Title: A Reexamination of Pottenger's Cats

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ABSTRACT

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From 1932 to 1942, Dr. Francis Marion Pottenger Jr. conducted a group of experiments to determine the effects of heat-processed food on cats. Cats fed the all-raw diet were healthy while cats fed the cooked meat diet developed various health problems. At the time of Pottenger's studies the heat labile amino acid taurine had not yet been identified as essential for cats. This paper identifies that the deficiencies Pottenger found in his cats correspond with those of a taurine deficiency and are the direct result of the lack of taurine in the feline diet.
Pub Med and EBSCO Host were utilized to obtain journal articles. The Pottenger papers were obtained from the Price-Pottenger Nutritional Foundation. The physiological effects of a cooked diet described by Pottenger in his papers were studied. His findings were compared with data from recent studies on taurine deficiency in the feline diet. Since Pottenger's studies, current research have shown that a taurine deficiency induces feline developmental abnormalities. Taurine deficiency is a strong explanation for the symptoms observed by Pottenger in his cat studies. Current research has linked Pottenger's observed developmental abnormalities to taurine deficiency in cats.

Keywords: taurine; cat; FM Pottenger; deficiency

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Psalm 139

INTRODUCTION

From 1932 to 1942, Dr. Francis Marion Pottenger Jr. conducted a group of experiments to determine the effects of heat-processed food on cats.[1,2] As part of the studies, one group of cats was fed a diet of two-thirds raw meat, one-third raw milk, and cod-liver oil while the second group was fed a diet of two-thirds cooked meat, one-third raw milk, and cod-liver oil. The cats fed the all-raw diet were healthy while the cats fed the cooked meat diet developed various health problems. His study published in 1946 sparked immense interest in raw food diets partially due to a better understanding of the aid of enzymes in digestion.[1]

The amino acid taurine is so named because it was first isolated from the bile of an ox and one of the first publications on this amino acid was in 1838 by DeMarcay.[3] At the time of Pottenger's studies taurine had not yet been identified as an essential amino acid for cats. This fact was not realized until 1975.[4,5] Taurine is heat labile and the act of cooking food that it is found in degrades the amino acid.[6] Today many cats thrive on a cooked meat diet where taurine has been added after cooking. Pottenger himself concluded that there was likely an "as yet unknown" protein factor that may have been heat sensitive.[1] This paper identifies that the deficiencies Pottenger found in his cats, namely near and farsightedness, cardiac lesions, increased incidence of stillbirths, low kitten survival, low birth weight, and poor development correspond with those of a taurine deficiency in cats and are the direct result of the lack of taurine in the feline diet.

METHODS

The Learning Resource Center of Logan College of Chiropractic was utilized to obtain peer reviewed literature that has been published on the topic of feline development and taurine deficiency. Literature on the topic of taurine and feline development began to be published in 1975, therefore the papers used for the current examination date from 1975 to the present. The peer reviewed search engines of Pub Med and EBSCO Host were utilized to obtain journal articles. The F.M. Pottenger Jr. papers describing the cat studies were obtained from the Price-Pottenger Nutritional Foundation. The physiological effects of a cooked food diet described by Dr. Pottenger in his papers were studied and his findings were compared with data from recent studies on taurine deficiency in the feline diet.

DISCUSSION

According to the Nutrient Requirements of Cats, revised edition, 1986, the taurine requirement for the domestic cat is 400 mg/kg for kittens and adult cats and 500 mg/kg for pregnant females. Cats lack the ability to synthesize adequate amounts of taurine from precursors.[7] This fact makes taurine an essential amino acid in the feline biological system and therefore it must be provided completely by the diet. It has been shown that cats with a diet consisting of heat processed foods have a lower plasma taurine concentration.[6,8-11] It is believed that the Maillard reaction produces products that promote an enteric flora that degrades taurine and reduces the recycling of taurine by the enterohepatic route. Another possibility of why there is lower plasma taurine concentration in heat processed diets is an excessive secretion of the hormone cholecystokinin or CCK brought about by lower protein digestibility.[12]

Throughout the ten years that Pottenger used to conduct the cat studies he observed six major deficiencies in cats that were fed cooked food diets. These six deficiencies are near and farsightedness, increased incidence of stillbirths, low kitten survival, low birth weight, and poor development. Each of these deficiencies will be discussed in order and current research will be presented to show the link between each deficiency and taurine insufficiency.

In 1975 Dr. K.C. Hays conducted two studies that observed retinal degeneration in kittens and adult cats fed a semi-purified diet containing casein. Casein is the name of a family of related phosphoproteins which are commonly found in mammalian milk, making up 80% of the proteins in cow milk and between 20% and 45% of the proteins in human milk.[13] As a food source, casein supplies amino acids, carbohydrates, and two inorganic elements, calcium and phosphorus.[14] The Hays studies were conducted in order to better understand the findings of a 1973 paper in which it was observed that kittens fed a diet with casein as the only protein source developed abnormalities in retinal function and structure.[15] Hays stated that his first study was "undertaken to document the earliest morphologic changes associated with this [retinal] degeneration" and "to determine whether the degeneration could be prevented or reversed by substituting other sources of protein for the casein in the semi-purified diet".[4] A total of 53 domestic cats were utilized in this experiment and it was found that all cats fed the casein diets had retinal degeneration while none of the cats fed a commercial diet or a diet containing lactalbumin substituted for casein showed any signs of degeneration. The study is concluded by stating that there is "unequivocal evidence that dietary casein is related to nutritionally induced retinal degeneration in cats" and that this degeneration could be brought about by "some unknown factor in casein or a property of the protein source itself".[4] The second paper published by Hays in 1975 presents taurine as the "unknown factor" that is lacking in casein and what leads

to retinal degeneration. "Amino acid profiles indicate that [retinal] degeneration is associated with a selective decrease in plasma and retinal taurine concentrations. A sulfur amino acid deficit in the casein diet combined with specific amino acid requirements of the cat appear related to this unique expression of taurine deficiency".[5] Hays continues with "the data reported demonstrate that taurine deficiency in the retina is associated with photoreceptor cell degeneration…".[5] The realization that taurine is an essential amino acid in the cat sparked a multitude of further research on the subject. In 1976 Dr. S.Y. Schmidt published a paper further supporting the discovery of Hays the year before. Schmidt states that "the present study has demonstrated that cats fed a casein diet have a selective decrease in plasma and retinal taurine concentrations".[16] She further supports previous findings with "these studies help to establish a biological role for taurine in maintaining photoreceptor cell function and viability in the cat".[16] By 1979 it was commonly understood that lack of dietary taurine lead to retinal degeneration in cats, and it was theorized that taurine deficiency may lead to physiological effects in the heart as well.[17] Further evidence supporting that taurine deficiency is the cause of near and far sightedness in the cat is found in papers by Drs. Sturman and Neumann. "The adult females consuming the taurine-free diet…did suffer from severe retinal degeneration, which was easily visible opthalmoscopically."[18] "Taurine is an essential amino acid in feline nutrition. Cats are unable to synthesize taurine from dietary precursors, and taurine deficiency results in progressive retinal disease. Taurine deficiency in cats is associated with the exclusive feeding of diets based on vegetable protein".[24] Additional supporting evidence is supplied by the Waltham books and Morris.[19-20,23] It is reasonable to conclude based on evidence provided by current research that the defects in visual pathways that Pottenger observed in his cats was due to taurine deficiency.

As mentioned previously, the link between taurine insufficiency and feline cardiac deficiency was proposed as early as 1979.[17] In 1987 P.D. Pion researched to see if there was a direct link between low plasma taurine and decreased taurine concentrations in the myocardium of the cat.[26] Pion proposed that decreased taurine concentrations in the myocardium lead to decreased myocardial mechanical function which lead to dilated cardiomyopathy (DCM) in the cat. It was concluded by Pion and colleagues that "chronic taurine depletion causes DCM in cats".[26] M.J. Novotny's 1994 study supports Pion's previous study with "dietary taurine deficiency is a cause of DCM in cats" but added to current knowledge with the following statement, "while DCM is observed in some cats, decreased systolic pump function and increased LV end systolic short axis diameter or more consistent findings".[25] Novotny concludes with "present data provide direct evidence for a causal relationship between dietary taurine deficiency and myocardial failure in cats".[25] Information found in the Waltham books and the work of Morris provide more evidence to support that taurine deficiency causes cardiac lesions in the cat.[19-20,23] Based on all of the evidence provided by current research it is reasonable to concluded that the cardiac lesions observed by Pottenger during autopsy were due to taurine deficiency.

Three of the observed deficiencies by Pottenger; higher incidence of stillbirth, lower kitten survival rate and lower birth weight, were researched in detail by Drs. J.A. Sturman and J.A. Dieter. Dr. Sturman conducted a large research study in 1986 to see the effects of taurine deficiency in pregnant female cats.[18] It was observed that "reproductive performance by the taurine depleted females was poor".[18] A more detailed look at Sturman's observations and conclusion reveal that taurine insufficiency plays a major role in reproductive and birth deficiencies in the cat. "All female taurine depleted cats experienced difficulty in completing full term pregnancies, frequently suffering fetal resorption or abortion. Those pregnancies that

reached term were often stillborn or live born of low birth weight with poor neonatal survival. No such reproductive difficulties were observed with females consuming this same diet supplemented with 0.05% taurine".[18] Sturman correctly states that "these results establish the importance of taurine in feline maternal nutrition".[18] In 1993 "the effect of long term dietary taurine insufficiency on reproductive function was studied in adult female domestic cats" by Dr. Dieter.[27] Dieter summarizes the results of his study by stating "increased resorption of fetuses, reduced litter size, and increased incidence of stillborn kittens was observed in queens while on taurine deficient diets."[27] J.G. Morris and colleagues state that "a deficiency of taurine in cats is expressed by…reproductive abnormalities occurring in the female, a high incidence of fetal resorptions and abortions, low birth weight and survival of live born young".[23] It should be noted that the Waltham books provide more evidence of increased incidence of stillbirths in cats with deficient taurine levels as well.[19-20] The reproductive and development abnormalities observed by Pottenger can clearly be explained with taurine deficiency.

The final deficiency that will be covered in this paper is developmental abnormalities. Pottenger described a number of developmental abnormalities including smaller diameter yet longer length hind leg bones, an overall smaller build and the presence of a "more nervous disposition".[2] Dr. J.A. Sturman conducted research on the effects of taurine deficiency on the development of the cat and a number of papers were published as a result. Sturman observed that kittens who were the offspring of taurine deprived mothers exhibited "a constellation of neurological abnormalities including abnormal hind leg development, a peculiar gait characterized by excessive abduction and paresis, and thoracic kyphosis readily visible by x-ray".[28] It was also observed that "kittens subjected to postnatal dietary taurine deficiency have a reduced rate of growth, which is almost entirely corrected by daily taurine feeding".[29] A third study published by Sturman and

colleagues concisely sums up the place taurine holds in cat development. In it the author states "these results establish the importance of taurine in feline maternal nutrion".[18] Abnormal growth and development including rate of growth, structural development and neurological function in taurine depleted cats has been observed and documented in other studies as well.[19-20,23,30] As has been stated previously, the developmental abnormalities observed by Pottenger can be explained by taurine deficiency in the cat.

Since the Pottenger studies, there have been a number of researchers who have shown through multiple studies that a taurine deficiency induces a number of pathologies in cats. Table 1 illustrates the deficiencies that Pottenger observed in his cats and listed next to these deficiencies are references from current research that have linked taurine deficiency to pathologies in the feline.

CONCLUSION

Taurine deficiency is a solid explanation for the symptoms observed by Pottenger in his cat studies from 1932 to 1942. Current research shows that there are undeniable similarities between taurine deficiency and the abnormalities, symptoms, and degeneration Pottenger reported in his cats that were fed the cooked meat diet. The only source of taurine for Pottenger's cats was meat and it is now well understood that cooking degrades this amino acid. This degradation leads to reduced bioavailability and biopotency of taurine. Therefore the cats that received the cooked meat diet were taurine deficient and the abnormalities observed by Pottenger were a result of this deficiency.

TABLE 1 - Pottenger's observed deficiency symptoms and corresponding taurine deficiency description references.

Symptom observed by Pottenger	Published corresponding taurine deficiency description
Defects in visual pathways.	Hayes, et al. 1975a[21] Hayes, et al. 1975b[22] Morris, et al. 1990[23] Neumann 1984[24] Rabin, et al 1973[15] Schmidt, et al 1976[16] Sturman, et al. 1986[18] Waltham 1993[19] Waltham 1994[20]
Cardiac lesions.	Morris, et al. 1990[23] Novotny, et al. 1994[25] Pion, et al. 1987[26] Waltham 1993[19] Waltham 1994[20]
Higher incidence of stillbirth.	Dieter, et al. 1993[27] Morris, et al. 1990[23] Sturman, et al. 1986[18] Waltham 1993[19] Waltham 1994[20]
Lower kitten survival rate.	Dieter, et al. 1993[27] Morris, et al 1990[23] Sturman, et al. 1986[18]
Lower birth weight.	Dieter, et al. 1993[27] Morris, et al 1990[23] Sturman, et al. 1986[18]
Developmental Abnormalities.	Sturman, et al. 1985[28] Sturman, et al. 1985[29] Sturman, et al. 1986[18] Morris, et al. 1990[23] Waltham 1993[19] Waltham 1994[20]

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