Laminitis is a serious, crippling disease of horses, ponies and donkeys. Severe and recurring cases of laminitis can reduce a horse's usefulness or result in the horse being destroyed to prevent further suffering. Treatment can require a lot of time and money (whether successful or not) and requires a good deal of energy from the carer for an extended period of time.

WHAT IS LAMINITIS?

Laminitis is a painful inflammatory condition of the tissues (laminae) that bond the hoof wall to the pedal (coffin) bone in the horse's hoof. It can affect any horse, of any age or sex, at any time of the year. Although it is traditionally considered a disease of fat ponies, laminitis can be triggered by a variety of metabolic or physical causes in any horse as discussed in 'Causes of laminitis'.

Laminitis is caused by weakening of the supporting lamina within the hoof, leading to painful tearing of the support structure suspending the pedal bone within the hoof. If laminitis is not treated promptly, the pedal bone drops (these cases are described as "sinkers") or the pedal bone can rotate downwards.

Laminitis and Founder are not the same. Laminitis can, but does not always result in Founder. The word Founder describes the sinking of the horse's foot. The sinking occurs when the laminar bond fails. The laminar bond is made up of two layers:

- The insensitive nonliving layer that grows from the coronary band (comparable to our finger nail).
- This is attached to the sensitive laminae (similar to the sensitive skin under our finger nails), which interlocks with insensitive tissue forming a remarkably strong bond.

The level of pain a horse demonstrates does not necessarily indicate either laminitis or founder. Some horses show tremendous pain while they are laminitic, and others show very little. The same may be true for foundering horses.

WHAT TO DO IF LAMINITIS OCCURS

- Treat laminitis as an emergency. Call us for advice
- Initial treatment is critical and can dramatically reduce the likelihood of founder
- Do not exercise your pony. If he is at pasture remove him to a deep shavings bed (10cm+ deep). If he has to travel a distance to a stable use a low loading trailer (medication may be appropriate first).
- After examining your pony we may give painkillers, a peripheral vasodilator - sedative to encourage him to lie down and fit frog supports and give other treatment if appropriate.
- Do not starve your pony as hyperlipaemia may develop. We will advise you on an appropriate diet; this will normally consist of limited forage and high fibre, low starch chopped feeds with plenty of fluids.
CAUSES OF LAMINITIS

Laminitis can be triggered by various causes. The most common causes are:

- Grain engorgement or excessive intake of pasture
- Sequel to infection. For example a mare that has recently foaled and has retained afterbirth
- Sequel to administration of excessive doses of certain drugs such as corticosteroids (e.g. Prednisolone)
- Cancer of the pituitary gland ("Cushings disease")

Less common causes include

- Concussion from riding on hard surfaces
- Excessive weight bearing by one leg due to severe injury or lameness in the opposite leg
- Stress from long distance transport

EFFECTS OF LOW GRADE LAMINITIS

Subclinical laminitis is the early stage of laminitis where structural changes have occurred within the hoof, without the horse being obviously lame or short in the stride. Subclinical laminitis is a likely cause of the following.

- Prominent growth rings on the hoof wall
- Dished hoof wall with flared out long toes (and often low compacted heals)
- Low grade seedy toe, sub solar haematomas that may lead to an abscess
- Crumbly white line
- Flaky soles and hoof edges broken away
- "Sore feet" in the front feet. More evident particularly on hard ground
- Shortened gait that doesn't improve with exercise and worsens with fast and hard work.

If you recognize any four of the above symptoms your horse may be suffering the effects of low-grade laminitis.

DIET AND LAMINITIS

Vets and horse owners alike have long been familiar with the association of lush pasture and laminitis in susceptible equines. Water soluble carbohydrates in grasses are implicated as triggers for dietary induced laminitis. Cereals (grain) contain high levels of starch. Starch has the same effect as the Water Soluble Carbohydrates found in grasses in causing laminitis.

Temperate grasses contain naturally large amounts of carbohydrate (sucrose, fructose, glucose, and fructans). It is the carbohydrate that is rapidly fermented that initiates the cascade resulting in laminitis. In most of Europe, USA & southern parts of Australia, temperate grasses are an important part of the diet of most equines, especially during the period Spring to Autumn. It is only recently that the association with high fructan levels and the onset of dietary induced laminitis was made.

Difficulties lie in predicting Fructan (a soluble carbohydrate) levels at any given point in time. The content of Fructans in grasses is highly variable. The actual amounts of sucrose and fructans vary from 5-50% of total dry matter. Large changes can occur within hours. The accumulation of these sugars is a highly dynamic, variable and environmentally responsive process. Temperate grasses may store 10-13% of their
total sugars reserves as starches. This leaves up to 90% being stored as sucrose or fructans. (Water-soluble carbohydrates)

Certain grass species seem to accumulate more fructans than others; perennial ryegrass may contain 12% fructans and cocksfoot only 2%. Temperature effects fructan accumulation, cold sunny days mean a high level of fructan accumulation. Grass stores more fructans in its stems than in its leaves so horses turned out on stubble after a hay crop can be eating a relatively large amount of fructans, conversely well managed fields which are grazed by sheep or cut will have a high leaf to stem ratio and potentially less fructans. Fertilisers should be avoided wherever possible

International research has shown that there is an interrelationship between diet, hindgut acidosis and low-grade laminitis.

**PREVENTION OF DIETARY INDUCED LAMINITIS**

"PREVENTION IS BETTER THAN CURE"

Restricting the horses' intake of lush green grass and grain can reduce the rate of lactic acid production. Easy to say, however in practice a pony in a field as sparse as a pool table can still suffer dietary induced laminitis.

Digestion of carbohydrates in Equines and its relevance to dietary induced laminitis.

Lush green grass, which carpets the countryside particularly in spring and autumn, or feeding high levels of grain, can induce laminitis in horses. This information is well known amongst horse owners and riders.

The first step is to understand what components of the horses diet create the risk of laminitis. What do grains and lush pastures have in common? Cereal grains contain abundant amounts of Starch a form of carbohydrate that is broken down to simple sugars such as glucose in the digestive tract. Lush green grass can have high levels of sugars that are directly available in the digestive tract.

- **GRAIN** contains Starch that are converted to Simple Sugars
- **LUSH GRASS** contains readily available Sugars

How does the horse usually digest its food?

This question provides us with the key to the cause of dietary induced laminitis. The horse digests its food in two ways

- **Simple digestion** Once the food has been eaten it is digested by enzymes in the first part of the digestive tract. The nutrients are then absorbed from the digestive tract.
- **Hind gut fermentation** Excess sugars and complex carbohydrates which require longer to digest move onto the large bowel for fermentation.

Normally the bacteria ferment away slowly and horse absorbs the products from the bowel for use as an energy source. When the simple sugars arrive in the large bowel it takes the bacteria no time to ferment them producing large amounts of acid very quickly - more quickly than they can be absorbed. This leads to build up of lactic acid in the hind gut. This is the first step towards laminitis. The process that follows is complex and there is still research being conducted as to the exact mechanism. Suffice to say that the build up of acid is the primary trigger.