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Abstracted from:

## Bacterial lipase and high-fat diets in canine exocrine pancreatic insufficiency: A new therapy of steatorrhea?

Suzuki A, Mizumoto A, Sarr MG, Dimagno EP. *Gastroenterology* 1997; 112: 2048-2055.

In humans, exocrine pancreatic insufficiency (EPI) is associated with steatorrhea. Even with pancreatic enzyme supplementation, steatorrhea persists. Porcine pancreatic lipase found in commercially available enzyme extracts is inactivated in the acidic environment of the stomach. Therefore, the majority of this orally administered enzyme is unavailable for fat digestion in the duodenum. In an effort to increase the effectiveness of enzyme supplementation, enzyme preparations have been preincubated with food, administered with antacids or H<sub>2</sub> blockers to decrease gastric acidity, or formulated with an enteric coating. An alternate approach for preservation of lipolytic

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# Use of Medium Chain Triglycerides in Clinical Nutrition

D. P. Laflamme, DVM, PhD, Dipl ACVN

Dietary fats are important as a source of essential fatty acids as well as a source of energy. Normally, dietary fat is efficiently and nearly completely digested and absorbed. However, during various disease conditions, fat digestion is compromised. This paper reviews the physiology of fat digestion and absorption, diseases associated with fat maldigestion and the role of dietary fat in the management of fat malabsorption problems.

Table 1: Causes of Diarrhea Associated with Fat Malabsorption<sup>8</sup>

Exocrine pancreatic insufficiency	Intestinal histoplasmosis	Pancreatitis
Gluten sensitive enteropathy	Intestinal lymphosarcoma	Severe cholestasis
Inflammatory small bowel disease	Lymphangiectasia	Small intestinal bacterial overgrowth
	Pancreatic acinar atrophy	Villus atrophy

Most dietary fat present in dog foods is in the form of long chain triglycerides (LCT), which are composed of fatty acids that contain between 16 and 22 carbon atoms. Fat is the most complex nutrient to digest and absorb. The bulk of fat digestion begins with the emulsification of fats by bile acids, creating minute, water-soluble particles which can be readily digested by pancreatic and enteric lipase. Pancreatic lipase is the enzyme of primary importance in the normal digestion of fats. Gastric lipase, which is secreted by the stomach, and enteric lipase, which is secreted by the intestinal mucosa, are also

capable of digesting LCTs. These become more important in dogs with exocrine pancreatic insufficiency.

The end results of luminal LCT digestion are glycerol, monoglycerides and lipid-soluble free fatty acids. Bile acids facilitate the formation of micelles (water-soluble aggregations of the monoglycerides and free fatty acids) which act as a transport medium to carry the digested fat particles to the mucosal surface for absorption. While bile acids are not essential for fat absorption, they do facilitate it. Approximately 97% of dietary fat is normally digested and

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absorbed while only about 50% to 60% is absorbed in the absence of bile acids.<sup>5</sup>

Free fatty acids and monoglycerides are passively absorbed upon presentation to the intestinal brush border. Within the enterocyte, free long chain fatty acids are re-synthesized into triglycerides. The newly formed LCTs are packaged with absorbed and synthesized cholesterol, lipid soluble vitamins and other lipids into chylomicrons. These protein-coated, water-soluble chylomicrons then enter into the intestinal lacteals and are transported via the lymphatic system to the venous blood.

Fat absorption occurs predominantly via intestinal cells located at the tip of the microvilli. These are also the cells most susceptible to mucosal injury. Therefore, fat malabsorption or maldigestion may occur as a result of mucosal damage or atrophy. Deficiencies in pancreatic

lipase or bile acids also result in fat maldigestion or malabsorption. Severe fat malabsorption is evident as steatorrhea. Less severe fat malabsorption may contribute to diarrhea and/or weight loss. Table 1 lists conditions which may cause diarrhea associated with fat malabsorption.<sup>8</sup>

Fat malabsorption leads primarily to a secretory diarrhea. Malabsorbed fats may be fermented by colonic bacteria to

## Fat is the most complex nutrient to digest and absorb.

produce hydroxylated fatty acids which stimulate excessive secretion of fluids into the intestinal lumen. When the volume of secretions overloads the colon's ability to reabsorb the water, the result is diarrhea.

Less commonly, fats may contribute to an exudative diarrhea due to obstruction of lymphatic lacteals. This may occur with lymphangiectasia, lymphosarcoma, intestinal histoplasmosis or other conditions. Mucosal hydrostatic pressure increases secondary to congestion of

lacteals with fat-laden chylomicrons resulting in production of a protein-rich exudate and decreased nutrient and fluid absorption.

A low-fat diet may help limit diarrhea associated with fat malabsorption. Unfortunately, since fat provides a significant proportion of calories in the normal diet, many dogs will lose weight on a low-fat diet unless there is adequate dietary compensation. A diet containing highly digestible carbohydrates and proteins may provide adequate calories for some patients while others require a more concentrated source of energy. For these dogs, supplementation with medium chain triglycerides (MCT) can provide essential dietary calories.

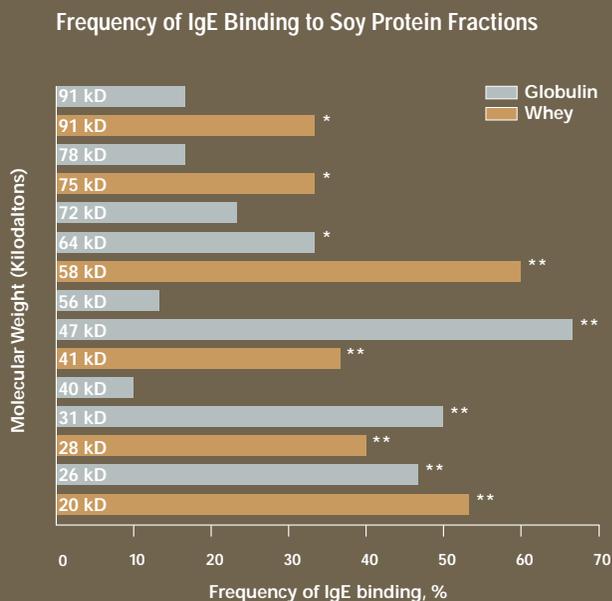
Medium chain triglycerides, such as found in milk fat or coconut oil, contain fatty acids with carbon chains from 6 to 12 carbons in length. These shorter chain lengths allow the MCTs to be digested and absorbed differently than LCTs (Table 2).

Gastric lipase, a normally insignificant enzyme in LCT digestion, is effective in hydrolyzing MCTs. Compared to other species, dogs have a higher level of

## Major allergens in soybean

(from: Awazuhara H., Kawai H., Maruchi N. *Clin. Exp. Allergy* 1997;27:325-332.)

The allergens in soy protein were identified by immunoblotting techniques using IgE and IgG4 from soybean sensitive patients and control subjects. The reactivity of the various soy protein fractions by molecular weight were then identified. The protein fractions showing a significant difference in IgE binding between soy sensitive and control subjects are shown in the graph to the right. Five *major* allergens were identified, with a molecular weight between 20 and 78 kilodaltons. Soy fractions with a molecular weight less than 20 kilodaltons are unlikely to elicit an allergic response.



\* Frequency of binding significantly different from normal controls, p<0.05  
\*\* Frequency of binding significantly different from normal controls, p<0.01

gastric lipase activity. Gastric lipase activity found in stomachs from dogs was considerably greater than that found in stomach tissue from rodents, pigs and primate species.<sup>6</sup> The enzyme is concentrated in the fundic mucosa and remains active despite the highly acidic gastric pH. While the amount of lipid hydrolysis in the stomach is normally less than 30%, it is thought to become more important in the absence of pancreatic lipase or in the presence of short and medium chain triglycerides. For example, the ability to digest MCTs

was reduced only about 25% in dogs with exocrine pancreatic insufficiency (EPI) while digestion of LCT fats in a commercially available low-fat diet was reduced by about 95% (Table 3).<sup>2</sup>

Medium chain triglycerides are readily hydrolyzed in the small intestine to form glycerol and free fatty acids. These fatty acids are water soluble and readily enter the enterocytes by diffusion. These characteristics may be beneficial in patients lacking bile acids, which are needed to facilitate normal transportation

of long chain fatty acids within the gastrointestinal lumen to the mucosal surface.

A large portion of absorbed MCT fatty acids pass directly from the enterocyte into the portal blood, bypassing the lymphatic system. Chylomicron lipid content was decreased by nearly 80% in humans when MCTs provided the dietary fat rather than LCTs.<sup>9</sup> This may be advantageous in patients with lymphatic congestion or disrupted lymphatic flow. However, excessive intake of fats, even MCTs, can increase chylomicron lipid content. Therefore, the total dietary fat should be controlled for patients with these conditions.

Following absorption, MCTs are readily metabolized for energy. Because of their short chain length, medium chain fatty acids pass easily into cells and are oxidized. It appears that MCTs are not incorporated into storage lipids, such as adipose tissue, but are preferentially oxidized for energy. Intravenously administered MCTs were oxidized at about twice the rate of LCTs.<sup>4</sup>

Concerns about the use of MCTs have been raised. Notably, toxicity was observed when excess quantities were

**Table 2: Summary of Digestion and Absorption of Long Chain and Medium Chain Triglycerides**

	LCT	MCT
Digestion		
Hydrolysis by gastric lipases	Slow	Fast
Hydrolysis by pancreatic lipases	Fast	Very Fast
Luminal transport		
Water solubility of free fatty acids	Low	High
Requires bile acid micellarization	Yes	No
Absorption		
Paracellular absorption	None	Some
Re-esterification and chylomicron formation	Yes	No
Primary transport route from gut	Lymphatic	Portal

## Clinical Application of Glutamine Therapy

Sarah K. Abood, DVM, PhD

The amino acid glutamine is readily synthesized in the skeletal muscle, lung and other tissues of normal healthy animals. It functions as a regulator of protein synthesis and a precursor for nucleic acid biosynthesis. It also shuttles nitrogen and carbon between tissues, and serves as a substrate for renal ammoniogenesis, hepatic ureagenesis, and gluconeogenesis. Glutamine

can be utilized by all cells of the body, but is especially important to rapidly dividing cells of the intestinal tract and the immune system.

In clinical patients that are not consuming adequate nutrition, short-term supplementation of glutamine administered in food or water may support the integrity of the small intestine. While controlled

clinical trials documenting beneficial effects of glutamine supplementation in veterinary patients are lacking, studies in laboratory animals and humans suggest that glutamine may have a protective effect in maintaining intestinal structure and function during disease. Criteria for selecting small animal patients to receive glutamine

may include 1) a history of vomiting, diarrhea, or anorexia for greater than five days, or 2) intestinal surgery or chemotherapy followed by greater than two days of fasting or anorexia.

Oral glutamine, provided at 0.5 gm/kg body weight/day, should be dissolved in water or mixed in food/gruel and divided into two or more daily servings. Because glutamine is unstable at room temperature for extended periods, any unconsumed water or



administered intravenously, resulting in a narcoleptic state. This condition was reversed by reducing the amount infused to a more suitable level.<sup>7</sup> At one time the use of MCTs in patients with liver failure was considered inappropriate. It was suggested that higher concentrations of free fatty acids would compete with tryptophan for binding sites on albumin, and aggravate hepatic encephalopathy. This theory has been evaluated and disproven. In fact, MCTs have been shown to enhance nutritional status and are especially recommended for use in patients with chronic liver failure.<sup>1,3,5</sup>

... the ability to digest MCTs was reduced only about 25% in dogs with pancreatic insufficiency while digestion of LCT fats was reduced by about 95%.

Medium chain triglycerides may be beneficial in the management of many conditions which cause fat malabsorption or deranged energy metabolism. Despite their considerable benefits, however, MCTs should never be used as the sole

food with added glutamine should be removed after 15 minutes and fresh water should be made available. The palatability of glutamine dissolved in water is similar to electrolyte solutions; most sick animals that are interested in drinking or eating usually will accept water/food with glutamine added. Glutamine may be purchased at a reasonable cost (Ajinomoto USA, Inc., Teaneck, N.J.), so that the cost to supplement a medium sized dog is less than \$0.50 per day.

The optimal dose and beneficial length of time for glutamine supplementation in veterinary patients is not known. Contraindications to glutamine administration have yet to be established but patients with liver disease or hepatoencephalopathy might be unable to dispose of the byproducts of glutamine metabolism: glutamate and ammonia. Glutamine supplementation also may be contraindicated in patients with chronic kidney disease because of abnormalities in acid/base

balance. Although the precise role that glutamine may play is unclear, there is evidence that it can be safely and economically administered to support intestinal function in patients that are unable or unwilling to consume complete diets. ❏

Adapted from Abood SK. Proceedings 1998 North American Veterinary Conference, Orlando, Florida.

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**Table 3: Effect of Exocrine Pancreatic Insufficiency on Nutrient Digestion in Dogs<sup>2</sup>**

	Percent Apparent Digestibility (Mean +/- SD)			
	Dry Matter	Protein	Fat	Carbohydrate
<b>Commercial Low Fat Diet with LCTs</b>				
<b>Normal Dogs</b>	<b>89.8 +/- 2.8</b>	<b>91.1 +/- 2.7</b>	<b>96.0 +/- 1.2</b>	<b>94.5 +/- 1.6</b>
<b>Dogs with EPI</b>	<b>44.5 +/- 14.1</b>	<b>25.7 +/- 26.2</b>	<b>1.3 +/- 27.7</b>	<b>65.4 +/- 3.1</b>
<b>Experimental Diet with MCTs</b>				
<b>Normal Dogs</b>	<b>91.6 +/- 2.4</b>	<b>86.8 +/- 4.8</b>	<b>93.7 +/- 2.5</b>	<b>96.9 +/- 0.6</b>
<b>Dogs with EPI</b>	<b>86.7 +/- 4.3</b>	<b>70.4 +/- 9.8</b>	<b>76.8 +/- 8.6</b>	<b>97.8 +/- 2.0</b>

LCTs = Long Chain Triglycerides; EPI = Exocrine Pancreatic Insufficiency; MCTs = Medium Chain Triglycerides

source of fatty acids. These lipids are devoid of essential omega-6 fatty acids. Therefore, it is critical that patients receive an appropriate source of essential fatty acids in addition to the MCTs. It is suggested that MCTs not exceed 50% of dietary fat for diets that will be fed chronically. ❏

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...decreases in serum urea nitrogen observed in association with high fiber diets were due to a decrease in protein digestibility.

## Urea Trapping and Its Importance in Dogs

Urea is produced in the liver and serves as a non-toxic form to transport amino nitrogen derived from the breakdown of proteins. The serum concentration of urea nitrogen (SUN) is influenced by a number of factors in normal animals. Among these is the amount of dietary protein consumed and absorbed from the gastrointestinal (GI) tract. Under normal circumstances, a prolonged reduction of protein intake or absorption will lead to a reduction in SUN while increased protein intake above maintenance levels will increase SUN.

Serum urea nitrogen is increased in patients with chronic renal failure (CRF). Historically, dietary protein restriction has been prescribed for CRF patients in an attempt to reduce SUN under the assumption that urea was toxic. Most modern investigators, however, consider that urea is not a "uremic toxin" per se, but is merely a marker that correlates reasonably well with clinical signs of uremia or adequacy of dialysis.<sup>1,3,5</sup> Therefore, dietary protein should not be reduced for the sole purpose of attaining a prescribed reduction in SUN concentration.<sup>5</sup>

Recently, inclusion of fermentable fiber in the diet has been suggested as a means of reducing SUN in patients with CRF.<sup>4</sup> Fermentable fibers stimulate bacterial proliferation. As the bacteria reproduce they metabolize non-absorbed dietary protein, sloughed cells, and urea which has diffused into the gut lumen as a source of nitrogen for protein. Dietary fiber also can decrease protein digestion and absorption, making more protein available to the GI microflora. The incorporation of protein and urea nitrogen into fecal bacteria results in less protein being absorbed and greater amounts of nitrogen excreted in feces. This has been shown to induce a slight reduction in SUN and may be interpreted as support for the urea "trapping" theory.

The urea trapping theory assumes: 1) there is a net flux of urea into the GI tract; 2) urea is incorporated into bacterial protein and excreted in feces rather than reabsorbed as ammonia; 3) the decrease in SUN is due to

GI loss of urea nitrogen rather than decreased absorption of dietary protein; and 4) the decrease in SUN is biologically significant and provides some benefit to the patient. However, a number of studies have raised doubts about these assumptions.

### Is there a net flux of urea into the gastrointestinal tract?

Investigators administered isotopically labeled urea intravenously and monitored animals for disposition of the labeled urea. Only a very small portion of labeled urea appeared in the feces.<sup>9</sup> The majority of labeled urea nitrogen was excreted in urine, with more of the label appearing in the urine of animals fed beet pulp compared to those fed corn starch. In other studies that actually measured flux of urea into the gastrointestinal tract, there was no significant net flux into or out of the gastrointestinal tract despite the inclusion of soluble or insoluble fiber in the diet, or elevated plasma urea concentrations.<sup>6-8</sup> Thus, the net movement of urea into the gastrointestinal tract is negligible.

### Is urea nitrogen excreted in feces or reabsorbed?

Urea in the gastrointestinal tract is either reabsorbed intact or metabolized by bacteria to produce ammonia or bacterial protein. Most of the ammonia reabsorbed via the portal bloodstream is reincorporated into urea in the liver. Thus a futile cycle occurs and enteric loss of urea is minimal due to the reabsorption of ammonia.<sup>3</sup> In dogs, urea nitrogen contributes 42% of the ammonium absorbed from the colon.<sup>11</sup> As noted in studies with isotopically labeled urea, very little plasma urea is excreted in the feces.<sup>9</sup>

### Is the decrease in SUN due to gastrointestinal excretion of urea nitrogen or decreased absorption of dietary protein?

Under normal feeding conditions, SUN levels can be decreased by decreasing the intake or absorption (digestibility) of dietary protein.<sup>9</sup> The addition of fibrous ingredients, such as beet pulp or oligosaccharides, has been shown to reduce dietary protein digestibility.<sup>2</sup> Addition of resistant starch, a form of fermentable dietary fiber, to the diet reduced true protein digestibility as measured by increased nitrogen remaining in ileal

digesta.<sup>4</sup> Fiber did result in increased fecal nitrogen excretion, although this was likely due to a combination of undigested dietary protein and increased bacterial protein. The mean fecal bacterial mass and, therefore, bacterial protein was 1.5 fold greater with addition of soluble fiber to the diet.<sup>1</sup>

This suggests that the decreases in SUN observed in association with high fiber diets were due to a decrease in protein digestibility. A reduction in protein digestion and absorption would provide the same effect as consuming a reduced quantity of total protein, which can lower SUN. In studies that observed a reduction in SUN from dietary fiber, the effect was pronounced only if dietary protein was low.<sup>10,12</sup> The effect of dietary protein content on SUN was much greater than the effect of dietary fiber.<sup>12</sup>

### Does the decrease in SUN provide some benefit to the patient?

While SUN concentration was significantly reduced in CRF patients through the addition of soluble fiber to the diet (mean +/- SEM = 44 +/- 5 mg/dL vs 50 +/- 6 mg/dL), there was no effect on mean serum creatinine concentration (4.4 +/- 0.8 mg/dL vs 4.5 +/- 0.8 mg/dL).<sup>1</sup> The lack of effect on serum creatinine suggests no effect on kidney function. While SUN is commonly used as a marker of kidney function, it is not thought to be a primary uremic toxin.

These data suggest that the SUN lowering effect of dietary fiber works by the same mechanism as low protein diets – it reduces the amount of protein available for absorption by the body. An incremental benefit over dietary protein restriction has yet to be demonstrated. ❏

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activity of enzyme preparations is the use of a lipase isolated from the bacteria, *Burkholderia plantarii*, which has been shown to maintain lipolytic activity under *in vitro* conditions mimicking the acid environment of the stomach.

Human and canine pancreatic and gastrointestinal physiologies are similar, therefore, the *in vivo* effectiveness of this bacterial lipase was determined in a canine model of pancreatic insufficiency. EPI was surgically induced by ligation and transection of all pancreaticoduodenal connections in four dogs. With induction of EPI, the coefficient of fat absorption (CFA) was dramatically reduced from 96% (pre-surgical) to 63%. When dogs were fed a diet containing 30% of calories from fat, supplementation of powdered bacterial lipase (range 30,000 IU to 600,000 IU) increased CFA in a dose-dependent manner. Supplementation at 600,000 IU of bacterial lipase corrected steatorrhea in two dogs and dramatically improved fat absorption in the other two dogs. When the dogs were supplemented with 300,000 IU bacterial lipase

with each meal and fed diets containing 18%, 23%, 33%, 43% and 47% of calories from fat, CFA was directly correlated with the percentage of fat in the diet. Without enzyme supplementation, fecal fat content increased significantly in dogs fed diets containing 43% and 47% fat. With 300,000 IU bacterial lipase supplementation, fecal fat content was significantly reduced to approximately 10 grams/24 hours for all diets.

The authors conclude that 300,000 IU of powder bacterial lipase ingested with high-fat meals corrects canine pancreatic steatorrhea and may abolish human pancreatic steatorrhea.

### Commentary by Helene Pazak, DVM, PhD

Clinical management of canine EPI includes feeding a highly digestible, low-fat and low-fiber diet and supplementation with pancreatic enzymes at each meal. Orally administered pancreatic enzymes have been successful in increasing the absorption of dietary protein, carbohydrate and fat. Even with appropriate enzyme therapy, however, fat absorption may be incomplete, and steatorrhea and diarrhea may develop in affected animals.

The results of this study suggest that bacterial lipase supplementation was effective in improving fat absorption and greatly reducing steatorrhea in dogs with EPI. In this study, proteolytic enzymes and amylase were not supplemented, and protein and carbohydrate absorption did not improve in the affected dogs. Bacterial lipase, used in conjunction with currently available enzyme preparations, may prove to be valuable in the treatment of affected canine patients. Finally, the results of this study suggest that excessive dietary fat restriction may not be warranted in dogs with EPI. Without enzyme supplementation, fecal fat content was approximately the same when the dogs were fed diets containing 18%, 23% and 33% of calories from fat, which is typical of diets containing approximately 6% to 14% fat on a dry matter basis. Fecal fat content increased dramatically when fed a diet containing 43% or 47% calories from fat. An optimal amount of dietary fat has yet to be confirmed for canine EPI patients. ❑

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