Taurine: an essential nutrient for cats

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ABSTRACT

The ß-sulphonic amino acid taurine is synthesised in animals from dietary sulphur amino acids. Cats exclusively use taurine to conjugate cholic acid rather than being able to use the alternate glycine conjugation. Since total body synthesis of taurine in cats is limiting, metabolic deficiencies of taurine occur when the dietary intake of taurine is restricted. A deficiency of taurine in cats is expressed by aberrant functions of a wide range of organ systems. Pathological changes occur in the eye, feline central degeneration; reproductive abnormalities occur in the female, a high incidence of fetal resorptions and abortions, low birth weight and survival of live-born young; growth rate in the new born kitten is depressed; heart induction of dilated cardiomyopathy and compromised immune function. All these conditions are prevented or reversed with adequate dietary taurine. While all tissues contain taurine, the concentration varies with the tissue. Generally plasma has been used to assess taurine status in cats, but the concentration of taurine in plasma varies widely. Food deprivation of cats given high taurine diets causes a marked fall in the concentration of taurine in plasma. Major changes in whole blood concentration do not occur as rapidly as plasma and therefore appear to be a superior diagnostic test for taurine status. An inadequate number of samples have been analysed to define a marginal taurine level from whole blood concentration to prevent clinical signs. The minimal dietary concentration of taurine to prevent clinical signs of efficiency is dependent on the type of diet. For commercial expanded (dry) cat foods a concentration of 1200 mg taurine/kg dry matter appears adequate. Higher concentrations are required in canned diets, 2000 to 2500 mg taurine/kg dry matter to supply adequate taurine. The reasons for the higher concentration of taurine required in canned foods is not due to availability of taurine in the classical context. Rather it appears that heating during the canning process produces products which increase enterohepatic loss of taurine.

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INTRODUCTION

A complete diet for a simple-stomached mammal has to contain nine or 10 α-amino acids. These amino acids are essential dietary constituents as they cannot be synthesised by the body from precursors normally present in the diet at a rate compatible with the animal's needs. A deficiency of any one of these α-amino acids generally results in non-specific clinical signs, such as depressed growth rate, and sub-optimal function of the animal. In addition to these 10 essential α-amino acids, cats also require a ß-sulphonic amino acid, taurine. A deficiency of this amino acid in the diet, in contrast to the 10 essential α-amino acids, results in a number of specific pathological changes. Taurine is not a normal constituent of proteins, but is associated with animal proteins, and is present in all animal tissues as a free amino acid at concentrations often greater than any other free amino acid. While the presence of taurine in the bile salts as taurocholic acid has long been recognised, the function of high concentrations of free taurine in the tissues of mammals has been elusive. A deficiency of taurine in cats has been shown to induce a wide range of clinical signs, involving disparate organ systems.

This paper will briefly discuss the reasons why cats require dietary taurine, changes which result from a diet deficient in taurine, some of the difficulties in assessing the taurine status of cats and adequacy of diets.

ESSENTIAL NATURE OF TAURINE FOR CATS

The essential presence of taurine in the diet of cats arises because the rate of synthesis does not meet the rate of loss of taurine from the body. That is, either rate of synthesis of taurine in cats is less than in other mammals, or rate of loss is greater, or possibly both. No major pathway of taurine metabolism has been identified in mammals other than its conjugation with bile acids to form bile salts. Cats, like most other animals, use taurine to conjugate the bile acids, but unlike most other

placental mammals, cats cannot substitute glycine when tauriné is limiting (Rentschler and others 1986). The obligatory use of taurine to produce bile salts results in a continuous loss of taurine from the body, proportional to that not recovered by the enterohepatic circulation.

Formation of bile salts depends on activation of cholic acid by the enzyme choloyl-CoA synthetase to produce choloyl-CoA. Choloyl-CoA reacts with glycine or taurine in the presence of the enzyme choloyl-CoA-amino acid N-acyltransferase to form the bile salts. Vessey (1978) examined the nature of the acyltransferase in the guinea pig, rabbit and human, by measuring production of bile salts from supernatant fractions of the liver. He found that while the affinities of the enzyme towards glycine and taurine varied, independent of whether the species produced bile salts containing predominantly glycine or taurine, the supernatant fraction from the livers of these animals could make at least some of the glycine conjugates. He suggested that the appearance of glycine conjugated bile salts in placental mammals is a result of two contributory factors. One is the appearance of a new or altered enzyme, an enzyme capable of using glycine as well as taurine. The other is that hepatic levels of taurine are low in some species that produce mainly glycine conjugates. To our knowledge the nature of bile acid-CoA amino acid N-acyltransferase in cats has not been investigated. From an evolutionary viewpoint, taurine would have been well supplied in the diet of a true carnivore, and any selection pressures towards modification of the enzyme would not have been present during the last 35 million years of the evolution of cats.

A proportion of the taurine secreted as cholyltaurine in the bile of mammals is recovered by the body in the enterohepatic circulation. In humans, estimates of the daily turnover of the bile acid pool through the enterohepatic circulation, range from four to eight (Brunner and others 1972) to five to 15 times per day (Berry and Reichen 1983). The size of the bile acid pool in cats has been reported to be 819 ± 149 μmol (Radberg and others 1987). The majority of the bile acids in cat bile are conjugated with taurine, so if a conservative turnover of six times per day was assumed, this would represent a total turnover of taurine of 5 mmol, or 600 mg taurine/day.

In the intestine, bacteria deconjugate bile salts. In humans, about 18 per cent of the glycine-conjugated bile salts are deconjugated at each enterohepatic cycle (Hepner and others 1972a, b). The deconjugation of taurine-conjugated bile salts is less than the glycine-conjugated bile salts, accounting for about 40 per cent of the cholyltaurine pool, or about 7 per cent per cycle, if it is assumed that the pool turnover is six times per day (Hepner and others 1973).

The only significant metabolic pathway of taurine in mammalian tissue which has been identified, is conjugation with bile acids. Therefore, a cat fed a diet containing a constant level of taurine should achieve an equilibrium situation where taurine excretion in faeces and urine should equal dietary intake plus synthesis. When such measurements are made often less than half the dietary intake of taurine is recovered in faeces and urine. The question arises – what happens to the taurine not accounted for in the balance measurements? Taurine is readily degraded by bacteria, particularly anaerobes. An anaerobe has been identified which is capable of using taurine as the sole carbon and nitrogen source (Ishimoto and others 1983). Bacterial overgrowth in the gut of humans has been associated with reduced levels of taurine in plasma, and rats with surgically created self-filling blind loops of the gut have been shown to have lower than normal plasma and retinal taurine concentrations, and visual dysfunction as indicated by electroretinography (Sheikh 1981). Incubation of taurine with faecal material from cats, under anaerobic conditions in vitro, causes extensive degradation of taurine. Measurements have not been made on the extent of taurine degradation in the gut of cats.

Another line of evidence supporting the importance of bacterial degradation comes from measurements on cats that have been given a pulse labelled dose of ^{14}C taurine in the diet. Up to 10 per cent of the label has been recovered as $^{14}CO_2$ (Hickman and others unpublished). These data suggests that a major, if not the primary reason for taurine being an essential nutrient for the cat, is the obligatory secretion in bile salts, which are deconjugated by intestinal bacteria with the subsequent degradation of the taurine.

As a deficiency of an essential nutrient occurs when the rate of loss exceeds the combined rates of synthesis and ingestion, many authors have suggested that a low rate of de novo synthesis by cats is the major factor rendering taurine to be an essential dietary constituent. The major site of taurine synthesis in most animals is probably the liver, but enzymes of the synthetic pathway, such as cysteine sulphinic acid decarboxylase, have been demonstrated in other tissues, such as brain and retina. While synthesis by the extrahepatic tissues may be important for those tissues, the liver appears to be the major site of synthesis for the body as a whole.

Several possible pathways have been proposed for taurine synthesis; the most widely accepted one is shown in Fig 1. As L-cysteine from dietary sulphur amino acids is the precursor of taurine in these pathways, the intake of sulphur amino acids should effect the magnitude of the precursor pool for taurine synthesis. We have shown, in long term experiments, that the concentration of

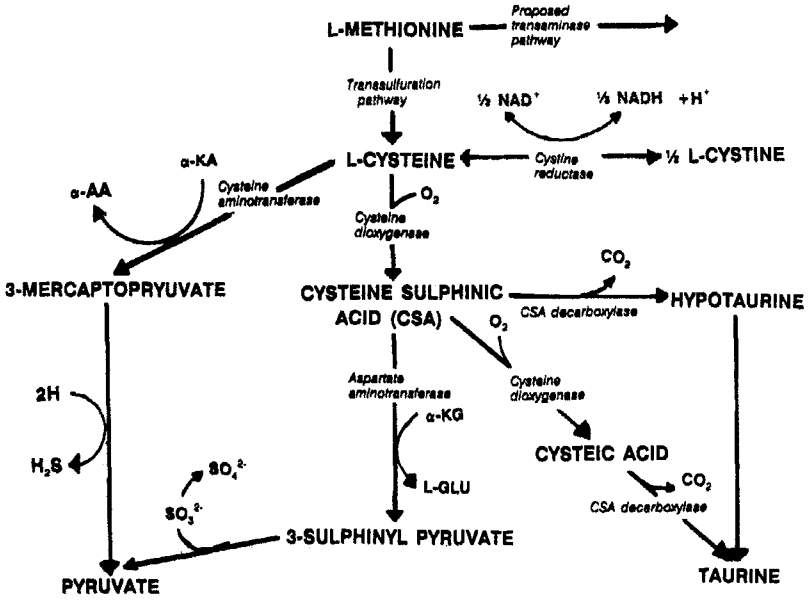

FIG 1. Alternate pathways of cysteine metabolism in cat liver. Note that taurine synthesis via cysteine sulphinic acid is only one of the possible pathways, higher fluxes in competitive pathways leading to pyruvate synthesis could provide the basis for taurine being an essential dietary requirement of cats. The cysteamine pathway of taurine synthesis appears to be insignificant in cats

taurine in the plasma of cats is elevated when the dietary concentrations of total sulphur amino acids is increased from 9 to 17 g/kg diet, irrespective of dietary taurine in the range of 0 to 500 mg/kg diet. Therefore, the intake of sulphur amino acids is an important factor affecting the rate of endogenous synthesis of taurine by cats.

While cysteine is the precursor for taurine synthesis, and cysteine is produced when methionine is metabolised by the transsulphuration pathway, the carbon in cysteine (and taurine) is not derived from methionine. In the metabolism of methionine via homocysteine, the sulphur of methionine is transferred to serine, to form cysteine. As the carbon in serine can come from non-amino acid precursors, radioactive taurine can be synthesised in animals following the administration of carbon-labelled carbohydrate precursors.

The major pathway of taurine synthesis in cat liver is postulated to involve the oxidation of cysteine to cysteine sulphinic acid (CSA). CSA may be either decarboxylated, transaminated or oxidised. Decarboxylation leads to hypotaurine, which can be further oxidised to taurine. Transamination of CSA results in 3-sulphinylpyruvate which spontaneously decomposes to pyruvate and sulphite. Oxidation of CSA produces cysteic acid which then may be decarboxylated to taurine. The predominant pathway may depend on the tissue involved. The apparent relationship between hepatic CSA decarboxylase activity and the capacity of animals to synthesise taurine supported the view that the CSA pathway is the major route of synthesis. Few measurements have been made of the activity of the enzymes in the pathways of taurine synthesis in tissues of cats. Hardison and others (1977) were unable to demonstrate that the liver from cats was capable of producing CSA, but demonstrated synthesis in rat liver. In the liver and brain of rats, CSA and cysteic acid are decarboxylated by the same enzyme, CSA decarboxylase or cysteate decarboxylase (Oja and Kontro 1982). Large variation in activity of these enzymes has been observed in the liver of various species. It has been suggested that the CSA decarboxylase activity may be the rate limiting step in the synthesis of taurine by the liver of cats. The activity of CSA decarboxylase in the liver of cats is low compared to rats. But, the activity of CSA decarboxylase in the liver of adult cats, reported by several investigators, is some 20-fold that in the liver of adult humans (Rassin 1982). We suggest that low activity of CSA decarboxylase may not be the only explanation for limited taurine synthesis in cats, but also high fluxes along alternate pathways of cysteine and, or, CSA metabolism which either do not lead to CSA production, or effectively maintain a low concentration of CSA in liver may limit taurine synthesis. Human vegans while maintaining significantly lower plasma concentrations of taurine than non-vegans, and excreting much less taurine in their urine than non-vegans (Laidlaw and others 1988), do not develop clinical signs of deficiency.

Taurine homoeostasis of cats depends on many factors including dietary intake, endogenous synthesis, turnover in the enterohepatic circulation, microbial degradation in the lower gut and urinary and faecal losses. Availability as applied to dietary taurine is not synonymous with availability of dietary α-amino acids or minerals. Some of the factors involved in taurine homoeostasis are shown in Fig 2.

TAURINE DEFICIENCY IN CATS

Hayes and others (1975) identified the first pathological condition in cats resulting from a deficiency of taurine. In the ensuing 15 years, a

further six abnormalities have been shown to result from an inadequate dietary intake of taurine which results in a low concentration in the tissues of the cat. These conditions can be conveniently classified in the following broad categories.

Feline central retinal degeneration (FCRD)

Attention was first drawn to this disease by Dr Patricia Scott, a pioneer in feline nutrition in the UK, in the course of her investigations into the vitamin A requirements of cats fed a diet based on casein as a protein source. Casein is virtually devoid of taurine, and also contains low concentrations of cysteine. A casein-based diet rapidly depleted cats of taurine and produced FCRD. Hayes and others (1975) also gave cats a casein-based diet and showed that feline central retinal degeneration was associated with a depletion of taurine in the retinal tissue. Lesions of taurine deficiency are visible on ophthalmoscopic examination. These lesions usually begin as small circumscribed areas in the area centralis, increase in size, become oval in shape and eventually involve the entire retina. Ultrastructural changes in the outer segments of the photoreceptor cells include vesiculation, disorientation and disintegration. In addition to retinal involvement, the rods of the tapetum lucidum which acts to reflect light back through the retina and increases retinal sensitivity to light are also affected by taurine deficiency. These changes collectively result in a diminished response of the retina to light as recorded by electroretinogram measurements.

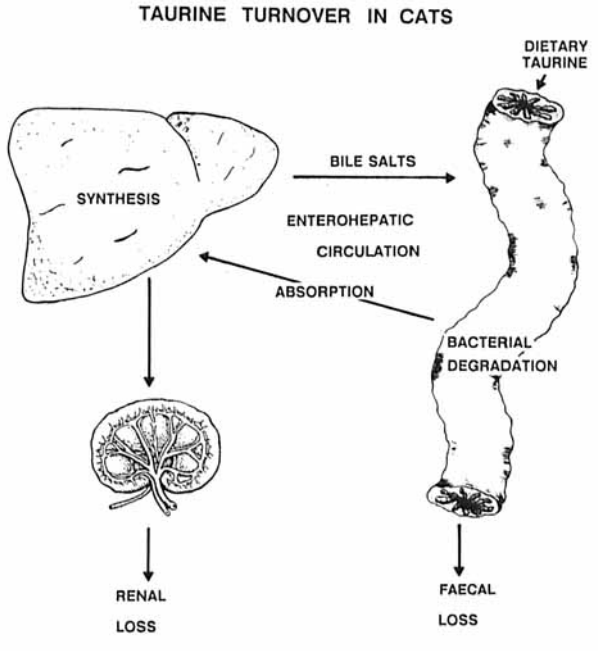

FIG 2. Taurine turnover in cats showing some of the factors affecting taurine status

Reproductive failure in the queen

Taurine deficiency has a profound effect on reproduction in the queen, leading to a reduction in the number of terminal pregnancies, number of live kittens born per pregnancy and decreased birthweight and survival of the kittens. In retrospect, poor reproductive performance in some catteries may have been caused by a taurine deficiency.

Sturman and others (1986) reported that of 18 queens given a taurine-free diet, 12 either resorbed or aborted their fetuses, whereas 17 of 18 given the same diet containing taurine reached full term. The taurine-deficient queens produced only 18 liveborn young of which eight survived, while the taurine-adequate queens produced 71 liveborn young of which 67 survived. Our experience has been that queens deficient in taurine, come into oestrus, mate with the tom and ovulate. Fertilisation and implantation appears to occur in a high percentage of matings. Pregnancy is maintained to approximately day 30, after which there is a high percentage of fetal abortions or resorptions. In a study at the University of California at Davis, Dieter and others (1988) reported that ovulation occurred in 29 out of 33 matings, in taurine deficient queens. Only 11 of the cats that ovulated (38 per cent) resulted in term deliveries, with a mean litter size of 2·7 kittens, of which only a mean of 2·1 were liveborn. Of the remaining queens that ovulated, 31 per cent had pregnancies that were aborted before term. In taurine-depleted queens that went to term, progesterone levels were consistently lower, but relaxin levels were similar to those in normal queens.

Development abnormalities in kittens

Sturman and others (1987) reported developmental abnormalities, such as severe hydrocephalus and anencephaly in aborted fetuses from queens given a taurine-deficient diet during pregnancy. The concentration of taurine in the brain of liveborn kittens to taurine-deficient queens was only 30 per cent of that in the brains of kittens born to taurine-supplemented queens. In stillborn kittens, the concentration of taurine in the brain was only about 15 per cent of that in kittens from queens given a taurine supplemented diet. Surviving kittens born to taurine-deficient queens exhibited abnormal development of the hindlimbs, a reduction in bulk of the limbs compared to age-matched controls and a gait characterised by excessive abduction and paresis. Kittens born to taurine-deficient queens also exhibited ventral curvature of the spine in the thoracic region, giving an appearance of a flattening of the thoracic cavity in the dorsoventral plane. Our experience indicates that the severity

of the curvature decreases with the age of the kitten and may not be apparent in the adult.

Histological abnormalities of the brains of kittens born to taurine-deficient queens is indicative that a deficiency of taurine causes delayed maturation of the brain. However, ophthalmoscopic examination of kittens of up to eight weeks of age from depleted queens, showed no gross lesions. Ultrastructural changes were apparent in the retina and tapetum (Imaki and others 1986).

Delayed growth in kittens

Taurine appears to be required for normal growth of kittens in the immediate post natal period. Sturman and others (1985) presented growth data for eight kittens born to queens given a taurine-free diet for eight weeks after birth. The bodyweight of these kittens was about 600 g compared to about 1000 g in control kittens. Taurine may be acting directly as a nutrient or enhancing utilisation of the fat in the milk by the kittens. Wang and others (1989) reported that supplementation of a kitten milk formula with a bile-salt-activated lipase, normally present in cat milk, doubled the growth rate of pre-weaned kittens. We have not found any significant effect of the level of taurine in the diet on the bodyweight gain of kittens following weaning.

Compromised immune function

Schuller-Levis and Sturman (1988) reported that taurine deficiency in cats results in a decreased number of total white cells ($9{\cdot}3 \pm 3{\cdot}4 \times 10^6$ vs $18{\cdot}2 \pm 4{\cdot}2 \cdot \times 10^6$), a shift in the polymorphonuclear and mononuclear cells, and a change in the sedimentation rate. Studies on polymorphonuclear cells isolated from taurine-deficient cats showed a significant decrease in zymosan induced peroxide production and a decreased phagocytosis and intracellular killing of *Streptococcus epidermis*, compared to cells from cats fed the same diet containing taurine. Serum gamma globulin in cats fed a taurine-free diet was also less than that in cats fed the same diet plus taurine.

Dilated cardiomyopathy

The association between low concentrations of taurine in the plasma of clinic cats (referred to the Veterinary Medical Teaching Hospital at the University of California at Davis), and cats receiving purified diets and dilated cardiomyopathy was reported by Pion and others (1987). The supplementation of the diet of these cats with taurine demonstrated that the cardiomyopathy was reversible (see the paper by Pion and Kittleson also in this Symposium publication).

Hearing loss in kittens

Davies and others (1989) reported that cats which have been depleted of taurine have reduced auditory brain stem evoked potentials indicating hearing loss. Similar responses have been reported in ferrets given ß-alanine to deplete body stores of taurine. Supplementation of the formula for low birthweight human infants with taurine has been reported to increase the rate of maturation of auditory evoked responses (Tyson and others 1989). Clinical cases of hearing loss in cats due to taurine deficiency have not been reported, however, this probably arises from the general lack of auditory testing of clinic animals.

ASSESSMENT OF TAURINE STATUS

As a deficiency of taurine has been shown to be responsible for a range of pathological states in cats, it is obvious that some clinical measure to assess the taurine status of a cat would be highly desirable. An ideal index of taurine adequacy would be the concentration of intracellular taurine in the tissue of interest. As a biopsy is impractical in most clinical situations, the concentration of taurine in plasma has generally been used as an index of taurine status. However, there are problems associated with the use of plasma to predict taurine status.

(1) Taurine in human blood is concentrated in the formed elements (Vinton and others 1987) particularly the granulocytes and platelets. Leakage of taurine from these formed elements into the plasma will inflate the observed value in plasma. Laidlaw and others (1987) reported concentrations of taurine in plasma, platelets, granulocytes, lymphocytes and erythrocytes of cats, but the different units used to express the concentration precluded calculation of taurine distribution in cat blood. However, the distribution of taurine in whole blood of children can be calculated from the values of Vinton and others (1987) and is shown in Table 1. Of the total taurine in whole blood, 0·83 is in the formed elements and only 0·17 is in plasma. Platelets contain the largest proportion of the taurine in whole blood followed by granulocytes, and both collectively account for nearly two-thirds of the total taurine in blood.

When blood is collected and allowed to stand at room temperature, taurine in the formed elements leaks into plasma and increases the apparent concentration. In our experience, placing samples of whole blood on ice immediately following collection gives slightly higher values than immediate separation of the plasma, presumably due to cold shock of the platelets.

Table 1. Taurine distribution in whole blood of children*

Constituent	Taurine concentration nmol/10^9 cells	Cells/litre whole blood	µmol/ litre	Fraction
Platelets	294	245·0 × 10^9	72·0	0·373
Lymphocytes	4845	3·6 × 10^9	17·4	0·091
Granulocytes	11,268	4·5 × 10^9	50·7	0·264
Erythrocytes	4·2	4·6 × 10^{12}	19·3	0·100
Plasma	52 µmol/ litre	630 ml	32·8	0·171
Total			192·2	1·000

*Calculated from taurine concentrations (Vinton and others 1987) and normal values for blood of children (Jandl 1987)

However, values obtained by this method are stable if the plasma is not separated from the cells for some hours, and we regard this as the procedure of choice when a number of blood samples are to be collected at one time. Alternatively, the blood cells can be separated from the plasma immediately after collection without cooling. As the platelets contain a high concentration of taurine, and sediment later than the erythrocytes, centrifugation should ensure sedimentation of all platelets from the plasma. Care should be taken not to include any of the 'buffy' layer in the plasma sample.

(2) The concentration of taurine in plasma can change markedly with time following food restriction for cats fed certain types of diets. Therefore, sampling procedures for cats fed these diets needs to be standarised in relation to feeding. When cats are given canned diets containing high levels of taurine there is a post prandial increase in the concentration in the plasma. As the kidney tubules of cats are capable of up and down regulation depending on taurine intake, cats given diets containing high levels will show a marked decrease in plasma taurine concentration on withholding of food. Whole blood changes far less, on a percentage basis, than plasma (Fig 3).

(3) The pattern of depletion and repletion in plasma does not immediately reflect changes in the whole blood or muscle concentrations of taurine. Fig 4 shows the depletion and repletion curves for lysed whole blood and plasma from cats given either a taurine-free diet or a diet containing 1·5 g taurine/kg. During depletion, the concentration of taurine in plasma fell more rapidly than that in whole blood, then rose more slowly than whole blood when a diet adequate in taurine was fed. Taurine concentration measured on biopsy samples of muscle more closely followed lysed whole blood than plasma taurine concentrations.

The relationship between whole blood and plasma taurine concentration, in cats given one of three commercial diets that contain an adequate level of taurine, is shown in Fig 5.

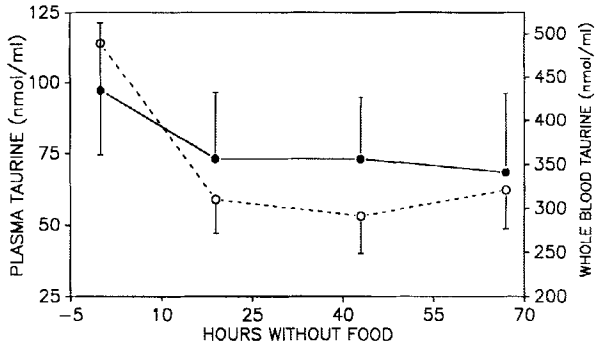

FIG 3. Changes in whole blood and plasma concentrations of taurine in cats, in relation to time of food deprivation. Whole blood shown as closed circles and solid line, plasma as open circles and dashed line

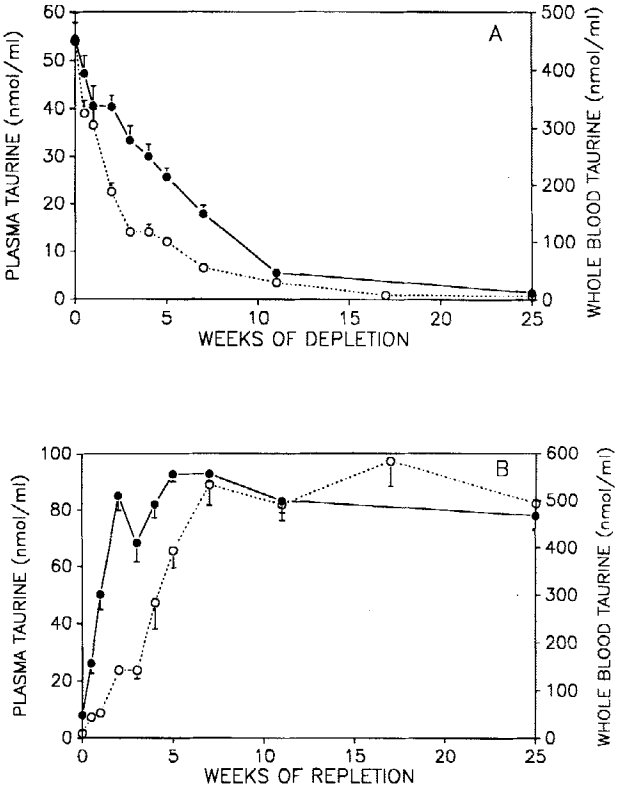

FIG 4. Changes in whole blood and plasma concentrations of taurine in cats. (A) During taurine depletion. (B) During taurine repletion. Whole blood shown as closed circles and solid line and plasma as open circles and dashed line

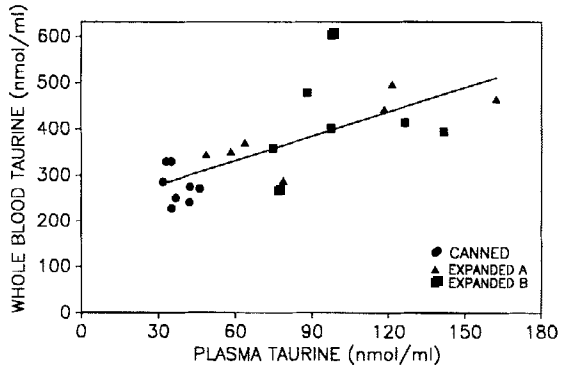

FIG 5. Relationship between whole blood and plasma concentrations of taurine of individual cats given one of three commercial diets

Whole blood concentration of taurine appears to be a superior index of taurine status than the concentration in plasma. Unfortunately, there are few data available on whole blood concentrations of taurine in taurine-adequate and taurine-deficient cats. It would appear that a concentration greater than 250 nmol/ml would indicate a satisfactory taurine status.

ADEQUACY OF DIETS

Veterinarians, nutritionists and pet food manufacturers who formulate diets for cats need to know the concentration of taurine in diets that will maintain levels in the tissues of cats to prevent diseases associated with deficiency. Taurine in diets is normally assayed by extraction of the diet with water or dilute acid. Therefore, the availability in the classical sense of taurine assayed by this method should approach 100 per cent. However, diets containing similar concentrations of taurine by analysis, support vastly different plasma levels of taurine. For example when canned and dry (expanded) diets containing similar concentrations of taurine are compared, expanded diets support much higher concentrations of taurine in plasma than canned diets.

Cats given the raw unprocessed ingredients of a canned diet also maintain higher concentrations of taurine than cats given the same diet after standard thermal processing (Cooke and others, unpublished data). As the taurine recovered by water extraction from the processed diet equals that before processing, the problem is not one of destruction or binding in processing. It appears that a substance produced during the canning process prevents absorption or increase microbial degradation of taurine in the lower intestine. The addition of glucose to the raw material before thermal processing results in a diet that causes a more rapid rate of decline in plasma concentration of taurine than when starch was added to the diet. One possible candidate, formed by the reaction of reducing sugars and protein in the presence of high moisture and high temperatures, are Maillard reaction products. These or other products formed may bind bile salts, and be responsible for preventing absorption of taurine of dietary or enterohepatic origin. Alternatively, the reaction products may support an anaerobic bacterial population more active in the fermentation of taurine in the lower gut. As estimates of the bile salts turnover in humans are in the range of five to 15 times a day, even a small change in the proportion of taurine degraded during each cycle of the enterohepatic circulation would result in a substantial loss of taurine from the total pool.

While a recommendation of a definitive minimal dietary concentration is not possible at this time, it would appear that concentrations of 1200 mg taurine/kg dry matter in expanded diets and 2500 mg taurine/kg dry matter in canned diets, are adequate under all situations examined to date.

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ABSTRACTS

Epizootiological association between feline immunodeficiency virus infection and feline leukaemia virus seropositivity

AN ELISA revealed that the prevalence of feline immunodeficiency virus (FIV) among a population of 521 cats was 11·3 per cent. The results of an ELISA for feline leukaemia virus (FeLV) was available for 156 of these cats and revealed that there was a significant association (P=0·008) between FIV infection and FeLV seropositivity. FeLV-positive cats were nearly four times more likely to be seropositive for FIV than FeLV-negative cats. The association remained significant (P=0·021) after the data had been adjusted for the effects of age and gender by using multiple regression analysis. The FIV-infected cats were older than the non-infected cats, and more of them were male.

COHEN, N. D., CARTER, C. N., THOMAS, M. A., LESTER, T. L. & EUGSTER, A. K. (1990) *Journal of the American Veterinary Medical Association* **197**, 220

Tick parasitism and antibodies to *Borrelia burgdorferi* in cats

TWENTY-TWO of 93 cats living in tick-infested areas of Connecticut and New York had one or more motile stages of the tick *Ixodes dammini* attached to them, and of the two larvae and 20 nymphs removed one larva and two nymphs were infected with the spirochaete *Borrelia burgdorferi*. Analyses by indirect immunofluorescent antibody staining or ELISA showed that 10 of 71 samples of serum obtained from the cats contained antibodies to the spirochaete, with maximum antibody titres of 1:256 and 1:2560, respectively. These titres are lower than those previously recorded in dogs and horses. In clinical studies of 30 of the cats there were nearly equal proportions of seropositive cats with limb or joint disorders which were not accompanied by fever, anorexia or fatigue (five of 21 cats) and cats which had these signs of illness but were not lame (two of nine cats).

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