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COMPARATIVE NUTRITION OF CATS AND DOGS

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INTRODUCTION

Although dogs and cats are commonly referred to as "companion animals," they serve as much more than mere companions in our society. Dogs are actively being used to aid blind and deaf people as well as those confined to wheelchairs. Dogs that are used to help the handicapped have been trained to open doors, pick up objects, transport things, and help a person stand, sit, and use a wheelchair. Dogs find missing people and escaped prisoners, and locate drugs and bombs. Playing a vital role in search and rescue work, trained dogs can find people buried beneath rubble during earthquakes, and they can even differentiate between cadavers and living bodies. During wars, dogs have been used to carry messages across enemy lines and to detect bombs and land mines.

There are approximately 58 million cats and 51 million dogs in the United States, representing 30 and 36% of households, respectively (122). Dog-owning households have an average of 1.5 dogs per household; cat-owning households, 2.2 cats (56). Reasons for pet ownership vary: of respondents who own dogs, 95% cite companionship; 48%, protection; 8%, hunting; 4%, breeding/showing; and 0.5%, aid for the disabled (19). Even the animals owned primarily as companions are not viewed as mere pets. Only 36% of cat owners view their cats as pets; 43% view them as family members; 12%, as friends; and 8%, as children (175).

The health and psychological benefits of owning pets have recently been recognized. In 1987 the NIH held a special conference to examine the health benefits of companion animals. Among the recognized psychological benefits of interacting with pets are decreased loneliness, increased self-esteem, increased interaction with others, and development of assertiveness (9). As a result, programs have been developed for the use of pets in therapy for emotionally disturbed children, criminally insane prisoners, and elderly people in nursing homes.

Companion animals are a vital part of our economy. Pet food sales in the US totalled approximately $8 billion dollars in 1989 (123). With a 329% increase in dollar volume since 1970, pet foods represent one of the most rapidly growing areas of the feed industry. Pet food manufacturing is regulated by the USDA, FDA, AAFCO, and state feed control officials.

The most common types of practical diets for dogs and cats are dry, semi-moist, and canned products. Ingredients include corn, corn gluten meal, soybean meal, wheat, rice, plant by-products, meat, and meat by-products. Pet foods contain a greater variety of ingredients, more flavor enhancers, and more by-products than do livestock diets. In addition, dry pet foods are almost always extruded. Therefore, processing losses can have a significant impact on the nutritional quality of the diet.

PROTEIN AND AMINO ACIDS, INCLUDING TAURINE

Young cats and dogs require a dietary source of the same 10 amino acids (105, 132) that are essential for other mammalian and avian species. Methionine can meet the dietary need for both methionine and cystine, but cystine can furnish up to 50% of the requirement for total sulfur-containing amino acids (SAA) in young kittens and puppies (27, 85, 159). Cats and dogs thus resemble other species in regard to cystine's capacity to reduce the amount of methionine required for growth (12). Tyrosine in young kittens and puppies, as in other species studied, can furnish up to 50% of the need for total aromatic amino acids (7, 106).

Whereas dogs resemble other simple-stomached species in protein-amino acid utilization, cats exhibit some peculiar idiosyncrasies, particularly in arginine and SAA metabolism. Also, cats digest protein from conventional diets less efficiently than dogs (94). Moreover, cats exhibit higher protein requirements than dogs, and this is likely due to the cat's high nitrogen requirement for maintenance (102). Nitrogen catabolic enzymes in cat liver are very high (134), and the enzymes that degrade amino acids are nonadaptive so that obligatory nitrogen losses occur even when cats are fed low-protein diets (133).

Arginine

Both cats and dogs exhibit a marked hyperammonemia when fed an arginine-free diet (44, 69, 108, 109). The consequences of arginine deprivation, however, are far more dramatic in the cat. Even a single arginine-free meal fed to cats may cause severe hyperammonemia two to five hours later. Other symptoms of ammonia intoxication include lethargy, emesis, hypersalivation, hyperactivity, hyperesthesia, ataxia, extended limbs, and exposed claws; the most severely affected animals exhibit hypothermia and bradypnea cyanosis, and some die (108, 109). Susceptibility of felids to arginine-deficiency hyperammonemia is related to their inability to synthesize ornithine in the intestine from glutamine or glutamic acid. The intestine is the primary site of de novo ornithine synthesis (135, 170, 171). Two felid mucosal enzymes involved in de novo ornithine synthesis are very low in activity: pyrroline-5-carboxylate synthase and ornithine aminotransferase (135). Morris and coworkers (111) demonstrated that ornithine addition to arginine-free diets of cats can completely prevent hyperammonemia, although it has no effect on weight loss. Thus, while provision of ornithine apparently stimulates hepatic uptake of ammonia (as carbamoyl phosphate) to accomplish urea (and arginine) synthesis, net synthesis of arginine does not occur. Hepatic arginase activity is so high that virtually all of the arginine formed in the liver is quickly catabolized to ornithine and urea. The ornithine taken up by the

kidney, a tissue where arginase activity is low (62, 131, 156–158, 171), cannot be converted to citrulline because the necessary enzyme, ornithine carbamoyltransferase, is not present in renal tissue (62, 131, 156–158).

Unlike ornithine, dietary citrulline can replace arginine in growing animals (44, 62, 111, 156, 157). Citrulline, in fact, will allow young animals to grow maximally (44, 62, 107, 111, 157, 158), although a greater quantity is necessary to achieve maximal growth than is the case with arginine (44). Windmueller & Spaeth (171), reported that 83% of the citrulline released by the intestine is taken up by kidney tissue where it is converted to arginine. Poor uptake of citrulline by the liver and good uptake by the kidney probably account for the efficacy of citrulline in replacing arginine as a growth enhancer and also for the failure of citrulline, at levels isomolar to arginine, to completely correct the hyperammonemia caused by consumption of an arginine-deficient diet (44).

A highly developed system of amino acid catabolism has evolved in strict carnivores such as the cat, mink (100), and ferret (51), perhaps because of their need for gluconeogenesis but also owing to an under-developed system for amino acid biosynthesis. Proline is a dispensable amino acid for both cats (132) and rats (130), but while it can reduce the rat's need for arginine (130), it does not reduce the need for arginine in cats (132). MacDonald and coworkers (102) suggest that ornithine aminotransferase is a constitutive enzyme in cat liver and kidney that leads to ornithine degradation instead of synthesis. Much of the glutamic acid semialdehyde produced from ornithine in this reaction is likely converted to proline via the enzyme pyrroline-5-carboxylate reductase. In contrast, ornithine aminotransferase is adaptive in the rat; thus ornithine catabolism is reduced in rats when a low protein or arginine-deficient diet is fed.

Arginine biosynthesis satisfies the need for arginine in adults of several mammalian species that have been studied (30, 59, 137, 172), but a dietary source of arginine is required by both adult cats (108, 109) and dogs (28). Cats, however, will die if fed an arginine-free diet, whereas adult dogs will merely show signs of unthriftness and will occasionally vomit. A marked orotic aciduria occurs in young or adult dogs fed an arginine-free diet (28, 44); in cats urinary orotate excretion is elevated only slightly under conditions of arginine deprivation (41). Thus, dogs, like rats (107), are susceptible to orotic aciduria, whereas cats, like pigs (62, 148), show only small increases in orotate excretion under conditions of arginine deprivation. Because orotic acid is formed in the cytosol, relative differences in the permeability of the mitochondrial membrane to carbamoyl phosphate may explain species differences in susceptibility to orotic aciduria caused by arginine deficiency.

The arginine requirement for maximal growth is considerably higher in cats than in dogs, and the arginine need for minimal urinary orotic acid excretion

is higher than that needed for maximal growth. Anderson et al (5) reported a dietary arginine requirement for maximal kitten growth of 0.83%, similar to the 0.80% growth requirement observed by Costello et al (41). The latter study, however, included urinary orotate as a criterion of response, and the data, though highly variable, suggested that an arginine level greater than 0.80% may be necessary to minimize orotate excretion. Kittens fed 0.4% arginine lost weight (−9.2 g per day) and excreted 11.5 mg orotate per day; those fed 1.0% arginine gained 13.4 g per day and excreted 2.1 mg orotate per day. For young puppies (44), a weight gain of 30 g per day and an orotate excretion of 116 mg per day were observed upon feeding a purified diet containing 0.2% arginine. Doubling the dietary arginine level to 0.4% increased weight gain to 143 g per day and decreased urinary orotate excretion to 47 mg per day. While increasing the dietary level of arginine to 0.6% of the diet elicited no further response in weight gain, urinary orotate continued to decrease, reaching a plateau excretion level of 1 mg per day. Thus, arginine-adequate diets result in similar quantities of orotic acid excretion in both cats and dogs, but arginine-deficient diets result in 100-fold elevations in orotate excretion in dogs but only 5-fold elevations in cats.

The kitten studies of Costello et al (41) involved much higher levels of nitrogen and lysine (2.24%) than the kitten studies of Anderson et al (5), where 0.80% lysine (the minimal requirement) was contained in the diet. High nitrogen diets that are also high in lysine could cause an increase in the arginine requirement. Excess lysine antagonizes arginine in dogs (45), decreases weight gain (partially reversible by additional arginine), and increases orotate excretion. Whether excess lysine antagonizes arginine in cats is not known. High nitrogen diets, however, have not increased amino acid requirements in the kitten (150) as has been shown to occur in other species (15, 21).

Sulfur Amino Acids

Cats have higher dietary requirements for SAA than dogs (85, 102, 159), and dogs utilize D-methionine and DL-hydroxymethionine more efficiently than cats (27, 36, 160). Methionine and cystine in the diet contribute importantly to acid-base balance in that most of the sulfur excreted from these compounds is voided in the urine as sulfate. An acidic urine is desirable for prevention of struvite urolithiasis in both cats and dogs. Hirakawa has shown that the excess methionine required to effectively lower urine pH of puppies causes a growth depression because of methionine imbalance (84). Thus, the popular veterinary practice of administering supplemental methionine to treat struvite urolithiasis in cats and stone formation in dogs could be dangerous.

Cats differ from dogs in at least two aspects of SAA metabolism: The cat excretes a branched-chain SAA, felinine, in the urine (47, 169), and cats but not dogs require a dietary source of taurine to prevent central retinal degenera-

tion (72, 97) and dilated cardiomyopathy (125). Felinine excretion is much higher in adult male than in adult female cats and is barely detectable in the urine of young kittens (129). Surprisingly, ^{35}S from ^{35}S-methionine or ^{35}S-cysteine was not incorporated into felinine (129). Thus, the route of felinine synthesis as well as its physiological significance is not known. Synthesis of felinine and taurine by cats has been estimated to consume only a small portion of the dietary SAA, assuming that the sulfur moiety of methionine and (or) cysteine must ultimately contribute sulfur for felinine synthesis. In young kittens, the portion of the SAA requirement needed for taurine and felinine synthesis probably does not exceed 5% of the total dietary SAA. Thus, taurine and felinine synthesis in the cat does not provide a reasonable explanation for why cats require much higher SAA levels in their diet than do dogs (102, 110, 118, 142).

Amino Acid Balance and Imbalance

Tolerance to excess dietary glutamic acid is lower in cats than in other species (48); this is probably due to the low activity of alanine aminotransferase in gut mucosa of cats (135). As much as 12% of chemically defined amino acid diets for pigs (37) and chicks (17) can be composed of glutamic acid (to provide amino nitrogen for synthesis of dispensable amino acids), and this source of dispensable nitrogen is as effective in promoting growth and nitrogen retention as a complete mixture of dispensable amino acids. A similar quantity of glutamic acid fed to cats causes a dramatic rise in free glutamate in blood plasma, and some cats vomit and develop thiamin deficiency (48, 49). The exact nature of the glutamate-thiamin interaction is not known, but excess glutamate ingestion does cause an increase in fecal excretion of thiamin (49).

Branched-chain amino acid antagonisms that have been observed in other species (18, 61, 145) have not been observed to the same extent in cats (102). Addition of 10% leucine to intact-protein diets of cats does not depress weight gain or food intake, whereas in pigs, for example, 6% supplemental leucine is growth depressing when added to a conventional corn-soybean meal diet (61).

Amino Acid Requirements

Work was done in the 1980s to establish minimal amino acid requirements for maximal growth of kittens and puppies. The bulk of this work was done at the University of Illinois and at the University of California (Davis) using purified crystalline amino acid diets. Because in most cases the diets used contained nitrogen and energy levels different from those used in practice (i.e. commercial diets), and because digestibility and bioavailability factors impact utilization of protein-bound amino acids in the commercial setting, a listing of minimal amino acid requirements for maximal growth has limited usefulness. Researchers in the United Kingdom have developed the concept of an "ideal

protein" requirement for pigs, and this circumvents many of the problems encountered in extrapolating requirements, based on purified diets, to a practical setting. Thus, the ideal protein concept attempts to establish the ideal ratio of indispensable amino acids (setting lysine at 100) based upon tissue needs for maximal growth and diet utilization. The ideal profile of amino acids is independent of dietary nitrogen and energy levels, both of which can influence the dietary needs for amino acids expressed in terms of dietary concentration.

Based upon the results of requirement studies done with cats (110, 118) and dogs (43, 85, 86, 117), ideal amino acid ratios are listed in Table 1, and for comparative purposes, these ratios are compared to the current best estimates of an ideal amino acid profile for pigs (2, 37, 148, 166) and chicks (11, 17, 70, 93, 101, 140). Use of the ideal protein concept can simplify diet formulation because one can formulate dog diets to a lysine need and cat diets to a SAA need. This concept assumes that most dog diets contain lysine at a level closest to its requirement, and that most cat diets contain methionine at a level closest to its requirement. Because some cat diets require fortification with crystalline methionine, it may prove useful to formulate such diets to meet minimal arginine and lysine requirements, and then fortify the diets with methionine to bring SAA to their desired ratio to lysine.

To provide points of reference for the key amino acids used to estimate desirable ratios to lysine, the SAA, arginine, tryptophan, and lysine needs of cats and dogs deserve special consideration. We assumed lysine requirements (% of a purified diet) of 0.80% for cats (118) and of 0.70% for dogs (86). Arginine requirements were estimated at 0.90% for cats (5, 41, 118) and at 0.50% for dogs (117), while SAA requirements were assumed to be 0.80% for cats (141, 159) and 0.45% for dogs (85). Tryptophan requirements were

Table 1 Ideal amino acid profiles (relative to lysine) for cats, dogs, pigs, and chicks

Amino acid	Ideal ratio by species			
	Cat	Dog	Pig	Chick
Lysine	100	100	100	100
Methionine + cystine	100	64	60	67
Tryptophan	19	22	18	16
Threonine	87	67	65	67
Arginine	112	71	42	105
Isoleucine	63	57	60	67
Valine	75	75	68	77
Leucine	150	100	100	111
Histidine	38	29	38	37
Phenylalanine + tyrosine	112	100	95	100

0.15% for cats (7, 118) and 0.16% for dogs (43). All of these requirement estimates were made with young kittens and puppies, and all were accomplished by feeding graded levels of the amino acid in question in purified amino acid diets containing, within species, the same level of metabolizable energy. Thus, the estimated ratios (to lysine) of SAA, arginine, and tryptophan in cats and dogs, though subject to some degree of interpretation, have considerable veracity.

In comparing cat and dog ideal amino acid ratios with pig and chick values, the most striking differences occur in arginine and SAA levels (relative to lysine) for cats. In general, the dog values, and the cat values for amino acids other than arginine and SAA, agree closely with the pig values, although the ideal arginine:lysine level in dogs exceeds that in pigs by a substantial amount. This finding may reflect the fact that dogs have a greater susceptibility than pigs to orotic aciduria caused by inadequate arginine feeding. Thus, the estimated arginine requirement of 0.5% of the diet (117) for dogs was based upon minimizing urinary orotate excretion (44). The high ratio of arginine to lysine in chicks results from the fact that avian species do not have a functional urea cycle and therefore cannot synthesize arginine in either the liver or kidney (157, 158). The high ratio of leucine to lysine in cats seems anomalous. More research is needed to establish whether cats require 50% more leucine than lysine. The high relative requirement for threonine in cats also needs verification.

Taurine

The cat is unique in its need for dietary taurine. Both inadequate synthesis (from methionine and cystine) and high physiological demand are responsible. However, decreased activity and concentration of cysteinesulfinic acid decarboxylase, the enzyme necessary for conversion of cysteine to hypotaurine, limits taurine synthesis to low levels in the cat (97, 173). Also some of the cysteine is shunted toward synthesis of felinine, a unique metabolite found only in cat urine (169). Felinine is thought to be involved in territorial marking or in the regulation of sterol metabolism (102). Cysteic acid, on the other hand, is an excellent precursor of taurine (60). In fact, there is evidence that at low levels of taurine intake, cysteic acid may be a better provider of peripheral taurine than taurine itself (Q. R. Rogers, 1990, personal communication). While taurine is converted to taurocholic acid in the liver, cysteic acid bypasses this step and may be used more directly as a taurine source by peripheral tissues. The physiological demand for taurine by cats is also remarkably high compared to other species. Most animals have the ability to respond to changes in dietary concentrations of taurine and glycine by altering the ratio of the bile acid conjugates containing these two amino

acids. However, cats rely almost exclusively on taurine for bile acid conjugation, regardless of the dietary concentration of taurine (127, 167).

Taurine metabolism and signs of taurine deficiency have been reviewed by others (6, 34, 65, 71, 87, 118, 152, 174). The newer taurine research is covered here, with emphasis on metabolic functions and effects of diet on taurine absorption, excretion, and status.

While attempting to ascertain whether cysteine could be used as a source of taurine in cats fed a taurine-deficient diet, Sturman et al (153) inadvertently produced cysteine toxicity. The neurotoxicity associated with excess cysteine consumption was found to be exacerbated when combined with taurine deficiency. All nine of the cats fed a diet containing 5% cysteine and no taurine exhibited signs of neurotoxicity and ultimately died. Only four of the cats fed the same level of cysteine in the presence of 0.05% taurine died; the remainder appeared normal. The biochemical mechanism of this interaction has not been determined. However, taurine appears to have a role in nervous system excitability and may have a neuroprotective role.

Taurine has recently been implicated in immunocompetency (143, 144), although its actual role remains to be elucidated. Prolonged taurine deficiency in the cat causes regression of follicular centers of spleen and lymph nodes, as well as depletion of reticular cells and mature and immature lymphocytes. Proliferative responses to the T-cell mitogen Con A are reduced, and phagocytosis declines. Taurine can act as a scavenger of strong oxidants such as hypochlorous acid and free radicals (174). As such, taurine may be viewed as a general detoxifier that protects cell membranes from oxidative damage (64, 65, 174). Antioxidant effects and membrane protection by taurine have been shown in rabbit spermatozoa (3) as well as in hepatic cells exposed to carbon tetrachloride (115).

Taurine deficiency has been shown to cause profound aberrations in feline reproduction and development. These effects, which have been summarized previously (151–155), include resorption and abortion of fetuses, stillbirths, low birth weight, and neurological abnormalities in surviving kittens. Proper brain development seems to be dependent on adequate taurine intake.

An oral dose of taurine is virtually 100% absorbed in the cat (54). Absorption occurs via both active and passive transport (39); during taurine deficiency, active transport increases relative to passive transport. The greatest rate and extent of absorption occurs in the jejunum.

Renal taurine transport is also responsive to dietary taurine concentration (120). During taurine deficiency in the kitten, urinary taurine excretion decreases markedly. Also, the taurine reabsorption rate (V_{max}) of brush border membrane vesicles increases and K_m decreases, signifying both an increase in transport sites and a greater affinity for taurine by renal tubules. Thus, the cat

conserves taurine during periods of limited taurine intake by modifying both taurine absorption and excretion.

Taurine deficiency is a widely recognized practical problem in cats fed commercial diets (124, 125). The dietary taurine requirement seems to be dependent on the diet fed and the parameter measured. Recently Pion and coworkers (125) reported that dilated cardiomyopathy in cats is related to low blood taurine levels. This condition is responsive to taurine supplementation. Curiously, dilated cardiomyopathy occurs in cats fed diets that were previously believed to contain more than adequate amounts of taurine (118).

A related phenomenon is that the requirement for taurine is higher in cats fed canned diets than in those fed dry commercial diets. Douglass et al (53) recently demonstrated that over twice as much taurine was required in a canned diet as in a dry diet to reach similar plasma or whole blood taurine concentrations. The mechanism for this interaction remains an enigma. Recovery of ^{15}N taurine, which was added to a canned diet before processing, was 100% (55). Likewise, Hickman et al (79) reported 88% recovery of crystalline taurine from a canned diet. Loss of taurine during processing is therefore minimal. Taurine remains in free form during processing (79). Hence, direct incorporation of taurine into a Maillard product during processing does not seem to be involved.

It is obvious that processing *per se* of cat diets is involved in producing the taurine-depleting effect. Researchers at the University of California at Davis (78) fed two diets with identical formulations; one diet was canned (heated at 120°C for 80 minutes) and the other frozen until used. While the heat-processed canned diet depleted plasma taurine, the nonheat processed diet did not. In addition, ileal chyme taurine, as a percent of taurine intake, was higher in cats fed the canned diet, but there was no difference in taurine excretion in urine. Expiration of ^{14}C taurine as $^{14}CO_2$ was higher in cats fed the canned diet (79), an indication of increased microbial degradation of taurine. Why more taurine would reach the lower intestine and be degraded by microbes with a canned than with a frozen diet is not known. As expected, urinary ^{14}C, as a percent of the dose, was lower in cats fed the canned diet. Isotope recovery ranged from 20 to 44% over the 14-day assay.

Douglass et al (53, 54) studied taurine metabolism in cats fed either a canned or a dry diet and given either an oral or an intravenous dose of ^{15}N taurine. The oral dose was either incorporated into the diet before processing or given as an oral supplement. In contrast to the previously discussed results with ^{14}C taurine, cats excreted 60% more ^{15}N in the urine when they were fed the canned instead of the dry diet (80% versus 50% of the dose, respectively). Similarly, cats fed the canned diet excreted a larger percentage of the oral than of the intravenous ^{15}N dose in the urine, and less in the feces, during the first six days of the trial. The ^{15}N results suggest that taurine reabsorption in the

renal tubule is decreased in cats fed canned diets. An inhibitor such as a Maillard product, which is formed during processing and then absorbed, could be responsible for the decreased renal tubule reabsorption of taurine (E. B. Fern, 1990, personal communication).

An integrated hypothesis for the taurine-depleting effect of canned diets is that much of the taurine may be degraded by intestinal microbes and the ^{14}C excreted as $^{14}CO_2$. The ^{15}N of microbially degraded taurine is probably absorbed and subsequently excreted in the urine. Further research should examine taurine labelled with both ^{14}C and ^{15}N. The vast differences in total isotope recovery between the two studies [28% of the ^{14}C was recovered (79) and 90% of the ^{15}N was recovered (54) in cats fed the canned diets] also suggest that both hypotheses may be correct; that is, some of the taurine is degraded by intestinal microbes and some is absorbed, but not reabsorbed, in the renal tubules.

Using ^{15}N stable-isotope-labelled taurine, Fern (1990, personal communication) calculated that the half-life of taurine in cats fed a canned diet was 4.2 days while the half-life of taurine in cats fed a dry diet was approximately 8 days. Likewise, the total body taurine pool was lower for cats fed a canned diet.

C. E. Wright and J. A. Sturman (1990, personal communication) measured taurine uptake in human lymphoblastoid cells from the same canned and frozen diets used by Hickman et al (78, 79). Taurine uptake was inhibited by a water extract of the canned but not the frozen diet. Clearly, an inhibitor of taurine uptake was formed during processing. No inhibitor was formed during cooking of synthetic diets. Therefore, a product(s) unique to canned diets is responsible for the effect. This product may inhibit taurine absorption in the intestine, affect reabsorption of bile acids, and/or affect reabsorption in the renal tubule. The fact that fecal ^{15}N was greater in cats given the intravenous dose than in those given the oral dose (54) indicates that something happens to taurine, but not taurocholic acid, in the intestinal tract.

One major difference between canned and dry diets is the presence of gums in canned products. Recent research indicates that soluble fiber components such as pectin and guar gum deplete hepatic taurine content, decrease urinary taurine excretion, and reduce cysteine dioxygenase activity in rats (88, 89). Meat by-products such as lungs, liver, and gullets are present in canned diets. Some component of these by-products may be altered during heat processing to form an inhibitor of taurine uptake.

Source of protein apparently affects taurine status. The size of the taurocholic acid pool and the irreversible loss of both taurine and cholic acid from the taurocholic acid pool were found to be higher in cats consuming soy protein than in those consuming casein (80). Whether similar differences exist for other protein sources remains to be elucidated. Differences in amino acid

pattern reaching the small intestine may influence cholecystokinin release. Consequently, bile flow would be altered and taurine status possibly jeopardized.

Regardless of the mechanism, it is clear that the taurine requirement of cats is different when dry and canned commercial products are fed. While the National Research Council (118) originally recommended 400 mg taurine per kg diet as the minimum requirement, cats consuming dry commercial diets require 1000–1200 mg taurine per kg diet, and cats consuming canned commercial diets require 2200–2500 mg taurine per kg diet on a dry basis (53, 124). Diets, regardless of type, should contain sufficient taurine to support plasma taurine concentrations of 60 nmol per ml (124).

CARBOHYDRATES AND FAT

Carbohydrate utilization has been studied in both dogs and cats. Like humans, adult dogs and cats display lactose intolerance (112, 113). Diarrhea is also prevalent in puppies consuming raw cornstarch (G. L. Czarnecki-Maulden, K. P. Boebel, D. H. Baker, University of Illinois, unpublished data). When formulating a purified diet for puppies, one must ensure that the cornstarch is gelatinized before inclusion in the diet. In contrast, pigs (128), chicks (17), rats (14), and cats (8) tolerate raw cornstarch. In commercial diets, corn must be cooked either before or after incorporation into the diet (77). Digestibility of potato and maize starch by cats is also improved by cooking (52).

An extensive review of carbohydrate metabolism and comparative activity of digestive enzymes was recently published (110). Of note is the observation that the cat appears to be in a continuous state of gluconeogenesis. In addition, both glucagon and insulin are less responsive to glucose in felids than in other species.

Initial studies indicated a dietary requirement for carbohydrate in the gestating dog (136). Bitches fed a carbohydrate-free diet exhibited hypoglycemia, ketosis, and poor pup survivability. However, subsequent research (20, 95) demonstrated that if dietary protein was high enough to meet energy needs, carbohydrates were not required.

Because of a deficiency of the enzyme delta six desaturase (103), cats require a dietary source of arachidonic acid. Arachidonic acid is only found in fats of animal origin. Therefore, either animal fat or a synthetic source of arachidonic acid must be included in the diet of cats.

MINERALS

Little is known about the mineral requirements of dogs and cats. It is generally accepted that both species have qualitative requirements for mineral elements that are the same as those established for other mammalian species

such as the rat and pig. Also, the plant-source factors, soluble fiber and phytate, probably reduce the bioavailability of mineral elements for cats and dogs in the same manner as has been shown for other species. Hence, "total" requirements for most mineral elements are considerably higher for cats and dogs fed grain and soy-based dry diets than for those fed purified research diets based upon casein or free amino acids (118).

Calcium and Phosphorus

The calcium (Ca) and phosphorus (P) requirements of dogs and cats are not well defined but seem similar to those of other species (117, 118). Hintz & Schryver (83) reported that cats maintain positive Ca balance with as little as 0.3% dietary Ca. However, bone fractures occurred in dogs fed diets containing 0.55% Ca (74, 75), even though the dogs were in positive Ca balance. Therefore, positive Ca balance is not a good indicator of Ca adequacy in the dog. Both Ca absorption and bone turnover increase in dogs fed a low Ca diet (74, 75). These adaptations are not adequate to maintain normal blood Ca.

The Ca:P ratio is at least as important as the actual dietary levels of Ca and P. Great Danes fed 0.55% Ca and 0.9% P (74) exhibited osteoporosis with fractures in the axial and abaxial skeleton. Bone turnover, Ca absorption, and Ca resorption were all higher in these dogs than in dogs fed 1.1% Ca. Lowering P to 0.50% (75) resulted in fewer bone fractures. Thus, the Ca:P imbalance was a primary cause of the skeletal abnormalities. Since fractures still occurred in Great Danes fed 0.55% Ca and 0.5% P, Hazewinkel et al (75) suggested the dog's Ca requirement must be greater than 0.55%. Bone abnormalities typical of excess Ca ingestion, however, were observed in dogs fed 1.1% Ca (74).

Numerous cases of Ca:P imbalance and nutritional secondary hyperparathyroidism have been reported in both cats and dogs (82, 83), primarily in animals fed diets rich in meat products. Meat products are rich in P but low in Ca.

Dogs cannot completely adapt to consumption of excess Ca. Hazewinkel (74) fed Great Danes 0.9% P with either 0.55, 1.1, or 3.3% Ca. Dogs fed the diet with 3.3% Ca developed osteochondrosis, radius curvus syndrome, and myelin degeneration. Calcium absorption was estimated to be the same in dogs fed 3.3% Ca as in those fed 1.1% Ca.

As a species, dogs are unusual in that adult weight ranges from 1 to 125 kg. The larger breeds exhibit a rapid growth rate that places added stress on the skeletal system. Skeletal defects are common in large breeds, but rarely occur in smaller dogs. Although many of these skeletal abnormalities have a genetic base, nutrition and growth rate can influence the severity of the lesions (98). Hedhammar et al (76) reported numerous skeletal abnormalities in growing Great Danes fed a highly palatable diet that was rich in Ca, P, protein, and metabolizable energy. Voluntary feed intake was double that recommended

by NRC (117). Lavelle (99) was unable to repeat these results with dogs consuming lower quantities of a more nutritionally balanced diet. Thus, it appears that overconsumption of an energy dense, high Ca, high P diet is detrimental to growing dogs. Hip dysplasia, osteochondrosis, wobbler's syndrome, and hypertrophic osteodystrophy have each been associated with overnutrition and fast growth rate in dogs (76, 98).

The P present in plant-source feed ingredients is largely present as phytate-P, a form of P considered to be largely unavailable to simple-stomached animals. The P in corn-, soy-, and wheat-based products that make up most dry dog and cat foods is probably no more than 30% bioavailable relative to commercial dicalcium phosphate (117–119). The P in meat products, on the other hand, is probably equal in bioavailability to the P contained in dicalcium phosphate.

Supplemental P is available to the commercial feed industry as monodicalcium phosphate, i.e. $Ca(H_2PO_4)_2 \cdot 2H_2O$ (16% Ca, 21% P), dicalcium phosphate, i.e. $CaHPO_4 \cdot XH_2O$ (22% Ca, 18.5% P), or defluorinated rock phosphate (32% Ca, 18% P). P bioavailability in these supplements, relative to monodicalcium phosphate set at 100%, is thought to be 90% in dicalcium phosphate and about 80% in defluorinated rock phosphate (165). Complicating the use of these values in practice is the fact that commercial dicalcium phosphates are mixtures of $CaHPO_4$ and $Ca(H_2PO_4)_2$. They also contain smaller quantities of other compounds that contribute Ca and P activity, e.g. $CaCO_3$, $FePO_4 \cdot 2H_2O$, $A1PO_4$, $Mg(H_2PO_4)_2 \cdot 4H_2O$, $CaSO_4 \cdot H_2O$, CaF, $Na_2H_2PO_4 \cdot 2H_2O$, and H_3PO_4, in addition to the calcium phosphate compounds $Ca(H_2PO_4) \cdot 2H_2O$, $CaHPO_4 \cdot H_2O$, and $CaHPO_4$ (13). Thus, perhaps Ca and P analysis is the only effective means of estimating the ratio of $CaHPO_4$ to $Ca(H_2PO_4)_2$. A rule of thumb in swine and poultry feed formulation (13) is to set the P bioavailability in the purchased feed-grade phosphate source at 100% (regardless of whether it is labelled dicalcium or monodicalcium phosphate) and then assume that the P bioavailability is also 100% in meat-source P, 90% in defluorinated P, and 30% in plant-source P.

Iron and Copper

The dietary iron (Fe) requirement for weanling puppies and kittens fed a casein-based purified diet is 80 mg per kg diet (31); this is approximately twice that needed by growing chicks fed a similar diet (147). Likewise, there are striking species differences in the utilization of feed sources of Fe. Cats utilize Fe from dried beef liver 350% more efficiently than Fe from ferrous sulfate (32). Chicks, on the other hand, utilize the Fe in dried beef liver at 90% efficiency relative to ferrous sulfate (32). The dried beef liver used in this study contained only 5% of the Fe as heme Fe. It seems reasonable that the cat may have evolved in a carnivorous manner that resulted in an enhanced ability to utilize hemosiderin and ferritin, the two major storage forms of Fe in

animal tissues. Plant sources of Fe such as corn gluten meal (32) are poorly utilized by cats (20% bioavailability) but well utilized by chicks (84% bioavailability).

Whether these differences hold true for other plant and animal sources of Fe remains to be determined. Mechanisms of heme and nonheme Fe absorption have not been investigated in cats. Both dogs and cats are more sensitive than other animal species to dietary excesses of Fe (116).

Ferrous sulfate and ferrous carbonate are commonly used as sources of Fe in pet foods. Pig and rat studies have shown that the Fe in ferrous carbonate is poorly utilized relative to the Fe in ferrous sulfate (4). In addition, the Fe present in feed-grade sources of calcium and phosphorous (dolomite, limestone, dicalcium phosphate, monocalcium phosphate, and soft rock phosphate) is 30–50% bioavailable (50). The commercial phosphate supplements contain large quantities of Fe (6500–9500 mg per kg) mostly as $FePO_4 \cdot 2H_2O$. Therefore, phosphate supplements can provide substantial quantities of bioavailable Fe to the diet. The bioavailability of Fe in ground oyster shell, steamed bonemeal, and soft rock phosphate is zero (50). Ferric oxide, which contains no bioavailable Fe, is commonly used as a source of color in pet foods.

Copper (Cu) requirements of dogs and cats do not appear to be substantially different from those of other species. Bedlington terriers and a number of other breeds, including West Highland white terriers and Doberman pinschers (161, 162), have exhibited an inherited defect in Cu metabolism that is similar to Wilson's disease in humans. Excess Cu accumulates in the liver with resultant hepatitis and cirrhosis. Affected animals have a decreased capacity for biliary Cu excretion. As with humans, management of this Cu toxicity problem involves reduction of Cu intake as well as use of chelating agents to decrease Cu absorption (164). Certainly, commercial dog foods containing substantial quantities of liver (rich in Cu) cannot be fed to affected animals.

Supplemental Cu is generally added to cat and dog foods as either copper oxide or copper sulfate ($CuSO_4 \cdot 5H_2O$). Feed-grade copper oxide is largely cupric oxide (CuO), a form of Cu thought to be totally unavailable to pigs (42) and chicks (16).

Zinc

In practice, zinc (Zn) deficiency is more common in dogs than in cats; this is probably not due to an inherent difference in Zn metabolism or requirement between the two species. Rather, when compared to commercial cat foods, dog foods contain higher levels of plant products, and hence phytate. Thus, bioavailability of Zn would likely be lower in dog foods than in cat foods. Zinc deficiency was produced in the cat by Kane et al (92). They concluded that the zinc requirement was between 15 and 50 mg per kg diet. Although

signs of Zn deficiency have been reported in the dog (138, 139, 146), a quantitative Zn requirement study has never been published.

Inherited metabolic defects in Zn absorption or metabolism have been reported in a number of dog breeds, particularly the arctic breeds (10, 24). The condition generally manifests as a Zn-responsive dermatosis that is not related to low dietary Zn intake. Zinc absorption is impaired in Alaskan malamutes with genetic chondrodysplastic dwarfism (24). While Zn is taken up into the intestinal cell, it is not released from a low molecular weight Zn-binding ligand and is, therefore, not available for transfer across the serosa.

The most common sources of zinc in commercial pet foods are $ZnSO_4 \cdot H_2O$ and ZnO. The bioavailability of Zn in ZnO is approximately 60% relative to that in $ZnSO_4 \cdot H_2O$ (168).

Zinc toxicity was recently reported in dogs consuming galvanized nuts from air-freight carrier cages (163) and in those consuming pennies minted after 1982 (126); both are very rich in Zn. Signs of toxicity included vomiting, jaundice, and hemolytic anemia.

Manganese and Selenium

There is virtually no published research concerning deficiency, requirements, or metabolism of manganese in either dogs or cats. Likewise, selenium (Se) requirement studies have not been published. High concentrations of Se occur in tuna-based canned cat foods (64). However, overt signs of Se toxicity have not been reported in cats consuming these products (22). Tuna-based canned cat foods may contain high amounts of mercury, which has a protective effect against Se toxicity (116). In addition, the bioavailability of Se in tuna may be low (55).

Potassium

The potassium (K) need of cats and dogs is easily met in dry formulated diets that contain a considerable quantity of soybean meal, an ingredient very rich in K (117, 118). Both canned and dry meat-based dog and cat foods are lower in K than those rich in soybean meal, and some of these diets may require K supplementation. Clearly, high protein diets increase the kitten's dietary requirement for K (81), probably because high protein diets lead to elevated sulfate excretion, which may cause increased K excretion via the kidney. Dow et al have recently shown that acidification of diets for adult cats (to decrease urine pH) may lead to K depletion as well (57, 58).

Magnesium-Feline Urologic Syndrome

Signs of magnesium (Mg) deficiency in the growing kitten and puppy include hyperirritability, convulsions, reduced bone and serum Mg, and aortic

calcification (26, 33). Magnesium deficiency is not likely to occur for either cats or dogs. The potential harmful effects of excess dietary Mg have recently been emphasized.

Excess Mg intake was previously linked to feline lower urinary tract disorder, commonly referred to as feline urologic syndrome (FUS). The syndrome is characterized by dysuria and hematuria as well as production of struvite (Mg ammonium phosphate) uroliths (25). Urethral blockage and death occurred in severe cases. Recurrence is common if preventive measures are not taken.

Early research targeted ingestion of excess levels of Mg as the primary cause of FUS. Other possible causative factors have been reviewed (118) and have largely been discounted. Initial studies used MgO or $MgCO_3$, compounds that also resulted in production of alkaline urine. When $MgCl_2$ was fed, cats produced acidic urine and FUS was not evident (25). Thus, urine pH was found to be much more important than Mg intake. At low urine pH (less than 6.5) struvite uroliths are soluble. As urine pH increases, the uroliths precipitate and clinical signs of FUS arise. Consequently, current emphasis is being placed on preventing and treating FUS by producing acidic urine. However, dietary manipulations that cause an extremely low urine pH for extended periods of time should be avoided. Urine pH should be monitored periodically, and cats should not be given urinary acidifiers if they are already being fed a diet that produces a urine pH of 6 to 6.4. Prolonged production of urine with a pH of 5.8 to 6.0 causes metabolic acidosis (35, 58, 91), negative potassium balance (35, 58), and bone resorption (35). Hypokalemia has been reported in cats fed commercially available diets (57), and the condition was exacerbated by dietary acidification (58). In addition, renal dysfunction was induced in cats fed an acidified, potassium-deficient diet (58).

The most common therapeutic acidifying agents are ammonium chloride and methionine. Both are toxic if misused or fed to young kittens (23, 63). Use of methionine to acidify urine of puppies also results in methionine-induced anorexia (84).

Commercial cat foods are often designed to produce acidic urine through the selective inclusion of acidifying ingredients such as phosphoric acid, which is an effective acidifier (91). The acid or alkaline contribution of an ingredient can be calculated. Kienzle et al (96) accurately predicted urine pH by calculating base excess (BE, mmol per kg dry matter): $BE = 2[Ca] + 2[Mg] + [Na] + [K] - 2[P] - 2[Met] - [Cl]$; mean urine $pH = 6.5 + 0.0024\ BE$ ($r = 0.96$). The equation does not take into account inorganic sulfate or cystine. If the methionine:cystine ratio or inorganic sulfate content of a diet differs from that employed by Kienzle et al (96), the predicted urine pH will not be accurate. An alternative method is to calculate dietary undetermined anion (dUA, milliequivalents per kilogram of dry matter) (68,

121); dUA = (Na + K + Ca + Mg) − (Cl + S + P). The ideal dUA has not been determined for either cats or dogs.

VITAMINS

Research on vitamins has revealed that the vitamin needs of cats and dogs differ little from those of pigs or rats. The exceptions worthy of note are vitamin A and niacin for cats, and thiamin as well as vitamin E for both cats and dogs. While vitamin E needs of cats and dogs are not greatly different from those of other species under normal dietary circumstances, commercial diets containing high levels of fish or fish oil require much higher levels of vitamin E activity to prevent steatitis, muscle wasting, impaired spermatogenesis, and other symptoms characteristic of vitamin E deficiency (38, 40, 67, 73, 114). Clearly, high dietary levels of polyunsaturated fatty acids stress the vitamin E requirement of cats and dogs. In the case of thiamin, the problem for cats and dogs relates less to thiaminase activity in uncooked fish than to thermal destruction of thiamin during processing of cat and dog foods. Commercial dry pet foods are extruded, and extrusion is known to cause losses in thiamin activity (29). Canned diets are also heated to high temperatures. Supplemental thiamin is available commercially as either thiamin·HCl or thiamin·NO_3. Adams (1) showed that the mononitrate form of thiamin retained 98% of its activity when present in a complete feed stored for 21 days at 40°C and 85% relative humidity; the hydrochloride form of thiamin, however, retained only 21% of its bioactivity when stored under similar conditions.

Cats unlike dogs require a dietary source of preformed vitamin A. Feline species lack the dioxygenase enzyme necessary to cleave β-carotene to vitamin A (66). As for niacin, felids contain all the necessary hepatic enzymes required for niacin synthesis from tryptophan, but the high activity of picolinic acid carboxylase (90) causes rapid removal of 2-aminomuconic acid semialdehyde from the niacin biosynthetic pathway (104). Thus, very little nicotinic acid is synthesized. Unlike dogs, cats die within 20 days when fed diets high in tryptophan but devoid of nicotinic acid (46).

There are voids in our knowledge of the effects of certain vitamins in cats and dogs. Without resorting to use of coumarin compounds, vitamin K deficiency has been difficult to produce in cats and dogs (117, 118). Recent attempts by Strieker (149) to produce vitamin K deficiency in cats were unsuccessful, even though diets with very low levels of vitamin K were fed for a prolonged period of time to kittens receiving high levels of antibacterial agents in an attempt to minimize intestinal synthesis of the vitamin.

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