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Since its discovery in 1975, taurine deficiency has been an important nutritional problem in cats. After the original findings of blindness and retinal degeneration, the number of clinical manifestations has increased to include reproductive failure, growth retardation and neurologic abnormalities in surviving kittens from taurine depleted queens. Cardiomyopathy, platelet hyperaggregation, and possibly immune function impairment are implicated as well.[1]

Taurine is a beta amino acid containing a sulfonic rather than a carboxylic group and thus cannot form a peptide bond. Many of the biological effects of taurine are mediated via activity on calcium and, as recent studies show, zinc.

The susceptibility of the feline species to taurine deficiency occurs because, unlike many other placental mammals, cats have very limited ability to synthesize enough taurine from other sulfur amino acids, and they are constantly secreting large amounts of taurine in their bile. The limited ability to produce taurine arises due to low availability of the enzyme CSA decarboxylase which is needed to convert the sulfur amino acid cysteine to taurine.[1,2] The bile acid pool in cats is unique since all bile acids are conjugated with taurine. Therefore, the cat has a continual dietary taurine requirement of 500-750 mg/Kg of dry matter per day to replace fecal losses resultant from the less than 100% recovery by the enterohepatic circulation.[2]

Since the feline species is unable to synthesize appreciable amounts of taurine, this amino acid is considered an essential nutrient in the dietary intake.[3,11] The most widely accepted consensus suggests a requirement of 500-750 mg/kg of diet dry matter per day. However, many variables are known to alter this recommended level. As the fiber content of the diet decreases, fecal taurine losses increase. This requires a corresponding elevation of taurine in the diet. Likewise, the taurine requirement is increased with a decreasing intake of other sulfur containing amino acids. Evidence also shows that cats fed dry cat food containing 500-750 mg of taurine/Kg of diet dry matter do not develop plasma taurine deficiency, while feeding canned food containing 2-10 times this level may result in low plasma taurine levels. This is thought to be due to an unknown absorption or retention problem. Therefore, cats fed canned foods may require more taurine added to their diets in order to maintain normal plasma levels.[2,3]

Taurine deficiency may occur from ignoring any of the above variables. It may also occur from feeding dog food to cats. In fact, the latter is the most common cause of taurine deficiency in the feline. Dog food contains lesser amounts of protein and therefore sulfur containing amino acids. Also, the majority of dog food contains ingredients from plant sources and less from animal sources than do cat foods. Plants are devoid of taurine, whereas fish and animal tissues are high in taurine and thus are widely used in cat food production.[1,2]

Among all the clinical manifestations of taurine deficiency, the retinopathy has been studied most intensely. lnadequate taurine intake by cats causes low retinal taurine concentration and central retinal degeneration that can result in irreversible blindness. The primary defect appears in the structural integrity of the photoreceptor cell and the underlying tapetum lucidum. It seems that taurine modulates the structural integrity by control over ionic flux of calcium, sodium, and potassium in the retina and possibly zinc via zinc-taurine cysteine interactions in the tapetum.[1,2,4,5,6,7,8]

When retinal taurine concentration is reduced to 50-75% of normal, the structure and

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function deteriorate progressively. However, because the liver is one of the major sources for retinal taurine, pronounced reductions in retinal taurine concentration occur only when liver and plasma taurine concentrations are near zero. Since the half-life of taurine in the liver is approximately 2-5 days, it would be expected that the liver content would be near zero only after taurine was eliminated from the diet for 4-12 weeks. Studies have shown that the half-life of taurine in the retina is about 30-88 days, and since taurine isn't metabolized in the cat retina, a greater than 50% reduction in retinal taurine concentration would occur 10-25 weeks after liver taurine was near zero. Upon consideration of the half-life of taurine in the liver and retina, it is possible to explain why some cats develop severe retinal malfunction 15-45 weeks after elimination of taurine from the diet.[8]

The retinal degenerative lesions in cats on taurine deficient diets are readily diagnosed by routine ophthalmoscopy. On ophthalmoscopic exam, visible lesions are bilateral and range in size from small focal atrophic areas in the area centralis to a generalized atrophic appearance of the entire retina with attenuation or loss of retinal vessels. These changes become evident when retinal taurine concentration is reduced to 20-30% of normal. After long term taurine deprivation, the lesion becomes progressively larger and the entire fundus is atrophic in appearance.[4,5,6,7,8]

Cats fed taurine deficient diets have also shown pronounced reductions in electroretinogram (ERG) amplitude even prior to detectable cell death. When retinal taurine concentrations are 20-30% of normal, the ERG amplitude is very low or undetectable and ultrastructural studies show widespread photoreceptor cell death. However, based on experimental studies, a change to a taurine adequate diet will prevent further progression of the disease. Therefore, routine ophthalmoscopic screening of cats is recommended in order for early detection.[8]

Dietary taurine deficiency has also been shown to suppress reproductive performance in queens. Although taurine deficient females come into estrus normally, they frequently resorb or abort their fetuses, and have stillborn or vital low birth weight kittens at term. Those kittens born alive have poor survival rate and grow at substantially slower rates than kittens from taurine supplemented queens. They also exhibit a number of neurological abnormalities such as defective cerebellar maturation, a peculiar gait characterized by excessive abduction and paresis, hydrocephaly, and anencephaly. Furthermore, kittens may show severe developmental abnormalities including abnormal hind limb development and thoracic kyphosis attributed to dysplasia of the cerebellar granular layer. This is a result of a failure in cell division and cell differentiation, both of which are dependant on the intracellular calcium pool. It is interesting to note that although queens fed a taurine deficient diet show reproductive failure, when switched to a taurine supplemented diet they return to the usual breeding schedule and produce live, normal kittens.[1,2,9,10]

Another important aspect of taurine deficiency is its effect on the myocardium. In 1987, Dr. Paul Pion, University of California-Davis discovered that all cases of feline dilated cardiomyopathy showed low plasma taurine concentrations. Those cases treated with taurine recovered normal function of the heart muscle.[3,11,12]

Feline dilated cardiomyopathy associated with low plasma taurine results in contractility failure due to abnormal calcium ion balance in the myocardial cell. Taurine modulates calcium movement through the sarcolemma, and by reducing myocardial calcium, protects the heart from injury. Taurine constitutes 50% of the free amino acid pool in the myocardium. In fact, the myocardium contains one of the highest concentrations of free taurine in the body with levels ranging from 100-400 times those found in plasma. In cases of congestive heart failure, the taurine concentration in the heart is increased above these levels.[3]

Taurine deficient cardiomyopathy of cats appears to require lengthy and severe taurine depletion. Studies have shown that a moderately severe depletion does not result in clinical evidence of heart failure, even when other signs of taurine deficiency exist. The level of plasma taurine below which a cat is at risk of developing dilated cardiomyopathy is 20 nmole/ml. To ensure a margin of safety, cats with levels less than 40 nmole/ml should be changed to another diet or supplemented with taurine.[3] In feline dilated cardiomyopathy, pulmonary and hepatic congestion are seen due to congestive heart failure. Pleural effusion is also common and occasionally pericardial and peritoneal ef-

fusion is seen. Because of calcium overload, there is a decrease in ventricular systolic function and pump failure occurs. Thus, the blood remains in the heart and causes a decrease in stroke volume and arterial pressure. The body attempts to increase its stroke volume by vasoconstriction, and sodium and water retention, which in turn results in ventricular dilation.

Dilated cardiomyopathy can be diagnosed using echocardiography. An end systolic diameter greater than 12 mm and a shortening fraction less than 35% is diagnostic provided there is no evidence of other underlying congenital, acquired, or functional cardiac abnormalities.[3] At the University of California Davis, cats with these echocardiographic criterion were supplemented with 5000mg taurine/Kg dry diet for 21 days. Every cat improved clinically during the first 2 week period of taurine supplementation, however 3-4 weeks of supplementation were needed before the cats improved echocardiographically.[3]

Taurine depletion also induces an increased thromboxane level and an increased incidence of collagen-induced aggregation of platelets, both of which are linked to intracellular calcium metabolism in the platelet. Since the feline species is highly prone to thromboembolic episodes, the importance of adequate taurine intake may prove to be prophylactic.[1]

Evidence for possible immune function impairment in taurine depleted cats has been reported. It appears that upon taurine depletion, the white blood cell taurine content also decreases and white blood cell stimulation by mitogens is depressed. However, this manifestation of taurine deficiency is less well defined and further studies are needed.[1]

Clinical confirmation of taurine deficiency requires a routine plasma amino acid or a specific high pressure liquid chromatography (HPLC) assay. Normal values for plasma taurine in the feline range from 60-120 umoles/l with an average being 80 umoles/l.[3] However, dietary intake must be considered when interpreting these results. For example, a cat consuming a fresh fish, meat, or poultry diet will have a higher plasma taurine concentration than a cat consuming a dry diet.

Taurine is an important amino acid affecting many body systems of the feline. Further studies of its effect on calcium metabolism in cells is needed to fully understand the impact that it may have on other body systems. Because calcium is involved in a wide range of body functions, the possibility for new clinical manifestations of taurine deficiency exists. With proper nutritional management this complex deficiency problem can be avoided.

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