Nestlé PURINA

Companion Animal Nutrition Summit

Tackling Myths About Pet Nutrition

March 21-23, 2013 | Atlanta, Georgia

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Preface

The Nestlé Purina Companion Animal Nutrition (CAN) Summit is a scientific meeting where experts gather from around the world to explore an important topic in veterinary nutrition. This year, the CAN Summit focused on "Tackling Myths About Pet Nutrition." Under this banner, several areas were explored.

Speakers from industry and academia discussed issues related to pet food safety and quality. Their presentations showed that commercial pet foods are safe, and the risk of a pet becoming ill due to pet food contamination is very small. Dr. Karyn Bischoff discussed her experience with three pet food recalls and reviewed steps that can be taken to help assure pet food quality. These include new FDA regulations and enhanced safety programs by pet food manufacturers. She also reviewed ingredient quality and sourcing, as well as important aspects of food processing on nutrition and food quality. Meanwhile, Dr. Mian Riaz described the balance between enhanced digestibility from proper cooking versus loss of nutrient quality from overcooking and other important benefits from food extrusion.

Numerous myths abound relative to the nutritional needs of cats, many which focus on the appropriate amounts of protein, fat and carbohydrate to include in their diets. A detailed series of presentations described feline metabolism and how cats respond to variations in macronutrients. Dr. Margarethe Hoenig and others showed how cats, like other species, adapt by altering metabolism and nutrient oxidation. Dr. Bob Backus explained the impact of the balance between fats and carbohydrates, showing a benefit of limiting dietary fat when avoiding obesity is the goal. The ability of cats to adjust to different protein intakes was explored, as was evidence that cats require even more protein than previously believed if preservation of lean body mass is the goal.

Presentations regarding canine nutrition included the role of nutrients and calorie control for growing puppies and a nutritionist's view about managing gastric dilatation volvulus. Other talks explored the differences in nutritional needs of different types of "working" dogs, and the concepts of "minimum" versus "optimum" nutrient levels for dogs of all ages and lifestyles.

Finally, Dr. Sandi Smith shared tools and tips for communicating with clients about belief-based issues, such as pet nutrition. She reviewed the importance of understanding your clients' attitudes, whether they relate to nutrition or care of their pet. With an understanding of their perspectives, you can learn to shape, reinforce or change their behavior, as may be appropriate.

Among the "take home" messages from these proceedings are: Properly processed commercial pet foods are safe, due to quality control programs; healthy cats benefit from a diet that includes abundant protein and a proper balance between carbohydrates and fat suggested as a ratio of 2:1; growing dogs should not be fed adult maintenance products as they may not provide sufficient essential nutrients; and changes in dietary composition that help moderate disease processes do not necessarily prevent disease. A review of many common myths can be found in these pages.

We hope that you enjoy this collection of papers providing current, practical nutrition information as well as emerging research findings.

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Product Safety and Pet Food Recalls

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Abstract

Although pet foods are generally safe, incidents of contamination have led to recalls. Commercial pet food and animal feed recalls accounted for <10% of the total food recalls in 2011 and 2012. Slightly more than half of those recalls were due to chemical contamination, misformulation or foreign material. The rest were due to *Salmonella* spp contamination. Eleven commercial pet food recalls in the U.S. between 1996 and 2010 were caused by chemical contaminants and misformulations. As a result of these pet food recalls, there has been an increase in government oversight of commercial pet food manufacturers and human food manufacturers, greater manufacturuing vigilance, and increased awareness among veterinarians and pet owners.

Glossary of Abbreviations

CCP: Critical Control Point
COA: Certificate of Analysis
DON: Deoxynivalenol
ELISA: Enzyme-Linked Imunosorbent Assay
FDA: Food and Drug Administration
FDAA: FDA Amendments Act
HACCP: Hazard Analysis and Critical Control Point
HPLC: High-Performance Liquid Chromatography
IARC: International Agency for Research on Cancer
LD: Lethal Dose
MRI: Magnetic Resonance Imaging
NIR: Near-Infrared
PTH: Parathyroid Hormone

Introduction

Only 1.7% of reported poisonings in dogs and cats have been attributed to pet foods, thus the risk of illness due to pet food contamination is very small.[1] Incidents of chemical contamination occur through microbial action, mixing error or intentional adulteration. Although rare, the effects of pet food contamination can be physically devastating for companion animals and emotionally devastating and financially burdensome for their owners. Whereas most people consume a varied diet, diluting the effects of chemical contaminants, companion animals tend to consume a more uniform diet, often coming from a single large bag or cans from a single lot, for an extended time. Many animal owners consider their dog or cat to be a vulnerable family member that needs to be protected.[2] Based on the author's experience, some pet owners undergo seemingly disproportionate guilt when pets become sickened or die after being unknowingly fed contaminated pet foods.

The Food and Drug Administration (FDA) is charged with assuring the wholesomeness of pet foods. The U.S. Congress passed the 2007 FDA Amendments Act (FDAAA) to improve responsiveness to contamination of pet foods and other products after adulteration of pet food with melamine and related compounds was identified that year. The FDAAA requires manufacturers to report incidents of possible contamination to the FDA within 24 hours and to investigate the cause and report their findings. If contamination is confirmed, the pet food is recalled. Recall initiation is usually voluntary by the manufacturer at the request of the FDA. The FDA can secure a court order to issue a recall if the manufacturer is reluctant, but this is rare because of the potential for bad publicity and litigation should a manufacturer refuse to initiate a recall.[1] Consumer complaints can be reported to local FDA consumer complaint coordinators or online (http://www.fda.gov/cvm/petfoods.htm). Local government agriculture or food safety agencies also should be alerted when contamination of a commercial product is suspected.

Samples for laboratory analysis in a pet food investigation include the suspected food and its packaging (or, if unavailable, lot numbers, manufacturing codes and other identifying information) and samples from the pet, such as blood, serum, urine, vomitus or gastric lavage fluids, and feces. If the animal should unfortunately die, a full necropsy is necessary, and the postmortem sample collection for histopathology and analytical chemistry should include fresh urine, adipose tissue, heart blood, fresh and fixed brain, liver, kidney, as well as fixed lung, spleen and bone marrow for histology.

There are three types of recalls involving chemical contaminants:

- Class I — reasonable probability that the contaminated food will cause adverse health consequences or death
- Class II — the contaminated food can cause temporary or medically reversible adverse health consequences but is unlikely to cause serious adverse health effects
- Class III — the contaminated food is unlikely to cause adverse health consequences

There were 22 Class I and II pet food recalls in the U.S.

over a 12-year period (1996 to 2008). Six were due to chemical contaminants: Two were caused by aflatoxin, two from excess vitamin D_3, one from excess methionine, and one from adulteration of food ingredients with melamine and related compounds.[3] Since 2008, there have been three cat foods recalled due to inadequate thiamine, a dog food recalled due to excessive vitamin D_3, and one dog and one cat food recalled due to contamination with aflatoxin. There also have been two FDA warnings since 2007 concerning a Fanconi-like renal syndrome in dogs after ingesting large amounts of chicken jerky treat products manufactured in China and one local recall in New York due to low-level contamination of these products with sulfa antibiotics.[4] Pet food contamination incidents are only rarely due to adulteration, as occurred with melamine and cyanuric acid. The melamine contamination investigation in 2007 led to the discovery that other cases of melamine poisoning had happened in companion animals across Europe and Asia and in Africa.[5-7]

Natural Contaminants

The most commonly isolated natural contaminants in pet foods are mycotoxins. Aflatoxins are the most common mycotoxins associated with pet food recalls in the U.S. Other mycotoxin contaminants also have been reported, however. There was a recall of dog food due to contamination with the mycotoxin deoxynivalenol (DON) in 1995. DON is produced on grain by *Fusarium* spp. under temperate conditions. Pet food DON concentrations of >4.5 ppm and 7.7 ppm were associated with feed refusal in dogs and cats, respectively, and concentrations ≥8 ppm cause vomiting in both species.[8,9] Animals recover quickly once the food is replaced, though supportive care is needed if gastroenteritis is severe.[9]

Aflatoxins are a group of related compounds sometimes produced as metabolites of various fungi, *Aspergillus parasiticus, A flavus, A nomius*, some *Penicillium* spp., and others. High-energy foods, such as corn, peanuts and cottonseed, are most often affected. Rice, wheat, oats, sweet potatoes, potatoes, barley, millet, sesame, sorghum, cacao beans, almonds, soy, coconut, safflower, sunflower, palm kernel, cassava, cowpeas, peas, and various spices also can be affected.[10,11] Aflatoxin production can occur on field crops or in storage. Temperature, humidity, drought stress, insect damage, and handling techniques influence mycotoxin production.[10] Use of aflatoxin-contaminated food commodities in the manufacture of pet foods has caused aflatoxicosis in pets, but improper storage of dog food and ingestion of moldy garbage also have been implicated.[12]

Both dogs and cats are very sensitive to aflatoxin.[11] The oral median lethal dose (LD_{50}) for aflatoxin in dogs is between 0.5 and 1.5 mg/kg.[13] The experimental oral LD_{50} for cats is 0.55 mg/kg, though field cases of aflatoxicosis in cats are not well documented.[11] The period of exposure and amount ingested are difficult to determine in field cases, but aflatoxin concentrations of 60 ppb in dog food have been implicated in aflatoxicosis.[13] Factors associated with increased susceptibility to aflatoxicosis include genetic predisposition, concurrent disease, age, and sex, with young males and pregnant females considered particularly susceptible.[13,14] No carcinogenic effects have been reported in cats and dogs, though aflatoxins are known to be carcinogenic in some species, including rats, ferrets, ducks, trout, swine, sheep, and rats, and are classified by the International Agency for Research on Cancer (IARC) as Class I human carcinogens.[11,15]

The presentation of aflatoxicosis in small animals appears to be acute, but exposure to contaminated foods can occur for weeks or months before the onset of clinical signs. Indeed, the author was involved in one case in which known contaminated food was removed from the diet of a dog approximately three weeks before clinical aflatoxicosis became evident though the dog was closely monitored during that period by the owner and veterinarian. Many dogs die within a few days of initial clinical signs, but illness can be protracted for up to two weeks.[14] Early clinical signs of aflatoxicosis include feed refusal or anorexia, weakness and obtundation, vomiting, and diarrhea. Later, dogs become icteric, often with melena or frank blood in the feces, hematemesis, petechia, and epistaxis.[11,16]

Complete blood cell count, serum chemistry including bile acids, and urinalysis can be helpful to rule out other causes of liver failure. Total bilirubin is increased in aflatoxicosis, and hepatic enzyme concentrations, including AST, ALP and GGT, are variably elevated.[13,14] Liver function tests, though not specific for aflatoxin, are often more helpful in supporting the diagnosis. Prothrombin time is increased due to decreased synthesis of clotting factors, and serum albumin, protein C, antithrombin III, and cholesterol concentrations are decreased.[16] Post-mortem lesions can be highly suggestive, though not pathopneumonic for aflatoxis in dogs. Common gross necropsy findings include icterus, hepatomegaly with evidence of lipidosis, ascites, gastrointestinal hemorrhage, and multifocal petechia and ecchymosis.[12,15,16] Histologic lesions in acute aflatoxicosis include fatty degeneration of hepatocytes with one to numerous lipid vacuoles. Centrilobular necrosis and canalicular cholestasis with mild inflammation are commonly reported.[11,15] The presence of aflatoxin in dog food or other implicated material is extremely helpful toward confirming the diagnosis, but due to the extended time between exposure and onset of clinical signs, the contaminated food is often gone. However, if aflatoxicosis is strongly suspected, the FDA and pet food manufacturer should be alerted in case further investigation is warranted.

The prognosis for dogs with clinical aflatoxicosis is guarded. Early intervention improves the prognosis, but many cases fail to respond to treatment.[11,12,16] Intervention includes replacing the suspect diet, patient assessment and stabilization, and use of liver protectants, such as silymarin (a mix of silybin and other flavolignans from milk thistle), S-adenosylmethionine (SAMe), or *N*-acetylcysteine.

Commercial grain is routinely screened for aflatoxin, but

sampling error is possible due to the uneven distribution of mold within the grain and other commodities. Aflatoxin and other mycotoxins are produced under specific climatic conditions, and increased monitoring is warranted during those years when conditions are right for increased mycotoxin production. Current analytical techniques utilize enzyme-linked immunosorbent assays (ELISAs), high-performance liquid chromatography (HPLC) and LC/MS to detect aflatoxin in raw ingredients. Further analysis can be done on the finished product to assure that mycotoxins are not present.

Misformulation

Misformulation is another cause of adverse reactions to pet foods in cats and dogs. Hypervitaminosis D and thiamine deficiency have been reported recently. Other misformulations have involved excesses of methionine and vitamin A. Excessive methionine was associated with anorexia and vomiting.[3] Misformulation of a feline research diet in Thailand in 2009 resulted in evident hypervitaminosis A (per communication, Dr. Rosama Pusoonthornthum). Hypervitaminosis A in cats and dogs has been reported with homemade diets and caused osteopathy, commonly affecting the axial skeleton (lesions also noted in the Thai cats), and often presents as lameness, paresis or paralysis due to entrapment of spinal nerves.[17] Some animals with hypervitaminosis A, even those severely affected, recover in the long term after they are placed on a new diet.

Vitamin D is an essential vitamin, The two major active forms of vitamin D in mammals are ergocalciferol (vitamin D_2) and cholecalciferol (vitamin D_3). There also is increasing use of 25-hydroxy vitamin D_3 in feeds for livestock. Oversupplementation and unintentional cross-contamination can cause vitamin D_3 excess in pet food.

Vitamin D poisoning occurred following prolonged ingestion of the contaminated food, usually after weeks of exposure. Cholecalciferol is rapidly absorbed from the gastrointestinal tract and transported to the liver, where it is rapidly broken down to 25-hydroxy vitamin D_3. This is further metabolized primarily to 1,25-dihydroxy vitamin D_3 (calcitriol) and 24,25-dihydroxy vitamin D_3 in renal proximal convoluted tubular epithelium. Calcitriol is the vitamin D metabolite that is most important in calcium-phosphorus metabolism.

Vitamin D has a major role in regulation of calcium and phosphorous. Clinical signs of vitamin D poisoning in pets include depression, weakness, anorexia, polyuria, and polydipsia. Diagnosis is based on clinical signs, decreased serum intact parathyroid hormone (PTH), increased total and ionized serum calcium, and serum 25-hydroxy vitamin D_3. Affected animals have gross and microscopic evidence of metastatic soft tissue mineralization on post-mortem examination and elevated concentrations of 25-hydroxy vitamin D_3 in kidneys.

Replacing the diet with uncontaminated food is often sufficient treatment for affected animals, but it can take weeks before clinical pathology changes resolve. More aggressive therapy to manage the imbalance of circulating calcium and phosphorus can include use of salmon calcitonin, pamidronate disodium and corticosteroids.[18]

There have been three recent cat food recalls in the U.S. due to inadequate thiamine supplementation. Thiamine is a required B vitamin (B_1). Monogastric animals like cats and dogs cannot synthesize thiamine, and because it is a water-soluble vitamin, there is no long-term storage in the body. Other factors, such as age and diet, affect the thiamine requirements for dogs and cats.[19] Pet foods should contain at least 5 mg/kg and 1 mg/kg thiamine on a dry matter basis for cats and dogs, respectively.[20] Thiamine deficiency in cats has been associated with a food containing 0.56 mg thiamine/kg, dry matter.[21]

Polioencephalomalacia describes the lesion associated with thiamine deficiency. Cats presenting during the 2009 recall exhibited anorexia, head tilting, dilated pupils, apparent blindness, circling, ataxia, seizures, positional ventroflexion of the head, and, in one case, marked extensor rigidity of the front limbs. All were responsive to thiamine treatment except the one with extensor rigidity. A study of puppies found that the first signs occurred after nearly two months on a thiamine-deficient diet and some individuals died before the abrupt onset of neurologic signs.[22]

Bilaterally symmetric changes in the central nervous system have been observed in affected dogs and cats using magnetic resonance imaging (MRI). Lesions were documented in the cerebellar nodulus, caudal colliculi and periaqueductal grey matter in dogs and in the red nuclei, vestibular nuclei, facial nuclei, and medial vestibular nuclei in cats.[23] MRI lesions correlated with those found on necropsy. The most common functional test for thiamine deficiency is erythrocyte transketolase activity, which has been used in humans and dogs, but no reference values are available for cats.[21] The reported thiamine pyrophosphate concentration is 32 μg/dL in healthy feline blood and 8.4 to 10.4 μg/dL in healthy canine blood.[22] Cats in the 2009 outbreak had blood thiamine pyrophosphate concentrations ranging from 2.1 to 3.9 μg/dL, but no samples from unaffected cats were analyzed.

Most affected animals respond to thiamine supplementation, as noted above. Thiamine hydrochloride is given parenterally at a dose of 100 to 250 mg/day for cats and 5 to 250 mg/day for dogs. After five days of parenteral dosing in cats, oral thiamine at 25 mg/day is continued for one month.[24] Improvement is usually rapid, with significant improvement observed within a few days and often complete recovery in one to 12 weeks.[21,23,25] However, persistent ataxia, hearing loss and positional nystagmus also are reported.[23]

Adulteration

Adulteration of pet foods is rare but was responsible for the largest pet food recall in U.S. history. Unknown to pet food manufacturers in the U.S. and other countries, melamine was

fraudulently added to some ingredients, which were produced by companies in China, in order to enhance the apparent protein content. Protein in pet foods is estimated based on the nitrogen content. Because melamine is 67% nitrogen by molecular weight, its addition increases the nitrogen content and thus the apparent protein content of the product. Melamine has numerous uses in industry and manufacturing, including pigment and polymer production but is not used as an ingredient in food production.

Early in 2007, there were several reports of renal failure in cats and dogs consuming commercial pet foods in the U.S. Clinical signs included inappetance, vomiting, polyuria, polydipsia, and lethargy. A large number of affected cats were on feeding trials at a laboratory.[26] A recall was initiated on March 15, and melamine was detected in the cat food two weeks later. Previous studies had proved melamine to have low oral toxicity, and it was not understood how or if melamine was producing morbidity and mortality, but this became clear later. Cyanuric acid, ammelide and ammeline, all structurally similar to melamine, also were detected in affected pet food products. The oral LD_{50} of melamine is 3200 mg/kg in male rats, 3800 mg/kg in female rats, 3300 mg/kg in male mice and 7000 mg/kg in female mice, but the combination of melamine and cyanuric acid is markedly more toxic to most animals than either compound alone. Cats fed diets containing 0.2% each of melamine and cyanuric acid had evidence of acute renal failure within 48 hours.[27]

The FDA investigation determined that wheat gluten and rice protein concentrates used in pet food production were intentionally mislabeled by Chinese exporters and actually contained wheat flour and poor-quality rice protein, respectively, mixed with melamine.[26] Samples of imported wheat gluten contained 8.4% melamine, 5.3% cyanuric acid, 2.3% ammelide, and 1.7% ammeline.[3] Eventually, >150 contaminated pet foods were identified, containing up to 3200 ppm melamine and 600 ppm cyanuric acid. Estimates of the number of pets affected range from hundreds to thousands. Many consider the 2007 pet food recall a sentinel event. A year later, melamine contamination of Chinese baby formula and other milk-based products was detected.

Clinical signs in cats ingesting contaminated food included inappetance, vomiting, polyuria, polydipsia, and lethargy. Urine specific gravities <1.035 and elevated serum urea nitrogen and creatinine concentrations were documented. Circular green-brown crystals were observed in urine sediment. Post-mortem examinations typically noted bilateral renomegaly and evidence of uremia. Microscopic lesions were primarily localized to the kidneys: renal tubular necrosis, tubular rupture and epithelial regeneration. The distal convoluted tubules contained large golden-brown birefringent crystals (15 to 80 micrometers in diameter) with centrally radiating striations, sometimes in concentric rings and smaller amorphous crystals.[26] Crystals from kidneys and urine contained 70% cyanuric acid and 30% melamine based on infrared spectra.[7] Melamine and cyanuric acid form crystals by binding to form a lattice structure. The optimal pH for this reaction is 5.8.[7]

Analysis of 451 cases matching the definition of melamine toxicosis found that 65.5% were cats and 34.4% were dogs. The case mortality rates were 73.3% and 61.5% for affected dogs and cats, respectively. Older animals and those with preexisting conditions were less likely to survive.[3] However, >80% of exposed cats during the original feeding trials survived with supportive care.[26]

Preventing Contamination

It is the manufacturer's responsibility to prevent contamination of pet foods.[28] This requires the use of safe ingredients from trustworthy sources, excellent sanitation, processing procedures that avoid contamination and destroy microbes, and excellent record keeping so that problems that occur can be traced. The Association of American Feed Control Officials (AAFCO) is composed of individuals responsible for enforcing laws related to animal feed and foods. The objective of the organization is to promote uniformity in the laws and enforcement of those laws. AAFCO publises a model of Good Manufacturing Practices (GMP) that can be used at the manufacturing or regulatory level to ensure food and feed quality. These include:

- Training of personnel
- Facilities and equipment maintenance
- Ingredient handling and tracking
- Packaging and labeling
- Storage and inventory of finished products
- Inspection and sampling of finished products
- Record keeping

A Hazard Analysis and Critical Control Point (HACCP) program, required by law, is used by manufacturers to identify points in the production process where contamination is most likely to occur. Steps to developing a HACCP program include:

- Consider all aspects of manufacturing, including ingredients, equipment, processes, storage, and distribution
- Analyze potential hazards based on probability and severity
- Control hazards at the critical control point (CCP)
- Document how CCPs will be monitored and problems handled

Obviously, the organization and cleanliness of the factory and personnel are of primary importance. Factories must be kept arthropod-free and rodent-free through insect- and rodent-proof construction, use of bait stations around the outdoor periphery, and, when needed, traps within the facilities. Surfaces within facilities should be routinely screened for contamination with *Enterobacteriacea* spp, many of which are human commensals. Personnel should wear clean uniforms and personal protective equipment with no visible long hair, buttons, jewelry, or other loose objects.

All ingredients received at the factory should be fit for purpose and within preset quality specifications. Certain ingredients come

with a certificate of analysis (COA) from the vendor. HACCP programs can require routine testing of ingredients, even those that come with a COA. Near-infrared (NIR) spectroscopy can be used as part of the routine testing program. A library of NIR spectra of all ingredients should be available. Matching the incoming ingredients' spectra to spectra in the library assures purity of the ingredients. The risk of misformulation is greatly minimized through rapid turnover of ingredients, highly automated production and constant maintenance of equipment.

Ingredients and food-grade chemicals should be kept in separate locations from nonfood-grade chemicals. Materials can be color coded. For example, those in white containers are finished product, yellow containers hold byproducts, gray containers hold waste, black contain nonfood material, etc.

Goals of HACCP in Pet Food Production

The goals of HACCP include consideration of all possible hazards associated with ingredients, manufacturing processes and storage. Three of the most important CCPs are the incoming raw ingredients, the thermal processing step (extrusion process for dry foods), and the external coating of kibble products.

Another goal of HACCP is to determine which hazards are of greatest importance due to probability and the severity of the problem. Mycotoxin contamination is a ubiquitous problem, and the mycotoxins of primary importance in the U.S. are aflatoxin, which can be lethal, and deoxynivalenol, which can cause significant morbidity and distress. The bacterial organisms of most concern based on pathogenicity, environmental persistence and likelihood of contamination are *Salmonella* spp and *Enterobacteriaceae*.

Mycotoxins can be produced in field and stored grain or in the stored final product, if fungal contamination is present. One CCP at the factory is the arrival of the grain. Mycotoxin contamination is prevented through thorough testing of multiple samples from all grain lots. Any grain containing 20 ppb or greater aflatoxin cannot be used due to FDA regulations. FDA also recommends that ingredients used in animal foods contain less than 5 ppm DON. Fungal contamination of the final product is prevented through factory cleanliness and pasteurization of the ingredients during manufacture.

The nature of certain raw ingredients makes it impossible to avoid bacterial contamination. A CCP for prevention of bacterial contamination, particularly *Salmonella*, is the extrusion process for kibble or the retorting process for canned pet foods. Prevention of bacterial contamination is achieved through pasteurization of the product. The extrusion process and retorting time and temperature are constantly monitored to be certain that adequate temperatures are reached to kill pathogenic bacteria.

Because it occurs after thermal processing, there is the risk for bacterial contamination during the coating process in dry foods. Therefore, all coatings must be certified as pathogen-free.

Some of the considerations for HACCP are mentioned here, but other aspects of manufacture, such as drying and bagging, also must be evaluated and routinely monitored. Monitoring of CCPs can be automated and all information stored in an accessible database that can be viewed at any time during or after production. HACCP data should be analyzed and confirmed to be within specification before a lot of product can be released. Product lots should be subsampled, and samples analyzed and warehoused for long-term stability determination and ease of retrieval.

Under this type of HACCP program, an error in any part of the manufacture, from formulation to final packaging or analysis of the final product for contaminants and nutritional value, warrants a stop in the production line. The problem is brought to the attention of the appropriate personnel, including on-site and off-site experts, for consultation. Production does not begin until a solution can be initiated.

Conclusion

With a myriad of possible contaminants, ranging from bacteria to fungal metabolites like aflatoxin and vomitoxin, to misformulations producing nutritional excesses and deficiencies, to adulteration with industrial chemical such as melamine and related compounds, it is impossible to predict the cause of the next pet food recall. Nevertheless, pet food recalls are quite rare compared to recalls for food products for human consumption, and compared to other causes, illnesses associated with pet food are rare. Pet food manufacturers have instituted HACCP and ingredient control practices that minimize the risk of contamination, mixing errors and adulteration, and assure product safety and quality. When rare contamination incidents occur, vigilance on the part of regulators, manufacturers and veterinarians is our major line of defense.

Acknowledgements: The author would like to thank Drs. S.Y. Sanderson and S. Gluckman for their work with the aflatoxin dogs, Dr. S. McDonough for his pathology work, Drs. K. Woosley and A. Hubbard for their work with the PEM cats, Dr. H.G. Kang for his thiamine analysis, Dr. R. Pusoonthornthum for information about hypervitaminosis A, Dr. R. Goldstein for his work with melamine-poisoned cats, and Dr. W. Rumbeiha for his expertise in vitamin D toxicosis, and Drs. L.H. Thompson and D.P. Laflamme for their expertise and assistance with this document.

References

1. Feng T, Keller LR, Wang L, et al. Product quality risk perception and decisions: Contaminated pet food and lead-painted toys. *Risk Anal*. 2010;30:15732-15890.

2. Dzanis D. Anatomy of a recall. *Top Comp An Med*. 2008;23: 133-136.

3. Rumbeiha W, Morrison J. A review of class I and class II pet

food recalls involving chemical contaminants from 1996 to 2008. *J Med Toxicol*. 2011;7:60-66.

4. Jerky treats from China could be causing illness in pets. *J Am Vet Med Assoc*. 2007;231:1183.

5. Bhalla V, Grimm PC, Chertow GM, et al. Melamine nephrotoxicity: An emerging epidemic in an era of globalilzation. *Kidney Intl*. 2009;75:774-779.

6. Cocchi M, Vascellari M, Galina A, et al. Canine nephrotoxicosis induced by melamine-contaminated pet food in Italy. *J Vet Med Sci*. 2010;72:103-107.

7. Osborne CA, Lulich JP, Ulrich JL. Melamine and cyanuric acid-induced crystaluria, uroliths, and nephrotoxicity in dogs and cats. *Vet Clin N Am Sm An.* 2008; 39:1-14.

8. Hughs DM, Gahl MJ, Graham CH, et al. Overt signs of toxicity to dogs and cats of dietary deoxynivalenol. *J An Sci.* 1999;3;693-711.

9. Puschner, B. Mycotoxins. *Vet Clin N Am Small Anim*. 2002;32: 409-419.

10. Meerdink GL. Mycotoxins. In *Clinical Veterinary Toxicology*. Plumlee KH (ed). Mosby, St. Louis, MO. 2004; 231.

11. Newbern PM, Butler WH. Acute and chronic effects of aflatoxin on the liver of domestic and laboratory animals: a review. *Cancer Res*. 1969;29:236.

12. Liggett AD, Colvin BM, Beaver BW, et al. Canine aflatoxicosis: A continuing problem. *Vet Hum Toxicol*. 1986;28:428-430.

13. Stenske KA, Smith JR, Shelly JN, et al. Aflatoxicosis in dogs and dealing with suspected contaminated commercial foods. *J Am Vet Med Assoc*. 2006;228:1686.

14. Miller DM, Wilson DE. Veterinary diseases related to aflatoxins. In *The Toxicology of Aflatoxins*. Eaton DL, Groopman JD (eds). Academic Press, San Diego, CA. 1994;347-364.

15. Bastianello SS, Nesbit JW, Willliams MC, et al. Pathological findings in a natural outbreak of aflatoxicosis in dogs. *J Vet Res*. 1987;64:635-640.

16. Dereszynski DM, Center S, Randolph JF, et al. Clinical and clinicopathologic features of dogs that consumed foodborne hepatotoxic aflatoxins: 72 cases (2005-2006). *J Am Vet Med Assoc*. 2008;232:1329-1337.

17. Polizopoulou ZS, Patsikas MN, Roubies N. Hypervitaminosis A in the cat: A case report and review of the literature. *J Feline Med Surg*. 2005;7:363-368.

18. Bischoff K, Rumbeiha W. Pet food recalls. *Vet Clin NA Sm An*. 2012;42(2):237-250.

19. Singh M, Thompson M, Sullivan N. Thiamin deficiency in dogs due to the feeding of sulphite-preserved meat. *Aust Vet J*. 2005;85:412-417.

20. Steel R. Thiamin deficiency in a cat associated with the preservation of 'pet meat' with sulfur dioxide. *Aust Vet J*. 2005;75:719-721.

21. Davidson, M. Thiamin deficiency in a colony of cats. *Vet Rec*. 1992;130:94-97.

22. Read DH, Harrington DD. Experimentally induced thiamine deficiency in Beagle dogs: Clinical observations. *Am J Vet Res*. 1981;42:984-991.

23. Garosi LS, Dennis R, Platt SR, et al. Thiamin deficiency in a dog: Clinical, clinicopathologic, and magnetic resonance imaging findings. *J Vet Intern Med*. 2003;17:719-723.

24. Plumb D. *Veterinary Drug Handbook*. PharmaVet Publishing, White Bear Lake, MN. 2002;4:788-789.

25. Studdert VP, Lubac RH. Thiamin deficiency in cats and dogs associated with feeding meet preserved with sulfur dioxide. *Aust Vet J*. 1991;68:54-57.

26. Cianciolo RE, Bischoff K, Ebel JG, et al. Clinicopathologic, histologic, and toxicologic findings in 70 cats inadvertently exposed to pet food contaminated with melamine and cyanuric acid. *J Am Vet Med Assoc*. 2008;233:729-737.

27. Puschner B, Poppenga RH, Lowenstine LJ, et al. Assessment of melamine and cyanuric acid toxicity in cats. *J Vet Diag Invest*. 2007;19:616-624.

28. Eirmann L, Cowell C, Thompson L. Pet food safety: The roles of government, manufacturers, and veterinarians. *Compendium*. 2012;E1-E3.

Ensuring Responsible Sourcing for a Safe, Quality and Sustainable Supply of Ingredients for Pet Diets

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Abstract

This review will focus on the procurement of quality ingredients and the guidelines needed to ensure responsible sourcing for the long term. In today's dynamic ingredient climate, pet food ingredients require constant monitoring regarding preferred suppliers, detailed ingredient specifications and quality testing programs. The monitoring is not only limited to quality parameters but also availability, traceability and, of course, sustainability. These and other ingredient sourcing considerations will be discussed.

Glossary of Abbreviations

HACCP: Hazard Analysis & Critical Control Points
ISO 22000: International Organization for Standardization 22000
NGO: Nongovernmental organization

Introduction

In today's market, pet food manufacturers need to consider multiple factors in the selection of vendors and the evaluation and monitoring of ingredients that are incorporated into pet products. A team approach (Figure 1) between technical and procurement is a method that is needed to ensure that all bases and details of the ingredients are considered for vendors to be approved.

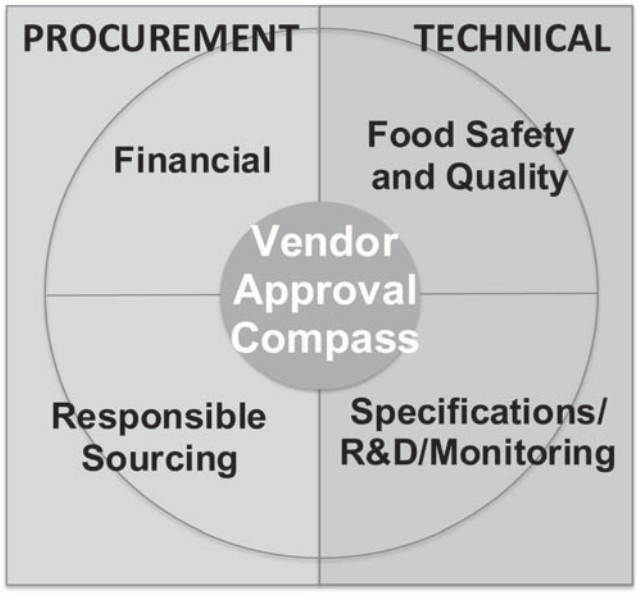

Figure 1

Technical Responsibility

Technical is responsible for ensuring that ingredients used in pet foods comply with local regulations and that the vendor's material meets these requirements. Routine on-site audits using the principles of ISO 22000 and HACCP conducted by technical personnel help to ensure that vendors meet the required regulations and quality. Along with food safety and quality, technical communities (R&D and application groups) have the responsibility to ensure that the ingredients used in pet foods meet detailed requirements outlined in an ingredient specification that is designed by the pet food manufacturer.

Depending on the individual ingredient, specifications may be, in part, based on trading standards outlined by local agriculture ministries or departments (e.g., USDA Corn Grades – Table 1) or other commonly recognized guidelines. Typically, pet food manufacturers include additional limits to parameters in specifications to meet safety or quality requirements. These parameters can be necessary to meet the needs of the application for which the ingredient is intended.

Safety of pet food ingredients involving compounds that may pose a health concern (e.g., mycotoxins) is the responsibility of Technical. Many of these type compounds have been defined where limits or recommended safe levels have been established by regulatory agencies. Pet food manufacturers need to continually work closely with their vendors and monitor incoming ingredients for these compounds to keep them within safe limits.

Complete ingredient specifications need to be fully understood by Procurement and vendors. Monitoring of parameters in an ingredient specification and auditing are key components to ensure the safety and quality of ingredients and compliance by vendors. Each ingredient has unique requirements for a technical audit. The more complex the ingredient, the more detailed the audit needs to be.

In addition, the frequency of a technical audit can vary based on ingredient type, confidence in the vendor and vendor location. Technical audit reports need to be complete and shared with both

Table 1: Grades and Grade Requirements of Corn

	Maximum Limits	Maximum Limits		
Grade	Test weight per bushel (pounds)	Heat-damaged kernels (percent)	Damaged kernels total (percent)	Broken corn and foreign material (percent))
U.S. No. 1	56.0	0.1	3.0	2.0
U.S. No. 2	54.0	0.2	5.0	3.0
U.S. No. 3	52.0	0.5	7.0	4.0
U.S. No. 4	49.0	1.0	10.0	5.0
U.S. No. 5	46.0	3.0	15.0	7.0
U.S. Sample Grade:				
U.S. Sample Grade is corn that: (a) Does not meet the requirements for grades U.S. No.1, 2, 3, 4 or 5; (b) Contains stones that have an aggregate weight in excess of 0.1 percent of the sample weight, two or more pieces of glass, three or more crotalaria seeds (*Crotalaria* spp.), two or more castor beans (*Ricinus communis L.*), four or more particles of an unknown foreign substance(s) or a commonly recognized harmful or toxic substance(s), eight or more cockleburs (*Xanthium* spp.) or similar seeds singly or in combination, or animal filth in excess of 0.20 percent in 1,000 grams; (c) Has a musty, sour or commercially objectionable foreign odor; or (d) Is heating or otherwise of distinctly low quality.				
Source: Grain Inspection Handbook – Book II; Corn Chapter 4, Corn Section Number 4.1, Section Grades and Grade Requirements, Page Number 4.2; U.S. Department of Agriculture, Grain Inspection, Packers and Stockyards Administration, Federal Grain Inspection Service 6/18/07.				

Procurement and the vendor. Audit deficiencies and corresponding corrective actions with timelines need to be completed as quickly as possible and agreed upon based on the severity of the deficiency. A follow-up audit or verifying evidence of completing the corrective action is the responsibility of Technical.

Roles of Procurement

Procurement has numerous roles beyond purchasing ingredients. Procurement is the key point of contact to the vendor on almost every facet. When engaging with a vendor, Procurement may request additional requirements of the vendor and have them agree upon (this is a "must" at Nestlé Purina). These may include:

- Supplier Business Principles (a code of conduct)
- Responsible Sourcing
- Traceability
- Sustainability
- Animal Welfare

If these types of requirements are deemed necessary, the first responsibility of Procurement would be to ensure that vendors acknowledge and agree to a set of business principles and a code of conduct. The principles would include items, but are not limited to, a responsibility to business integrity, sustainability, labor standard, safety and health, and environment. Secondly, Procurement must conduct an audit of these principles and/or responsible sourcing criteria including the corrective action plans and verification or follow-up of these actions. Continual monitoring of vendors using periodic audits is a part of Procurement's responsibilities regarding business principles.

Procurement has an obligation to define the true origin of the ingredients purchased. Traceability is critical in order to understand the processing or handling that ingredients have been subjected. Sourcing and quality requirements for ingredients need to be maintained for each upstream touch point or handling step. Procurement along with Technical must outline the degree of upstream auditing and monitoring necessary to ensure integrity of the ingredients purchased.

All industries should share the responsibility of global sustainability and the impact their respective ingredients have on the environment. Pet food manufacturers along with ingredient vendors need to work with regulatory and environmental groups to understand the impact they have on the environment. Reputable consultants and nongovernmental organizations (NGOs) can be of great assistance to pet food manufacturers in identifying sustainability issues and outlining guidelines to follow and require of vendors. Currently, specific fish species and palm oils are two examples of ingredients with high potential sustainability issues.

Procurement and Technical need to have a clear understanding with vendors to ensure proper steps and measures are in place to address sustainability. Approved techniques in securing raw material (e.g., fish harvesting practices) and harvesting quotas need to be enforced to avoid impact on the environment from a sustainability standpoint. Vendor techniques need to be monitored

through periodic audits along with analytical monitoring of ingredients supplied. This is a responsibility of both Procurement and Technical.

Pet food manufacturers have responsibility to assess vendor techniques and methods regarding ingredients they purchase originating from animals. Animal welfare and treatment of animals need to align with established guidelines and practices outlined by regulatory and governmental agencies. As with sustainability, reputable consultants and NGOs can be of great assistance to pet food manufacturers to ensure vendors use approved techniques in the handling and treatment of animals. Procurement and Technical need to account for animal welfare through the auditing process not only from the direct vendor but also for upstream touch points in the product stream.

Finally, a three-way understanding of the quality requirements and sourcing requirements is agreed upon.

- The ingredient vendor has the responsibility to consistently meet these requirements outlined by both Technical and Procurement.
- Technical has the responsibility to monitor the standards and quality limits outlined to meet the needs of the products for which the ingredient is intended.
- Procurement has the responsibility to monitor and enforce the requirements and corrective actions determined through the monitoring and auditing process.

Preferred Vendors

Cost of ingredients on a unit basis is important in any consideration of vendors. However, preferred vendors are not necessarily those that provide the lowest competitive ingredient unit price. Pet food manufacturers typically establish a preferred vendor list of those that have established a record of consistent compliance to meeting ingredient requirements and service performance.

Preferred vendors can and often do result in the overall lowest delivered cost when considering the time and efforts associated with poor vendor compliance and performance. The efforts and costs associated with dealing with delivered ingredients that do not meet specifications can be enormous. Below is a short list of some of the typical issues of poor performing vendors that have an impact on cost other than unit price for the ingredient:

- Late delivery — resulting in loss of production
- Delivery of the wrong ingredient — resulting in loss of production and/or manufacturing out of specification product and recall
- Delivery of ingredients out of specification — resulting in loss of production
- Routinely having issues with meeting requirements — resulting in a higher level of monitoring and added costs

Currently, the ingredient industry is characteristic of dynamic pricing fluctuations and for some ingredients availability is an issue. The demand for ingredients is highly competitive within and between industries. The economic changes taking place in Asia, Latin America and many developing countries have also had an impact on the availability of ingredients. Increased internal growth of food production in developing regions and movement of raw material production location from one region to another also are creating ingredient availability issues in some areas.

These conditions along with changes in buying power among industries are causing the pet food industry to search for new ingredients, new sources of nutrients and additional importation for ingredient solutions. Now, more than ever, pet food manufacturers need to know their vendors and work more closely with them to ensure that ingredient safety and quality specifications are maintained and that responsible sourcing methods are current and within the established guidelines to meet today's needs.

Ingredient Myths That Have Altered the Course of Pet Food: Byproducts, Synthetic Preservatives and Grain

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The number of myths about ingredients used in pet food is near limitless. For those trained in nutrition or medicine, they often seem the stuff of pulp fiction. Despite this, there are a few deeply held misunderstandings bordering on "urban legend" that seem to have more emotional impact than others. From the author's perspective developing and formulating pet foods for nearly 20 years, these myths have been so pervasive that they have altered the direction of pet food product development, not always to the benefit of the pet or owner. These misunderstood ingredient categories are often prominent "No list items" one finds on a package. They are oft maligned as cheap, harmful or even toxic and include the "Byproducts," "Artificial Preservatives" and "Grains" like corn and wheat. Of course, there are other prominent ingredient myths. These include: Citric acid causes bloat; salt is used as a flavoring; euthanized pets end up in meat and bone meals; carrageenan and sodium selenite are carcinogens; vitamin K3/menadione leads to kidney disease; beet pulp causes "red coat"; wild-caught, free-range and organic ingredients are more nutritious than farm-raised meats; and the list goes on and on. Byproducts, preservatives and grains seem to be maligned more than they deserve. What do we know about these ingredients, what is the thread of truth that undergirds the myth, and is there anything that can be done to rectify the situation?

Byproducts

Of course, the term "byproduct" sounds inferior right from the start. By the Association of American Feed Control Officials (2012) definition, they are "secondary products produced in addition to the principal product." In a zero-sum world of "first or worst," one would assume that second is undesirable, but are these compounds really second rate?

Consider the following: By the letter of the regulations, the byproduct moniker really only applies to organ meats (viscera and entrails) and to heads and feet of poultry. It doesn't apply to ingredients like beet pulp, wheat bran, soybean meal, or distillers dried grains and solubles per se. But clearly under the overarching definition, these ingredients should be considered byproducts. Most of the ingredients we use to produce pet foods (and even many human foods) are the secondary products, aka byproducts. The crux of the matter is a regulatory definition, not a common sense one.

On another point, byproduct organ meats include such parts of the anatomy as the glandular stomach, small intestine, large intestine, heart, liver, lungs, spleen, kidney, bladder, udder, and others. These were part of canid and felid diets long before man came onto the scene. Following a successful hunt our pets' ancestors, and their wild cousins of today, ripped into the soft underbelly of their prey as a first course. This is probably because it's an easier entry point to the body cavity, but also because these organs represent a rapid source of nourishment. For small wild felids that subsist primarily on a diet of rodents, they will eat their prey whole — organs included. In this day of "wild," "primordial" and "ancestral" diets, it is all the more surprising that organ meats have the connotation of being an inferior product. The data on the topic would suggest quite the contrary. As it turns out, organ meats have a high-nutrient density, favorable nutrient profile and are highly digestible. For example, Aldrich and Daristotle (1998) reported that chicken viscera and viscera plus hearts and liver had a protein quality similar to chicken meat, and Cramer, et al. (2007) reported that pork, beef and sheep lungs had a superior protein quality to that of chicken and fish.

Much of the organ meats today wind up in the various rendered protein meals, such as poultry byproduct meal, meat and bone meal, pork meal, lamb meal, or fish meal. These byproduct organ meats are processed in USDA inspected facilities and are intended for human consumption. The discrimination against their use is a Western bias, not a nutritional or safety matter. Today, there are a few products extolling the virtue of organ meats in canned and dry extruded pet food applications, so perhaps the negative connotation of nondescript byproducts has begun to wane. As Angele Thompson put it in a recent review article (2008), "There is a lot more to a cow than a hamburger and to a chicken than chicken breast." The "more" she was referring to are organ meats. Opening our minds to their use in dog and cat diets clearly expands our base of quality raw materials, provides flavor the pet relishes, and supports the quality of a nutritionally complete pet diet. As it relates to organ meats in pet food, the ski slope mantra says it best, "no guts, no glory."

Artificial Preservatives

The growing progression of natural products in the market has been strong and steady since the mid-1990s. It seems that today this is the "price of entry" into this competitive market. The avoidance of all compounds that are supposedly "synthetic" is not without its value from a safety perspective. There is a strong distrust of the chemical industry and residues in our foods and water stream. This fear is not completely unfounded because we've had some issues, but the modern chemical industry also has played a big part in liberating us from the tethers of subsistence living, increased our life expectancy due to advances in medicine, and made it possible to enjoy a wider range of travel, entertainment and leisure. But reliance on chemistry turns to outright avoidance when we consider synthetic chemicals in our or our pets' food.

The primary point of concern with the use of synthetic compounds in pet foods surrounds the antioxidant preservative ethoxyquin. This is a hindered phenolic amine that was initially introduced by Monsanto. It found favor in a number of applications as a commercial and industrial antioxidant preservative, ranging from retarding oxidation in food fats and preventing destruction of vitamins to retention of color in spices to preventing scald on apples and preserving rubber and plastics. Despite having passed Food and Drug Administration (FDA) and Environmental Protection Agency safety, efficacy and environmental impact studies, and having been fed safely to dogs at super-physiological levels, there have been concerns expressed by various activist groups, yet there isn't solid evidence that this compound when used at approved levels is harmful.

In the late 1980s and early 1990s, there were a number of anecdotal reports to the FDA by pet owners. These came principally from breeders reporting reproductive problems, cancers, itchy skin, etc., in their pets (Dzanis, 1991). Most of these claims have since been refuted, but not before forcing additional safety tests. A third series of studies (the first and second were completed in 1958 and 1964, respectively) were conducted as a voluntary 3.5-year, two-generation feeding trial of Beagle dogs in which ethoxyquin was included in the diet at 0, 180 and 360 ppm. This study, completed in 1996, concluded that no real issues were noted. However, the FDA in 1997 requested a voluntary reduction of ethoxyquin from 150 to 75 ppm in complete dog foods.

Given the level of sensitivity and perception in the market, they could have just as well outlawed the compound. While the FDA didn't outright say it was unsafe, it was the message consumer groups took away. The net outcome started the march toward "naturally preserved" foods and swept other preservatives like BHA, BHT (antioxidant) and potassium sorbate (mold inhibitors) out of favor for use in pet foods.

The challenge is that nearly 70% of the market is dry extruded or baked foods that are packaged in bags and boxes with an expectation of a one-year shelf life or better. These products are susceptible to oxidation leading to staling, rancidity and nutrient losses. Compound that with the current trend of retailers requesting an 18-month shelf life. The problem is the leading "natural" option is based on mixed tocopherols (vitamin E compounds) as the platform for the natural alternative preservatives. Many of the suppliers of natural preservative systems have demonstrated that mixed tocopherol-based systems can achieve a targeted shelf life for ingredients and pet foods (e.g., one year), but the dose and cost of mixed tocopherols can be five to 10 times that of synthetic antioxidants, such as BHA, BHT or ethoxyquin. Plus, mixed tocopherol systems are not quite as robust when exposed to more extreme conditions (Gross, et al.,1994) or as forgiving if underapplied. Further, the natural-based products are derived from the soybean oil refining industry and have themselves never been validated as safe and effective under the same premise as the synthetic preservatives. While some may be pleased that naturally preserved foods are now more common, we've not necessarily improved the quality, value or overall safety of the food for our pets in the process.

Grains — Corn and Wheat

The modern pet food industry has for the most part evolved from the livestock feed industry. Because of these beginnings, pet food, especially dry pet foods, have predominately been produced from animal protein meals like meat and bone meal in combination with grains and vegetable protein concentrates like corn gluten meal and soybean meal. No longer do we look at our pets as mere animals (livestock), but rather as a member of the home, the "furry children" as it were. With this changing status some of the key ingredients in their food have been scrutinized more. It's not surprising that common ingredients like corn and wheat have received some blame for pet nutrition and health issues, but the myth that grains cause food hypersensitivities in all pets may be unfounded. Rather than a grain issue, it could be an individual animal issue.

If we isolate this to just corn and wheat, what do we know? From a nutritional perspective, corn is readily utilized with consistent results (Murray, et al., 1999; Twomey, et al., 2002; Walker, et al., 1994; Carciofi, et al., 2004). Not unlike other plant sources, it has an incomplete amino acid profile with lysine, methionine and tryptophan as the first limiting amino acids, but this can readily be offset with a legume protein like soy. Contrary to claims on the Web, there are very few case reports of food hypersensitivity or food intolerance to corn reported in the literature (Hand, et al., 2000). On the positive side, yellow No. 2 corn derives its name from the pigments zeaxanthin and β-carotene, carotenoids that add color to foods and support to nervous and immune systems. Corn also is a rich source of the essential fatty acid linoleic acid (C18:2n-6).

Wheat is not merely a source of nutrients, it also imparts "architecture" to the food. In general, consumers put wheat into

the "carb category" and don't give it much more consideration. This isn't too far off since whole wheat is about 50% starch and wheat flour is about 70% starch (the difference being removal of the bran and germ layers during processing). The real story for wheat is not in the starch fraction, rather what makes wheat truly unique among the grains is protein. Generally speaking, it has a high-protein content (up to 18% in some varieties), most of which is gluten. Gluten is a mix of proteins (e.g., glutenin and gliadin), which, on average, comprise about 85% of the total protein found in wheat. Gluten is indelibly connected with "rising" in bread; it imparts viscosity and elasticity to dough and allows it to trap the CO2 bubbles produced by the fermentation of yeast. Once baked, the dough around these bubbles solidifies to form cells and create texture. Therein lays an opportunity that has yet to be fully exploited by pet food. From a nutritional perspective, wheat is well-utilized (Sunvold and Bouchard, 1998; Murra, et al., 1999; Bouchard and Sunvold, 2000) with lysine, methionine and threonine, the first limiting amino acids. Wheat is one of the top three ingredients typically identified with food hypersensitivities in dogs. Much of this can be attributed to gluten. For example, there is a specific intolerance in Irish Setters similar to celiac disease in humans. Celiac disease is an inflammatory disease of the small intestine associated with gluten intake. This gluten sensitivity is likely immune-mediated, but the underlying mechanisms are not fully understood at this point.

Clearly the issues with the grains are not nutritional per se but linked to specific animals predisposed to sensitivities. Whether this can be identified within certain animals before it becomes an issue is something for nutragenomics to address. In the meantime, corn and wheat appear to be suitable sources of nutrition for the bulk of our pets. Whether that plays well to the marketing manager or the pet owner might be more of a question.

Conclusions

The myths surrounding ingredients used in pet foods have led us down a path of more segmentation in the market. This has created new product categories, including the so-called "No Byproducts," "Holistic," "Natural," "Organic," Grain Free," and "Limited Ingredient Diet." It has increased the depth and range of product offerings on top of age, life stage and breed/type-based diets. So, the outcome hasn't all been bad. As with many marketing strategies, these things tend to cycle. One day pet owners might request foods for their pets made from byproducts and grains that were preserved with artificial preservatives — maybe for the first kennel on Mars.

References

Association of American Feed Control Officials. Official Publication. 2012.

Aldrich CG, Daristotle L. Petfood and the economic impact. *Proc Calif An Nutr Conf*. 1998;140-148.

Bouchard GF, Sunvold GD. Effect of dietary carbohydrate source on postprandial plasma glucose and insulin concentration in cats. In *Recent Advances in Canine and Feline Nutrition. Proc Iams Nutr Symp*. 2000;3:91-101

Carciofi AC, Takakura FS, Prada F. Evaluation of starch sources for canine diets. *Proc of the Alltech Nutr Biotech in the Feed and Food Industries*. Lyons and Jacques (eds). Abstract Supplement. 2004;8.

Cramer KR, Greenwood MW, Moritz JS, et al. Protein quality of various raw and rendered byproduct meals commonly incorporated into companion animal diets. *J An Sci*. 2007;85:3285-3293.

Dzanis DA. Safety of ethoxyquin in dog foods. *J Nutr*. 1991;121: S163-S164.

Gross KL, Bollinger R, Thawnghmung P, et al. Effect of three different preservative systems on the stability of extruded dog food subjected to ambient and high temperature storage. *J Nutr*. 1994;124:2638S-2642S.

Hand, Thatcher, Remillard, et al. *Sm An Cli Nutr*. Mark Morris Institute. Topeka, KS. 2000;4.

Murray SM, Fahey Jr. GC, Merchen NR, et al. Evaluation of selected high-starch flours as ingredients in canine diets. *J An Sci*. 1999;77:2180-2186.

Thompson A. Ingredients: Where Pet Food Starts. *Top Comp An Med*. 2008;23:127-132.

Twomey LN, Pethick DW, Rowe JR, et al. The use of sorghum and corn as alternatives to rice in dog foods. *J Nutr*. 2002;132: 1704S-1705S.

Walker JA, Harmon DL, Gross KL, et al. Evaluation of nutrient utilization in the canine using the ileal cannulation technique. *J Nutr*. 1994;124:2672S-2676S.

Impact of Extrusion on Pet Food Nutrition

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Abstract

Extrusion technology offers several benefits for producing pet foods. Other than economic benefits, the most important benefit is in the area of pet food nutrition. Extrusion cooking will improve the digestibility of raw starch and other ingredients, help to improve the functionality of proteins, and destroy or eliminate the anti-nutritional factors found in ingredients used for pet food production. Extrusion cooking also helps to minimize the enzymatic rancidity of the ingredients, and most importantly, the primary function of extrusion cooking is pasteurization of the feed ingredients, reduction of microbial populations, and destruction of pathogenic organisms.

Extrusion is a process used extensively in the pet food industry to produce products that are low density and light in texture, such as dog and cat foods. Ingredients or mixes are moved through a barrel, where they are guided by a single- or double-screw configuration with increasing restrictions, and then forced through a die, thereby generating heat and pressure. As the product exits the die, sudden expansion results from the pressure differential.

At the same time, we want to make sure the products being produced are safe for handling and consumption. There are several myths about extrusion claiming that high-temperature cooking destroys the nutritional value of the ingredients. It is true, to some extent, if someone is not careful with the extrusion operation, but the extrusion cooking process is a moderate temperature/short-time process. In general, extrusion cooking conditions do not destroy the nutritional value of the ingredients. On the other hand, this cooking helps cook the starch, denature the protein, destroy the anti-nutritional factor and insect larvae, eliminate the enzymatic rancidity, reduce the toxins, and pasteurize the raw material so there are no microbes in the finished product. Extrusion does not affect fat availability, and new extruder designs have much improved vitamin retention.

Extrusion Effect on Starch

Native starch is a good texture stabilizer and regulator in food systems, but low-shear resistance, thermal resistance and high tendency toward retrogradation limit its use in some feed applications. A wide range of techniques is being used by the industry for processing various feed materials. Processing leads to an alteration in the food structure and also influences the nutritional characteristics of the feed including starch digestibility. The outcome of some recent studies related to different processing techniques and their effect on starch digestibility is presented in Table 1. Anguita, et al. (2006) observed that extrusion provoked a decrease in particle size compared to raw samples and affected digestibility. Traditional and conventional processing methods were compared with extrusion cooking, and their effects on bean starch digestibility were studied by Alonso, et al. (2000). Extrusion produced a higher increase in starch digestibility than other processing methods.

Effect of Extrusion on Protein

Denaturation of protein is the thermal processing of protein that lowers protein solubility and destroys the biological activity of enzymes and toxic proteins. For most mature species, lowering protein solubility renders the protein more digestible. Many species, however, immediately after birth will assimilate more protein if it is available in a highly soluble form. On occasion, studies have indicated that the adult stage (such as in shrimp) also can effectively utilize soluble proteins. Extrusion cooking can be managed to process protein to the degree of solubility desired. Researchers at Wageningen University and TNO recently looked at the effect of different extrusion conditions and product parameters. According to their findings, total lysine and other amino acids were unaffected by the extrusion conditions employed. Extrusion conditions have a clear impact on the reactive lysine content with the ratio of reactive to total lysine increasing from 0.71 to 0.80 and higher as a result of extrusion and temperature (All About Feed, 2007).

De-Bittering Proteins

The most effective process of extrusion cooking involves the application of heat and moisture. Research confirms that an effective method of reducing the intensity of the raw flavor is to treat with steam. During extrusion processing, raw material and protein are treated with steam in preconditioners, and this has a de-bittering effect on the raw material. Research also shows that when differential diameter preconditioners (DDC) were used for making pet food, palatability was increased three to four times compared to pet food made with a single- or double-shaft pre-

Table 1. Effect of Processing on Starch Digestibility

Processing	Starch Digestibility	Reference
Cooking	34a	Roopa and Premavalli (2008)
Pressure Cooking	41a	
Autoclaving	39.7a	
Re-autoclaving	37.3a	
Puffing	33.4a	
Roasting	37.2a	
Baking	7.2a	
Frying	11.2a	
Germination	15.4a	
Malting	15.5a	
Toasting	31.8a	
Gamma irradiation	75.2a	Chung and Liu (2009)
Sheeting of pasta dough (3 passes)	156b	Kim, et al. (2008)
Sheeting of pasta dough (45 passes)	217b	
Dehulled beans	151c	Alonso, et al. (2000)
Germinated (48 h) beans	178c	
Extruded beans	306c	
Popped amaranth seeds	112d	Capriles, et al. (2008)
Cooked amaranth seeds	96d	
Flaked amaranth seeds	120d	
Extruded amaranth seeds	93d	

a Expressed as rapidly and slowly digestible starch (%)
b Expressed as rapidly and slowly digestible starch (%)
c Expressed as starch digestibility (%)
d Expressed as hydrolysis index (%)

Adopted from Singh, et al. (2010)

conditioner. A DDC design will give more retention time, thus increase the exposure of steam to the raw material.

Destruction of Trypsin Inhibitor

Raw soybeans cannot be used as such for animal feed or pet food because they contain several anti-nutritional factors. These factors are: trypsin and chymotrypsin inhibitors; phytohaemagglutinins (lectins); urease; allergenic factors; and lipases and lipoxygenases. Full, fat soybeans are thermally processed to destroy anti-nutritional factors and to increase oil availability while preserving the nutritional quality of the protein.

The major anti-nutritional factor of concern in raw soybeans are trypsin inhibitors as the others are generally destroyed or reduced if the trypsin inhibitor is destroyed. Trypsin inhibitor is a protease that interferes with protein digestibility in most animals and humans, and nutritionists have documented this effect conclusively. This protease enzyme can be inactivated by heat treatment. A reduction of at least 85% of the trypsin inhibitor units is considered necessary by feed technologists to avoid nutritional problems. Destruction of trypsin inhibitor can be achieved utilizing either "wet" or "dry" extrusion systems. A dry extrusion process is one in which mechanical energy from the extruder main drive motor is the only energy used to process the soybeans. A wet extrusion process not only uses mechanical energy but also steam that is injected into either a preconditioner or the extruder barrel. Either system can successfully process full, fat soybeans to be used in feed application or to reduce the levels of anti-nutritional factors in a complete pet food diet.

Soybean meal will have a similar anti-nutritional profile to full, fat soybeans. The reduction in anti-nutritional factors depends on the level of steaming prior to flaking/extraction and the level of toasting after extraction. Lightly toasted soybean meal will have significant levels of anti-nutritional factors. On the other hand, soy concentrates and soy isolates have further processing that usually reduces anti-nutritional levels below that which is of concern.

Effect of Extrusion on Total Glucosinolates and Phenols

The University of Novi Sad, Serbia, (Sakac, et al. 2006.) conducted a study on the influence of extrusion on total glucosinolates and total phenols in rapeseed. The application of the dry extrusion process on heat treatment of rapeseed in combination with the examined agricultural crops (corn, wheat, barley, triticale, alfalfa) led to the conclusion that applied heat treating results in feeds of satisfying nutritional-chemical profile, with the reduction of total glucosinolates in the range of 10 to 15% and preservation of total phenols. Many vegetable protein sources used in pet food nutrition also contain residual anti-nutritional factors that are reduced during extrusion of pet food diets.

Effect of Extrusion on Vitamin Stability

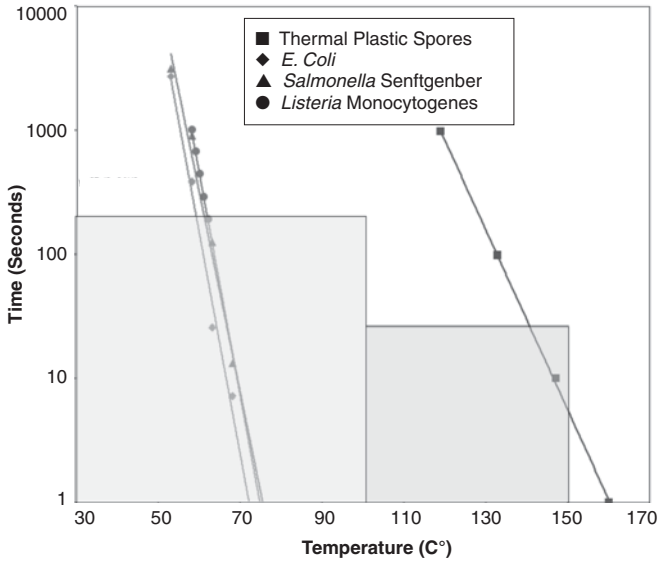
Depending on the particular vitamin and the process parameters employed during extrusion and drying, considerable degradation of vitamins can occur. The fat-soluble vitamins A, D and E are more stable during extrusion than the water-soluble vitamin C. The range of losses of vitamin varies, depending upon the vitamin source and the processing condition, such as increasing or decreasing extrusion moisture, decreasing or increasing residence time, decreasing and increasing extrusion temperature, and lower or higher mechanical energy inputs. Those processes that employ high-shear stress conditions during extrusion (direct expanded cereals and snacks, textured vegetable proteins) have very high losses during extrusion and drying. These products are fortified after extrusion/drying through external coating applications. Processes employing medium-shear stress conditions (pet

foods, aquatic foods, livestock feeds) will destroy 12 to 20% of the vitamins during processing. These products are fortified prior to extrusion with 12 to 20% overages to supply the required levels. Attempts have been made to apply vitamins externally during the coating steps, but losses are still substantial due to oxidation. New thermal extruders are much better regarding retention of vitamins and their stability. Initial results show that when using a thermal extruder, vitamin stability was increased significantly. Thermal extrusion technology depends on thermal energy rather than mechanical energy (shear) as the primary energy source. Many vitamins have been protected during their manufacturing steps to resist degradation. These "protective coatings" are destroyed when subjected to shear. The protective coatings remain more intact when thermal extrusion processes are employed.

Sanitation Via Extrusion

While many processing technologies result in an agglomerated feed, only a few have sufficient energy inputs to ensure food safety. Food safety is a major factor in choosing extrusion-based methods over traditional pelleting methods. Extrusion is a hydro-thermal process where the critical process parameters of retention time, moisture, and thermal and mechanical energy inputs can be varied over a wide range. The feed industry has considered a provision to mandate that all feeds are pasteurized and some form of extrusion will likely be implemented as the processing method of choice. The moderate temperature/short-time extrusion cooking process is able to accommodate a wide range of raw materials that might otherwise be discarded as unqualified material. Although extrusion does not completely eliminate toxins and other anti-nutritional or anti-growth factors, in many cases these substances or their activity is reduced to permit some level of incorporation into the recipe for animal feed.

Studies at Texas A&M University indicate that the extrusion process will reduce aflatoxin levels. Research at the University of Nebraska indicates that certain temperatures of extrusion are sufficient to reduce fumonisin levels. Studies also have indicated that viruses, molds and other pathogenic organisms can be destroyed by the operating parameters employed during extrusion. However, very little published data is available on this subject and there is a need for carefully designed studies to investigate the effects of the extrusion process.



Courtsey of Wenger Mfg.

Pasteurization and *Salmonella* Control with Extrusion

The feed industry is acutely aware of the need to eliminate the possibilities of foodborne illnesses from microbial contamination that can occur at any point along the food chain. Mandating that all feed be sterilized through processing was even considered by the U.S. government as a means to ensure public safety. Even though the practical aspect of such a mandate is questionable, feed processing techniques, such as extrusion, are in place that could fill the requirements of this type of program.

The graph "Thermal Death Time Studies" indicates the time/temperature relationship required to destroy common pathogenic microbes found in the feed and food industries. Note that *E. coli, salmonella* and *listeria* are destroyed if a temperature of 77º C is achieved for a one-second time interval during processing. The first, larger square in the graph outlines the average time/temperature possible in a DDC preconditioner (100o C for 120 seconds). The second, smaller square outlines the operating window for the extruder barrel. Note that the normal time/temperature employed in preconditioning and extrusion of pet foods and aquatic feeds will easily pasteurize feedstuffs and foodstuffs.

Effect of Extrusion on Microbial Population

Table 2 shows the effect of extrusion on microbial populations. Research shows that extrusion cooking can reduce the total plate count, coliform, mold count, *clostridium*, and *listeria*. The main

Table 2. Effect of Extrusion on Microbial Populations

Microbe	Raw Recipe Before Extrusion	Product After Extrusion
TPC (cfu/g)	240,000	9,300
Coliform	22,600	<10
Mold Count	54,540	<10
Clostridium	16,000	<10
Listeria	Positive	Negative
Salmonella	Negative	Negative

Courtsey of Wenger Mfg.

factor that contributes to the destruction of microbial is time, temperature, pressure, and mixing (turbulence in the extruder barrel).

Conclusion

In general, extrusion improves the nutritional quality of the raw material either making it more digestible or eliminating and destroying some toxic compound. The effect of extrusion on nutritional quality depends upon what kind of extruder is used to make pet food. Some extruders may provide too much shear and may damage some of the nutritional quality, but a majority of the extruders, if operated correctly, will enhance the nutritional quality of the pet food. Therefore, the myth that extruders always destroy the nutritional quality of pet food is not correct. On the contrary, an extruder will produce nutritionally balanced and safe pet food by pasteurizing the feed ingredients and enhancing digestibility.

References

Alonso R, Aguirre A, Marzo F. Effect of extrusion and traditional processing methods on antinutrients and *in vitro* digestibility of protein and starch in faba and kidney beans. *Food Chem*. 2000; 68:159-165.

Anguita M, Gasa J, Martı´ n-Oru´ e SM, et al. Study of the effect of technological processes on starch hydrolysis, nonstarch polysaccharides solubilization and physicochemical properties of different ingredients using a two-step *in vitro* system. *An Feed Sci and Tech*. 2006;129:99-115.

Capriles VD, Coelho KD, Guerra-Matias AC, et al. Effects of processing methods on amaranth starch digestibility and predicted glycemic index. *J of Food Sci*. 2008;73: H160-H164.

Chung HJ, Liu Q. Effect of gamma irradiation on molecular structure and physicochemical properties of corn starch. *J of Food Sci*. 2009;74:C353-C361. http://www.allaboutfeed.net/Nutrition/Diet-Formulation/2007/11/Extrusion-effect-on-quality-of-dog-food-AAF001318W/. Accessed Feb. 13, 2013.

Kim EHJ, Petrie JR, Motoi LM, et al. Effect of structural and physicochemical characteristics of the protein matrix in pasta on *in vitro* starch digestibility. *Food Biophy*. 2008;3:229-234.

Roopa S, Premavalli KS. Effect of processing on starch fractions in different varieties of finger millet. *Food Chem*. 2008;106:875-882.

Sakac MB, Filipovic SS, Borojevic CM, et. al. The influence of extrusion on total glucosinolates and total phenols in rapeseed. *Acta Agriculturae Serbica*. 2006;11:19-27.

Singh J, Dartois A, Kaur L. Starch digestibility in food matrix: A review. *Trends in Food Sci and Tech*. 2010;21:168-180.

Making Pet Food: Quality Assurance

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Abstract

This discussion will outline the concepts and structure of a Quality/Food Safety program that includes various processes and procedures to help ensure that the production and distribution of commercial pet food occurs in a safe, consistent manner, is compliant to appropriate regulation, and meets customer and consumer expectations.

Glossary of Abbreviations

CCP: Critical Control Point
GMP: Good Manufacturing Practices
HACCP: Hazard Analysis Critical Control Point
OPRP: Operational Prerequisites
PRP: Prerequisite Programs

In the development of an operational quality program, consideration should be given to the prioritization of programmatic quality system elements, among which there are many. There are three key expectations of commercial pet food: The product and package is food safe and is compliant to regulation, and the product is produced in a consistent manner, conforming to the respective specifications, including nutritional attributes. Focusing with first priority on the key elements that deliver these three expectations will help ensure a sustainable program that builds trust in the marketplace. The following list of quality system elements are core to the deliverable of the prioritization discussed above and will be reviewed in detail.

- Training
- Documentation
- Supplier Quality Assurance
- GMP – Product Protection
- GMP – Sanitation
- GMP – Pest Control
- GMP – Biosecurity
- HACCP
- Monitoring Scheme
- Instrument and Equipment Calibration
- Laboratories and Test Methods
- Release System
- Status Control
- Traceability, Lot Identification, Coding
- Recall and Crisis Management
- Corrective Action Program

Training and Documentation

Training and Documentation are fundamental to a quality program and are intrinsic to every other element mentioned above. One cannot exist without the other. The technical expertise required in our increasingly automated world with sophisticated technology is considerable. Respect must be given so as not to underestimate the resources, time and commitment to ensure that staff is appropriately trained and that there is a verification program that demonstrates competency. It is not uncommon that when quality gaps or failures are investigated, training was found to be insufficient.

The rigor of documentation is not only essential, it can be burdensome. Care must be taken that there is an appropriate balance between documentation and the practices on the factory floor. With the increasing documentation demands of regulatory requirements and various certifications (if any), maintenance of a documentation program can be overwhelming and even detrimental if not carefully managed. With the progressive move toward paperless systems and the usage of hyperlinks, change control can be a difficult challenge. It is easy to get tripped up with inconsistency among documented policies, procedures, methods, etc., if there is not a robust program to manage documentation and change control. The application and execution of a documentation program must be constantly reviewed.

Supplier Quality Assurance

One of the most discussed topics about pet foods is ingredients. There are a number of ingredient categories that are part of pet food formulas, each with its own unique contribution. Each material must go through its own risk assessment to identify the associated potential hazards. The outcome of the assessment then helps define what programmatic efforts are needed to support the ongoing assurance that the material used is safe, compliant to regulation and meets the defined specification.

Among the applicable programs would be surveillance and monitoring, including samples, questionnaires and surveys, site audits, and finally, sampling and testing upon receipt. While packaging is uniquely different, there are many different types and structures of packaging, and the appropriate risk assessment must be conducted as such.

GMPs

The acronym stands for **G**ood **M**anufacturing **P**ractices. Not only are GMPs a "good" thing to do as the title suggests, manu-

facturers of pet food must comply with GMP regulation, 21 CFR Part 110. Adherence to GMPs is not only a compliance concern, it also serves as the basis of discipline and respect for what is eventually the consumption of foodstuffs that all consumers partake. The maintenance and sustainability of a sound GMP program is not simple or easy. Sanitation, pest management, housekeeping, deep cleaning, product protection, and biosecurity are the key elements to the program and must be maintained throughout the facility. There have been numerous GMP gaps/failures identified in regulatory citations to various manufacturers providing fair warning for insufficient programs.

HACCP

The acronym stands for **H**azard **A**nalysis **C**ritical **C**ontrol **P**oint. The framework concept and fundamental principles of HACCP have been around for decades and have been perhaps the most discussed and debated among industry, academia and regulatory bodies. One of the most debated topics has been "What should or shouldn't be a CCP (Critical Control Point)?" Should GMPs be a CCP, for example? Thus, early HACCP plans had dozens of CCPs.

Over time, with much collaboration and dialogue, the HACCP approach has evolved into a full complimentary program covering multiple areas outlining increasing prioritization and consequence. Among the multiple areas that a HACCP program covers are PRPs (Prerequisite Programs), OPRPs (Operational Prerequisites) and CCPs, each being determined with the help of decision tree analysis. The level of severity escalates with loss of control from PRPs to OPRPs to CCPs.

Monitoring Scheme

In basic terms, this is Quality Control on the factory floor, from ingredient receiving through production and packaging, warehousing and distribution. A properly constructed monitoring scheme outlines the following: What is being checked, where it is being checked, by whom and at what frequency, the respective method/analysis, the applied limits, the documentation of record, and the action to take if results are outside the limits.

The construction of a Monitoring Scheme can be either sequential, by position or a combination of both. The application of a well-functioning Monitoring Scheme provides a number of things. First, it defines in unambiguous terms what is expected of the operator and where the boundaries start and stop. It also is a platform for training and change management. If the scheme is followed and executed as designed, it will reduce or prevent unacceptable deviations.

Instrument and Equipment Calibration

Increasing reliance is given to equipment and instrumentation operating the processes on the factory floor. Support is provided by complex programming logic through PLCs, which are programmable logic control systems. With this automation, a robust calibration program becomes ever more essential. Calibration of measuring and test equipment is simply verification that the output or results are accurate.

Prioritization within a calibration program must be given to that which ensures in the following order: personnel and food safety, quality and consistency, and lastly, monitoring and performance trending.

Laboratories and Test Methods

In factory operations, laboratory results provide part of the means necessary to determine product acceptability. Additionally, laboratory testing can be a support mechanism, providing valuable data that can aid the factory in improvement opportunities through monitoring and trending.

Proficiency in analytical testing for laboratory associates is ensured, in part, through a collaborative program whereby participants perform laboratory tests on a controlled sample. The results are compared with those generated by other analysts in the same or other laboratories using appropriate statistical procedures. Competency is determined by the results falling within a certain predefined range from the statistical mean. This approach helps ensure that sound methodology is being used and good laboratory practices are being followed, including internal control plans relevant to the methodology.

Release System and Status Control

A formal Release System and accompanying Status Control designate levels of authority for decisions to release product to the marketplace. In an appropriately designed Release System, products are automatically produced and warehoused in a restrictive status, meaning product cannot be shipped without the formal decision of a predefined authority. This authority is ultimately given to the factory manager who would typically delegate to the quality assurance manager.

Formal release by a designated authority happens when the designate has reviewed every preselected release norm for a given lot of product, has made the determination that said lot meets all release norms, and changes the status from restricted to available. Although quality control is happening in the background from the preceding element discussed and all results are within acceptable range, the final accountability rests with the release agent to ensure the product is fit for distribution.

Traceability, Lot Identification, Coding

With products being distributed for interstate commerce, it is essential to have an effective, efficient means by which to trace product that is moving through trade channels. One of the prerequisites is to have an accurate, legible code on the package. Package coding provides communication that is twofold: It informs the consumer of the product shelf life or "best if used by date," as well as factory specific information that is traceable back to the specific lot of production, including date, time, line number, etc.

Another essential element is an effective, accurate finished product traceability program that provides results in a timely manner. This application should be electronically based with well-trained staff to generate the necessary reporting and should be tested per the frequency outlined in the company policy. Time is of the essence when the decision has been made to retrieve product from the trade, and having such a system will pay great dividends should such an unfortunate situation arise.

Recall and Crisis Management

Having a well-documented and practiced Recall and Crisis Management program in the event of a marketplace situation, whether a food safety, regulatory or quality concern, is vital to protect the consumer. Quick and competent action also can minimize impact to the business. Many functional roles within an organization play a key part in the successful execution of product removal from the marketplace. Generally, the quality organization coordinates activities, working in concert with corporate communications, the face to the public; consumer services, the touch point to the consumer; the sales group, working with their respective customer partnerships; and the logistics and warehousing group, managing the flow of product returns.

Timing is critical in a crisis situation. High-level decisions need to be made by key stakeholders. When such decisions are made, the operational component needs to be executed flawlessly. Having the appropriate systems and applications are of utmost importance.

Corrective Action Program

Preventive quality assurance means that no unsatisfactory situation must be allowed to continue and that no quality failures are tolerated without attempting to remove the source of the hazard or failure.

The person(s) responsible for defining the procedures, performing the root cause analyses and then implementing the corrective action should be skilled in the application of problem-solving. The results of the corrective measures taken should be monitored over a period of time to verify that they are effective and have eliminated the problem. Corrective action plans may be the impetus for changes in suppliers of ingredients, product specifications, equipment design, and manufacturing instructions, to name a few.

In conclusion, it bears repeating that the aforementioned quality system elements do not comprise the entirety of a full and complete quality program. There are many other components that are complimentary and were not discussed here. The premise of this discussion was based on understanding the prioritization of the elements that deliver the three key expectations of commercial pet food: food safety, regulatory compliance, and product quality and consistency. One thing is common to all consumers: the demand for food safety and an interest in knowing how the related corporation assumes responsibility. People want to buy from companies they can trust.

Fuel for Felines: Cats and Carbohydrates

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Introduction

The physiology of cats regarding the metabolism of carbohydrates is similar in many respects to that of other mammals, but with key differences. Cats are obligatory carnivores and, as such, their natural diet consists primarily of fat and protein, and a small amount of carbohydrate. A bird or a mouse consists of approximately equal amounts of fat and protein and <5% carbohydrates. Commercial diets, however, contain on average 33% carbohydrates in dry food and 15% carbohydrates in canned food (Forrester D, et al. Consensus Statement. ACVIM. 2011). Cats have alterations in their metabolism that may lead one to conclude that they are ill-equipped to deal with dietary carbohydrates. It is for those reasons that it has long been discussed in the lay and scientific literature that carbohydrates cause obesity and diabetes mellitus. The premise is that high-carbohydrate intake drives overproduction of insulin resulting in excess fat deposition and obesity. High-carbohydrate intake has also been blamed for inducing chronic hyperglycemia, which increases demand on the beta cells to secrete insulin and leads to beta cell failure and diabetes. In this review, we will examine the validity of those statements in cats by focusing on physiologic mechanisms involved in carbohydrate use.

Glossary of Abbreviations

GK: Glucokinase
GLUT5: Glucose Transporter 5
IVGTT: Intravenous Glucose Tolerance Test
SGLT1: Sodium-Glucose Transporter 1

1. Physiology of Tasting, Digestion and Absorption of Carbohydrates

Sweet taste receptors in most mammals are heteromers consisting of T1R2 and T1R3 proteins. In cats, T1R3 is an expressed and likely functional receptor, whereas T1R2 is an unexpressed pseudogene. Because cats lack functional sweet receptors, they are neither attracted to nor show avoidance to the taste of sweet carbohydrates and high-intensity sweeteners, such as saccharin and cyclamate, and they avoid stimuli that taste bitter or sour to humans.[1]

Cats also lack salivary amylase, the enzyme involved in the initial digestion of starch. However, the effect of this is not known because amylase is also found in feline pancreas and chyme.[2] Compared to dogs, the activity of intestinal disaccharidases, such as sucrose and maltase, is lower in cats, whereas lactase activity is lower in some parts of the feline small intestine and much higher than in dogs in others.[3] Despite these differences, cats are capable of digesting cooked starch and various carbohydrates with an apparent digestibility of >94% in one study[4] and 89 to 100% in another.[5]

In the intestine, the sugars glucose and galactose, products of dissaccharidase digestion, are absorbed into enterocytes by Na+/Glucose Co-Transporter 1 (SGLT1) against an electrochemical gradient, whereas fructose is absorbed by facilitated diffusion by glucose transporter 5 (GLUT5). All are extruded into blood by GLUT2, a high-Km, low-affinity glucose transporter with characteristics similar to the GLUT2 in liver, pancreas and kidney. Many species can upregulate the capacity of the intestine to absorb glucose in response to high concentrations of dietary carbohydrate.[3,6-9]

It was shown in a study by Buddington and co-workers[6] that cats are unable to upregulate intestinal sugar absorption. However, these studies need to be evaluated with caution because the number of animals used was small (two and three per group on a high-protein and high-carbohydrate diet, respectively) and the cats were only 3 months old. Batchelor and co-workers showed that cats express SGLT1, and they suggested that the level of SGLT1 was sufficient for absorbing the carbohydrate content of their natural diet.[3] They also showed that the sweet receptor subunit T1R2 was not expressed in the feline intestine, limiting the capacity to upregulate the transport of sugars with increased intake.

While they concluded that this inability to upregulate the intestinal capacity to transport glucose suggests that high-carbohydrate diets are unsuitable for cats, they did not document that cats cannot upregulate glucose transport. What they did show is that the V_{max} of cats is about 50% that of dogs. However, the affinity for glucose is higher. It is impossible to deduce from this or other studies which concentration of sugars must be present in the lumen of the small intestine after the ingestion of carbohydrate-containing diets to exceed V_{max}, and whether such concentration would be reached with commercially available diets. Currently, there is no information on this topic, to my knowledge.

2. Physiology of Glucose Phosphorylation: Cats Lack Glucokinase

Glucokinase, the high-capacity, low-affinity glucose-phosphorylating enzyme, which has a Km for glucose of approximately 10 mmol/L, is present in brain, liver, beta cells, and the intestinal tract in humans and most other vertebrates. This enzyme is also called the glucosensor in beta cells and the brain because it is the rate-limiting enzyme for insulin and neurotransmitter release.[10,11] Cats do not have glucokinase, however, the activity of other hexokinases, which have a higher affinity for glucose than glucokinase, and other enzymes within the glycolytic pathway is upregulated.[12-14]

Regarding insulin secretion, a lack of beta cell glucokinase would be similar to maturity onset diabetes of the young type 2 (MODY2) in people, which is characterized by mild to severe hyperglycemia, depending on the mutation of the GK gene.[15] Healthy cats, however, do not show persistent hyperglycemia. In fact, cats have fasting glucose concentrations that are not different from those of humans or other mammals, and they are able to respond very rapidly to an IV or oral glucose bolus with insulin release.[16,17]

Phenotypically, there is great similarity in the insulin response of cats compared to other species, including the insulin secretion pattern. It has been suggested that glucose clearance seems to be delayed in cats compared to dogs,[18] based on two studies where a high dose of glucose (1 g/kg body weight) was administered during an intravenous glucose tolerance test (IVGTT) and a return to baseline was seen at 60 minutes in dogs[19] and at 90 minutes in cats.[16] The response to lower glucose dosages, however, appears similar among cats, dogs and humans,[16,20,21] although it has been shown in one study using a glucose dose of 500 mg/kg that baseline levels were not reached at 60 minutes. The variation in glucose values in that study were large during the last hour of testing and ranged from severe hypo- (12 mg/dl) to hyperglycemic values (223 mg/dl).[22] It is quite possible that in cats the high-dose IVGTT leads to attenuation of insulin-receptor kinase activity and signaling pathways involved in insulin-mediated glucose uptake leading to a delay in glucose clearance.[23,24] However, because glucose disposal is not only a function of insulin secretion and insulin-dependent glucose uptake but also includes glucose uptake by insulin-independent means and elimination through the kidney, other mechanisms may contribute to the slower clearance at high-glucose concentrations.

Insulin concentrations should not be compared among cats and other species in a quantitative way because of the lack of a feline-specific insulin assay. Based on the qualitative insulin response pattern to a glucose stimulus, it appears that cats compensate well for the lack of glucokinase. We also can assume that the lack of hepatic glucokinase is compensated by the upregulation of other hexokinases, as it has been shown that hyperglycemia is also the clinical sign for animals with isolated hepatic glucokinase deficiency,[25] and is, in part, due to impaired hepatic glycogen synthesis.[26] It has been documented that liver glycogen content in cats is similar to that of humans[27] and except for glucokinase, a deficiency of the other hexokinases that are responsible for the bulk of peripheral glucose uptake, has not been described.

The study by Curry and co-workers[28] is frequently cited as documentation that the beta cell response of cats differs from that of other species and that insulin release is higher with amino acids than with glucose. However, results from that study show that following a low dose of glucose, insulin secretion is higher during the first phase, whereas the second phase is equally stimulated by amino acids or low-dose glucose. Although the authors concluded that amino acids are more potent insulin secretagogues in the cat compared to other species, such a conclusion is confounded by the use of different concentrations and the nonuse of amino acids without glucose in the perfusion medium. Furthermore, no attempt was made to establish a dose-response relationship for the amino acid or glucose effect.

3. Physiology of Glucose Metabolism: Cats Can Adapt to Different Macronutrients

The notion that cats are unable to adjust their metabolism and are always gluconeogenic is based primarily on the results from a study by Rogers, et al. (1977) in which three cats fed a low- or high-protein diet lacked the ability to adapt levels of enzymes regulating amino acid catabolism, gluconeogenesis and ureagenesis.[29] Another study also showed that the gluconeogenic capacity of cats on a high-protein diet was already high in the fed state and no further increase was seen during fasting; however, in the same study, it was shown that cats have metabolic flexibility, because similar to omnivorous animals when fed a diet with higher carbohydrate content, they increased gluconeogenesis when fasted.

A further indication that cats can adjust their metabolic fluxes was the fact that cats on a high-carbohydrate diet have higher glycogen deposits and lower phosphoenolpyruvate kinase activity than those on a high-protein diet.[30] Results from other studies also support the notion that cats can adapt to variations in macronutrients in the diet. In several studies, it was shown that cats can adapt to increased protein by increasing amino acid oxidation and the activation of related enzymes;[31-33] other investigators have shown that cats can adapt to varying dietary fat concentrations.[34]

In our laboratory, we also have documented that cats show metabolic flexibility and increase glucose oxidation, glycogenesis and lipogenesis.[35] In lean male cats, the respiratory exchange ratio increased to >1 during a euglycemic hyperinsulinemic clamp indicating that these cats can replete their glycogen and lipid stores in response to insulin. In a recent study from our laboratory, we showed a difference between hepatic glucose fluxes when measured in the fasted and postprandial states, although little

effect of different diets was seen, likely because their differences in macronutrient composition were not large.[27] We also showed that the magnitude of postprandial gluconeogenesis and glycogenolysis in cats is not different from that seen in people. Six hours after food intake, glycogenolysis in cats contributed about 45% to total glucose production and about 55% to gluconeogenesis; in people, after intake of a 1000 Kcal meal, almost identical values were seen at approximately the same postprandial period,[36] demonstrating the importance of gluconeogenesis even in the postprandial state of omnivorous humans.

4. Physiology Meets Pathology?

Few studies of insulin and glucose concentrations after a meal have been conducted. Unfortunately, the majority have not been performed in a blinded fashion and may therefore be subject to bias. For obvious reasons, including differences in ingredients and manufacturing, as well as amounts of Kcal (i.e., the percentage of the daily food requirement), it is difficult to compare dietary studies. In one study, different amounts of starch led to dose-dependent increases in glucose levels, yet even with the highest amount of starch (34% dry matter) glucose concentrations remained well within the normal glucose concentrations range of cats.[37] Similarly, when different carbohydrate sources were examined, glucose concentrations remained in the low range of normal.[5] Even with extremely high levels of dietary carbohydrates, the majority of the glucose concentrations during a 24-hour observation period were well within the normal glucose range in lean cats.[38]

A recent publication showed that cats stay well within the normal glucose range during a 24-hour period.[39] Unfortunately, many times, any increase in postprandial glucose is incorrectly called hyperglycemia even when glucose concentrations never exceed the normal range. It appears unlikely and has not been shown that such normal glucose concentrations would have detrimental effects even long term. It is known that even in MODY2 patients with mild hyperglycemia, long-term glucose control remains stable for many years. Deteriorations were only seen when those patients gained excessive weight.[40]

Feeding studies often require animals to eat a calculated amount of food, which frequently is different than their maintenance requirement, within a short period of time and after a long fasting period. This is dissimilar from the normal eating behavior of a cat, and, therefore, results may not reflect what might be seen naturally. To overcome this potential pitfall, cats were monitored for several days with a continuous glucose monitoring system. It was documented that there was little daily variation in glucose concentration and that lean as well as obese cats, except for one obese diabetic cat, stayed well within the lower half of the normal glucose range after intake of a dry commercial diet with a carbohydrate content of 47% dry matter.[39]

Studies examining whether increasing dietary carbohydrate leads to higher postprandial insulin secretion have not shown consistent results. It has been speculated that a higher, long-term insulin secretion rate would lead to insulin resistance. It is questionable whether insulin concentrations elicited by glucose concentrations within the normal range would have long-term detrimental effects. It also has not been documented that diets with higher carbohydrate content lead to a change in insulin sensitivity.[27, 41]

Learning Points

The major culprit of the development of obesity and diabetes is likely not the carbohydrate content of the diet but rather the amount that the cats are fed. In a recent unpublished study, we found that the large majority of already obese and even diabetic cats still had unlimited access to food. This implies that the greatest need is for improved client education. This is especially important in view of the findings, which indicate that initially, with increasing obesity, cats increase their metabolic rate, but the rate becomes slower when body weight gain is >60% and/or obesity exists longer term.[42]

References

1. Li X, Li W, Wang H, et al. Cats lack a sweet taste receptor. *J Nutr.* 2006;136(7 Suppl):1932S-1934S.

2. Kienzle E. Carbohydrate metabolism of the cat. 1. Activity of amylase in the gastrointestinal tract of the cat. *J An Physiol An Nutr.* 1993;69:92-101.

3. Batchelor DJ, Al-Rammahi M, Moran AW, et al. Sodium/glucose cotransporter-1, sweet receptor, and disaccharidase expression in the intestine of the domestic dog and cat: Two species of different dietary habits. *Am J Physiol Regul Integr Comp Physiol.* 2011;300:R67-R75.

4. Thiess S, Becskei C, Tomsa K, et al. Effects of high-carbohydrate and high-fat diet on plasma metabolite levels and on IV glucose tolerance test in intact and neutered male cats. *J Feline Med Surg.* 2004;6(4):207-218.

5. de-Oliveira LD, Carciofi AC, Oliveira MC, et al. Effects of six carbohydrate sources on diet digestibility and postprandial glucose and insulin responses in cats. *J An Sci.* 2008;86(9): 2237-2246.

6. Buddington RK, Chen JW, Diamond JM. Dietary regulation of intestinal brush-border sugar and amino acid transport in carnivores. *Am J Physiol Regul Integr Comp Physiol.* 1991;261: R793-R801.

7. Dyer J, Al-Rammahi M, Waterfall L, et al. Adaptive response

of equine intestinal Na+/glucose co-transporter (SGLT1) to an increase in dietary soluble carbohydrate. *Pflügers Arch.* 2009; 458:419-430.

8. Shirazi-Beechey SP, Hirayama BA, Wang Y, et al. Ontogenic development of lamb intestinal sodium-glucose co-transporter is regulated by diet. *J Physiol.* 1991;437:699-708.

9. Wood IS, Dyer J, Hofmann RR, et al. Expression of the Na+/glucose co-transporter (SGLT1) in the intestine of domestic and wild ruminants. *Pflügers Arch.* 2000;441:155-162.

10. Matschinsky FM. Perspectives in diabetes. Glucokinase as glucose sensor and metabolic signal generator in pancreatic P-cells and hepatocytes. *Diabetes.* 1990;39:647-652.

11. Levin BE, Routh VH, Kang L, et al. Neuronal glucosensing: What do we know after 50 years? *Diabetes.* 2004;53:2521-2528.

12. Ballard FJ. Glucose utilization in mammalian liver. *Comp Biochem Physiol.* 1965;14:437-443.

13. Arai T, Kawaue T, Abe M, et al. Comparison of glucokinase activities in the peripheral leukocytes between dogs and cats. *Comp Biochem Physiol C Pharmacol Toxicol Endocrinol.* 1998;120:53-56.

14. Tanaka A, Inoue A, Takeguchi A, et al. Comparison of expression of glucokinase gene and activities of enzymes related to glucose metabolism in livers between dog and cat. *Vet Res Comm.* 2005;29:477-485.

15. Hussain K. Mutations in pancreatic ß-cell glucokinase as a cause of hyperinsulinaemic hypoglycaemia and neonatal diabetes mellitus. *Rev Endocr Metab Disord.* 2010;11:179-183.

16. Hoenig M, Alexander S, Holson J, et al. Influence of glucose dosage on interpretation of intravenous glucose tolerance tests in lean and obese cats. *J Vet Intern Med.* 2002;16:529-532.

17. Hoenig M, Jordan, ET, Ferguson, DC, et al. Oral glucose leads to a differential response in glucose, insulin and GLP-1 in lean versus obese cats. *Domest Anim Endocrinol.* 2010;38: 95-102.

18. Hewson-Hughes AK, Gilham MS, Upton S, et al. Postprandial glucose and insulin profiles following a glucose-loaded meal in cats and dogs. *Br J Nutr.* 2011;106(Suppl 1):S101-S104.

19. Church DB. A comparison of intravenous and oral glucose tolerance testing in the dog. *Res Vet Sci.* 1980;29:353-359.

20. Hahn RG, Ljunggren S, Larsen F, et al. A simple intravenous glucose tolerance test for assessment of insulin sensitivity. *Theo Bio and Med Mod.* 2011;8:1-12.

21. Kaneko JJ, Mattheeuws D, Rottiers RP, et al. Glucose tolerance and insulin response in diabetes mellitus in dogs. *J Sm An Pract.* 1977;18:85-94.

22. Appleton DJ, Rand JS, Priest P, et al. Determination of reference values for glucose tolerance, insulin tolerance and insulin sensitivity tests in clinically normal cats. *Am J Vet Res.* 2001;62: 630-636.

23. Pillay TS, Xiao S, Olefsky JM. Glucose-induced phosphorylation of the insulin receptor: Functional effects and characterization of phosphorylation sites. *J Clin Invest.* 1996;97:613-620.

24. Olefsky JM., Nolan JJ. Insulin resistance and non-insulin-dependent diabetes mellitus: cellular and molecular mechanisms. *Am J Clin Nutr.* 1995;61:980S-986S.

25. Zhang YL, Tan XH, Tan, MF, et al. Establishment of liver-specific glucokinase gene knockout mice: A new animal model for screening anti-diabetic drugs. *Acta Pharmacol Sin.* 2004;25: 1659-1665.

26. Gilberto Velho G, Petersen KF, Perseghin G, et al. Impaired hepatic glycogen synthesis in glucokinase-deficient (MODY-2) subjects *J Clin Invest.* 1996;98:1755-1761.

27. Hoenig M, Jordan ET, Glushka J, et al. Effect of macronutrients, age and obesity on 6- and 24-h postprandial glucose metabolism in cats. *Am J Physiol Regul Integr Comp Physiol.* 2011;301:R1798-R1807.

28. Curry DL, Morris JG, Rogers QR, et al. Dynamics of insulin and glucagon secretion by the isolated perfused cat pancreas. *Comp Biochem Physiol.* 1982;72A:333-338.

29. Rogers QR, Morris JG, Freedland RA. Lack of hepatic enzymatic adaptation to low- and high-dietary protein in the adult cat. *Enzyme.* 1977;22:348-356.

30. Kettelhut IC, Foss MC, Migliorini RH. Glucose homeostasis in a carnivorous animal (cat) and in rats fed a high-protein diet. *Am J Physiol Regul Integr Comp Physiol.* 1980;239:R437-R444.

31. Russell K, Lobley GE, Millward DJ. Whole-body protein turnover of a carnivore, *Felis silvestris catus*. *Br J Nutr.* 2003; 89:29-37.

32. Russell K, Murgatroyd PR, Batt RM. Net protein oxidation is adapted to dietary protein intake in domestic cats (*Felis silvestris catus*). *J Nutr.* 2002;132:456-460.

33. Green AS, Ramsey JJ, Villaverde C, et al. Cats are able to adapt protein oxidation to protein intake provided their requirement for dietary protein is met. *J Nutr.* 2008;138:1053-1060.

34. Lester T, Czarnecki-Maulden G, Lewis D. Cats increase fatty acid oxidation when isocalorically fed meat-based diets with increasing fat content. *Am J Physiol Regul Integr Comp Physiol.* 1999; 277:R878-R886.

35. Hoenig M, Thomaseth K, Waldron M, et al. Fatty acid turnover, substrate oxidation, and heat production in lean and obese cats during the euglycemic hyperinsulinemic clamp. *Domest Anim Endocrinol.* 2007;32:329-338.

36. Petersen KF, Price T, Cline GW, et al. Contribution of net hepatic glycogenolysis to glucose production during the early postprandial period. *Am J Physiol.* 1996;270(1 Pt 1):E186-E191.

37. Hewson-Hughes AK, Gilham, MS, Upton S, et al. The effect of dietary starch level on postprandial glucose and insulin concentrations in cats and dogs. *Br J Nutr.* 2011;106:S105-S109.

38. Coradini M, Rand JS, Morton JM, et al. Effects of two commercially available feline diets on glucose and insulin concentrations, insulin sensitivity and energetic efficiency of weight gain. *Br J Nutr.* 2011;106(Suppl 1):S64-S77.

39. Hoenig M, Pach N, Thomaseth K, et al. Evaluation of long-term glucose homeostasis in lean and obese cats using continuous glucose monitoring. *Am J Vet Res.* 2012;73:1100-1106.

40. Martin D, Bellanné-Chantelot C, Froguel P, et al. Long-term follow-up of oral glucose tolerance test–derived glucose tolerance and insulin secretion and insulin sensitivity indexes in subjects with glucokinase mutations (MODY2). *Diab Care.* 2008;31(7): 1321-1323.

41. Kley S, Hoenig M, Glushka J, et al. The impact of obesity, sex, and diet on hepatic glucose production in cats *Am J Physiol Regul Integr Comp Physiol.* 2009;296:R936-R943.

42. Hoenig M, Pach N, Thomaseth K, et al. Cats differ from other species in their cytokine and antioxidant enzyme response when developing diabetes. *Obesity.* doi:10.1002\oby.20306. (Epub ahead of print)

Feline Nutrition: What Is Excess Carbohydrate?

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Abstract

The domestic cat (*Felis domesticus*) is metabolically adapted to low-carbohydrate diets that provide about 2% of metabolic energy (ME). These adaptations limit the rate and the amount of carbohydrate utilization in the cat compared to omnivorous species. High-carbohydrate intake and carbohydrate excess have been implicated in certain health disorders, but evidence for a cause and effect relationship is lacking. Because of the variability in carbohydrate source, processing, nutrient interaction, and the health status of cats, the identification of uniform recommendations for carbohydrate excess is not possible. While recommendations to limit carbohydrates to 4-8 g/100 Kcal ME are published, evidence that higher carbohydrate intake represents an excess is lacking.

Glossary of Abbreviations

AAFCO: Association of American Feed Control Officials
BW: Body Weight
DM: Dry Matter
ME: Metabolic Energy
NEFAs: Non-Esterified Fatty Acids

Introduction

The nutritional requirements of the domestic cat exemplify evolutionary adaptation to a strict carnivorous diet and predatory lifestyle. The feral domestic cat (*Felis domesticus*) eats a wide variety of prey, including small mammals, birds, insects, reptiles, and other animals. Because this prey-directed diet is composed exclusively of animal tissues, the typical composition of the natural diet has limited carbohydrate availability. The primary source of carbohydrates in the natural diet of adult feral cats is derived from tissue glycogen and the gut contents of the prey. As the cat became domesticated, the nutritional profile of the diet changed to include larger proportions of carbohydrates from milk products, sugars, grains, and other vegetable matter. In contrast to a whole rat carcass (carbohydrate 1.18% DM, dry matter; 0.18 g/100 Kcal),[1] carbohydrates in commercial cat foods may provide up to 50-fold the amount found in the natural diet. Dry cat foods commonly provide 30-40% ME (8-10 g/100 Kcal) as carbohydrates or 33-45% DM. The divergence of nutritional profiles of commercial cat foods from the cat's natural diet has led to debate regarding the impact of high carbohydrate feeding in this obligate carnivore with concerns that these levels represent a carbohydrate excess.

While definitions for carbohydrate content of cat foods are not standardized, published classifications for various levels have suggested high (> 50% ME), moderate (26-50% ME), low (5-25% ME) and ultra low (< 5% ME).[2] This definition of carbohydrate level refers to the content of simple carbohydrates (digestible starches and sugar) in foods as opposed to complex carbohydrates, such as fibers or resistant starches, that are largely undigested. This discussion will focus on digestible carbohydrates. Despite the nutritional adequacy of modern commercial cat foods, consumption of high-dietary carbohydrate levels are suggested to alter energy metabolism, promote insulin resistance, and contribute to increased risk for obesity, diabetes mellitus and other health disorders in the cat.[3] Many studies have been published evaluating the impact of various dietary carbohydrate levels on changes in body weight, on plasma glucose and insulin concentrations, and on insulin sensitivity and biomarkers for disease risk. While the majority of studies do not support a direct cause-and-effect relationship of carbohydrate intake to risk for obesity or diabetes, the diversity of study designs, animal populations, feeding methods, and diet variables allows for alternate interpretations of study findings.[a] Because changes in dietary carbohydrate content require changes in the proportion of protein and/or fat, it is difficult to isolate carbohydrate's effect. The question remains whether some levels or sources of carbohydrates are excessive and deleterious to the health of the cat.

Feline Carbohydrate Metabolism

Carbohydrates are included in feline commercial foods as a readily available energy substrate, a source of dietary fiber or for functional properties in the processing of foods. Gums, mucilage and starch serve as gelling agent in canned foods; starch aids expansion and kibble structure in dry extruded diets; and simple sugars reduce water activity to help preserve semimoist foods and treats. The proportion of carbohydrates in commercial foods varies with lower levels typically found in canned foods and moderate to high levels found in dry foods. Most domestic cats

consume increased carbohydrate levels compared to their wild or feral counterparts, which derive approximately 2% ME from carbohydrates.[4]

Feline Requirement for Carbohydrates

Adult cats have evolved to a diet that is high in protein and low in carbohydrates. Nursing kittens consume up to 20% of their energy in the form of lactose, while the natural diet of adult cats is < 5% DM carbohydrate.[5] Similar to most mammals, adult cats do not have a dietary requirement for carbohydrates, although maximum lactation performance and minimization of weight loss are enhanced by higher carbohydrate intake in lactating queens.[6]

Evaluation of carbohydrate requirements in nursing neonatal kittens is not reported. Feeding studies using AAFCO protocols were conducted in kittens fed two raw meat formulations compared to a low-carbohydrate canned food. Health parameters and growth rates in kittens fed 0%, 3.9% or 7.3% DM carbohydrate were similar among female kittens and highest in male kittens consuming the 0% DM carbohydrate diet.[7] It is clear that kittens greater than 8 weeks of age have no dietary carbohydrate need if adequate protein is supplied to support gluconeogenesis.

Physiological Adaptations of Carbohydrate Metabolism

Several metabolic adaptations in carbohydrate metabolism differentiate the cat from more omnivorous species. Cats, like all other mammals, require metabolic glucose to support cellular requirements for ATP production in the glucose-requiring tissues, such as nervous tissues, red blood cells, renal medulla, and active reproductive tissues. Efficient metabolism of proteins via hepatic gluconeogenesis sustains blood glucose levels in the absence of dietary carbohydrates.

Several physiological changes in cats attest to their adaptation to low-carbohydrate diets. A cat's ability to rapidly digest and absorb sugars and carbohydrates is reduced through elimination, reduction or lack of adaptation of glucose metabolizing enzymes.[8] Cats lack salivary amylase, which is designed to initiate the digestion of starches, and in one study, were found to have pancreatic amylase levels that are 5% those of dogs.[9] Brush border disaccharidase activity is 40% that of dogs.[10] Disaccharidases, amylase, along with intestinal sugar transporters, are nonadaptive to changes in dietary carbohydrate levels.[8-12]

Enzymatic changes within the liver reflect the cat's evolutionary adaptation to a diet low in carbohydrates, especially simple sugars like glucose and fructose. The cat has low levels of hepatic glucokinase, which limit the ability to efficiently metabolize large glucose loads following dietary intake.[8,11] Reports of limited fructose utilization resulting in fructosemia and fuctosuria following high sucrose or fructose intake have implied there is a lack of active fructokinase in the liver of cats.[13] Interestingly, feline liver demonstrates fructokinase distribution similar to other animals and a higher activity of hexokinase compared to canine liver.[15,16] Therefore, altered enzyme activities do not necessarily limit total capacity for hepatic glucose uptake in cats as other pathways may be active; however, the rate of hepatic glucose disposal appears to be slowed.

Hepatic protein catabolic enzymes appear constitutively active and provide continuous carbon skeletons (ketoacids) for gluconeogenesis or oxidation. These adaptations facilitate ongoing gluconeogenesis to sustain blood glucose levels during consumption of a low-carbohydrate diet.[8,15]

Digestion and Utilization of Carbohydrates

The above metabolic adaptations have led many to suggest poor carbohydrate utilization by the cat and a state of adaptive insulin resistance described as "the carnivore connection."[3] In reality, cats readily digest, absorb and utilize many types and levels of dietary carbohydrates. Carbohydrate digestion in commercial foods is reported to be greater than 90% for starches and greater than 94% for most sugars, although some studies have reported values as low as 36% for uncooked potato starch.[13,14,17,18] While the impact of microbial fermentation of undigested starches in the colon likely overestimates apparent carbohydrate digestibility compared to true carbohydrate digestibility, the difference is not so great as to discount carbohydrates' high digestibility and intestinal uptake. Lactose digestion declines in weaning kittens, with both a decrease in intestinal lactase production and down-regulation of sugar transporters.[12] Kittens tolerate up to 6 grams lactose/kg per body weight (BW) during nursing,[5] while adult cats can consume more than 1.3 g/kg BW of lactose/day without adverse gastrointestinal signs.[13] Starch intake up to 5 g/kg BW/day is readily digested and was suggested by Keinzle[19] as an upper limit for the cat, while de-Oliveira demonstrated excellent starch digestibility up to 6.7 g/kg BW/day in adult cats.[18]

Experimental feeding studies using uncooked starches and high levels of sugars demonstrated limited capacity for rapid absorption or utilization of these ingredients in cats. Carbohydrate digestibility varies based on the starch or sugar source, processing, particle size (fine grinding versus course meal), and the diet matrix.[13,14,17-19] Thus, defining a level of carbohydrate excess is specific to many ingredient and processing criteria along with specificity of the effect.

Carbohydrate Excess

While cats do not require dietary carbohydrates, identifying a level of carbohydrate excess is not straightforward. The definition of a carbohydrate excess presumes a negative health effect from consumption above a defined level. When maximum capacity for carbohydrate digestion is exceeded, excessive fermentation and osmotic effects of undigested sugars in the gastrointestinal tract lead to bloating, gas production, discomfort, and diarrhea. Excess lactose consumption (greater than 1.3 g/kg BW/d) and above 5 g/kgBW/day for simple sugars and certain starches represents

a maximum intake for some adult cats without an increase in blood biomarkers of altered carbohydrate metabolism and various monosaccharide loss in the urine.[13,18] However, most cats tolerate complex starches in the food matrix at levels of a least 6.7 g/kg BW/d with no observable effect.[18]

Excess dietary carbohydrate also may be described as exceeding the practical amount of carbohydrate added to a food that will imbalance the nutrient profile by limiting the additions of other ingredients and diluting nutrient content. Based on the AAFCO nutrient guidelines[29] for adult cats, diets containing carbohydrates that are approximately 60% or greater DM or ME risks nutrient unbalance. Lesser carbohydrate amounts — 40% ME or 51% DM[19] — would impact kitten diets due to their higher protein requirements. Foods with marginal nutrient concentration or bioavailability may be altered by lower levels of carbohydrates, depending on the source and the impact of the altered nutrient digestion. Protein digestibility is sometimes reduced by high-carbohydrate feeding while the availability of certain minerals, e.g., phosphorus and magnesium, may actually increase with increased carbohydrate feeding.[21]

Carbohydrate Intake and Relationship to Disease

Levels of carbohydrate intake and source may influence metabolic function or disease parameters. Dietary carbohydrates, primarily in the form of sugars, have resulted in changes in water balance, glucose metabolism, secretion of gut incretins, and adipokines. While changes are observed in various metabolic pathways, evidence does not support carbohydrate excess as a factor but more likely reflects adaptive changes to altered nutrient profiles.

Carbohydrates and Urinary Tract Disorders

High-sugar diets may alter urinary tract health by exceeding the renal threshold for monosaccharides and disaccharide excretion resulting in increased water loss by osmotic diuresis or increasing risk for disease. Glycosuria was demonstrated in cats fed simple sugars from 11-40% DM, while cooked starches that were up to 40% of diet had no effect on renal glucose excretion or postprandial blood glucose level in healthy cats.[13] Renal histologic changes were noted in the kidney of a cat fed high sucrose (7.2 g/kg BW; 36.1 % DM).[13]

The impact of carbohydrate intake was reported in an epidemiological study by Lekcharoensuk, et al.[22] Cats fed increased carbohydrates in canned diets (mean carbohydrate 7.84 g/100 Kcal ME) were compared to the reference group (.52-4.15 g/100 Kcal ME). Cats fed increased carbohydrates were at increased risk of calcium oxalate urolithiases, while carbohydrate intake had no influence on struvite urolithiases.

Ocular Effects of Carbohydrates

Galactose toxicity was evident in intake of 5.6 g/kg BW in a single cat. Cataracts were observed in one cat fed 39% galactose and resolved with diet discontinuation.[13] Similar findings have been reported for other species but at higher levels of galactose. Thus, 5.6 g/kg BW of galactose intake clearly is in excess, but the threshold for such effect in cats is unknown.

Role in Satiety

Previous studies suggested high protein: low carbohydrate diets improved satiety by reduction of appetitive behaviors in cats. However, the effect of feeding carbohydrate levels at 0.94 g/100 Kcal (4.5 % DM) versus 10.2 g/100 Kcal (31.3% DM) demonstrated no difference in owner satisfaction related to appetitive behaviors in pet cats undergoing a weight -loss plan.[23]

Role in Obesity

Numerous laboratory and epidemiological studies have been published evaluating the impact of diet profile on the rate of weight gain and loss and the impact of neutering, energy requirements, glucose levels, and insulin response in healthy cats. While the results are not uniformly consistent, the major risk factors suggest excess energy consumption, neutering and limited exercise are the greatest risk factors for obesity.[a] Epidemiological studies suggest that high-carbohydrate, low-fat dry foods do not favor the development of obesity but that feeding energy-dense, high-fat dry foods in caloric excess is a dietary risk factor.[24] A benefit to feeding high-protein, low-carbohydrate foods during weight loss have been noted. There appears to be an energetic benefit to feeding high-protein diets for maintenance of lean body mass and the thermic effect of proteins. Most studies support the effect of weight loss and obesity reduction toward improved insulin sensitivity in obese cats.[a]

A recent paper investigated therapeutic low-carbohydrate foods and a moderate carbohydrate-high fiber for the management of diabetes mellitus compared to a maintenance high-carbohydrate diet. In five cats, a reduction of postprandial glycemia and insulin levels along with increased non-esterified fatty acids (NEFAs) were observed.[25] While the study is limited by the small number of cats, the findings suggest that low-carbohydrate intake or fiber fortification were beneficial as independent factors in improving glycemic regulation. Similar to this observation, mean plasma glucose concentrations were greater in cats fed 12.1 g carbohydrate/100 Kcal compared to 8.3 g or carbohydrate 3.2g /100 Kcal, estimated ME of 48%, 33% or 12.8%, respectively.[26]

An increase in fat and simple carbohydrate levels in rats and people are suggested to alter the gut microbiome toward lower levels of bacteroidetes and higher firmicutes, a pattern termed "obese microbiota" because of improved energy extraction from the diet in this microbial environment.[27,28] Diet is known to influence the microbial population of the feline intestine, but the influence of carbohydrates on an "obese microbiome" in cats has not been described.

While most studies suggest a limited role of carbohydrates

in the cause of obesity, the above studies suggest carbohydrates in excess of 12.1 g/100 Kcal (48% ME) may be inappropriate in obese cats at risk for insulin resistance and impaired glucose tolerance.

Role in Diabetes Mellitus

Low- to moderate-carbohydrate diets are beneficial in the management of diabetes mellitus. Utilizing the cat's natural metabolic adaptations to provide a steady source of glucose from the liver via gluconeogenesis and avoiding postprandial fluctuations in glucose absorption appears beneficial in managing diabetic cats.

Sustained hyperglycemia by IV glucose infusion contributes to glucose toxicity of the feline beta cell leading to decreased glucose sensing, impaired insulin secretion, hydropic degeneration, and beta cell death.[29] Models of sustained hyperglycemia, 29 mmol/l (522 mg/dl) by IV glucose infusion results in transient diabetes mellitus in the cat.[b] In addition to glucose toxicity, chronic hyperglycemia leads to chronic stimulation of insulin secretion from beta cells. In people, carbohydrate intolerance, insulin resistance and hypersecretion of insulin increase the risk of beta cell exhaustion, pancreatic amyloid accumulation and overt Type 2 diabetes mellitus. A similar relationship to high-carbohydrate feeding has been suggested to occur in the cat and may contribute to the development of diabetes mellitus although evidence to support this hypothesis is lacking.

The lowest level of glucose infusion required to initiate glucose toxicity in normal cats has not been fully identified but appears to be that amount needed to increase blood glucose to approximately 29 mmol/l (522 mg/dl). Lower levels of sustained hyperglycemia (17 mmol/l (310 mgl/dl) did not result in glucose toxicity.[b] Postprandial elevation of blood glucose in nonstressed healthy cats is unlikely to reach such levels. Dietary carbohydrate intake across a range of available carbohydrate sources providing 1 g/kg of available carbohydrate was not associated with significant increases in plasma glucose in healthy cats.[c] Dietary carbohydrate ingestion resulted in minimal postprandial blood glucose change when diabetic cats were allowed to eat free-choice.[30] In normal cats fed once daily, plasma glucose levels increased (mean levels 6.2 mmol/l (112 mg/dl) and 4.7 mmol/l (85 mg/dl), respectively, from eight or more hours postprandially and remained elevated up to 12 hours following the rise.[26,31,d] Further study is needed to assess the relationship of these findings to cats that are fed dietary carbohydrate twice daily, cats that are fed free-choice, or cats that are obese and insulin-resistant.

In diabetic cats treated with long-acting insulin and low-carbohydrate foods, several studies have resulted in variable improvements in glucose regulation and improved insulin sensitivity. The high rate of diabetic remission appears to more closely relate to control of obesity, but improved blood glucose regulation and reversal of glucose toxicity with low carbohydrate may contribute to the positive outcome. While diabetic regulation was similar in diets containing carbohydrate levels of 3.5 g/100 Kcal (12% ME) and 7.6 g/100 Kcal (26% ME), remission rates were increased in cats eating the lowest carbohydrate level.[32] In a study by Hall[33] comparing 1.1 g and 7.9 g/100 Kcal, a reduction in serum fructosamine levels was observed when feeding the lowest carbohydrate, but no differences in other parameters of glucose regulation or remission rates were noted.

At present, a finite limit of carbohydrate intake for the treatment of diabetic cats is uncertain. From experience and the limited studies, it would appear diabetic regulation may benefit from feeding low-carbohydrate levels below 20% ME or 25% DM. However, feeding diets with moderate-carbohydrate levels, especially when supplemented with fiber, provides similar glycemic control. A level of carbohydrate excess for the diabetic cat remains uncertain but would appear to be greater than 8.3 g/100 Kcal in low-fiber foods and possibly higher in fiber-fortified foods.

Summary

Diet composition is known to alter metabolic processes, health and disease risk. The domestic cat has adapted to a naturally low-carbohydrate diet (< 2% ME) with resulting limitations on carbohydrate utilization or rate of glucose disposal compared to more omnivorous species. Cats have no dietary requirement for carbohydrates, yet commercial diets commonly provide levels of carbohydrates well above the evolutionary nutritional profile. It has been suggested that high-carbohydrate levels, especially those in dry foods, predispose cats to disorders including glucose intolerance, insulin resistance, obesity, and Type II diabetes mellitus. To date, no solid evidence is available to support these hypotheses.

Current studies suggest that cats have a rate-limiting capacity to digest or utilize large amounts of simple sugars but tolerate a wide range of starches and complex carbohydrates. Carbohydrate excess is evident for certain simple sugars at levels greater than 1.3 g/kg BW/day, with maximum intake of rapidly available starch limited to 4 g/kg BW/day. Practical limits based on formulation constraints suggest an approximate maximum of 60% ME carbohydrates in adult diets and a maximum of 40% ME carbohydrates in kitten diets as higher carbohydrate levels would limit protein inclusion and thereby create a nutritionally deficient diet.

High-carbohydrate intake does not appear to increase the risk for obesity. Energy excess, low-energy expenditure, neutering, and high-fat intake have the highest association with obesity development in cats. Remission of Type 2 diabetes mellitus in cats may benefit from low-carbohydrate foods along with insulin to improve glycemic control. Suggested limits on dietary carbohydrate for cats with Type 2 diabetes mellitus are 20% ME (5.8 g/100 Kcal ME; 25% DM) in low-fiber foods. While higher levels of carbohydrate levels combined with dietary fiber provide acceptable control and remission rates of Type 2 diabetes mellitus, a finite level of carbohydrate excess has yet to be defined.

Footnotes

a ACVIM Consensus Statement: The role of dietary carbohydrate in causing and managing feline obesity and diabetes mellitus. In 29th Annual ACVIM (American College of Veterinary Internal Medicine) Forum. 2011.

b Link KRJ, Rand JS. Glucose toxicity in cats. *J Vet Intern Med*. 1996;10:185.

c Cave NJ, Monroe JA, Bridges JP. Dietary variables that predict glycemic response to whole foods in cats. *Comp Cont Ed for Vet*. 2008;30:57. (Abstract)

d Coradini MA, Rand JS, Morton JM, et al. Delayed gastric emptying may contribute to prolonged postprandial hyperglycemia in meal-fed cats. In 24th Annual ACVIM Forum. 2006.

References

1. Vondruska JF. The effect of a rat carcass diet on the Urinary pH of the cat. *Comp An Prac*. 1987;1:5-9

2. Rucinsky R, Cook A, Haley S, et al. AAHA diabetes management guidelines. *J Am An Hosp Assoc*. 2010;46(3):215-224.

3. Zoran DL. The carnivore connection to nutrition in cats. *J Am Vet Med Assoc*. 2002;221:1559-1567

4. Platinga EA, Bosch G, Hendriks WH. Estimation of dietary nutrient profiles of free-roaming feral cats: possible implications for nutrition of domestic cats. *Brit J Nutr.* 2011;106:S35-S48.

5. Meyer H. Lactose intake of carnivores. *Wiener Tierarztliche Monatsschrift*. 1992;79:236-441.

6. Piechota T, Rogers QR, Morris JG. Nitrogen requirement of cats during gestation and lactation. *Nut Res*. 1995;15:1535-1546.

7. Hamper BA. Nutritional adequacy and performance of raw food diets in kittens. Doctorate dissertation, University of Tennessee, Knoxville. 2012;67-78.

8. MacDonald ML, Rogers QR. Nutrition of the domestic cat, a mammalian carnivore. *Ann Rev Nutr*. 1984:4521-4562.

9. Kienzle E. Carbohydrate metabolism of the cat. 1. Activity of amylase in the gastrointestinal tract of the cat. *J Am Physio and An Nutr*. 1993;69:92-101.

10. Kienzle E. Carbohydrate metabolism of the cat. 4. Activity of maltase, isomaltase, sucrose, and lactase in the gastrointestinal tract in relation to age and diet. *J Am Physio and An Nutr*. 1993;70:289-296.

11. Baker DH, Czarnecki-Maulden GL. Comparative nutrition of cats and dogs. *Ann Rev Nutr*. 1991;11:239-263.

12. Buddington RK, Diamond J. Ontogenetic development of nutrient transporters in cat intestine. *Am J Phys Soc*. 1992; 263(5):G605-G616.

13. Kienzle E. Carbohydrate metabolism of the cat. 3. Digestion of sugars. *J Am Physio and An Nutr*. 1993; 69:203-210.

14. Kienzle E. Effect of carbohydrates on digestion in the cat. *J Nutr*. 1994; 124:2568S-2571S.

15. Tanaka A, Inoue A, Takeguchi T, et al. Comparison of expression of glucokinase gene and activities of enzymes related to glucose metabolism in livers between dog and cat. *Vet Res Comm*. 2005;29(.6):477-485.

16. Washizu T, Tanaka A., Sako T, et. al. Comparison of the activities of enzymes related to glycolysis and gluconeogenesis in the liver of dogs and cats. *Res Vet Sci*. 1999;67(2):205-206.

17. Morris JG, Trudell J, Pencovic T. Carbohydrate digestion by the domestic cat (*Felis catus*). *Br J Nutr*. 1977;37(3):365-373.

18. De-Oliveira LD, Carciofi AC, Oliveira MCC, et al. Effects of six carbohydrate sources on diet digestibility and postprandial glucose and insulin responses in cats. *J An Sci*. 2008; 86(9): 2237-2246.

19. Kienzle E. Studies to intestinal and intermediary metabolism of carbohydrates (amylum in different origin and preparation, mono- and disaccharides) in cats (*Felis catus*). *Tierarztlichen Hochschule Hannover*. 1989: 219-222. (dissertation)

20. Association of American Feed Control Officials. Official Publication. 2012.

21. National Research Council Subcommittee on Dog and Cat Nutrition. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006.

22. Lekcharoensuk C, Osborne CA, Lulich JP, et al. Association between dietary factors and feline calcium oxalate and magnesium ammonium phosphate uroliths. *J Am Vet Med Assoc*. 2001; 219: 1228-1237.

23. Cline M, Witzel A, Moyers T, et al. Comparison of high-fiber and low-carbohydrate diets on owner-perceived satiety of

cats during weight loss. *Am J An Vet Sci*. 2012;7:218-225.

24. Lund EM, Armstrong PJ, Kirk CA, et al. Prevalence and risk factors for obesity in adult cats from private U.S. veterinary practices. *Intern J Appl Res Vet Med*. 2005;3(2):88-96.

25. Mimura K, Mori A, Lee P, et al. Impact of commercially available diabetic prescription diets on short-term postprandial serum glucose, insulin, triglyceride, and free fatty acids concentrations of obese cats. *J Vet Med Sci*. 2013 (March 1).

26. Hewson-Hughes AK, Gilham MS, Upton S, et al. The effect of dietary starch level on postprandial glucose and insulin concentration in cats and dogs. *Brit J Nutr.* 2011;106:S105-S109

27. Turnbaugh PJ, Ley RE, Mahowald, MA, et al. An obesity-associated gut microbiome with increased capacity for energy harvest. *Nature*. 2006;444(7122):1027-1131.

28. Turnbaugh PJ, Bäckhed F, Fulton L, et al. Diet-induced obesity is linked to marked but reversible alterations in the mouse distal gut microbiome. *Cell Host Microbe*. 2008;3(4):213.

29. Dohan FC, Lukens FDW. Lesions of the pancreatic islets produced in cats by administration of glucose. *Science*. 1947; 105:183.

30. Martin GJW, Rand JS. Food intake and blood glucose in normal and diabetic cats fed *ad libitum*. *J Fel Med Surg*. 1999; 1(4):241-251.

31. Mori A, Sako T, Lee P, et.al. Comparison of three commercially available prescription diet regimens on short-term postprandial serum glucose and insulin concentrations in healthy cats. *Vet Res Comm*. 2009;33(7):669-680.

32. Bennett N, Greco DS, Peterson ME, et al. Comparison of a low carbohydrate-low fiber diet and a moderate carbohydrate-high fiber diet in the management of feline diabetes mellitus. *J Fel Med Surg*. 2006;8(2):73-84.

33. Hall TD, Mahony O, Rozanski EA, et al. Effects of diet on glucose control in cats with diabetes mellitus treated with twice daily insulin glargine. *J Fel Med Surg*. 2009;11:125-130.

Optimal and Natural as Rationale for Selecting Dietary Energy Distribution in Carbohydrate and Fat

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Abstract

Metabolism of dietary protein, fat and carbohydrate provides cats with energy, which they need and which compels them to eat. While most energy is derived from fat and carbohydrate, an optimal ratio of these nutrients is not established and may vary with nutritional aim. Only a few health-related variables are studied for determining optimal proportions of the nutrients. An energy ratio of carbohydrate to fat of 2 to 1 in dry diets may be optimal for reducing obesity risk. Clear evidence of optimal ratios in canned diets and for reducing diabetes risk is lacking and worthy of investigation.

Objectives

1. The primacy of energy in providing nutrition to cats.
2. Nutrients that provide energy and an unavoidable interdependence.
3. Rationale guiding mixture of fat and carbohydrate used in providing energy.
4. Optimal energy provision from fat and carbohydrate.
5. Gaps in understanding and future directions.

The Primacy of Energy in Providing Nutrition to Cats

More than 40 substances in or derived from food are necessary for life, growth, maintenance, and repair of body tissues.[1] Qualitatively and quantitatively unique among these substances is energy, which animals obtain from chemical bonds of metabolizable substances in food. With respect to quantity, the need for energy constitutes the largest mass of a diet, second only to water. For highly digestible diets, around 90% of the dry matter mass may be utilized in meeting energy need (Figure 1). Hence, the largest nutrient cost in feeding animals is invested in providing energy.

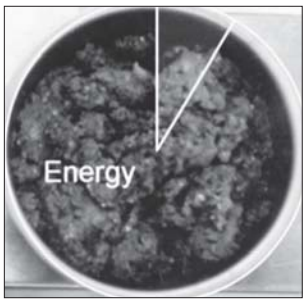

Figure 1. Dry matter mass proportion of diet needed to meet energy requirement.

Also unique about energy, animals need energy and this need compels them to eat. Said another way, most animals, including cats, eat to meet perceived energy needs,[1] though many other factors appear contributing.[2] The significance of this condition for feeding cats is great. Foremost is that the nutritional needs of cats can be met if all other nutrients in their diet are kept in proper proportion relative to dietary energy. With this proportionality, nutritional requirements of normal healthy cats are met or exceeded when they eat until they are no longer hungry. By contrast, malnutrition may readily result when energy-dense foods are added to a diet without regard for nutrient proportions with respect to energy. One of the many benefits of feeding commercially manufactured diets rather than home-prepared diets is the likelihood of matching energy with other nutrient needs.

Nutrients That Provide Energy and an Unavoidable Interdependence

Energy is derived primarily from metabolism of three nutrients: protein, fat and carbohydrate. Dietary carbohydrate in this context is predominately bioavailable starches and/or glycogen. Though provision of energy is not a property commonly ascribed to protein, utilization of protein ultimately yields metabolizable energy, as much energy as an equivalent mass of dietary carbohydrate. On a mass equivalency basis, dietary fat provides substantially more energy than protein and carbohydrate, on average 2.25 times more energy. Hence, diets that are energy dense are diets that are high in fat.

Because of the variety in energy-containing nutrients, the energy needs of cats may be met with a myriad of combinations of protein, fat and carbohydrate. This latitude has two important consequences. First, it prompts a question: *Is there an optimal combination of dietary protein, fat and carbohydrate for providing energy?*

In seeking an answer to this question, another question arises: *What outcome or outcomes should guide selection of an optimal combination?*

Answers to these questions vary depending how optimal is defined.

Another consequence of the variety in nutrient energy sources

is that change in the concentration of one nutrient will obligately change the proportion of one or both of the other nutrients. This interdependence limits the range of combinations of protein, fat and carbohydrate. A clinically relevant example is the aim to reduce carbohydrate in diets for cats with diabetes mellitus.[4] Such reduction must be accompanied by an energy equivalent increase in fat and/or protein (Figure 2). In diabetes, better glycemic regulation might be gained with a low-carbohydrate diet, but reciprocally increased fat in exchange for carbohydrate might further exacerbate existing insulin resistance. High dietary fat favors increased circulating non-esterified fatty acid concentration and body adiposity, both which reduce insulin sensitivity.[5] Alternatively, increasing protein in exchange for carbohydrate expectedly reduces alimentary glucose entry from carbohydrate, but it also increases protein catabolism, which favors glucose entry from hepatic gluconeogenesis.

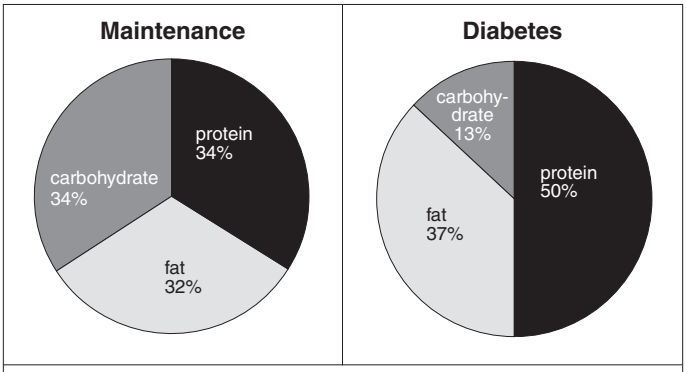

Figure 2. Energy distributions in protein, fat and carbohydrate in feline commercial dry diets formulated for diabetes (Purina Veterinary Diets® DM Dietetic Management® Feline Formula) and maintenance (Purina® Cat Chow® Complete Formula).

Rationale Guiding Mixture of Fat and Carbohydrate Used in Providing Energy

While there is heightened interest in feeding home-prepared diets, most cats are fed commercially prepared diets of the dry and canned (or wet) types.[6] It is common for cats to receive both canned and dry products, but energy needs mostly are met with dry diets. The popularity of feeding dry compared to canned diets relates to economy, convenience and consistency of palatability when left out for *ad libitum* consumption.

Dry compared to canned diets are generally, but not exclusively, higher in carbohydrate and lower in protein and fat. Energy from protein in maintenance dry diets appears fairly uniform across many commonly fed products. This condition is indicated from a survey in recent years of seven commercial feline dry diets of brands reported to account for two-thirds of the U.S. market share.[7] Protein energy in diets of this survey was a mean of 33% and ranged from 29 to 36% of dietary energy (Figure 3). Compared to protein, substantially more variation was observed in fat and

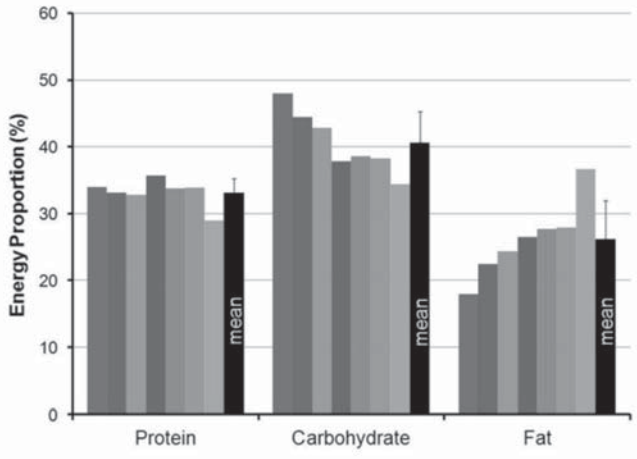

Figure 3. Energy distribution in protein, fat and carbohydrate in surveyed feline commercial dry diets.[7]

carbohydrate. Energy from carbohydrate was estimated to range from 34 to 48%, while energy from fat was generally lower and more variable, with some diets containing twice as much fat as others (18 to 37% of metabolizable energy).

The similarity of protein content in dry commercial diets likely reflects an aim of manufacturers to be above an "optimal" minimum while keeping products economical. Sources of protein typically are more costly than those of fat and carbohydrate. Results of a classic paradigm in nutrition form the basis of optimal, which, in this context, optimal is a range of protein concentrations. An optimal minimum is indicated from analysis of the relationship between a variable affected by the nutrient and the dietary concentration of the nutrient. An example of this definition is easy to understand for determining the growth requirement of kittens, when growth rate is plotted against dietary protein concentration.[8] With less than optimal dietary protein concentration, growth rate increases with increasing protein concentration (Figure 4B). A lower limit or minimum of optimal is identified when growth rate does not further increase in response to increasing dietary protein concentration (Figure 4C). Compared to dietary protein, optimal ranges of fat and carbohydrate are not as clearly identifiable. This difficulty reflects the nature of fat and carbohydrate as energy-yielding substances.

Though carbohydrate is one of three possible sources of energy, which is required, a dietary requirement for carbohydrate has not been established for cats. Hence, much flexibility in carbohydrate content is allowable in feline diets. In contrast, dietary requirements for fat have been established, but the requirement is low, much lower than what is common to commercial diets, for both dry and canned. Thus, for fat, as with carbohydrate, there is nutritional flexibility for a wide variation in allowable concentrations.

Although carbohydrate is not classically defined as nutritionally essential, its abundance in dry feline diets relates to manu-

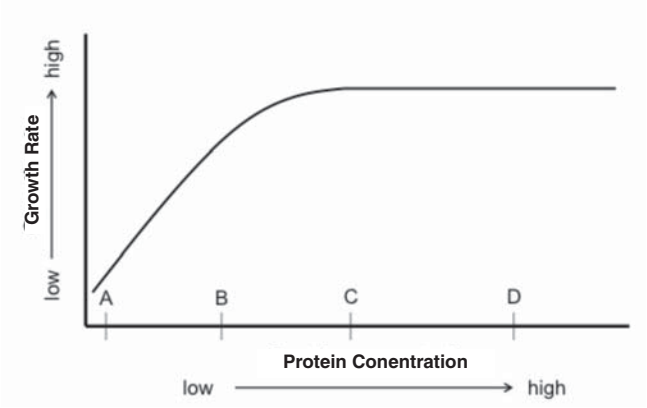

Figure 4. Stylized plot of growth rate as a function of dietary protein concentration.

facturing need and aim to produce economical diets that meet the nutritional need for energy. A substantial cost (~17%) in owning a cat is providing nutrition,[9] and a large cost in providing nutrients is in energy. Carbohydrate is an inexpensive source of energy. The carbohydrate in dry diets is mostly starch of cereal or root crop origin. Extrusion technology used in producing dry diets requires that ingredients contain sufficient starch to form a gelatinized dough.[10] After cooking under high pressure in moist heat, the starch-containing dough of extruded diets expands into stable shapes when cut and dried.

Optimal Energy Provision from Fat and Carbohydrate

Postulated effects of excessive dietary carbohydrate and fat in the pathogenesis of human diseases have elicited concern about similar pathogenic roles for carbohydrate and fat in cats. A noteworthy example of cross-species speculation is the Carnivore Connection Hypothesis.[11] During the 20 years since its description, the hypothesis has been influential on thinking about the soundness of feline dry diets.[12,13] Proposed by physicians, the hypothesis asserts that obesity and type-2 diabetes mellitus develop in people genetically adapted to a "carnivorous diet" who consume modern diets that are high in processed carbohydrates, in particular, starches and sugars. The mechanism posited for the diet-disease relationship of the hypothesis is chronic hyperinsulinemia developing in response to abundant alimentary glucose entry against a genetic background of insulin resistance.

By citing a report describing diabetes mellitus in cats, authors of the Carnivore Connection Hypothesis infer that cats, as model carnivores, develop obesity when given diets supposed "unnaturally" high in carbohydrate.[11] Additionally inferred is that resulting obesity coupled with continued high dietary carbohydrate feeding causes diabetes in cats. The nutrient proportion in a "natural" diet of cats is assumed in the hypothesis to be optimal for prevention of diabetes.[13] Here, energy from protein, fat and carbohydrate in natural diets is inferred to be similar to that in wild prey that cats might consume today. For reference, a medium-size, whole mouse would provide 54, 41 and 5% of metabolizable energy as protein, fat and carbohydrate,[14] whereas metabolizable energy of these nutrients in feline dry diets cited in the survey above distinctly differ from those in a whole mouse.

Evidence is lacking for optional being a "natural" nutrient profile, or importantly, what is speculated to be a natural nutrient profile for cats. It should be appreciated that the Carnivore Connection Hypothesis assumes that selection pressure has not modified metabolic propensities of cats. The cats of today have long been selected for aesthetic and behavioral traits for life with humans. During the last 50 years, cats have increasingly been fed dry type diets while being kept by humans. Heritable adaptations to dry diets that are advantageous for survival and reproduction on the diets would seem probable.

Epidemiological studies of pet cats seem to indicate that risk for obesity is not reduced by the energy-containing nutrient profile of presumed natural diets. Several surveys have compared body condition among cats given canned and dry diets.[15-17] For canned diets, the energy distribution in protein, fat and carbohydrate is similar to that in prey that cats might eat, e.g., rabbits, mice, voles, rats, birds, reptiles, fish, and invertebrates.[18] The surveys show no significant relationship between diet type and overweight body condition. These findings appear contrary to supposing that natural is optimal for body weight maintenance.

In considering findings for canned diets, it appears unlikely that obesity in cats is caused by greater carbohydrate in dry diets compared to that believed in natural diets. Other factors appear more relevant to obesity in cats, most notably neutering. An average body weight gain of 25 to 30% is observed after neutering if food presentation is not limited (Figure 5).[19] A gain of 20 to 25% or more from ideal would be considered to have health consequences.[20]

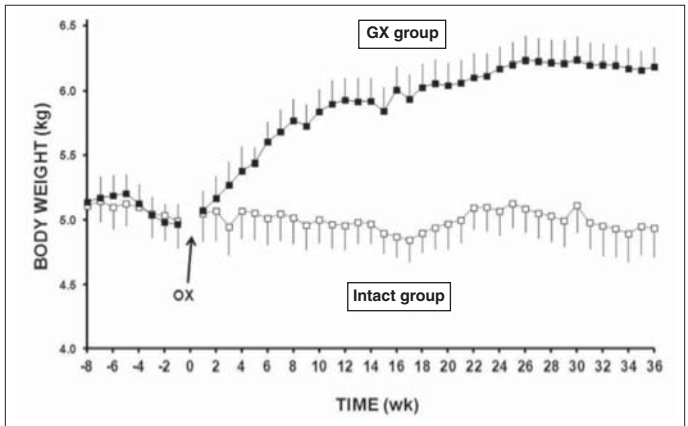

Figure 5. Mean body weights of intact and gonadectomized (GX) adult male cats before and after gonadectomy.[19] Error bars represent SEM, n = 8. *Different from intact cats, P < 0.05.

After protein requirement is met in a dry diet formulation, the balance of energy needs can be provided with a myriad of combinations of carbohydrate and fat. In appreciating this latitude, a few investigators have examined whether obesity risk can be reduced with an optimal energy ratio of carbohydrate to fat. In one study, addition of fat in exchange for carbohydrate and protein increased undesired body weight gain in neutered cats.[21] These results supported previous conjecture that dietary fat rather than carbohydrate is obesogenic. However, the results were not definitive. Protein was not held constant, only immature, neutered cats were studied, and only two nutrient mixtures were evaluated.

In a later study, protein was held constant at 33% of dietary energy and four energy ratios of carbohydrate to fat were evaluated.[22] The findings indicated that increasing fat in place of carbohydrate on an equivalent energy basis causes weight gain. When cats were sexually intact in the study, a threshold concentration of dietary fat was found for promoting body weight gain. Weight gain occurred when energy from fat was quite high, 64% (Figure 6). Significant gains were not observed when fat proportions were lower, ≤44% of dietary energy. Also studied in this work was whether gain after neutering might be mitigated by an ideal dietary carbohydrate to fat ratio. The findings were disappointing in that none of the ratios tested prevented weight gain after neutering. However, the degree of weight gain did vary across the ratios studied. Regression analysis indicated post-neutering weight gain might be minimized if the energy ratio from carbohydrate and fat was about 2 to 1 (Figure 7). Perhaps coincidently, the nutrient profiles of many feline maintenance dry diets are close to this ratio.

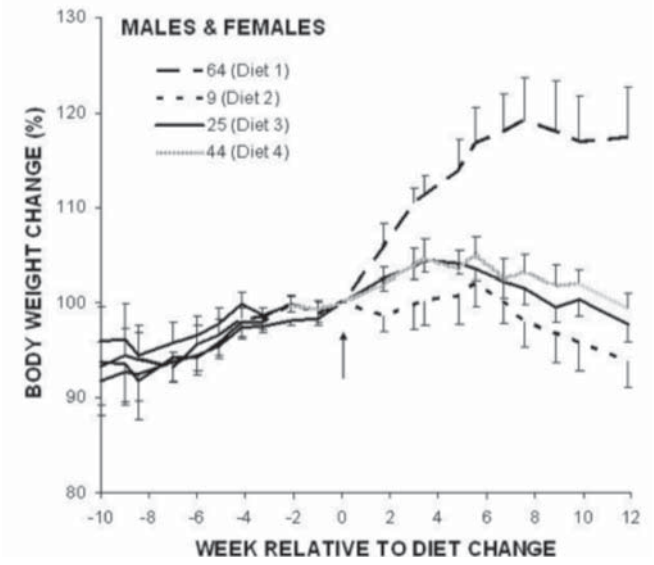

Figure 6. Percentage changes in body weight of sexually intact adult cats given purified diets (1-4) of varying fat content (9 to 64% metabolizable energy as fat).[22] Values are means of observations of five to six cats.

Results of a recent study of neutered cats confirm that increasing dietary fat and not carbohydrate favors weight gain.[23] Though only two diets were evaluated, the study was unique in that energy densities between the diets were similar. The study findings indicated that while energy density and fat concentration of dry diets typically increase in parallel, it is fat concentration and not energy density that is most favoring of weight gain.

Gaps in Understanding and Future Directions

The optimal ratio of carbohydrate to fat to reduce the risk for undesired weight gain may vary with dietary matrix. Evidence supporting dietary fat as obesigenic is inconsistent for canned diets.[15,24] Further, though feline canned diets are typically higher in fat than dry diets, body condition of cats is not consistently reported to differ between the diet types. For canned diets, factors other than fat would appear more influencing of body condition. Canned diets relative to dry diets are 3.5 to 4.5 times less energy dense because of their characteristically high moisture content.[25] Energy dilution by moisture may substantially reduce intake of canned diets. A recent study reports that dehydration of a canned diet may increase intake when the diet is presented twice daily.[26]

Optimal carbohydrate to fat ratio may depend on the response variable selected for study. Only a few variables appear to have been studied. A suggested variable to optimize is reduction of risk for diabetes mellitus. However, any basis for study of this variable is speculative. After controlling for body condition and age, clear evidence is lacking that dietary carbohydrate or fat proportion causes or elevates risk for diabetes. Postprandial blood glucose is reported to rise with increasing dietary carbohydrate proportion, but the meaning to the health of cats and risk for

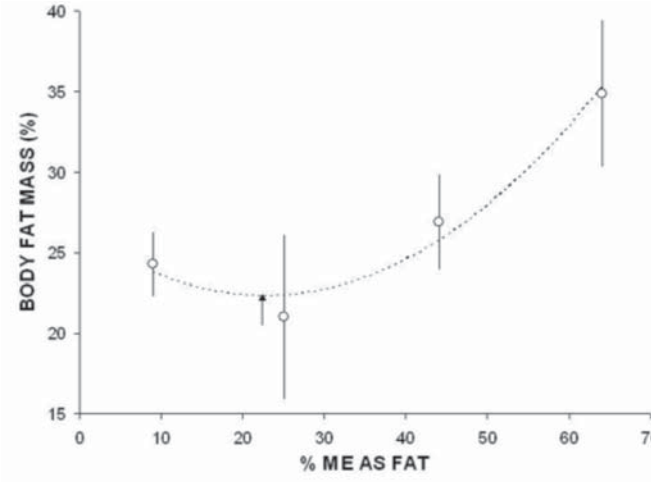

Figure 7. Body weight percentage as fat in adult male and female cats as a function of dietary fat content as metabolizable energy (ME).[22] Values are means (n = 5 to 6) with their standard errors represented by vertical bars. Plotted line is quadratic regression function. Arrow indicated function minima.

diabetes is elusive. Extraordinary pre-meal food withholding (~24 hours) is used to demonstrate the dietary carbohydrate effect.[3,27] As ambush feeders, cats relative to other species may more acutely conserve glucose with food deprivation. With feeding resumed, postprandial hyperglycemia may reflect slow disposal of glucose by metabolism previously adapted to glucose conservation.

An unhealthy postprandial glycemic level is not established for cats. Though human hyperglycemia levels are referenced for cats, species differences are notable. Blood glucose concentrations in normal healthy animals of many species[28] would be indicative of a prediabetic state in humans.[29]

For the most extreme elevations in postprandial glycemia, dietary protein concentration is low,[3] or extraordinarily low, on par with that typical of maintenance dog foods, 21% of dietary energy.[23] It is rational to suggest that future studies may reveal that optimal carbohydrate to fat ratio changes with dietary protein content.

The compositions of dietary carbohydrate and fat may importantly affect study outcomes on optimal dietary proportions. The rate of digestion of starches will depend on processing and proportions of amylose and amylopectin.[30,31] Among dietary fats, dietary fatty acid profile varies and thereby is potentially modifying of discoverable effects. Insulin sensitivity of overweight cats may be modulated by long-chain omega-3 fatty acid content.[32] Fatty acids vary in their utilization; some are oxidized more than they are stored. The fatty acid, α-linolenic acid, which is low in commercial feline diets and substantially higher in wild prey that cats may eat, appears more readily oxidized than many other fatty acids in dietary fat.[7,33]

Summary

Presently, there appears to be great latitude in proportions of fat and carbohydrate that may be used to provide dietary energy once other nutrient requirements are met. Few health-related variables have been studied for determining optimal dietary proportions of fat and carbohydrate. The most studied variable is body condition. For dry diets, which most cats are fed, increasing dietary fat in exchange for carbohydrate appears to favor overweight conditions. In sexually intact cats, a threshold proportion of fat to carbohydrate appears to exist. Beyond this threshold, increasing the fat to carbohydrate ratio promotes undesired weight gain. In neutered cats, a carbohydrate to fat energy ratio of 2 to 1 may minimize undesired weight gain. The influence of the fat to carbohydrate energy ratio may be obscured by other variables in a dietary matrix, perhaps most importantly palatability. These variables include moisture content, proportion of energy from protein, and compositions of fat and carbohydrate.

References

1. National Research Council. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006:22-27.

2. Hewson-Hughes AK, Hewson-Hughes VL, Miller AT, et al. Geometric analysis of macronutrient selection in the adult domestic cat, *Felis catus. J Exp Biol*. 2011;214:1039-1051.

3. Hewson-Hughes AK, Gilham MS, Upton S, et al. The effect of dietary starch level on postprandial glucose and insulin concentrations in cats and dogs. *Br J Nutr*. 2011;106(Suppl 1):S105-S109.

4. Bennett N, Greco DS, Peterson ME, et al. Comparison of a low carbohydrate-low fiber diet and a moderate carbohydrate-high fiber diet in the management of feline diabetes mellitus. *J Feline Med Surg*. 2006;8:73-84.

5. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*. 2006; 444:840-846.

6. Remillard RL. Homemade diets: Attributes, pitfalls and a call for action. *Top Comp An Med*. 2008;23:137-142.

7. Backus RC, Thomas DG, Fritsche KL. Comparison of inferred n-3 and n-6 polyunsaturated fatty-acid composition of feral domestic cat diets with the composition in commercial feline extruded diets. *Am J Vet Res*. 2013. (in press)

8. Smalley KA, Rogers QR, Morris JG, et al. The nitrogen requirement of the weanling kitten. *Br J Nutr*. 1985;53:501-512.

9. American Society for the Prevention of Cruelty to Animals (ASPCA). Pet care costs. http://www.aspca.or/adoption/pet-care-costs.aspx. Cited Feb. 9, 2012.

10. Dzanis DA. Petfood types, quality assessment and feeding management. In *Petfood Technology*. Kvamme JL, Phillips TD (eds). Watt Publishing, Mt. Morris, IL. 2003:68-73.

11. Miller JC, Colagiuri S. The carnivore connection: Dietary carbohydrate in the evolution of NIDDM. *Diabetologia*. 1994; 37:1280-1286.

12. Rand J. Current understanding of feline diabetes: Part 1, pathogenesis. *J Feline Med Surg*. 1999;1:143-153.

13. Zoran DL. The carnivore connection to nutrition in cats. *J Am Vet Med Assoc*. 2002;221:1559-1567.

14. Douglas TC, Pennino M, Dierenfeld ES. Vitamins E and A, and proximate composition of whole mice and rats used as feed. *Comp Biochem Physiol Comp Physiol*. 1994;107:419-424.

15. Russell K, Sabin R, Holt S, et al. Influence of feeding regimen

on body condition in the cat. *J Sm An Pract*. 2000;41:12-17.

16. Allan FJ, Pfeiffer DU, Jones BR, et al. A cross-sectional study of risk factors for obesity in cats in New Zealand. *Prev Vet Med*. 2000;46:183-196.

17. Courcier EA, O'Higgins R, Mellor DJ, et al. Prevalence and risk factors for feline obesity in a first opinion practice in Glasgow, Scotland. *J Feline Med Surg*. 2010;12:746-753.

18. Fitzgerald BM. Diet of domestic cats and their impact on prey populations. In *The Domestic Cat: The Biology of its Behavior*. Turner DC, Bateson P (eds). Cambridge University Press, Cambridge, UK. 1988:123-147.

19. Kanchuk ML, Backus RC, Calvert CC, et al. Weight gain in gonadectomized normal and lipoprotein lipase-deficient male domestic cats results from increased food intake and not decreased energy expenditure. *J Nutr*. 2003;133:1866-1874.

20. Laflamme DP. Companion Animals Symposium: Obesity in dogs and cats: What is wrong with being fat? *J An Sci*. 2012;90:1653-1662.

21. Nguyen PG, Dumon HJ, Siliart BS, et al. Effects of dietary fat and energy on body weight and composition after gonadectomy in cats. *Am J Vet Res*. 2004;65:1708-1713.

22. Backus RC, Cave NJ, Keisler DH. Gonadectomy and high dietary fat but not high dietary carbohydrate induce gains in body weight and fat of domestic cats. *Br J Nutr*. 2007;98:641-650.

23. Coradini M, Rand JS, Morton JM, et al. Effects of two commercially available feline diets on glucose and insulin concentrations, insulin sensitivity and energetic efficiency of weight gain. *Br J Nutr*. 2011;106(Suppl 1):S64-S77.

24. Lester T, Czarnecki-Maulden G, Lewis D. Cats increase fatty-acid oxidation when isocalorically fed meatbased diets with increasing fat content. *Am J Physiol*. 1999;277:R878-R886.

25. Shi Z, Tang G. Development of palatants for canned petfoods. In *Petfood Technology*. Kvamme JL, Phillips TD (eds). Watt Publishing, Mt. Morris, IL. 2003:180-182.

26. Farrow H, Rand JS, Morton JM, et al. Postprandial glycaemia in cats fed a moderate carbohydrate meal persists for a median of 12 hours — Female cats have higher peak glucose concentrations. *J Feline Med Surg*. 2012;14:706-715.

27. Wei A, Fascetti AJ, Villaverde C, et al. Effect of water content in a canned food on voluntary food intake and body weight in cats. *Am J Vet Res*. 2011;72:918-923.

28. Washington IM, Van Hoosier G. Clinical biochemistry and hematology. In *The Laboratory Rabbit, Guineas Pig, Hamster, and Other Rodents*. Suckow, MA, Stevens KA, Wilson RP (eds). Elseviser Academic Press, Amsterdam. 2012:57-116.

29. American Diabetes Association. Diabetes basics, prediabetes FAQs. http://www.diabetes.org/diabetes-basics/prevention/pre-diabetes/pre-diabetes-faqs.html. Cited Feb. 8, 2012.

30. Morris JG, Trudell J, Pencovic T. Carbohydrate digestion by the domestic cat (*Felis catus*). *Br J Nutr.* 1977;37:365-373.

31. Brewer LR, Cai L, Shi YC. Mechanism and enzymatic contribution to *in vitro* test method of digestion for maize starches differing in amylose content. *J Agric Food Chem*. 2012;60:4379-4387.

32. Wilkins C, Long RC Jr, Waldron M, et al. Assessment of the influence of fatty acids on indices of insulin sensitivity and myocellular lipid content by use of magnetic resonance spectroscopy in cats. *Am J Vet Res*. 2004;65:1090-1099.

33. Veltri BC, Backus RC, Rogers QR, et al. Adipose fatty acid composition and rate of incorporation of alpha-linolenic acid differ between normal and lipoprotein lipase-deficient cats. *J Nutr*. 2006;136:2980-2986.

Protein Metabolism: Adaptation

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Abstract

Scientific research has shown that as carnivores, cats have obligatory requirements for nutrients that are not essential for many other mammals. A higher maintenance requirement for protein is one example. This has been attributed to the inability to downregulate nitrogen catabolism secondary to the consumption of an almost-exclusive vertebrate prey diet. Studies support that the feline can adapt protein oxidation to dietary intake provided their protein requirement is met. However, their inability to downregulate nitrogen catabolism may not fully explain their high-protein requirement. One recently proposed model suggests that the cat has a high-protein requirement because of its high-endogenous glucose demand that is met by obligatory amino acid-based gluconeogenesis. It is postulated that the high-protein requirement is the result of amino acids entering gluconeogenesis to supply the glucose needs of the brain and other tissues requiring glucose.

Glossary of Abbreviations

AP: Adequate Protein
HP: High Protein
LP: Low Protein
ME: Metabolizable Energy
MP: Medium Protein

The domestic cat (*Felis catus*) is the only member of the family *Felidea* in which nutritional requirements have been studied extensively.[1] Scientific research has shown that as carnivores, cats have obligatory requirements for nutrients that are not essential for many other mammals. Their nutritional and metabolic idiosyncrasies are believed to be the result of evolutionary adaptations to a diet consisting mainly of animal tissue.[1] As a result, there are some nutrients, such as arginine, taurine, niacin, and vitamins A and D, that are nonessential in many mammals that are essential in felines.[1] The essentiality of these nutrients is often the result of synthesis rates *in vivo* that are insufficient to meet their needs.

Carnivores also have a higher maintenance requirement for dietary protein compared to noncarnivores.[2] The significant difference in protein requirements between carnivores and omnivores is demonstrated by the findings that the rat can maintain body weight, nitrogen balance and carcass nitrogen when fed diets containing 3.5 to 4.5% metabolizable energy (ME) as protein,[3,4] whereas the minimum protein requirement for maintenance in the cat is 16% ME.[2,5]

Dietary protein is required to provide essential amino acids and the nitrogen needed to synthesize dispensable amino acids and other nitrogen-containing compounds. Therefore, is the cat's high-protein requirement due to a high requirement for one or more essential amino acids or dispensable nitrogen?[1] Research supports that the essential amino acid requirements for the growing kitten generally are similar to other growing mammals.[1,6] Cats are able to control the activity of enzymes in the first irreversible step of essential amino acid degradation to some extent, explaining why they do not have a high requirement for essential amino acids.[1,7]

This leaves the possibility that the protein requirement is driven by the need for dispensable nitrogen. Early findings to support a high requirement for dispensable nitrogen came from an *in vitro* study reporting no difference in the activity of some hepatic aminotransferases and urea cycle enzymes in cats consuming high- (54% ME) and low-protein (14% ME) diets.[8] The result is a continuously high rate of nitrogen loss compared to other species that adapt to variations in protein calories by regulating protein oxidation and turnover.[9] Similar metabolic inflexibility has been reported in other carnivores, such as vultures, barn owls, alligators, and trout.[10-14] Conversely, studies in mink have reported that rates of amino acid decarboxylation and protein oxidation are regulated based on protein intake, even when protein is supplied near or below the requirements.[15-17] These findings support some metabolic flexibility in carnivores.[18] Studies conducted in omnivorous and herbivorous species fed high- and low-protein diets under similar experimental conditions have reported significant changes in enzyme activities.[5,19-25]

Based on the Rogers, et al. (1977) study, the high-protein requirement in cats seems to reflect a high obligatory rate of protein oxidation caused by an inability to downregulate the enzymes of urea cycle synthesis in order to conserve nitrogen when consuming a low-protein diet.[8] Reports that cats have higher endogenous nitrogen excretion on a protein-free diet, compared to omnivores,[26] and during times of food deprivation[27] provide further support for this hypothesis. It is important to point out a few limitations of this classic *in vitro* study. The enzyme

assays used in this project were optimized for rats and not cats,[28,29] and enzyme activity was reported as maximal activity, which may or may not reflect physiological conditions. The cats in this study were fed adequate protein concentrations, thus the findings did not test metabolic flexibility at or below requirements.[28,29]

Rogers and Morris (2002) suggest that the basis for the difference between cats and other noncarnivorous animals can be better understood in the context of the mechanisms available to the animal to conserve nitrogen when necessary or to oxidize the surplus when provided.[30] The mechanisms are based on four levels of control of urea cycle enzymes:

1) substrate regulation based on the amount of nitrogen entering the cycle from ammonia or aspartate;
2) allosteric regulation of carbamoylphosphate synthase 1 by N-acetyl glutamate (NAG);
3) control of the urea cycle by increasing and decreasing ornithine; and
4) up- and downregulation of the enzymes involved in urea synthesis.[30]

These researchers argue that while the first three levels of regulation are present in the cat, it is the fourth level, the inability to downregulate the nitrogen catabolic enzymes, that results in obligate nitrogen loss and a high-nitrogen requirement.[30] Therefore, the concentration of urea cycle intermediates may be more important in the control of ureagenesis.[30,31]

Future work based on that study provide additional details but also raise additional questions. An *in vivo* study by Lester, et al. (1999) reported no change in protein oxidation in a group of cats fed diets with varying amounts of energy coming from protein.[32] One potential limitation of the study was that protein oxidation was calculated rather than measured by indirect calorimetry. The end result may have represented nitrogen flux rather than oxidation.[28] Other *in vitro* and *in vivo* studies reported that cats have some ability to adapt to increases in dietary protein intake in terms of ureagenesis and gluconeogenesis.[5,28,31,33] However, none of the studies evaluated diets supplying protein at a concentration less than the cat's minimum requirement.

In one of these *in vivo* studies, using indirect calorimetry, Russell, et al. (2002) investigated substrate oxidation when cats were fed moderate- (35% ME) or high-protein (52% ME) diets.[28] Both treatment groups exceeded the cat's minimum requirement of protein (16% ME) for maintenance.[28] They found that protein oxidation increased when cats were fed the high-protein diet. The authors concluded that cats are more capable of adjusting protein metabolism than previously thought based on the enzyme data.[28] However, only diets exceeding the minimum requirement for protein were evaluated, and adaptation to these protein concentrations could easily be explained by allosteric and substrate/intermediate level regulation of the urea cycle and/or change in liver size.[5]

A second study evaluated protein oxidation in cats fed diets with protein concentrations below, at and above their requirement to test their ability to adapt substrate oxidation to dietary macronutrient concentration.[5] Semi-purified diets containing protein at 7.5% (low protein, LP), 14.2% (adequate protein, AP), 27.1% (medium protein, MP), and 49.6% (high protein, HP) of calories were fed in a modified cross-over design.[5] Using indirect respiration calorimetry and nitrogen balance to measure substrate oxidation, they reported that the ratio of protein oxidation: protein intake was higher when cats consumed the LP diet compared to the other three diets.[5] Provided the diet contained adequate protein to meet the cat's minimum requirement, protein oxidation closely matched protein intake, a finding consistent with that reported by Russell, et al. (2002).[5,28]

To explore the possibility that protein oxidation exceeded intake due to poor energy intake when the cats were consuming the LP diet, the investigators ran a follow-up study. They fed the MP diet (27.1% protein calories) in the same amounts as the cats voluntarily reduced their intake while being fed the LP diet. They determined that protein oxidation for this energy-restricted subgroup was similar to cats fed the MP diet to meet their energy needs. This finding suggests that energy balance may be independent of the cat's inability to adapt protein oxidation to low concentrations of dietary protein.[5] Overall, the findings supported their hypothesis that cats would adapt protein oxidation to dietary intake provided their protein requirement was met; however, if dietary intake was below their protein requirement, cats would be unable to decrease protein oxidation enough to maintain nitrogen balance.[5]

Overall, the limited metabolic flexibility in cats to adapt to low-protein diets may be the result of evolutionary adaptations to a diet consisting primarily of protein.[1] The ability to upregulate the urea cycle aids in protecting against ammonia toxicity after a high-protein meal and permits the utilization of the carbon skeletons from amino acids for gluconeogenesis.[5] This high rate of protein oxidation only becomes a detriment when consuming a diet in which the protein content is below the cat's minimum requirement. In this situation, the cat exceeds its ability to adapt and faces a negative nitrogen balance, whereas most omnivores would continue to thrive.[5]

However, the findings from these studies may have another meaning when considered in the context of Waterloo's explorations of the dual regulation of the urea cycle by substrate supply (reactive regulation) and urea cycle enzyme adaptations (adaptive regulation).[9] Herein, the argument is that adaptive changes to urea cycle enzymes are not necessary given the rapid and automatic regulation of carbamoylphosphate synthase 1.[9] Applied to the cat, it may be that the feline reacts rather than adapts to dietary protein although with the same net result.[34]

The general model of protein turnover in mammals proposed by Waterloo (1999) states that the lower limit of amino acid catabolism is dictated by the rate of whole-body protein turnover

and obligatory nitrogen loss.[9,35] One study evaluating urea kinetics in the cat reported protein turnover to be one-half to one-third that in other mammals.[31,35] The results did not explain the cat's need to catabolize amino acids at the high rates reported in numerous other studies.[31,35] The authors concluded that the high-protein requirement of the cat remains unexplained but is probably not due solely to its inability to downregulate hepatic protein catabolism in response to variations in dietary protein intake.[31,34]

More recently, a model was proposed by Eisert[35] to explain this paradox. In summary, the model says, "… cats do not have a high-protein requirement per se, but rather a secondarily high elevated protein requirement in response to a high endogenous glucose demand."[35] The hypotheses that serve as the foundation for the model include:

1) The cat has a relatively large brain for its size and hence a secondarily high metabolic demand for glucose. The cat has developed specific metabolic strategies that do not include hyperketonemia to meet this demand while consuming a low-carbohydrate diet.
2) Amino acids enter gluconeogenesis at a rate to meet the endogenous glucose demand independent of dietary carbohydrate intake (obligatory gluconeogenesis).
3) Obligatory amino acid-based gluconeogenesis results in endogenous nitrogen losses that exceed the amount predicted for a carnivore the size of the cat and therefore increase the minimum protein requirement in cats above that of other noncarnivorous species.[35]

A review of the natural feline diet based on published databases concluded that a prey-based diet supplies insufficient carbohydrates to meet the high, ongoing endogenous glucose demand of the cat.[35] This endogenous glucose demand stems from the cat's relatively large brain for a mammal of its size.[35,36] Using published data on brain mass and whole blood-glucose utilization, a predictive allometric model was developed to compare the glucose demand of the cat brain with other mammals.[35-37] The result was that with the exception of primates, the relative brain-glucose demand of the cat expressed on a metabolic body weight basis was the greatest in all the mammals evaluated.[35] The brain-glucose demand reported by this model represented 30% of the measured gluconeogenesis in the cat following an overnight fast.[35,38] It is proposed that the discrepancy between the amount of carbohydrate derived from the cat's natural diet and the cat's high-glucose needs is met through endogenous gluconeogenesis.[35]

Eisert (2011) continues to explore whether the glucose requirement of the cat brain is a sufficient explanation for the cat's elevated protein requirement. In order to evaluate this, the theoretical nitrogen costs of brain-glucose demand calculated from the brain-glucose demand of the cat estimated in the predictive allometric model is compared to the endogenous urinary nitrogen losses reported in the cat.[26,35] Endogenous urinary nitrogen losses were used as a proxy for total amino acid oxidation. Using this theoretical approach, there was close agreement in cats between nitrogen loss predicted from brain-glucose demand and published endogenous urinary nitrogen losses.[26,35] The author concludes that this finding supports the hypothesis that the cat's high-endogenous nitrogen losses are the consequence of augmentation of its minimal nitrogen losses by obligatory gluconeogenesis.[35]

Two potential criticisms to the model are raised and addressed by the author.[35] The first is that obligate gluconeogenesis reduces metabolic flexibility and leaves the cat incapable of adapting to a low-protein diet. This point is refuted by offering the hypothesis that the risks of a transient negative nitrogen balance in a feline consuming a high-protein diet are relatively small compared to the compromise in brain function or other organ systems due to low-glucose concentrations. The second criticism is why maintain obligatory gluconeogenesis in domestic cats provided with high-carbohydrate, low-protein diets? Eisert (2011) argues that the modern cat is likely consuming a diet that provides at least 30% of the calories from protein.[35] Therefore, failure to adapt to a higher carbohydrate diet by reducing gluconeogenesis from protein is unlikely to carry a risk of protein deficiency.[35] This is further underscored by evolutionary pressure to maintain the current metabolic status quo.[35]

In conclusion, the cat's inability to downregulate hepatic catabolic capacity at low-protein intakes may only partially explain this carnivore's high-nitrogen requirement. An emerging argument suggests that cats have evolved a high capacity for gluconeogenesis from amino acids to solve the dilemma of how to survive on a high-protein, prey-based diet as a small mammal with a large brain.[35] While arguably the overarching model proposed by Eisert (2011) requires more direct scientific support, the hypotheses and ideas are intriguing and provide a platform for future studies. Certainly this model emphasizes the interrelatedness and interdependence of protein, fat and carbohydrate in the feline diet. Future studies are needed to better understand how dietary composition impacts nutrient utilization and requirements.

References

1. Morris, JG. Idiosyncratic nutrient requirements of cats appear to be diet-induced evolutionary adaptations. *Nutr Res Rev.* 2002; 15:153-168.

2. National Research Council. Nutrient Requirements of Cats and Dogs. National Academy Press, Washington, D.C. 2006.

3. Sheehan PM, Clevidence BA, Reynolds LK, et al. Carcass nitrogen as a predictor of protein requirement for mature female rats. *J Nutr.* 1981;111:1224-1230.

4. Baldwin JK, Griminger P. Nitrogen balance studies in aging rats. *Exp Gerontol.* 1985;20:29-34.

5. Green AS, Ramsey JJ, Villaverde C, et al. Cats are able to adapt protein oxidation to protein intake provided their requirement for dietary protein is met. *J Nutr.* 2008;138(6);1053-1060.

6. Rogers QR, Morris JG. Essentiality of amino acids for the growing kitten. *J Nutr.* 1979;109:718-723.

7. Rogers QR, Morris JG. Why does the cat require a high-protein diet? In *Nutrition of the Dog and Cat*. Anderson RS (ed). Oxford: Pergamon Press. 1980;45-66.

8. Rogers QR, Morris JG, Freedland, RA. Lack of hepatic enzymatic adaptation to low and high levels of dietary protein in the adult cat. *Enzyme.* 1977;22:348-356.

9. Waterlow JC. *Protein Turnover*. CABI Publishing, Cambridge, MA. 2006

10. Walton MJ. Metabolic effects of feeding a high protein/low carbohydrate diet as compared to a low protein/high carbohydrate diet to rainbow trout *Salmo gairdneri*. *Fish Physiol Biochem*. 1986;1:7-15.

11. Myers MR, Klasing, KC. Low glucokinase activity and high rates of gluconeogenesis contribute to hyperglycemia in barn owls (*Tyto alba*) after a glucose challenge. *J Nutr.* 1999; 129:1896-1904.

12. Coulson RA, Hernandez T. Alligator Metabolism: *Studies on Chemical Reactions* In Vivo. Oxford: Pergamon Press, U.K. 1983.

13. Cowey CB, Cooke DJ, Matty AJ, et al. Effects of quantity and quality of dietary protein on certain enzyme activities of rainbow trout. *J Nutr.* 1981;111:336-345.

14. Migliorini RH, Lindner C, Moura JL, et al. Gluconeogenesis in a carnivorous bird (black vulture). *Am J Physiol.* 193:225:1389-1392.

15. Fink R, Tauson A, Chwalibog A, et al. Effects of substitution of dietary protein with carbohydrate on lactation performance in the mink (*Mustela vison*). *J An Feed Sci.* 2004;13:647-664.

16. Matthiesen CF, Blache D, Thomsen PD, et al. Effect of late-gestation, low-protein supply to mink (*Mustela vison*) dams on reproductive performance and metabolism of dam and offspring. *Arch An Nutr.* 2010;64:56-76.

17. Tauson AH, Fink R, Chwalibog A. Protein as glucose precursor in lactating mink (*Mustela vison*) estimated by decarboxylation of 1-13C-labelled leucine. In *Proceedings of the 15th Symposium on Energy Metabolism in Animals*. Chwalibog A, Jakobsen K (eds). Wageningen Academic Publishers, Wageningen, The Netherlands. 2001;261-264.

18. Matthiesen CC, Blache PD, Thomsen PD, et al. Foetal life protein restriction in male mink (*Neovision vison*) kits lowers post-weaning protein oxidation and the relative abundance of hepatic fructose-1,6-bisphosphatase mRNA. *Animal*. 2012;6: 1:50-60.

19. Schimke RT. Differential effects of fasting and protein-free diets on levels of urea cycle enzymes in rat liver. *J Biol Chem*. 1962;237:1921-1924.

20. Schimke RT. Studies on factors affecting the levels of urea cycle enzymes in rat liver. *J Biol Chem.* 1963;238:1012-1018.

21. Chandra M, Singh B, Soni GL, et al. Renal and biochemical changes produced in broilers by high-protein, high-calcium, urea-containing, and vitamin-A-deficient diets. *Avian Dis*. 1984;28:1-11.

22. Koutsos EA, Smith J, Woods LW, et al. Adult cockatiels (*Nymphicus hollandicus*) metabolically adapt to high protein diets. *J Nutr*. 2001;131:2014-2020.

23. Featherston WR, Freedland RA. Influence of dietary protein and carbohydrate levels on liver enzyme activities in quail. *J Nutr*. 1973;103:625-634.

24. Rosebrough RW, Steele NC, McMurtry JP. Effect of protein level and supplemental lysine on growth and urea cycle enzyme activity in the pig. *Growth*. 1983;47:348-360.

25. Payne E, Morris JG. The effect of protein content of the diet on the rate of urea formation in sheep liver. *Biochem J*. 1969; 113:659-662.

26. Hendriks WH, Moughan PJ, Tarttelin MF. Urinary excretion of endogenous nitrogen metabolites in adult domestic cats using a protein-free diet and the regression technique. *J Nutr*. 1997; 127:623-629.

27. Biourge V, Groff JM, Fisher C, et al. Nitrogen balance, plasma-free amino acid concentrations and urinary orotic acid excretion during long-term fasting in cats. *J Nutr*. 1994;124: 1094-1103.

28. Russell K, Murgatroyd PR, Batt RM. Net protein oxidation is adapted to dietary protein intake in domestic cats (*Felis silvestris catus*). *J Nutr*. 2002;132:456-460.

29. Russell K, Lobley GE, Rawlings J, et al. Urea kinetics of a carnivore, *Felis silvestris catus*. *Br J Nutr*. 2000;84:597-604.

30. Rogers QR, Morris JG. Upregulation of nitrogen catabolic enzymes is not required to readily oxidize excess protein in cats. (Letter to the editor) *J Nutr*. 2002;132:2819-2820.

31. Russell K, Lobley GE, Millward DJ. Whole-body protein turnover of a carnivore, *Felis silvestris catus*. *Br J Nutr*. 2003; 89:29-37.

32. Lester T, Czarnecki-Maulden G, Lewis D. Cats increase fatty-acid oxidation when isocalorically fed meat-based diets with increasing fat content. *Am J Physiol*. 1999;349:53-59.

33. Silva SVPS, Mercer JR. Effect of protein intake on amino acid catabolism and gluconeogenesis by isolated hepatocytes from the cat (*Felis domestica*). *Comp Biochem Physiol B*. 1985; 80:603-607.

34. Russell K. Reply to Rogers and Morris (Author reply) *J Nutr*. 2002;132:2812-2821.

35. Eisert R. Hypercarnivory and the brain: Protein requirements of cats reconsidered. *J Comp Physiol B*. 2011;181:1-17.

36. Röhrs M, Ebinger P. Verwildert ist nicht gleich wild: Die hirngewichte verwilderter haussäugetiere [Feral animals are not really wild: The brain weights of wild domestic animals.] *Berl Münch Tierärztl Wochenschr*. 1999;112:234-238.

37. Harper AM, Jennett S. Cerebral blood flow and metabolism. Manchester University Press: St. Martin's Press, Manchester, NY. 1990.

38. Kley S, Hoenig M, Glushka J, et al. The impact of obesity, sex, and diet on hepatic glucose production in cats. *Am J Physiol*. 2009;296:R936-R943.

Determining Protein Requirements: Nitrogen Balance Versus Lean Body Mass

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The concept that components of the body are continually being replaced has been around since the 6th century B.C., according to which the structure of the body was continuously broken down and being replaced by new structures and substances derived from food.[1] We now understand that this applies appropriately to the proteins in the body, and to the concept of protein turnover. Protein turnover refers to the catabolism and synthesis of endogenous proteins.[1] Within limits, this dynamic process is adaptable to the availability of amino acids for protein synthesis, hence it is influenced by sufficiency of dietary protein as well as the protein reservoirs provided by lean body mass (LBM).[2] This paper will address what is sufficient protein for adult cats, and how this is best measured.

Glossary of Abbreviations

AAFCO: Association of American Feed Control Officials
DEXA: Dual-Energy X-Ray Absorptiometry
LBM: Lean Body Mass
ME: Metabolizable Energy
NRC: National Research Council

Adequate dietary protein is required to provide essential amino acids and nitrogen for the synthesis of other amino acids, endogenous proteins and nitrogenous compounds. However, what constitutes "adequate dietary protein" or "minimum protein requirements" depends, in part, on the method of assessment. The requirements for growing animals are typically based on maximizing growth rates in post-weaning animals. In adults, minimum protein requirements are defined as the smallest amount of protein intake that will maintain nitrogen balance.[3,4] Nitrogen balance is defined as nitrogen intake equal to losses and is determined as the difference between total nitrogen intake and nitrogen loss in an adapted subject. It is assumed that all nitrogen intake comes from dietary protein, so nitrogen is measured rather than protein. In practice, only nitrogen intake and urine and fecal losses are measured, while skin, hair, sweat and other minor losses are ignored or estimated.[3-5] Studies are performed using various amounts of protein, and test subjects are allowed to adapt to the low-protein intake before the nitrogen balance study is performed since the body adapts to lower "habitual" protein intake.[1,6]

Other, less frequently used measures of protein adequacy include measures of protein turnover or maintenance of LBM.[1,2,5,7-9] Lean body mass, especially skeletal muscle, provides the protein reserves to support protein turnover.[2,10,11] The daily flux of amino acids that constitutes protein turnover is quantitatively large, involving about five to 10 times the protein provided from the daily dietary protein requirements.[8]

Most species adapt to reduced protein intake by reducing protein turnover and amino acid oxidation, and utilizing LBM for essential protein synthesis.[1,2,10] Proteins from the blood, liver and intestinal cells are utilized during acute protein deprivation, while muscle and skin are the major sources of amino acids during chronic protein deprivation.[1,8,11] Thus, inadequate protein intake over extended periods can result in a reduction of LBM despite reduced protein turnover.[1,2,10,11] This can be clinically significant since reduced protein turnover or loss of LBM appears to leave the subject in a compromised state with increased risk for mobidity or mortality.[2,7,9-13]

Homeostatic adaptation to dietary protein intake allows most adult animals to maintain nitrogen balance at a wide range of protein intakes.[1,14,15] However, it has previously been shown that cats have a reduced capacity to regulate the activity of transaminases and urea cycle enzymes, thus they have a limited ability to adapt to low-protein intakes and to conserve protein nitrogen.[16] Therefore, one would expect cats fed inadequate dietary protein to be in negative nitrogen balance and to demonstrate a loss of LBM, while cats fed adequate diets should be in a neutral or positive nitrogen balance and maintain LBM. The current recommendation by the Association of American Feed Control Officials (AAFCO) for protein in adult cat foods is 26% of the diet dry matter or 65g protein/1000Kcal metabolizable energy (ME).[17] However, the criteria used to reach this recommendation are unclear.

STUDY 1: Comparison of Nitrogen Balance to Lean Body Mass in Adult Cats

Twenty adult neutered male cats (ages 4 to 8 years) completed a study designed to compare estimates of protein requirements using nitrogen balance or maintenance of LBM assessed by dual energy X-ray absorptiometry (DEXA).[18] The cats were fed one of three diets (approximately 3.9 Kcal (ME)/gm) with protein contents of 5.7, 7.3 or 9.5g/100Kcal ME for two months. Intake was recorded daily and body weight recorded weekly throughout the study. At the beginning and end of the study, all urine and feces were collected during a 96-hour period for the nitrogen balance assessment. DEXA was performed at the beginning and end of the study to determine body composition.

By the end of the eight-week period, all cats completing this study had adapted and were able to maintain nitrogen balance. Based on regression of the data, an average daily protein intake of 1.5g protein/kg body weight should be sufficient to maintain nitrogen balance.[18] These results are similar to those reported by others who showed adult cats maintained nitrogen balance or had minimal endogenous nitrogen loss at about 1.4g to 1.7g protein/kg body weight.[2,16]

The average daily protein intake needed to maintain LBM in this study was 5.2g protein/kg body weight.[18] This is more than three times the amount needed to maintain nitrogen balance. A similar relationship between nitrogen balance and protein turnover, another marker of protein adequacy, was observed in dogs. In that study, young Beagle dogs required 0.9 g protein/kg body weight for nitrogen balance compared to 2.5 g needed to maximize protein turnover.[12] In the same study, older Beagles (ages 12 to 13 years) required 1.25 g and 3.75 g protein/kg body weight to maintain nitrogen balance or protein turnover, respectively.

STUDY 2: Six-Month Study on the Effect of Protein Intakes on Lean Body Mass in Geriatric Cats

Forty adult neutered male and female cats (ages 7 to 17 years) were divided into four groups balanced for gender, age and LBM to complete a six-month study on the effects of different protein levels on maintenance of LBM assessed by DEXA (unpublished). The cats were individually fed to maintain body weight with one of four diets (approximately 4.0 Kcal ME/gm), containing 7.4, 9.0, 10.4 and 12.75g protein/100Kcal ME. Intake was recorded daily and body weight recorded weekly throughout the study. Any cat that lost more than 10% of its initial body weight was removed from the study. DEXA was performed at the beginning, at three months and at six months to determine body composition.

At the end of three months (n = 39), linear regression of protein intake on LBM indicated a mean daily intake of 4.8g protein/kg body weight was needed to maintain LBM. At six months (n = 38), the regression indicated 5.4g protein/kg body weight was needed to maintain LBM. These results are not different from the 5.2g protein/kg body weight derived in the two-month study in younger adult cats and suggest that the true minimum requirement for adult cats, based on the amount needed to maintain LBM, is within the range defined in these studies.

STUDY 3: One-Year Study Evaluating Protein Intake Effects on Lean Body Mass in Geriatric Cats

The six-month study previously described was continued until one year, and DEXA was repeated on all cats that were not excluded for health reasons or due to body weight loss of >10%. Thirty of the initial 40 cats were included in the final DEXA analysis (n = 5, 8, 7 and 10, respectively, for increasing protein levels).

A simple linear regression of protein intake to LBM was no longer significant, so stepwise analyses were performed to determine which factors most influenced the observed change in LBM in these cats. Energy intake (Kcal/kg body weight) had no effect on change in body weight or LBM. The single largest factor influencing loss of LBM in these cats was initial age. This has been previously observed in numerous studies in multiple species: LBM decreases with age.[2,20,21] However, as has also been observed, loss of LBM can be influenced by protein intake.[2,11,20,22] Multiple regression of the data from these cats indicates a relationship defined as: % change in LBM = -4.890 – (1.525* initial age, yr) + (1.754 * protein intake, g/kg body weight); $r = 0.466$, $p < 0.05$. Based on this equation, the protein requirements to maintain LBM increase with age in cats (Table 1), but given that some decrease in LBM appears to be a normal aging effect, it is not clear that a target of complete preservation of LBM in geriatric cats is reasonable.

Table 1: Effect of Age on Theoretical Amount of Protein Needed to Preserve Lean Body Mass*

Initial Age (years)	Apparent Daily Protein Requirement (g/kg body weight)
8	4.17
10	5.91
12	7.65
14	9.38
16	11.12

*Some loss of LBM is normal with aging, so complete preservation of LBM in geriatric cats may not be possible.

Discussion

A major limitation to nitrogen balance data is that the intakes sufficient to maintain nitrogen balance are not necessarily adequate for optimum health.[9,11,23] The assay cannot differentiate between animals maintained in a relatively depleted state or in a condition in which the tissue proteins are maximal, so it should be considered an inadequate measure of protein requirements.[7,12] In the studies presented or summarized here, although nitrogen balance data indicated that about 1.5 g protein/kg body weight should be adequate to maintain nitrogen balance, more than three times that amount was needed to maintain LBM in adult cats.

The current recommendation by AAFCO for protein in adult cat foods is 26% of the diet dry matter or 65g protein/1000Kcal metabolizable energy.[17] Assuming an average calorie intake of 60Kcal ME/kg body weight for adult cats, this would equate to approximately 3.9g protein/kg body weight. The National Research Council (NRC) guidelines indicate a minimum daily protein requirement and a recommended daily protein allowance of 2.5 and 3.13g protein/kg body weight, respectively.[24] Based on the results of the studies presented here, the current AAFCO and NRC recommendations may be adequate to support nitrogen

balance, but appear to be inadequate to support protein turnover and LBM.

An interesting observation from the cat studies reported here was the effect of dietary protein on weight loss: Cats fed low-protein diets tended to lose body weight to a greater degree than those fed higher protein diets despite no significant differences in energy intake. In study three, for example, only the group fed the highest protein diet had no cats removed owing to weight loss of >10%. In study one, percent weight loss increased in a linear manner with decreasing protein intake.[18] While this may seem like an advantage for obesity management if it did result in lower body fat, a loss of LBM or an undesired loss of weight due to low-protein diets would not be advantageous. Previous research suggests that unexplained weight loss, especially in geriatric cats, can be the first sign of an impending terminal condition.[21] Whether increased protein intake could play a role in delaying the onset of disease requires further research. However, loss of LBM is associated with increased morbidity and mortality in multiple species,[2,11,13,25-28] and increased protein intake can slow or reduce the loss of LBM.[2,11] Additional research is needed to better define optimum protein requirements for adult cats of various ages.

References

1. Waterlow JC. *Protein Turnover*. CAB International Publishing, Oxfordshire, UK. 2006.

2. Wolfe RR. The role of dietary protein in optimizing muscle mass, function and health outcomes in older individuals. *Brit J Nutr*. 2012;108:S88-S93.

3. Earle KE. Protein and amino acid requirements of the adult cat. In *Proc of the 12th Ann Kal Kan Sym for Treatment of Sm An Disease*. 1988;87-90.

4. Tesaari P. Nitrogen balance and protein requirements: Definition and measurements. In *Cachexia and Wasting: A Modern Approach*. Mantovani G (ed). Springer-Verlag, Milan, Italy. 2006:73-79.

5. Campbell WW, Johnson CA, McCabe GP, et al. Dietary protein requirements of younger and older adults. *Am J Clin Nutr*. 2008;88:1322-1329.

6. Williams CC, Cummins KA, Hayek MG, et al. Effects of dietary protein on whole-body protein turnover and endocrine function in young adult and aging dogs. *J An Sci*. 2001;79: 3128-3136.

7. Allison JB, Wannemacher RW. The concept and significance of labile and overall protein reserves of the body. *Am J Clin Nutr.* 1965;16:445-452.

8. Swick RW, Benevenga NJ. Labile protein reserves and protein turnover. *J Dairy Sci*. 1976;60:505-515.

9. Young VR, Marchini JS. Mechanisms and nutritional significance of metabolic responses to altered intakes of protein and amino acids, with reference to nutritional adaptations in humans. *Am J Clin Nutr*. 1990;51:270-289.

10. Wolfe RR. The underappreciated role of muscle in health and disease. *Am J Clin Nutr*. 2006;84:475-482.

11. Volpi E, Campbell WW, Dwyer JT, et al. Is the optimal level of dietary protein intake for older adults greater than the recommended dietary allowance? *J Gerontol A Biol Sci Med Sci*. 2012. doi:10.1093/Gerona/gls229.

12. Wannemacher RW, McCoy JR. Determination of optimal dietary protein requirements of young and old dogs. *J Nutr*. 1966;88:66-74.

13. Szulc P, Munoz F, Marchand F, et al. Rapid loss of appendicular skeletal muscle mass is associated with higher all-cause mortality in older men: The prospective MINOS study. *Am J Clin Nutr*. 2010;91:1227-1236.

14. Pannemans DLE, Halliday D, Westerterp KR, et al. Effect of variable protein intake on whole-body protein turnover in young men and women. *Am J Clin Nutr*. 1995;61:69-74.

15. Waterlow JC. Nutritional adaptation in man: General introduction and concepts. *Am J Clin Nutr*. 1990;51:259-263.

16. Rogers QR, Morris JG, Freedland RA. Lack of hepatic enzyme adaptation to low and high levels of dietary protein in the adult cat. *Enzyme*. 1977;22:348-356.

17. Association of American Feed Control Officials. Official Publication. 2012.

18. Laflamme DP, Hannah SS. Discrepancy between use of lean body mass or nitrogen balance to determine protein requirements for adult cats. *J Fel Med Sur*. 2013 (in press)

19. Hendriks WH, Moughan PJ, Tarttelin MF. Urinary excretion of endogenous nitrogen metabolites in adult domestic cats using a protein-free diet and the regression technique. *J Nutr.* 1997;127: 623-629.

20. Kealy RD. Factors influencing lean body mass in aging dogs. *Comp Cont Edu Sm An Pract*. 1999;2(11K):34-37.

21. Perez-Camargo G. Cat nutrition: What's new in the old? C*omp Cont Edu Sm An Pract*. 2004;26(Suppl 2A):5-10.

22. Nguyen P, Leray V, Dumon H, et al. High-protein intake affects lean body mass but not energy expenditure in nonobese neutered cats. *J Nutr*. 2004;134:2084S-2086S.

23. Reeds PJ, Hutchens TW. Protein requirements: From nitrogen balance to functional impact. *J Nutr*. 1994;124(9 Suppl):1754S-1764S.

24. National Research Council Subcommittee on Dog and Cat Nutrition. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006.

25. Fujita S, Volpi E. Nutrition and sarcopenia of aging. *Nutr Res Rev*. 2004;17:69-76

26. Cosqueric G, Sebag A, Ducolombier C, et al. Sarcopenia is predictive of noscomial infection in care of the elderly. *Brit J Nutr*. 2006;96:895-901.

27. Cupp CJ, Kerr WW. Effect of diet and body composition on life span in aging cats. *Proc Nestlé Purina Companion Animal Nutrition Summit: Focus on Gerontology*. Clearwater Beach, FL. 2010.

28. Han SS, Kim KW, Kim KI, et al. Lean mass index: A better predictor of mortality than body mass index in elderly Asians. *J Am Geriatr Soc*. 2010;58:312-317.

How Important Is Protein for Weight Management?

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Abstract

It has been proposed that higher protein diets more similar to the cat's "natural diet" may be beneficial in preventing and treating obesity. Research in humans suggests that high-protein diets can help in weight management due to their effects on satiety, energy expenditure and lean body mass. Data in cats support that high-protein diets have positive effects in feline weight-loss plans and in prevention of weight regain. However, there currently is no evidence that high-protein diets fed *ad libitum* can be helpful in preventing weight gain either by decreasing voluntary energy intake or by increasing energy expenditure.

Glossary of Abbreviations

BCS: Body Condition Score
DEXA: Dual Energy X-Ray Absorptiometry
kJ: Kilojoules (1 Kilocalorie = 4.18 Kilojoules)
NRC: National Research Council

Importance of Weight Management

Obesity is defined as an excessive fat accumulation that has an adverse effect on health. Some of the reported adverse effects of increased body fat (above 25 to 30% excess body fat) on the health of people are cardiovascular diseases (heart disease and stroke), diabetes mellitus, osteoarthritis, and some cancers.[1] Obesity and overweight also are a concern in veterinary medicine. In one study with Labrador Retrievers, even being moderately overweight was associated with a reduced life span and earlier chronic health problems, such as arthritis.[2]

Even though obesity has not yet been associated with a shorter life span in cats, epidemiological data suggest that obesity is linked to several health problems, such as diabetes mellitus, dermatological problems, lameness, and hepatic lipidosis.[3,4]

Obesity in veterinary medicine is diagnosed using body condition score (BCS) systems. Other more objective measures of body fat, such as isotope dilution or DEXA, although useful in a research setting, are not practical or common in clinical practices. The more common BCS systems are the 5-point and 9-point scales. The latter has been validated for its use in cats[5] where a BCS of 5 is considered ideal, a BCS of 6 to 7 is overweight, and a BCS of 8 to 9 is obese. It is estimated that each point above 5 represents approximately an increase of 10 to 15% body weight.

The prevalence of this condition is high both in humans and in dogs and cats. In 2008, the World Health Organization estimated that 1.4 billion adults were overweight, and 500 million overweight adults were considered obese.[1] As for cats, the reported prevalence can vary widely depending on the study (Table 1). In the United States, two studies reported the prevalence to be approximately one-third of the population (29%[6] and 35%[3]), but it could be even higher.

Risk factors more commonly reported[3,6,7,8,9] for feline obesity are age (middle-aged cats); gender (male); underestimation of BCS by owners; and sexual status (sterilized). There are other suggested risk factors, but not all studies agree, thus there is debate regarding their importance. These include living indoors,[6] *ad libitum* feeding,[9] and the use/nonuse of dry premium foods.[3]

To summarize, obesity and overweight are highly prevalent and have negative effects on feline health. Despite the fact that obesity is considered a multifactorial disease, it always results from an energy imbalance, where intake is higher than expenditure. Controlling dietary energy intake is an important tool for the prevention and treatment of this condition.

Protein and Weight Management: Weight Loss

Classically, in order to prevent or treat obesity, the goal has been to use diets lower in energy density. Fat and fiber have been the nutrients receiving most of the attention both in human and veterinary medicine.

Fat is the most energy-dense nutrient — it provides more than twice the energy compared to protein and carbohydrates — and there is research suggesting that high-fat diets promote more weight gain than high-carbohydrate diets in cats.[10] Fiber is very low in energy density — we assume it provides none, although that is likely untrue — and it provides bulk and volume to the diet. Fiber, then, can potentially help with satiety, although research in cats is lacking in this aspect. Complex carbohydrates like whole-grain cereals have classically been recommended for weight loss because they provide more fiber and their starch is less readily available.

However, in recent years, new weight-loss regimes in humans have proposed that a better diet for weight loss is high in protein (+/- high fat) and low in carbohydrates, without any need for calorie restriction. In order to evaluate its efficacy and due to the concern about the long-term effects of such diets, there has

Table 1: Selected Reported Prevalence of Feline Overweight and Obesity

Study	Date of Publication	Geographical Location	Number	Prevalence	Risk Factors
Scarlett, et al.[6]	1994	Northeast U.S. veterinary clinics	>2,000	24%	Indoor living, inactivity, middle age, male, neuter, mixed breed, some dry foods
Russell, et al.[9]	2000	London (U.K.) households	136	52%	Neuter, middle age, frequent treating, *ad libitum* feeding
Allan, et al.[36]	2000	Households, urban area (Palmerston North, New Zealand)	202	25.8%	Longer leg length, owners underestimating BCS
Lund, et al.[3]	2000	U.S. veterinary clinics	8,159	35%	Neuter, male, premium or therapeutic food, breed
Colliard, et al.[8]	2009	National Veterinary School of Alfort Teaching Hospital (Paris, France)	385	26.8%	Male, neuter, owners underestimating BCS
Cave, et al.[7]	2012	Households, urban area (Palmerston North, New Zealand)	200	27%	Age, longer leg length, owners underestimating BCS

been a growing number of research papers evaluating the role of protein in weight control in humans.[11-13] The beneficial effects of protein on weight control in humans are attributed to its effect on satiety and energy intake, its thermogenic effect, and its sparing effect on lean body mass.

Several studies have compared diets, both energy-restriction and *ad libitum* diets, for weight loss in humans. The results suggest that when energy intake is restricted, there are no marked differences, depending on the type of diet, on weight-loss outcomes. However, when *ad libitum* feeding is allowed, high-protein diets (25 to 30% of total calorie intake) seem to have an advantage facilitating weight loss and body fat compared to higher carbohydrate, normal protein diets (15% of energy intake).[12,13] This suggests that their main effect is through reduction of voluntary feed intake rather than through an increase in energy expenditure.[13] There is some data to suggest that even moderate increases in dietary protein (15% versus 18% on an energy basis) may have a positive effect on weight maintenance after weight loss.[14] More research still is needed in this area, especially considering that most studies are short term (months).

Of course, doubt still exists whether the higher protein content or the lower carbohydrate content of these diets is responsible for any effect. One study[15] in humans compared four diets: high protein-low carbohydrate (20% and 25% on an energy basis, respectively), high protein-normal carbohydrate (20% and 50%), normal protein-low carbohydrate (10% and 25%), and normal protein-normal carbohydrate (10% and 50%). Weight loss was similarly successful with all four diets, although both high-protein diets resulted in a greater degree of weight loss.

Protein and Weight Management: Weight Maintenance

In regards to body weight maintenance and, in particular, in never-obese subjects, studies are much scarcer. A study published in 2005[16] recruited normal weight or slightly overweight human subjects and provided them with controlled amounts of a 15% protein, 35% fat diet on an energy basis for two weeks, and then switched to a higher protein and lower fat diet (30% and 20%, respectively) for two more weeks. Then, they were fed the high-protein diet *ad libitum* for 12 weeks. The individuals reported a decrease in hunger when fed the isocaloric high-protein diet, and they decreased their energy intake and lost weight when fed this diet *ad libitum* without any changes in energy expenditure. These results suggest that a higher protein diet, at least in the short term, can have an important effect on voluntary feed intake in non-obese humans and be potentially useful in the prevention of obesity.

Martens and collaborators[17] provided people of varying degrees of body mass index with three isocaloric diets on a crossover design with 5%, 15% and 30% protein (on an energy basis) in substitution for carbohydrates for 12 weeks each that were fed without calorie restriction. They did not note differences in satiety, hunger or fullness. All subjects lost weight during the experiment, without any effect of diet, but the energy deficit was higher in the high-protein treatment, suggesting that these individuals underate relative to their energy balance and without feeling hungrier.

The authors concluded that this is evidence that protein intake is more regulated than energy intake, which could explain why the high-protein group did stop eating before covering their energy requirements. They did not see any difference in body weight,

but this may have been due to a lean body mass sparing effect, although they did not measure body composition.

Proposed Mechanisms

Protein, Satiety and Energy Intake

Several studies in humans[11,12] suggest that dietary protein levels higher than 30% calories, compared with the average of 15% calories, may have a higher post-meal satiating effect. The limitations of these studies are that the majority are short term, which may not be maintained over time; most of them used very high-protein diets, thus not practical; and not all of them controlled other nutrient variations between treatments.

Possible explanations for this potential effect of protein on satiety include effects of amino acids on appetite-regulating hormones or an increased body temperature and oxygen consumption.[13]

Thermogenic Effect of Protein

There are losses of energy from foodstuffs, or crude energy, until it is deposited in the body[18] (Figure 1). Fecal losses, urinary and gas losses, and heat production losses all occur before we obtain the energy that will be available for use and deposition, or the net energy. Net energy systems are, in general, too complex to use, so with most species we work with digestible and metabolizable energy systems. In humans and in companion animals, we use metabolizable energy systems. The main difference between net energy and metabolizable energy is heat production or thermogenesis. This implies that there always is a certain error when estimating the energy content of foodstuffs and macronutrients.

There are several aspects to thermogenesis, and one of them is the thermic effect of feeding or diet-induced thermogenesis, which is described as the increase in energy expenditure associated with a meal. That is, the energy expended to digest, absorb and metabolize the ingested nutrients. This depends mostly on the energy content of the meal and can account for 5 to 15% of the daily energy expenditure.[19]

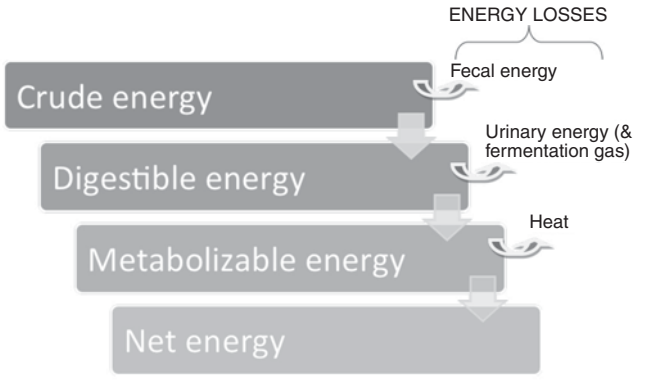

Figure 1

It has been suggested that protein increases diet-induced thermogenesis at least twice as much as carbohydrates and fat.[19] Thus, the metabolizable energy content of a foodstuff may be overestimating the amount of net energy available. Possible explanations for the higher thermogenesis of protein are based on the relative energy inefficiency of this macronutrient and include the cost of protein turnover, which increases with the consumption of high-protein diets, gluconeogenesis and urea synthesis.[12] Qualitatively, its effect on energy expenditure is estimated to be small. Eisenstein and Roberts[11] in their review of the use of high-protein diets in humans have estimated that a 30% protein diet (on an energy basis) versus a 15% protein diet would result in an increase in expenditure of 96 kJ (approximately 23 Kcal) per day. But this difference could be significant over the long term if not accompanied by an increase in energy intake. Diet-induced thermogenesis also has been proposed to contribute to the effect of high-protein diets on satiety.[13]

Protein and Lean Body Mass

Some studies in people have found an effect of high-protein diets (>25% protein calories) on lean body mass conservation during weight loss, but others have not.[1] Lean body mass maintenance during and after weight loss is important since, compared to fat mass, the energy expenditure of this tissue is higher and may help prevent weight regain.

Protein and Weight Management in Cats

Protein also has received attention for weight management in companion animals for the same reasons mentioned above. Moreover, this subject has received particular attention in cats due to the fact that they are obligate carnivores with high-protein requirements.

Plantinga and collaborators[20] proposed that the usual prey of feral cats (rabbits, rodents, small birds, small reptiles, etc.) is quite higher in protein (51.9% of the total daily metabolizable energy) than average commercial dry food (30 to 40% of calories). Two recent studies with colony domestic cats[21,22] suggested that these animals, if given the choice, can regulate their macronutrient intake to aim for a high-protein content (around 50% of total daily calories).

Thus, the idea has emerged that a high-protein diet closer to what felines have evolved to eat in the wild may have beneficial effects in several aspects, including the maintenance of adequate body weight.

In the next sections, different published experiments regarding the effect of dietary protein on weight management will be discussed. Table 2 summarizes the main characteristics of the experiments cited. The diet composition expressed as % of protein, fat and carbohydrate as a percentage of metabolizable energy has been calculated using the modified Atwater factors of 14.63 kJ/g (3.5 Kcal/g), 35.53 kJ/g (8.5 Kcal/g) and 14.63 kJ/g

Table 2: Summary of Characteristics of Feline Studies on the Effect of Protein on Body Weight and Composition

Reference	Age	Average Initial Body Condition	Sterilized	Length of Experimental Period	Calorie Distribution of Control Diet			Calorie Distribution of High-Protein Diet		
					% Protein	% Fat	% Carbohydrates	% Protein	% Fat	% Carbohydrates
Nguyen, et al., 2002[23]	adults	4.4/5	?	5 months	44.4	21.6	34	50.5	18.5	31
Russell, et al., 2002[27]	adults	? normal	Yes	50 days	35.3	61.8	2.9	51.9	44.6	3.5
Russell, et al., 2003[28]	adults	? normal	Yes	14 days	20	69.8	10.2	70	20	10
Nguyen, et al., 2004[29]	adults	3.4/5	Yes	6 months	27.0	25.2	47.8	46.5	30.4	23.1
Laflamme and Hannah, 2005[24]	adults	7.9/9	?	Up to 6 months	36.5	26.7	36.8	46.4	25.7	27.9
Hoenig, et al., 2007[25]	adults	12 lean, 16 obese	Yes	4 months	26.5	36.7	36.8	41.7	31.4	22.9
Green, et al., 2008[30]	adult	normal	No	14 days	27.1	39	34	49.6	39.2	11.2
Vasconcellos, et al., 2009[26]	adults	8.6/9	Yes	Minimum 4 months	31.3	23.1	45.6	41.5	22.7	35.8
Vester, et al., 2009[31]	adults	6/9	Yes	6 months	32.4	44	23.6	45.7	49.4	4.9
Vester, et al., 2012[32]	kittens	?	No	4 months	32.4	44	23.6	45.7	49.4	4.9
Michel, et al., 2005[33]	adults	Lean and obese	Yes	3 months	36.1	22.9	41.0	49.7	37.4	12.9
Wei, et al., 2011[34]	adults	7.6/9	Yes	4 months	27.1	44.1	28.8	47.3	44.5	8.2

(3.5 Kcal/g) for crude protein, crude fat and nitrogen free extractives, if this information was not directly provided in the study. Percentages of macronutrients are expressed on an energy basis unless otherwise specified.

Protein and Weight Loss in Cats

Four studies have evaluated the effect of two protein levels on weight-loss program outcomes in an experimental setting.[23-26] None of these studies found a difference in total weight loss, mainly because of the way the experiments were designed. All of them aimed for a constant weekly weight loss.

Three of them measured body composition, and two of them[24,26] found a positive effect of the higher protein diet on body composition, where the cats on the high-protein diet lost more fat and less fat free mass compared to the controls. In these studies, the protein content of the high-protein diet was 46.4% on an energy basis (versus 36.5% on the control diet)[24] and 41.5% (versus 31.3% on the control diet).[25] The composition of the weight that was lost with the higher protein diets was in both cases approximately 91% fat mass and 9% fat free mass, whereas the weight lost by control cats had a composition of 77% and 23% fat and fat free mass for the Vasconcellos study[26] (31.3% dietary protein) and of 81% and 19% in the Laflamme and Hannah study[24] (36.5% protein).

In the third study, Nguyen and collaborators[23] did not find this positive lean body mass sparing effect. Possible explanations include the small number of animals, the fact that their experimental diets differed in more than their protein and carbohydrate ratio (ingredients, energy density and fiber content were different), and the control diet was already higher than 40% protein, a similar level to the high-protein diet in the study from Vasconcellos and collaborators. The composition of the weight lost with the high-protein diet in this study also was different (76% fat mass and 24% fat free mass), but the weight loss rate also was higher (1.6% body weight loss per week, compared to the 1% in both other studies), which also can influence lean body mass loss.

The fourth study (from Hoenig and collaborators[25]) did not measure body composition, but it did measure fat depots (abdom-

inal and subcutaneous) with magnetic resonance imaging and found that obese cats fed the high-protein diet during weight loss (41.7% protein) lost more fat from those depots compared to the cats eating the control diet (protein content of 26.5%). Since there were no differences in body weight, it can be hypothesized that the cats on the high-protein diet conserved more lean body mass.

Regarding the effect of protein on baseline energy expenditure, neither the Nguyen[23] nor the Hoenig[25] studies found a difference between their diets. The Nguyen study[23] also estimated protein turnover and did not find a difference according to diets, although both diets were quite high in protein and the number of cats per group was only three. Laflamme and Hannah[24] did not measure energy expenditure, but did not find any difference between diets in the estimated energy intake to achieve weight loss.

The Vasconcellos study did find a difference in the energy intake necessary to lose weight and to maintain body weight after weight loss, with the high-protein diet requiring less-strict energy restriction (i.e., lost the same weight with a higher metabolizable energy intake), especially during the first weeks of weight loss. This study experimentally measured the metabolizable energy intake of the diets used instead of estimating it from their chemical composition. This may account for the differences in this study respective of the others. Also, the way of expressing energy intake may account for differences since no study expressed it the same way: The Vasconcellos study expressed energy intake as $kJ \cdot kg^{-0.4} \cdot d^{-1}$; the Laflamme study expressed it as $kJ \cdot d^{-1}$; the study from Nguyen expressed it as kJ·kg target body $weight^{-1} \cdot d^{-1}$; and the Hoenig study as $Kcal \cdot kg^{-1}$

Protein and Weight Maintenance in Cats

There are several, mostly short-term, controlled studies, on the effect of different protein intakes without accompanying weight loss on several outcomes in lean, never-obese adult cats;[2-32] two in lean and in obese cats[25,33] (one of these studies is also discussed in the previous section since it included a post-weight loss phase for the obese group); one in obese cats;[34] and one in ex-obese cats[26] (also discussed in the weight-loss section since the animals were submitted to weight loss before being kept weight stable for four months).

Three of the studies in lean cats[27,28,30] were performed to evaluate the effect of different dietary protein concentrations on protein metabolism: oxidation[27,30] and turnover,[28] so their main objective was not to evaluate the effect of protein on weight. However, weights were reported, and it is worth looking into them even though they were short-term studies.

The protein oxidation studies performed indirect calorimetry to estimate protein oxidation and energy expenditure. The Russell study[27] had two experimental canned diets, with little carbohydrate content, so they differed mostly on fat and protein. Both provided protein above the National Research Council (NRC) requirement,[18] at 35.3% and 51.9% on an energy basis (their respective fat content was 61.8% and 44.6%). The diets were fed *ad libitum* 14 days before measurements. During these 14 days, all cats lost a little bit of body weight (no difference between treatments), but there was a statistically significant (although numerically slight) increase in food intake and thus estimated energy intake for the cats eating the high-protein diet expressed as $kJ \cdot kg^{-.1}$ During the 18-hour calorimetry run, no differences in energy expenditure (expressed as kJ per 18 hours) were found. These results may indicate that the high-protein diet had a lower net-energy density compared to the control diet, so they need to eat more to maintain the same body weight with the same energy expenditure, but we need to consider the short-term nature of the study and the low number of cats.

The Green study[30] used a higher number of cats and had four semi-purified experimental diets, in this case differing in protein and carbohydrate content, keeping the fat constant at approximately 40% of the energy. The treatments were low protein (7.5%), adequate protein (14.2%), medium protein (27.1%), and high protein (49.6%), where the low protein was below and the adequate protein was at the NRC requirement.[18] The cats adapted for several weeks to the medium-protein diet, and then basal measurements were taken. The diets were fed *ad libitum* for at least 14 days before measurements, similar to Russell and collaborators,[28] providing another short-term study.

Regarding body weight, the researchers found that cats eating the low-protein diet had a lower energy intake and subsequently lost weight, which the authors attributed to either a palatability problem or a metabolic response to the low-protein/high-carbohydrate content. There were no differences in body weight and estimated energy intake between the medium- and high-protein groups (similar to Russell and collaborators). This study also measured body composition by isotope dilution, and there was no effect of the different diets on fat mass and fat free mass during the duration of the trial. Energy expenditure did not differ between the medium- and high-protein diets. Protein oxidation did differ between treatments: The higher the protein intake, the higher the protein oxidation, and, in all cases, intake matched oxidation except when protein requirements were not met and oxidation exceeded intake.

In the protein turnover study,[28] cats were fed one of two diets for 14 days. The diets were homemade and differed on fat and protein content (70% versus 20% protein on an energy basis) and carbohydrate was constant at 10%. Both groups lost a little bit of body weight, but there was no difference between the treatments as there also was no difference for energy intake. Thus, in the very short term, there is no marked effect of high-protein diets on body weight and body composition in lean adult cats.

Two studies[29, 31] measured body composition in sterilized adult cats using dry diets. Nguyen and collaborators[29] fed two diets

differing in protein and carbohydrate (27% versus 46.5% protein) to spayed and neutered adult non-obese cats for six months. The cats were not fed *ad libitum*: The daily amount was calculated to maintain body weight (209 $kJ \cdot kg^{-1} \cdot d^{-1}$). They did not observe any effect of diet on energy intake, since it was fixed, or energy expenditure, that was measured by indirect calorimetry. There were no differences in final body weight either. However, cats on the medium-protein diet lost weight, while cats on the high-protein diet did not. Both lost fat mass (approximately 5%, no difference between treatments), and the high-protein cats increased their lean body mass content (4%), whereas the medium-protein group had a stable lean body mass. In this longer term study, it seems a higher protein diet compared to a moderate-protein diet resulted in an increase of lean body mass. However, under controlled calorie intake conditions, they did not lose weight, while the moderate-protein diet group did. Moreover, the moderate-protein cats lost fat and not lean body mass, which was preserved.

The second study by Vester and collaborators[31] focused on the six-month period after ovariohysterectomy. Both diets differed in protein (32.4% versus 45.7%) and carbohydrate, but they also differed mildly in fat, energy density and markedly in fiber content. The female cats, after the surgery, were fed these two diets *ad libitum*, and body composition and physical activity were evaluated. Both groups increased their food intake and body weight after surgery and decreased their physical activity, with no differences between diets at any time points. When expressing food intake as estimated energy intake ($Kcal \cdot^{d-1}$), there was a trend for cats on the high-protein diet to consume more energy throughout the experiment. There were no differences in body composition measured by DEXA after six months. In this study, a high-protein diet also higher in fat and energy density and lower in fiber was not protective against the weight gain associated with spaying. This could be expected since higher protein/low carbohydrate diets usually are higher in energy density compared to other diets due to the increase in the proportion of energy coming from fat in these types of diets.

The same research group performed a study in kittens using the same experimental diets.[32] Kittens were fed these diets *ad libitum* from weaning until 4 months of age. The queens had been fed the respective diets through gestation and lactation. The investigators did not find any effect of diet on birth weight, body weight gain and energy intake.

Looking at these three medium-term (months) studies, the effect of consuming a high-protein diet on body weight in normal weight cats is not very remarkable. The results regarding body composition in adults are contradictory. One study found an increase in lean body mass, and the other study did not find such a difference. Those studies are hard to compare since their diets and experimental designs were so different. The Hoenig study discussed previously[25] also evaluated the effect of diet on stable weight lean and obese cats for four months before placing the obese cats on a weight-loss plan. In this case, food intake was restricted to maintain stable body weight, so no differences in food intake or body weight could be investigated, but heat production per metabolic body size was not affected by diet (41.7% verus 26.5% dietary protein) neither on the lean nor the obese cats, although there was a trend for an increase in heat production in lean cats eating a high-protein diet that was not observed in fat cats. Abdominal and subcutaneous fat depots were not affected by diet in lean or obese cats, although as mentioned diet did have an effect on the amount of fat lost during the weight-loss process.

There is another study with both lean and obese cats, although less controlled, by Michel and collaborators.[33] These colony cats used to be fed *ad libitum* and the aim of the study was to determine if time-limited feeding (four hours a day) would help with weight control (their average BCS was 7/9). They used two commercial feline dry diets differing in protein (36.1% versus 49.7%), fat and carbohydrate content and also differing markedly in fiber (18 versus 3 g/100 Kcal) and energy density (320 versus 411 Kcal/100 g). The same amount in grams of each diet was offered to each group.

Cats were stratified according to body weight and BCS and were group-fed so there was no food intake data and the only measured outcome was body weight for 12 weeks. They found that the cats fed the higher protein diet were weight stable (both lean and obese), while the control cats lost some weight (not large enough to affect BCS), and this was due mainly to the overweight cats losing weight on the less energy-dense diet. The weight of lean cats fed the control diet was not changed.

Since the amount of calories offered was higher with the high-protein diet, the authors decided to test this diet again. Both groups were fed the same diet, but one group was fed the same amount as the previous trial, and a second group was offered lower amounts of the diet comparable to the average energy offered with the control diet in the previous trial. In this situation, the obese cats significantly lost weight after 12 weeks with the "lower calorie" offering, while the lean cats in the same regime were weight stable. On the other hand, the lean cats fed a "higher calorie" regime gained weight, while obese cats maintained weight. Though the results should be taken with caution since the diets were quite different and the cats were group-housed, a higher protein, lower cabohydrate diet did not seem to positively affect body weight in lean or obese cats. Thus, the total amount of calories offered seems to be more important than the specific caloric distribution of the diet.

Wei and collaborators[33] evaluated the effect of two dry diets on energy balance and body composition in obese cats (BCS higher than 6/9) fed *ad libitum*. The diets differed mainly in protein and carbohydrate (27.1% versus 47.3% protein) and had a constant fat content of approximately 44%. Before obtaining baseline values, the cats were fed the control diet for three to four

months. The experimental diets were fed four months before measurements were repeated.

This study found that food intake — and, consequently, estimated energy intake — immediately increased when the cats were switched from the moderate-protein diet to the high-protein diet, and this was maintained for the whole period. This suggests that this particular high-protein diet was more palatable to the cats. After four months of *ad libitum* intake, body weight, body composition, which was measured by isotope dilution, and BCS score did not differ between treatments; however, energy expenditure, both resting and total energy expenditure, measured by indirect calorimetry was higher for the high-protein diet. However, since energy intake also was increased, there was no effect on body weight.

Energy expenditure may have been higher due to several reasons, such as increased diet-induced thermogenesis, which was not measured in the study, and the authors suggest that the fact that the cats in the study were obese and more sedentary may explain why energy expenditure was affected by diet when it has not been seen in studies with lean cats where a higher activity and total energy expenditure may mask small changes. This is in contrast to the Hoenig study,[25] where the very modest effect of high-protein diets on heat production was seen only in lean cats although the difference was not statistically significant.

With the limited research available, feeding higher protein diets in obese animals fed *ad libitum* does not seem to promote weight loss or changes in body composition. There is not enough data to know if high-protein diets fed in restricted amounts during a weight-loss plan would result in higher energy expenditure. This would be interesting to know and could add to the importance of high-protein diets on weight loss.

Finally, Vasconcellos and collaborators, in their weight-loss study, also studied the effect of the diet used during weight loss for weight maintenance. The BCS was 6.4/9, so these would be "slightly overweight ex-obese" cats. The cats lost weight with two different diets (31.3% versus 41.5% protein), and after they lost 20% of their initial body weight, they were maintained at a stable body weight for four months, all with the same diet (41% protein). They found that the metabolizable energy intake necessary to maintain body weight increased with time, indicating that the suggested drop in energy requirements post-weight loss[35] may be alleviated with time. Moreover, this was affected by the diet used during weight loss. Cats that lost weight with the higher protein diet maintained their body weight with a higher food and energy intake — in other words, their energy requirements were greater — which can be helpful in keeping the cats fuller and thus help prevent weight regain.

Implications

It is inevitable that diets differing in protein differ at least in one other macronutrient, mostly carbohydrate in the studies cited, so it is hard to decide which is responsible for the observed effects. However, the diets used in all these studies, both human and animal, differ not only in macronutrient content but also in fiber content and type, protein source, ingredients, moisture content, physical form, processing methods, among others. Of course, each study has different definitions of a high-protein or moderate-protein diet. This is inevitable but needs to be considered. Accounting for the short- and medium-term nature of most of the research, we should exercise caution when making blanket statements regarding the effects of "high-protein" diets.

The current evidence suggests that higher protein levels (>40% on an energy basis) are important to maintain lean body mass during weight loss in cats, and it may help blunt the decrease in energy expenditure associated with weight loss in "ex-obese" cats. This is important since lean body mass has a higher energy expenditure and may help prevent undesired weight regain. More research is needed to figure out if these diets have an effect on energy expenditure during weight loss since these studies are not in agreement. Currently, there is no data to deduce if high-protein diets have a satiating effect or increase diet-induced thermogenesis in cats during weight loss.

As for weight maintenance and prevention of weight gain, the current short- and medium-term studies do not support a weight-gain prevention effect of high-protein diets (>40%) in lean or obese cats. One study found an increase in the proportion of lean body mass when feeding a high-protein diet on controlled amounts but not the others, so no conclusions can be drawn at this point to a potential benefit via a leaner body composition. One study found an increase in energy expenditure in cats fed a high-protein diet compared to cats fed a more moderate-protein diet,[33] which could support an effect of protein on diet-induced thermogenesis or another energy component, but others did not find this effect.[25,27,30] As these latter studies were either performed with lean cats or the intake was not *ad libitum*, it is hard to compare them. More research is needed to figure out if any aspect of energy expenditure is significantly affected by high-protein diets.

As for their effect on energy intake, the results seem to be different to those in humans since in the studies cited where animals were fed *ad libitum*, the food and energy intake was actually higher in the cats fed a high-protein diet and this does not support a satiating effect of protein. Wei and collaborators[33] hypothesize that in cats, obligate carnivores that have evolved eating very high-protein prey, such a satiating effect could be potentially dangerous.

References

1. http://www.who.int/mediacentre/factsheets/fs311/en/. (accessed Dec. 12, 2012)

2. Kealy RD, Lawler DF, Ballam JM, et al. Effects of diet restriction on life span and age-related changes in dogs. *J Am Vet Assoc*. 2002;220:1315-1320.

3. Lund EM, Armstrong PJ, Kirk CA, et al. Prevalence and risk factors for obesity in adult cats from private U.S. veterinary practices. *Intern J Appl Res Vet Med*. 2005;3:88-96.

4. Scarlett JM, Donoghue S. Associations between body condition and disease in cats. *J Am Vet Med Assoc*. 1998:212:1725-1731.

5. Laflamme DP. Development and validation of a body condition score system for cats: A clinical tool. *Feline Pract*. 1997;25:13-18.

6. Scarlett JM, Donoghue S, Saidla J, et al. Overweight cats: Prevalence and risk factors. *Int J Obes Relat Metab Disord*. 1994;18(Suppl 1):S22-S28.

7. Cave NJ, Allan FJ, Schokkenbroek SL, et al. A cross-sectional study to compare changes in the prevalence and risk factors for feline obesity between 1993 and 2007 in New Zealand. *Prev Vet Med*. 2012;107:121-133.

8. Colliard L, Paragon BM, Lamuet B, et al. Prevalence and risk factors of obesity in an urban population of healthy cats. *J Feline Med Surg*. 2009;11:135-140.

9. Russell K, Sabin R, Holt S, et al. Influence of feeding regimen on body condition in the cat. *J Sm An Pract*. 2000;41:12-17.

10. Backus RC, Cave NJ, Keisler DH. Gonadectomy and high dietary fat but not high dietary carbohydrate induce gains in body weight and fat of domestic cats. *Br J Nutr*. 2007;98:641-650.

11. Eisenstein J, Roberts SB, Fallal G, et al. High-protein weight-loss diets: Are they safe and do they work? A review of the experimental and epidemiological data. *Nutr Rev*. 2002;60:189-200.

12. Halton TL, Hu FB. The effects of high-protein diets on thermogenesis, satiety and weight loss: A critical review. *J Am Coll Nutr*. 2004;23:373-385.

13. Morenga LT, Mann J. The role of high-protein diets in body weight management and health. *Br J Nutr*. 2012;108:S130-S138.

14. Westerterp-Plantenga MS, Lejeune MP, Nijs I, et al. High-protein intake sustains weight maintenance after body weight loss in humans. *Int J Obes Relat Metab Disord*. 2004;28:57-64.

15. Soenen S, Bonomi AG, Lemmens SG, et al. Relatively high protein or "low-carb" energy-restricted diets for body weight loss and body weight maintenance? *Physiol Behav*. 2012;107: 374-380.

16. Weigle DS, Breen PA, Matthys CC, et al. A high-protein diet induces sustained reductions in appetite, *ad libitum* caloric intake, and body weight despite compensatory changes in diurnal plasma leptin and ghrelin concentrations. *Am J Clin Nutr*. 2005; 82:41-48.

17. Martens EA, Lemmens SG, Westerterp-Plantenga MS. Protein leverage affects energy intake of high-protein diets in humans. *Am J Clin Nutr*. 2013;97:86-93.

18. National Reseach Council. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006.

19. Westerterp KR. Diet induced thermogenesis. *Nutr Metab*. 2004;1:1-5.

20. Plantinga EA, Bosch G, Hendricks WH. Estimation of the dietary nutrient profile of free-roaming feral cats: Possible implications for nutrition of domestic cats. *Br J Nutr*. 2011;106(Suppl 1): S35-S48.

21. Hewson-Hughes AK, Hewson-Hughes VL, Miller AT, et al. Geometric analysis of macronutrient selection in the adult domestic cat, *Felis catus*. *J Exp Biol*. 2011;214:1039-1051.

22. Hewson-Hughes AK, Hewson-Hughes VL, Colyer A, et al. Consistent proportional macronutrient intake selected by adult domestic cats (*Felis catus*) despite variations in macronutrient and moisture content of foods offered. *J Comp Physiol B*. 2012. (E-pub ahead of print)

23. Nguyen P, Dumon H, Martin L, et al. Weight loss does not influence energy expenditure or leucine metabolism in obese cats. *J Nutr*. 2002;132:S1649-S1651.

24. Laflamme DP, Hannah SS. Increased dietary protein promotes fat loss and reduces loss of lean body mass during weight loss in cats. *Intern J Appl Res Vet Med*. 2005;3:62-68.

25. Hoenig M, Thomaseth K, Waldron M, et al. Insulin sensitivity, fat distribution, and adipocytokine response to different diets in lean and obese cats before and after weight loss. *Am J Physiol Regul Integr Comp Physiol*. 2007;292:227-234.

26. Vasconcellos RS, Borges NC, Gonçalves KNV, et al. Protein intake during weight loss influences the energy required for weight loss and maintenance in cats. *J Nutr*. 2009;138:855-860.

27. Russell K, Murgatroyd PR, Batt RM. Net protein oxidation is adapted to dietary protein intake in domestic cats (*Felis silvestris catus*). *J Nutr*. 2002;132:456-460.

28. Russell K, Lobley GE, Millward DJ. Whole-body protein turnover of a carnivore, *Felis silvestris catus*. *Br J Nutr.* 2003; 89:29-37.

29. Nguyen P, Leray V, Dumon H, et al. High-protein intake affects lean body mass but not energy expenditure in nonobese neutered cats. *J Nutr*. 2004;134:S2084-S2086.

30. Green AS, Ramsey JJ, Villaverde C, et al. Cats are able to adapt protein oxidation to protein intake provided their requirement for dietary protein is met. *J Nutr*. 2008;138:1053-1060.

31. Vester BM, Sutter SM, Keel TL, et al. Ovariohysterectomy alters body composition and adipose and skeletal muscle gene expression in cats fed a high-protein or moderate-protein diet. *Animal*. 2009;3:1287-1298.

32. Vester BM, Belsito KR, Swanson KS. Serum metabolites, ghrelin and leptin are modified by age and/or diet in weanling kittens fed either a high- or moderate-protein diet. *An Sci J*. 2012;83:426-433.

33. Michel KE, Bader A, Shofer FS, et al. Impact of time-limited feeding and dietary carbohydrate content on weight loss in group housed cats. *J Feline Med Surg*. 2005;7:439-355.

34. Wei A, Fascetti AJ, Liu KJ, et al. Influence of a high-protein diet on energy balance in obese cats allowed ad libitum access to food. *J An Physiol An Nutr*. 2011;95:359-367.

35. Villaverde C, Ramsey JJ, Green AS, et al. Energy restriction results in a mass-adjusted decrease in energy expenditure in cats that is maintained after weight regain. *J Nutr*. 2008;138: 856-860.

36. Allan FJ, Pfeiffer DU, Jones BR, et al. A cross-sectional study of risk factors for obesity in cats in New Zealand. *Prev Vet Med*. 2000;46:183-196.

Balanced Feeding Assists Healthy Growth in Dogs

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Abstract

The main factors affecting the healthy growth of dogs, such as proper calcium and phosphorus supplies as well as energy consumption and its impact on the growth curve, are discussed in the context of nutrient requirements of dogs compared to those in man. Published data on the consequences of excessive or deficient mineral supply and growth intensity also will be reviewed.

Glossary of Abbreviations

BCS: Body Condition System
BW: Body Weight
Ca: Calcium
DOD: Developmental Orthopedic Diseases
NRC: National Research Committee
P: Phosphorus
PTH: Parathyroid Hormone

Calcium and Phosphorus

Dogs have higher needs for some minerals, such as calcium (Ca) and phosphorus (P), and seem to be more sensitive to deficient or excessive nutrient supplies when compared to other species, especially man. This is true for adult dogs, but specifically for dogs during growth.

The existing requirements for dogs and humans may differ in format and heights according to the recommendations you choose, but one thing becomes obvious: Adult dogs seem to need considerably more calcium than adult humans (Table 1).

Calcium Requirements and Calcium Deficiency

Why do dogs need so much more Ca?

It is a fact that the body needs to keep the blood Ca levels in narrow ranges. Therefore, a sensitive regulation is mandatory to avoid far-reaching, and possibly life-threatening, hypo- or hypercalcaemia. Next to increasing digestibility, which does not seem to occur at least in adult dogs, a deficient supply of Ca and P may be counteracted using the body stores, i.e., activating bone resorption. Currently, it is not fully known how long it takes a dog to efficiently activate bone resorption. It also is within the realms of possibility that skeletal reservoirs with different availability, i.e., for *ad hoc* or for long-term use, leave the dog widely unaffected by day-to-day variability of mineral supply. However, even adult dogs are susceptible to osteomalacia as a consequence of prolonged Ca deficient feeding.[4, 5]

What happens to humans with a long-term calcium deficient diet?

It is not really farfetched to declare a widely spread unbalanced and incomplete nutrient supply in some Western countries with increasing consumption of food rich in (animal) fat and carbohydrates but low in vitamins and certain minerals. For example, the 2008 nutrition report of the Deutsche Gesellschaft für Ernährung documented deficient intake of some vitamins, fiber and Ca in most children and adolescents. However, reports of clinically and obviously relevant developmental problems due to mineral-deficient nutrition are scarce. Even the Ca deficiency in combination with the often (too) high body weight and too steep growth curve do not lead to a relevant incidence of developmental orthopedic diseases (DOD) in children comparable to dogs.

A growing dog will most probably develop severe clinical skeletal problems when fed amounts of Ca sufficient for children of a similar body weight. Growing dogs may be more sensitive due to the higher speed of growth and, therefore, higher needs for tissue accretion. Dogs are multipar and, therefore, it is comprehensible that reproducing bitches have higher needs for Ca and P. But even the recommended allowance for maintenance for an adult man, including safety margins, is far less than the net requirement of a dog of the same body weight. The question is not if a dog provided with the amount of Ca sufficient for humans (calculated on BW or per energy unit basis) will develop clinical problems but rather how fast this will happen.

Table 1: Daily Calcium Requirement Based on the Same Body Weight During Maintenance, Growth and Reproduction in Dog and Man

Maintenance	**Adult Dogs**	**Adult Man**	**Factor**
mg/d (70kg BW)*	3146	1000	3.1
Growth	**Growing Dogs (<14w)**	**Children (~2y)**	**Factor**
mg/d (12kg BW)#	4384	600	7.3
Reproduction	**Adult Dogs**	**Adult Women**	
mg/d (70kg BW)*	19844	1000	19.8

* Calculated for a dog with a body weight of 70kg due to only one recommendation for an average adult man (estimated mean body weight of 70kg)
\# Calculated for an actual body weight of 12kg and an adult body weight of 70kg

Recommendations from Deutsche Gesellschaft für Ernährung,[1] NRC[2] and FEDIAF.[3]

Do we have enough knowledge and data to postulate whether such a huge difference exists? If so, is this difference due to a less efficient Ca digestibility in the dog? How about regulation and adaptation to the respective form of the diet or the origin of the existing data, i.e., the design of digestion trials? Do we have to take into account how the data were established?

First, we should have a look into the existing requirements including safety margins and safe upper limits. Even though many papers in this field are published and on first sight the information status seems to be excellent or at least sufficient due to limited information, for example, on bioavailability of Ca and P, the deriving recommendations on Ca and P supply remain rather speculative. How is that possible?

Presumably many factors may affect the bioavailability of the major minerals, predominantly Ca and P, in dogs. Among these are age, performing stage (growth, maintenance, reproduction, etc.), body weight, and breed of the animal. Another affect comes from dietary factors, such as diet composition, source of minerals, concentration of the respective mineral in the diet, as well as the concentration of other minerals known to influence the digestibility of the mineral in question (interactions determined, for example, by the Ca/P ratio), processing of the diet and last, but not least, the duration of feeding the respective diet, the least understood and realized factor. The compilation of sound data on Ca and P bioavailability, therefore, includes the uniformity of trials or, better, the knowledge and quantification of all factors that may modify the results.

A recently submitted work comprising a meta-analysis of digestibility trials in dogs and cats on Ca and P digestibility[6] yields much better insight on the bioavailability of Ca and P in dogs and cats. The Ca metabolism in adult cats and dogs is regulated by parathyroid hormone (PTH), calcitonin and metabolites of vitamin D. Unlike hindgut fermenters, such as horses or rabbits, dogs and cats are thought to balance their Ca status through the regulation of digestibility and not, for example, through excretion of excess Ca via the kidneys. According to the current scientific consensus, cats and dogs are able to decrease the intestinal mineral absorption in case of a dietary excess and to increase it in case of deficient supply[7] in order to maintain equilibrium. However, the meta-analysis showed that this is not the case.[6] Two explanations are possible in this case: The duration of the trials commonly used to determine the Ca balance is too short to initiate regulation of Ca digestion, or the dogs (at least at maintenance) are completely unable to adapt the digestibility of Ca comparably to other species, such as horses and rabbits.

First: The duration of the trials and therefore the exposure to the diet may be too short for the dog to modulate the digestibility. Normally, digestion trials last four to eight weeks. If there is no adaptation of Ca digestibility in this period, as shown from Mack, et al.,[6] the blood levels must be regulated through bone resorption in case of deficient Ca supply and Ca excretion via urine in case of Ca excess, respectively. In this case, data of those "normal" balancing trials are quite useless in regard to the resulting data of Ca digestibility — just because they are too short.

Second: Dogs are not able to adapt the digestibility of Ca, even after a longer period of a deficient or excessive Ca supply. This would cause depletion of the skeletal stores and result sooner or later in possible clinical problems.

Using the wolf as progenitor as a basic model for understanding the background of Ca metabolism may help to understand that the availability of Ca in the diet is important. All primal, natural diets consisted of whole prey animals. Therefore, there was no need to upregulate the efficiency of Ca digestibility as more than enough bony material was available. Even the much higher need for Ca of growing and reproducing dogs may be explained by this image: The alpha dogs feed on the more valuable meaty parts, whereas the bitches are next and the youngsters feed on a higher proportion of skeleton, thereby (involuntarily) increasing their Ca intake. The same background may be responsible for the inability to synthesize vitamin D in dogs and cats or better the lack of need to be able to do so.

As a consequence of these factors affecting the bioavailability of the major minerals Ca and P, it is mandatory to establish the maximum possible uniformity (for comparability reasons) regarding the digestibility trials taken into account (animals, diet, trial design, etc.) in order to have valid data to base the recommendations for these minerals. If dogs are able to adapt the digestibility of Ca, and if so, how long it takes to do so, needs to be clarified, and therefore, more research is required. This knowledge is the basis for a healthy feeding of dogs, especially in case of insufficient or excess Ca intake.

Calcium Excess

Rearranging the point of view on research papers and clinical cases dealing with diet-induced developmental skeletal diseases may help to understand the complex situation and sometimes seemingly conflicting information. In the literature, plenty of papers describe the detrimental effect of excess Ca on the skeletal development in growing dogs.[8,9,10-14, etc.] What the underlying trials of most of those papers have in common is an excessive Ca supply to the trial animals (puppies) without a concomitant increase of P in the diet, resulting in a wide Ca:P ratio > 2:1 that seems to trigger signs of developmental orthopedic diseases in growing dogs of certain breeds, mostly Great Danes. It is a well-known fact that the amount of Ca in the diet has an impact on the P digestibility.[15] Also, Mack, et al.[6] stated that the fecal P excretion was strictly correlated to fecal Ca excretion in dogs and cats. In other trials with more balanced Ca:P ratios through elevated P supply, no or definitely less-severe clinical signs of DOD were caused.[13,14,16,17]

Therefore, not the Ca excess itself seems to be the only and major problem for the skeleton, but the consequences of a wide Ca:P ratio on the bioavailability of P and, accordingly, a possible

clinical consequence of a secondary P deficiency triggered by a Ca excess.[18] This explanation is not really new. It was first published in 1931 by Marek and Wellmann[19] who were summarizing their findings about the detrimental effects of excess Ca, concluding that the signs, such as lameness or deviation of limb axes, were more detrimental when the P content in the diet was not increased concurrently. It has been reported that two German Shepherd Dog puppies developed severe clinical signs of skeletal problems, confirmed by radiological and histological findings, as well as hypophosphataemia after being fed excess Ca and a normal amount of P. Severe clinical signs of DOD were observed in a Fox Terrier puppy after adding $CaCO_3$ to its diet, and these signs disappeared after $CaCO_3$ was exchanged with bone meal, which led to an increase in the P supply and a balanced Ca:P ratio.[19]

Taking that information into account, it is possible to conclude that mainly the combination of a Ca excess and a marginal P supply, i.e., a secondary P deficiency, may cause DOD, especially in puppies of large and giant breeds. However, a certain sensitivity of some breeds against a Ca excess supposedly exists alongside a co-factor to breed size. Such a breed difference may help to explain the practical experience that some dogs develop the multifactorial-caused DOD when others stay (clinically) healthy under the same conditions. This also is shown by a study on the effects of a Ca excess combined with an increased P supply on skeletal development in two different dog breeds during growth: The measurements of bone lengths and widths in radiographs of the forearm of Beagles and Foxhound-crossbred dogs at 6 weeks of age, and again after a period of overexposure to Ca at about 27 weeks of age, revealed a growth-reducing influence only in Beagles, without influence on clinical parameters of skeletal health.[16]

The hypothesis that DOD in growing dogs is related to the co-factor "P supply" was reinforced by the results of a study of P deficiency in growing Beagles and Foxhound crossbreds.[20] The puppies received a diet providing approximately 40 to 50% of the recommended P allowance[2] (approximately 3.5% DM) while the Ca supply met the requirements, resulting in a Ca:P ratio above the recommended ratio. In this trial, some puppies of both breeds developed severe clinical signs of DOD showing extremely bowed legs. These signs were reversible by phosphorus repletion.

That excess Ca is much or may be only more hazardous to growing dogs when it is accompanied by a low or a marginal P supply also makes sense in respect to the natural diet of the ancestors of the modern dog: In a pack of hunting dogs or wolves, those low in the hierarchy, such as puppies and young dogs, are feeding on the remains of larger prey, i.e., mainly connective tissue and bones. A certain sensitivity against Ca excess would only make sense here if the digestibility of minerals from bones would be quite low or the dogs would be able to excrete excessive amounts via urine without greater harm (possibly after a period of extra storage of Ca in the skeleton).

Forming a hypothesis, it is more likely that dogs, including growing dogs over wide ranges, are quite unsusceptible against Ca excess because their natural diet contains high quantities of Ca and P. However, a certain degree of Ca excess, maybe after a certain period of time, may have a negative effect on its own, especially on the healthy skeletal development influenced by breed, growth curve, micro-trauma, training intensity, the source and bioavailability of Ca and P, supply of other nutrients, and possible other factors. On the other hand, dogs seem to have only a limited ability to increase the digestibility of Ca and P and, therefore, may be prone to exhibit clinical signs of deficiency. The latter part of the hypothesis also would explain why dogs need so much more Ca compared to humans. As in many other areas, the warning to draw conclusions based on knowledge from human physiology is justified. Dogs are no barking humans!

Energy and Growth Development

Another main factor to consider in the healthy upbringing of dogs is energy supply. It is common knowledge that next to age and body weight, the energy requirements in growing dogs are influenced by a number of factors, such as breed, activity, health status, etc. This leads to the main conclusion that the correct energy supply for the individual puppy can only be determined through monitoring the individual weight development. If the body weight lies within a range of the recommended growth curve, the risk of overfeeding and excessive body weight is minimal. In this context, it is necessary to emphasize that substantial limitations exist in puppies with regard to a common body condition scoring (BCS) system. Not only that the body fat content measured by DEXA is not necessarily in accordance with the predicted content using the BCS system,[21] a low BCS may easily be found in a puppy that is too heavy for its age. This is due to the fact that especially puppies of breeds with a high growth potential use excess energy for growth and not fat accretion. Here, all detrimental effects of a too high body weight on a growing skeleton may act while the puppy itself has a skinny appearance. Because the size of an animal is often mistaken for beauty, good health and strength, especially in large and giant breeds, growing dogs of those breeds often are too heavy.[22] Here, restrictive feeding is required to let the dog grow more slowly but healthfully.

The recommendations for energy supply during growth given in the NRC[2] overestimate the *de facto* needs,[23] which may lead to wrong feeding recommendations. Dog owners following feeding guides based on existing predictions for energy needs, therefore, may overfeed their puppies. Additionally, most of the owners add a lot of treats and snacks to their puppies' daily rations for training or bonding reasons. But not only an excessive energy supply and a resulting forced growth development with detrimental effects may result. When the average commercial diet is designed to meet all nutrient requirements, presuming an

energy consumption that in reality is overestimated, the nutrient supply will be insufficient when the puppy eats less. If, for example, the puppy requires only 70% of the presumed average daily energy to grow according to the recommendation, it will consume 70% or less (also subject to the amount of snacks and treats) of the presumed amount of diet and therefore only 70% of the nutrients. Even when safety margins are incorporated, the nutrient supply is probably marginal or insufficient. This also is the reason it is not recommendable to use diets meant for maintenance/adult dogs during growth. In most of those products, the nutrient density is not sufficient for puppies let alone for those individuals with below-average energy requirements. The consequences of feeding such inappropriate diets to growing dogs, such as signs of DOD, is something we regularly see in our nutrition consultation practice.[24]

Other Nutrients with Impact on Skeletal Development

Other nutrients, such as vitamins A and D and trace elements, including zinc, copper, etc., have a possible negative impact in case of deficiency or excess, respectively, and therefore have to be considered, especially in diets for growing dogs. Other nutrients, such as protein, are overrated in regard to their impact. Often a warning is expressed that a protein excess impairs the healthy growth of puppies, and there are products on the market advertised by indicating the restricted protein concentration for a healthy growth, although it was demonstrated that this effect does not exist.[24] This is partly true for the presumed detrimental effect of a vitamin A excess on skeletal development. We learned recently[25] that the safe upper limit in puppies is 26fold higher than expected.[2]

As a consequence of these factors, the recommendation for practical feeding remains that one should try to meet the requirements at least for energy, Ca and P in a puppy as accurately as possible. This leads to the question if it is possible to create a diet that is perfect for all breeds, ages, activities, life stages, etc., but also to the insight that special requirements need to be addressed with matching products. A careful choosing of a suitable product is crucial especially during the very sensitive life stage of growth.

References

1. Ernährungsbericht 2008. Deutsche Gesellschaft für Ernährung e.V. 2009. http://www.dge.de/modules.php?name=News&file=article&sid=914.

2. National Research Council. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006.

3. The European Pet Food Industry Federation. Nutritional guidelines for dogs and cats. 2012. http://www.fediaf.org/press-area/press-releases/news-detail.

4. Becker N, Kienzle E, Dobenecker B. Calcium deficiency: A problem in growing and adult dogs — Two case reports. *Tierarztl Prax Ausg K Kleintiere Heimtiere*. 2012;24(40;2): 135-139.

5. De Fornel-Thibaud P, Blanchard G, Escoffier-Chateau L, et al. Unusual case of osteopenia associated with nutritional calcium and vitamin D deficiency in an adult dog. *J Am An Hosp Assoc*. 2007;43(1):52-60.

6. Mack JK, Alexander LG, Morris PJ, et al. Demonstration of uniformity of true calcium digestibility in the adult dog and cat. *J of Nutr*. 2013 (submitted)

7. Von Engelhardt W, Breves G. In *Physiologie der Haustiere (Physiology of domestic animals)*. Enke Verlag, Stuttgart, Germany. 2010(3rd ed).

8. Hazewinkel HAW, Goedegebuure SA, Poulos PW, et al. Influences of chronic calcium excess on the skeletal development of growing Great Danes. *J Am An Hops Assoc*. 1985;135:305-310.

9. Hazewinkel HAW, Hackeng WHL, Bosch R, et al. Influences of different calcium intakes on calciotropic hormones and skeletal development in young growing-dogs. *Front Horm Res*. 1987;17: 221-232.

10. Hazewinkel HAW, Brom WE, Van't Kloster A. Calcium metabolism in Great Dane dogs fed diets with various calcium and phosphorus levels. *J Nutr*. 1991;121:S99-S106.

11. Voorhout G, Hazewinkel HAW. A radiographic study on the development of the antebrachium in Great Dane pups on different calcium intakes. *Vet Radiol*. 1987;28:152-157.

12. Goedegebuure SA, Hazewinkel HAW. Morphological findings in young dogs chronically fed a diet containing excess calcium. *Vet Pathol*. 1986;23:594-605.

13. Schoenmakers I, Hazewinkel HAW, Voorhout G, et al. Effects of diets with different calcium and phosphorus contents on the skeletal development and blood chemistry of growing Great Danes. *Vet Rec*. 2000;147:652-660.

14. Goodman SA, Montgomery RA, Titch RB, et al. Serial orthopaedic examinations of growing Great Dane puppies fed three diets varying in calcium and phosphorus. In: *Recent Advances in Canine and Feline Nutrition. Iams Nut Sym Proc*. Reinhart GA, Carey DP (eds). Orange Frazer Press, Wilmington, NC. 1998;63-70.

15. Jenkins K, Phillips P. The mineral requirement of the dog: The relation of calcium, phosphorus and fat levels to minimal calcium and phosphorus requirements. *J Nutr*. 1960;70:241-246.

16. Dobenecker B, Kasbeitzer N, Flinspach S, et al. Calcium-excess causes subclinical changes of bone growth in Beagles but not in Foxhound-crossbred dogs, as measured in X-rays. *J An Physiol An Nutr*. 2006;90(9-10):394-401.

17. Stephens LC, Norrdin RW, Benjamin SA. Effects of calcium supplement and sunlight exposure on growing Beagle dog. *Am J Vet Res*. 1985;46:2037-2042.

18. Dobenecker B. Factors that modify the effect of excess calcium on skeletal development in puppies. *Br J Nutr*. 2011;106(1): S142S14-5. doi: 10.1017/S0007114511002959.

19. Marek J, Wellmann O. In: *Die Rhachitis — Pathologischer Teil*. Gustav Fischer Verlag, Jena, Germany. 1931.

20. Dobenecker B, Kienzle E. Normal calcium and low phosphorus intake in puppies leads to extremely bowed legs which are reversible by phosphorus repletion. *ESVCN Congress Proc*. Vienna. 2008:49.

21. Dobenecker B. Comparison of body weight, body condition score and pelvic circumference in dogs to predict adipose tissue as measured by DEXA. *ESVCN Congress Proc*. Vienna. 2008:24.

22. Dobenecker B, Kienzle E, Köstlin R, et al. Mal- and over-nutrition in puppies with or without disorders of skeletal development. *J An Physiol An Nutr*. 1998;80:76-81.

23. Dobenecker B, Endres V, Kienzle E. Energy requirements of puppies of two different breeds for ideal growth from weaning to 28 weeks of age. *J An Physiol An Nutr*. 2011. doi:10.1111/j.1439-0396.2011.01257.x. (Epub ahead of print)

24. Kölle P, Jacobs S, Klesty C, et al. Calciumunterversorgung eines Hundewelpen durch Fütterung eines calciumreduzierten Welpenalleinfutters. *Tierärztl Prax*. 2006;34(K):104-107.

25. Nap RC, Hazewinkel HAW, Voorhout G, et al. The influence of the dietary protein content on growth in giant breed dogs. *VCOT*. 1993;6(1):5-12.

26. Morris PJ, Salt C, Raila J, et al. Safety evaluation of vitamin A in growing dogs. *Br J Nutr*. 2012;28(108;10):1800-1809. doi:10.1017/S0007114512000128. (Epub, 2012)

The New Age of Working Dogs: Different Jobs, Different Diets

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Nutrition research in working dogs has focused primarily on sprinting Greyhounds and endurance sled dogs. More recent investigation into other sporting arenas like agility dogs, olfactory-task oriented dogs (detection dogs and Foxhounds), and other event dogs (field trial to dock diving dogs) has shown that potentially these dogs have different dietary or supplement needs for peak performance. Unfortunately, in the past 10 years as performance endeavors have changed and evolved, little research has been done examining nutrition and its role in these athletic endeavors. As a result, optimal nutrition information relies heavily on sled dog and Greyhound research and the practices of lay people. Dispelling myths and molding research findings into everyday practices remains a challenge.

Energy for Exercise

Increasing physical activity and the effects of training have been extensively studied in dogs during treadmill exercise. Maximal oxygen utilization (VO_2 max) shows variability between 5.8 to 10.3 L/kg/hr in conditioned dogs. The highest VO_2 max was found in Alaskan Husky purpose-bred sled dogs (10.3 L/kg/hr), and purpose-bred Foxhounds were shown to have a VO_2 max of 6.7-6.8 L/kg/hr.[1-3] For all practical intents and purposes, an average 20 kg Foxhound or Alaskan sled dog working at maximal oxygen consumption burns approximately 700 to 900 kilocalories per hour. In the field, there are other factors to consider, such as external temperature, thermal regulation, variability in terrain, and incline versus decline. Treadmill exercise reveals that there is a loss of efficiency in energy utilization with increased incline due to the need for vertical rise increasing overall Kcal expenditure. Larger dogs need to exert more energy to break the fall that occurs on decline.[4] Uneven or poor footing (snow and sand) as well as load bearing result in increased energy expenditure.[5]

Dietary calculations and metabolic studies have suggested that Alaskan Huskies training in the northeast United States showed an energy expenditure of approximately 4000 to 5000 Kcals when training and racing an average 10 km per day, suggesting approximately 440 Kcals/kg.$^{0.75}$ [6,7] Orr suggested an intake of approximately 4400 Kcals when pulling a heavy load over ice 32 km per day (270 Kcals/ kg$^{0.75}$).[8] Double-labeled water studies suggest that dogs running an average of 79 km a day over eight days expended approximately 438 Kcals/kg$^{0.75}$ body weight[9]; however, in extreme racing conditions with dogs running at a speed of approximately 7 km an hour approximately 14 hours per day over five days (total 490 km) at temperatures between -10 to 35^{o}C suggested the Kcal expenditure in 18 dogs averaged 1052 ± 192 Kcals/kg$^{0.75}$ body weight per day.[10]

Field studies examining energy expenditure of working Greyhounds have shown that the average 32 to 35 kg Greyhound expends approximately 2050 to 2160 Kcals per day. These studies take into account the typical training regimen for a racing Greyhound, which includes being penned or caged with daily sprint training in enclosed paddocks for approximately 30 minutes and two races a week.[11,12] Interestingly, studies by Hill and colleagues suggested that feed restriction during racing from the normal of approximately 155 Kcal/kg$^{0.75}$ body weight down to a restricted regimen of only 137 Kcal/kg$^{0.75}$ resulted in decreased racing times, making mild feed restriction advantageous.[12] Though speculative, it is possible that agility, dock diving, earthdog, flyball, sprint sled racing, short-distance skijoring, lure coursing, and field trial dogs may benefit from similar feeding practices before and during competition.

Dietary Protein — Beyond Energy

Nitrogen retention studies are the gold standard for protein sufficiency and rely on nitrogen balance as the measure of adequacy; however, most canine nitrogen retention studies have not examined the consequences on lean mass. Necessary protein content in the diet has been well-studied in both racing Greyhounds and sled dogs. Dietary protein helps maintain musculoskeletal integrity and appropriate total protein, albumin and red blood cell status. The hematocrit and serum albumin tend to decrease with training and racing, which appears to be a result of the overtraining syndrome in endurance dogs.[6,13] Adequate protein intake may be helpful in ameliorating this condition. Studies examining protein consumption and its role in maintaining red blood cell counts and hematocrit in training sled dogs have postulated that approximately 30% of the metabolizable energy (70 to 80g protein/1000 Kcals) should come from highly digestible animal-based protein.[13] Four groups of sprint-racing sled dogs exercising approximately 60 km in fieldwork per week plus treadmill training each week on four diets comprising 18%

ME protein (48g/1000 Kcals), 24% ME protein (60g/1000 Kcals), 30% ME protein (75g/1000 Kcals), and 36% ME protein (90g/1000 Kcals) from an initial diet of approximately 26% ME protein were examined. After 12 weeks of feeding each diet routine, complete blood counts, serum chemistries, VO_2 max, and physical assessment were performed. Six of eight dogs in the lowest protein diet (18% ME) sustained musculoskeletal injuries and showed a drop in VO_2 max.[3]

Querengaesser and colleagues examined diets of approximately 72 and 85g protein/1000 Kcals and showed that there was no difference in the hematocrit decline over a six-month period of training, but the higher protein group had elevated post-exercise hematocrit, further suggesting that protein may affect parameters associated with performance.[14] Another study of mongrel dogs exercised four hours per day at 12 km/hr compared soy protein versus fish- and meat meal-based protein at approximately 35% of the ME. After three weeks, this study showed decreased hematocrit and increased red blood cell fragility in the soybean meal-fed dogs.[15] These data suggest that endurance dogs should receive minimally 70g/1000 Kcals (approximately 26% of ME) of a highly digestible protein source that is adequately balanced with essential amino acids, and no upper limit of protein consumption has been defined during rigorous exercise.

In sprinting dogs, the picture may be slightly different as Hill and colleagues performed studies suggesting that racing Greyhounds perform better on lower protein diets of 63g/1000 Kcals versus 106g/1000 Kcals.[16] These diets substituted carbohydrate for the protein in an isocaloric exchange of nutrients; therefore, the enhanced performance may have been due to the increased carbohydrate in the diet, not the lack of protein. Though it appears that approximately 60g/1000 Kcals may be adequate for racing Greyhounds, further decreases in protein have not been evaluated and are contrary to current feeding practices. Most Greyhounds are provided 0.25 to 0.5 kg of raw or cooked meat mixed with dry commercial dog food, approaching 106g/1000 Kcals to meet their energy requirements.[17,18] Without firm recommendations or studies in sprinting dogs other than Greyhounds, a reasonable recommendation is that most sprinting and intermediate activity dogs receive minimally 60g/1000 Kcals consumed (22 to 24% ME).

Dietary Fat and Carbohydrate — Energy and Demands

Unlike athletic humans, athletic dogs appear to function well and remain healthy on higher fat diets. The generation of energy from fat is up to 70% of the ME used during long-duration exercise, suggesting a propensity for fat utilization due to the dog's high aerobic activity in skeletal muscle and increased mitochondrial density as compared to humans.[19] Beagles running at low to moderate intensity increased their time to exhaustion by approximately 25 percent when provided diets with 55 to 81g/1000 Kcals of fat versus 33g/1000 Kcals.[20] Kronfeld, Hammel and colleagues showed that dogs performed equally well on diets containing absolutely no carbohydrate compared to two diets with increasing carbohydrate content.[6,7] Further studies in trained and untrained sled dogs showed that when comparing a high-carbohydrate (162g/1000 Kcals; 59% ME), low-fat (18g/1000 Kcals; 14% ME) diet to a high-fat (70g 1000 Kcals; 58% ME), low-carbohydrate (43g/1000 Kcals) diet, there was no difference in muscle glycogen storage. Interestingly, the dogs on the high-fat diet showed diminished muscle glycogen consumption with exercise.[21] Endurance Huskies racing approximately 100 km per day over five days showed immediate glycogen depletion, with an increase in skeletal muscle glycogen and gradual depletion of skeletal muscle triglyceride, further suggesting that the longer these endurance dogs run, the more they adapt to fat utilization sparing muscle glycogen.[22]

Fat consumption can supply approximately 60 to 70% of the ME, and in times of extreme demand, fat may supply up to 85% of the ME, particularly in endurance sled dogs; however, many of today's activities do not require this type of excessive fat use and adaptation. In fact, many may benefit from limiting fat consumption similar to findings in Greyhounds. Toll and colleagues have shown that Greyhounds on a high-carbohydrate diet (46% ME versus 6% ME) were 0.4km/hr faster.[23] The high-carbohydrate diets contained only 31% ME fat, while the high-fat diet consisted of 75% ME as fat. These results taken together with previous reports suggesting higher carbohydrate diets enhance performance imply that approximately 30% ME fat and 24% ME protein with the remaining ME from carbohydrate seems adequate for racing Greyhounds and possibly other sprinting athletes, such as agility, dock diving, flyball, earthdog, and lure-coursing dogs. Fortunately for dog owners, this type of dietary breakdown results in a product that would be approximately 24 to 28% dry matter protein, 12 to 14 % dry matter fat, and 45 to 50% carbohydrate, which is similar to many commercial adult pet foods on the market.

Dietary Fat — Beyond Fuel

Very little information regarding optimal dietary fats for canine athletes is available, but there has been some speculation that chain length and saturation can affect a variety of issues from inflammation to oxidative potential during exercise.[24] Typical fatty acids found in meats and most vegetable oils are 16 carbons and greater, which require free fatty acid absorption and repackaging through microsomal transferase enzymes into triglycerides for transportation and storage. Medium-chain triglycerides when digested liberate eight to12 carbon fatty acids that undergo some direct absorption into the bloodstream and are transported via albumin to cells for metabolism. This has led to speculation that medium-chain triglycerides in the form of coconut and palm oils can be utilized more rapidly at the initiation of exercise leading to further glycogen sparing.[24] This does not appear to

be the case in dogs, and one pilot study in athletic dogs showed no improvement in fat utility using medium-chain triglycerides; therefore, it cannot currently be recommended as a strategy in fat adaptation.[24]

Many performance animals, including Foxhounds, hunting dogs and service detection dogs, rely on their detection capabilities; therefore, the potential for increased polyunsaturated fatty acids in olfaction should be discussed. A small study showed that olfactory performance appeared to be enhanced or maintained when dogs were provided a base diet in which dietary fat sources were switched from animal-based fat to corn oil-based fat; however, the results of this study truly showed that conditioning was more important for detection capabilities.[25] As a follow-up to this study, a larger study was performed whereby 17 conditioned Labrador Retriever detection dogs were examined in a 3-by-3 Latin square design and were provided three different diets containing: 26% ME protein and 28% ME fat (typical adult maintenance diet); 26% ME protein and 57% ME fat (typical performance diet); or 18% ME protein and 57% ME fat (typical adult diet plus supplemental corn oil) for 12 weeks. The dogs then underwent a treadmill exercise stress test at 12.5 km/hr at 2.5% incline for 30 minutes and were tested for alterations in blood gases, vitals, complete blood count, serum chemistry, and cortisol status. Results suggest that the lower ME protein diet (18% ME) dogs actually have a significantly lower body temperature while running and during recovery. More interestingly, when utilizing GLM statistical modeling examining parameters such as time, age, gender, and conditioning in a standardized olfactory testing procedure, the only parameter to show significant influence on increased olfactory acuity was being fed the 18% ME protein and 57% ME corn oil-enriched diet (Gillette and Angle, personal communication). Exactly what led to the increased olfactory acuity remains to be determined; however, current postulation is that increased polyunsaturated fats in the olfactory epithelium may lead to better sensory signaling and/or a decrease in core body temperature, while a lower protein diet allowed for better thermoregulation and more nasal breathing rather than open-mouthed panting, which could explain greater delivery of aerosolized particles to the olfactory bulb. Regardless, these findings warrant further investigation into moderate-protein, high-fat diets for performance dogs dependent on olfactory stimuli (hunting dogs, Foxhounds, detection dogs) for success.

Carbohydrates — Timing and Strategy

The use of carbohydrate as a major dietary substrate makes sense in sprinting animals like Greyhounds, with approximately 40 to 50% of the ME in the diet as highly digestible carbohydrates. Endurance sled dogs need less than 10% of the ME as carbohydrate, as there are no definitive carbohydrate requirements.[5,6] Considerable attention has been given to the source of carbohydrate used in commercial diets suggesting that more complex dietary carbohydrate sources, such as barley and sorghum, may provide a more gradual release of glucose to the bloodstream improving the glycemic index.[26] Carbohydrate sources, such as corn and rice, cause a more rapid absorption and moderate spike in serum glucose. It is still unclear how this would benefit the performance canine, particularly since feeding regimens (discussed later) are unlikely to take advantage of available glucose. The lack of carbohydrate in endurance athlete diets warrants the concept invalid. In general, sprint performance canine owners prefer rapidly absorbable carbohydrate sources with minimal total dietary fiber to decrease fecal bulk.

Studies performed in sled dogs have definitively shown that post-exercise supplementation with a maltodextrin supplement at 1.5g/kg body weight within 30 minutes of exercise increases skeletal muscle glycogen within four to 24 hours.[27,28] In both studies, it was evident that this dosing returned muscle glycogen to baseline concentrations before exercise, while one study showed that without supplementation skeletal muscle glycogen content was only 50% of baseline concentrations the following day.[28] Based on this information, post-exercise carbohydrate repletion is recommended in dogs running anywhere between five minutes and three hours per day at high intensity, particularly when expected to perform similarly the following day.

In the human athletic arena, protein with carbohydrate is often provided post-exercise. The protein is thought to help curb skeletal muscle proteolysis after intermediate exercise.[29] This approach has not been examined in the canine performance arena, and only one study has examined whether the addition of 0.5 mg/kg body weight of protein as a chicken liver hydrolysate to maltodextrin at 1.5 g/kg body weight improved muscle glycogen repletion, and it did not.[28] In humans, the use of whey-based protein in young athletes post-exercise appears to help in retention of lean mass and possibly improves glycogen repletion in some situations,[28] but this concept remains unstudied sufficiently in performance canines with some data suggesting diminished muscle turnover.[30]

Dietary Fiber

Insoluble fiber results in fecal bulk but has the capacity to act as a binding agent that can improve fecal quality when diarrhea is a problem. Soluble fiber has the capacity to alter the large intestinal microflora and potentially increase the absorptive surface of the small and large intestines through villous hypertrophy. This has been used strategically in canid athletes with stress-related diarrhea. Soluble fiber tends to be a matrix on which certain bacterial families, including *Bifidobacteria, Lactobacillus* and *Streptococcus*, thrive.[31,32] These bacterial families ferment soluble fiber sources and liberate volatile fatty acids that promote colonocyte regeneration and may improve recovery from diarrhea.[32] Therefore, many of the enteric formulas of commercial dog food use small amounts of gums, soy fiber, fructooliogsachharides,

other oligosaccharides, and mixed insoluble and soluble fiber sources to improve fecal quality and intestinal absorptive capabilities. The amount of soluble fiber added is generally around 1% of dry matter in the diet since overfermentation can result in deteriorating fecal quality, particularly in performance dogs that can be considered "hard keepers."[32,33] In many instances, the use of psyllium husk powder on feed is used in exercising canines to improve exercise-related stress diarrhea. Psyllium husk fiber is unique since it is a mucilage with water-binding properties and much like insoluble fiber provides a matrix that improves fecal quality. It is often recommended to start with approximately 4 grams of psyllium (1 rounded teaspoon of fine powder) per day, titrating upward, not exceeding 16 grams per day in a typical 20 to 30 kg canine athlete.[34]

Feeding Strategies in Canine Athletes

Feeding patterns can affect performance. Frequency and time of feeding become important not only to decrease fecal bulk but also to maximize metabolites that are typically used for the activity. Sprinting dogs running less than 10 minutes during a single bout of exercise will benefit from modest feed restriction 24 hours prior to exercise (decreasing total meal by 20%) during single-day events to decrease fecal bulk. Although some advocate small meals rich in carbohydrate before exercise to provide glucose as fuel for impending exercise, there is little data to support this strategy in dogs. These sprinting and intermediate athletes, particularly agility and field trial dogs, for example, that perform multiple bouts of exercise in a day may benefit from post-exercise carbohydrate in small amounts immediately after a bout of exercise when expected to undertake another bout within two to three hours. If repetitive bouts are close together, this may not be advised to avoid vomiting or regurgitation. During multiple days of competition, post-exercise glycogen repletion is advised within 30 minutes of the last bout of exercise for the day to replete muscle glycogen.[35]

Intermediate athletes exercising typically once a day for 20 to 120 minutes generally rely on both glycogen and fat for energy generation, therefore it is recommended that they be fed diets moderate in fat (40 to 60% ME) and carbohydrate (15 to 25% ME) and high in protein (30% ME). This represents a wide variety of activities from six-mile sprint sled dog races and 20-minute field trials to 30-mile sprint sled dog races and 90-minute hunting competitions, whereby the former category may benefit from only 40% ME fat while the latter category may benefit from 60% ME fat. This overall dietary approach allows adequate muscle glycogen repletion during training and helps increase mitochondrial volume with the moderate to higher fat content. Fat will be used as a primary fuel at rest and within 20 minutes of exercise allowing for glycogen sparing, providing a reserve fuel source when these athletes are asked to run above 60% of VO_2 max. These athletes will benefit from post-exercise carbohydrate supplementation to restore muscle glycogen concentrations, particularly during multiple-day events.[27,28] In an effort to promote fat use and lipolysis, feeding a single meal each day may be advantageous with that meal given about two hours post-exercise. Modest feed restriction (20%) the day before racing will prevent defecation during exercise, promote lipolysis and decrease fecal bulk.[12,35] However, if dealing with a line of dogs or breed predisposed to bloat, or gastric dilitation and volvulus, care should be taken to not feed larger meals immediately after exercise and twice daily feeding may be more appropriate.

Endurance athletes (i.e., Foxhounds and sled dogs) tend to be fed one or two large meals each day during training. These meals should be approximately 30% ME protein, 60% to 70% ME fat and negligible carbohydrate (less than 10% ME). During heavy training, some sled dogs will be running up to 60 miles two to three times a week interspersed with shorter exercise bouts. The ration will likely compromise approximately 50% commercial dog food, with the rest as high fat, preferably cooked meat (%vol/% vol basis). This is considered necessary to achieve the caloric density and digestibility needed for competitive racing and long-term hunting. Since carbohydrate is not a primary fuel for exercise in these athletes, post-exercise carbohydrate supplementation for glycogen repletion is not recommended. Foxhounds and various coonhound breeds that course through fields daily and pointing dogs asked to hunt for multiple hours may benefit from post-exercise carbohydrates, particularly if they are not trained regularly, since they will rest for a significant time (greater than eight hours) between exercise bouts.

Another classification of athlete would be the so-called "weekend warrior" or the typical military dog that sits untrained or minimally trained in kennels for extended periods of time and will then be deployed into the field for active duty. These dogs largely represent law enforcement dogs, detection dogs, some hunting dogs, and even some event dogs in homes and kennels. Although there is no definitive dietary strategy, we should likely feed these dogs according to their expected workload in the field, and in many of these cases, nutrition will not be able to make up for the lack of training from owners and trainers with these types of dogs. These owners and policymakers should be coached on appropriate training that will provide benefits that far outweigh dietary alterations or performance-enhancing supplements.

Dietary Supplementation

There has been significant discussion surrounding the idea of antioxidant of vitamin and mineral supplementation in exercise. Much of this was initiated by pilot studies reporting that serum antioxidants, such as vitamins C and E, were depleted in serum post-exercise and that oxidative damage due to exercise may be the culprit.[13,36,37] However, after many years of examining various supplements in endurance Huskies and Greyhounds, including vitamins C and E, there has been no consensus regarding their

efficacy of preventing oxidative damage or protecting skeletal muscle from increased permeability changes reflected as creatine kinase activity or decreasing exertional rhabdomyolysis.[38-40] In fact, in racing Greyhounds the supplementing of vitamins C and E have been associated with increased racing times.[41,42] The lack of efficacy in antioxidant or pro-energetic supplementation to enhance performance or decrease biochemical manifestations of muscle damage has led to ambiguity regarding their efficacy as antioxidants for sporting dogs.[43-45] Newer insights into maintaining muscle integrity and improving energetics have been attempted through supplements such as β-hydroxy-β-methylbutyrate, resveratrol and betaine.[46-50] Rodent and human studies have shed light into new paradigms in mitochondrial function and improved energetics that may prove useful in dogs with appropriate research in the future.

References

1. Musch TL, Haidet GC, Ordway GA, et al. Dynamic exercise training in Foxhounds. I. Oxygen consumption and hemodynamic responses. *J Appl Physiol*. 1985;59:183-189.

2. Ordway GA, Floyd DL, Longhurst JC, et al. Oxygen consumption and hemodynamic responses during graded treadmill exercise in the dog. *J Appl Physiol*. 1984;57:601-607.

3. Reynolds AJ, Reinhart GA, Carey DP, et al. Effect of protein intake during training on biochemical and performance variables in sled dogs. *Am J Vet Res*. 1999;60:789-795.

4. Taylor CR, Caldwell SL, Rowntree VJ. Running up and down hills: Some consequences of size. *Science*. 1972;178:1096-1097.

5. Taylor RJF. The work output of sledge dogs. *J Physiol*. 1957; 137:210-217.

6. Hammel EP, Kronfeld DS, Ganjam VK, et al. Metabolic responses to exhaustive exercise in racing sled dogs fed diets containing medium, low or zero carbohydrate. *Am J Clin Nutr*. 1977;30:409-418.

7. Kronfeld DS, Hammel EP, Ramberg CF, et al. Hematological and metabolic responses to training in racing sled dogs fed diets containing medium, low and zero carbohydrate. *Am J Clin Nutr*. 1977;30:419-430.

8. Decombaz J, Jambon M, Pigueet C, et al. Energy intake and expenditure of sled dogs during the Alpirod race 1995. *Proc Sec An Sled Dog Vet Med Assoc Symp*. Grandjean D, Vanek J (eds). 113-118.

9. Orr NWM. The feeding of sledge dogs on Antarctic expeditions. *J Nutr*. 1966; 20:1-12.

10. Hinchcliff KW, Reinhart GA, Burr JR, et al. Metabolizable energy intake and sustained energy expenditure of Alaskan sled dogs during heavy exertion in the cold. *Am J Vet Res*. 1997;58: 1457-1462.

11. Hill RC, Bloomberg MS, Legrand-Defretin V, et al. Maintenance energy requirements and the effects of diet on performance in racing Greyhounds. *Am J Vet Res*. 2000;61:1566-1573

12. Hill RC, Lewis DD, Scott KC, et al. Mild food restriction increases the speed of racing Greyhounds. *J Vet Intern Med*. 1999;13:281.

13. Kronfeld DS, Adkins TO, Downey RL. Nutrition, anaerobic and aerobic exercise and stress. In *Nutrition of Dog and Cat: Waltham Sym*. Burger IH, Rivers JPW (eds). 1989; 133-145.

14. Querrengaesser A, Iben C, Leibetseder J. Blood changes during training and racing in sled dogs. *J Nutr*. 1994;124: 2760S-2764S.

15. Yamada TM, Tohori T, Ashida T, et al. Comparison of effects of vegetable protein diet and animal protein diet on the initiation of anemia during vigorous physical training in dogs and rats. *J Nutr Sci Vitaminol* (Tokyo). 1987;33:129-149.

16. Hill RC, Lewis DD, Scott KC, et al. The effects of increased protein and decreased carbohydrate in the diet on performance and body composition in racing Greyhounds. *Am J Vet Res*. 2001;62:440-447.

17. Kohnke JR. Nutrition in the racing Greyhound. In *Canine Sports Medicine and Surgery*. Dee JF, Bloomberg MS, Taylor RA (eds). W.B. Saunders Co., Philadelphia, PA 328-336.

18. Hill RC. The nutritional requirements of exercising dogs. *J Nutr*. 1998;128:S2686-S2690.

19. Wakshlag JJ, Cooper BJ, Wakshlag RR, et al. Biochemical evaluation of mitochondrial respiratory chain enzymes in canine skeletal muscle. *Am J Vet Res*. 2004;65:480-484.

20. Downey RL, Kronfeld DS, Banta CA. Diet of Beagles affects stamina. *J Am An Hosp Assoc*. 1980;16:273-277.

21. Reynolds AJ, Fuhrer L, Dunlap HL, et al. Effect of diet and training on muscle glycogen storage and utilization in sled dogs. *J Appl Phys*. 1995;79:1601-1607.

22. McKenzie EC, Hinchcliff KW, Valberg SJ, et al. Assessment of alterations in triglyceride and glycogen concentrations in muscle tissue of Alaskan sled dogs during repetitive prolonged exercise. *Am J Vet Res*. 2008;69:1097-1103.

23. Toll PW, Pieschl RL, Hand MS. The effect of dietary fat and carbohydrate on sprint performance in racing greyhound dogs. In *Proc of the Eighth Intl Racing Greyhound Sym*. North American Veterinary Conference. 1992;1-3.

24. Reynolds AJ. The role of fat in the formulation of performance rations: Focus on fat sources. In *Recent Advances in Canine and Feline Nutrition, Vol II*. Hayek MG, Lepine AJ, Sunvold GD (eds). Orange Frazer Press, Wilmington, OH. 277-281.

25. Altom EK, Davenport GM, Myers LJ, et al. Effect of dietary fat source and exercise on odorant-detecting ability of canine athletes. *Res Vet Sci*. 2003;75:149-155.

26. Sunvold GE, Bouchard GF. The glycemic response to dietary starch. In *The Recent Advances in Canine and Feline Nutrition, Vol II*. Hayek MG, Lepine AJ, Sunvold GD (eds). Orange Frazer Press, Wilmington, OH. 23-131.

27. Reynolds AJ, Carey DP, Reinhart GA, et al. Effect of post-exercise carbohydrate supplementation on muscle glycogen repletion in trained sled dogs. *Am J Vet Res*. 1997;58:1252-1256.

28. Wakshlag JJ, Sneddon KA, Otis AM, et al. Effects of post-exercise supplements on glycogen repletion in skeletal muscle. *Vet Therap*. 2002;3:226-234.

29. Betts JA, Williams C. Short-term recovery from prolonged exercise: Exploring the potential for protein ingestion to accentuate the benefits of carbohydrate supplements. *Sports Med*. 2010; 40:941-959.

30. Okamura K, Doi T, Hamada K, et al. Effect of amino acid and glucose administration during post-exercise recovery on protein kinetics in dogs. *Am J Phys*. 1997; 272:E1023-E1030.

31. Whelan K, Schneider SM. Mechanisms, prevention and management of diarrhea in enteral nutrition. *Curr Opin Gastroenterol*. 2011;27:152-159.

32. Wakshlag JJ, Struble AM, Simpson KW, et al. Negative fecal characteristics are associated with pH and fecal flora alterations during dietary change in dogs. *Int J App Res Vet Med*. 2010;9: 278-283.

33. Gagné JW, Wakshlag JJ, Simpson KW, et al. Effects of a synbiotic on fecal quality, short-chain fatty acid concentrations, and the microbiome of healthy racing sled dogs. *J An Phys An Nutr*. (in press)

34. Leib MS. Treatment of chronic idiopathic large-bowel diarrhea in dogs with a highly digestible diet and soluble fiber: A retrospective review of 37 cases. *J Vet Intern Med*. 2000;14:27-32.

35. Reinhart GA. Nutrition for Sporting dogs. In *Canine Sports Medicine and Surgery*. Dee JF, Bloomberg MS, Taylor RA (eds). W.B. Saunders Co., Philadelphia, PA. 348-356.

36. Toll PW, Gillette RL. *The Canine Athlete in Small Animal Clinical Nutrition*. Hand MS, Thatcher CD, Remillard RL, Roudebusch P, Novotny TL (eds). Walsworth Publishing, Marceline, MO. 2010;5:323-357.

37. Grandjean D. Nutrition for Sled dogs. In *Canine Sports Medicine and Surgery*. Dee JF, Bloomberg MS, Taylor RA (eds). W.B. Saunders Co, Philadelphia PA. 336-347.

38 Piercy RJ, Hinchcliff KW, Morely PS, et al. Association between vitamin E and enhanced athletic performance in sled dogs. *Med Sci Sports Exerc*. 2001;33:826-833.

39. Piercy RJ, Hinchcliff KW, Morely PS, et al. Vitamin E and exertional rhabdomyolysis during endurance sled dog racing. *Neuromuscul Disord*. 2001;11:278-286.

40. Scott KC, Hill RC, Lewis DD, et al. Effect of oral alpha tocopherol acetate supplementation on vitamin E concentrations in Greyhounds before and after a race. *Am J Vet Res*. 200;62:1118-1120.

41. Hill RC, Armstrong D, Browne RW, et al. Chronic administration of high doses of vitamin E appears to slow racing Greyhounds. *FASEB J*. 15:A990.

42. Marshall RJ, Scott KC, Hill RC, et al. Supplemental vitamin C appears to slow racing Greyhounds. *J Nutr*. 2002;132:1616S-1621S.

43. Piercy RJ, Hinchcliff KW, DiSilvestro RA, et al. Effect of dietary supplements containing antioxidants on attenuation of muscle damage in exercising sled dogs. *Am J Vet Res*. 2000; 61:1438-1445.

44. Baskin CR, Hinchcliff KW, DiSilvestro RA, et al. Effects of dietary antioxidant supplementation on oxidative damage and resistance to oxidative damage during prolonged exercise in sled dogs. *Am J Vet Res*. 2000;61:886-891.

45. Dunlap KL, Reynolds AJ, Duffy LK. Total antioxidant power in sled dogs supplemented with blueberries and the comparison of blood parameters associated with exercise. *Comp Biochem Physiol A Mol Integr Physiol*. 2006;143:429-434.

46. Armstrong LE, Casa DJ, Roti MW, et al. Influence of betaine consumption on strenuous running and sprinting in a hot environment. *J Strength Cond Res*. 2008;22:851-860.

47. Trepanowski JF, Farney TM, McCarthy CG, et al. The effects of chronic betaine supplementation on exercise performance, skeletal muscle oxygen saturation and associated biochemical parameters on resistance trained man. *J Strength Cond Res*. 2011;25:3461-3470.

48.Dolinsky VW, Jones KE, Sidhu RS, et al. Improvements in skeletal muscle strength and cardiac function induced by resveratrol during exercise training contribute to enhanced exercise performance in rats. *J Phys*. 2012; 590:2783-2799.

49. Rowlands DS, Thompson J. Effects of β-hydroxy- β-methylbutyrate supplementation during resistance training on strength, body composition, and muscle damage in trained and untrained young men: A meta-analysis. *J Strength Cond Res*. 23: 826-836.

50. Wilson JB, Lowery RF, Joy JM, et al. β-hydroxy- β-methylbutyrate free acid reduces markers of exercise induced muscle damage and improved recovery in resistance-trained men. *Br J Nutr*. 2013, Jan 1-7. (Epub ahead of print)

Feeding Practices of Dog Breeders in the U.S. and Canada

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Abstract

Feeding practices of dog breeders throughout the reproductive cycle can influence fertility and litter survival, as well as have long-term consequences on the health of puppies. Therefore, we sought to determine the prevalence of dog breeders who feed their animals diets that meet currently accepted nutritional standards for reproduction and early development, and to investigate factors that influence their feeding practices.

Using a Web-based questionnaire, we surveyed dog breeders from the U.S. and Canada who were 18 years and older and who breed at least one litter every two years. Information collected included: feeding practices during three life stages (pre-pregnancy, gestation/lactation, and puppy growth); criteria for diet selection; and sources of diet information. Nutritional adequacy of commercial diets was determined by AAFCO (Association of American Feed Control Officials) statements on product labels. During gestation/lactation, 1,659 respondents (85%) reported feeding commercial diets, while 294 (15%) reported feeding home-prepared diets. Approximately 17% (131 of 759) of breeders who provided the AAFCO statements from their commercial diet products were feeding inadequate diets intended only for supplemental feeding or for a different life stage. Unsubstantiated marketing claims, particularly those referring to diet ingredients, were reported by many respondents as being factors that influence their feeding practices. Although 68.5% of the breeders viewed veterinarians as a trusted source of diet information, only 34.5% reported consulting them for nutrition information. Given the number of breeders feeding inadequate diets and the influence of non-refereed sources of information on their feeding decisions, a more proactive role by veterinarians in providing nutritional guidance to dog breeders may be necessary.

Etiopathogenesis of Canine GDV: A Nutritionist's Interpretation of the Evidence

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Abstract

A definitive understanding of the etiopathogenesis of canine gastric dilatation-volvulus, also known as bloat, remains elusive. While commercial dry dog foods have been theorized as a causative factor, there is little evidence that the form or composition of food has a major influence on risk. Feeding the at-risk dog multiple times per day may have some preventive effect, but prophylactic gastropexy is likely to be most effective in managing risk.

Introduction

Gastric dilatation in dogs is characterized by the onset of rapid accumulation of gas or air in the stomach. This condition is often associated with gastric volvulus, i.e., a varying degree of malposition or torsion of the stomach in the abdominal cavity. Although it is thought that dilatation generally precedes volvulus, both gastric dilatation with and without volvulus are thought to be caused by the same underlying disease process. Most reports do not attempt to differentiate the two manifestations, but rather refer to them collectively as gastric dilatation-volvulus (GDV). Regardless, the increase in intragastric pressure as a result of accumulation of gas in the stomach can lead to gastric necrosis, decreased venous return, metabolic acidosis, cardiac arrhythmias, hypovolemia, disseminated intravascular coagulation, and cardiogenic shock. A case of GDV is most often fatal without urgent and aggressive therapy, usually with surgical correction of the torsion (if it exists), but in any case with a gastropexy to prevent recurrence. Even with medical intervention, short-term mortality rates reported in more recent papers range from 10 to 16%, and may be much higher when other procedures are performed concurrently (e.g., partial gastrectomy).[1,2]

GDV occurs predominantly in giant- and large-breed dogs, with the likelihood of a purebred show dog in either category developing GDV over its lifetime to be 22 and 24%, respectively (up to 42% in Great Danes).[3] The etiopathogenesis of GDV remains elusive, but by all accounts appears to be multifactorial in origin. Both dietary and nondietary factors have been implicated as contributing to the incidence of occurrence. Because the onset of GDV in an individual animal is infrequent and unpredictable, well-controlled studies to investigate the direct effect of a hypothesized causative factor on occurrence of GDV are difficult to conduct. As a result, most reports offer either epidemiological evidence of risk factors or results of studies looking at the effect on a presumed cause of GDV (e.g., delayed gastric emptying time). Although the effect of food and/or feeding is often the subject of study, few contributions to the literature have been offered by veterinary nutritionists.

Food-Related Risk Factors

Much effort has been spent in investigation of food as a potential cause of GDV, particularly with respect to type and composition of food. Early postulation was that dry dog food, especially those largely comprised of processed soy and cereal grains, was a primary factor in the etiopathogenesis of GDV.[4-6] The theory was that unlike the diet of wild or feral carnivores, whose diet would be high in animal protein and "animal roughage" (poorly digestible parts of carcasses such as bone, cartilage, fur, feathers, etc.), the modern extruded commercial diet was unsuitable for maintenance of optimum gastric structure and function. The high levels of fermentable carbohydrates characteristic of dry dog foods served as a substrate for gastric flora (including *Clostridium perfringens*), which could be responsible for the gas formation.

A study tested this hypothesis by comparing the feeding of commercial dry dog food versus a raw "meat and bone ration" to eight Irish Setter dogs either once or three times daily (2 X 2 Latin square design) for up to approximately two years.[6] One dog died approximately six months into the study and was replaced. The raw ration, intended to mimic the diet of wild carnivores, consisted of whole dressed, roughly chopped chicken, ground horse meat, whole apple, bran, and vitamins and minerals. No effects due to diet or frequency of feeding were seen on pentagastrin-induced gastric secretion. Postprandial serum gastrin levels increased in dogs fed once daily versus three times, but no effect of diet type was observed and all values remained within the normal range. Dogs eating commercial food once daily showed greater gastric dimensions post-feeding and greater stomach weight and larger residual food volume in the stomach two hours post-feeding at the termination of the study.

The authors postulated that once daily feeding of a commercial

dry dog food could cause GDV by virtue of repeated extension and eventual enlargement of the stomach, delaying normal emptying, and in combination with the rapidly fermentable carbohydrates in the food could account for the rapid production of gas in the stomach and onset of GDV. The dog that died six months into the study was in the commercial food/once daily group and did suffer GDV on multiple occasions before death, hence adding credence to this suggested etiopathogenesis. However, due to the small number of animals (two per group) and the high prevalence of GDV in the breed, it is difficult to conclude whether there was a true effect due to diet/frequency or whether it was just coincidence.

Subsequent work has shown that neither a higher prominence of soy and grain ingredients in dry dog food nor a higher percentage of metabolizable energy from carbohydrates influenced GDV risk.[7,8] Also, other work counters the contention that cereal-based dry diets result in a delay in gastric emptying, a theorized influencing factor in GDV. A study comparing effects of a canned meat-based diet with a dry cereal-based diet (either fed as is or with added water) failed to show a significant difference between groups with respect to patterns of gastrointestinal motility or half-time for gastric emptying.[9] Another study showed that movement of particulate markers from the stomach was slower in dogs fed a fortified all-meat wet food versus a cereal-based dry food moistened with evaporated milk.[10]

An Internet survey did find consumption of dry dog food to be associated with increased risk of GDV.[11] However, that risk was lowered with supplemental fish or eggs in the diet. A case-control study found inclusion of table scraps in an otherwise dry food diet also lowered the risk compared to dry food alone, but the effect of adding canned food or moistening the dry food was not significant.[12] A study of feeding practices in Irish Setters found that feeding a single food type, although not necessarily dry food, increased the risk of GDV.[13]

While one study found that the relative predominance of either animal-sourced protein ingredients or soy and cereal ingredients did not influence GDV risk, the presence of a fat or oil (animal or vegetable origin) listed among the first four ingredients (and associated higher metabolizable energy contribution from fat) was linked to a significant increased risk of GDV.[7] The authors hypothesized that a high-fat diet could delay gastric emptying compared to high-protein or -carbohydrate diets and contribute to the etiopathogenesis.

The inclusion of citric acid in dry dog foods was implicated as a risk factor for development of GDV, particularly if the food was moistened.[14] Citric acid is often used as a component in "natural" fat preservative systems. Regardless, this finding was reported on a preliminary basis only and did not appear in the finished paper as published, so its relevance appears moot.

In addition to food composition, food volume has been reported to be a factor in risk of GDV. Dogs fed a larger volume of food per meal expressed as a proportion of body weight had a higher risk, regardless of the number of meals fed daily (although the combination of large volume and once daily feeding further increased the risk).[8]

Food particle size also has been reported to have an effect. Large pieces of meat (greater than 30 mm in size) added to dry commercial kibble, canned meat-based foods or home-prepared foods appeared to decrease the risk of GDV, while added ground or small pieces of meat did not have that effect.[15] While the authors suggest that this decrease in risk may be due to a mechanical effect of large particles, the means by which this occurs was unclear. While large particles could theoretically slow the rate of consumption, food intake time (i.e., the time needed to finish a meal completely) was measured but not found to be a contributing risk factor in this study.

Feeding-Related Risk Factors

In addition to food type, the number of daily feedings also has been implicated in the etiopathogenesis of GDV.[6] Some studies have found a positive association between feeding one meal per day with increased risk of GDV,[12,13] while another failed to make that association.[15] Similarly, rapid eating behavior has been implicated as a factor in GDV.[12,16] However, other studies found no evidence of increased risk.[13,15]

Common advice for dogs at risk for GDV is to limit activity after eating. However, one study failed to find an associated risk.[15] In fact, playing with other dogs or "running the fence" after meals was associated with a decreased risk of GDV.[11]

Another common piece of advice to decrease the odds of GDV is to feed the dog with a raised feeding bowl, reportedly to minimize aerophagia. However, even with confounding taken into account (e.g., owners of high-risk dogs are more likely to implement this feeding management advice), using a raised bowl was found to increase, not decrease, risk.[16]

Other Risk Factors Not Related to Food or Feeding

Breeds identified with a predilection for GDV include (but certainly is not limited to): Great Danes, Irish Wolfhounds, Bloodhounds, Irish Setters, Akitas, and Standard Poodles.[3] In a report from New Zealand, the odds of a case of GDV presented to a veterinary office being a Huntaway (a large working farm dog) was 19 times higher than the odds of a control (trauma case) being a Huntaway.[7] "Deep-chested" dogs, i.e., those with a higher thoracic depth-to-width ratio, and those thin for their respective breed standard may be at greater risk.[3,16] There also may be a familial component, as dogs with a first-degree relative with a history of GDV (including a sire or dam, but especially a sibling or an offspring) are at increased risk.[16]

Age appears to be a factor, with odds of occurrence increasing over time.[11,13,15,16] Risk of GDV has been reported to be increased in males in one study[12] but intact females in another,[11] while

others reported no influence of gender or neuter status.[15,18]

Stress caused by kenneling, travel or other activities has been implicated with increased risk of GDV.[13,19] A "happy" personality as opposed to fearfulness or anxiety appears to decrease risk.[12]

The minimum and maximum daily atmospheric pressure on the day of a GDV event and the maximum pressure the day before the event were positively associated with the probability of an incident of GDV.[20] An increased incidence of GDV has been reported to occur during the spring in pet dogs,[11] during the summer in working farm dogs[17] and during the winter in military dogs.[21]

Conclusions

At this time, there appears to be a paucity of sound guidance for the veterinary nutritionist to offer in terms of management of risk of GDV in susceptible dogs via dietary manipulation. While early theories incriminated dry dog food containing highly fermentable carbohydrates as an etiologic factor, only modest evidence has surfaced to corroborate the premise that the form, ingredients or nutritional composition of the food have a significant impact on risk. In other words, there is little basis upon which to advise against the feeding of commercial dry foods containing grains or soy as a means to mitigate the risk of GDV. On the other hand, several reports suggest that adding "something" to a dry diet (e.g., fish, eggs, table scraps, large chunks of meat) could lower the risk. The mechanism by which use of these dietary additions would have an impact on risk is not understood. However, it is a relatively easy practice to implement, provided the added foods are not used in excess as to unbalance the total ration. The addition of a complete and balanced canned food to the dry diet would avoid the possibility of unbalancing the diet, but a lowering of risk by this practice has not been reported.

An ingredient/nutrient in dry dog foods that has been identified as a potential risk factor for GDV is fat. However, recommendations to avoid products that list fat or oil as a major ingredient and/or where fat is the major energy contributor appear contrary to other advice. For example, it also has been reported that lowering the volume of food fed per meal may have protective effects. The addition of fats to a dry food facilitates increased energy density of the food compared to protein or carbohydrates, so higher fat products generally allow for lower volumes to be fed. Also, addition of table scraps or other foods to a dry dog food often increases total fat content of the diet. Finally, a dog that is thin for its breed standard appears to be at greater risk for GDV. This condition normally would be addressed by increasing caloric intake, usually achieved by increasing dietary fat content.

If a lower energy density food is offered, the volume of food per meal also can be effectively reduced by increasing the number of meals per day. Although not all studies reported increased risk with once-a-day feeding, it is a repeated finding, so multiple daily feedings appear to be a prudent practice that is easy to implement. Reports on the effect of food intake time on risk of GDV are mixed. Food bowls designed to slow the rate of eating are available and would not appear contraindicated. On the other hand, feeding from raised bowls may actually increase risk.

Surgical rather than nutritional intervention may be key to effective prevention of GDV. In dogs that suffered an episode of GDV, gastropexy decreased the rate of recurrence from 54.5 to 4.3% and increased median survival time from 188 to 547 days.[22] Endoscopically assisted gastropexy is advocated as a safe and reliable means of prophylaxis in dogs at high risk for GDV.[23]

References

1. Beck JJ, Staatz AJ, Pelsue DH, et al. Risk factors associated with short-term outcome and development of perioperative complications in dogs undergoing surgery because of gastric dilatation-volvulus: 166 cases (1992-2003). *J Am Vet Med Assoc*. 2006;229:1934-1939.

2. Mackenzie G, Barnhart M, Kennedy S, et al. A retrospective study of factors influencing survival following surgery for gastric dilatation-volvulus syndrome in 306 dogs. *J Am An Hosp Assoc*. 2010;46:97-102.

3. Glickman LT, Glickman NW, Schellenberg DB, et al. Incidence of and breed-related risk factors for gastric dilatation-volvulus in dogs. *J Am Vet Med Assoc*. 2000;16:40-45.

4. Van Kruiningen HJ, Gregoire K, Meuten DJ. Acute gastric dilatation: A review of comparative aspects, by species, and a study in dogs and monkeys. *J Am An Hosp Assoc*. 1974;10:294-324.

5. Rogolsky B, Van Kruiningen HJ. Short-chain fatty acids and bacterial fermentation in the normal canine stomach and in acute gastric dilatation. *J Am An Hosp Assoc*. 1978;14:504-515.

6. Van Kruiningen HJ, Wojan LD, Stake PE, et al. The influence of diet and feeding frequency on gastric function in the dog. *J Am An Hosp Assoc*. 1987;23:145-153.

7. Raghavan M, Glickman NW, Glickman LT. The effect of ingredients in dry dog foods on the risk of gastric dilatation-volvulus in dogs. *J Am An Hosp Assoc*. 2006;42:28-36.

8. Raghavan M, Glickman N, McCabe G, et al. Diet-related risk factors for gastric dilatation-volvulus in dogs of high-risk breeds. *J Am An Hosp Assoc*. 2004;40:192-203.

9. Burrows CF, Bright RM, Spencer CP. Influence of dietary composition on gastric emptying and motility in dogs: Potential involvement in acute gastric dilatation. *Am J Vet Res*. 1985;46: 2609-2612.

10. Banta CA, Clemems ET, Krinsky MM, et al. Sites of organic acid production and patterns of digesta movement in the gastrointestinal tract of dogs. *J Nutr*. 1979;109:1592-1600.

11. Pipan M, Brown DC, Battalion CL, et al. An Internet-based survey of risk factors for surgical gastric dilatation-volvulus in dogs. *J Am Vet Med Assoc*. 2012;240:1456-1462.

12. Glickman LT, Glickman NW, Schellenberg DB, et al. Multiple risk factors for the gastric dilatation-volvulus syndrome in dogs: A practitioner/owner case-control study. *J Am An Hosp Assoc*. 1997;33:197-204.

13. Elwood CM. Risk factors for gastric dilatation in Irish Setter dogs. *J Sm Anim Pact*. 1998;39:185-190.

14. http://www.moonstruckmeadows.com/Bloat%20(GDV)%20Study.htm. (accessed Jan. 16, 2013)

15. Theyse LFH, van de Brom WE, van Sluijs FJ. Small size of food particles and age as risk factors for gastric dilatation volvulus in Great Danes. *Vet Rec*. 1998;143:48-50.

16. Glickman LT, Glickman NW, Schellenberg DB, et al. Nondietary risk factors for gastric dilatation-volvulus in large and giant breed dogs. *J Am Vet Med Assoc*. 2000;217:1492-1499.

17. Hendricks MM, Hill KE, Cogger N, et al. A retrospective study of gastric dilatation and gastric dilatation and volvulus in working farm dogs in New Zealand. *NZ Vet J*. 2012;60:165-170.

18. Glickman LT, Glickman NW, Perez CM, et al. Analysis of risk factors for gastric dilatation and dilatation-volvulus in dogs. *J Am Vet Med Assoc*. 1994;204:1465-1471.

19. Burrows CF, Ignaszewski LA. Canine gastric dilatation-volvulus. *J Sm An Pract*. 1990;31:495-501.

20. Levine M, Moore GE. A time series model of the occurrence of gastric dilatation-volvulus in a population of dogs. *BMC Vet Res*. 2009;5:12.

21. Herbold JB, Moore GE, Gosh TL, et al. Relationship between incidence of gastric dilatation-volvulus and biometerorologic event in a population of military working dogs. *Am J Vet Res*. 2002;63:47-52.

22. Glickman LT, Lantz GC, Schellenberg DB, et al. A prospective study of survival and recurrence following the acute gastric dilatation-volvulus syndrome in 136 dogs. *J Am An Hosp Assoc*. 1998;34:253-259.

23. Dujowich M, Keller ME, Reimer SB. Evaluation of short- and long-term complications after endoscopically assisted gastropexy in dogs. *J Am Vet Med Assoc*. 2010;236:177-182.

Preventing Disease Through Nutrition: Reducing Nutrients to Prevent Disease?

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Introduction

In 2006, the National Research Council (NRC) of the National Academy of Sciences published an update on the nutrient requirements for dogs and cats.[1] The document makes recommendations as to how much of each nutrient should be included in the diet to maintain health in normal dogs and cats and reviews studies in which the recommendations are based. Thus, the NRC recommendations remain the best available resource for published evidence as to how much of each nutrient should be included in the diet to prevent disease.

Glossary of Abbreviations

AAFCO: Association of American Feed Control Officials
AI: Adequate Intake
CKD: Chronic Kidney Disease
FEDIAF: European Pet Food Industry Federation
MCT: Mast Cell Tumor
MR: Minimum Requirement
NRC: National Research Council
RA: Recommended Allowance
SUL: Safe Upper Limit

The recommendations are based on a traditional paradigm in which performance, such as growth or maintenance of health, is compromised when there is an inadequate amount of an essential nutrient in the diet but improves as the amount of nutrient in the diet increases until it reaches a plateau. Performance remains high and animals remain healthy as nutrient density increases until the nutrient starts having a negative or toxic effect whereupon performance or health declines. Thus, there is a minimum and maximum amount or concentration of each nutrient that should be included in the diet to maintain health. It is evident from this paradigm that inclusion of too little or too much of a nutrient can impair health, but animals are capable of adapting to varying amounts of most nutrients in their diet so the difference between minimum and maximum requirements is quite wide for many nutrients.

Some authors and companies have suggested that this difference between minimum and maximum may be smaller than is suggested by short-term feeding studies, i.e., that there may be an "optimum" concentration in the diet that will prevent disease when food is fed for long periods of time. The NRC recommendations allow for the fact that different measures of "performance" may give different minimum and maximum recommendations, and thus account for any role that diet may play in the prevention of disease but are limited in scope by a lack of long-term feeding studies. Long-term prospective feeding studies are prohibitively expensive to perform because they require a large number of animals to determine a statistical difference between treatments unless it is possible to reliably induce some abnormality. Thus, there currently is no information to support a narrow "optimum" range that is different from that suggested by the NRC recommendations.

Nevertheless, the NRC recommendations and published studies do have limitations. Many minimum and maximum nutrient amounts have not been determined with accuracy. Making pet food is a complex process, animals and ingredients are not uniform, and many factors such as nutrient bioavailability, animal body size and condition, life stage, activity, and the presence of disease can influence both maximum and minimum requirements. Pet foods for a varied population of animals must include wide safety margins, therefore, to accommodate differences among ingredients and animals.[1]

The NRC guidelines also are expensive to purchase and are not easy to interpret. This has allowed many unfounded interpretations that lack logic. In particular, the distinction between preventing disease with nutrition and therapeutic nutritional intervention has become blurred. A myth has arisen that changes in diet that are important in the management of a disease may also prevent disease occurrence. This assumption does not bear scrutiny, and the NRC recommendations specifically exclude discussion of the nutritional management of dogs and cats with disease because prevention and treatment are not necessarily related. Changes in the diet that are important in diabetes mellitus management may not be the same ones that affect the development of diabetes mellitus. Limiting intake of phosphorus slows the progression of chronic kidney disease (CKD) but does not necessarily prevent the onset of CKD.

Senior diets provide a particular case in point. The NRC guidelines provide recommendations for growth, adult maintenance, pregnancy and lactation, but there are no recommendations for older patients because there are almost no studies of the effect of old age on the requirement for any nutrient except energy. Some senior diets contain reduced amounts of some nutrients because some diseases, such as CKD, are more common in older patients.

It does not follow, however, that *all* older patients have kidney disease or that *all* older patients benefit from nutrient restriction. On the contrary, the NRC guidelines note that animals with lower than average energy needs may need to consume more nutrient-dense foods, i.e., foods with greater nutrient-to-calorie ratios, in order to provide the essential nutrients they require. As energy requirements decline with age in dogs, it can be inferred that older dogs may require a more nutrient-dense diet. Similarly, a breed-specific diet designed to treat a disease that is common in a particular breed may not benefit members of the breed that do not suffer from that particular disease.

This review examines the scientific foundation for claims that some nutrients should be included in the diet either above or below the minimum and maximum amounts recommended by the NRC for normal dogs and cats to reduce the risk of disease. The review relies heavily on the material reviewed in the 2006 guidelines, and the reader is referred to that text for the references that support assertions derived from that publication. Additional references are provided for studies that have been published since that report.

Minimum NRC Recommendations and Their Limitations

The 2006 NRC guidelines define a minimum requirement (MR) as the minimal concentration or amount of a *bioavailable* nutrient that will support a defined physiological state. A safety factor is added to the MR to give a recommended allowance (RA) for foods formulated from normal pet food ingredients. The safety factor is designed to allow for normal variation in nutrient bioavailability in typical pet food ingredients. Gradually increasing amounts of nutrient have to be fed to dogs and cats while measuring performance to accurately establish an MR. When an MR cannot be established in this fashion but a pet food containing a nutrient at a low concentration has been fed without resulting in signs of deficiency, then that concentration was used to establish an adequate intake (AI). This AI is defined as a concentration or amount of a nutrient that had been demonstrated to support a defined physiological state. The RA was then established based on the AI without any additional safety factor because the AI was established using pet food ingredients.

Regulators and the general public often do not appreciate that these are guidelines and thus are not firm values and that animals consuming less than the RA may remain healthy and animals consuming more than the RA may develop deficiency problems. The safety factor is an educated guess that makes assumptions about normal pet food ingredients. The MR also represents the mean for a population of animals after a short-term feeding trial. Individual animals may have a lower or higher MR than that reported. It is quite possible, therefore, that a diet containing lower concentrations than an RA established from an MR may support a healthy animal. Similarly, a nutrient that is less available than normal would need to be included at a higher concentration than the RA to maintain health.

It is often not appreciated that individual energy requirements need to be taken into account when interpreting the NRC guidelines. Most requirements were established with laboratory animals consuming diets of average energy density so the NRC guidelines assume that the diet contains 4 Kcal/g and is being consumed by dogs requiring 130 Kcal/kg body weight$^{0.75}$ daily or cats requiring 100 Kcal/kg body weight$^{0.67}$ daily for maintenance. Nevertheless, energy requirements vary widely with activity and environmental conditions and also among individuals maintained under similar conditions. How nutrient requirements vary as energy requirements vary is mostly unknown: The amount of nutrient required for a given body weight may increase, may stay the same, or may decrease as energy needs increase or decrease among individuals and under different physiological conditions. In the face of this uncertainty, the guidelines recommend taking the conservative option that nutrient requirements relative to body weight stay the same when energy requirements differ from expected norms. Thus, to ensure adequate intake of nutrients, the nutrient density in the diet relative to energy must increase when a pet dog or cat are able to maintain body condition while consuming less than average amounts of energy. Similarly, the nutrient density relative to energy of potentially toxic nutrients should decrease in the diet of very active dogs that need more than average amounts of energy.

In general, therefore, it is wise when formulating a diet for a diverse population of dogs or cats with a wide range of energy requirements to include nutrients at concentrations well above the minimum and well below the maximum suggested by the NRC, i.e., that the concentrations should be maintained within a narrower range than that suggested by the NRC recommendations. This allows for variations in energy requirements as well as any deviations in formulation or the quality of ingredients. If a diet is being fed that closely approximates the RA, then the amount of food being consumed becomes an important consideration. Under such circumstances, close monitoring is recommended and a nutritional specialist may need to be consulted who can evaluate an individual's needs when deciding what to feed.

Safe Upper Limits

For some nutrients, the 2006 NRC guidelines also report a safe upper limit (SUL), which is defined as the maximal concentration or amount of a nutrient that has not been associated with adverse effects. Higher amounts may or may not be safe, but data are lacking. The SULs give some indication of the maximum amount that may be included in a diet safely, but some SULs are known quite precisely from toxicological studies, whereas others are known less precisely. These SULs have the same limitations as the MRs and RAs, so it may be possible to feed higher amounts than the SUL with impunity. This was shown recently for vitamin A.

The 2006 NRC set the SUL for retinol at 12,500 IU/Mcal because 12,500 IU/Mcal has been fed to dogs without causing problems, whereas 550,000 IU/Mcal has been shown to cause toxicity. At the time, it was not known whether a higher concentration than 12,500 IU/Mcal might be safe but the SUL did not preclude that possibility. A recent study reported no abnormal development when growing puppies were fed 100,000 IU/Mcal,[2] so the SUL can now be increased to 100,000 IU/Mcal. Nevertheless, nutrient concentrations close to the SUL may need to be reduced if animals are consuming more food than such studies report or the SULs assume.

The tables in the NRC guidelines give relatively few SULs because there have been few reports of adverse effects from feeding large quantities of most nutrients. Thus, SULs are provided in the tables only for some amino acids, fat, calcium, sodium and chloride, and vitamins A and D. There also are some recommendations hidden in the text that were not included in the tables. For example, SULs for selected carbohydrates can be found in the carbohydrate and fiber chapter.

Relative proportions of nutrients also are important. Thus, the relative proportions of protein, cations and anions affect urine pH which in turn can affect the SUL of various other minerals. A myth persists that magnesium (Mg) must be limited to prevent struvite stones forming in cat urine, but no SUL is provided for Mg in cats because increasing Mg in the diet is much less important when urine pH is low. Similarly, a myth persists that magnesium, phosphorus and protein need to be restricted in dogs with struvite stones, yet almost all struvite stones in dogs result from urinary tract infections. Thus, no SUL is provided for Mg in dogs because treatment of infection rather than a change of diet is needed to prevent struvite urolith recurrence. Furthermore, the NRC guidelines do not give an SUL for phosphorus because the effect of low calcium to phosphorus ratios on bone density appears to result from calcium deficiency and not phosphorus excess. Nevertheless, experimental and case-control studies clearly suggest that the relative proportions of moisture, protein, sodium, potassium, calcium, phosphorus, and magnesium and urine acidifying potential of the diet are associated with altered risk for struvite and calcium oxalate urolith formation in dogs and cats.[3-6] Limiting purine intake while maintaining a more alkaline urine also is important in preventing stone formation in Dalmatians that are prone to developing ammonium urate uroliths.[7,8] Unfortunately, most of these studies have not reported food intake or detailed nutrient analyses of the diets, so it is difficult to ascertain how variation in energy requirements may play a role in any of these situations.

Should Fat-Soluble Vitamins Be Increased in the Diet to Prevent Disease?

Several studies in humans and rodents have sought to determine whether increased intake of fat-soluble vitamins may benefit the prevention or treatment of disease. It is important to remember, however, that most of these studies are concerned with deficiency versus adequacy because many people in developing countries and some people in developed countries consume diets that are deficient in some nutrients. Pets in developed countries mostly consume commercial diets that are designed to be complete and balanced, so the issue of concern is not deficiency versus adequacy but whether additional vitamins above the minimum recommended by a regulating body (NRC, AAFCO, FEDIAF) would be beneficial. The effect of supplementation to overcome deficiency is only relevant when pets are fed home-prepared recipes that are not complete or balanced or commercial foods that do not conform to standard recommendations.

Thus, for example, large parenteral doses of vitamin A have reduced the prevalence of diarrhea and respiratory infections and reduced the morbidity and mortality of measles in malnourished children in developing countries,[9-11] but there is no evidence that supplementation of vitamin A above that recommended by the NRC prevents disease in pets in developed countries fed commercial diets. On the contrary, many commercial diets contain liver and large amounts of vitamin A.

Of more immediate concern is whether the RA for vitamin D should be increased. The 2006 NRC RA for vitamin D is based on the central role vitamin D plays in calcium and phosphorus homeostasis, but it is now recognized that calcitriol plays an additional unrelated noncalcemic role in cell proliferation and differentiation, particularly in immunity and the induction of apoptosis in cancer cells *in vitro*.[12-14] A cross-sectional study of Labrador Retrievers found that mean 25 (OH) vitamin D3 concentrations were 13% lower in dogs with mast cell tumors (MCT) than in dogs without MCT suggesting that low blood concentrations of vitamin D might be a risk factor for MCT development. Despite this, the calculated dietary vitamin D intake was not statistically different between the two groups.[15] Furthermore, high-dose oral calcitriol induced remission in four of 10 dogs (one complete remission, three partial remissions), but the majority experienced toxicity, necessitating discontinuation of the trial.[14] At this time, therefore, there is insufficient data to recommend more vitamin D than is recommended by the 2006 NRC.

Some studies have shown changes in immune function and oxidation status in dogs and cats fed increased amounts of vitamin A, carotenoids, vitamins E and C, and various other antioxidants in various combinations, but none to date have shown that supplementation above NRC recommendations reduce the prevalence of disease.[16-21] Changes in immune cell proliferation or increasing circulating antibodies in response to stimulation do not necessarily enhance the ability of an animal to resist disease. Similarly, the benefits of increasing antioxidant concentrations in the blood are equivocal because preventing oxidation may prevent stimulation of protective mechanisms against oxidation during training.[1] In racing Greyhounds, for example, high doses

(1 g daily) of vitamin C appeared to reduce performance.[22]

Old Beagles supplemented with a cocktail of antioxidants (1050 ppm dl-alpha tocopheryl acetate, 260 ppm L-carnitine, 128 ppm dl-alpha lipoic acid, 80 ppm vitamin C and 1% each of spinach flakes, tomato pomace, grape pomace, carnitine, and citrus pulp)[23,24] or alpha-lipoic acid combined with L-carnitine showed improvements in learning and retention of learned behavior.[25] This improvement was increased in animals receiving behavioral enrichment.[26] Middle-aged cats fed increased amounts of vitamins E and C and fish oil and slightly increased amounts of B vitamins and arginine also showed improved cognitive testing.[27] Which parts and what doses of these cocktails are required for these effects and whether pet cats show a similar effect has yet to be determined.

Should Protein, Phosphorus and Sodium Be Restricted to Prevent the Development of Kidney Disease?

To quote from the NRC guidelines: "Satisfactory maintenance and maximal growth and reproduction of dogs and cats can be achieved on a wide variety of concentrations of amino acids in purified diets, using either free amino acids, amino acids in purified proteins, or proteins from common feed ingredients incorporated into dry expanded or canned diets; that is no upper limit is known." A SUL for lysine is suggested for dogs to be >5 g/Mcal because an antagonism between lysine and arginine has been reported when purified lysine was added to change the ratio of lysine to arginine. No SUL is given for other amino acids for dogs because no adverse effects have been reported at high doses. SULs are suggested for many amino acids in growing kittens, but most are imprecise and are termed as being more than a certain concentration. In practice, it would be almost impossible to exceed the SUL for these amino acids using normal mixed-protein diets and the ratio of amino acids would not change with increasing protein in the diet.

Racing Greyhounds fed a mixed-protein diet ran more slowly when the protein content of the diet increased from 63 to 96 g/Mcal, but the protein was substituted for carbohydrate so it is possible that this effect could have been the result of a decrease in available carbohydrate.[28] On the other hand, racing sled dogs are fed a very high-protein, high-fat diet without showing any tendency to develop kidney disease, despite consuming enough food to provide up to 1050 Kcal/kg$^{0.75}$ daily.[1] For example, sled dogs consuming about 440 Kcal/kg$^{0.75}$ daily have been fed a mixed-protein diet containing up to 102 g protein/Mcal for several months without showing important changes in blood parameters.[29] Overweight dogs and cats also have preserved more lean body mass during weight loss and have not shown untoward effects while being fed high-protein diets over weeks to months.[30]

Prospective controlled clinical trials have shown that median survival is prolonged more than twofold in dogs and cats with CKD when they are fed commercial kidney diets with less protein and phosphorus, moderate amounts of sodium and sometimes containing fish oil compared to when they are fed normal maintenance diets.[31,32] In an experimental model of CKD in dogs, phosphorus restriction and fish oil addition without protein restriction have been shown to slow progression of the disease.[33-35] All these feeding trials have started with animals with a serum creatinine of more than 2 mg/dL, i.e., equivalent to the International Renal Interest Society's stage 3 CKD in dogs and high stage 2 to stage 3 CKD in cats. Furthermore, a case-control study found that pet cats were at increased risk of CKD when fed *ad libitum* or consuming more ash and were at decreased risk when fed increased dietary fiber, magnesium, protein, and sodium.[36] Thus, there currently is no evidence that normal cats or dogs or even those dogs and cats with unrecognized early non-azotemic non-proteinuric CKD would benefit from protein or phosphorus restriction.

Unlike foods for human consumption, which often contain more than 2 g Na/Mcal, most commercial pet foods contain moderate amounts of sodium (0.5-2 g/Mcal). Nevertheless, therapeutic diets containing higher concentrations of sodium (2-3 g/Mcal) are being fed to cats to promote diuresis, lower the concentration of urine solutes and thus reduce the risk of urolith formation. The long-term safety of this approach has yet to be adequately evaluated, however, and some have questioned whether animals, especially those with chronic kidney disease, can excrete the increased sodium. If this is the case, then hypertension may ensue and promote the development or progression of CKD. Nevertheless, healthy sedentary dogs are capable of excreting enormous amounts of sodium (approximately 66 g of sodium/Mcal).[37] Even sled dogs racing in the cold consuming huge amounts of food to support their energy requirements would not exceed this amount if the diet contained less than 8 g/Mcal. Furthermore, in an experimental model of CKD in dogs, restricting sodium to about 0.5 g/Mcal was associated with moderate hypertension compared to feeding a high-sodium diet, containing about 3 g/Mcal.[38] In a cat model of CKD and in cats with naturally occurring CKD, arterial blood pressure was unaffected by markedly increasing dietary sodium intake.[39,40] Nevertheless, all these studies were short term, so the safety of this approach has yet to be determined. Currently, it would seem that sodium restriction is not necessary in normal animals and is probably not necessary in animals with IRIS stage 2 or above CKD because animals will have dilute urine.

Maintenance of Health by Prevention of Obesity

Maintaining a lean body condition remains the best method of adjusting the diet to maintain health. Obesity has been associated with orthopedic, endocrine, cardiac, respiratory, neoplastic, urinary, reproductive, and dermatological disease and reduced resistance to infection in dogs and cats.[30,41] Associations do not necessarily reflect causality, but obesity can cause insulin resistance in both dogs and cats and can result in noninsulin- dependent

diabetes mellitus in cats.[42] Blood pressure also increases, and lipoprotein profiles change slightly in overfed dogs.[43-45] A prospective randomized controlled trial involving Labrador Retrievers clearly demonstrated that restricting food intake by 25% so that dogs maintained a lean body condition increased their life span by almost two years compared to dogs that ate more and were modestly overweight. Lean dogs also developed osteoarthritis later and required medications for pain three years later than overweight dogs.

Nevertheless, changes in blood pressure and lipoprotein concentrations are relatively modest in dogs and cats. Most of the lipoproteins in fasted dogs and cats are HDL, whereas those in humans are mostly LDL, and the prevalence of atherosclerosis and cardiac infarction in dogs and cats is very low. Thus, weight gain may not cause dogs and cats to suffer the same consequences as humans with metabolic syndrome. Labrador Retrievers also are prone to arthritis, and it remains to be determined whether food restriction has such dramatic effects on life span in smaller dogs and other breeds that are less prone to degenerative joint disease.

Conclusion

The NRC recommendations remain the best published source of information concerning how much of each nutrient should be included in a diet to maintain health, but it is important to take into account factors such as energy requirements, ingredient quality, and food processing when deciding how much of any nutrient should be included in the diet. Changes in dietary composition that help moderate disease processes do not necessarily prevent disease. Thus, it is better to include moderate amounts of nutrients in the diet and it is probably unwise to reduce nutrient concentrations in the hope of preventing disease. On the other hand, restricting energy intake to maintain a lean body condition has been shown to help maintain health and prolong life.

References

1. National Research Council Subcommittee on Dog and Cat Nutrition. Nutrient requirements of dogs and cats. National Academy Press, Washington, D.C. 2006.

2. Morris PJ, Salt C, Raila J, et al. Safety evaluation of vitamin A in growing dogs. *Br J Nutr*. 2012;108:1800-1809.

3. Lekcharoensuk C, Osborne CA, Lulich JP, et al. Associations between dry dietary factors and canine calcium oxalate uroliths. *Am J Vet Res*. 2002;63:330-337.

4. Lekcharoensuk C, Osborne CA, Lulich JP, et al. Associations between dietary factors in canned food and formation of calcium oxalate uroliths in dogs. *Am J Vet Res*. 2002;63:163-169.

5. Lekcharoensuk C, Osborne CA, Lulich JP, et al. Association between dietary factors and calcium oxalate and magnesium ammonium phosphate urolithiasis in cats. *J Am Vet Med Assoc*. 2001;219:1228-1237.

6. Stevenson AE, Blackburn JM, Markwell PJ, et al. Nutrient intake and urine composition in calcium oxalate stone-forming dogs: comparison with healthy dogs and impact of dietary modification. *Vet Ther*. 2004;5:218-231.

7. Bijster S, Nickel RF, Beynen AC. Comparison of the efficacy of two anti-uric acid diets in Dalmatian dogs. *Acta Vet Hung*. 2001;49:295-300.

8. Bartges JW, Osborne CA, Lulich JP, et al. Canine urate urolithiasis. Etiopathogenesis, diagnosis, and management. *Vet Clin North Am Small Anim Pract*. 1999;29(xxii-xiii):161-191.

9. Chowdhury S, Kumar R, Ganguly NK, et al. Effect of vitamin A supplementation on childhood morbidity and mortality. *Ind J Med Sci*. 2002;56:259-264.

10. Zaman K, Baqui AH, Yunus M, et al. Malnutrition, cell-mediated immune deficiency and acute upper respiratory infections in rural Bangladeshi children. *Acta Paediatr*. 1997;86:923-927.

11. Huiming Y, Chaomin W, Meng M. Vitamin A for treating measles in children. *Cochrane Database Syst Rev*. 2005:CD001479.

12. Artaza JN, Sirad F, Ferrini MG, et al. 1,25(OH)2vitamin D3 inhibits cell proliferation by promoting cell cycle arrest without inducing apoptosis and modifies cell morphology of mesenchymal multipotent cells. *J Steroid Biochem Mol Biol*. 2010;119:73-83.

13. Schauber J, Dorschner RA, Coda AB, et al. Injury enhances TLR2 function and antimicrobial peptide expression through a vitamin D-dependent mechanism. *J Clin Invest*. 2007;117:803-811.

14. Malone EK, Rassnick KM, Wakshlag JJ, et al. Calcitriol (1,25-dihydroxycholecalciferol) enhances mast cell tumour chemotherapy and receptor tyrosine kinase inhibitor activity *in vitro* and has single-agent activity against spontaneously occurring canine mast cell tumours. *Vet Comp Oncol*. 2010;8:209-220.

15. Wakshlag JJ, Rassnick KM, Malone EK, et al. Cross-sectional study to investigate the association between vitamin D status and cutaneous mast cell tumours in Labrador retrievers. *Br J Nutr*. 2011;106(Suppl 1):S60-S63.

16. Hall JA, Tooley KA, Gradin JL, et al. Effects of dietary n-6 and n-3 fatty acids and vitamin E on the immune response of healthy geriatric dogs. *Am J Vet Res*. 2003;64:762-772.

17. Heaton PR, Reed CF, Mann SJ, et al. Role of dietary antioxidants to protect against DNA damage in adult dogs. *J Nutr*. 2002;132:1720S-1724S.

18. Kim HW, Chew BP, Wong TS, et al. Dietary lutein stimulates immune response in the canine. *Vet Immunol Immunopathol*. 2000;74:315-327.

19. Kim HW, Chew BP, Wong TS, et al. Modulation of humoral and cell-mediated immune responses by dietary lutein in cats. *Vet Immunol Immunopathol*. 2000;73:331-341.

20. Chew BP, Park JS, Wong TS, et al. Dietary beta-carotene stimulates cell-mediated and humoral immune response in dogs. *J Nutr*. 2000;130:1910-1913.

21. Dunlap KL, Reynolds AJ, Duffy LK. Total antioxidant power in sled dogs supplemented with blueberries and the comparison of blood parameters associated with exercise. C*omp Biochem Physiol A MolIntegr Physiol*. 2006;143:429-434.

22. Marshall RJ, Scott KC, Hill RC, et al. Supplemental vitamin C appears to slow racing Greyhounds. *J Nutr*. 2002;132:1616S-1621S.

23. Cotman CW, Head E, Muggenburg BA, et al. Brain aging in the canine: a diet enriched in antioxidants reduces cognitive dysfunction. *Neurobiol of Aging*. 2002;23:809-818.

24. Milgram NW, Zicker SC, Head E, et al. Dietary enrichment counteracts age-associated cognitive dysfunction in canines. *Neurobiol of Aging*. 2002;23:737-745.

25. Milgram NW, Araujo JA, Hagen TM, et al. Acetyl-L-carnitine and alpha-lipoic acid supplementation of aged Beagle dogs improves learning in two landmark discrimination tests. *FASEB J*. 2007;21:3756-3762.

26. Milgram NW, Head E, Zicker SC, et al. Learning ability in aged Beagle dogs is preserved by behavioral enrichment and dietary fortification: a two-year longitudinal study. *Neurobiol of Aging*. 2005;26:77-90.

27. Pan Y, Araujo JA, Burrows J, et al. Cognitive enhancement in middle-aged and old cats with dietary supplementation with a nutrient blend containing fish oil, B vitamins, antioxidants and arginine. *Br J Nutr*. 2012:1-10.

28. Hill RC, Lewis DD, Scott KC, et al. Increased dietary protein slows racing Greyhounds. *J Vet Int Med*. 1998;12:242.

29. Kronfeld DS, Hammel EP, Ramberg CF, et al. Hematological and metabolic responses to training in racing sled dogs fed diets containing medium, low, or zero carbohydrate. *Am J Clin Nutr*. 1977;30:419-430.

30. Laflamme DP. Companion Animals Symposium: Obesity in dogs and cats. What is wrong with being fat? *J An Sci*. 2012; 90:1653-1662.

31. Jacob F, Polzin DJ, Osborne CA, et al. Clinical evaluation of dietary modification for treatment of spontaneous chronic renal failure in dogs. *J Am Vet Med Assoc*. 2002;220:1163-1170.

32. Elliott J, Rawlings JM, Markwell PJ, et al. Survival of cats with naturally occurring chronic renal failure: effect of dietary management. *J Sm An Pract*. 2000;41:235-242.

33. Brown SA, Crowell WA, Barsanti JA, et al. Beneficial effects of dietary mineral restriction in dogs with marked reduction of functional renal mass. *J Am Soc Nephrol*. 1991;1:1169-1179.

34. Brown SA, Brown CA, Crowell WA, et al. Beneficial effects of chronic administration of dietary omega-3 polyunsaturated fatty acids in dogs with renal insufficiency. *J Lab Clin Med*. 1998;131:447-455.

35. Finco DR, Brown SA, Crowell WA, et al. Effects of aging and dietary protein intake on uninephrectomized geriatric dogs. *Am J Vet Res*. 1994;55:1282-1290.

36 Hughes KL, Slater MR, Geller S, et al. Diet and lifestyle variables as risk factors for chronic renal failure in pet cats. *Prev Vet Med*. 2002;55:1-15.

37. Ladd M, Raisz LC. Response of the normal dog to dietary sodium chloride. *Am J Physiol*. 1949;159:149-152.

38. Greco DS, Lees GE, Dzendzel G, et al. Effects of dietary sodium intake on blood pressure measurements in partially nephrectomized dogs. *Am J Vet Res*. 1994;55:160-165.

39. Buranakarl C, Mathur S, Brown SA. Effects of dietary sodium chloride intake on renal function and blood pressure in cats with normal and reduced renal function. *Am J Vet Res*. 2004;65:620-627.

40. Kirk CA, Jewell DE, Lowry SR. Effects of sodium chloride on selected parameters in cats. *Vet Ther*. 2006;7:333-346.

41. German AJ. The growing problem of obesity in dogs and cats. *J Nutr* .2006;136:1940S-1946S.

42. Hoenig M. Comparative aspects of diabetes mellitus in dogs and cats. *Mol and Cell Endocrinol*. 2002;197:221-229.

43. Bodey AR, Michell AR. Epidemiological study of blood pressure in domestic dogs. *J Sm An Pract*. 1996;37:116-125.

44. Briand F, Bailhache E, Andre A, et al. The hyperenergetic-fed obese dog, a model of disturbance of apolipoprotein B-100 metabolism associated with insulin resistance: kinetic study using stable isotopes. *Metabolism*. 2008;57:966-972.

45. Tvarijonaviciute A, Ceron JJ, Holden SL, et al. Obesity-related metabolic dysfunction in dogs: a comparison with human metabolic syndrome. *BMC Vet Res*. 2012;8:147.

Communicating with Clients to Teach, Reinforce or Change Pet Beliefs

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Abstract

Gaining insights into pet owner beliefs about issues and procedures in your practice can aid in behavioral compliance. Understanding beliefs will help inform whether you need to teach, reinforce or change client beliefs, attitudes and behaviors. Two theoretical frameworks, the Health Belief Model (HBM) and the Transtheoretical Model of Change (TTM), are offered to help you in this process. Each framework is contextualized within the pet owner-provider relationship. Understanding client beliefs and attitudes about an issue or procedure will help you to be a more strategic and effective advocate for pet health.

Glossary of Abbreviations

HBM: Health Belief Model
TTM: Transtheoretical Model of Change

Introduction

This paper will teach you about persuading and communicating with clients about their pet beliefs, attitudes and behaviors. You will learn why it is important to assess whether clients do not know much about an issue or whether they hold correct or incorrect beliefs about the topic. After you assess client perceptions, you can teach new facts to those who do not know much about the topic to reinforce correct beliefs or try to change incorrect beliefs as ultimately this affects attitudes and behavior.

First, persuasion and the basic building blocks of persuasion, beliefs and attitudes, are defined. The relationship of beliefs and attitudes to behavior is then explored. Although compliance with your suggestions may be your primary goal with your clients, understanding that lasting change depends on changing beliefs and attitudes is important to the long-term health of the pets and your continuing relationship with your clients. Communication skills training can enhance your practice.[1]

Persuasion

Persuasion has been generally defined as an attempt to shape, change or reinforce a target's beliefs, attitudes or behaviors,[2] or as an active, noncoercive attempt to change another's beliefs, attitudes and behaviors.[3] When you meet with clients and attempt to get them to comply with your recommendations for their pets, you are engaged in persuasion. Whether they engage in the recommended behavior, and for how long, is partly a function of their beliefs and attitudes about the behavior you want them to perform and your ability to listen and infer their beliefs.[4]

Beliefs

Beliefs are the bedrock of persuasion, and they can be defined as a description of what a person assumes to be true or false about the issue at hand.[3] Beliefs can be correct or incorrect, and incorrect facts can be termed "myths" in that they are popular beliefs that are unfounded or false.[5] Beliefs are learned, and they are systematically related to one another within a category. Two relevant categories are beliefs about spay/neuter and pet nutrition. Several commonly held beliefs in each category are listed along with whether they are true or false.

Spay/Neuter Beliefs and Facts

1. Early spay/neuter will stunt growth of the pet. (False)
2. A pet's temperament will change after spay/neuter. (False, but hormonally driven behaviors will be lessened.)
3. Neutering a male will make him less likely to roam or fight with other cats. (True)
4. A spayed female will live an average 18 months longer than an unsprayed female. (True)

Pet Nutrition Beliefs and Facts

1. Food is the leading cause of allergies. (False, it accounts for about 10% of allergies.)
2. Homemade food is best for pets. (False, homemade diets can be good, but it is difficult to make a diet that is complete and balanced at home.)
3. Raw foods are best for pets. (False, salmonella is one common problem.)
4. Soy is unusable in an animal's body. (False, quite a bit of data says otherwise.)
5. Commercial pet foods lack enzymes necessary for normal digestive function. (True, but animals make and secrete their own digestive enzymes and do not need such enzymes from the diet.)

Clients who hold certain beliefs or multiple beliefs in each category are likely to have a positive or negative attitude about your recommendations for their pets.

Attitudes

Attitudes are feelings about a certain person, object, issue, or behavior. They are general and enduring positive or negative feelings, and they result in a readiness to think, feel or act in a particular way.[2,3] While attitudes cannot be observed directly, they can be inferred. Attitudes often influence behaviors. For example, clients who hold the first two incorrect beliefs about spay/neuter will likely have a negative attitude about the procedure. They may envision a small, lazy pet as a result of the procedure. Understanding client beliefs and attitudes about an issue or procedure will help you to be a more strategic and effective advocate for pet health.

Shaping, Reinforcing or Changing Behavior

An influence attempt can function to *shape, reinforce* or *change* behavior. Shaping, or teaching, occurs when the target has no established pattern of behavior and does not know facts relevant to the issue.[2] The process allows the communicator to set goals for target behavior, providing guidance for a desired behavior that is not currently in the target's repertoire. In the context of veterinary care, this teaching function is common for first-time or new pet owners who are navigating the process of pet ownership. In many cases, the clients have yet to establish pet care practices (i.e., feeding regimens, vaccination schedules). Initial veterinary consults with a new pet owner can afford opportunities in which shaping or teaching new facts or beliefs can take place.

Reinforcement occurs when persuasive messages explicitly or implicitly advocate enactment of a behavior that the target already has adopted, or aim to strengthen adherence to existing behaviors that are based on correct knowledge.[2] In such cases, the target has the requisite knowledge and is already engaging in the desired behaviors, and the goal is to encourage maintaining such behaviors or, in some cases, increasing their frequency. For example, a veterinarian may praise a pet owner for being diligent about following prescribed recommended dental care for a new dog. Given that the target (i.e., dog owner) is engaging in the desired behavior, the goal of the reinforcement is to highlight those actions as desired and to strengthen commitment. Reinforcement may also serve to strengthen such behaviors. For example, that owner may be diligent about initial dental care but tend to get lax about dental care as the pet ages. In this context, reinforcement can take place to emphasize the continued need for dental care to promote pet health.

Change occurs when an influence attempt functions to cause a target to stop enacting one behavior and to adopt a different behavior,[2] and the behavior is often based on incorrect knowledge and a negative attitude toward the healthy behavior. A change attempt is employed when targets are engaging in behaviors that are potentially detrimental to a pet's health. For example, owners giving excessive table scraps to their pets can contribute to the animals' poor nutrition and obesity. A veterinarian addressing an owner of an obese pet might engage in change attempts to influence the owner to stop the behavior of giving table scraps and replace it with more healthful behaviors, such as a low-calorie treat associated with training or pet exercise.

In order to engage in the appropriate influence attempt, it is important to assess the current state of the client and pet. The assessment can inform whether the provider should engage in communication to *shape, reinforce* or *change* current behavior. This can be done through formative research, experience with clients in general or with the specific owner, or an informal assessment during the client-provider interactions. In conducting a needs assessment, a patient-centered approach to directing care can be established.

The path between beliefs and behaviors is not always clear. The Health Belief Model is one framework that can be used to guide the discussion of beliefs toward the engagement in a wide range of target behaviors.

Health Belief Model

The Health Belief Model is a commonly utilized framework for explaining and predicting the acceptance of health-care recommendations.[9,10] Though originally developed to assess engagement in preventive measures for early detection of disease, the HBM has been extended to a wide range of health practices including patients' responses to symptoms and compliance with medically prescribed courses of action.[10-12] The HBM provides a framework in which five major factors influence whether one engages in prescribed health behaviors. They are: perceived susceptibility, perceived severity, perceived benefits, perceived barriers, and cues to action.

Perceived Susceptibility. The first component of the HBM is perceived susceptibility, or the belief that one, in this case a pet, is vulnerable to a condition or disease.[13] Susceptibility is measured at the individual level given the potential range of perceptions. Within any given health concern, there are those who perceive high vulnerability, while others may perceive immunity regardless of actual susceptibility. Taking action to avoid a condition or disease requires perceived susceptibility.[12] In addition to feeling susceptible, the health threat needs to be perceived as causing at least moderate harm.[12,14]

Perceived Severity. Perceived severity is the belief that a health threat would inflict harm or discomfort.[10] Similar to perceptions of susceptibility, severity also varies. Severity may be judged by the degree of physiological and psychological damage a condition or disease may cause as well as the degree to which the disease disrupts daily life.[12] According to the HBM, in order for an individual to take action, the health threat needs to be perceived as moderately severe.[12,14] Within the model, the combined levels of perceived susceptibility and severity create a force or readiness to act.

Perceived Benefits. While perceived susceptibility and severity create a readiness to act, the path or type of action taken is

determined, in part, by the beliefs about perceived benefits. An individual is likely to engage in a behavior that will aid in reducing perceptions of susceptibility and severity of a given health threat.[12] By evaluating a course of action, individuals develop perceptions of the potential benefits gained. For example, clients living in a densely populated mosquito area with high reports of heartworm cases (i.e., susceptibility) who are aware of the disease or infection that heartworm can cause (i.e., severity) might seek the benefits of a recommended heartworm preventive for their pets.

Perceived Barriers. Along with perceived benefits to engage in a given health behavior, another factor that directs a path of action are the beliefs about perceived obstacles to that behavior. Within the HBM, perceived barriers incorporate potential negative aspects of, or impediments to, engaging in a given health behavior. In order for individuals to engage in a given health behavior, they must perceive it to be both beneficial and efficacious. Thus, many individuals may perceive certain health activities to have many benefits yet at the same time feel they are unable to accomplish them.[10,12,15] Barriers can be physical, psychological, emotional, or financial.[9] In the case of pet ownership, many of the target barriers can be financial. Reported barriers to preventive health measures, such as flea, tick and heartworm treatments, are how costly they are. Certain recommended foods, modified in nutrients or specialized for certain medical conditions, may be ideal yet financially unobtainable. Procedures such as allergy testing also can be costly, whether real or perceived, and may deter pet owners from compliance.

Individuals compare perceptions of health benefits to perceived barriers to identify a preferred course of action. Thus, according to HBM, a "sufficiently threatened" individual would not likely engage in the target behavior unless it was perceived as personally feasible and efficacious.[12,14]

Cues to Action. While perceptions of susceptibility, severity, benefits, and barriers direct a course of action, cues to action encourage individuals to act on that preferred path. Cues to action are specific stimuli necessary to trigger the audience's readiness for the preventive behavior.[10,12,16] The strength of the cues to action largely depends upon an individual's perception of susceptibility and severity. If individuals feel they or their pet is highly susceptible to a condition or disease they perceive as severe, the cue to action may only need to be subtle, such as a poster in the examination room. Conversely, if individuals perceive themselves to be fairly immune to a health threat or that the health threat is limited in severity, the cues to action will likely need to be more intensive, such as a well-argued plea for the action. Last, demographic and socio-psychological variables must be considered to identify factors that may influence an individual's perceptions and decision-making process.[10]

The HBM predicts that the interaction among beliefs about perceptions of susceptibility, severity, benefits, and barriers determines the effectiveness of a health behavior message as well as the likelihood of engaging in the target behavior. In sum, the combined levels of perceived susceptibility and severity create a force or readiness to act. The perceived benefits minus perceptions of barriers establish a likelihood of engaging in a given course of action. Taken together, the HBM predicts, in general, that as the individual assessment of risk increases, so does behavioral compliance.[10] The HBM has been applied to a wide range of health behaviors in attempt to better understand decision-making surrounding that behavior.[9,10,14,17-19]

When considering behavior change, another theoretical framework useful to guide conversational interventions with clients is the transtheoretical model of change. It may be the case that clients perceive pet susceptibility and the outcomes to be severe and the benefits to outweigh the barriers, yet not be ready to take action.

Transtheoretical Model of Change

One theoretical approach to attempt to predict and explain behavior change is the transtheoretical model of change.[20] The TTM predicts that change occurs through a six-stage progress: precontemplation, contemplation, preparation, action, maintenance, and termination. Each stage is structured by a temporal dimension of readiness to act with differences based on the degree of intention and behavior performed. The stages progress from *precontemplation* (no intention of engaging in target behavior) to *contemplation* (thinking about engaging in the behavior within the next six months), *preparation* (making small changes in action toward target behavior), *action* (engaging in target behavior but only recently), *maintenance* (meeting criterion for target behavior for six months or longer), and *termination* (no longer engaging in target behavior).

The TTM employs a client-centered approach in recognizing that people vary on their readiness to enact a target health behavior. Whether it is individual or situational differences contributing to that stage, catering messages and intervention strategies to the stage of the client may increase receptiveness. For example, pet owners in the precontemplation stage may be those who do not know that an issue exists with their pets and will need a different approach (shaping) than those who do not plan to engage in the target behavior due to incorrect myths held on the topic (changing). Further, pet owners already engaging in the target behavior will need another type of intervention, reinforcement messages, to encourage the continuation of their actions.

With regard to individuals and their pets, there are not only ideal target health behaviors but also many factors that impede or attenuate owner ability to follow through and later maintain certain actions. The benefit of examining behavior change through the transtheoretical lens is that change is not dichotomous but rather a dynamic and continuous phenomenon.[20] Although it was originally thought that individuals progressed through stages in

a linear fashion, it is now recognized that individuals move through stages in a cyclical fashion, progressing at some points and regressing at others in an effort to create change that works with their lifestyle.

Conclusion

Assessment tools such as short questionnaires or screening questions can be used to assess pet owner beliefs about issues and procedures in your practice. Gaining insights to client beliefs will help inform whether you need to teach, reinforce or change their beliefs, attitudes and behaviors. Two theories were offered to help you in this process. The HBM suggests that communicating pet susceptibility and severity can clarify a recommended course of action. It further highlights that through enhancement of client efficacy and assisting the elimination of barriers, greater behavioral compliance will likely occur. The TTM gives insights to individual differences and readiness to modify existing actions. Provider consideration of the stages of readiness will aid in more patient-centered approaches and the tailoring of behavioral recommendations. Understanding client beliefs and attitudes about an issue or procedure will help you to be a more strategic and effective advocate for pet health.

References

1. Abood SK. Effectively communicating with your clients. *Top Comp An Med*. 2008;23:143-147.

2. Miller GR. On being persuaded: Some basic distinctions. In *Persuasion: New Directions in Theory and Research*. Rolloff ME, Miller GR (eds). Sage, Beverly Hills, CA. 1980:11-28.

3. O'Keefe DJ. *Persuasion: Theory and Research*. Sage, Thousand Oaks, CA. 2002(2nd ed).

4. Bonvicini K, Abood SK. Communicating with the client: Enhancing compliance. *Hills Global Sym on Feline Care*. 2006. Toronto.

5. http://www.merriam-webster.com/dictionary/myth.

6. Caldwell F. Pets best insurance blog true or false? Top five beliefs about spay and meuter. Posted: September 11, 2012.

7. Laflamme DP. Commercial pet foods – The bottom line. *Proc N Am Vet Con*. 2004. Orlando, FL

8. Laflamme DP. I read it on the Internet: Nutritional myths and misperceptions. *Proc Geo Vet Med Assoc*. 2010. Sandestin, FL.

9. Brown LK, DiClemente RJ, Reynolds LA. HIV prevention for adolescents: Utility of the health belief model. *AIDS Educ Prev*. 1991;3:50-59.

10. Janz NK, Becker MH. The health belief model: A decade later. *Health Educ and Behav*. 1984;11:1-47.

11. Kirscht JP. Research related to the modifications of health beliefs. *Health Educ Monogr*. 1974;2:455-469.

12. Rosenstock IM. The health belief model: Origins and correlates. *Health Educ Monogr*. 1974;2:336-353.

13. Rimal RN, Real R. Perceived risk and efficacy beliefs as motivators of change. *Hum Commun Res*. 2003; 29:370-399.

14. Rosenstock IM, Strecher VJ, Becker MH. Health belief model and HIV risk behavior change. In *Preventing AIDS: Theories and Methods of Behavioral Interventions*. DiClemente RJ, Peterson JL (eds). Plenum Press, New York. 1994:5-24.

15. Ajzen I. The theory of planned behavior. *Organ Behav Hum Decis Process*.1991;50:179-211.

16. Mattson M. Toward a reconceptualization of communication cues to action in the health belief model: HIV test counseling. *Commun Monogr*. 1999;66:240-265.

17. Harrison JA, Mullen PD, Green LW. A meta-analysis of studies of the health belief model with adults. *Health Educ Res*. 1992;7:107-116.

18. Rosenstock IM, Strecher VJ, Becker MH. Social learning theory and the health belief model. *Health Educ and Behav*. 1988;15:175-183.

19. Chew F, Palmer S, Kim S. Testing the influence of the health belief model and a televised program on nutrition behavior. *Health Commun*. 1998;10:227-245.

20. Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: Toward an integrative model of change. *J Consult Clin Psychol*. 1983;51:390-395.

Appendix | 2013 CAN Summit: Tackling Myths About Pet Nutrition

Speakers

Product Safety and Pet Food Recalls

Karyn Bischoff, DVM, DABVT, Cornell University

Dr. Karyn Bischoff completed her veterinary degree at the University of Illinois at Champaign-Urbana, and then did a residency in toxicology at Oklahoma State University and was certified by the American Board of Veterinary Toxicology. Since then, she has had further training and experience in anatomic pathology and laboratory animal medicine. Dr. Bischoff has been the diagnostic toxicologist at the New York State Animal Health Diagnostic Center and a faculty member of the Cornell University College of Veterinary Medicine since 2004. During that time, she has been involved in three pet food recalls.

Ensuring Responsible Sourcing for a Safe, Quality and Sustainable Supply of Ingredients for Pet Diets

Steve Binder, PhD, Nestlé Purina PetCare

Dr. Steve Binder earned his doctorate in cereal chemistry from Kansas State University. As a cereal scientist, he has been involved in the evaluation and application of ingredients in animal feeds and the associated processing of these materials since 1975. Currently, he is Director for Ingredients in the Product Development Department of Nestlé Purina PetCare.

Ingredient Myths That Have Altered the Course of Pet Food: Byproducts, Synthetic Preservatives and Grain

Greg Aldrich, PhD, Kansas State University

Dr. Greg Aldrich is an independent nutritionist specializing in foods, ingredients and nutrition for companion animals, as well as associate professor and coordinator of the Pet Food Program at Kansas State University. His research program is focused on pet food safety and process effects on nutrition and shelf life. He also provides diet formulation, nutritional advice, product support, and technical communications related to pet foods and their development. Dr. Aldrich writes a monthly column for *Petfood Industry* magazine on ingredient issues and is a frequent speaker at industry and scientific forums. He has had a lifetime fascination with feeding animals and has spent more than 25 years studying and working in the animal nutrition field. He received his doctorate in nutrition from the University of Illinois and a master's degree from the University of Missouri. He has held several industry management and technical positions, including with Co-op Feeds, the Iams Co., Kemin Industries Inc., and Menu Foods Ltd. Dr. Aldrich is a member of the American Society of Animal Science, American Society for Nutrition, and the American Academy of Veterinary Nutrition. Dr. Aldrich has been on the faculty at K-State since April 2012. He and his wife, Susan Aldrich, Ph.D., have managed their consulting firm, Pet Food & Ingredient Technology Inc., from Topeka, Kan., since 2003.

Impact of Extrusion on Pet Food Nutrition

Mian N. Riaz, PhD, Texas A&M University

Dr. Mian N. Riaz earned a bachelor's and a master's degree in food technology from the University of Agriculture in Faisalabad, Pakistan, and received a doctorate degree in food science from the University of Maine in 1992. He is the Director of the Food Protein R&D Center and Head of the Extrusion Technology Program and graduate faculty in the Food Science and Technology Program at Texas A&M University. Dr. Riaz has published five books, 17 chapters and more than 100 papers on extrusion and related topics. Three of his books are in the area of extrusion: Extruders in Food Application; Extruders and Expanders in Pet Food, Aquatic and Livestock Feed; and Extrusion Problem Solved. Dr. Riaz is a frequent speaker at international and national conferences and meetings, and has delivered more than 150 presentations in 47 countries. Each year, he organizes four courses in the area of extrusion. Dr. Riaz has been with Texas A&M University for 20 years and has a teaching appointment in the Nutrition and Food Science Department.

Making Pet Food: Quality Assurance

Adrian Palensky, Nestlé Purina PetCare

Adrian Palensky is Vice President of Quality Assurance for Nestlé Purina PetCare, North America and Latin America, where he leads a team of quality professionals in the areas of food safety, compliance and quality operations. Mr. Palensky grew up on a family farm in eastern Nebraska, understanding the importance of animals and the commitment and responsibility as a caretaker. He attended the University of Nebraska, earning a degree in industrial and management systems engineering. Mr. Palensky has over 25 years of experience in pet food quality assurance.

Fuel for Felines: Cats and Carbohydrates

Margarethe Hoenig, Dr.med.vet, PhD, University of Illinois

Dr. Margarethe Hoenig earned her veterinary degree from Hannover University in Germany and her doctorate degree in

pathology from the University of Pennsylvania. After completing an internship at the University of California-Davis, she returned to the University of Pennsylvania to complete a residency in internal medicine. She was a senior research associate at the New York State College of Veterinary Medicine at Cornell University before joining the faculty at the University of Georgia, where she worked until 2008. Currently, a professor at the University of Illinois, Dr. Hoenig has received several awards for her research and has written numerous publications on obesity, diabetes and thyroid disease.

Feline Nutrition: What Is Excess Carbohydrate?

Claudia Kirk, DVM, PhD, DACVIM, DACVN, University of Tennessee

Dr. Claudia Kirk received her veterinary degree from the University of California at Davis, where she continued her training and completed her doctorate degree as well as a residency in both internal medicine and nutrition. She worked in the pet food industry with Hill's Pet Nutrition before accepting a position at the University of Tennessee, where she currently is a professor and head of the Department of Small Animal Clinical Sciences. Dr. Kirk is board-certified by the American College of Veterinary Internal Medicine and the American College of Veterinary Nutrition. She has written numerous publications related to feline and canine nutrition.

Optimal and Natural as Rationale for Selecting Dietary Energy Distribution in Carbohydrate and Fat

Robert C. Backus, DVM, PhD, DACVN, University of Missouri

Dr. Robert C. Backus received his veterinary degree from the University of California-Davis, where he continued his training and completed master's and doctorate degrees, as well as a residency, in veterinary nutrition. Dr. Backus currently is an associate professor and Director of the Nestlé Purina Endowed Small Animal Nutrition Program at the University of Missouri. Dr. Backus is an author of numerous publications related to feline and canine nutrition.

Protein Metabolism: Adaptation

Andrea Fascetti, VMD, PhD, DACVN, DACVIM, University of California-Davis

Dr. Andrea Fascetti graduated from the University of Pennsylvania School of Veterinary Medicine, and then completed an internship and medicine residency at The Animal Medical Center in New York City. She holds a doctorate degree in nutrition from the University of California-Davis. A Diplomate of both the American College of Veterinary Internal Medicine and the American College of Veterinary Nutrition, Dr. Fascetti currently is professor of nutrition at the University of California-Davis and service chief for the Nutrition Support Service in the Veterinary Medical Teaching Hospital.

Determining Protein Requirements: Nitrogen Balance Versus Lean Body Mass

Dottie Laflamme, DVM, PhD, DACVN, Nestlé Purina PetCare

Dr. Dottie Laflamme received her veterinary degree from the University of Georgia, where she also completed a master's degree in nutrition and a doctorate degree in nutrition and physiology. She completed her clinical nutrition residency as an ALPO postdoctoral fellow in clinical nutrition. Dr. Laflamme is a Diplomate and Past-President of the American College of Veterinary Nutrition. She is an author on over 200 scientific and technical publications and has been a speaker at numerous veterinary, research and continuing education programs worldwide. She has worked for Purina — first Ralston Purina, now Nestlé Purina — in the Research and Development Department since 1990, and has served in numerous research and management positions. Currently, she is a Communications Specialist in veterinary nutrition, and her research focus is therapeutic nutrition, especially obesity management. Dr. Laflamme lives in the Blue Ridge Mountains of western Virginia.

How Important Is Protein for Weight Management?

Cecilia Villaverde, BVSc, PhD, DACVN, DECVCN, Universitat Autònoma de Barcelona

Dr. Cecilia Villaverde received her veterinary degree in 2000 from the Universitat Autònoma de Barcelona in Spain, where she also obtained her doctorate degree in animal nutrition in 2005. After two years as a postdoctoral research fellow in feline nutrition at the University of California-Davis, she completed a two-year residency there in small-animal clinical nutrition at the Veterinary Medical Teaching Hospital. Since 2010, she has been board-certified in veterinary nutrition by the American College of Veterinary Nutrition and the Europoean College of Veterinary and Comparative Nutrition. Currently, Dr. Villaverde is chief of service of the Servei de Dietètica i Nutrició, Fundació Hospital Clínic Veterinari at the Universitat Autònoma de Barcelona.

Balanced Feeding Assists Healthy Growth in Dogs

Britta Dobenecker, Dr.med.vet., DECVCN, Ludwig-Maximilians University

Dr. Britta Dobenecker earned her veterinary degree and completed her doctoral thesis in veterinary nutrition at the veterinary school in Hannover, Germany. She is boarded by the European College of Veterinary and Comparative Nutrition and is currently on the faculty at Ludwig-Maximilians University in Munich, Germany, serving as a specialist and senior lecturer in animal nutrition and dietetics. Dr. Dobenecker's research has focused on the effects

of mineral supplies on skeletal development in puppies and renal health in cats as well as ration calculation and nutrition consultation. She serves on the scientific advisory board for the European Pet Food Industry Federation (FEDIAF), which establishes the nutritional guidelines for pet foods in Europe.

The New Age of Working Dogs: Different Jobs, Different Diets

Joseph J. Wakshlag, DVM, PhD, DACVN, DACVSMR, Cornell University

Dr. Joseph J. Wakshlag completed a veterinary degree at Cornell University, where he continued studies in both pathology and nutrition. He earned a doctorate degree in pharmacology and subsequently completed a residency in clinical nutrition. Dr. Wakshlag is board-certified by the American College of Veterinary Nutrition and the American College of Veterinary Sports Medicine and Rehabilitation. Currently, he is assistant professor of clinical nutrition at Cornell University College of Veterinary Medicine. His research interests include carotenoids and fatty acid metabolism in cancer cell biology and metabolism in working dogs.

Etiopathogenesis of Canine GDV: A Nutritionist's Interpretation of Evidence

David A. Dzanis, DVM, PhD, DACVN, Regulatory Discretion Inc.

Dr. David A. Dzanis is CEO of Regulatory Discretion Inc., a consulting firm for the food, feed and supplement industries in matters relating to nutrition, regulation and labeling. He r eceived his veterinary degree from Purdue University in 1982 and his doctorate degree in veterinary nutrition from Cornell University in 1989. Between 1989 and 1998, Dr. Dzanis served as a veterinary nutritionist for the Food and Drug Administration (FDA) and was a member of the Association of American Feed Control Officials (AAFCO) Pet Food Committee. He is a Diplomat and current Secretary of the American College of Veterinary Nutrition and a Past-President of the American Academy of Veterinary Nutrition, and was a member of two National Research Council committees on dog, cat and horse nutrition. Dr. Dzanis writes a monthly column on regulatory issues for *Petfood Industry* magazine.

Preventing Disease Through Nutrition: Reducing Nutrients to Prevent Disease?

Richard Hill, VetMB, PhD, DACVIM, DACVN, University of Florida

Dr. Richard Hill earned his veterinary degree from Cambridge University in the U.K. and his doctorate degree from the University of Florida. He completed clinical training in both internal medicine and veterinary nutrition and is boarded by both the American College of Veterinary Internal Medicine and the American College of Veterinary Nutrition. Dr. Hill currently is an associate professor at the University of Florida. His research interests include gastroenterology and clinical nutrition, particularly energy requirements. He served on the National Research Council's committee on dog and cat nutrition and is an author on the current Nutrient Requirements of Dogs and Cats.

Communicating with Clients to Teach, Reinforce or Change Pet Beliefs

Sandi W. Smith, PhD, Michigan State University

Dr. Sandi W. Smith earned her doctorate degree in communication from the University of Southern California. She is Director of the Health and Risk Communication Center and professor in the Department of Communication at Michigan State University, as well as associate editor of *Health Communication*. Her work has been funded by NSF, HRSA, NCI, NIEHS, and the U.S. Department of Education. Dr. Smith's research interests center on the impact of communication on health behavior, such as the prevention and detection of breast cancer, persuading people to enroll on state organ donor registries and to engage in family discussion about organ donation, and encouraging college students to consume alcohol moderately. Her research frequently appears in the *Journal of Health Communications* and *Health Communication*, as well as in *Communication Monographs, Human Communication Research* and the *Journal of Communication*. Her book with Steve Wilson, *New Directions in Interpersonal Communication Research*, recently received the G.R. Miller Outstanding Book Award from NCA. Dr. Smith also has been honored with the Distinguished Faculty Award at Michigan State University and received the B. Aubrey Fisher Mentorship Award from ICA.