The effect of dietary taurine content on the plasma taurine concentration of the cat

BY K. E. EARLE AND P. M. SMITH

Wulthani Centre for Pet Nutrition, Freeby Lane, Waltham on the Wolds,
Melton Mowbray, Leics LE14 4RT

(Received 20 April 1990 – Accepted 10 December 1990)

The essential role of taurine in the diet of the cat has been well documented and a deficiency of this nutrient is known to be responsible for a number of clinical conditions. At present, the National Research Council (1986) recommendation for the minimum dietary requirement of the kitten is 400 mg/kg dry matter and for a pregnant cat is 500 mg/kg dry matter. However, these minima were established by feeding the animals on semi-purified diets and, therefore, may be inappropriate for other dietary regimens. The aim of the present study was to investigate the effect of feeding a series of specially prepared diets containing different concentrations of taurine (canned diets 1475–5750 mg/kg dry matter, dry diets 811–1240 mg/kg dry matter) on the plasma taurine concentration of the cat. All diets were fed solus for 6 weeks and plasma taurine concentration was measured every 2 weeks. The results showed that to maintain plasma taurine values in the ‘normal’ range (>60 μmol/l), a canned diet must supply at least 39 mg taurine/kg body-weight per d and a dry diet at least 19 mg/kg body-weight per d; a cat fed on a semi-purified diet need only achieve a daily intake of at least 10 mg taurine/kg body-weight to maintain an adequate circulating level of taurine. The cause of the reduced availability of taurine from these diets is not yet known. Furthermore, repletion of plasma concentration above 60 μmol/l can be achieved within 2 weeks of feeding an adequate diet.

Plasma taurine: Cat nutrition: Dietary taurine.

The evidence for establishing the essential role of taurine in feline metabolism can be traced back more than 25 years (Scott et al. 1964) when it was reported that corneal and retinal lesions developed in cats fed on a vitamin A-deficient diet based on casein. Supplementing the diet with vitamin A did not prevent lesions but meat-based diets did alleviate the problem. Over the next 10 years various authors reported degenerative retinopathy in cats maintained on casein-based diets which could not be corrected with vitamin supplementation (Morris, 1965; Rabin et al. 1973; Hayes et al. 1975a). The first indication that taurine was the key nutrient in feline degenerative retinopathy came from a series of studies showing that normal retinal structure and function were maintained if the basal casein diet was supplemented with taurine (8 g/kg) or cats were fed on commercially-available dry cat foods containing 1 g taurine/kg (Hayes et al. 1975a; Berson et al. 1976; Schmidt et al. 1976; Aguirre, 1978).

In the early 1980s a number of studies were carried out to identify the cat’s minimum requirement for taurine (Burger & Barnett, 1982; Rogers & Morris, 1982) and the effect of various dietary sulphur amino acids on its synthesis (Knopf et al. 1978; Sturman et al. 1978). It was shown that there was limited synthesis of taurine in the cat and that there was a close relationship between dietary intake and circulating levels of taurine (Burger & Barnett, 1979; O’Donnell et al. 1981; Laidlaw et al. 1987). During this period it was also demonstrated that taurine was a key nutrient for normal reproductive performance of cats.
K. E. EARLE AND P. M. SMITH

(Sturman et al. 1985a, b, 1986, 1987). Cats fed on a taurine-deficient diet had a greater likelihood of foetal resorption early in the gestation period than cats fed on a diet containing taurine. Kittens produced by the taurine-deficient queens were of lower birth weight and had a reduced growth rate compared with the control group. In view of these findings the US National Research Council (1986) recommended a minimum requirement of 400 mg/kg dry matter (DM) in the diet for growth and maintenance and 500 mg taurine/kg DM for reproduction. However, these minima were derived from studies with semi-purified diets where taurine was assumed to have a high availability and, therefore, may not be applicable to other dietary regimens.

High concentrations of taurine can be measured in various tissues including the heart, retina, central nervous system and skeletal muscle, although they are not normally monitored to assess taurine status. Plasma taurine concentration has been measured routinely as it has been shown that, in the cat, plasma levels are sensitive to dietary intake (Hayes, 1988). The cat's propensity for developing a low plasma taurine concentration may be as a consequence of their limited biosynthesis of taurine and their total dependence on taurine to form bile acids. Several workers showed that plasma levels of greater than 40 μmol/l were indicative of normal retinal taurine accumulation (Schmidt et al. 1977; Barnett & Burger, 1980; O’Donnell et al. 1981). A number of workers demonstrated that cats having a plasma taurine concentration less than 20 μmol/l showed signs of feline degenerative retinopathy (Aguirre, 1978; Barnett & Burger, 1980) or dilated cardiomyopathy (Pion et al. 1987), or both. Values recorded for ‘normal’ cats differ but, in general, plasma taurine concentrations greater than 60 μmol/l were considered adequate (Pion et al. 1987; Monson, 1989). There was some indication that, for plasma taurine values to remain above 60 μmol/l, canned cat foods should contain at least 2000 mg taurine/kg DM and dry, extruded foods at least 1000 mg/kg DM (Pion et al. 1989), therefore identifying a reduced bioavailability of taurine from these foods as compared with a semi-purified diet. The aim of the present study was to investigate the effect of feeding a series of specially-prepared cat foods, for a period of 6 weeks, on the plasma taurine concentration of the cat.

METHODS

Three groups of seven British, domestic, short-haired, adults cats all born and reared at the Waltham Centre for Pet Nutrition were used in these studies. They had been vaccinated against feline infectious enteritis, feline calici virus and feline viral rhinotracheitis and had been reared on commercial cat food since weaning. All cats were given veterinary examinations at the beginning and end of each trial; no cats were observed to have signs of retinal degeneration throughout this period. The groups were fed to appetite on a total of fifteen diets, twelve of which were made from meat-based materials that had been canned and cooked to an Fo equal to 15, (heat processing equivalent to 121°C for 15 min), the other three were dry, extruded products. Plasma taurine concentration was measured on fasting blood samples at the beginning of trial (week 0) and every fortnight for a total of 8 weeks. All food and plasma samples were analysed for taurine by the methods described later pp. 228–229. After 6 weeks on the test diet, all cats were fed on the diet containing 3550 (SE 95.0) mg taurine/kg DM for 2 weeks to ensure repletion of plasma taurine. At the end of the 8 week period all cats were re-randomized between treatments before the next test.

Analysis

Taurine was analysed in blood samples taken from the cephalic vein of cats fasted for 16 h. Approximately 3 ml blood was collected in a heparinized tube and immediately centrifuged. An accurately weighed 1 ml sample of plasma (care taken not to remove buffy coat) was
### Table 1. List of dietary treatments for each of the three groups of cats, listed in chronological order

<table>
<thead>
<tr>
<th>Dietary taurine content† (mg/kg dry matter)</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1750</td>
<td>1875</td>
<td>1475</td>
<td></td>
</tr>
<tr>
<td>4500</td>
<td>3750</td>
<td>4425</td>
<td></td>
</tr>
<tr>
<td>1950</td>
<td>2500</td>
<td>3000</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2900</td>
<td>5750</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2325</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>967‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>811‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1240‡</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* There was always a period of at least 2 weeks repletion between each trial.
† The formulation of all foods was based on meat, poultry, offals, wheat flour and fortified with vitamins and minerals. Composition of diets; g/kg dry matter: crude protein 40–50, fat 25–40, carbohydrate 2–50 (higher levels of carbohydrate in the dry, extruded diets). The replenishing diet was of similar composition (crude protein 45.5, fat 26.8, carbohydrate 13-1 g/kg dry matter) and supplemented with taurine to a level known to overcome the poor availability of this nutrient, 3550 (SE 95.0) mg/kg dry matter.
‡ Dry, extruded diet.

### Table 2. Effect of dietary taurine content (mg/kg dry matter (DM)) on fasting plasma taurine concentration (μmol/l)

(Mean values with their standard errors for seven cats fed on canned diets)

<table>
<thead>
<tr>
<th>Dietary taurine† (mg/kg DM)</th>
<th>Taurine intake (mg/kg BW per d)</th>
<th>Period of feeding (weeks)</th>
<th>Mean</th>
<th>SE</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8†</th>
<th>SED</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>1475</td>
<td></td>
<td></td>
<td>23.7</td>
<td>1.5</td>
<td>99.9</td>
<td>32.6***</td>
<td>25.8***</td>
<td>25.1***</td>
<td>130.6</td>
<td>14.5</td>
<td>24</td>
</tr>
<tr>
<td>1750</td>
<td></td>
<td></td>
<td>31.5</td>
<td>3.3</td>
<td>78.0</td>
<td>65.9</td>
<td>61.3</td>
<td>73.8</td>
<td>152.8***</td>
<td>16.5</td>
<td>24</td>
</tr>
<tr>
<td>1875</td>
<td></td>
<td></td>
<td>31.3</td>
<td>0.5</td>
<td>84.6</td>
<td>78.3</td>
<td>80.6</td>
<td>35.8**</td>
<td>93.6</td>
<td>12.9</td>
<td>24</td>
</tr>
<tr>
<td>1950</td>
<td></td>
<td></td>
<td>20.4</td>
<td>0.3</td>
<td>92.6</td>
<td>70.9</td>
<td>73.6</td>
<td>54.8*</td>
<td>81.3</td>
<td>13.6</td>
<td>24</td>
</tr>
<tr>
<td>2325</td>
<td></td>
<td></td>
<td>60.1</td>
<td>1.6</td>
<td>75.6</td>
<td>80.0</td>
<td>98.6</td>
<td>98.6</td>
<td>93.8</td>
<td>10.4</td>
<td>24</td>
</tr>
<tr>
<td>2500</td>
<td></td>
<td></td>
<td>35.9</td>
<td>1.6</td>
<td>71.1</td>
<td>77.9</td>
<td>84.6</td>
<td>72.4</td>
<td>100.6</td>
<td>12.4</td>
<td>24</td>
</tr>
<tr>
<td>2900</td>
<td></td>
<td></td>
<td>53.0</td>
<td>2.1</td>
<td>82.9</td>
<td>70.0</td>
<td>172.0***</td>
<td>66.4</td>
<td>146.3***</td>
<td>15.7</td>
<td>24</td>
</tr>
<tr>
<td>3000</td>
<td></td>
<td></td>
<td>52.9</td>
<td>2.3</td>
<td>66.6</td>
<td>76.6</td>
<td>100.4***</td>
<td>61.6</td>
<td>104.3***</td>
<td>7.7</td>
<td>24</td>
</tr>
<tr>
<td>3750</td>
<td></td>
<td></td>
<td>63.1</td>
<td>1.0</td>
<td>101.0</td>
<td>252.6***</td>
<td>202.6***</td>
<td>129.7</td>
<td>77.3</td>
<td>23.2</td>
<td>23</td>
</tr>
<tr>
<td>4425</td>
<td></td>
<td></td>
<td>59.1</td>
<td>2.3</td>
<td>61.7</td>
<td>87.0</td>
<td>184.7***</td>
<td>132.7***</td>
<td>62.5</td>
<td>27.1</td>
<td>20</td>
</tr>
<tr>
<td>4500</td>
<td></td>
<td></td>
<td>59.1</td>
<td>2.3</td>
<td>61.7</td>
<td>87.0</td>
<td>184.7***</td>
<td>132.7***</td>
<td>62.5</td>
<td>27.1</td>
<td>20</td>
</tr>
<tr>
<td>5750</td>
<td></td>
<td></td>
<td>102.5</td>
<td>2.8</td>
<td>56.9</td>
<td>123.1***</td>
<td>272.1***</td>
<td>171.7***</td>
<td>75.4</td>
<td>16.2</td>
<td>24</td>
</tr>
<tr>
<td>Group total</td>
<td></td>
<td></td>
<td>80.6</td>
<td></td>
<td>107.3***</td>
<td>121.8***</td>
<td>84.3</td>
<td>99.6**</td>
<td>12.9</td>
<td>281</td>
<td></td>
</tr>
</tbody>
</table>

**BW, body weight.**
† For details of dietary treatment, see Table 1.
‡ Cats were fed on a product containing 3550 (SE 950) mg taurine/kg DM for the preceding 2 weeks.
All mean values were compared along the same horizontal row with the value for week 0: Student's t test.
* P < 0.02; ** P < 0.01; *** P < 0.001.
mixed with an equal volume of sulphosalicylic acid (0.46 mol/l) and 0.5 ml norleucine (0.5 mmol/l), internal standard. All samples were stored frozen (−18°C) before taurine analysis. Before analysis the samples were thawed and the protein precipitate removed by centrifugation; the supernatant fraction was adjusted to pH 2.2 with lithium hydroxide (3 mol/l) buffer. A 0.1 ml sample was separated by ion-exchange chromatography into its constituent amino acids by an amino acid analyser (LKB Alpha-Plus, Cambridge) and taurine was detected after a post-column reaction with ninhydrin at a wavelength of 570 nm. Norleucine was used as an internal standard and had a coefficient of variation 4%. The detection limit for plasma was 5 μmol/l and for food samples, 250 mg/kg DM.

Sample preparation differed slightly for food samples: 2.5 g of the food sample was mixed with 150 ml hydrochloric acid (0.1 mmol/l) and 2.5 ml norleucine (10 mmol/l) for 5 min in a laboratory food mixer (Silverson, Chesham, Bucks.). All the liquid was transferred to a 250 ml volumetric flask and made-up to volume with de-ionized water. The solution was allowed to settle before 3 ml supernatant fraction were removed; adjusted to pH 2.2 with lithium hydroxide buffer (3 mol/l). A 0.1 ml sample was then separated as described previously.

Statistical analysis

All values were expressed as means with their standard errors. All data were analysed by standard parametric statistical tests using 2-way ANOVA and the Student's t test (SAS, 1985).

RESULTS

The body-weights of the three groups of cats (seven in each group, eleven males, ten females in total) were closely monitored throughout the trial period. Their initial mean body-weights were: group I 3.63 (SE 0.23) kg, group 2 3.57 (SE 0.29) kg, group 3 4.06 (SE 0.39) kg. Their mean body-weights remained fairly stable, over the test period and at the end of the trial were not significantly different from their initial weights: group 1 4.42 (SE 0.48) kg; group 2 3.90 (SE 0.25) kg, group 3 4.79 (SE 0.55) kg.

Table 1 shows the list of feeding trials, in chronological order, carried out on each of the three groups of cats.

Table 2 shows the dietary taurine content (mg/kg DM) of the twelve canned test diets, ranked in descending order, and the fasting plasma taurine concentrations (μmol/l) throughout each trial. At the beginning and end of each feeding period (weeks 0 and 8), all mean plasma taurine values were greater than 40 μmol/l, and the group mean value was 80-6 μmol/l. Fasting plasma taurine values were recorded every 2 weeks throughout the test period. There were no significant differences between the initial plasma values (week 0); however, there were a number of significant differences across the dietary treatments. The groups fed on diets containing 1475, 1875 and 1950 mg taurine/kg DM had a fasting plasma concentration significantly lower than their initial value, at the end of the test period (week 6). When dietary content was greater than or equal to 3750 mg taurine/kg DM plasma taurine concentration was significantly higher than the initial level after 6 weeks feeding. There was no significant change from the mean fasting plasma taurine concentration after 6 weeks feeding, where dietary content was in the range 2325–3750 mg taurine/kg DM. Fig. 1 shows the relationship between dietary taurine content (mg/kg DM) and the mean plasma taurine concentration (μmol/l) for the 6-week test period. A dietary taurine concentration of greater than 2000 mg/kg DM would result in a plasma taurine concentration greater than 60 μmol/l. Fig. 2 shows the relationship between mean daily taurine intake (mg/kg body-weight) and the percentage change in plasma taurine concentration from the initial fasting concentration (see Table 2) after 6 weeks of feeding.
Fig. 1. Effect of dietary taurine content (mg/kg dry matter (DM)) on the mean plasma taurine concentration (μmol/l), after a 6-week period of feeding the test diet. Values are means for seven cats for a total of twelve trials. For details of diets, see Table 1. The line of best fit by least squares regression was $Y = 0.037X - 6.82$, $r = 0.90$, $df = 10$, $P < 0.001$.

Fig. 2. Effect of mean daily taurine intake (mg/kg body-weight) on the mean change in plasma taurine concentration (%) from the initial group mean value of 80.6 μmol/l. Values are means for seven cats for a total of twelve trials. For details of diets see Table 1. The line of best fit by least squares regression was $Y = 2.44X - 94.5$, $r = 0.86$, $df = 10$, $P < 0.001$.
the test product. When dietary intake exceeded 39 mg taurine/kg body-weight per d there was a net increase in the circulating concentration of taurine.

Values were recorded at the end of a 2-week repletion period when all cats were fed on a diet containing 3550 (SE 95.0) mg taurine/kg DM (Table 2). There were no significant differences between the fasting plasma values at week 8, however, group means for weeks 6 and 8 (84.3 v. 99.6 μmol/l, P < 0.01) were significantly different.

The groups fed on diets containing less than 2000 mg taurine/kg DM, during the test period showed a significant increase in their circulating plasma taurine concentration after 2 weeks on the repletion diet. The fasting values had returned to the pretrial levels at week 8. The groups fed on diets containing at least 3750 mg taurine/kg DM showed a significant decrease in their plasma taurine concentration following 2 weeks solus feeding of the repletion diet, the week 8 plasma values were not significantly different from those at week 0. All cats had a plasma taurine concentration greater than 60 μmol/l at the end of the study.

Table 3 shows the dietary taurine content (mg/kg DM) of the three dry, extruded diets, ranked in descending order, and the fasting plasma taurine concentration (μmol/l) throughout each trial. All cats had a fasting plasma taurine concentration greater than 60 μmol/l at the beginning and end of each trial (weeks 0 and 8). There was a statistically-significant change in plasma taurine concentration with the dietary regimen of 811 mg taurine/kg DM from week 0 to week 2, but not over the following 6-week period. There was a significantly lower concentration of plasma taurine at week 4 in the test group fed 967 mg taurine/kg DM than during all other time periods. When dietary taurine content was increased to 1240 mg/kg DM there was a statistically-significant increase in the circulating level of taurine at weeks 2, 4 and 6 as compared with the values at weeks 0 and 8. The mean daily taurine intake for this third dietary treatment was significantly greater than those for the groups fed on the diets containing 811 and 967 mg taurine/kg DM (19.4 v. 209.2, 23.0 v. 209.2 mg/kg body-weight per d; P < 0.05), which would account for the higher circulating concentration of taurine during this test.

DISCUSSION

The cats used in the present study showed no significant fluctuations in their mean body-weights as all the test diets, when fed ad lib. supplied the necessary 290–335 kJ/kg body-weight (National Research Council, 1986) needed for weight maintenance. The diets were prepared, not only to provide sufficient metabolic energy, but to supply a wide range of dietary taurine intake (23.7–102.5 mg/kg body-weight from a canned diet, 19.4–209.2 mg/kg body-weight from a dry diet).

The early studies, using semi-purified diets, concluded that maintaining a plasma taurine concentration of greater than 40 μmol/l over an extended period would reduce the risk of retinal degeneration and reproductive abnormalities. This was achieved by ensuring a minimum dietary taurine content of 500 mg/kg DM. Findings from the present study show that, when feeding cats test diets that have been prepared in the same way as commercially-available canned cat foods, a much greater taurine intake is needed to sustain plasma taurine concentration above 40 μmol/l. There were a number of test diets which resulted in at least one cat having a plasma level of less than 40 μmol/l after 6 weeks solus feeding, and where the diet contained more than 500 mg/kg DM (1475, 1750, 1875 and 1950 mg/kg DM). The mean daily taurine intakes of these cats were between 20.4 and 31.5 mg/kg body-weight. Therefore, using a value for the fasting plasma concentration of 40 μmol/l as the lower limit of the normal range likely to lead to retinal degeneration, the diet should ensure a daily taurine intake of at least 32 mg/kg body-weight.
Table 3. Effect of dietary content (mg/kg dry matter (DM)) on fasting plasma taurine concentration (μmol/l)
(Mean values with their standard error for seven cats fed on dry, extruded diets)

<table>
<thead>
<tr>
<th>Dietary taurine content (mg/kg DM)</th>
<th>Taurine intake (mg/kg BW per d)</th>
<th>Period of feeding (weeks)</th>
<th>Mean SE</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8†</th>
<th>SED</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>811</td>
<td>19.4</td>
<td>168.8</td>
<td>114.3</td>
<td>128.1</td>
<td>121.3</td>
<td>128.1</td>
<td>18.6</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>967</td>
<td>23.0</td>
<td>107.3</td>
<td>128.2</td>
<td>92.2*</td>
<td>138.0</td>
<td>126.7</td>
<td>13.9</td>
<td>13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1240</td>
<td>209.2</td>
<td>73.4</td>
<td>153.6***</td>
<td>131.1**</td>
<td>213.1***</td>
<td>124.6**</td>
<td>15.5</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group total</td>
<td></td>
<td>117.0</td>
<td>132.4</td>
<td>119.8</td>
<td>160.7*</td>
<td>126.4</td>
<td>24.6</td>
<td>61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BW, body-weight. † For details of dietary treatments, see Table 1. ‡ Cats were fed on a product containing 3550 (SE 95.0) mg taurine/kg DM for the preceding 2 weeks. All mean values were compared along the same horizontal row with the value for week 0: Student’s t test. * P < 0.02, ** P < 0.01, *** P < 0.001.

Pion et al. (1987) showed that fasting plasma taurine concentrations less than 20 μmol/l were indicative of severe taurine deficiency and they were able to record a significant reduction in cardiac function. However, plasma taurine must be maintained below 20 μmol/l for an extended period (months/years) before clinical signs are apparent. A number of the test diets resulted in at least two cats having plasma values below 20 μmol/l after 6 weeks solus feeding (1475, 1750 and 1875 mg/kg DM). It is unlikely, however, that this had any lasting effect on these individuals as their plasma levels were repleted above 40 μmol/l within 2 weeks of feeding an adequate diet.

Although plasma taurine concentrations of 40 μmol/l are often used as the cut-off limit indicative of taurine deficiency, in a short-term feeding trial it may be more realistic to assess plasma levels somewhat higher as indicative of ‘normal’ values. A circulating plasma taurine concentration of greater than 60 μmol/l was considered to be more appropriate as the lower limit (Pion et al. 1987; Monson, 1989). The canned test diets providing more than 2000 mg taurine/kg DM resulted in plasma taurine concentrations greater than 60 μmol/l (Fig. 1) after 6 weeks feeding. Also, when daily taurine intake exceeded 39 mg/kg body-weight, there was a net increase in the mean circulating levels of plasma taurine compared with the initial (week 0) group mean values (Fig. 2).

Where the dietary taurine content, during the 6-week trial period had been inadequate to maintain the fasting plasma taurine concentration of all the cats above 60 μmol/l, there was a significant increase in the plasma values following 2 weeks on the repletion diet. The repletion diet contained 3550 mg taurine/kg DM and provided a daily taurine intake of 50 mg/kg body-weight, which was higher than the taurine-deficient canned, test diets (1475–1950 mg taurine/kg DM; equivalent to 20.4–31.5 mg/kg body-weight per d). Test diets supplying more than the 3550 mg taurine/kg DM of the repletion diet resulted in plasma levels at the end of the test period higher than for weeks 0 and 8. The repletion diet used in all the trials was identical, to enable all values at the end of the study to return to their prettrial level.

The values for the dry, extruded test diets indicated that a dietary intake of 19.4 and 23.0 mg taurine/kg body-weight per d would maintain the plasma taurine concentration at the initial level, i.e. greater than 60 μmol/l, after 6 weeks feeding. This was considerably lower than the 39 mg taurine/kg body-weight per d needed for the canned test diets. When daily taurine intake increased to 209.2 mg/kg body-weight there was a significant increase...
in plasma taurine values; however, the increase was no greater than that observed for a canned test diet supplying 102.5 mg taurine/kg body-weight. These results suggest that there may be an upper limit for the circulating concentration of taurine in the cat; however, more information is needed from diets providing greater than 100 mg taurine/kg body-weight per d to confirm this observation. At present it can be concluded that a dry, extruded diet providing more than 811 mg taurine/kg DM will maintain 'normal' circulating levels of plasma taurine. This confirms earlier work where cats fed on commercially-available dry foods containing 900 mg taurine/kg DM prevented retinal degeneration in cats (Aguirre, 1978).

In view of the role of taurine as an essential nutrient for the cat, it was necessary to supply five times more taurine in a canned test diet and almost twice the level in a dry test diet than that known to be effective in a semi-purified diet to maintain normal plasma taurine concentration. This observation inevitably begs the question as to the possible adverse effects of long-term feeding of high levels of taurine. In a series of multigeneration studies, taurine intakes as high as 7 g/kg body-weight per d were shown to have no adverse effect on the growth, reproductive performance or histology of a group of rats (Takahashi et al. 1972a, b). The dietary taurine intakes measured in the present study were well below this level (maximum was 0.21 g/kg body-weight per d) and, therefore, not considered to be excessive.

Conclusions

The aim of the study was to investigate the effect of feeding a series of specially-prepared test diets on the plasma taurine concentration of the cat. Values collected over a period of 20 months have shown that the test diets, although containing a greater concentration of taurine than recommended for semi-purified diets, do not necessarily maintain plasma taurine concentrations above 40 μmol/l. Short-term feeding trials of 6 weeks duration allowed an evaluation of each test diet, although it is acknowledged that extended periods of many years may be necessary to observe the clinical signs of taurine deficiency. It has enabled us to conclude that a canned cat food with a dietary taurine content of 2000 mg/kg DM or greater, and supplying at least 39 mg taurine/kg body-weight, would maintain plasma taurine values above 60 μmol/l. If the diet supplies less than 2000 mg/kg DM it is likely that some cats will become taurine deficient. The availability of taurine from these specially-prepared canned diets was approximately five times lower than from a semi-purified diet.

The dry, test diet which supplied the cats with a daily intake of greater than 19 mg/kg body-weight were able to maintain their circulating taurine concentration at an adequate level. These diets contained around twice as much taurine as an adequate semi-purified diet.

Repletion of plasma taurine concentration occurred within 2 weeks of feeding a supplemented diet containing 3550 mg taurine/kg DM, indicating the transient nature of the deficiency state in the present study. Previous workers have observed clinical signs of taurine deficiency only after extended periods of deprivation.

When considering the results of the present study and those of other workers showing the reduced availability of taurine from commercially-prepared cat foods, it was interesting to note that it was necessary to increase dietary taurine content above a higher threshold level than for semi-purified diets before normal plasma taurine levels were achieved, assuming of course, that plasma levels below 60 μmol/l are indicative of reduced taurine status. This would indicate that the poor availability of taurine, especially from the canned test diets, was due not only to a reduced gastrointestinal absorption caused, possibly, by competition at the appropriate binding site but to the excessive wastage of taurine in the digestive tract. Once the dietary intake was increased beyond 39 or 19 mg taurine/kg body-
weight for the canned and dry diets respectively, the taurine was able to overcome these two mechanisms. Investigations continue at the Waltham Centre to evaluate the possible cause of the reduced bioavailability of taurine from these foods.

REFERENCES


Printed in Great Britain