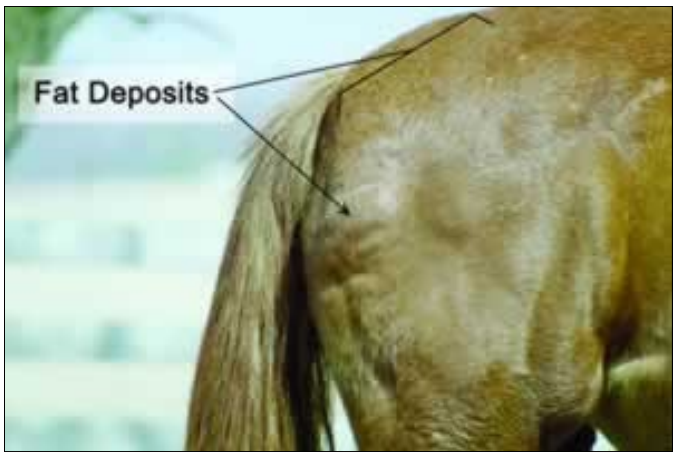
THE EASY KEEPER: Myth and Dangers

by Dr. Joseph Thomas

Easy Keeper has become such a common term in the horse world that when it's used a majority of horse people quickly visualize a fat horse with a cresty neck and a rump full of fat deposits. Promptly on the tail of this image is the apparently correct interpretation that this is a horse primed for laminitis.

Results of excessive triglycerides



As horse owners, we are advised to get that weight off fast by severely limiting food intake as though just overfeeding them is the cause of the weight condition. This is a dangerous misconception because these horses aren't just fat and prone to laminitis because of their weight. Actually, an Easy Keeper is a horse with a metabolic disease primarily involving the pancreas and liver that interferes with the digestion and absorption of fats and free fatty acids.1 These horses have distinct blood work profiles that closely resemble people with Type II Diabetes who also have the identical issues with fat absorption. Until the metabolic problem is dealt with at the source of the disease the Easy Keeper is always going to be an Easy Keeper and taking this horse's food away is not only cruel it is dangerous.

Laminitis and the Type II Diabetes Connection

Laminitis is a metabolic disease that has striking similarities to Type II diabetes mellitus. This is the type of diabetes that does not necessarily require insulin shots but rather comes later in life and is usually controlled by diet and glucose lowering medications. By definition, "Diabetes mellitus (DM) is a group of diseases in which blood glucose levels are elevated because of deficient insulin and/or abnormal insulin action."2 Laminitis, according to the insulin resistant (IR) model, also has elevated glucose in the bloodstream yet is considered to have elevated insulin as well.3

My research has shown that in the very early stages of both laminitis and DM, insulin is elevated with an associated decrease in glucose levels. As the diabetic process progresses, and during the IR phase of laminitis, the pancreas continues its insulin secretion, injuring and killing off the beta cells (the cells in the islets of the pancreas that secrete insulin) by overstimulation, eventually leading to either normal or decreased levels of insulin and increased levels of glucose in the blood. This insulin deficiency and blood

glucose excess leads to both DM in people and laminitis in horses.

A primary function of insulin is to impel glucose into all the cells of the body to provide life sustaining energy and nourishment. When there is too much glucose in the blood stream, there is not enough getting into the cells. In horses that means the laminae cells as well. The glucose starvation of these cells results in the separation and stretching of the laminae with likely coffin bone rotation.4

The Liver's Involvement in the Creation of the Easy Keeper

In both laminitis and DM the metabolism of fats and carbohydrates, a function of the liver and pancreas, is seriously impaired. The liver is the largest organ in the horse and, though rarely talked about in laminitis, it is the most important site for the formation of glucose and storage of glycogen, which readily converts to glucose as needed by the body to satisfy its energy needs. Under the influence of insulin and a hormone produced by the pancreas, the liver also regulates plasma glucose concentration.

Given that blood glucose is elevated due to carbohydrate metabolic dysfunction, the liver attempts to regulate this excess through the synthesis of carbohydrates into a neutral fat known as triglycerides. Essentially, these fats function as storage units for excess glucose and carbohydrates. As they are filled and more are produced in the liver, triglycerides become elevated (blood test reference range of high normal to above normal). When this happens, the triglycerides transform the liver into a fatty state that then pumps out fat globules to the muscles in the form of fatty cysts. These fat cells then become enlarged and more resistant to insulin's influence.5 They eventually end up in the adipose [fat] tissue of the body giving a swollen appearance and the familiar cresty neck, fat deposits in the rump, shoulder area and above the horse's eyes.

Bile and Intestinal pH

Another important function of the liver is the secretion of bile. In people bile is concentrated in the gallbladder before pouring into the small intestine. Given that horses do not have a gall bladder the liver's bile duct empties directly into the small intestine. The importance of this information is that bile is responsible for making the contents of the small intestine more alkaline, i.e. less acidic.

The most reliable blood test for liver function assesses bilirubin (the orange-yellow pigment in bile) in three aspects: total bilirubin, direct bilirubin and indirect bilirubin. My research has shown a clear pattern in these test results in laminitic horses: Total bilirubin tends to be low and direct bilirubin tends to be high. Given this bilirubin pattern, the bile duct is always affected because direct bilirubin rises from interference with the flow of bile from the liver. This means that the flow of bile is impaired in these horses.

So with less bile, the small intestine becomes more acidic. Given a familiar laminitic trigger, a fructan (sugar) rich pasture, and a horse with a predisposition towards laminitis in this pasture, this information on bile's pH becomes more than important.

Blood Test Results and Symptom Comparisons

The Beginning of the Disease Process Prior to External Symptoms

Blood Tests	Laminitis		Diabetes Mellitus	
	Results	Symptoms	Results	Symptoms
Glucose	Normal to Low Normal	None	Normal to low normal	None
Insulin	High Normal to Out of Range		High Normal to Out of Range	
Disease Advances with Time				
Glucose	High Normal to Out of Range	Increased thirst and fluid intake with increased urination	High Normal to Out of Range	Increased thirst and fluid intake with increased urination
Insulin	Decreases: Normal to Low Normal		Decreases: Normal to Low Normal	
Triglycerides	High Normal to Out of Range	Fat Deposits Become Visible	High Normal to Out of Range	Weight Gain Begins
Total Bilirubin	Low Normal	Intestinal pH begins to drop creating an acidic environment	Normal	None
Direct Bilirubin	High Normal			
Disease Progression Due to Glucose Overload				
	Carbohydrates, Grain, & Fructans		Sugars & Carbohydrates	
Glucose	Elevates Out of Range	Glucose Deprivation to Laminae Cells Resulting in Separation	Elevates Out of Range	Vision Problems, Peripheral Neuropathy plus more
Insulin	Low Normal to Normal	Pancreatic Beta Cells Die-Off Under Increased Load to Secrete Insulin	Low Normal to Normal	Pancreatic Beta Cells Die-Off Contributing to Glucose Elevation
Triglycerides	Elevates Out of Range	Fat Deposits Larger and More Dense		
Total Bilirubin	Low to Out of Range	pH Continues to Lower. Acidic Conditions Increase Dramatically	Normal	None
Direct Bilirubin	High to Out of Range			
Extreme Uncontrolled Metabolic Conditions				
Total Bicarbonate	Low Out of Range	Potential Progression for Metabolic Acidosis: Refer to text.		
Anion Gap	High Out of Range			

This diagram shows the progressive pattern of blood test results and the associated symptoms for Laminitic horses and people with Diabetes Mellitus given no regulation of diet and no glucose lowering intervention. The Results presented are relative ranges taken from the author's patient records and the blood work profiles of horses benefiting from Dr. Thomas' Chinese herbal Laminitis Intervention Program. These are relative rather than absolute values that represent patterns derived from the blood work panels analyzed.

Currently, it is understood that the small intestine does not contain the appropriate bacteria to assimilate fructans. Accordingly, the fructans, unable to be digested in the small intestine, move on their digestive path to the hindgut (colon) where they quickly ferment. During this fermentation process, the lining of the intestine is damaged with an accompanying drop in pH thereby creating an acidic environment. In the course of this process, toxins are released into the bloodstream. When they reach the hoof and meet with expanded blood vessels, the toxins are expedited to the inner hoof wall releasing a floodgate response of MMP's (matrix metalloproteinases, an enzyme), resulting in separation and stretching of the laminae.6

The bilirubin profile apparent in my research shows that there is an acidic environment in the intestines due to a liver bile dysfunction that is an unrecognized component of the pre-existing metabolic syndrome. Depending on the magnitude of the direct bilirubin elevation, which is another way of saying depending on the relative severity of the individual horse's metabolic syndrome, the intestines are already proportionally acidic relative to the bile problem. With a comparatively low pH already present before the horse ingests that lush grass, the drop in pH from the hindgut must have an even greater impact on the subsequent chain of events.

Metabolic Acidosis: The Danger Behind the Myth

The traditional advice from well-meaning horse people is to seriously restrict the food of Easy Keepers. Clearly it is important to limit their intake of carbohydrates, grains and fructans, but to limit their intake of hay is not advisable. These horses are heavy due to their underlying metabolic condition, similar to the obesity often associated with Type II diabetics. If you deprive them of enough food to lose weight rapidly, not only does it not affect the underlying physiological cause of the weight problem, it also may put them at risk.

All horses have a standing pool of hydrochloric acid sitting in the lower stomach. The evolutionary purpose of this pool of stomach acid is to assist in digestion as horses by their nature are foragers and therefore eat most of their waking hours. When they do not eat, this acid is not absorbed. For horses with a metabolic syndrome, whether laminitis or Cushing's, who already have acidic intestines, this situation can become critical.

There is a pathological condition called acidosis that results from the accumulation of acid or the depletion of the alkaline reserve (measured by a blood test called Total Bicarbonate - low in laminitic horses). By dramatically reducing the horse's diet to a point nearing starvation, the horse's body starts metabolizing their own fat stores very rapidly, breaking them down to fatty acids. This creates a highly acidic environment resulting in something referred to as metabolic acidosis. This is very similar to the Acidosis resulting from 'uncontrolled' DM. In both instances, large quantities of 'ketone bodies' (three acidic substances that are produced by fatty acid and carbohydrate metabolism in the liver) collect in the urine and tissues of the body, depleting the bicarbonate reserves that are to keep the pH alkaline. Metabolic acidosis occurs in horses from both 'starvation' and a progressively 'uncontrolled' metabolic disease.

A horse or person in metabolic acidosis can become comatose and die, preceded by severe respiratory difficulties. It is unknown how many horses die

in this manner as it is often misdiagnosed. It may not be frequent, but metabolic acidosis in horses occurs more often than is known or talked about – one horse is too many.

Management

The point of drawing your attention to the similarities between diabetes and laminitis is to underline the importance of understanding laminitis as a metabolic disease. Horses with laminitis or Cushing's live along a continuum within the disease process. There are horses that seem to always be on the edge of an episode yet never move into full-blown lameness and other horses that end up with coffin bone rotation in the dead of winter without any apparent trigger. Similarly, some people with DM can control their disease merely by diet regulation while others must use diet and hypoglycemic medications (drugs to lower blood glucose) of varying magnitudes. For both, the management of the disease is a way of life. As long as a horse has a propensity towards this disease, excess glucose in the blood is going to go along with glucose deprivation to the laminae. Whether through some external or internal trigger the end result is going to be laminae separation and stretching and no amount of hoof anti-inflammatory treatment is going to cure this disease – the source of this disease is presented in the horse's blood work and not in the hoof.

Through my research on laminitis and my twenty-five years experience in Chinese Medicine, I have developed a program of herbal formulas (Laminitis Intervention Program) which is designed to change the course of the patterns described. Each herb used in these formulas has been prepared with pharmacological precision and has been chosen for its unique qualities and contribution to the overall effectiveness of the formula.

My work is rooted in Chinese Medical Theory which views the horse as a whole rather than a group of parts working in isolation. In the same sense, my herbal formulas are created as a whole, not just a combination of single herbs that do different things. Therefore, to effectively arrive at an herbal solution to laminitis, every single herb of the twenty-five in the formula must work in concert. Individual herbs were chosen for their intricate functions, the way they relate to one another and their impact on the involved organ system. For example, in the foundation formula there is a group of herbs that work together with the liver and pancreas to improve the digestion of carbohydrates, decrease blood glucose levels, and improve the insulin-glucose connection. Another group of herbs, in this same formula, improves bile secretion from the liver thereby decreasing the acidity of the intestinal contents and raising the pH of the intestines, making them more alkaline. This is only a brief description of the conceptual framework of the formulation process and is in no way representative of the entire picture.

This program of proprietary Chinese herbal formulas is consistently improving the lives of laminitic horses along the entire continuum of the disease process as it has been presented in this article.

Footnotes:

1. Fatty acids and insulin resistance in muscle and liver. P. Kovacs, M. Stumvoll. Best Pract Res Clin Endocrinol Metab. (2005)Dec;19(4):625-35.

2. Clinical diagnosis and management by laboratory methods. J.B. Henry, MD, W.B. Saunders Company.
3. Equine internal medicine; S.M. Reed, W.M. Bayly, D.C. Sellon. (2004) Saunders Company.
4. Decreased glucose metabolism causes separation of hoof lamellar in vitro: a trigger for laminitis. M.A. Pass, S. Pollitt, and C.C. Pollitt; Equine Vet J Suppl (26)(1998)133-138.
5. Dysfunctional fat cells, lipotoxicity and type 2 diabetes. R.A. DeFronzo. Int J Clin Pract Suppl (2004)Oct(143):9-21.