

## MINERALS

There is a paucity of data on the cat's qualitative and minimal quantitative requirements for minerals. Successful growth and reproduction have been achieved with purified diets containing a mineral mixture similar to that required by growing rats and pigs (e.g. 150, 152). The cat's quantitative requirements for essential minerals therefore do not greatly exceed those of these species, and the cat does not have a requirement for any mineral that is not essential for these species. However, these findings do not preclude the possibilities of peculiarities in the transport, metabolism, or storage of minerals by cats and of a lower quantitative requirement for a particular mineral.

When cats consume the whole bodies of their prey, they obtain all necessary dietary minerals, but when the soft tissues are consumed without the skeleton, the diet is deficient in calcium. A report of skeletal abnormalities in large felids in the London Zoo was made by a surgeon of Middlesex Hospital as early as 1888 (31). He reported that "when cubs which early manifest signs of rickets were promptly fed on bone dust and cod liver oil they made good recoveries." Presumably these lions were given an all-meat diet without bones. This observation is of particular interest as it predates the demonstration by Sir Edward Mellanby in 1919 that rickets could be prevented by cod liver oil. It took another 70 years before research and clinical reports appeared describing the effects of all-meat diets on the induction of the disease now referred to as nutritional secondary hyperparathyroidism (NSHP) in both wild captive and domestic carnivores (46, 78, 107, 139, 192, 200, 226).

The metabolism of calcium and phosphorus in the cat does not appear to differ markedly from that in other mammals. The frequency of occurrence of NSHP in cats appears to be the result of the combination of the low calcium content of animal soft tissues, which are highly palatable to the cat, and the fact that owners frequently feed animal tissues as the sole diet.

The quantitative requirements of the cat for magnesium have not been defined, but excessive dietary intakes of magnesium have been shown to predispose male cats to the formation of urinary calculi composed of struvite (magnesium ammonium phosphate), which cause subsequent urethral obstruction. Thus it has been suggested that high intakes of magnesium are an etiological factor in producing FUS (70, 127).

The potassium requirement of growing kittens has been shown to be influenced by the protein level of the diet (111). The estimated potassium requirement of kittens consuming a purified diet based on 33% soy protein was 0.3%, and for 68% soy protein, 0.5%. Potassium deficiency was evidenced by hypokalemia, anorexia, retarded growth, emaciation, lethargy, locomotive disorders, and unkempt fur.

Cats undoubtedly require dietary sources of sodium and chlorine, but quantitative requirements of these elements have not been experimentally defined. Salt (sodium chloride) has been used to increase water consumption and hence urinary output to aid in preventing FUS (13, 36, 97).

The quantitative dietary requirements of the cat for iron have not been studied, although Spray & Widdowson (239) measured the amount of iron in the cat at various stages of development. The requirement for copper appears to be approximately 5 mg/kg diet (69), which is similar to that of other mammals. Copper deficiency induced in post-weanling kittens resulted in a decrease in the concentration of copper and Cu-Zn-superoxide dismutase (SOD) in plasma. No changes were observed in Mn-SOD, packed cell volume, or coat color or condition in these copper-deficient kittens. However, when the deficiency was induced by giving pregnant queens a deficient diet (less than 1 mg copper/kg diet), histochemical changes in the aortas of neonatal kittens were observed that indicated connective tissue defects. The latter changes occurred without depressions in tissue copper levels. This is in contrast to observations of weanling kittens given the copper-deficient diet; changes in tissue copper levels were apparent in this group. It also appears that the hematopoietic system of the adult cat, in contrast to those of other mammalian species, is resistant to changes induced by a low copper status.

There have been two reports (4, 130) of experimentally induced zinc deficiency in the cat. The clinical signs of the deficiency are similar to those described in other mammals (249); however, while the dermal lesions appear to be less severe in cats than in species like the pig, a mild deficiency of zinc, which caused no apparent growth retardation and allowed reproduction in a female, caused defective spermatogenesis in male cats (130). There is no information on the availability to cats of zinc in natural products or in commercial cat foods, but some studies suggest that it may have low availability (4; Q. R. Rogers, J. G. Morris, unpublished results).

Iodine deficiency has been reported in zoo felines (78, 187) and in domestic cats (94). Clinical signs include hypertrophy of the thyroid, alopecia, abnormal calcium metabolism, and fetal resorption. An interaction has been reported between calcium metabolism and iodine (200, 222, 226). Iodine supplementation reduced clinical signs of the deficiency in cats given all-meat or low-calcium diets. It has been stated that euthyroid cats can tolerate relatively high doses of iodine (223), but over a period of 42 months, one clinic has found hyperthyroidism in 131 cats between 6 and 20 years of age (184). Clinical signs of hyperthyroidism included polyphagia, increased activity, polydipsia, polyuria, vomiting, and increased base circulating levels of thyroxine and triiodothyronine. It is not known whether the concentration of iodine in commercial cat foods is an etiological factor in producing this disease.



There are no published studies on the requirements of the cat for manganese, selenium, chromium, nickel, silicon, vanadium, molybdenum, fluorine, or tin. These elements have been shown to be essential in other species (178).

## VITAMINS

### *Fat-Soluble Vitamins*

**VITAMIN A** The most unusual aspect of fat-soluble vitamin nutrition in cats is that preformed vitamin A is a dietary essential. The conversion of carotenes to retinol, which occurs in the intestinal mucosa in other species including the dog (91, 180), is negligible in the cat (3, 86). As a result, neither dietary nor intravenously administered  $\beta$ -carotene can prevent the development of vitamin A deficiency (86). Because preformed vitamin A is not present in plant lipids, the cat requires a source of animal fat (in particular, organ meats) to satisfy its vitamin A requirement. Coombes has reported that the fox, another carnivore, may also be unable to utilize carotenoids as a source of vitamin A (52).

There is some information regarding tissue levels of vitamin A in cats. The kidneys of cats contain approximately ten times as much vitamin A as do the kidneys of other animals (148, 158); however, liver stores of vitamin A are more closely related to dietary status (158, 224). The high levels of vitamin A in cat kidney are not found in other Felidae (110).

The quantitative requirement of the cat for vitamin A has not been precisely defined. Scott & Scott (228) concluded that approximately 2000 IU/day were necessary for normal reproduction. However, other workers (J. G. Morris, Q. R. Rogers, unpublished results) found that 6000 IU/kg diet met the requirement for normal reproduction in female cats. Vitamin A toxicity occurs frequently in cats fed diets consisting largely of raw liver (229) and can be induced in kittens given supplements of 100,000 IU daily for four weeks (44).

**VITAMIN E** Vitamin E deficiency in cats was first observed by Cordy & Stillinger (54) in kittens fed a canned, fish-based diet. Deficient cats develop steatitis, in which the depot fat becomes firm and yellow-orange in color. This painful deposition of ceroid pigment is irreversible and leads to death if not treated. Cordy (53) showed that the deficiency could be prevented by 20 or 40 mg/day of D,L- $\alpha$ -tocopherol. Almost all naturally occurring cases of vitamin E deficiency have been in cats fed diets high in fish (especially red tuna), which contains large amounts of highly unsaturated fatty acids (47, 173). Today, all cat foods composed of red tuna meat are fortified with vitamin E.

**VITAMIN D** Although vitamin D is essential for preventing rickets in kittens (89), the requirement is probably quite low. When kittens were fed vitamin

D-deficient diets that contained 1% calcium and 1% phosphorous, only one cat out of seven was still alive after 21 months (89). When the diet contained 2% calcium and 0.65% phosphorous, six out of nine cats survived. Rickets developed in vitamin D-deficient cats after 4–5 months, but in those cats that survived the first 12 months of the study, there were spontaneous remissions. In these studies, cholecalciferol (250 IU twice a week) prevented rickets. Scott (224) reported that clinical cases of rickets occur rarely and only in kittens kept in the dark. Rivers et al (197) reported that levels of vitamin D<sub>3</sub> in plasma of vitamin D-deficient kittens shielded from ultraviolet light declined initially but then tended to increase with age. They reported no signs of bone disease after 9 months. Excess vitamin D<sub>3</sub> (2.5 mg/kg diet) caused calcification of soft tissues (197), which indicates that large doses of vitamin D are toxic for cats, as they are for other mammals.

**VITAMIN K** The essentiality of vitamin K for cats has not been established. Reber & Malhotra (188) observed that an irradiated beef diet, which caused hemorrhage in weanling rats, did not have adverse effects on cats. The level of vitamin K was calculated to be 60 µg/kg of solids.

### *Water-Soluble Vitamins*

In omnivores, the requirement for niacin can be met by dietary tryptophan. The pathway of tryptophan metabolism in liver has a branch point at  $\alpha$ -amino- $\beta$ -carboxymuconic- $\epsilon$ -semialdehyde, and this intermediate can be converted either to quinolinic acid, which is then metabolized to niacin, or to picolinic acid. In most mammals, the activity of picolinate carboxylase is quite high, so most tryptophan is diverted away from the niacin-synthesizing pathway. The activity of picolinate carboxylase in cats is 30–50 times higher than in rats, so there is almost no synthesis of niacin (120, 244). Cats or kittens fed a diet lacking niacin develop signs of deficiency that cannot be reversed by the administration of additional tryptophan (61). DaSilva et al (61) found that when tryptophan was administered orally or subcutaneously in doses of up to 600 mg/day, there was no significant increase in urinary N-methylnicotinamide excretion. Leklem et al (143) gave higher doses of tryptophan (1 g/kg body weight/day) or kynurenine (0.25 g/kg body weight/day) and found a slight increase in N-methylnicotinamide excretion, but the increase was only approximately 3% of that seen in rats. These authors also showed that less than 1% of a dose of L-[<sup>14</sup>C]tryptophan could be recovered as niacin metabolites in cat urine (142). There appear to be other peculiarities in tryptophan metabolism in cats, in that 80% of the radioactivity in urine of cats given labelled tryptophan could not be identified as any of the known tryptophan metabolites.

The efficiency of conversion of tryptophan to niacin varies widely among species: it is greatest in rats, less so in humans, and negligible in cats. Poston

& Combs (186) reported that picolinate carboxylase activity is even higher in trout and salmon than in cats. Despite the fact that cats do not convert tryptophan to niacin, their requirement for niacin does not appear to exceed that of other species; however, the cat depends on animal tissues as a source of niacin, because nicotinamides are present in lower quantities in plant tissues.

The cat requires thiamin (73), riboflavin (141), pantothenic acid (88), pyridoxine (87), biotin (39), folacin (60), and vitamin B<sub>12</sub> (135). Ascorbic acid is synthesized in adequate amounts and is not a dietary essential (59). Vitamin B<sub>12</sub> is low in diets of vegetable origin, and an all-vegetable diet would necessitate supplementation with this vitamin (135). Deficiencies of some vitamins occur with relative frequency: thiamin deficiency is easily produced by feeding fish products (especially carp and herring) in which the thiaminase has not been destroyed (238).

Choline is required by kittens when the level of dietary methionine is just enough to meet the methionine requirement (218). There is evidence that the choline requirement of the kitten, as of other species, can be met in part by excess dietary methionine (12), but it is not known whether dietary choline is completely dispensable. There are reports that inositol is required by cats (224), but this has not been investigated thoroughly.

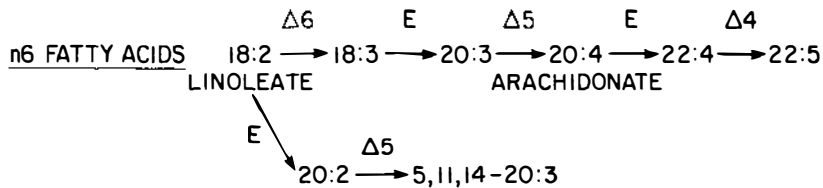
## OTHER FOOD CONSTITUENTS

Benzoic acid, a food preservative, is extremely toxic to cats. Bedford & Clarke (26) showed that doses of benzoic acid in excess of 0.45 g/kg body weight led to hyperesthesia, muscular tremors, depression, and often, death. In many species, benzoic acid is detoxified with glycine to form hippuric acid and with glucuronic acid (258). Cats are able to form glycine conjugates but they have low levels of glucuronyl transferase in liver (71). This may also explain the toxicity and slow metabolism of many drugs by cats (33, 253). Cats excrete unusual conjugates in which quinoline-2-carboxylic acid, a typical aromatic acid, is combined with glycyltaurine or glycylglycine (125, 126). The significance of these reactions is not known.

## ESSENTIAL FATTY ACIDS

### *Metabolism*

**LACK OF Δ6 DESATURATION** The essential fatty acid (EFA) requirements of most mammals can be met entirely by linoleate. In the liver, linoleate is converted to arachidonate by alternating desaturation and elongation (Figure 3). The first indication that this scheme might be different in cats came in 1975, when Rivers, Sinclair & Crawford (199) reported that cats fed purified diets containing linoleate but lacking arachidonate had extremely low levels of



**Figure 3** Pathways of linoleate metabolism in mammals. In the liver of the cat there is almost no  $\Delta 6$ -desaturase activity, so arachidonate is a dietary essential. E= elongation,  $\Delta$ = desaturation.

arachidonate in plasma lipids. The authors suggested that cats were unable to synthesize arachidonate from linoleate. They also predicted that the cat would require arachidonate in the diet.

Hassam et al (101) showed that the enzyme defect was at the  $\Delta 6$ -desaturase, the first step in the conversion. In cats given injections of [ $1-^{14}\text{C}$ ]linoleate, very little radioactivity was incorporated into arachidonate. Sinclair et al (231) confirmed these results and showed that less than 0.5% of the radioactivity from [ $^{14}\text{C}$ ]linoleate recovered from liver lipids was associated with fatty acids having four double bonds.

Most of the information about EFA metabolism in cats has been obtained by studying the effects of different diets on the fatty acid compositions of blood and tissue lipids. When cats are fed purified diets containing safflower seed oil as the only source of fat, the level of arachidonate in blood lipids decreases with time (80). At the same time, linoleate comprises nearly 50% of the fatty acids in plasma lipids (150, 199, 231).

In cats fed a diet low in linoleate, the level of linoleate in plasma and erythrocytes decreases with time and levels of n7 and n9 fatty acids (16:1n7, 18:1n9, 20:1n9, and 20:3n9) increase (150). These fatty acids are synthesized de novo in liver, and an increase in their concentration is a sign of EFA deficiency (114). Although the amount of 20:3n9 synthesized in linoleate-deficient cats is very small compared to rats, for example, the synthesis of 20:3n9 from 18:1n9 suggested that the cat might be able to synthesize some arachidonate from linoleate, because the same enzymes are responsible for both conversions. Therefore, Sinclair et al (232) suggested that dietary linoleate could provide arachidonate in cats, whereas Rivers & Frankel (196) concluded that the amount of arachidonate that could be synthesized would be insignificant.

If linoleate were converted to arachidonate, the inclusion of a source of linoleate in an otherwise EFA-deficient diet would result in increased levels of arachidonate in cat tissues. However, examination of the fatty acid compositions of plasma, erythrocyte and liver lipids of cats fed diets with or without safflower seed oil showed that there was no net increase in arachidonate when safflower seed oil was included in the diet (150). Thus, it appears that the

activity of the  $\Delta 6$ -desaturase is so low as to be nearly saturated with substrate even in linoleate deficiency, so there is negligible synthesis of arachidonate in cat liver.

This conversion, however, may occur in cat testes. Levels of arachidonate and longer-chain n-6 fatty acids are higher in testes lipids of cats fed a diet containing linoleate than in those fed a linoleate-deficient diet (152). However, the arachidonate in testes is apparently not released into the bloodstream.

**$\Delta 5$ -DESATURATION** Sinclair and coworkers (231) showed that a  $\Delta 5$ -desaturase is present in cat liver. They injected cats with 20:3n6 labelled with  $^{14}\text{C}$  in the 2-position and found radioactivity in liver arachidonate. This indicated that 20:3n6 could be converted to arachidonate.

The presence of a  $\Delta 5$ -desaturase results in the appearance of 5,11,14-20:3 in the tissues of cats fed diets containing linoleate (231; see Figure 3). Levels of 20:2n6 and 5,11,14-20:3 in blood increase as the level of linoleate increases (149, 150). Although other mammals can also synthesize 5,11,14-20:3 (248), this fatty acid usually is not detectable in animal tissues. In human erythrocyte lipids, the level is only 0.1% of total fatty acids (M. L. MacDonald, unpublished results); in cat erythrocyte lipids, the level is almost 3% (150). The synthesis of relatively high amounts of 5,11,14-20:3 by cats probably results from the extremely low activity of the  $\Delta 6$ -desaturase, which allows linoleate to accumulate and to be shunted toward 5,11,14-20:3.

Sinclair (231, 232) suggested that the cat might be able to synthesize arachidonate by  $\Delta 8$ -desaturation of 20:2n6 (11, 14-20:2) to form 20:3n6 (8, 11, 14-20:3), but since there is negligible synthesis of arachidonate by any route, both the  $\Delta 8$ - and  $\Delta 6$ -desaturases apparently have extremely low activities in cat liver.

The presence of a functional elongase and  $\Delta 5$ -desaturase (231) means that 18:3n6 should be a useful precursor of arachidonate. Frankel & Rivers (81) used evening primrose oil, which contains 18:3n6, to supplement cats that had previously been fed a diet containing 25% safflower seed oil as the only source of fat. After five days, there were increases in the levels of 18:3n6 and 20:3n6 in plasma phosphatidylcholine, but levels of 20:4n6 were unchanged. At that time, the authors concluded that cats lack a  $\Delta 5$ -desaturase and were unable to convert 20:3n6 to 20:4n6. However, later studies by Frankel (80) showed that the level of arachidonate in plasma lipids increased only after evening primrose oil was fed for 10 weeks or more. Sinclair et al (231) showed that when the methyl ester of 18:3n6 was fed to cats, the levels of 20:3n6 and 20:4n6 in erythrocyte lipids increased, which is consistent with the  $\Delta 5$ -desaturation of 20:3n6.

*EFA Deficiency in Cats*

**ARACHIDONATE DEFICIENCY** Rivers and coworkers (193, 194, 195, 199) reported signs of EFA deficiency in cats fed a diet high in linoleate but lacking in arachidonate. The deficiency signs included poor growth, "staring" fur, skin lesions around the mouth and on the hocks, slow wound healing, fatty liver, increased susceptibility to infections, poor reproductive performance, underdeveloped testes, and cleft palates and other defects in kittens born to deficient dams. Later, Frankel (80) showed that growth was not abnormal as long as respiratory infections were prevented.

These early studies were done without the benefit of more recent knowledge of the nutritional requirements of cats. The diets used by Rivers et al (199) lacked taurine and  $\alpha$ -tocopherol. Stephan & Hayes (240) suggested that the deficiency signs could have resulted from vitamin E deficiency. The deficiencies of taurine and vitamin E were corrected in later studies (195), but levels of other nutrients may have been marginal. Examination of the diets they used (195), showed that level of zinc in the mineral mix was only 25% of the NRC requirement (177); the diets also lacked several other trace elements. Since many signs of zinc deficiency are similar to those described above e.g. poor wound healing, perioral skin lesions, reproductive failure, and cleft palate (130; E. Kane, Q. R. Rogers, J. G. Morris, unpublished results), it was difficult to determine which signs were attributable to arachidonate deficiency alone. Although supplementation with evening primrose oil resulted in arachidonate synthesis and caused female cats to come into estrus (81), reproductive performance was not improved (80), which suggests that there may have been a deficiency of another nutrient.

MacDonald et al (151, 152) also found that cats fed diets containing linoleate (5% safflower seed oil) but lacking arachidonate become arachidonate-deficient. Platelet aggregation was impaired in arachidonate-deficient cats, and there was occasional thrombocytopenia (151). Female cats had few litters; kittens were born dead and underdeveloped (152), but there were no apparent birth defects. When the same cats were supplemented orally with ethyl arachidonate, reproductive performance improved greatly; kitten survival increased from 0 to 75%. In cats fed a purified diet with animal fat added to provide arachidonate at 0.04% of dietary kilocalories, reproduction was essentially normal. When other diets containing as little as 0.01% of kilocalories as arachidonate were used, reproductive performance was not always completely normal but was better than it was when arachidonate was absent. Reproductive failure in females is therefore an extremely sensitive index of arachidonate deficiency, and the arachidonate requirement for normal reproduction appears to be very low (152).

**LINOLEATE DEFICIENCY** Because there is negligible synthesis of arachidonate from linoleate in cat liver, the cat is a useful model for studying the specific physiological roles of these two fatty acids. Levels of linoleate can be altered without changing levels of arachidonate in tissues. The results of studies by Frankel & Rivers (81) led them to conclude that "18:2n6 lacks EFA activity in the cat, and that the syndrome encountered was a dietary deficiency in some or all of the derived EFA." They noticed that the condition of the skin and coat was worse in cats fed diets containing hydrogenated coconut oil (lacking linoleate) than in those fed diets containing safflower seed oil (high in linoleate); however, they attributed this to accentuation of arachidonate deficiency by the saturated fatty acids in the coconut oil (194).

Sinclair et al (232) reported that cats fed a diet containing saturated beef fat developed signs of EFA deficiency i.e. lethargy, slow growth, dry, scaly skin, and skin lesions. They also reported that safflower seed oil reversed the deficiency signs (153), but their data have not been published.

MacDonald et al (150) found that cats fed a diet low in linoleate (0.3% of total kilocalories) had low feed efficiencies, scaly skin, hair loss, enlarged fatty livers, and markedly increased rates of water loss through the skin. Necropsies revealed degeneration of the kidneys, testes, and adrenals and hyperkeratosis of the skin (149, 152). These problems were prevented by dietary linoleate (5% safflower seed oil), which demonstrated that linoleate is an EFA for the cat.

### *EFA Requirements*

The EFA requirements of cats have not yet been precisely determined. Although linoleate at 6.7% of kilocalories is more than adequate for growth and normal skin condition (150), the minimum requirement is probably less than this. Calculations of the double-bond index of liver lipids (149) based on comparison to data from rats (115, 157) indicate that the linoleate requirement of the cat may be near 2.5% of dietary energy in the absence of dietary arachidonate.

The discussion of quantitative requirements is complicated by two factors: (a) Arachidonate alone will probably meet the total EFA requirement of the cat, as it does in the rat (115, 157), so the linoleate requirement of the cat will be negligible if there are high levels of arachidonate in the diet. (b) With linoleate at 4.8% of dietary energy and arachidonate at 0.04%, reproductive performance is essentially normal (152), which indicates that 0.04% of kilocalories is near the minimum requirement for arachidonate. However, low levels of dietary linoleate would be expected to increase the arachidonate requirement. The reason for this is that both linoleate and arachidonate are active in maintaining membrane integrity (117). Therefore, although a source of arachidonate is indispensable for the cat, linoleate might spare or reduce the

arachidonate requirement. Further studies are needed to quantitate the linoleate and arachidonate requirements when both are present in the diet.

An observation that deserves further study is that although linoleate prevents liver enlargement due to fatty infiltration, the neutral fat content of liver remains higher than normal (149). Because levels of linoleate in tissues are near maximal with diets containing 5% safflower seed oil, higher levels of dietary linoleate would not be expected to have further beneficial effects. This raises the question of whether a source of fatty acid that is more polyunsaturated than linoleate is needed to minimize the accumulation of neutral fat in liver. In rats, liver fatty acid synthesis is regulated in part by levels of polyunsaturated fatty acids (221), and arachidonate may be more effective than linoleate (45). Dietary arachidonate or other polyunsaturated fatty acids may be required by the cat for normal liver fat metabolism. Before a minimum arachidonate requirement can be assessed, a study is needed of the amount of neutral fat in the livers of cats fed different dietary levels and combinations of linoleate and arachidonate.

The lack of  $\Delta 6$ -desaturation, which prevents the synthesis of arachidonate, would also prevent the conversion of 18:3n3 (linolenate) to longer-chain n3-fatty acids. Rivers et al (199) suggested that these fatty acids may also be required by cats. Because n3-fatty acids can be converted to eicosanoids that can have various pharmacological effects, the cat would be useful in the study of the physiological roles of these fatty acids.

The domestic cat is not the only carnivore that lacks the ability to desaturate fatty acids at the  $\Delta 6$  position. The lion (198), the turbot (a carnivorous fish) (182); and even an insect carnivore, the mosquito (58), have similar inability to synthesize arachidonate. This metabolic peculiarity appears to be correlated with species that are strict carnivores regardless of their position on the phylogenetic tree.

## SUMMARY

From the foregoing discussion of the nutritional requirements and some of the metabolic anomalies of the cat, it is clear that the cat is adapted to eating a carnivorous diet. It may, however, have less capability than omnivores and herbivores to adapt to wide ranges in dietary composition. For example, the lack of ability to synthesize sufficient vitamin A from carotene, ornithine from glutamic acid, arachidonate from linoleate, and taurine from cysteine results from a complete deletion or severe limitation of the enzyme or pathway that makes each nutrient. Other nutrient requirements, such as the absolute requirement for niacin and the high protein requirement, appear to result from the high activity of one or more enzymes and the fact that these enzymes are not adaptive in the cat. For example, the cat cannot decrease picolinic carboxylase in order



to force tryptophan toward the niacin-synthetic pathway (244) nor can it decrease the urea cycle enzymes when dietary protein is decreased in the diet in order to conserve nitrogen (209).

Indeed, the cat appears to have less capability to adapt to most changes in dietary composition because it cannot change the quantities of enzymes involved in the metabolic pathways (209). This evolutionary development has resulted in more stringent nutritional requirements for cats than for omnivores such as the rat, dog, and man. What little evidence exists for other carnivore species leads us to suggest that this pattern may well be common among other strict carnivores.

The metabolic differences between the cat and omnivores provide the researcher with a useful animal model for studying the biochemical basis of some nutrient requirements. For example, because there is no significant conversion of linoleate to arachidonate in cat liver (101, 150, 231), the physiological functions of linoleate can be determined independent of it having a role as a precursor of arachidonate (150). This has not been possible with other species. It is anticipated that further studies of the nutrition of the cat will increase our understanding of metabolic adaptation and nutrient functions.

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