DIETARY TAURINE REQUIREMENT OF CATS IS DETERMINED BY MICROBIAL DEGRADATION OF TAURINE IN THE GUT

James G. Morris, Quinton R. Rogers,
Seungwook W. Kim, and Robert C. Backus

Department of Molecular Biosciences
School of Veterinary Medicine
University of California, Davis CA 95616

INTRODUCTION

Taurine is an essential dietary constituent for cats because in vivo synthesis is limited, and cats have an obligatory requirement for taurine for the conjugation of bile salts. Dogs and cats use only taurine to conjugate bile acids, but dogs unlike cats, appear to have a rate of synthesis adequate to meet their needs. The basis for the lower synthetic capacity in cats than in dogs and other animals has not been fully elucidated. The activity of cysteine dioxygenase is low in the liver of cats which results in limited production of cysteine sulfinate and synthesis of taurine. Other contributing factors are the low activity of cysteine sulfinate decarboxylase and the high activity of aspartate aminotransferase which transaminates cysteine sulfinate to pyruvate, rather than allowing it to be decarboxylated to hypotaurine and hence to taurine (29).

Taurine is essential for the maintenance of normal function of the visual, auditory, cardiac and reproductive systems of cats, as well as for development (32,37,39). The concentration of taurine in the diet that will maintain a eutaurine status is dependent on the type of diet. For expanded dry diets, a concentration of 1000 mg taurine/kg diet is adequate, whereas for canned diets concentrations as high as 2500 mg taurine/kg dry matter are required (5,30). Taurine in both the canned and expanded diets is in the free state and can be extracted by water, so the difference between diets is not one of indigestibility of taurine rendering it unavailable for absorption. This paper will focus on the role of the gastrointestinal tract and dietary components, in particular protein, which have a dominant role in determining the utilization of taurine in a diet, and hence the dietary requirement of cats for taurine.

R. J. Huxtable et al. (eds.), *Taurine in Health and Disease*

TAURINE BALANCE

As taurine is not degraded by mammalian enzymes, taurine balance of cats at equilibrium might be expected to be zero; that is, the taurine in feces and urine should be equal to taurine ingested and synthesized. When taurine balances measurements are taken on cats given diets containing fixed amounts of taurine for long periods, less than half the dietary taurine is recovered in the feces and urine. Examples of such balance studies are shown in Tables 1a and b. The proportion of ingested taurine recovered in the urine and feces varied from about half (0.55) for a purified diet based on casein, to only 0.07 to 0.08 for the commercial diets. The partition of the excreted taurine between urine and feces indicated that diets with the highest recoveries had the greatest urinary taurine excretions, whereas those with the lowest recoveries had the lowest urine taurine excretion.

Table 1a. Taurine balance of cats given two commercial canned diets.

	Food A	Food B
	Taurine (mg/day)	
Intake	85.8 ± 8.7	56.9 ± 2.4
Excretion		
Urine	30.9 ± 8.0	9.8 ± 4.0
Feces	11.1 ± 7.8	7.3 ± 1.7
Recovery (feces + urine)	40.2 ± 8.0	17.2 ± 3.0
Proportion recovered	0.47	0.30

From reference (3). Data are means ± SEM.

Table 1b. Taurine balance of four groups of cats given either one of two purified diets or a commercial diet before and after thermal processing

	Casein purified	Soy purified	Cooked commercial	Frozen commercial
	Taurine (mg/day)			
Intake	60.7 ± 12.0	52.0 ± 13.0	101 ± 8.4	104 ± 14.3
Excretion				
Urine	33.1 ± 9.1	3.2 ± 1.0	1.8 ± 0.4	4.0 ± 0.9
Feces	1.5 ± 0.9	2.7 ± 1.0	6.2 ± 2.1	2.7 ± 0.9
Recovery (feces + urine)	34.7 ± 9.5	5.9 ± 1.7	8.0 ± 2.3	6.7 ± 0.9
Proportion recovered	0.55 ± 0.08	0.13 ± 0.04	0.08 ± 0.02	0.07 ± 0.00

All diets contained 1.5 g taurine/kg dry matter. Data are means ± SEM.

In contrast to animals, some microbes are able to degrade taurine. Kondo and associates (23) reported that extracts from a bacterium able to grow on taurine as the sole source of carbon, nitrogen and sulfur converted taurine to sulfoacetaldehyde (HO_3S-CH_2-CHO). Two organisms with taurine aminotransferases have been studied. A taurine:α-ketoglutarate aminotransferase (EC 2.6.1.55) has been isolated and characterized from *Achromobacter superficialis* (40). This enzyme catalyzes the transamination of taurine with α-ketoglutarate

to yield sulfoacetaldehyde and L-glutamate. A strain of *Pseudomonas aeruginosa* TAU-5 has a taurine:pyruvate aminotransferase that catalyses the production of alanine and sulfoacetaldehyde. The latter then undergoes a lyase reaction to produce sulfite and acetate (33). The sulfite is presumably oxidized and excreted as sulfate (38). Sulfoacetaldehyde may also be metabolized to isethionate (7).

When a pulse dose of ^{14}C labeled taurine was given to cats and the expired CO_2 collected and analyzed, it was found that 100 times more label was recovered in the CO_2 from cats given a thermally processed canned diet (that caused taurine depletion) than from cats given the same diet in the unprocessed state (16). These observation indicated that processing a diet had a marked effect on the extent of taurine degradation in the gut. It was not possible from these observations to quantify the taurine degraded. $^{14}CO_2$ could have come from CO_2 produced by the microbes directly or from oxidation of products of taurine degradation such as acetate which enters the body pool and may only be partially oxidized in the period of observation.

Similar overall recoveries of taurine from purified and cooked and frozen diets are presented in Table 1b. However, these diets supported very different blood concentrations of taurine. The whole blood taurine concentration of cats given the four diets are presented in Table 2. While three of the diets (two purified and frozen commercial) produced only a slight fall in taurine concentration in the blood, the cooked diet resulted in marked depletion in whole blood taurine concentration. These results indicate that the cooked diet, relative to uncooked or purified diets, was associated with a greater degree of degradation of taurine in the gut.

Table 2. Concentration of taurine (μmol/liter) in the whole blood from groups of cats given purified diets containing different protein sources or a commercial diet before and after thermal processing

Sampling time	Casein purified	Soy purified	Cooked commercial	Frozen commercial
Initial	554 ± 47	521 ± 23	448 ± 50	446 ± 67
After 42 days	518 ± 62	493 ± 1	186 ± 34	387 ± 38
Difference	28 ± 36	28 ± 31	261 ± 28*	58 ± 44

All diets contained 1.5 g taurine/kg dry matter. Data are means ± SEM. *Significantly different from other 3 groups ($p < 0.05$).

Binding of bile salts to indigestible dietary constituents was considered as a possible mechanism whereby some diets deplete cats of taurine. Anion exchange resins such as Cholestyramine® or Colestipol® bind bile acids (24) in the intestinal lumen and have been used as hypocholestrolemic agents in humans (13). When these resins are included in the diet of cats, rapid depletion of taurine occurs, as the resins bind taurocholate in the lumen of the intestine, prevent it participating in the enterohepatic recycling, and resulting in its excretion in the feces.

The effect of the inclusion of 20 g of Cholestyramine®/kg dry matter in a purified diet containing 1500 mg taurine/kg dry matter is shown in Figure 1. Both plasma and whole blood concentrations of taurine declined rapidly in the group receiving the resin. Supplementation with additional taurine to bring the total concentration to 3500 mg taurine/kg diet overcame the effect of the resin on whole blood and plasma taurine concentrations. Bile salt turn over time in humans is shortened when Cholestyramine® is included in the diet.

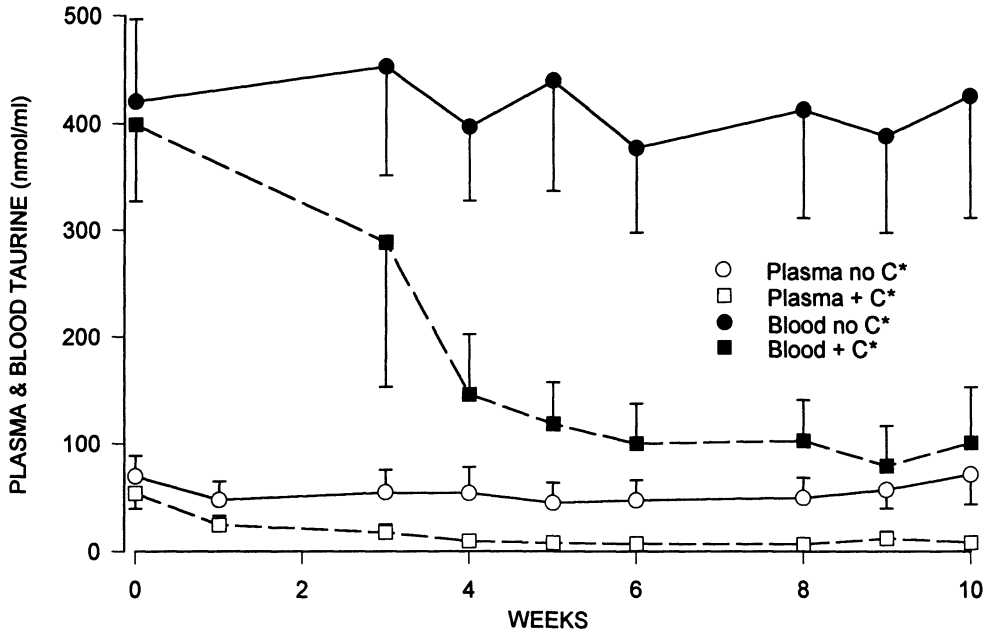

Figure 1 Changes in plasma and whole blood concentrations of taurine in cats given a purified diet containing 1500 mg of taurine/kg dry matter with and without 20 g Cholestyramine®/kg diet. C* in legend = Cholestyramine®. (Morris, M.L., Rogers, Q.R and Morris, J.G. unpublished data).

EFFECT OF LEVEL AND SOURCE OF PROTEIN IN THE DIET ON TAURINE STATUS OF CATS

When cats are given expanded (dry) diets and canned diets containing similar concentrations of taurine on a dry matter basis, those cats receiving the expanded diets consistently maintain higher concentrations of taurine in plasma and whole blood than cats given the canned diets. As canned diets generally contain higher levels of protein and fat than expanded diets, and the protein is of different origin, the effect of level and source of protein in the diet on the taurine status of cats was examined. We had previously reported that the level and source of fat in the diet had only a minor effect on the concentration of taurine in plasma (4). Two experiments were conducted using purified diets containing 1000 mg taurine/kg diet dry matter and different sources and levels of protein. In the first experiment, the proteins examined were isolated soy protein versus casein. Two groups each of 4 mature cats were given purified diets containing 100 mg taurine and either 250 or 500 g casein/kg of dry matter. It can be seen from Table 3 that after 6 weeks there were no significant differences in plasma concentration of taurine from initial values or between the two groups, although the group of cats given the 500 g casein/kg diet had a slightly lower plasma concentration. The protein source of the diet was then changed from casein to isolated soy protein maintaining the same levels of protein in the diet. There was a marked decline in the concentration of taurine in the plasma of the cats given the 500 g soy protein/kg diet whereas the concentration in the plasma of the group given the 250 g soy protein /kg diet declined only slightly.

In a similar experiment, five groups each of 6 mature cats (3 males and 3 females) were given purified diets containing either a low level of protein (300 g casein/kg diet dry

Table 3. Plasma concentrations of taurine (μmol/liter) in cats given diets containing casein for 0 to 6 weeks then isolated soy protein from 6 to 12 weeks

	<-----Casein------>	<---Isolated soy protein--->	
Protein in diet (g/kg)	Initial	6 weeks	12 weeks
	Plasma taurine (μmol/liter)		
250	91.4 ± 15.2	84.1 ± 10.3	71.0 ± 11.8
500	101.5 ± 14.3	73.7 ± 12.3	22.3 ± 3.8*

Data are means ± SEM. *Significantly different from 12 week value for 250 g/kg diet.

matter with either 0, 20 or 40 g of soybean cotyledon fiber) or a high level of protein (600 g of casein or isolated soybean protein/kg diet dry matter) for 14 weeks. Food intake was controlled to 26 g dry matter/kg body weight/day to equalize taurine intakes. Cats given the low casein diet had the highest concentration of taurine in plasma (86.8 ± 7.1 μmol/liter), which was significantly greater than cats receiving the high casein diet (63.9 ± 7.2 μmol/liter) which in turn was significantly greater than cats given the high soy group (34.2 ± 2.6 μmol/liter). Whole blood concentrations of taurine were not significantly different.

The addition of soy cotyledon fiber (Fibrim®), which had been shown to have a hypocholestrolemic effect and to increase fecal bulk in humans (28,34), had no significant effect on plasma taurine concentration. These levels of fiber would include those present in normal cat foods, other than low energy cat foods. This finding is in agreement with another report where soluble fiber was not found to have an effect on plasma taurine concentration of cats (1).

When the results were analyzed for the effect of sex on plasma taurine no sex effect was present at zero time. However, by 14 weeks there was a significant ($P< 0.05$) sex effect, males having a higher plasma concentration of taurine than females. These results indicate the need for care in the design of experiments involving cats of different sexes.

The results from the above experiments demonstrated a marked effect of the source and level of protein in the diet on the taurine status of cats. Three hypotheses were advanced to explain the protein effect. Certain proteins (a) modify the microflora in the gut resulting in greater degradation of taurine, or (b) bind taurine or taurocholic acid and render it unavailable for absorption, or (c) cause greater production of bile acids through stimulation of cholecystokinin release. In order to test the first hypothesis the following experiment was undertaken. Two groups of mature cats were given a heat-processed canned diet containing 1500 mg taurine/kg diet dry matter. This diet was known to cause depletion of taurine in cats. The food intake of both groups was restricted to 18 g dry matter/kg body weight /day to equalize the intake of taurine. One group of cats had 50 mg of tetracycline and 25 mg of procaine penicillin G added daily to the diet, the other group received no antibiotics. After 5 weeks the treatments were crossed over. Plasma and whole blood concentrations of taurine at the initial, 5 week (cross-over) and final 10 week samplings are shown in Table 4. In the first phase of the crossover, plasma taurine concentration declined more rapidly in the cats not receiving antibiotics than those receiving antibiotics. Within one week after the cross-over, the antibiotic treatment resulted in an increase in plasma taurine concentration which continued until 10 weeks. In contrast, the taurine concentration in the plasma of cats that had been receiving the antibiotic treatment declined more rapidly after the antibiotics were withdrawn.

The concentration of taurine in whole blood followed a similar response to plasma showing that there was a depletion of taurine in the formed elements of blood as well as in plasma. As whole blood and muscle taurine are correlated (6, 26) this decrease in whole blood taurine would indicate depletion of taurine in muscle, the largest pool of body

Table 4. Concentrations of taurine in plasma and whole blood of cats and urinary taurine excretion in a cross-over experiment with and without antibiotics added to the diet

Initial	Treatment	Cross-over (5 weeks)	Treatment	End (10 weeks)
		Plasma taurine (μmol/liter)		
116 ± 6	No antibiotics	26 ± 6	Antibiotics	67 ± 10
109 ± 6	Antibiotics	77 ± 7*	No antibiotics	16 ± 2*
		Whole blood taurine (μmol/liter)		
546 ± 8	No antibiotics	292 ± 30	Antibiotics	368 ± 21
559 ± 11	Antibiotics	431 ± 20*	No antibiotics	226 ± 11*
		Urinary taurine (μmol/day)		
365 ± 34	No antibiotics	54 ± 8	Antibiotics	95 ± 10
396 ± 23	Antibiotics	135± 19*	No antibiotics	35 ± 5*
		Fecal taurine (μmol/day)		
92 ± 10	No antibiotics	175 ± 15	Antibiotics	93 ± 6
88 ± 8	Antibiotics	53 ± 4*	No antibiotics	205 ± 8*

Data are means ± SEM. *Significantly different from equivalent value for no antibiotic group.

taurine. Urinary excretion of taurine by cats is an index of taurine adequacy (31). In both treatments, urinary excretion declined in the first 5 weeks, but the rate of decline was less in the group of cats receiving antibiotics. After the cross-over, the urinary excretion of cats receiving antibiotics increased, whereas the group now without antibiotics continued to decline. In contrast to urinary taurine excretion, the fecal excretion of taurine was greatest in cats not receiving antibiotics. Fecal excretion of bile acids by cats was also decreased by the antibiotic treatment (Table 5). The reason for the reduction in fecal taurine and bile acid excretion is not known. Possibly the lower activity of cholyltaurine hydrolase (Table 5) resulted in less taurocholate being cleaved and more absorbed.

This experiment highlights the importance of the microflora in determining the taurine status of cats. When microbial activity was inhibited by antibiotics there was a decreased cholyltaurine hydrolase activity in the feces, and presumably in the intestine, which decreased the rate of taurocholic acid hydrolysis and taurine degradation, and allowed more taurine to be recycled by the enterohepatic circulation. Greater recovery of taurine was reflected in the higher plasma and whole blood concentrations of taurine and in the greater urinary excretion of taurine in the antibiotic-treated cats.

Measurements of the half life of taurocholate and taurine in anaerobic cultures from the colon of cats indicates that hydrolysis proceeds at a faster rate than taurine degradation. The $t_{½}$ for taurocholate was 2.7 h compared to 15.5 h for taurine. Antibiotics have a profound effect on the activity of the cholyltaurine hydrolase which in turn probably reduces the quantity of taurine degraded in the intestine. Batt and co-workers (21) have reported that clinically healthy cats fed a commercial diet have high bacterial counts in the proximal small intestine at endoscopy. The numbers of bacteria in these cats fulfilled the established criteria for small intestinal bacterial overgrowth in man and dogs.

In another experiment 12 adult cats were given a purified diet containing 435 g casein and 800 mg taurine/kg. The cats were paired into two groups, one group of cats was given the diet ad libitum, while the other group had the food intake controlled to half that of the ad libitum pair mate. Food intake had no effect on plasma or whole blood taurine status of the cats. The concentration of taurine in the diet was then reduced to 500

Table 5. The effect of antibiotics in the diet on fecal excretion of bile acids and cholyltaurine hydrolase activity

Initial	Treatment	Cross-over (5 weeks)	Treatment	End (10 weeks)
		Fecal bile acids (μmol/day)		
122 ± 15	No antibiotics	235 ± 18	Antibiotics	184 ± 5
131 ± 9	Antibiotics	106 ± 11*	No antibiotics	358 ± 17*
		Cholyltaurine hydrolase activity[a]		
220 ± 38	No antibiotics	279 ± 54	Antibiotics	94 ± 19
191 ± 41	Antibiotics	42 ± 10*	No antibiotics	331 ± 29*

[a] nmol of cholic acid released/min/g dry feces. * Significantly different ($p < 0.05$) from equivalent value for no antibiotics group.

mg/kg diet which reduced plasma taurine concentration but there was no difference between treatments. These results support the thesis that the amount of taurine degraded in the gut is proportional to intake of food.

PROTEIN SOURCE AND EFFECT OF PROCESSING

The above studies indicated that the difference between diets in their ability to maintain the taurine status of cats was related to the source of protein in the diet and the type of processing it had undergone. Four groups of mature cats were given different purified nutritionally complete diets each containing 400 g casein and 800 mg taurine/kg. The diets were prepared by four processing methods:

(1) Unprocessed diet (control) containing starch as the carbohydrate source.
(2) Same diet as (1) + increased vitamins, moistened and autoclaved for 30 minutes.
(3) Same diet as (1) + increased vitamins except that 50 g glucose was substituted for starch in each kg of diet, moistened and autoclaved for 30 minutes.
(4) Casein component of the diet was separately autoclaved with glucose and the resulting brown material containing Maillard product was ground and added to the diet as the source of protein.

Each cat was individually housed and given 18 g dry diet/kg body weight to maintain a constant intake of taurine. Food refusals were negligible. After 5 weeks, antibiotics (50 mg of tetracycline and 25 mg of procaine penicillin G) were added daily to the diets for a further 5 weeks.

The effect of the dietary treatments on plasma, whole blood, fecal and urinary taurine is shown in Table 6. The results for weeks 0 to 5 show that processing the protein in a purified diet has a profound effect on the plasma and whole blood taurine concentrations of cats. The concentrations of taurine increased in the groups of cats given the unprocessed (control) and the autoclaved complete diet in which the carbohydrate was starch. This indicates that heat processing a purified diet without glucose (reducing sugar) has only a minor effect on taurine status. In contrast, the concentrations of taurine declined in plasma and in whole blood of cats given the diets autoclaved with glucose. The effect of autoclaving with glucose was independent of whether the complete diet was autoclaved, or only the casein and glucose were autoclaved separately to produce Maillard products. The difference in the response of plasma and whole blood taurine concentration of cats given the autoclaved starch and the autoclaved glucose-containing diets is presumed to be

a consequence of the Maillard products formed in both the glucose-containing diets. Reducing sugars in the presence of heat and moisture readily form Maillard products with proteins, especially those high in lysine such as casein. Maillard products are known to be of lower digestibility than untreated protein.

Table 6. Concentrations of taurine in plasma and whole blood from cats given purified diets containing 400 g casein and 800 mg taurine per kg

Method of processing	Initial <--no antibiotics-->	Week 5 <--antibiotics--> added	Week 10
	Plasma taurine concentration (μmol/liter)		
Control	78 ± 13	90 ± 6[a]	107 ± 5[a]
Starch autoclaved	80 ± 11	70 ± 11[a]	89 ± 4[b]
Glucose autoclaved	79 ± 6	36 ± 2[b]	84 ± 4[b]
Maillard product	78 ± 8	46 ± 5[b]	94 ± 4[a,b]
	Whole blood taurine concentration (μmol/liter)		
Control	509 ± 24	541 ± 25[a]	579 ± 14
Starch autoclaved	543 ± 34	524 ± 35[a]	580 ± 9
Glucose autoclaved	535 ± 41	357 ± 11[b]	556 ± 15
Maillard product	507 ± 12	368 ± 7[b]	577 ± 13

Means in the same column with different superscripts are significantly different ($P<0.05$). Each diet was processed by one of four methods. From week 0 to week 5 the diet did not contain antibiotics, whereas from week 5 to week 10 antibiotics were included.

The addition of antibiotics to these diets (weeks 5 to 10) restored the plasma and whole blood concentrations of taurine in the cats receiving the two diets containing casein autoclaved with glucose, indicating that the taurine-depleting mode of action of these casein-glucose diets had a large microbial component. Antibiotics also slightly increased plasma, but not whole blood, taurine of the control and autoclaved diet without added glucose.

When the diets did not contain antibiotics (weeks 0 to 5), fecal excretion of taurine by cats given the two glucose-containing diets was over twice that of cats given the control and autoclaved starch diet. The effect of the treatments on urinary taurine excretion was a mirror image of the fecal losses. As less taurine was lost in the feces from cats fed the control and starch autoclaved diets than the glucose-containing autoclaved diets, more was available for urinary excretion in these cats. When antibiotics were added to the diets, fecal excretion of taurine was greatly reduced to a level that was similar for cats in all treatments. Similarly, urinary output of taurine was enhanced by antibiotics, presumably reflecting the decreased degradation of taurine in the gut.

The cholyltaurine hydrolase activity of the feces of the cats (Table 7) show that activity was higher in the feces from cats receiving the glucose-containing diets than the control and starch autoclaved diet. The Maillard products supported either a greater microbial mass or changed the population to one with a greater cholyltaurine hydrolase activity. Antibiotics added to the diets caused a marked reduction in cholyltaurine hydrolase activity of the feces such that the activity was similar across all diets. The red-

Table 7. The effect of dietary processing on the cholyltaurine hydrolase activity in the feces and fecal bile acid output of cats given four casein containing diets with and without antibiotics

Method of processing	No antibiotics	Antibiotics
	Cholytaurine hydrolase (nmol cholate/min/g dry feces)	
Control	260 ± 34[a]	108 ± 33
Starch autoclaved	308 ± 23[a]	142 ± 37
Glucose autoclaved	520 ± 41[b]	125 ± 22
Maillard	568 ± 36[b]	149 ± 44
	Fecal bile acid excretion (μmol/day)	
Control	46 ± 4[a]	20 ± 4
Starc autoclaved	88 ± 2[b]	32 ± 7
Glucose autoclaved	120 ± 4[c]	44 ± 5
Maillard	124 ± 8[c]	38 ± 7

Means with different superscripts are significantly different ($P < 0.05$).

duction in activity was greatest for cats with the highest initial activity; that is, cats receiving the glucose-containing diets.

Fecal bile acid excretion of the cats not receiving antibiotics virtually doubled for those given the starch autoclaved versus control diet, and again increased when Maillard products were produced by autoclaving the complete diet or only the protein portion with glucose. It is not possible to distinguish which of the rate variables (bile secretion, absorption, or degradation) changed, but the Maillard products probably promoted a greater rate of bile secretion through greater stimulation of cholecystokinin release. Intact proteins are required for stimulation of cholecystokinin release in rats (25), but release in dogs is stimulated by a simple solution of two amino acids (phenylalanine and tryptophan) and fat. In rats, cholecystokinin release is inhibited by both the presence of pancreatic enzymes (12, 27) and presence of bile acids (11) in the intestine. As Maillard products are poorly digested, they would probably bind proteolytic enzymes and free cholecystokinin-releasing peptide (monitor peptide) (9) found in pancreatic juice to bind to sites in the duodenum and jejunum (41) and stimulate cholecystokinin release by interacting directly with the cholecystokinin-containing cells (26). If cholecystokinin had a major role, then maintenance of a greater difference between the control and heat processed diets would be expected when antibiotics were given.

The decreased total bile acid output in feces of cats given antibiotics is in agreement with the observed lower output in germ-free versus conventional rats (14). The half life of bile acids reported for germ-free rats is considerably longer (11 days) than for conventional rats (2 days) fed the same diet (14). Diet has a marked effect on bile acid excretion in germ-free as well a conventional rats. Fecal output of bile acids is increased two to three fold when the diet is changed from a purified to a rat chow diet (15).

Most values for urinary taurine excretion in the literature are for free taurine excretion. However, taurine in urine, as well as feces, is present both as free and as bound taurine. Total taurine (free + bound) may be measured after the sample has been digested for 12 h in 6N HCl. We have found variable proportions of free to bound taurine in the urine of cats given both commercial and purified diets. Across diets, the proportion of bound taurine ranged from 34 to 95% (5,16,17,18,19). The proportion bound did not appear to be related

to total taurine excretion as suggested by Glass et al. (10). These authors reported that, for cats given purified diets based on casein, increments of crystalline taurine added to the diet increased free taurine excretion while bound taurine remained relatively constant.

We have not investigated the nature of the taurine conjugates in cat urine or feces. Taurine is known to be a constituent of a number of small peptides such as gamma-glutamyl-taurine which acts as a hormone in the parathyroid gland (8). Quinaldylglycyltaurine, a metabolite of quinaldic acid and kynurenic acid has been identified in cat urine (22). When physiological doses of retinoic acid were administered to rats, 60% of the dose appeared in the bile as retinotaurine and 10% and 2% of the dose was excreted in urine and feces, respectively (35). It appears that retinotaurine is an excretory form of retinoic acid, as it has no biological activity in rats (36). However, if all the retinol ingested by cats was excreted as retinotaurine, it would not make a significant contribution to taurine loss.

Many animals use taurine to conjugate xenobiotics before excretion. As cats have a reduced ability to undertake the glucuronide conjugation of small phenols and aromatic acids, conjugation with taurine may be more extensively used by cats than other species to eliminate xenobiotics. Cats given phenylacetic acid excrete it in urine as a taurine conjugate (20) and taurine is used by rats, mice and ferrets to conjugate 2-naphthylacetic acid (2) before urinary excretion. Taurine is a major conjugator for phenylacetic acid in ferrets and birds, but strangely glycine, and not taurine, was reported to be its main conjugator found in cat urine.

CONCLUSIONS

Over half the dietary taurine requirements of adult cats is required to replace the taurine degraded by the intestinal microbes. The quantity of taurine degraded in the gut is primarily dependent on the source and amount of protein in the diet and the method by which the protein is processed. In purified diets, casein maintains higher concentrations of taurine in plasma than isonitrogenous levels of isolated soy protein. Addition of oral antibiotics to a taurine-depleting diet decreased taurine loss in feces, increased plasma and whole blood concentrations of taurine, and increased urinary taurine excretion compared to the diet without antibiotics. Current knowledge suggests that thermal processing in the presence of reducing sugars produces Maillard products which increases microbial degradation of taurine in the intestine. Maillard products may either provide an environment that favors higher numbers of taurine-degrading bacteria, or increase taurine exposure to bacteria. Maillard products may bind proteolytic enzymes in the intestine which permits monitor peptide to stimulate cholecystokinin-containing cells to release cholecystokinin, which in turn increases bile entry into the small intestine, and exposure of taurine to microbial degradation.

REFERENCES

1. Anantharaman, G., Vuichoud, J., Gicquello, P., and Rabot, R., 1992, The effect of dietary gelifying agents on bile acid excretion and taurine status in cats, *Veterinary International* 1:45-46.
2. Caldwell J., 1981, The current status of attempts to predict species differences in drug metabolism, *Drug Metabolism Reviews* 12:221-237.
3. Cooke, J.A., Rogers, Q.R., and Morris, J.G., 1988, Urinary and fecal excretion of taurine by cats fed commercial canned diets, *FASEB J.* 2: A1617 (abs).
4. O'Donnell J.A. Rogers, Q.R., and Morris, J.G., 1981, Effect of diet on plasma taurine in the cat, *J. Nutr.* 111:1111-1116.
5. Earl, K.E., and Smith, P.M., 1991, The effect of dietary taurine content on the plasma taurine concentration of the cat, *Brit. J. Nutr.* 66:227-235.

6. Edgar, S.E., Hickman, M.A., Marsden, M.M., Morris, J.G., and Rogers, Q.R., 1993, Dietary cysteic acid serves as a precursor of taurine in cats, *J. Nutr.,* (in press).
7. Fellman, J.H., Roth, E.S., and Fujita, T.S., 1978, Taurine is not metabolized to isethionate in mammalian tissue, *in:* "Taurine and Neurological Disorders", Barbeau, A., and Huxtable, R.J., eds., Raven Press, New York, pp. 19-24.
8. Feuer, L., Torok, L.J., Kapa, E., and Csaba, G., 1978, The effect of gamma-glutamyl-taurine (litoralon) on the amphibian metamorphosis, *Comp. Biochem. Physiol.* 61C:67-71.
9. Fushiki, T., Kajiura, H., Fukuoka, S-I., Kido, K., Semba, T., and Iwai, K., 1989, Evidence for an intraluminal monitor in rat pancreatic enzyme secretion: reconstitution of the pancreatic response with dietary protein, trypsin and the monitor peptide, *J. Nutr.* 119:622-627.
10. Glass, E.N., Odle, J., and Baker, D.H., 1992, Urinary taurine excretion as a function of taurine intake in adult cats, *J. Nutr.* 122: 1135-1142.
11. Gomez, G., Upp, J.R., Lluis, F., Alexander, R.W., Poston, G.J., Greeley, G.H., and Thompson, J.C., 1988, Regulation of the release of cholecystokinin by bile salts in dogs and humans, *Gastroenterology* 94:1036-1046.
12. Green, G.M., and Lyman, R.L., 1972, Feedback regulation of pancreatic enzyme secretion as a mechanism for trypsin inhibitor-induced hypersecretion in rats, *Proc. Soc. Exp. Biol. Med.,* 140:6-12.
13. Grundy S.M., 1972, Treatment of hyercholestrolemia by interference with bile acid metabolism, *Arch. Internal Medicine* 130:638-648.
14. Gustafsson, B.E. Bergström, S., Lindstedt, S., and Norman, A., 1957, Turnover and nature of fecal bile acids in germfree and infected rats fed cholic acid-24-^{14}C. Bile acids and steroids 4I, *Proc. Soc. Exp. Biol. Med.* 94:467-471.
15. Gustafsson, B.E., and Norman, A., 1969, Influence of the diet on the turnover of bile acids in germ-free and conventional rats, *Brit. J. Nutr.* 23:429-442.
16. Hickman, M.A., Rogers, Q.R., and Morris, J.G., 1990, Effect of processing on the fate of dietary [^{14}C]taurine in cats, *J. Nutr.* 120:995.
17. Hickman, M.A., Bruss, M.L., Morris, J.G., and Rogers, Q.R., 1992, Dietary protein source (Soybean vs. casein) and taurine status affect kinetics of the enterohepatic circulation of taurocholic acid in cats, *J. Nutr.* 122:1019-1028.
18. Hickman, M.A., Rogers, Q.R., and Morris, J.G., 1992, Taurine balance is different in cats fed purified and commercial diets, *J. Nutr.* 122:553-559.
19. Hickman. M.A., Morris, J.G., and Rogers, Q.R., 1992, Intestinal taurine and the enterohepatic circulation of taurocholic acid in the cat, *in:* "Taurine: Nutritional Value and Mechanisms of Action", Lombardini, J.B., Schaffer, S.W., and Azuma, J., eds., *Plenum Press, New York,* pp. 45-54.
20. James, M.O., Smith, R.L., Williams, R.T., and Reidenberg, M., 1972, The conjugation of phenylacetic acid in man, sub-human primates and some non-primate species, *Proc. Royal Soc. Lond. B* 182:25-35.
21. Johnston, K., Lamport, A., and Batt, R.M., 1993, An unexpected bacterial flora in the proximal small intestine of normal cats, *Vet. Rec.* 132:362-363.
22. Karhara, M., and Price, J.M., 1961, Quinaldylglycyltaurine: a urinary metabolite of quinaldic acid and kynurenic acid in the cat, *J. Biol. Chem.* 236: 508-511.
23. Kondo, H., Anada, A., Oshsawa, K., and Ishimoto, M., 1971, Formation of sulfoacetaldehyde from taurine in bacterial extracts, *J. Biochem.* 69:621-623.
24. Kritchevsky, D., and Story J.A., 1974, Binding of bile salts in vitro by nonnutritive fiber, *J. Nutr.* 104:458-462.
25. Liddle, R.A., Green, G.M., Conrad, C.K., and Williams, J.A., 1986, Proteins but not amino acids, carbohydrates, or fats stimulate cholecystokinin secretion in the rat, *Am. J. Physiol.* 251:G:243-248.
26. Liddle, R.A., Misukonis, M.A., Pacy, L., and Balber, A.E., 1992, Cholecystokinin cells purified by fluorescent-activated cell sorting respond to monitor peptide with an increase in intracellular calcium, *Proc. National Academy of Sciences,* USA 89:5147-5151.
27. Louie, D.S., May, D., Miller, P., and Owyang C., 1986, Cholecystokinin mediates feedback regulation of pancreatic enzyme secretion in rats, *Amer. J. Physiol.* 250:G252-259.
28. Lo, G.S., 1990, Physiological effects and physio-chemical properties of soy cotyledon fiber, *in:* "New Developments in Dietary Fiber", Furda, I., and Brine, C.J., eds., Plenum Press, New York.
29. Morris, J.G. and Rogers, Q.R., 1992, The metabolic basis for the taurine requirement of cats, *in:* "Taurine: Nutritional Value and Mechanisms of Action", Lombardini, J.B., Schaffer, S.W., and Azuma, J., eds., Plenum Press, New York, pp. 33-44.
30. Morris, J.G., Rogers, Q.R., and Pacioretty, L.M., 1990, Taurine: an essential nutrient for cats, *J. Small Anim. Prac.* 31:502-509.

31. Odle, J., Glass, E.N., Czarnecki-Maulden, G.L., and Baker, D.H., 1992, Urinary excretion of taurine as a function of taurine intake: Potential for estimating taurine bioavailability in the adult cat, *in:* "Taurine: Nutritional Value and Mechanisms of Action", Lombardini, J.B., Schaffer, S.W., and Azuma, J., eds,. *Plenum Press, New York, pp.* 55-62.
32. Pion, P.D., Kittleson, M.D., Rogers, Q.R., and Morris, J.G., 1987, Myocardial failure in cats associated with low plasma taurine: A reversible cardiomyopathy, *Science* 237:764-768.
33. Shimamoto, G., and Berk, R.S., 1990, Taurine catabolism. III Evidence for the participation of the glyoxylate cycle, *Biochim. Biophys. Acta* 632:399-407.
34. Shorey, R.L., Day, P.J., Willis, R.A., Lo, G.S., and Steinke, F.H., 1985, Effect of soybean polysaccharide on plasma lipids, *J. Am. Diet. Assoc.* 85:1461.
35. Skare, K.L., Schnoes, H.K., and DeLuca, H.F., 1982, Bilary metabolites of all-trans-retinoic acid in the rat:isolation and identification of a novel polar metabolite, *Biochemistry* 21:3308-3317.
36. Skare, K.L., Sietsema, W.K., and DeLuca, H.F., 1982, The biological activity of retinotaurine, *J. Nutr.* 112:1626-1630.
37. Sturman J.A., 1993, Taurine in development, *Physiol. Rev.* 73:119-147.
38. Sturman, J.A., Hepner, G.W., Hoffman, A.F., and Thomas, P.J., 1975, Metabolism of [^{35}S] taurine in man, *J. Nutr.* 105:1206-1214.
39. Sturman, J.A., Palachal, T., Imaki, H., Moretz, R.C., French, J., and Wisniewski, H.M., 1987, Nutritional taurine deficiency and feline pregnancy outcome, *in:* "The Biology of Taurine Methods and Mechanisms", Huxtable, R.J., Franconi, F., and Giotti, A., eds., Plenum Press, New York, pp.113-134.
40. Toyama, S., Misono, H., and Soda, K., 1978, Properties of taurine:α-ketoglutarate aminotransferase of *Achromobacter superficialis*. Inactivation and reactivation of the enzyme, *Biochem. Biophys. Acta* 523:75.
41. Yamanishi, R., Kotera, J., Fushiki, T., Soneda, T., Saitoh, T., Oomori, T., Satoh, T., and Sugimoto, E., 1993, A specific binding of the cholecystokinin-releasing peptide (monitor peptide) to isolated rat small-intestine cells, *Biochem. J.* 291:57-63.