Canine Nutrition NEWSLETTER

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Protein is an essential nutrient for dogs and amino acids play an important role in the body. For more information about the structure and function of protein and amino acids, please read:

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Current strategies for managing hepatic disease all target to reduce the risks of hepatic encephalopathy. But is this a relevant strategy for most dogs with liver disease? This article explains the pathology of hepatic encephalopathy, its causes and treatment, with a discussion on its relevance in different forms of hepatic disease. For more information on hepatic encephalopathy, please read:

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The liver is one of the most complicated organs in the body, responsible for more than 500 different essential tasks. Although it has an amazing ability to regenerate itself after suffering damage, the consequences are severe when the damage exceeds the organ's ability to compensate. Dietary intervention can be beneficial. For an overview of the dietary management of liver disease, including 8 home-made recipes for dogs with liver disease, please see:

Dietary Management of Canine Liver Disease
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Once again Laura has found a great research paper. This study challenges NRC's most recent guidelines for iodine requirements in cats. There are people who would use this study to argue that NRC recommendations are unreliable. In fact, this study illustrates the opposite. It shows the value of peer reviewed studies, of scientific scrutiny, scientific challenge and the value of empirical evidence. This study epitomizes good science – the advancement of knowledge through carefully executed studies. For more info, please see:

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Protein and Amino Acids – Structure and Function

By Elizabeth Pask, PhD

Protein makes up approximately 15-30% of your dog's diet. Protein has many roles in the body. In the following article we are going to examine the various functions of proteins.

Functions of Different Proteins in the Body

Protein has a wide variety of functions in the body. The largest store of protein and amino acids in the body is ***structural protein*** (i.e. skeletal muscle). Skeletal muscle is responsible for moving our arms and legs and provides us with the ability to eat, play and move around. However this is not the only function of protein in the body.

Enzymes are proteins. Enzymes are responsible for many functions including digesting proteins that we ingest. Some hormones are proteins.

Protein hormones (also called peptide hormones) control a variety of functions in the body by controlling the turning on and off of enzymes. One common peptide hormone is Insulin. Insulin is responsible for controlling the level of blood sugar in our body.

Immune proteins help recognize bacteria, viruses and other foreign bodies and then help the body get rid of these potential pathogens.

Transport proteins are responsible for moving molecules around our body. Hemoglobin is an example of a transport protein. Hemoglobin transports oxygen from the lungs to the rest of the body. Another transport protein is called albumin. Albumin transports a variety of substances around the body.

What is Protein?

All protein is made up of small chemicals called amino acids. Protein can be a variety of sizes and shapes. The shape and size of the protein is determined by genetic code. Proteins have a hierarchy of structure. Simple proteins can be a strand of amino acids joined together (called a peptide) – this is called the primary structure. With larger proteins the strand of amino acids will coil back on each other to form a secondary structure like an elastic band that has been over-wound (called an alpha-helix or beta-sheet). Proteins can be larger and more complex and form a tertiary structure which is multiple sheets and helixes bound together to form a glob like structure. Proteins can be even larger still and multiple globs can bind together. For example the blood protein called hemoglobin contains 4 globular protein structures bound together. This structure allows it to effectively carry oxygen from the lungs to the rest of the body.

This structure is very important. Different proteins will have different structures and as a result they will have different jobs to do in the body. The structure of the protein is determined by genetic code. Genetic code tells the cell which amino acids to join together. If the wrong amino acids gets put in by accident they the whole structure of the protein the cell is building can chance. Therefore having the right amino acids available to the body at the time it wants it is crucial for the health of your dog. If there is not enough of the right amount of amino acids available then the body will start breaking down muscle protein in order to fulfill the requirement. This is why dogs that have been starved have little muscle on their body.

Amino Acids: The Building Blocks of Protein

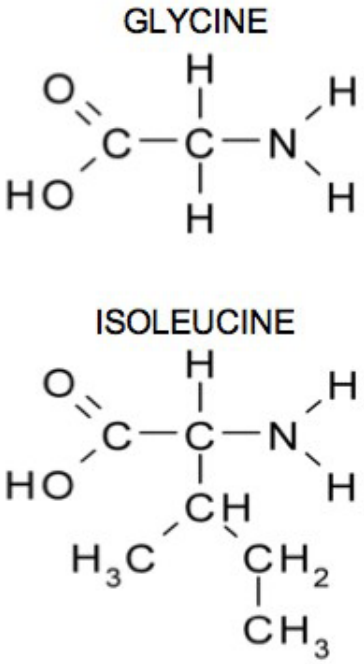


Figure 1

Figure 1 (Left): Example structures of amino acids. Notice that the top part of both amino acids is the same – a carboxyl group attached to a central carbon attached to an amino group. Notice that these two amino acids have different "side chains". Glycine's side chain is a simple hydrogen atom, while isoleucine, as one of the branched chain amino acids, has a side chain with a branching carbon structure.

Protein is essential in the diet because of these amino acid building blocks it contains. The body has a requirement for 10 amino acids and if the diet cannot provide them an amino acid deficiency will exist. This deficiency will result in the body using amino acid stores in an attempt to balance the deficiency. Therefore it is essential that the diet provide a good quality source of protein that contains these essential amino acids. Some diets may contain a variety of protein sources to ensure that sufficient amounts of amino acids are present in the diet. The amount of a particular amino acid required will vary with the life stage of the dog. Growing, pregnant or nursing dogs will have slightly different requirements when compared to senior dogs. In addition, a dog with a chronic disease or canine athletes may have a greater requirement for some amino acids called branched chain amino acids.

Table 1: The names of essential amino acids

Isoleucine	Leucine	Lysine	Methionine	Phenylalanine
Threonine	Tryptophan	Valine	Histidine	Arginine

Amino Acid Metabolism

Dietary protein is digested in the small intestine and amino acids are absorbed across the wall of the upper small intestine called the jejunum. Once in the blood the amino acids are primarily taken up by the liver. Once in the liver about 57% of the amino acids are broken down, 20% will be used to make proteins (such as albumin) and the remaining 23% of amino acids are released in to the blood stream. When the liver becomes distressed or diseased it can impact the metabolism of amino acids. As a result when the body is under a disease state the amount and type of amino acids should be altered so that the body can be supported nutritionally.

Branched Chain Amino Acids and the Canine Athlete

As mentioned above some body conditions can result in a higher requirement for branch chain amino acids. Canine athletes are dogs that are training and competing on a daily or weekly basis (ie top level agility, field or sled dogs). Similar to human athletes, canine athletes are pushing their bodies to their maximum ability.

Branch chain amino acids are a group of amino acids that have a branched structure. Valine, leu-

cine and isoleucine are branched chain amino acids. These amino acids are essential for proper growth, maintenance of the body and the body requires more of them during times of physical stress (i.e. during athletic performance). Branch chain amino acids are mainly metabolized mainly by muscle. Leucine stimulates protein synthesis and reduce protein breakdown in muscle. During exercise muscle is under stress which can result in muscle catabolism and damage. Leucine helps prevent this breakdown. In addition Leucine is one of the few amino acids that can be used as a fuel source by the body which helps to spare muscle glucose. In addition to helping with muscle integrity and glucose sparing BCAAs are thought to help with cognitive function in active dogs by aiding the brain in taking up the amino acid tryptophan. Tryptophan is used by the brain to make serotonin, which is the neurotransmitter that is responsible for making us happy and relaxed. When dogs are happy and relaxed they perform better. In a recent study researchers fed agility dogs a BCAA supplement prior to their run. The dogs that were supplemented with the BCAA had fewer errors over their course and the greatest improvement was seen in mature dogs (≥8 years). As a result there may be an improvement in performance for older dogs when supplemented with BCAA prior exercise.

"Olive Peabody"

Testimonial

For the last two months, I've been feeding my dog Olive Peabody the Vegetarian Diabetic 1 recipe from the Complete and Balanced recipe book. It has literally been a life saver.

When we moved to a new city at the end of June, Olive got extremely sick. At first I thought she had stopped eating because she was stressed out from the move. When she continued to refuse food, I took her to my wonderful veterinarian. He diagnosed Olive with diabetes. It had reached medical crisis because she hadn't been eating. After she was hospitalized for a couple of days, I brought her home with cans of commercial brand prescription diet food for diabetic dogs. She refused to eat both brands offered by the animal hospital. Getting her to eat again was critical in bringing her out of the medical crisis. After trying to cajole her into eating the canned food and even force feeding her for several days, I asked my veterinarian about a home-made diet. After speaking with Hilary, he recommended I feed the Vegetarian Diabetic Recipe along with Hilary's Blend supplement. Olive loves the peanut butter and tomato sauce recipe! She has never been a great eater, but now the food disappears as soon as I put down her plate. Once she started eating regularly again, we could get her glucose levels stabilized. Now I think she's healthier than she's ever been. The Complete & Balanced cookbook and Hilary's Blend supplement are a big part of that. I cannot express how grateful I am.

Nick Porter

Hepatic Encephalopathy in Dogs

By Hilary Watson BSc

Current dietary strategies for managing dogs with liver disease focus on minimizing the risks of hepatic encephalopathy. This partially evolved from human medicine. Hepatic encephalopathy (HE) occurs in up to 50% of humans with cirrhosis of the liver and its prognosis is very poor. For humans, the 3-year survival rate following the first episode of HE is 15%. So in managing humans with liver disease, a major focus has been on preventing HE. The commercial veterinary hepatic diets have followed suit. Is this an appropriate dietary strategy for dogs with liver disease? The answer is yes and no. Let's begin with a review of HE.

Hepatic encephalopathy (HE)

HE can be defined as all neurological symptoms in patients with liver disease that cannot be explained by other pathologies. HE occurs because the liver fails in its usual role of removing and detoxifying neurotoxins present in the blood. Signs of HE include seizures, tremors, head pressing, blindness, behavioural changes, cognitive impairment and poor coordination. HE can progress to coma and death.

Four different (and interconnected) hypotheses have been proposed to explain how HE develops.

The ammonia hypothesis suggests that HE is due to a high concentration of ammonia in the blood. Ammonia is generated in various ways. Normal metabolism results in the release of nitrogen from protein as ammonia. This ammonia is normally converted into urea by the liver. Urea is then removed from the body by the kidneys in urine. In liver disease, the liver's ability to convert ammonia into urea may be reduced by up to 80%. Ammonia concentration begins to rise in the blood.

Since about 50% of the body's ammonia pool is found in skeletal muscle, muscle wasting (which commonly occurs in liver disease) results in the release of ammonia into the blood.

Bacteria in the gut are another significant source of ammonia. Undigested protein in the gut is de-aminated by intestinal bacteria. The released ammonia can pass back into the dog's body. High protein intake and gastrointestinal bleeding also contribute to high concentrations of ammonia in the blood.

Ammonia in the blood easily crosses the blood-brain-barrier. In the brain, excessive ammonia (NH3) drives these two reactions to the right:

NH3 + alpha-ketoglutarate + NADH ===> glutamate + NAD
NH3 + glutamate + ATP ===> glutamine + ADP + P

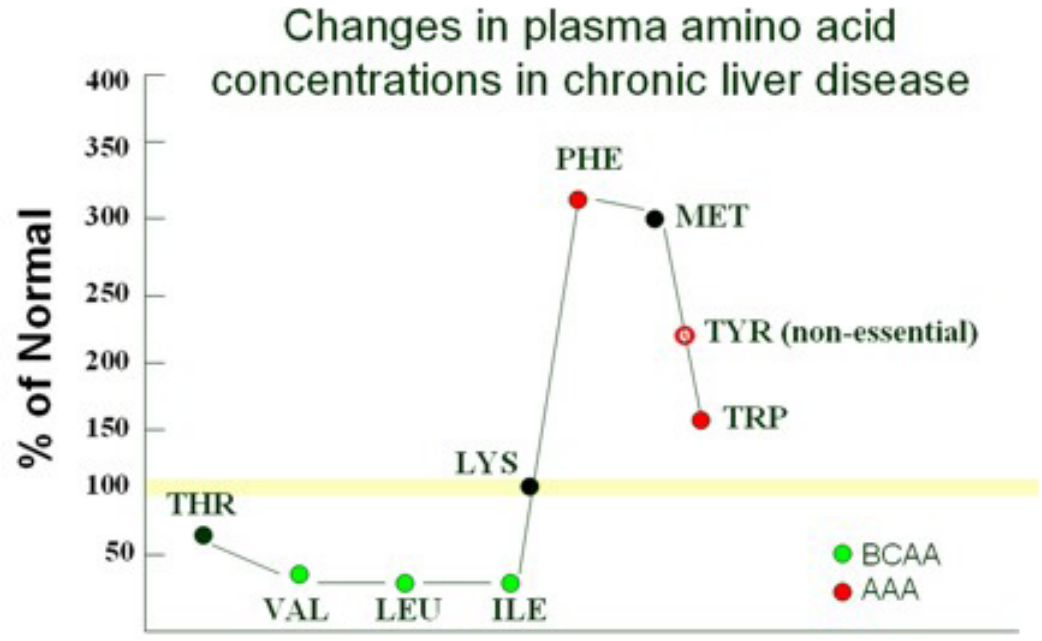
Alpha-ketoglutarate is a key intermediate in Kreb's cycle (ie the chain of reactions that turns food into energy/ATP in cells). As alpha-ketoglutarate is depleted in brain cells, energy/ATP production is interrupted which can lead to coma or death. The above reactions also cause an accumulation of glutamine in the brain. In the cerebrospinal fluid and brain cells of humans with liver disease, glutamine is 5 to 10 times more concentrated than in healthy people. While glutamine itself is not neurotoxic, it is an osmolyte, meaning it draws water. Glutamine accumulation in astrocytes (brain cells) causes swelling and brain edema. Glutamine accumulation in the brain also appears to facilitate the uptake of aromatic amino acids by the brain.

The synergistic neurotoxin hypothesis suggests that it's not just ammonia that causes HE but

rather a combination of multiple harmful substances including mercaptans, indoles, scatoles, as well as ammonia. These other neurotoxins are mostly produced by bacteria in the intestines. Controlling "bad" and promoting "good" bacteria in the gut may help to reduce the production of these compounds. Prebiotics and probiotics may also be beneficial in altering the type of bacteria present in the gut.

The false neurotransmitter hypothesis suggests that increased concentrations of gamma amino butyric acid (GABA) and benzodiazepam-like substances in the brain cause HE. Albumin is a carrier protein for many potentially encephalopathic substances including benzodiazepines and the amino acid tryptophan. Liver patients often have reduced levels of albumin in the blood (hypoalbuminemia), resulting in these substances circulating unbound in the blood, and crossing more freely into the brain. In addition, the cerebral accumulation of manganese (which is excreted in bile in healthy individuals) has also been implicated in abnormal neurotransmitter synthesis in humans with liver disease.

The amino acid hypothesis suggests that an abnormal balance between branched chain amino acids (BCAA) and aromatic amino acids (AAA) may lead to altered synthesis of neurotransmitters in the brain. In liver disease, blood concentrations of the branched chain amino acids (leucine, isoleucine and valine) fall, while those of the aromatic amino acids (phenylalanine, tyrosine and tryptophan) rise (see figure). In healthy individuals, AAA are extracted from the blood and metabolized by the liver, whereas BCAA bypass the liver and are metabolized by muscle and the brain. In liver disease, the liver fails to metabolize AAA, while muscle, brain and peripheral tissues continue to burn BCAA for fuel. This causes the ratio of BCAA:AAA to drop in the blood of liver patients. This occurs whether HE is present or not.



Cerra, et al; JPEN, 1985

The drop in BCAA and rise in AAA has two serious consequences. First, because BCAA (particularly leucine) promote protein synthesis and inhibit protein catabolism, a drop in BCAA can exacerbate to the loss of lean body mass. Second, because the AAA and BCAA share a common transporter across the blood-brain-barrier, a drop in the BCAA:AAA ratio causes an increase in the cerebral uptake of the aromatic amino acids. AAA can serve as substrates for the synthesis of some of the "false neurotransmitters" mentioned above.

Aromatic amino acid (AAA) precursor	Normal neurotransmitters	"False" neurotransmitters
Tyrosine	Dopamine	Tyramine Octopamine
Phenylalanine	Norepinephrine	Phenethyamine Phenylethanolamine
Tryptophan	Serotonin	Tryptamine

Current Dietary Management Strategies for Liver Disease in Dogs Target HE Prevention

The current dietary strategies for managing liver disease in dogs are almost all targeted at preventing HE. These include restricting protein (to minimize ammoniagenesis), using vegetable protein and avoiding meat protein (to reduce AAA intake), using dairy proteins such as cheese (to increase BCAA intake) and increasing fat (to provide non-protein calories to spare the use of protein for energy). Adjunctive therapies also target the same goal. These include feeding lactulose (which acidifies the digestive tract, causing ammonia to be converted into ammonium which cannot be reabsorbed from the gut into the dog's body) and using antibiotics like neomycin (which kills ammonia-producing bacteria in the gut). While there are other nutritional parameters relevant to managing liver patients (ie zinc, copper, vitamins etc), the current basis for managing liver patients and the primary goal of the veterinary therapeutic liver diets is to prevent HE. This may not be appropriate in many cases of dogs with liver disease. The risk of HE in dogs is much lower than that of cirrhotic humans.

To be clear, some dogs with liver disease are definitely at risk of developing HE, those with portosystemic shunts for example, and dogs with cirrhosis or juvenile fibrosing liver disorders. But many other types of liver disease are at much lower risk of developing HE. Here is a list of liver diseases that are NOT typically associated with HE in dogs:

1) Chronic active hepatitis
2) Major bile duct occlusion
3) Primary or metastatic neoplasia
4) Microvascular dysplasia
5) Vacuolar hepatopathy, secondary to:
- ***a) Chronic stress***
- ***b) Glucocorticoid therapy***
- ***c) Diabetes mellitus***
- ***d) Hepatocutaneous syndrome***
- ***e) Gastrointestinal inflammation***
- ***f) Inflammatory bowel disease***
- ***g) Pancreatitis***
- ***h) Hyperadrenocorticism, Hypoadrenocorticism***
- ***i) Hyperthyroidism, Hypothyroidism***
- ***j) Immune-mediated hemolytic anemia***
- ***k) Septicemia***
- ***l) Shock***
- ***m) Right-sided heart failure***
- ***n) Protein-losing enteropathy***
- ***o) Starvation***
- ***p) Non-hepatic neoplasia (lymphosarcoma etc)***
- ***q) Extra-hepatic infections***
- ***r) Severe dental disease***

The Relevance of HE in Managing Canine Liver Disease

With canine liver disease, there are probably two broad categories of patients – those at high risk of developing HE (porto-systemic shunts, liver cirrhosis) and those who are at less risk of developing HE (most others forms of liver disease). Many dietary strategies will apply to both categories. For example, increasing the BCAA:AAA ratio in the food is beneficial to both types of dogs. For dogs that are not at risk of HE, increasing the BCAA:AAA ratio will help to preserve lean body mass. For dogs at risk of HE, increasing this ratio will have the additional benefit of helping to minimize the production of "false neurotransmitters" in the brain.

The key difference in managing these two categories of patients is the degree to which protein needs to be restricted. For dogs at risk of HE, protein intake needs to be severely restricted while maintaining a positive nitrogen balance. For dogs that are not at risk of HE, a higher protein intake is possible and may be preferable, especially in cases where fat digestion is impaired (dogs with underlying problems of lipid metabolism, dogs with concurrent pancreatitis, inflammatory bowel disease, diabetes mellitus etc). For dogs at risk of HE, a protein intake of 2.2-2.5 g/kg BW/day is recommended as a starting point. This intake may need to be increased if a subsequent evaluation of body condition, serum albumin or creatinine suggests that the dog is in a negative nitrogen balance. In these cases, if protein intake needs to be increased, protein tolerance may be improved with the adjunctive treatments mentioned earlier (antibiotics such as metronidazole or neomycin, lactulose and/or prebiotic fibre). Although 2.2-2.5 g protein/kg BW/day is in the same ballpark as that recommended for renal disease, renal diets are not good choices for dogs with liver disease. Renal diets are highly restricted in phosphorus (well below AAFCO minimum). While this is appropriate for renal patients it is not appropriate for liver patients.

For dogs that are not at risk of HE, a somewhat higher target of 2.5-3.0 g protein/kg BW/day may give better results. Remember that the body weight of the dog affects the protein intake per kilogram of body weight. A large dog will consume less protein per kg BW/day than a smaller dog fed the same diet. (See page 51-52 and table 21 in my cookbook for a more detailed explanation of this concept).

Testimonial

Kona

Our toy poodle Kona was diagnosed with a liver condition (MVD) in December 2008. We switched her to a commercial food designed for dogs with liver disorders. I was concerned that with a compromised liver she would be less able to deal with potential risks so I started making food for her. My vet pointed out that I should be giving her a balanced diet and recommended Hilary's Blend. Hilary provided a custom recipe for Kona that incorporated catfish, cheese and tofu as the protein sources. A year and a half later Kona is thriving. She has gone from 8½ lbs to a healthy 10 lbs. Her coat is thicker and she has tons of energy. She can go for a 10 km hike, chasing her ball the whole way, and still want to play. I feel better having control over her diet and knowing that the recipe ensures complete nutrition for her liver condition. I have total confidence that her home cooked diet will keep her feeling healthy and give her as long a life as possible without creating unnecessary complications.

Glenn and Jill Baker

Dietary Management of Canine Liver Disease

By Hilary Watson BSc

The liver is one of the most important and complicated organs in the body. It plays a role in digestion, metabolism and storage of many nutrients and it is also the site of detoxification of waste products. In all, the liver performs over 500 different essential tasks in the body and although it has an amazing ability to repair itself, once damage has exceeded the organ's ability to compensate, the effects are far reaching. The chart below gives a list of some of the roles of the liver in normal metabolism, as well as the associated symptoms that occur with liver failure.

	Role of the healthy liver	Associated symptom in liver disease
Protein metabolism	Synthesis of plasma proteins including albumin, transport proteins, coagulation proteins, acute phase proteins Urea synthesis (detoxification of ammonia)	Increased catabolism of proteins, reduced production of plasma proteins Increased peripheral utilization of branched chain amino acids Decreased urea production (reduced ability to detoxify ammonia which can lead to hepatic encephalopathy)
Carbohydrate metabolism	Glycogen metabolism and storage Blood glucose control (via metabolism of insulin and glucagon) Gluconeogenesis	Decreased glycogen synthesis by liver and skeletal muscle Glucose intolerance and insulin resistance Hypoglycemia due to inability to regulate blood glucose
Lipid metabolism	Synthesis of fatty acids, triglycerides and ketones Excretion of cholesterol and bile acids	Increased breakdown of fat Increased turnover of fatty acids Some ketogenesis In cholestatic liver disease, bile acid concentration in the gut is decreased which can inhibit fat digestion
Vitamin metabolism	Storage and/or activation of fat soluble vitamins A, D, E, K, Synthesis and/or activation of B-complex vitamins	Decreased uptake of fat soluble vitamins from the gut, decreased conversion of vitamins to their active forms, deficiencies of B-complex vitamins
Mineral metabolism	Storage of iron, zinc, copper, excretion of copper and manganese in bile	Zinc deficiency, copper and manganese toxicity
Digestive function	Synthesis of bile acids	Impaired ability to digest and absorb fat from the digestive tract
Detoxification function	Detoxification and excretion of ammonia, bilirubin, copper, steroid hormones	Development of hepatic encephalopathy as a result of decreased ability to detoxify, copper storage toxicity

Dietary Management of Dogs with Liver Disease

In broad terms, the dietary goals in liver disease are to support the liver by 1) supplying adequate energy and nutrients to avoid malnutrition, 2) correcting metabolic abnormalities and 3) reducing the liver's workload.

Energy Requirements

Adequate energy is required to maintain body weight and avoid the catabolism of body proteins to supply energy. The type and severity of liver disease influence a dog's energy requirements. Patients with chronic liver disease are generally hypermetabolic (see Appendix 4 in my cookbook for energy calculations for hypermetabolic dogs). Those with acute hepatic injury generally have increased protein and energy requirements as a result of tissue synthesis and repair, but their requirements are typically lower than hypermetabolic dogs. Most dogs with vacuolar hepatopathies need only maintenance energy intake however stress, inflammation, infection or other factors may increase energy requirements above maintenance. The goal should always be to provide enough energy to maintain ideal body weight, and enough protein to maintain a positive nitrogen balance. Calorie and protein intake should be monitored and adjusted as necessary. Owners should be involved in monitoring their dogs to ensure that any changes are noticed early.

	Chronic liver disease	Acute hepatic injury	Vacuolar hepatopathy
Energy requirements	Hypermetabolic calories (appendix 4)	Above maintenance but not as high as hypermetabolic	Maintenance but may require more calories with stress, inflammation, infection

Protein Requirements

Protein requirements in hepatic disease vary according to the type and severity of liver disease. Liver disease can increase the break-down of body proteins thus requiring a higher protein intake to maintain a positive nitrogen balance. Dogs with chronic hepatitis, acute hepatitis or ischemic liver damage are all likely to have a protein requirement significantly higher than minimum maintenance values. Dogs at risk of hepatic encephalopathy (HE) need a highly restricted protein intake but care needs to be taken to ensure that a positive nitrogen balance is always maintained. It is not enough to choose a diet that provides adequate protein, it is also critical to monitor food intake to ensure that adequate protein and energy are being consumed by the dog. If the dog is consuming too few calories to meet his increased energy needs, he will catabolize lean body tissues to supply the missing energy. This will result in the loss of lean tissue even if adequate protein is being provided by the diet. It is therefore important to monitor food intake to ensure that both protein and energy requirements are being met.

Meat Protein Versus Plant Protein

Proteins containing heme groups (ie red meats, organ meats) are generally considered to be more encephalogenic than proteins of plant or dairy origin. Dogs with liver disease fed diets based at least partially on dairy and plant proteins have been shown to have lower blood ammonia concentrations than those fed meat-based diets. These findings may relate to other aspects of these ingredients other than the protein – plant ingredients are higher in dietary fibre and the lactose in dairy ingredients may be beneficial as an energy source.

Soybean/tofu has been recommended for dogs with liver disease and it is generally well tolerated by most dogs. Soy protein has the advantage of being somewhat lower in methionine, an amino acid that accumulates in liver disease. However, soy protein can form hydrophobic peptides in the gut that bind to bile salts preventing their reabsorption and increasing the rate of loss in the feces. For this reason, soy-based recipes may not be ideal for managing dogs with impaired bile production or secretion, or impaired fat digestion or absorption. In these cases, other types of beans or nuts may be better options.

Fat Requirements

Fat has the advantage of providing energy-dense non-protein calories which can help to maintain body weight especially in anorexic patients. Fat is also highly palatable to dogs which helps to encourage food intake. However, high fat intake may not be appropriate for all dogs with liver disease. Dogs with vacuolar hepatopathies often have underlying problems with lipid metabolism, and they may be dealing with concurrent pancreatitis, inflammatory bowel disease or diabetes mellitus. Some forms of liver disease, ie biliary cirrhosis, bile duct occlusion etc, affect the production or transport of bile acids. Dogs with this type of liver disease can end up with steatorrhea (fatty stools) if fat intake is too high. So while high fat intake has many advantages and may be appropriate for many dogs with liver disease, it may not be appropriate in all cases. Having both high fat, moderate fat and low fat options allows veterinarians to choose the best option for each individual case.

Copper and Zinc

Certain breeds of dogs (ie Bedlington Terriers, West Highland White Terriers, Doberman Pinschers and others) are genetically predisposed to copper storage disease, a condition similar to Wilson's Disease in humans. In dogs with this disease, copper accumulates in the liver causing liver damage. Because copper is normally deposited in the liver and because excess copper is normally excreted from the body in bile, copper often accumulates in the livers of dogs with liver disease even if they don't technically have copper storage disease. With copper storage disease, copper accumulation in the liver is the primary disease and liver tissue is damaged as a result. In most forms of liver disease, copper accumulation in the liver is secondary to liver disease, generally because copper excretion in bile is impaired. In either case, copper restriction is advisable. Concurrent zinc supplementation is also recommended.

There are many benefits to zinc supplementation. Zinc has a protective effect against many hepatotoxic agents (including bromobenzene, acetaminophen, carbon tetrachloride, as well as copper). Doses of 50-100 mg of elemental zinc/day were well tolerated in dogs ranging in weight from 14-18 kg. Although blood zinc concentrations are not a reliable indicator of zinc deficiency, blood concentrations do accurately reflect systemic zinc toxicity. It is therefore recommended that serum zinc concentrations be measured before zinc supplementation is started and then again 7 days, 14 days, 2 months and 6 months after supplementation has been initiated. While zinc acetate, zinc gluconate and zinc sulfate can all be used, zinc acetate is believed to be best tolerated by dogs with liver disease.

Chicory Root Extract – Pre-biotic Fibre

At one time it was thought that a low residue, low fibre diet would help liver patients by reducing the generation of toxin in the gut. Investigations in humans with liver disease have since shown that diets high in fibre and plant proteins are beneficial for liver patients. Fermentation of fibre in the gut increases a patient's nitrogen tolerance and reduce the risks of HE. In particular, supplementation with pre-biotic fibre such as that found in chicory root extract, can reduce ammonia production in the gut, increase fibre fermentation and lead to the production of beneficial short chain fatty acids (SCFAs). Although SCFA were once thought to contribute to HE, they are no longer considered encephalogenic agents. SCFA are readily absorbed by colon cells and contribute to mucosal health. They lower the pH in the lumen which causes ammonia to be converted to ammonium which cannot be reabsorbed back into the dog's body. In this way, SCFAs and pre-biotic fibre help to improve nitrogen tolerance in dogs with liver disease.

Frequency of feeding

Frequent feeding of small meals (4-6/day) lowers the amount of nutrients or metabolites requiring hepatic processing at a single time, thereby imposing less metabolic demand on the liver. Multiple small meals a day can help to increase energy intake and prevent inappetance in liver patients.

Liver Recipes

Liver Recipe 1 (Very low protein, moderate fat)

	%	grams
Apple, with skin, no core	18.2	182
Blueberries	3.7	37
Brussels sprouts	3.8	38
Cod liver oil	0.5	5
Egg, hard-boiled	15	150
Kidney beans, canned	12.8	128
Pears, with skin, no core	16.3	163
Peas thawed from frozen	16	160
Safflower oil	0.5	5
Yogurt, plain, low fat	12.2	122
HILARY'S BLEND	1	10
	100	1000

Protein*	50 g/1000 kcal
Fat	35 g/1000 kcal
ME	87 kcal/100g
% ME protein	20%
% ME fat	31%
% ME CHO	49%
BCAA	9.40 g/1000 kcal
AAA	4.62 g/1000 kcal
BCAA:AAA	2.0 : 1
Copper	2.92 mg/1000 kcal
Zinc	43.78 mg/1000 kcal

* note this is below AAFCO healthy minimum

Liver Recipe 2 (Very low protein, low fat)

	%	grams
Apple, with skin, no core	36	360
Brussels sprouts	15.2	152
Cod liver oil	0.5	5
Lentils, boiled	12	120
Pears, with skin, no core	12.1	121
Peas thawed from frozen	18.1	181
Safflower oil	0.5	5
Turkey breast, cooked	4.6	46
HILARY'S BLEND	1	10
	100	1000

Protein	53 g/1000 kcal
Fat	17 g/1000 kcal
ME	76 kcal/100g
% ME protein	21%
% ME fat	15%
% ME CHO	64%
BCAA	8.57 g/1000 kcal
AAA	4.02 g/1000 kcal
BCAA:AAA	2.1 : 1
Copper	3.62 mg/1000 kcal
Zinc	49.35 mg/1000 kcal

Liver Recipe 3 (Low protein, moderate fat)

	%	grams
Apple, with skin, no core	24.5	245
Brussels sprouts	4.5	45
Cod liver oil	0.5	5
Egg, hard-boiled	25	250
Pears, with skin, no core	16.3	163
Peas thawed from frozen	16	160
Yogurt, plain, low fat	12.2	122
HILARY'S BLEND	1	10
	100	1000

Protein	56 g/1000 kcal
Fat	39 g/1000 kcal
ME	88 kcal/100g
% ME protein	22%
% ME fat	35%
% ME CHO	43%
BCAA	10.62 g/1000 kcal
AAA	5.32 g/1000 kcal
BCAA:AAA	2.0 : 1
Copper	2.69 mg/1000 kcal
Zinc	43.40 mg/1000 kcal

Liver Recipes cont'd

Liver Recipe 4 (Low protein, high fat)

	%	grams
Apple, with skin, no core	5.5	55
Carrots, raw	11.0	110
Catfish, farmed, cooked	19.3	193
Cheese, cheddar	8.5	85
Cod liver oil	0.5	5
Rice, cooked	43.9	439
Safflower oil	0.5	5
Salmon oil	0.9	9
Tofu, firm, w/Cal sulfate	8.4	84
HILARY'S BLEND	1.5	15
	100	1000

Protein	56 g/1000 kcal
Fat	50 g/1000 kcal
ME	150 kcal/100g
% ME protein	22%
% ME fat	45%
% ME CHO	33%
BCAA	10.65 g/1000 kcal
AAA	5.37 g/1000 kcal
BCAA:AAA	2.0 : 1
Copper	2.54 mg/1000 kcal
Zinc	38.38 mg/1000 kcal

Liver Recipe 5 (Moderate protein, moderate fat)

	%	grams
Apple, with skin, no core	18	180
Brussels sprouts	7.6	76
Catfish, farmed, cooked	12.6	126
Cheese, cheddar	7.5	75
Cod liver oil	0.3	3
Egg noodles, cooked	8	80
Pears, with skin, no core	16.3	163
Peas thawed from frozen	16	160
Safflower oil	0.5	5
Yogurt, plain, low fat	12.2	122
HILARY'S BLEND	1	10
	100	1000

Protein	58 g/1000 kcal
Fat	43 g/1000 kcal
ME	111 kcal/100g
% ME protein	23%
% ME fat	39%
% ME CHO	38%
BCAA	11.05 g/1000 kcal
AAA	5.25 g/1000 kcal
BCAA:AAA	2.1 : 1
Copper	2.36 mg/1000 kcal
Zinc	36.17 mg/1000 kcal

Liver Recipe 6 (Moderate protein, low fat)

	%	grams
Brussels sprouts	19	190
Chickpeas, canned	36.9	369
Cod liver oil	0.25	2.5
Peas thawed from frozen	21.8	218
Soybeans, boiled	8.6	86
Yogurt, plain, low fat	12.2	122
HILARY'S BLEND	1.25	12.5
	100	1000

Protein	60 g/1000 kcal
Fat	19 g/1000 kcal
ME	95 kcal/100g
% ME protein	24%
% ME fat	17%
% ME CHO	59%
BCAA	9.91 g/1000 kcal
AAA	5.10 g/1000 kcal
BCAA:AAA	1.9 : 1
Copper	3.99 mg/1000 kcal
Zinc	52.37 mg/1000 kcal

Liver Recipes cont'd

Liver Recipe 7 (Moderate protein, high fat)

	%	grams
Apple, with skin, no core	18	180
Brussels sprouts	19	190
Butter	1	10
Cheese, cottage, low fat	37.6	376
Cod liver oil	0.5	5
Egg noodles, cooked	16	160
Peanut butter, smooth	6.4	64
HILARY'S BLEND	1.5	15
	100	1000

Protein	66 g/1000 kcal
Fat	46 g/1000 kcal
ME	117 kcal/100g
% ME protein	26%
% ME fat	41%
% ME CHO	32%
BCAA	12.35 g/1000 kcal
AAA	6.71 g/1000 kcal
BCAA:AAA	1.8 : 1
Copper	2.98 mg/1000 kcal
Zinc	45.94 mg/1000 kcal

Liver Recipe 8 (Moderate protein, high fat)

	%	grams
Apple, with skin, no core	5.5	55
Carrots, raw	11.0	110
Cheese, cheddar	8.5	85
Chicken liver, cooked	20.3	203
Cod liver oil	0.5	5
Rice, cooked	39.0	390
Safflower oil	0.5	5
Salmon oil	0.9	9
Tofu, firm, w/Cal sulfate	12.3	123
HILARY'S BLEND	1.5	15
	100	1000

Protein	66 g/1000 kcal
Fat	49 g/1000 kcal
ME	154 kcal/100g
% ME protein	26%
% ME fat	44%
% ME CHO	30%
BCAA	12.83 g/1000 kcal
AAA	6.59 g/1000 kcal
BCAA:AAA	1.9 : 1
Copper	3.03 mg/1000 kcal
Zinc	41.32 mg/1000 kcal

Each month, we'll review one nutrition research study published within the last 2 months. These reviews won't be limited to canine nutrition. We may review human, equine, livestock and zoo animal nutrition research if we find it interesting and relevant. The common denominator is that each study will be recently published in a peer-reviewed scientific journal and they will all provide new insight into some concept of nutrition.

Research Study of the Month

By Laura Scott MSc

Title of study:	*The feline dietary iodine requirement is lower than the 2006 NRC recommended allowance*
Authors:	*K. J. Wedekind, M. E. Blumer, C. E. Huntington, V. Spate and J. S. Morris*
Journal:	*Journal of Animal Physiology and Animal Nutrition*
Issue:	*2010 August 94:527-539*
Species:	*Cat*
Link:	*http://www3.interscience.wiley.com/journal/122681601/abstract*

Background information

Iodine is a required nutrient which is a major constituent in thyroid hormones. This is turn plays a role in regulation of metabolic rate, and in cell growth and differentiation during development.

The National Research Council (NRC) publishes their Nutrient Requirements books for various species. The requirements in these books are determined by a committee of qualified experts who review the published literature for the species in question. In 2006 the NRC published Nutrient Requirements of Dogs and Cats. The Association of American Feed Control Officials (AAFCO) is a U.S. based non-regulatory group that publishes model regulations, a list of approved feed ingredients, and standards for pet foods in the United States. The nutrient standards from AAFCO are determined by an expert sub-committee.

There is a discrepancy between the AAFCO (0.35 mg I/kg) and NRC (1.4 mg I/kg) recommended allowances for iodine for cats. The authors of this paper report that the studies used to determine the NRC iodine requirement failed to meet key criteria for defining nutrient requirements.

Purpose of this study and study design

The goal of this study was to determine the iodine requirement of adult cats. Forty-two adult cats were fed a low iodine basal diet for 4-7 weeks and then were randomly assigned to one of 7 experimental diets. The diets were identical except for the level of iodine supplied (0.15 mg/kg – 9.2 mg/kg). Cats remained on the experimental diet for one year. Iodine concentration in serum, urine, feces, complete blood count, serum chemistries and serum thyroid hormone profiles were all analyzed.

Study results

No significant effects of dietary iodine were seen in bodyweight, serum chemistries, thyroid hormone profiles or food intake. Fecal and urinary iodine were linear functions of iodine intake. The minimum urinary iodine concentration that defines iodine adequacy in humans was seen in the diets that supplied 0.46 mg I/kg. A significant decrease in free thyroxine was seen at the highest dietary iodine level (9.2 mg I/kg). Based on the daily urinary I concentration and scintigraphy, the iodine requirement derived from this experiment was 0.46 mg I/kg diet. This number is similar to the requirements for other species, such as humans and dogs.

My thoughts

Nutrient requirements are studied and define so we can be sure of providing our animals with optimal nutrition. Feeding too little of a nutrient and causing a deficiency or too much and providing an excess can both have negative impacts on health. If nutrient requirements are not properly determined it can be difficult to properly formulate a diet. The requirement for iodine which was determined in this study was lower than the NRC minimum requirement and higher than the AAFCO minimum requirement. As these are both used

as the basis for formulating diets, and since diets are often formulated in excess of the minimum requirement, it is important to be careful if adding any supplements containing iodine to your cat's diet. Products like seaweeds are naturally high in iodine and adding too much may provide your cat with an excess of iodine, leading to signs of toxicity. Ensuring your cat's diet is properly formulated and consulting a pet nutritionist before making changes is the best course of action.

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