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Taurine Requirement of the Cat

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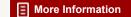
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This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

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PREFACE

In 1978, the Panel on Cat Nutrition, Subcommittee on Laboratory Animal Nutrition of the Committee on Animal Nutrition produced a report on the <u>Nutrient Requirements of Cats</u>. In this report, research on taurine as a requirement for the prevention of Central Retinal Degeneration (CRD) was discussed. However, the lack of research on lower limits of taurine for the prevention of CRD deterred the authoring panel from recommending a taurine requirement value.

The Director of the Division of Animal Feeds, Bureau of Veterinary Medicine, Food and Drug Administration, requested that the Committee on Animal Nutrition review the latest research data on taurine and if possible provide an estimate of the dietary taurine needs of the cat. The Committee on Animal Nutrition convened an ad hoc panel that has reviewed the latest research data on taurine and has produced this report.

Ad hoc Panel on Taurine Requirement of the Cat

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TAURINE REQUIREMENT OF THE CAT

Central retinal degeneration (CRD) in the cat and the association of this condition with limited dietary taurine intakes have been reviewed (NRC, 1978). While a qualitative requirement for taurine was apparent, at least when dietary sulfur amino acid (SAA) concentrations were low, published data were not sufficient to set a minimum taurine requirement. Observations with both purified and commercial-type diets suggested that 1,000 ppm taurine in the dry cat diet was adequate to prevent CRD, but this level probably does not represent the minimum need. Until more definitive information is available, the following discussion has been developed to guide those formulating diets for cats.

When cats were fed experimental diets containing zero concentrations of taurine, CRD was induced at five different laboratories (Hayes et al., 1975a; Anderson et al., 1979; J.G. McLean, School of Veterinary Science, University of Melbourne, Parkville, Victoria, Australia, personal communication, 1980; Burger and Barnett, 1980; Rogers et al., 1980a). The incidence of ophthalmoscopically visible lesions varied from 12 to 100 percent. Undoubtedly, a higher mean incidence would have been reported if abnormal electroretinography (ERG) were uniformly used as a criterion for CRD, since ERG is a much more sensitive indicator of degeneration. It also may be relevant that CRD incidence was inversely related to dietary SAA concentrations.

Taurine is synthesized by the cat to a limited extent (Schmidt et al., 1977; Sturman et al., 1978; Knopf et al., 1978), and there is some evidence that the amount of taurine synthesis is dependent upon the level of dietary SAA (Berson et al., 1976; O'Donnell et al., 1980). Rogers et al. (1980b) have found a low incidence of grossly visible CRD in kittens fed diets containing 0 ppm taurine and 1.5 to 2.0 percent SAA, and some kittens exhibited no visible CRD and had normal ERG amplitudes after receiving diets containing 0 ppm taurine and 2 percent SAA for 1 year. The highest incidence of CRD was found in kittens fed diets with 0 ppm taurine and less than 1.0 percent SAA (Hayes et al., 1975b; Anderson et al., 1979). Hayes et al. (1975b) noted a 100 percent incidence of CRD in kittens fed a casein-based diet (low SAA, 0.6 percent) and no CRD when kittens were fed a lactalbumin-based diet (higher SAA and 6 ppm taurine). Suboptimal blood plasma levels of taurine have been associated with the occurrence of CRD (Hayes et al., 1975a; Schmidt et al, 1976, 1977; Berson et al., 1976), and O'Donnell et al. (1980) have shown that plasma taurine was lower in adult cats fed a diet with 0.78 percent SAA than in adult cats fed a diet containing 1.55 percent SAA. On the other hand, supplementation of weanling kittens with methionine or cystine in a casein-based diet (1.5 to 1.6 percent total SAA) had no beneficial effect on plasma taurine levels after 23 weeks, even though plasma methionine or cystine levels were about double their usual concentrations. Furthermore, CRD was detected in these kittens by a 75 percent reduction in ERG amplitude, even though retinal changes visible with the ophthalmoscope were minimal (Berson et al., 1976). The age of the cat may also influence the degree to which SAA spare taurine. Adult cats fed a methionine-supplemented diet exhibited an improved bile acid taurine profile compared to cats fed the unsupplemented diet or kittens fed the diet with or without supplemental methionine (Rabin \underline{et} \underline{al} ., 1976).

Factors that influence the enterohepatic circulation of taurine may also affect dietary taurine requirements. Since the cat conjugates taurine almost exclusively with bile acids (Rabin et al., 1976), any factor that decreases absorption of taurocholic acid from the gut, such as dietary fiber, may increase the quantitative dietary taurine need.

Although data are still insufficient to set a minimum taurine requirement, fragmentary information can be used to develop a tentative recommendation. Rogers et al. (1980a) fed kittens purified diets containing 33 percent protein, 1.55 to 2.0 percent SAA and 0 ppm taurine and induced CRD in 15 percent of the kittens by 1 year. The addition of 50 ppm taurine to this purified diet prevented CRD based on ophthalmoscopic examination. Burger and Barnett (1980) fed a purified diet containing 23 percent crude protein, 1.53 percent SAA and 0 or 1,500 ppm taurine to 6-month-old kittens for 11 months. There was a 50 to 56 percent incidence of CRD in cats receiving no taurine and a 0 percent incidence in those receiving 1,500 ppm. Furthermore, when taurine-depleted cats were given 200 ppm taurine, plasma taurine levels failed to rise and there was considerable progression of the retinal lesions in two of three cats that had developed CRD during taurine-depletion. When cats, previously fed 1,500 ppm taurine, were fed diets containing 200 ppm taurine, plasma taurine levels fell from above 200 μM to 30 μM or less within 12 weeks. No CRD was noted during 30 subsequent weeks of observation. After 43 weeks, however, one of five previously normal cats transferred from 1,500 to 200 ppm taurine showed early stages of CRD (I.H. Burger, Animal Studies Centre, Freeby Lane, Waltham-on-the-Wolds, Melton, Mowbray, Leicestershire, England, personal communication, 1980). Taurine-depleted cats that were given 500 ppm taurine showed a transient plasma taurine rise to 55 μM after 12 weeks and then declined to less than 30 µM. In cats previously fed 1,500 ppm taurine, plasma taurine declined from about 150 to 30-60 µM when fed 500 ppm taurine. However, 500 or 1,000 ppm dietary taurine for 30 weeks had no effect on the severity of previously developed CRD in the depleted cats, and no CRD appeared over 67 weeks in those cats previously fed 1,500 ppm taurine for 11 months.

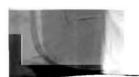
There are no published reports concerning cats that have been fed commercial-type diets with graded levels of taurine for extended periods and that have been examined for CRD. However, during the past year, Rogers and Morris have received three reports from practicing veterinarians who have clinically diagnosed CRD in cats fed commercial diets for periods of 2 to 6 years. Blood samples taken from two of these cats revealed plasma taurine levels of 1.1 and 3 μ M. Taurine concentrations in the diet brands consumed by these cats (but purchased from a local supplier) were 180 to 260 ppm (dry basis). The manufacturers reported SAA concentrations of 1.1 to 1.2 percent (dry basis). It has been established that plasma taurine concentrations are highest at dietary taurine levels



Inhe standard diet test protocol of the Association of American Feed Control Officials would not detect an inadequacy of dietary taurine because the test protocol does not involve sufficient time for deficiency signs to appear.

of 1,000 ppm or more (Burger and Barnett, 1980; O'Donnell et al., 1980). Schmidt et al. (1977), using isotopically labelled taurine, demonstrated that taurine accumulation by the retina can be correlated with plasma taurine concentration and concluded that a plasma taurine level of 40 μ M was indicative of normal retinal taurine accumulation. Kittens that have developed CRD at the Rogers and Morris laboratory (University of California, Davis) have plasma taurine levels consistently below 5 μ M. When fed purified diets, containing 30 percent protein, 1.5 percent SAA, and 50 to 200 ppm taurine, for extended periods of time, plasma taurine levels were 10 to 15 μ M. Dietary taurine levels of 500 ppm resulted in 25 to 50 μ M taurine in plasma. Burger and Barnett (1980) reported similar findings except their plasma taurine assay resulted in values that were 50 to 100 percent higher than those of Rogers and Morris.

From these incomplete data, it would appear that the minimal dietary taurine requirement for cats is between 250 and 1,000 ppm, dependent upon SAA levels and other factors. Studies are in progress (Rogers et al., 1980b) that, within 1 to 2 years, should provide a basis for a more definitive statement of taurine need. In the interim, based on plasma taurine responses to dietary taurine² and considering that SAA concentrations of some commercial cat diets may not provide for sufficient taurine synthesis and that fecal taurine losses may be greater when commercial rather than purified diets are fed, it is suggested that cat diets should contain at least 500 ppm taurine (dry basis).





²Hemolysis can obscure differences in plasma taurine levels since erythrocytes have 5 to 40 times greater taurine concentrations than plasma (Burger and Barnett, 1980).