



# Canine Nutrition NEWSLETTER

March 2010



## In this Issue

The loss of lean muscle tissue in kidney disease is usually caused by metabolic acidosis. The metabolism of nutrients, particularly proteins and fats, creates acid in the body. The kidney is responsible for excreting excess acid from the body in urine. In kidney disease, acid excretion is impaired often leading to metabolic acidosis. The emergency response to metabolic acidosis is to activate body protein breakdown. Correcting acidosis by feeding an alkalizing buffer such as potassium citrate or potassium bicarbonate is the best way to prevent muscle break-down and the loss of lean body tissue in dogs with kidney disease. For more information, see:

***Nutritional Management of Cachexia in Dogs with Kidney Disease***  
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Cachexia in cancer patients is mediated by cytokines produced both by the host animal and by the tumor itself. Nutrient metabolism is altered resulting in lean tissue loss, weight loss, and in some cases, inappetance. Appropriate dietary management can not only reverse cachexia, it can also be specifically designed to support the host without feeding the tumor. For more information, see:

***Nutritional Management of Cachexia in Dogs with Cancer***  
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When most owners think of skin supplements, they think of oils. Yet certain B-vitamins play a critical role in supporting the integrity of the epidermal skin barrier. Niacin (vitamin B1) and pyridoxine (vitamin B6) both are involved in the production of skin ceramides – the molecules that act as “mortar” binding epidermal skin cells together and preventing bacteria and allergens from entering the dog’s body. For more information, see:

***Strengthening the Skin Barrier with B-vitamins***  
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***The Successful Nutritional Management of Canine Lymphoma: Case Report for “Bailey” Allin*** ..... [Page 11](#)

Several hundred children are treated for rickets each year in the United Kingdom and 50% of the adult population has insufficient levels of vitamin D in the winter and spring. This is a serious public health issue. This January 2010 review focuses on the vitamin D status of people in the United Kingdom, recommended daily intakes, risk factors for deficiency, as well as explaining how vitamin D status can be assessed and deficiencies treated. For more information, see:

***Research Study of the Month***  
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# Nutritional Management of Cachexia in Dogs with Kidney Disease

By Hilary Watson BSc

Cachexia is defined as physical wasting with loss of muscle mass caused by disease. Dogs with advanced cancer, cardiac disease and kidney disease may become cachectic. Anorexia (lack of appetite) and cachexia often occur together. Cachexia is not the same as starvation. A healthy dog's body can adjust to starvation by slowing down its use of nutrients, but in dogs with cachexia, the body does not make this adjustment. Muscle wasting can occur even with increased food intake.

The underlying mechanism for cachexia in kidney patients is not the same as that for cancer or cardiac disease. In dogs with chronic kidney disease, cachexia is usually caused by metabolic acidosis. Correcting the acid-base balance will generally resolve cachexia in kidney patients.

Dogs with kidney disease have an impaired ability to excrete phosphorus from the body, so phosphorus accumulates in the dog's body. This is why we restrict phosphorus intake in dogs with kidney disease. The balance between calcium and phosphorus is carefully maintained in the body. When phosphorus levels are too high relative to calcium, parathyroid hormone is produced to try to correct the imbalance. In kidney patients reduced excretion of phosphorus leads to an imbalance in the body's calcium to phosphorus ratio, which stimulates the production of parathyroid hormone. This condition is known as "renal secondary hyperparathyroidism". Hyperparathyroidism can lead to soft tissue mineralization, loss of bone mass and it accelerates the progression of kidney disease. We can double the life expectancy of a dog with renal disease by severely restricting the dog's phosphorus intake, thereby maintaining a normal balance between calcium and phosphorus in the dog's body.

The impaired ability of the kidneys to excrete phosphorus has consequences for the dog's acid-base balance. Acid is produced through normal nutrient metabolism, particularly the metabolism of fats and proteins. In normal dogs, much of this acid is lost from the body in urine as phosphoric acid (figure 1). When urinary phosphorus excretion is reduced, the excretion of acid is reduced. This leads to the accumulation of acid in the body, a condition known as metabolic acidosis.

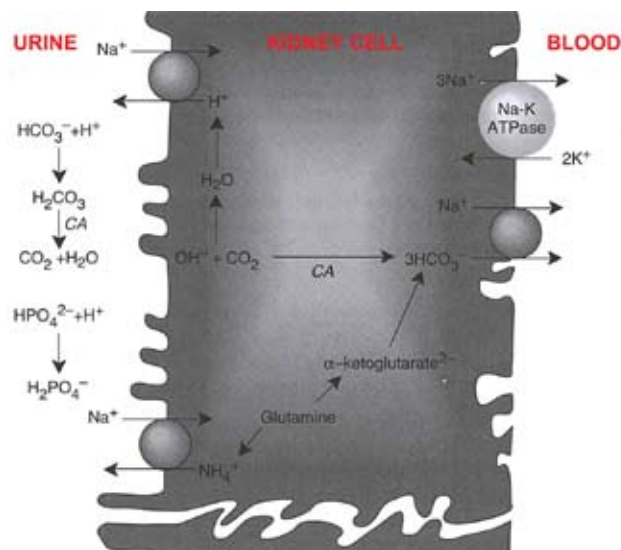


Figure 1 – the kidney is responsible for maintaining acid-base balance in the body

**Acid (H<sup>+</sup>) is produced in the body through normal metabolism. The kidney cells are responsible for excreting excess acid from the body in urine. This excess acid either reacts with bicarbonate to form CO<sub>2</sub> plus water, or it is bound to phosphorus and excreted as phosphoric acid. When phosphorus excretion is impaired in kidney disease, an alternative pathway involving the amino acid glutamine is used. In this case, acid is bound to NH<sub>3</sub> and is excreted as ammonium (NH<sub>4</sub>). The glutamine used in this process is derived from the break-down of body proteins including muscle.**

Metabolic acidosis has a serious impact on health. Enzymes, cellular reactions, transport molecules, metabolic processes all proceed most efficiently within a very narrow range of optimal pH. The normal pH range for dog blood is 7.31 – 7.42. A pH that is even slightly outside this range has serious metabolic consequences for the dog. Because pH balance is so important for body function, there are several emergency mechanisms that are designed to correct imbalances in acid-base status.

The primary emergency metabolic response to metabolic acidosis is the activation of a protein degradation pathway so that the amino acid glutamine is released from muscle. Glutamine is an amino acid that has an extra ammonia (NH<sub>3</sub>) group which is combined with acid to produce ammonium (see figure 2). Protein degradation to release glutamine is the body's normal emergency response to excess acid in the body. Mitch et al (1994) studied the phenomenon in rats and stated "the loss of lean body mass in kidney

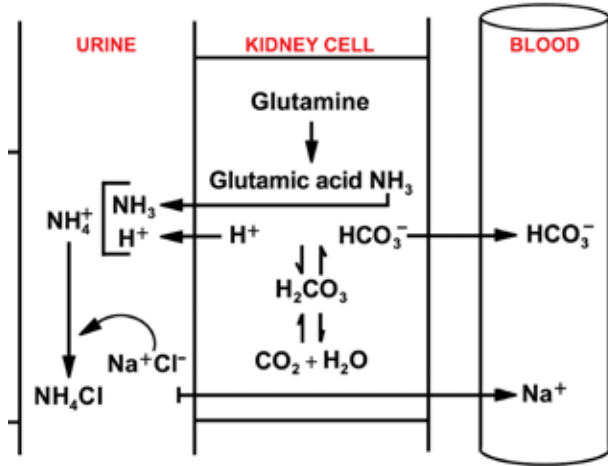


Figure 2

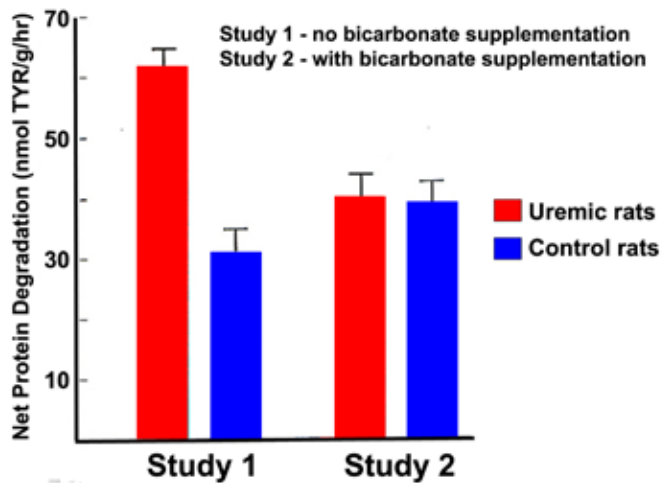
*When excessive acid accumulates in the body of a dog with kidney disease, muscle is broken down to yield the amino acid glutamine, which is converted into glutamic acid, thereby releasing ammonia (NH<sub>3</sub>). This ammonia is combined with acid (H<sup>+</sup>) into ammonium (NH<sub>4</sub><sup>+</sup>) which is lost from the body as ammonium chloride (NH<sub>4</sub>Cl). Bicarbonate (HCO<sub>3</sub><sup>-</sup>) is released back into the blood, helping to restore normal acid-base balance.*

disease appears to be a consequence of a normal metabolic response to acidosis that persists until acidosis is corrected". This metabolic response leads to whole body protein break-down, impaired protein synthesis (which can lead to reduced levels of the blood protein albumin), as well as resistance to the normal muscle-building effects of insulin and growth hormone. Activation of this catabolic pathway leads to muscle wasting and weight loss.

Protein break-down yields glutamine for acid excretion, but it also releases all the other amino acids from the muscle. The release of these amino acids into the blood is equivalent to feeding the dog a high protein diet. In other words, although the body is breaking down body proteins to yield glutamine for acid excretion, the by-product of this process is the release of all the other amino acids into the dog's blood stream, which contribute to the symptoms of renal disease (i.e elevated blood urea nitrogen, azotemia, uremia). This makes the dog feel ill and can lead to the development of anorexia. The dog stops eating because when he eats, acid accumulates in his body, which causes body proteins to be broken down, which causes a build-up of uremic toxins in the blood, which makes the dog feel sick. It's a negative feedback loop that will make the dog reluctant to eat future meals.

Since many dogs with kidney disease are also dealing with hypertension, additional sodium is contraindicated for dogs with kidney disease. For this reason, sodium bicarbonate is not a good choice for dogs with kidney disease. However, potassium citrate and potassium bicarbonate are both excellent buffers for kidney

Figure 3 - Correcting acidosis prevents muscle break-down in rats with experimentally induced kidney disease (May et al 1987)



*In 1987, May et al measured net protein degradation (ie how much muscle was broken down) in healthy rats (blue bars) and in rats with experimentally induced kidney disease (red bars). In study 1, there was no dietary intervention. The researchers simply measured the difference in muscle degradation and noted that the rats with kidney disease (blue bars) lost a great deal more muscle mass than did healthy rats (red bars). This study showed that without dietary intervention, acid accumulation in the body of kidney patients leads to muscle protein degradation.*

*In study 2, May et al supplemented both groups of rats with sodium bicarbonate. Bicarbonate is a buffer - it neutralizes acid in the body. The researchers found when they supplemented with bicarbonate, muscle degradation was reduced in the rats with kidney disease. There was no dif-*

*ference in the rate of muscle degradation between normal rats and those with kidney disease when both groups were supplemented with a buffer. In other words, muscle wasting in kidney disease was prevented by neutralizing excess acid by supplementing with a buffer.*



patients. Potassium often becomes depleted in kidney patients, so supplementing with a buffer that contains potassium is beneficial to the kidney patient.

I recommend that all dogs with kidney disease be supplemented with potassium citrate at a rate of 150 mg per kilogram of body weight per day. Alternatively potassium bicarbonate can be used at the same rate. Feeding a buffer will neutralize excess acid, prevent metabolic acidosis, prevent the loss of lean body mass, and will prevent anorexia, azotemia and other renal symptoms caused by body protein catabolism.

Cachexia associated with kidney disease is a result of the body's normal emergency response to metabolic acidosis. Correcting acidosis by supplementing with a buffer such as potassium citrate or potassium bicarbonate will help to prevent the muscle loss and inappetence commonly associated with renal disease.



"Tasha"

## Testimonial

*Tasha my 7 year old Sheltie has suffered from digestive problems for years. Thanks to Hilary Watson's cookbook "Complete & Balanced: 101 Healthy Home-made Meals for Dogs" and HILARY'S BLEND supplement, Tasha is now enjoying and thriving on very digestible and delicious home-cooked meals. The recipe book is meticulously put together, easy to follow and definitely worth purchasing. Tasha suggests you try her favourite – Recipe R10 – Beef and Potatoes on page 78.*

*Fay & Tasha,  
Richmond, BC*

# Nutritional Management of Cachexia in Dogs with Cancer

By Elizabeth Pask PhD (candidate)

Cancer is one of the hardest words to hear from your veterinarian. As our health management and nutrition improve our dogs are living longer and thus we are hearing the diagnosis of cancer more and more. Unfortunately by the time the cancer is diagnosed treatment is often not an option. However there maybe some nutritional changes we can make to help your dog maintain his or her quality of life for longer. The number one reason why cancer patients are euthanized is a loss of appetite. We often assume that a loss of appetite is related to severe pain or the dog “doesn’t want to live anymore”. This may not be true in all cases. Sixty eight percent of cancer patients will experience weight loss of more than 5% of their body weight. There are a variety of metabolic changes that take place in the dog’s body when a tumor starts growing. This collection of changes results in a syndrome called “cancer cachexia”.

## What is Cachexia?

The word “cachexia” comes from the Greek term meaning “bad condition”. The syndrome is seen in patients with cancer but it can also be seen in patients with chronic heart failure and kidney failure. Patients with cachexia have lethargy, loss of appetite and muscle wasting. In addition they often have a poor response to treatment and reduced survival time. Previously it was thought that this syndrome was caused by a lack of food. However the metabolic changes seen in cachectic patients do not match the changes seen in patients with starvation.

## Table: Differences Between Cachexia and Starvation

The biggest thing to remember about the difference between cachexia and starvation is that we can carefully re-feed a victim of starvation and achieve a full recovery. We cannot do that with cachexia patients. You cannot reverse cachexia simply by feeding more food. Why not? Well the answer lies in how the cancer’s presence affects your dog’s metabolism.

## How Tumors Affect Carbohydrate Metabolism

	Cachexia	Starvation
Appetite	↓	↑
Energy expenditure (while resting) (REE)	↑	↓
Amount of skeletal muscle	↓	No change
Amount of body fat	↓	↓
Size of liver	↑	↓
Insulin level	↑	↓

Adapted from QJMED, 2005 98:779

Tumor cells steal energy from the host. The tumor cell would prefer to use blood glucose as its





energy source. As the tumor cell metabolizes the glucose it produces lactic acid as a byproduct. Lactic acid is then released in to the blood stream of the dog. The dog's liver is then forced to use energy to metabolize the lactic acid. The end result is a net energy gain by the tumor and a net energy loss by the dog.

In addition to changes in carbohydrate metabolism there are also changes in protein and fat metabolism.

## How Tumors Affect Protein Metabolism

All protein is made up of amino acid building blocks. Amino acids bond together forming chains. These chains then fold to form a more complex structure. The complex structures then join to form muscle protein and other proteins in the body. The sequence of amino acids is important. If the correct number and types of amino acids are not available then the body will start breaking down protein stores in the muscle and liver in order to find and use the correct amino acid. When an animal has cancer the tumor will snatch some amino acids and use them but it won't take all types of amino acids. The tumor has now created an imbalance in the amino acids in the dog. The dog's body will now have to use energy to break down its protein stores in order to make the proteins that it needs to survive. When designing diets we can limit the amounts of amino acids that the tumor likes and maximize the amino acids that the dog needs thus starving the tumor.

## How Tumors Affect Fat Metabolism

In a normal animal, fat from the diet is metabolized for energy. Excess dietary fat is stored for later use. In times of starvation and cachexia, fat deposits are broken down and used for energy. In cachexia excess fat is not stored it is used by the body for energy. Tumors have difficulty using fat as an energy source so the high utilization of fat stores may be the body compensating for the tumor taking substantial amounts of energy from the body. However, this change in fat metabolism can have long term damaging effects on the body which can result in suppression of the immune system. The suppression of the immune system leads to decreased survival time and an increased susceptibility to infection.

## Cachexia and Cytokine Production

The metabolic dysfunction in protein, fat and carbohydrate metabolism leads to an increase in whole body metabolism (hyper-metabolism). This hyper-metabolism results in muscle wasting and a general inefficiency in nutrient utilization by the dog's body. What is to blame for this metabolic dysfunction? We can blame the tumor but cachexia often remains after tumors are removed. The real culprit is the combination of the tumor's presence and body's response to the tumor's invasion. The tumor's presence causes an increase in the number of cell messengers called cytokines. Cytokines are small molecules released by immune cells. Their job is to modify the metabolism of cells in their immediate vicinity.

During cachexia several pro-inflammatory cytokines are produced by the body (see figure 1), namely IL-1 (interleukin 1), IL-6 (interleukin 6), and TNF (tumor necrosis factor). In addition to the cytokines that the body produces the tumor also produces PIF (proteolysis inducing factor) and LMF (lipid mobilizing factor). PIF and LMF are small molecules that are released into the blood from the tumor and they cause an increase in muscle and fat breakdown. It is believed that these molecules are responsible for much of the metabolic dysfunction that ultimately causes the cachexia. In small doses and in confined areas the pro-inflammatory cytokines are powerful tools in fighting infection. However in large doses over the whole body pro-inflammatory cytokines have been linked to a variety of chronic illness including cachexia. Often the negative effects of the cytokines are more damaging to the dog than the effects of the disease itself.

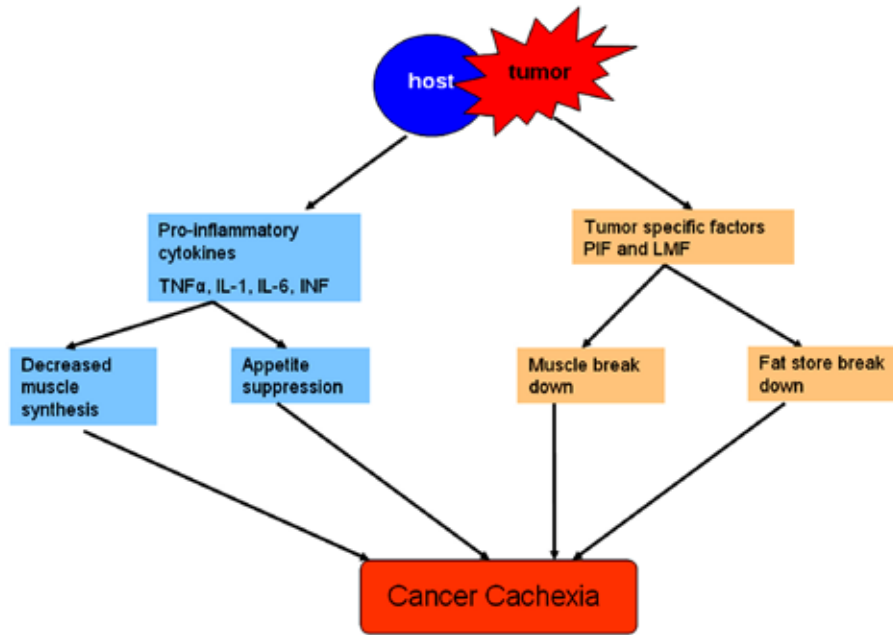


Figure 1 Cancer cachexia is caused by pro-inflammatory cytokines that are produced both by the dog (host) and by the tumor. (adapted from QJMED 2005, 98:779)

So what can be done? Well as mentioned above we can limit the amount of carbohydrates in the dog's diet to limit the amount of energy we are supplying to the tumor. We can also provide appropriate levels of protein with the right amino acid profile to prevent the tumor from stealing amino acids from the host. We can also use fat as the primary energy source because the tumor can't utilize fat for energy. But we are still stuck with the troublesome cytokines. Currently much nutritional research is focused on the potential for certain nutrients to down-regulate cytokine production. EPA (eicosapentanoic acid), an omega-3 fatty acid found in fish oil, is showing particularly promising results.

## EPA, Cachexia and the Fight Against Cytokines

EPA is a long chain polyunsaturated fat found in fish oil with the best source of EPA being salmon oil. In a recent study, human patients with advanced pancreatic cancer and cachexia were given a supplement containing 2.2 grams of EPA (approx. 31mg/kg body weight/day). Prior to supplementation all patients were losing weight at an approximate rate of 2.9 kg/day. After 3 months of supplementation patient weight stabilized and no further weight loss was seen. In a similar study cancer cachexia patients gained weight after 3 weeks of supplementation and continued to gain weight throughout the study. In addition the study reported that patients had an increase in appetite, a reverse of muscle wasting, a decrease in IL-6 and PIF. A reduction in the inflammatory cytokines results in an increase in muscle and a decrease in fat breakdown.

In a similar study, cachetic rats were fed a diet containing 1g of fish oil/kg of diet (approx. 10mg/rat/day). Rats fed fish oil had a significant decrease in the pro-inflammatory cytokines IL-6 and TNFα when fed compared to the control animals fed a normal diet.

In a study in dogs with cachexia due to heart disease, dogs were fed a diet containing 27 mg/kg/day of EPA for eight weeks. Following the 8 weeks of feeding the dogs had a significant reduction in the pro-inflammatory cytokine IL-1 however there was no change in the amount of TNFα.





## Dietary treatments

There is some question as to the optimal amount of EPA that should be in the diet of a cancer patient. There is a limit as to how much EPA can be added to a commercial pet food. High levels of EPA cannot be added to the surface of kibble since omega-3 fatty acids are unstable and prone to rancidity when exposed to air. As well, the manufacturing constraints of extrusion prohibit the production of extremely low starch kibble (ie less than 5% of calories coming from carbohydrates) which is ideal for dogs with tumors. While canned diets are an option, again the instability of omega-3 fatty acids makes the canning process less than ideal. Home-made diets on the other hand do allow for the inclusion of very high levels of EPA from salmon oil. A home-made recipe can easily be formulated to deliver less than 5% of calories from carbohydrates, while supplying adequate protein with an appropriate amino acid balance, and delivering the majority of calories from fat, effectively limiting the nutrients available for the tumor, and maximizing the nutrients feeding the host. This type of recipe can reverse cachexia in the dog, leading to the return to normal body weight and normal metabolism.

While reversing cachexia does not cure the dog of cancer, it may significantly extend the dog's life expectancy and dramatically improve the dog's quality of life.

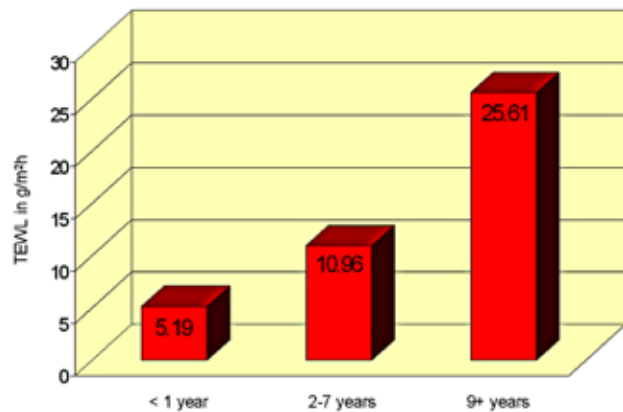


# Strengthening the Skin Barrier with B-vitamins

By Hilary Watson BSc

When most owners think about skin supplements, they generally think about oils. While it is true that oils can improve the luster of a dog's coat, certain B-vitamins also play a critical role in promoting skin health.

The skin is an important barrier between the dog's body and the external environment. Healthy skin prevents bacteria on the skin surface from entering the dog's body and the skin barrier prevents moisture from inside the dog's body being lost to the environment. Dogs with a weakened skin barrier have dry, flaky skin and are more susceptible to skin infections.



Adrian Watson, Tim Fray, Sally Beyer & Peter Markwell, 2001

Figure 1 – Trans-epidermal water losses (TEWL) increase as a dog ages.

The integrity of the skin barrier can be evaluated by measuring trans-epidermal water loss (TEWL). TEWL is water lost from the surface of the dog's skin. With healthy skin, very little water is lost from the skin surface. With unhealthy skin, moisture lost from the skin results in dry, flaky skin. This increases shedding and predisposes the dog to skin infections. Measuring TEWL is therefore good way of assessing the health of the skin barrier. A low TEWL indicates a healthy skin barrier.

A study done at the Waltham Centre for Pet Nutrition in England (Watson et al, 2001) showed that even in healthy

dogs, TEWL increases as a dog ages (figure 1). Dogs who were 2-7 years old lost twice as much water from their skin surface as dogs who were less than a year old. Dogs over 9 years of age lost five times more water from their skin surface than did the youngsters. This study showed that even in healthy dogs, the skin barrier weakens as the dog ages.

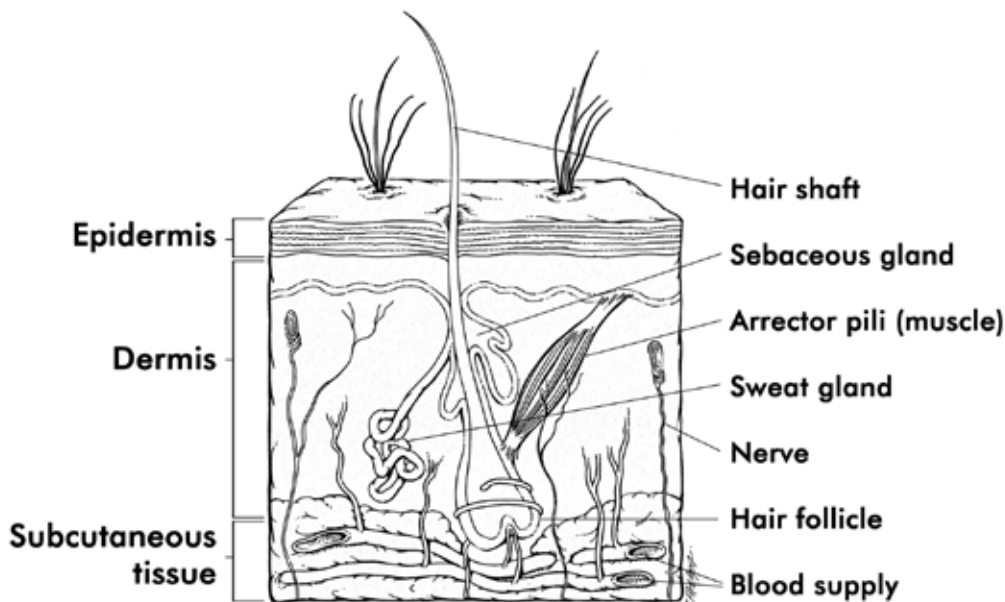


Figure 2 – The anatomy of dog skin





Figure 2 shows the structure of a dog's skin. The hair follicles, sebaceous glands and sweat glands are located in the dermis layer. The outermost layer is called the epidermis. It is this layer that forms the barrier between the dog's body and the external environment.

Figure 3 shows an enlargement of the epidermis layer. Skin cells are produced at the base of the epidermis inside the dog's body. As new skin cells are produced at the base, older cells are pushed outwards towards the surface. At the skin surface, the cells flatten, die and are shed as dandruff. This process is called desquamation.



Figure 3 Anatomy of the epidermis

In a healthy dog, desquamation takes about three weeks. With skin disease, the process is greatly accelerated. For example, in humans with psoriasis, desquamation takes only 7 days. The dry scaly skin of psoriasis is caused by skin cells dying 3 times faster than healthy skin.

The weakest link in the skin barrier is not the skin cells themselves, but rather is the space between skin cells. Think of the epidermis as a brick wall. The skin cells are the bricks. Holding the bricks together is the "mortar". Bacteria and allergens are more likely to travel into the dog's body through the mortar than through the skin cells. In the case of the epidermis, the mortar is made up of lipids and ceramides. The integrity of the skin barrier is therefore dependent on the integrity of the lipids and ceramides holding the skin cells together.

This is where certain B-vitamins play an important role. Another study at the Waltham Centre (Watson et al, 2001) showed that supplement with niacin (B1) and pyridoxine (B6) increased skin ceramide production (see figure 4). Not only do each of these vitamins increase ceramide production, but together they have a synergistic effect. Supplementing these two vitamins at levels well above AAFCO will strengthen the skin barrier by enhancing the production of ceramides in the epidermis.

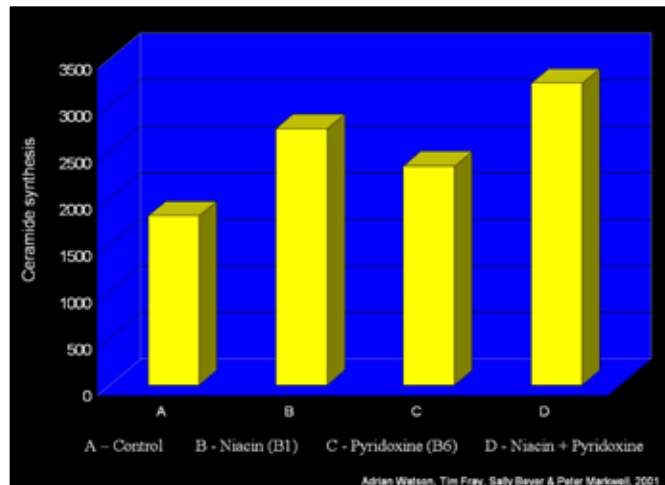


Figure 4 - Skin ceramide production can be enhanced by B-vitamin supplementation

The AAFCO Nutrient Profiles for Dogs specifies how much of each nutrient a dog needs for health. These profiles are based on research done in young healthy dogs. The AAFCO minimum for niacin is 3.3 mg per 1000 kcal, and for pyridoxine 0.29 mg per 1000 kcal. Niacin and pyridoxine above these values may be beneficial to the skin of older dogs and dogs with sensitive skin.

The recipes in the Complete & Balanced cookbook provide approximately 30 mg of niacin per 1000 kcal and 3 mg of pyridoxine per 1000 kcal – about 10 times more than the AAFCO minimum. Owners report healthier coats, less shedding, less dandruff and these improvements are typically seen within 7-10 days of changing to home-cooking.

# Successful Nutritional Management of Canine Lymphoma

## Case Report for “Bailey” Allin

Bailey is an 8.5 yr old M/N Golden Retriever who came down with generalised lymphadenopathy (prescapular LN 7 cm x 4 cm in size), and was diagnosed with multicentric lymphoma by FNA and pathology. At the onset of his cancer he was 78lbs and a body condition of 3/5 (ideal). He had no other illness at that time, except underlying atopy (which was controlled with serum allergy injections for previous 2 yrs.) and gastric hypomotility (controlled with Metoclopramide 5 mg q12h). He was borderline anaemic, but otherwise non-clinical for lymphoma.



“Bailey”

He started on the Madison-Wisconsin chemotherapy protocol November 11, 2009, under the guidance of Dr. Kevin Finora. Over the next 2 weeks he developed cancer cachexia, lost about 8 lbs and went to a body condition score of 1.5-2/5. He looked like a skeleton in Golden Retriever fur. He usually maintained on 2.75 cups/day of Hills Prescription Diet j/d. I had increased him to 6 cups daily, plus 2 eggs and Hills Prescription Diet n/d canned. He still lost 4lbs per week. He had no energy, was not playful and seemed depressed. His lymphadenopathy responded within one week of treatment and his lymph nodes returned to normal. At this point, he became anorexic and was fading away.

I started cooking a custom home-made recipe by Hilary Watson on November 28th, 2009 (see recipe on next page) and, although transitioning slowly, Bailey had more energy within 3 days of starting the new diet. Over the next 4 weeks this dog put all his former weight back on and an extra 5 lbs. His temporal muscles filled back out to normal, and he returned to a happy, active, spunky golden retriever with a coat that has never been this shiny and smooth.

Dr. Finora could not believe that the initially progressive anaemia of chronic disease was no longer progressive and has even improved to the point where it is almost back to normal not even a month into the diet. He says that normally cancer dogs on chemotherapy do not regenerate until the chemotherapy is completed.

I truly feel that Bailey would have been euthanised due to quality of life and wasting away towards the end of November had he not had this nutrition change and dramatic response. I owe Hilary a lot for helping my dog get back to his normal active, happy self.

Submitted by:  
Dr. Ines Allin D.V.M.  
Bracebridge Animal Hospital  
Bracebridge, Ontario.





# Recipe for "Bailey"

## CUSTOM RECIPE DETAILS

Date: 26-Nov-09  
 Clinic: Bracebridge Animal Hospital  
 Dr: Ines Allin  
 Client: Ines Allin  
 Dog: Bailey

INGREDIENTS (lymphoma reci	%	grams	Approx.
Almonds	4.7	47	1/2 cup ground
Butter	7.1	71	5 tbsp
Catfish, farmed, cooked	27.2	272	390 g raw
Cheese, cheddar	5.7	57	1/2 cup shredded
Cod liver oil	0.9	9	2 tsp
Eggs, hard-boiled	20.0	200	4 large
Liver, beef, cooked	25.0	250	350 g raw
Safflower oil	2.7	27	2 tbsp
Salmon oil	2.7	27	2 tbsp
HILARY'S BLEND*	4.0	40	4 scoops
	<b>100.0</b>	<b>1000</b>	

\*formerly THE BALANCER supplement

## ENERGY

Metabolizable energy: 275 kcal/100g  
 % ME from protein: 24%  
 % ME from fat: 71%  
 % ME from carbohydrate: 5%

**Current weight of dog:** 35.0 kg

<b>Feeding Guides:</b>	kcal/day	grams/day	days 1000g will last	Protein (g/kgBW/day)	EPA (g/day)
Average adult:	1367	<b>497</b>	2.0	2.4	2.1
Hypermetabolic adult:	1799	<b>654</b>	1.5	3.1	2.8

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: *Diagnosis and management of vitamin D deficiency*

Authors: *Simon H.S. Pearce and Tim D. Cheetham*

Journal: *British Medical Journal*

Issue: *Vol. 340, 142-147, January 2010*

Species: *Humans*

Link: [http://www.bmj.com/cgi/content/extract/340/jan11\\_1/b5664](http://www.bmj.com/cgi/content/extract/340/jan11_1/b5664)

## Background information

Vitamin D is a group of fat soluble prohormones. The group is made up primarily of vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol), as well as their metabolites. Vitamin D can be obtained from the diet or from sun exposure. Vitamin D plays an important role in calcium and phosphorus metabolism and is required for normal bone growth and maintenance. Deficiency will lead to rickets in children and osteomalacia in adults. In addition, a number of other conditions such as cancer, autoimmune disorders and metabolic syndrome have been associated with low vitamin D levels. There is an increased risk of vitamin D deficiency in northern regions as there is less exposure to sunlight.

## Purpose of this study and study design

Several hundred children are treated for rickets each year in the United Kingdom and 50% of the adult population has insufficient levels of vitamin D in the winter and spring. This is a serious public health issue and the authors of this study wanted to present a clinical review of the published articles on Vitamin D. They gave priority to more recently published works, as well as those that were systemic reviews, meta-analyses, and clinical guidelines. This review focuses on the vitamin D status of people in the United Kingdom, recommended daily intakes and risk factors for deficiencies. In addition, it looks at how deficiency can be determined and treated.

## Study results

In fair skinned people, 20-30 minutes of sun exposure 2-3 times per week is sufficient to meet vitamin D requirements, however much of North America and western Europe is too far north to get adequate UVB rays 6 months of the year resulting in 50% of the adult population in the UK being vitamin D insufficient and 16% being severely deficient. There are number of additional risk factors for vitamin D insufficiency including pigmented skin, strict sunscreen use, being elderly, obese or institutionalized and a vegetarian diet. Adults with vitamin D deficiency will present with pain and proximal muscle weakness, and may have low bone density or osteopenia and secondary hyperparathyroidism. Assay of serum 25-hydroxyvitamin D is the best way to determine vitamin D status. An increased risk of mortality and diabetes, cancer and cardiovascular disease were associated with vitamin D insufficiency in several studies.

Treatment of vitamin D deficiency is typically with high doses of vitamin D initially and then a maintenance dose for life, while insufficiency can be corrected with a maintenance dose. In addition, increased public awareness and fortification of foods may be necessary to prevent further cases of vitamin D insufficiency.

## My thoughts

As a fat soluble vitamin, vitamin D can cause toxicity at high levels. However, this study shows that insufficiency is a far greater risk in the human population. Although vitamin D's role in diseases such as cancer, diabetes and immune dysfunction has not been investigated in dogs, vitamin D still plays a very important role in calcium and phosphorus metabolism and is a required nutrient. Homemade diets that are not prop-





erly formulated with vitamin D sources such as oily fish or fish oil are likely to be deficient in vitamin D.

As this study demonstrates, humans are not always good at ensuring their own diet is complete and balanced. Our dogs have a great advantage here; pet nutritionists can use diet formulation software to ensure the diets they create are meeting all of the dog's nutritional needs and prevent deficiencies and excesses.

## Ask the Pet Nutritionist

Do you have a question that you'd like to see answered in this newsletter? Submit your question to [hwatson@completeandbalanced.com](mailto:hwatson@completeandbalanced.com)

## Coming Next Month

- Digestive Tract Physiology and immunology
- Limited Antigen Recipes and Elimination Trials
- Allergy Thresholds – The Link Between Food and Inhalant Allergies

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