



# Understanding and Avoiding Laminitis and Founder in Horses

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## Introduction

Horses and other equids have a unique anatomy whereby their entire body weight is supported by the equivalent a single fingernail on each foot. The horse's hooves are bound to the last digit of each front and hind limb by a set of feathery-looking structures called sensitive laminae which lock into grooves on the inside of the hoof walls. These grooves are called the insensitive laminae. Whenever a horse's sensitive laminae, which are absolutely essential for normal weight bearing and locomotion, become inflamed and sore, then the horse is said to have laminitis. When the sensitive laminae are damaged to the point that they begin to detach from the insensitive laminae and last bone of the foot, called the coffin bone or third phalanx rotates downward, the horse is said to be foundered. Horses with acute painful laminitis may recover a reasonable degree of soundness if the coffin bone rotation is minimal, however, once a horse is significantly foundered, it is unlikely that it will ever recover full soundness. Therefore, it is important for horse owners to manage their horses to reduce the likelihood that laminitis will ever occur.

As part of the 2008 Australian Equine Science Symposium in Gold Coast Queensland during June 4-6, 2008, several of the world's leading experts on equine laminitis presented recent research and recommendations for prevention, which will be summarized here. One of those experts was Dr. Patricia A. Harris of the Equine Studies Group of the Waltham Centre for Pet Nutrition in Melton, Mowbray, UK.

## "Thrifty" horses

Dr. Harris has been investigating features of horses that are "thrifty" or prone to being fat, and also prone to developing laminitis. She has been trying to determine why horses and ponies that tend to maintain body weight easily and become obese when consuming seemingly small excesses of feed are also prone to laminitis and founder. One aspect of these horses that is currently being studied is the function of the glucose transporter proteins that are important for carrying glucose to skeletal muscles and adipose tissues. These glucose transporter proteins are acted upon by insulin to control the amount of glucose that is carried in the blood versus delivered to the tissues. The main transporter protein that carries glucose to muscle and adipose tissue is called glut-4, and appears to be inhibited by a gene in certain ponies that are prone to laminitis. It is conceivable that it may have been beneficial for ponies with the "thrifty gene" in the wild, under harsh conditions of feed restriction. Blocking glut-4 would leave more available glucose for use in tissues like the endometrium (lining of the uterus) that do not require glut-4 to transport glucose in order to support pregnancy when times were tough. However, under modern conditions of feed abundance, these animals are more prone to high levels of visceral fat and elevated portal fatty acids which the liver converts to glucose.

## Insulin insensitivity

It has been found that horses that are insulin insensitive are much more prone to laminitis than normal horses. Most people realize that insulin is secreted by the pancreas in response to rising blood glucose concentrations, either after a meal or after glucose is synthesized from fatty acids. As mentioned earlier, insulin activates glut-4 to return blood glucose concentrations to normal by transporting glucose into tissues. Horses that require much more insulin than the average horse to return blood glucose concentrations to normal are said to be "insulin insensitive". What many people didn't realize was that the blood glucose curves recorded after meals in insulin insensitive horses are identical to those in horses that are normal. However, the "normal" blood glucose curve after a meal in an insulin insensitive horse is achieved by secreting greatly elevated concentrations of insulin to overcome the inhibited glut-4.

Dr. K.E. Asplin of the University of Queensland, along with several co-workers, reported on a very interesting study that found that experimentally-induced high insulin concentrations could directly cause laminitis in normal



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Page 2 of 2, Written: July 2008

ponies whose blood glucose concentrations were maintained at normal levels through glucose infusion. Prior laminitis research by this group had presupposed that glut-4 was required for glucose transport to sensitive laminae, which have a very high glucose demand. It was hypothesized that inactivated glut-4 might be “starving” the sensitive laminae of glucose and causing laminitis. However, genetic testing of sensitive laminae in horses found no expression of glut-4, and only expression of glut-1, another glucose transporter protein that does not require insulin to transport glucose to tissues. Therefore, glut-4 inactivation does not lead to glucose deprivation in the sensitive laminae. However, the results of this study suggested that the high insulin concentrations in “insulin insensitive” horses may be the direct cause. More research will need to be done to confirm this hypothesis.

### Laminitis-inducing feeds

Even in horses and ponies that are metabolically normal, prior research has suggested that over-used glut-4 may become resistant to the action of insulin simply from chronic overfeeding of starchy and sugary feeds. It has also been known for many years that laminitis can be reliably produced by intentionally overfeeding horses, even just once; using excessive amounts of cereal grains. It is always wise to limit grain feeding to horses that are either overweight or those that have ever experienced an episode of laminitis after grain feeding.

Even though overfeeding of grain is probably the single best-known cause of laminitis among horse owners, it is very likely that most laminitis and founder cases occur in horses and ponies out on pasture. Once again, the “thrifty” or “easy-keeper” and “insulin insensitive” equines are the ones most prone to “grass founder”. Most of the research on grass founder in horses in recent years has focused on a class of plant sugars called “fructans” in pasture grasses. A study reported by Dr. S.R. Bailey of the University of Melbourne, Parkville, Victoria, Australia, found that dietary fructans produced exaggerated insulin responses in ponies genetically prone to laminitis. Insulin concentrations after feeding high concentrations of fructans to genetically laminitis-prone ponies averaged 5.5 times higher than in normal ponies. This is more powerful evidence that both genetics and nutrition are key elements in the development of this devastating disorder, and that certain forages alone may pose a danger to many horses and ponies that are genetically prone to laminitis.

Several suggestions were offered at the Australian Equine Science Symposium to combat grass founder in horses and ponies that are predisposed to the condition:

1. Use varieties of pasture grasses that tend to be lower in fructan concentrations.
2. Fructan concentrations tend to be lower in pasture grass at night, so allow horses to graze at night and limit daytime grazing.
3. The top of grass shoots tend to be lower in fructans than the bottom, so the use of grazing muzzles that partially restrict grazing to the upper portion of shoots may be helpful.
4. Don't allow horses to graze frosted pastures. Frosted pastures have higher levels of fructans because the plants can still photosynthesize, but growth is diminished, so high levels of the sugars build up that don't get used for growth.
5. Fructan concentrations can likewise increase after drought, although the reduced total yield of forage tends to diminish the danger. However, don't allow a founder-prone horse to be turned out on a droughty pasture that has not been grazed extensively, as this can also result in consumption of dangerous concentrations of the plant sugars.
6. Soaking hay for 30 minutes in cold water before feeding can reduce the fructan concentration by up to 50%, and can be very helpful for horses and ponies that are prone to laminitis.