THE RELATIONSHIP BETWEEN EQUINE DIET AND PRESENTATION OF
LAMINITIS

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ACKNOWLEDGEMENTS

This thesis is the result of a beautiful day during my time shadowing at the Cleveland Equine Clinic. As I walked to get Dr. Genovese’s next patient, I quickly realized that the horse in front of me was a very old familiar horse. It was Bubba! The horse I fell in love with as a little girl. I even spent every Saturday volunteering to clean horse stalls to be able to afford riding lessons on Bubba. The horse that had brought me so much joy as a little girl, came into the Cleveland Equine Clinic to be treated for laminitis; I realized how badly I wanted to be part of the cure for laminitis – so other little girls would never have to see their old friends suffer from such a painful disease. I want to thank Dr. Adam Leff, Dr. Margaret Leslie, Dr. Mark Kershner and Dr. Leslie Heaphy for serving on my defense committee. Additionally, I would like to thank my mentors Dr. Ronald Genovese and Dr. Adam Leff for their support and guidance during my completion of The Relationship Between Equine Diet and Presentation of Laminitis and during my undergraduate career. Most of all, I would like to thank Bubba for being the most inspirational horse a girl could ever ask for.
CHAPTER 1. ABSTRACT

Abstract

Equine laminitis is a disease characterized by inflammation of the laminar tissues in the equine hoof. Inflammation of the laminar tissues can lead to failure of the laminae to act as a connective tissue that holds the distal phalanx bone in place within the hoof capsule. The purpose of this study was to investigate the dietary predispositions of equine laminitis patients and compare the diets of laminitis patients with the diets of a reference group. Owners with horses diagnosed with and under treatment for laminitis were surveyed in regards to their equine husbandry management techniques. Owners reported age, height, weight, breed, sex, exercise schedule, weekly time spent in pasture, specific grain type fed, specific hay type fed, any supplements given and owners noted if field grasses/other forages were present within the pastures that their horses had access to. After comparing the equine management techniques between groups one point of variance became clear --- laminitis patients were constantly allowed to be in pastures, while the reference group horses (if ever in pasture) were never in pasture for more than 8 hours a day. Such a difference in management techniques suggests that pasture field grasses may be the source of nonstructural carbohydrates (that are metabolized into sugars) that predispose these horses to insulin resistance and may eventually result in laminitis. Avoiding excessive nonstructural carbohydrates by preventing grazing while in pasture with grazing muzzles or changes in equine diets will revolutionize how laminitis is avoided until further research results in a more efficient treatment.
CHAPTER 2. INTRODUCTION

Introduction

Equine laminitis, a vascular disease defined by inflammation of the laminar tissue, has been historically difficult to treat due to the lack of consistent information regarding specific trigger factors of the disease. Though many trigger factors have been studied, no singular trigger factor has been identified in all cases of equine laminitis. The problem with many of the models used in previous laminitis studies is that the horses studied had experimentally induced laminitis rather than naturally occurring laminitis. This study attempts to identify specific dietary elements that may increase the patients’ risk of presenting symptoms of laminitis. By analyzing shared dietary elements between two groups of equine patients: the laminitis patients and the equine control group, researchers hope to identify any common dietary trends that are responsible for predisposing horses for laminitis.

The suspected culprit, excessive sugar, was closely examined due to many literature sources suggesting that carbohydrate overload is the primary cause of most cases of laminitis. The sugar content in each horse’s diet was compared to the patient’s exercise level and whether or not the patient in question presented symptoms of laminitis.
An understanding of the anatomy of the equine hoof is essential to understanding the development and progression of laminitis. The laminae are finger-like protrusions of tissue. In the equine foot, this tissue is found between the pedal bone (distal phalanx) and the hoof wall. Essentially, the tissue provides blood to the hoof and keeps the pedal bone in place. Two types of lamina exist. The first is the *dermal* laminae (the sensitive laminae). The second is the *epidermal* laminae (the insensitive) laminae. Each foot contains roughly 600 primary laminae; and each of the primary lamina contains approximately 100 secondary laminae.

Laminitis begins when blood flow to the lamina is reduced, resulting in cell death and eventually the breakdown of the laminae. Breakdown of the laminae leads to separation of the bond between the hoof wall and the pedal bone. When the breakdown of the laminae results in detachment and the rotation of the pedal bone (distal phalanx/P3) on its own axis, the laminitis condition is referred to as founder (Bhatt et al, 2014).

*According to A Review of the Pathophysiology and Treatment of Acute Laminitis:*
Pathophysiologic and Therapeutic Implications of Endothelin-1, approximately 75% of laminitic horses treated at a university hospital did not return to athletic soundness; the majority of these horses were ultimately euthanized due to the severity of pain associated with separation of the sensitive and insensitive laminae resulting in rotation and/or distal displacement of the distal phalanx. According to this review, laminitis has widespread implications for equine welfare as well as the equine performance industry. It is estimated that 15% of horses in the United States are afflicted with laminitis over the course of their lifetime, and 75% of these horses develop severe or chronic lameness and debilitation that necessitates euthanasia. These represent a substantial number of horses in the United States and worldwide that succumb to the devastating effects of laminitis.

Diagnosis and treatment of laminitis is estimated to cost approximately $8 million every year; with the monetary loss of euthanized animals each year subsequent to complications of laminitis is approximately an additional $5 million (Eades et al, 2002). According to a survey conducted in the United States, 13% of horse owners and equine operations had reported laminitis related issues with their horses in the previous 12 months (USDA, 2000).

As indicated in a study done by Hinckley and Henderson in 1996, a survey of 113,000 horses demonstrated 7.1% prevalence in the UK (Bailey et al 2003). In the same study, Hinckley and Henderson noted that pasture-induced laminitis appears to be the most common cause of laminitis in the UK. From these implications, this study intends to analyze how excessive dietary sugar may be commonly associated with laminitis presentation. One of the key pathophysiological characteristics of laminitis presents in all
working theories and experimental models - the loss of cellular shape and attachment to the basement membrane of the laminae. Upon loss of cellular shape and attachment, the basement membrane is lysed by the matrix metalloproteinase enzymes (MMPs) (Bhatt et al 2014 and Pollitt, 1996).

Controversy exists in understanding blood flow patterns present in laminitis. Some investigators believe that blood is pooling in the digits, whereas others hypothesize that blood is inhibited from entering the digits during the acute phases of the disease. Several investigators have observed that ischemia or reduced blood flow to the foot occurs in laminitis. Ischemia initiates a complex cascade that results in loss of function. Included as a primary event in this cascade is necrosis due to anoxia. (Hood et al, 1999)

Acute laminitis is a systemic disease, which is only finally manifest as a condition of the foot (Bailey et al 2003 and Hood et al 1999a). Laminitis is diagnosed, as being in various stages based on the severity of pain and lameness the horse is experiencing.

Sub-clinical laminitis: is typically not associated with lameness symptoms, but rather presents evidence of laminar damage such as stretched white lines on the hoof and/or divergent hoof rings.
Mild clinical laminitis: presents some signs of lameness, such as difficulty turning or difficulty standing and walking on hard ground. At this stage, the horse’s gait seems normal on soft ground and/or while walking in straight lines.

Moderate clinical laminitis: occurs when patient is unwilling to walk or pick up feet for regular hoof cleanings. A behavior called “paddling” in the equine community is typically observed at this point. “Paddling” is described as a gait abnormality in which the horse will shift weight from foot to foot showing continuous discomfort. This stage can be diagnosed with a slightly increased respiration rate and typically a strong digital pulse upon physical examination.

Severe clinical laminitis: Total refusal to move and/or pick up feet. Typically patients at this stage of the disease will be observed lying down frequently. Upon physical examination, heart rates are generally found over 80 bpm and respiration rates are typically over 60 breaths/minute. These values are exceptionally high compared to normal: heart rates are generally around 40 bmp and respiratory rates are generally around 16 breaths/minute. Sweating is typically observed at this point, muscles are hardened. Body temperature is usually normal upon physical examination unless SIRS laminitis is occurring (guard against dehydration and impaction colic seen in horses with severe pain not eating and drinking).

Perhaps the most common theory of laminitis trigger factors exists in the Metabolic/Endocrine Theory. This theory focuses on insulin resistance and its pathophysiologic implications in equine patients. Insulin is a major regulatory hormone used in the metabolism of glucose and fat, growth, inflammation management, vascular
function and tissue remodeling. Essentially, insulin resistance works by altering the insulin signaling pathways needed for metabolism. Such alterations in glucose and fat metabolism pathways are believed to predispose horses to laminitis. Insulin resistance alters insulin signaling pathways by decreasing the insulin action in particular resistant pathways while simultaneously increasing insulin signaling in pathways that remain unaffected due to compensatory hyperinsulinemia (Bhatt et al, 2014). Insulin resistance is believed to be responsible for many of the changes observed in extreme cases of laminitis when hoof separation presents. Vasculature compromise, inflammation and glucose availability changes are commonly observed in hoof-wall separation during the advanced stages of laminitis. Insulin resistance presents similarly in humans as altered insulin signaling is observed in reduction of glucose availability. Such a reduction in availability to insulin-sensitive cells is often observed in tandem with vasoconstriction, endothelial damage and an inflammatory response. (Bhatt et al, 2014). Further research by the Australian Equine Laminitis Research Unit conducted by Christopher Pollit has established a direct link with insulin resistance in ponies with experimentally induced laminitis.

Other theories exist including the Enzymatic Theory, Inflammatory Theory, Mechanical/Traumatic Theory and Vascular Theory. Enzymatic Theory focuses on the established activation of lamellar matrix metalloproteinase enzymes (MMPs) … believed
to be important in the pathway that allows for basement membrane separation in the bond between the epidermal and dermal lamellae. This theory suggests that with increased digital blood flow (from inflammation) cytokines or some other trigger factors are brought to the digit thus envoking the activation and production of MMPs (Pollit and Daradka, 1998). *Inflammatory Theory* was studied through two experimental models the starch model and the black walnut extract model. In both models, increased neutrophil and platelet levels were observed in the laminitic subjects. This is believed to be the result of the up-regulation of inflammatory cytokines (Blikslager et al., 2006). Both models presented symptoms of endotoxemia/sepsis (Bhatt et al., 2014 and Belknap et al., 2009). Although there are many specific models that depict potential disease mechanisms and trigger factors of laminitis, only 5 trigger factors have reliably induced laminitis experimentally including starch, black walnut, oligofructose, fructose and insulin.

*Mechanical/Traumatic Theory* is believed to be the result of excessive mechanical overload. In such situations, non-weight bearing injuries (like fractures) may cause direct mechanical lamellae trauma. Upon acquiring the injury, patients often have excessive weight bearing issues during recovery that result in destructive lamellae changes or blood supply changes that can indirectly cause damage to the laminae (Bhatt et al., 2014).

*Vascular Theory* is defined primarily by blood flow abnormalities. Clinically it is most commonly associated with rapid bounding digital pulsation and increased temperature in the hoof, pastern, and surrounding area. Essentially the inflammatory process is believed to result in endothelial activation and dysfunction (Bhatt et al., 2014).
Laminitis Diagnosis: The absence of clinical signs during the development phase makes laminitis identification, treatment and prevention difficult until the disease has moved past its initial phases. The most common identification factor in recognizing laminitis is associated with lameness, bounding digital pulse and a “laminitic stance” (figure 3) in which the equine patient is reluctant to fully bear weight. This stance is typically associated with initial laminitis in the forefeet where about 65% of the horse’s weight is supported. To alleviate pain associated with weight bearing, horses stand with their feet placed more forward than normal and most of their weight supported by their hind legs (where laminitis does not typically develop until later). If forced to walk, the horses generally arch their backs and place hind limbs forward; this is done to minimize painful downward rotation force of the deep flexor tendon on the distal phalanx (Bhatt et al., 2014).

Three primary changes are observed on the exterior of the limb in question – coronary band changes, sole changes and hoof wall changes. Generally lamellar attachment failure results in the distal phalanx moving downward into the hoof. As this occurs, the coronary subunits move downward as well. The coronary band is observed with deficits and the sharp edge of the proximal hoof may be palpable dorsally. In severe cases, the laminae are no longer able to suspend the distal phalanx and skin may separate at the hairline of the coronet and exude serum (Bhatt et al., 2014). Sole changes may occur with a convex bulbous shape in the hoof indicating a drop in the distal phalanx due to laminar failure. In severe cases, the P3 bone can even puncture through the hoof wall (at which point euthanasia is the only option for owners). Hoof wall changes such as
slowed growth of the dorsal hoof wall accompanied by normal to excessive heel growth can result in concentric ring production below the coronet band visible on the hoof surface. In severe cases, this can result in the upturning of the hoe and toes can become unnaturally long making movement even more painful.

Radiographs are often collected to identify the hoof distal phalangeal distance (HDPD). In normal horses, HDPD never changes and is usually around 25% of the distal phalanx palmar cortex length (Bhatt et al., 2014). An increase in HDPD generally indicates early stages of laminitis. Documenting changes in shifting or rotation of the distal phalanx are absolutely crucial for determining prognosis. Thickness of the dorsal hoof wall and connective tissues must be studied relative to foot size and size of the P3 bone.

**Therapies/Prescriptions used in Treatment:** Multiple laminitis treatments exist, including drug therapies such as administration of anti-inflammatory drugs, anti-endotoxin therapy, vasodilators, antithrombotic therapy. Due to the wide array of suspected laminitis causes, many potential treatments exist. Corrective trimming and shoeing and surgical procedures can be used to correct some cases of laminitis in addition to drug therapies. Though various treatment regimens can be effective in acute cases of the disease, the prognosis is largely determined by the quality of laminae tissue remaining after damage has occurred.

Haemodynamic disturbances in tissue oxygenation and metabolic changes in hindgut flora, along with metalloproteinase activities may be responsible for the presentation of laminitis in many cases (Bailey et al., 2004). Most treatments focus on
damage limitation. Icing the feet has been advocated for because vasoconstriction caused by cold temperature is believed to prevent toxins from reaching the laminae in the foot (Bailey et al., 2004 and Pollitt and Davies, 1998). Anti-inflammatory drugs are typically administered at the same time as cold therapy in order to prevent toxins from entering the tissues.

Treatment of laminitis caused by vasospastic-ischemia should focus on limiting the severity of lesions. Treatment essentially includes the use of vasoactive agents like alpha adrenergic blockers that increase digital cutaneous perfusion and of agents that limit the severity of reperfusion injury (Hood et al, 1999). Many common vasodilators include phenothiazine derivatives such as acepromazine and phenoxybenzamine; these derivatives are believed to work by blocking 5-HT receptors and adreno-receptors but this has not been extensively studied (Bailey et al., 2004). Non-selective vasodialator drugs such as acepromazine and nitric oxide typically have short-lived effects due to tissue desensitization (Vincent et al., 1992). Fibrin antagonists are sometimes used to prevent platelet activation and aggregation (Weiss et al, 1998 and Bailey et al., 2004). Dihydropyridine calcium channel blockers may cause red blood cell deformities and improve blood flow thus decreasing disease severity (Hood et al., 2002). Essentially, multiple mechanisms exist with varying degrees of effectiveness in reducing haemodynamic disturbances on tissue oxygenation observed in laminitis treatments.

**Goals of this study:** Using information from clinical cases of laminitis and through owner survey information of horse diet and exercise the relationship between horse diet and the risk and severity of laminitis was investigated. In addition, using
results from this study and current research literature, a management plan for horse owners was developed to help prevent laminitis.
CHAPTER 3. MATERIALS & METHODS

Materials & Methods

Horse owners were surveyed and asked to give the following information about their horses: general physical examination information, dietary information, exercise schedules, turn out/pasture time schedules and medical histories. General physical information was collected including name, age, height, weight, breed and sex. Dietary information collected included specific feed/grain, and hay/forage offered.

Hay content was determined by the cutting of hay harvested. According to livestock nutrition author Heather Smith Thomas, the cutting of hay plays an important role in the nutritional content of various forages. Hay can be found in either 1st, 2nd or 3rd cuttings; this distinguishes the forages’ stage of growth during harvest. 1st cut alfalfa generally has high stem content. It is common to find weeds present in this particular cutting of hay. The second-cut alfalfa generally has a higher stem to leaf ration, and is lower in crude protein content. Typically, 2nd cutting alfalfa has 16 percent crude protein. The 3rd cutting of hay has a high leaf-to-stem ration due to the slowed growth that occurs during the beginning of the cool season. Specific cutting of hay was recorded in addition to specie(s) fed (Thomas, 2013).

Nutritional information for the commercial food types were gathered from the company’s web site (Purina and Nutrena). Data was also collected on two other non-commercial feed sources from the findings of Kentucky Equine Research, Inc indicating...
average nutritional values for rolled oats and beet pulp. Dietary information provided by owners was on a volume basis (cups, etc.) and was converted to weight by estimating that one quart of commercial feed was equivalent to one pound of feed. The conversion from scoops to pounds was provided by a blog from the feed company, *Nutrena*. *Nutrena* stated that 4 cups = 1 quart = 1 lb. According to *Nutrena’s* Feed Room Blog, a quart holds roughly 1 pound of horse feed.

Calculations were based on the grain averages collected from feed companies such as *Nutrena, Tribute* and *Purina*. Additionally, sources such as the Kentucky Equine Research, Inc. were consulted for average “home-grown” grain values.

According to Ohio State University College of Veterinary Medicine, on average, horses that are constantly in pasture graze at a rate of approximately 0.77 pounds of field grass/hour, while horses that are only in pasture for around 8 hours a day graze at approximately 1.32 pounds of field grass/hour. Using these averages, calculations for grazing calories consumed daily in both groups were completed as follows: *Laminitis Group*: 24 hours (0.77lbs of field grass/hour)(245 calories/lb of field grass) = 4,527.6 average calories consumed/day from field grass alone. *Reference Group*: 8 hours (1.32lbs/hour) (245 calories/lb field grass) = 2,587.2 calories consumed/day from field grass alone.
Additionally, exercise and time spent turned out in pasture was reported by owners. This included the average amount of time that the horse was ridden during the week as well as if the horse had access to a pasture. Exercise schedules were ranked based on a scale from 0 to 3 and defined as follows:

Level 0: Horse is not regularly worked

Level 1: Horse is worked 1-3 days a week at least 1-2 hours each day; including turn out/lounging.

Level 2: Horse is worked 3-5 days a week at least 1-2 hours each day; including turn out/lounging.

Level 3: Horse is worked for 6 days a week at least 1-2 hours each day; including turn out/lounging.

Of the horses with reported pasture time, the amount of time spent in pasture as well as the type of pasture was recorded. Two pasture types were reported: pastures with field grass and pastures without field grass. Time of day spent in pasture was also
recorded since several recent studies have suggested that the time of day in which a horse grazes can influence how much sugar is present within the grasses being consumed.

After collecting the dietary and exercise information, the general medical history of each subject was collected from the horses medical records with a special emphasis on if the horses had acquired any of the following diseases: laminitis/founder (severity was indicated as well as diagnosis and treatments), cribbing, Cushing’s disease/Equine Metabolic Syndrome, “Coggin’s”/Equine Infectious Anemia, lameness (not related to laminitis) and any other known diseases or conditions were reported. In addition treatment plans including medications, physical therapy, corrective shoeing, etc. were noted for each horse.

Lameness evaluations are a key part of the diagnosis and prognosis of laminitis patients. Lameness evaluations of all equine patients seen at the Cleveland Equine Clinic used in this study were conducted by Dr. Genovese and observed and assisted by the author of this study. Overall lameness evaluation included: flexion tests, recognizing gait abnormalities, and radiographs, as well as data collected while sitting in on Dr. Genovese’s owner consultations which included explaining radiograph findings, treatment plans with drug therapies, alternative shoeing, ice-baths, physical therapies and exercise plan changes.

Lameness data was analyzed using the scale developed by the American Association of Equine Practitioners. Patients were then ranked based on the severity of their lameness. Scale is defined as follows:
0: Lameness is not perceptible under any circumstances.

1: Lameness is difficult to observe consistently apparent regardless of circumstances (under saddle, circling, inclines, hard surface, etc)

2: Lameness is difficult to observe at the walk or while trotting in a straight line.

3: Consistently observe lameness at trot under all circumstances.

4: Obvious at the walk.

5: Lameness produces minimal weight bearing in motion or at rest or the lameness produces the complete inability to move.

Other data collected on laminitis patients included a pinch test, and evaluations of rotation, drop, and separation. The pinch test was completed on patients suspected of having laminitis. Hoof testers are used to apply pressure to the patient’s foot to look for sensitivity within the hoof capsule (figure 4). Identifying the sensitive area of the foot is crucial for designing a treatment plan. If the horse moves away quickly or pins its ears or presents any other behavior indicating pain, the horse is considered positive. If the horse does not react to the pinchers, the horse is negative. Scores were added to the overall value of lameness from the lameness evaluation. Horses that reacted in pain during the pinch test received a score of 1. Horses that did not react received a score of 0.
Rotation of the pedal bone for each laminitis patient was determined through a veterinary examination. Rotation was identified via radiographic findings. Owners reported their veterinarian’s diagnoses. Horses presenting rotation were assigned a value of 1. Horses not presenting rotation were assigned a value of 0.

Drop of the pedal bone for each laminitis patient was determined through a veterinary examination. Like rotation, drop of the pedal bone was also determined via radiographic findings. Owners reported their veterinarian’s diagnoses. Horses presenting with a drop in the pedal bone a commonly referred to as having “foundered”. Horses that had “foundered” were assigned a value of 1. Horses that did not have any drop were assigned a value of 0.

Separation between the bond of the hoof wall and pedal bone due to laminar necrosis was also determined via veterinary examination. Horses that presented separation were assigned a value of 1. Horses that did not present separation were assigned a value of 0.
CHAPTER 4. RESULTS

Results:

Data were collected on a total of ten horses, five horses presenting with clinical laminitis (the laminitis group) and 5 horses presenting with non-laminitis conditions (the reference group). The laminitis group consisted of 3 American Quarter Horses, 1 Shetland pony and 1 mixed breed pony. The reference group consisted of 2 Arabians, 1 Percheron/Dutch Warmblood, 1 Arabian/Hackney Horse and 1 Arabian/Saddlebred. Ages of the laminitis group ranged from 16 to 36. Ages of the reference group ranged from 5 to 14. Heights were measured in hands. Each hand is equal to 4 inches or 10.16 cm. Laminitis group heights ranged from 11 to 16 hands. Reference group heights ranged from 14.3 to 16.3 hands. The laminitis patient group was composed of 1 female and 4 male horses (all gelded). The reference group contained 2 females, 1 stallion and 2 geldings (neutered males)

An analysis of diet and exercise was conducted on data collected from horse owners to determine if differences exist between laminitis horses and the reference horse group. Grain, hay, and forage (determined by pasture time and pasture type) characteristics were examined as well as the amount of exercise each horse received.

Exercise values within the reference group ranged from minimal to high performance. The horse with the least exercise was hand walked for 3 hours a week only, while the most active horse was ridden by a competitive rider 6 days a week for multiple
hours per day. The laminitis patients ranged from minimal to mild work. The least active horse had severe arthritis and although living on pasture, walked minimally. The most active horse was used for mild work 2-3 days a week for 45 minutes at a time. Overall, the laminitis horses were exercised less than the reference group horses (figure 6).

Food consumption was compared based on estimates of daily caloric intake (figure 5). Laminitis horses averaged approximately 2X the total daily calories as compared to the reference horses, and had approximately 7X the caloric intake of field grasses. Calories from grain consumed were similar between laminitis horses and reference horses. As a measure of overall diet and health, the ratio of daily total caloric intake to exercise was calculated. Laminitis horses had a much higher ratio than the reference group horses indicating a more sedentary “glutinous” lifestyle (figure 7).

The overall comparison of the grain fed to horses was conducted using nutritional information provided by the manufacturer (figures 8 & 9). The grain comparison showed that the average percent of starch was greater in the diets of the reference horses. The largest difference in the grain given to horses was in the starch fraction. The reference horses had an average of 17% starch, while the laminitis group had an average of 12% starch. The other principle components of grain showed only small differences. Fiber content average was higher in the reference group with a value of 20% while the laminitis group had a value of 18%. The protein content was identical in both groups at 12%. The crude fat was higher in the reference group at 7%, while the laminitis group average was 5%. The reference group had an average of 7% sugar in their grain, while the laminitis group had an average of 6%. Comparison of the individual components of the
carbohydrates in the grain consumed showed only modest differences between the groups. Laminitis horses consumed more starch and fiber than the reference horses (figure 10). The nonstructural carbohydrate component of grains showed very little differences between the two groups.

All horses received hay as part of their diet in addition to grain. The reference group received 2nd cutting timothy/alfalfa hay mix. Laminitis horses hay diet was more varied with horses receiving the following hay types: 1st, 2nd and 3rd cutting timothy/alfalfa hay mix, oat hay and pure alfalfa hay. The reference group horses were all fed 2nd cutting timothy/alfalfa mixed hay. The 2nd cutting has a higher stem to leaf ratio than first cutting hay.

Forage availability was another consideration in analyzing the dietary content of the laminitis and reference groups. Forage availability is characterized by the availability of plant material in pastures with various species of field grasses and legumes. Horses allowed to roam in pasture (or “turned out”) have free choice of pasture grasses and legumes. All laminitis horses were in constant turn out (allowed to roam in pasture), while only 1 of the control group horses was regularly turned out for 8 hours a day. Pasture time was 24 hours a day for all horses in the laminitis group. In the laminitis group all but one patient had full access to field grasses during the day.

Another goal of the study was to examine how aspects of diet and exercise might be related to the severity of laminitis. Factors related to diet and exercise were examined for relationship with laminitis as reflected in the lameness scale. The lameness scale reflects one aspect of the severity of laminitis. Trends were found with three factors: the
amount of NSC (non-structural carbohydrates) in the diet, the amount of calories in the
diet from grain, and the horse’s weight. The strongest correlations were observed with
NSC and grain calories ($R^2$ 0.85 and 0.87 respectively; figures 13 and 14). A weaker
correlation was observed with horse weight (figure 15).
Figure 6: Average daily caloric intake.

Figure 7: Average daily exercise.

Figure 8: Ratio of total daily caloric intake to average daily exercise.
Figure 9: Average grain content among laminitic horses.

Figure 10: Average grain content among reference horses.
Figure 11: comparison of reference group and laminitis group grain content.

Figure 12: Hay cutting averages based on 1\textsuperscript{st}, 2\textsuperscript{nd} or 3\textsuperscript{rd} hay harvest.

Figure 13: Comparison between laminitis and reference group of average amount of time in pasture daily.
Figure 14: Lameness score vs. nonstructural carbohydrate content of grain in laminitis patients

Figure 15: Lameness score of laminitis patients compared to caloric content of grain

Figure 16: Lameness score vs. weight of laminitis patients
Discussion:

The purpose of collecting data from horse owners was to study which diet and exercise predisposing factors might be present in cases of naturally occurring laminitis. It has generally been known that diet can play a role in the development of laminitis. Recent research suggests that the development of laminitis might be linked to specific carbohydrates especially the sugar fraction, as well as insulin resistance.

After analyzing the grain composition and comparing hay types little difference was found in the specific dietary values. The carbohydrate fraction representing sugars and starches (NSC fraction) did not differ between laminitis and reference horses. However, a large difference between groups was observed after calculating the average amount of time each group spent in their pastures. This difference in pasture time suggests that a potential source of excessive sugar causing insulin resistance and predisposing horses to laminitis was coming from the excessive exposure to sugars in field grasses. The laminitis horses spent most of their time at pasture where they had access to field grasses.

While both the grains and hay types fed were similar between the reference group and laminitis patients, the reference group horses had far less access to sources with high nonstructural carbohydrate content, which is commonly found in lush field grasses, according to Joe Pagan of the Kentucky Equine Research Unit and Kathryn Watts of the
Rocky Mountain Research Center. Perhaps the largest concerns in allowing horses to graze excessively, is in the nonstructural carbohydrate content of grasses.

There are two primary types of carbohydrates, structural and nonstructural. According to the Kentucky Equine Research Unit, structural carbohydrates are carbohydrates that are resistant to the horse’s digestive enzymes. These carbohydrates are found in the cell walls of plants and must be treated with the equine’s enzymes; and eventually fermented by the horse’s gut flora for the nutritional content to be utilized. Nonstructural carbohydrates are more readily available to the horse. These carbohydrates exist primarily as monosaccharides. Monosaccharides are carbohydrate subunits composed of primary sugars such as fructose, glucose and galactose. These carbohydrates are found in the non-fibrous tissues of field grasses. (Pagan et al). Monosaccharides are the only form of carbohydrates that can be absorbed from the intestine. The more complex carbohydrates must be broken down into simple sugars before the animal can utilize the carbohydrates. There are several examples of nonstructural carbohydrates that are disaccharides and polysaccharides. Disaccharides are completely digested in the small intestine of the horse. This is not the case for one of the most important polysaccharides in the equine diet, starch. Starches are long strings of glucose molecules in both straight chains, amylose, and branched chain structures, amylpectin. Starches are converted into two maltose molecules by the enzyme amylase. Maltose, sucrose and lactose are split into two monosaccharide units by the disaccharide enzymes maltase, sucrase and lactase. The horse’s ability to produce amylase, the enzyme needed to digest these into subunits, is limited. Lactose is another disaccharide, composed of one glucose molecule and one
galactose molecule. Lactose is the sugar of milk. It also promotes the development of acidophilic organisms in the intestine and opposes the growth of undesirable putrefactive bacteria in foals. Lactose also has been shown to favor calcium and phosphorus assimilation, so it is the sugar of choice for the suckling foal. As horses age, their ability to digest lactose decreases, so large intakes of lactose in the adult horse may lead to diarrhea. An overview of the types of carbohydrates found in horse diets is given in Table I.

<table>
<thead>
<tr>
<th>Monosaccharides</th>
<th>Examples</th>
<th>Monosaccharide Subunits Present</th>
</tr>
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</table>
| Monosaccharides | **6 carbon sugars**: glucose, fructose and galactose.  
**5 carbon sugars**: arabinose, xylose and ribose. | ----- |
| Disaccharides   | **Sucrose** (table sugar)  
**Lactose** (milk sugar)  
**Maltose** (malt sugar) | **Sucrose**: glucose and fructose  
**Lactose**: glucose and galactose  
**Maltose**: glucose and glucose |
| Polysaccharides | **Amylopectin** (branch starch) | Glucose subunits in both. |

*Table I: Sugars and Subunits of Nonstructural Carbohydrates*
According to Tania Cubitt, PhD with Performance Horse Nutrition of Middleburg Va, field grasses contain largest quantities of sugar within the bottom 3 inches of the blade. The time of day directly affects the sugar content of field grasses. One particular type of sugar that has received a great deal of attention in recent laminitis research is found in soluble fibers, fructans. Fructans are classified based on the subunits present within the fiber; two subunits exist: inulin and oligofructose. Forages can vary significantly in their nutritional content. Season, climate, time of day, species of grass and even height of grass can alter the concentration of sugars within the grass that is most easily available to horses. Data analysis of the equine management techniques in the surveyed laminitis patients showed one major husbandry difference between the reference group and the laminitis group. All of the laminitis patients were in pastures for 24 hours per day. With the exception of only one patient, all of the lamanitic horses had constant access to field grasses. Therefore, it can be assumed that these horses had access to, and likely consumed, field grasses even in their various sugar rich phases. According to the Animal Health Foundation, the content of sugar within a single grass blade can very significantly throughout the year and throughout every day. Such access provides horses with excessive quantities of fructans, which are polysaccharide sugars composed primarily of the subunits, fructose and glucose. These excessive sugars are especially a concern when horses are grazed in lush grasses, grasses in early spring and early fall, grasses that are 3 inches tall or less, as well as grasses that have just been shocked by a freeze. Excessive sugars found in field grasses provide an abnormally high concentration of glucose molecules to the GI tract. A single horse is only able to digest so much glucose before
developing insulin resistance.

Non-structural carbohydrates aid in the increase of blood glucose levels, which lead to an increase in blood insulin levels. High insulin levels are only useful during the horse’s response to predatory situations, when the horse may need to escape to ensure its survival. Otherwise, high insulin levels are associated with diseases such as hyperactivity, insulin resistance, metabolic syndrome, obesity, developmental orthopedic disease and ulcers. Many of these diseases are observed in tandem with laminitis presentation. Elevated insulin levels can also make Cushing’s syndrome more difficult to manage. It is likely that many of the above problems develop chronically over a long period of time, especially obesity and insulin resistance (Geor et al. 2010).

In observing that laminitis can be induced in healthy ponies by maintaining supraphysiologic circulating insulin (1,000–1,100 mU/L) concentrations for 2 to 3 days suggests that hyperinsulinemia may play a direct role in the pathogenesis of laminitis in susceptible animals. Therefore, laminitis may be triggered in a chronically insulin-resistant horse or pony under conditions that exacerbate IR or hyperinsulinemia, for example, the grazing of pasture with high nonstructural carbohydrate content, as is found in seasonal and daily differences (during spring season after pastures are stressed by drought or frost, consumption of other feeds rich in starch and sugars (grains, sweet feeds), overfeeding that induces or worsens obesity, and the administration of corticosteroids (Geor et al. 2010).

Equine veterinarians have long recognized an association between metabolic abnormalities and an increased risk of laminitis. This is particularly obvious in horses
with obesity and insulin resistance. The insulin-resistant phenotype is strongly linked with a predisposition to laminitis presentation. Non-structural carbohydrates may vary well be the link between nutritional content that causes insulin resistance and predisposes horses to laminitis. Although wild horses survived primarily off of forages and grass based diets, laminitis seems to have become a disease of domestication and poor pasture management. One such disorder of domestication is the disease of obesity caused by high caloric intake and a sedentary lifestyle (lack of exercise). Laminitis horses in this study exercised less and consumed more calories than did the non-laminitis horses.

In addition to excessive access to field grasses, diseases such as insulin resistance, may have been present (at least in early stages) in several of the laminitis patients. This was suspected because as is often the case in humans, horses can have insulin resistance for many years before they show obvious symptoms. Identifying the symptoms of insulin-resistant ponies and horses facilitates preemptive avoidance measures in two ways. These strategies focus on the improvement of insulin sensitivity (such as caloric restriction, exercise management, and drug therapies) and the strategies minimize exposure to environmental factors known to induce laminitis (such as restricted access to lush pasture grasses and the elimination of grains and sweet feeds from the diet entirely) (Geor et al., 2010).

Pratt et al., 2006 demonstrated that feeding a diet rich in NSC for 6 weeks resulted in decreased insulin sensitivity and impaired glucose tolerance. Physical conditioning lessened the effects of the high NSC diet on insulin sensitivity, as evidenced by their return to baseline, but did not mitigate the impaired glucose tolerance.
Tribute feed company nutrition specialists observed the rate at which NSCs reach the small intestine in two different feed types to analyze variation in metabolism rates. The two forms of feed included both hay and a concentrated feed, each containing 15% NSCs. The rate of entrance into the small intestine varies drastically between the two groups. The concentrated feed enters the small intestine at a much higher rate than the hay. Understanding the difference in metabolism provides insight into equine husbandry management techniques.

Allowing for slower glucose/insulin response from the body (as is the case in the hay example), allows for a more gradual reaction to insulin as the hormone facilitates the metabolism of dietary sugars. Thus, decreasing the risk of creating dietary induced insulin resistance can be accomplished via slow administration and decreased quantities of NSCs. It is also important to recognize there is a tremendous variation in how individual horses respond to nonstructural carbohydrates at various concentrations. Horses in training, competition, growing or lactating have a higher NSC tolerance than the less active mature horse (especially horses with metabolic/endocrine disease). Excessive grazing could very well be the source of excessive sugar suspected in laminitis patient diets.

The results of this study demonstrated that the diets of laminitis patients and the reference group patients varied most significantly in one aspect, the “turn-out”/pasture time which can be an indirect measure of the availability of field grasses. While the type of grasses consumed was not measured in this study, it is a safe assumption that horses at pasture feed on grasses over other types of plant forage. This would lead to a potentially
higher consumption of NSC by the laminitis horses. This coupled with a higher total caloric intake and a lower level of exercise, the laminitis horses did present with multiple risk factors for developing laminitis. Additional evidence to suggest that NSC plays a role in laminitis was found in this study by the strong correlation between the amount of NSC consumed in grain and the severity of the laminitis.
Management Techniques for Laminitis Prevention: In addition to examining predisposing factors involved in the development of laminitis, another goal of this study was to summarize the information found in this study and other laminitis and equine nutrition studies and convey this information to horse owners in a non-scientific manner. This management section is composed of techniques for recognizing predispositions, techniques for making a horse comfortable while waiting for emergency veterinary assistance as well as techniques for the long-term management of chronic laminitis.

1. Understanding which horses are most “at risk” for developing laminitis

Horses of all breeds, sexes, sizes and ages are at risk for laminitis. However, management techniques can influence whether or not the risks of predisposition factors will manifest into laminitis. Predisposition factors include obesity (high caloric intake and lack of exercise), having certain diseases such as endocrine/metabolic disease. According to Animed Veterinary Hospital & Equine Unit, these endocrine/metabolic diseases include: insulin resistance, Cushing’s disease, Pituitary Pars Intermedia Disease (PPID), Equine Metabolic Syndrome (EMS). Mechanical trauma, such as concussive forces (as seen when the hoof hits hard ground repeatedly) from intense exercise over long periods of time can drastically increase risk of laminitis. This is due to the cell death that can be caused and eventually lead to the separation of the tissue within the hoof capsule. Injuring the sensitive laminae repeatedly can result in necrosis (tissue death), which can
accelerate laminitis presentation. Improper shoeing can also function in a similar manner as uneven footing may provide excessive pulling on the medial or deep digital flexor tendons which can destabilize and move the distal phalanx bone (the last bone in the leg) out of place. See Smartpak’s *The Anatomy of Laminitis in Horses* informational video for a demonstration of how intimately linked the pulling of tendons is with overall laminar health ([https://www.youtube.com/watch?v=ubUMx2SrK4Y](https://www.youtube.com/watch?v=ubUMx2SrK4Y)). Like endocrine/metabolic syndromes, colic is often observed in tandem with laminitis especially in cases following carbohydrate overload. Colic is a very general term for abdominal upset in horses, like a terribly painful stomachache. Upon eating excessive carbohydrates or breaking into a grain bin, colic can initiate a metabolic response that can indirectly lead to laminitis. As is the case with endocrine/metabolic disease, toxins produced by the change in the horse’s hindgut bacteria result from the initial carbohydrate overload. The new bacterial colonies (now composed of more gram-positive bacteria) produce toxins, which get absorbed into the horse’s blood stream. This introduction of toxins is accelerated by the horse’s immune response, swelling of the laminae; the introduction of toxins causes cell death in the laminae and eventually results in tissue degradation (Belknap et al., 2010).

**Signs to look for:** If your horse has regional adiposity, a history of metabolic/endocrine disease (such as Cushing’s syndrome, Equine Metabolic Disease and insulin resistance), changes in the hoof structure or obesity, contact your veterinarian to discuss a prevention plan. Horses with insulin resistance or metabolic disease often exhibit an arched neck and fatty deposits in the eye socket region and above the horse’s tail. Regional adiposity occurs when a horse accumulates localized regions of fat
commonly resulting in an arched neck, eye sockets looking more concave than convex and sometimes localized fat above the poll of the horse’s tail. These fatty deposits found in these specific areas often indicate early stages of metabolic/endocrine diseases.

Hoof wall/white line changes such as white line disease may suggest laminitis. Bone rotation occurs in many cases after the destruction or weakening of the laminae from white line disease. When the white line on a horse’s hoof changes in size, this can be an early stage indicator of laminitis. A white line becomes most visible on newly trimmed feet; in which the stratum medium layer of the hoof is visible (this is the white line). If untreated, advanced cases allow for the degradation of the hallow space that forms between the inner and outer layer of the hoof/hoof wall; at which point the wall pulls away from the rest of the foot.

Change in hoof shape from concave to convex (suggests founder has occurred). Lameness, particularly lameness in which a gait abnormality is established in observing the horse stretch its feet farther out farther in front of itself than it usually does with its hind legs further forward than normal. When horses take the traditional “laminitic” stance, they often bear the majority of their weight on their hind legs to alleviate pressure on the sensitive laminae since laminitis generally presents more severely in the front hooves. Think about the consistency of a hard pear versus an over ripe pear; the hard pear is structurally very solid and takes substantial force to break it, while an overly ripe pear is soft and “squishy” and has a very limited structure. Healthy laminae are like the hard pear; they provide support and stabilization for the pedal bone and keep the bone in place.
However, like the soft pear, damaged laminae do not properly support the pedal bone, and can be easily destroyed by excessive concussive forces.

2. Measuring carbohydrates in horse feed

It is preferable to indirectly estimate the sugar content of feeds and forages by measuring several components of a feed and calculating non-structural carbohydrate (NSC) content. The equation to calculate NSC on an as fed basis is: NSC (%) = 100 - moisture (%) - crude protein (%) - crude fat (%) - neutral detergent fiber (NDF) - ash (%). This calculation will measure all forms of sugar in the feed including simple sugars as well as polysaccharides such as starch. All of these carbohydrates will be absorbed as simple sugars if they are digested by the horse’s digestive enzymes, so nutritionally speaking they can be lumped into this single category. Typically, the NSC content is lowest in straw and mature hays. Legume hays are usually higher in NSC than grass hays and cereal grains have the highest concentration of NSC. Molasses is also high in NSC, containing a level between corn and barley, but its overall. As a rule, the lower the cell wall content of a feed, the higher the NSC and energy density. This is because horses digest over 95% of the NSC and typically only about 40-50% of the cell wall. There are certain feedstuffs, however, that contain much more digestible cell wall such as beet pulp and soy hulls.
3. Prevention is the best medicine

**Poor management** can accelerate predispositions directly into the early stages of laminitis. Management techniques to avoid include: providing excessive access to and consumption of lush field grasses, poor shoeing, providing inadequate exercise in combination with excessive calories, and the use of black walnut bedding (Geor et al. 2010). A poor understanding of pasture biology is the primary factor in equine management that may accelerate laminitis presentation and disease progression

4. So your horse has been diagnosed with laminitis...

**Long term:**

Consider making feed/orage changes under the supervision of your veterinarian. To encourage exercise without grazing, put a grazing muzzle on your horse before turning him/her out to pasture. Change your horse’s shoeing if your veterinarian believes your horse needs corrective shoeing. Changing the feeding program for each laminitis patient from a diet high in NSCs (nonstructural carbohydrates) such as high-corn, high-molasses sweet feed to a diet low in nonstructural carbohydrates and high in fiber may delay the onset of laminitis or reduce the severity of laminitis symptoms (Geor et al., 2010). Overweight horses benefit from the exercise of roaming in pastures; to give a horse the benefit of exercise without the additional nonstructural carbohydrates of field grasses, a grazing muzzle can be attached to the horse’s halter to stop grazing activities.
If you suspect an episode: Hygain, an Australian feed company, has an excellent video on how to respond to early laminitis symptoms (see link below). It is crucial to understand that laminitis is excruciatingly painful for horses and regardless of the stage, should ALWAYS be treated as a medical emergency.

(https://www.youtube.com/watch?v=U36lEO1ZUy8)

The 3 Simple Steps of Emergency Laminitis Care:

1.) Pull the horse’s shoes to minimize damage to the hoof.

2.) Wrap the feet with vet wrap to provide support.

3.) Ice the feet.
Bailey, S, *Current research and theories on the pathogenesis of acute laminitis in the horse*: Department of Veterinary Basic Sciences, Royal Veterinary College, North Mymms, Hatfield, Hertfordshire, UK 2003


White, *EQUINE LAMINITIS*, Marion duPont Scott Equine Medical Center VA-MD Regional College of Veterinary Medicine Virginia Tech Leesburg, Virginia 22075 2015;


Eades, *A Review of the Pathophysiology and Treatment of Acute Laminitis: Pathophysiologic and Therapeutic Implications of Endothelin-1*, 2002


Harris, P, *Countermeasures for Pasture-Associated Laminitis in Ponies and Horses*, The WALTHAM International Nutritional Sciences Symposia; 2012


Insulin Resistance, January; 2014; Available from:

Gayle, R; Nutrena The Feed Room Blog January; 2013; Available from:

Smith Thomas, H, All Hay Is Not Equal: Choose Your Livestock’s Carefully Hay is the mainstay diet for our livestock. Learn the intricacies of hay types, nutritional content and quality before purchasing your next load. January; 2013; Available from:

Fonnesbeck, P, Effect of Diet on Concentration of Protein, Urea Nitrogen, Sugar and Cholesterol of Blood Plasma of Horses. January; 1969; Available from:

Feeding Suggestions For Horses, January; 1987; Available from:
NUTRITION FUNDAMENTAL SERIES: NONSTRUCTURAL CARBOHYDRATES


APPENDIX

Case Studies:

The control group consisted of multiple horses of various breeds, ages, sizes and backgrounds. These horses were patients at Cleveland Equine Clinic for reasons not related to laminitis.

1. Horse Name: Dax
   Height: 15.1 h
   Weight: 900 lbs
   Age: 14
   Breed: Arabian/Hackney horse mix
   Sex: M (gelding)
   Exercise schedule: level 1
   Turn out/pasture time: None
   Diet: ¼ pound of HealthyEdge and 2 flakes 2nd cutting timothy alfalfa mix hay twice daily.
   Medical history: Cribber. Frequent gas colic (medical, not surgical colic). No history of lameness

2. Horse Name: Gypsy
   Height: 15 h
   Weight: 850 lbs
Age: 12
Breed: Arabian
Sex: F
Exercise schedule: level 1
Turn out/pasture time: None
Diet: ¾ pound of *HeathyEdge* and 2 flakes 2nd cutting timothy alfalfa mix hay twice daily.
Medical history: injury related (3/5) lameness (from trailer accident). No history of laminitis.

3. Horse Name: Brigandell

Height: 16.3 h
Weight: 1,200 lbs
Age: 5
Breed: Percheron/Dutch Warmblood cross
Sex: M (gelding)
Exercise schedule: level 3
Turn out/pasture time: 8 hours daily on field grass (weather permitting)
Diet: 2 scoops rolled oats and 4 flakes alfalfa/timothy hay daily
Medical history: none (pre-purchase examination)
4. Horse Name: Elvis

Height: 14.3 h
Weight: 800 lbs
Age: 5
Breed: Arabian
Sex: M (stallion)
Exercise schedule: level 3
Turn out/pasture time: none
Diet: ¾ pound of *HeathyEdge* and 2 flakes 2nd cutting timothy alfalfa mix hay twice daily.
Medical history: lameness due to splint found on radiographs (RF leg). Eye ulcer.

5. Horse Name: Hallie

Height: 15.2
Weight: 950
Age: 10
Breed: Arabian/Saddlebred
Sex: F
Exercise Schedule: 2
Turn out/pasture time: 0
Medical History: bone chip RH (no lameness).
**Laminitis Patients:** these patients were collected from the Cleveland Equine Clinic and from local private barns in the Ohio/Pennsylvania region. One additional subject was treated in California.

6. Horse Name: Badger
   - Height: 15.1 h
   - Weight: 1,000 lbs
   - Age: 20
   - Breed: American Quarter Horse
   - Sex: M (gelding)
   - Exercise Schedule: level 2
   - Turn Out/Pasture time: constant turn out with field grass
   - Diet: 1 lb 12% Nutrena pelleted grain daily with round bale of 2\textsuperscript{nd} cutting alfalfa/timothy hay mix and field grass access
   - Medical History: laminitis with 10-degree rotation in each foot at age 8.
   - Diagnosis: founder/laminitis
   - Treatment Plan: Bute, corrective shoeing, cold soaking hooves to reduce inflammation.

7. Horse Name: Shandy
   - Height: 12 h
   - Weight: 400 lbs
Age: 36
Breed: pony mix
Sex: F
Exercise Schedule: level 0 (due to medical issues)
Turn Out/Pasture time: constant
Diet: 2/3 lb senior pellets and 2 flakes 1st cutting alfalfa/timothy hay mix (tender pieces not full patty of hay)
Medical History: extreme arthritis, founder and lamintis
Diagnosis: founder/laminitis
Treatment Plan: Bute, not in work.

8. Horse Name: Doc
Height: 15.2
Weight: 980 lbs
Age: 26
Breed: American Quarter Horse
Sex: M (gelding)
Exercise Schedule: none – retired for 8 years.
Turn Out/Pasture time: constant turn out.
Diet: 1 lb 12% senior feed once daily and 2 c. beet pulp mixed with 2 T maple syrup drizzle daily. Summer – all field grass. Winter – square bale of 2nd and 3rd cutting hay.
Medical History: shoulder trauma lameness from car accident at age 4.5  
(movement was not completely limited, but lost chunk of shoulder). Sound to ride  
from age 8-16. Respiratory issues when stalled year round.  
Diagnosis: hoof wall separation and extreme laminitis (5/5).  
Treatment Plan: .5 g Bute with 1 oz glucosamine daily.

9. Horse Name: Lightning McQueen

Height: 11 h

**Weight:** 450 lbs

Age: 16

Breed: Shetland pony

Sex: M (gelding)

Exercise Schedule: level 2

Turn Out/Pasture time: overnight turn out (8-12 hours)

Diet: 1 lb Purina senior feed with 2 flakes of 1st cutting alfalfa/timothy mix hay twice daily.

Medical History: during the summer, turned out over night on field grass (grass is  
usually richer in sugar at night). Moved barns to pasture with grass that had even  
higher sugar content.

Diagnosis: laminitis (3/5)

Treatment Plan: diet change (no more field grass) and bute
10. Horse Name: Tripp

Height: 16 h

Weight: 1,200 lbs

Age: 16

Breed: American Quarter Horse

Sex: M (gelding)

Exercise Schedule: level 2.

Turn Out/Pasture time: constant turn out, no field grass present.

Diet: 1 lb equine senior twice daily. 2 flakes alfalfa hay (am) with 2 flakes oat hay (pm)

Medical History: no other

Diagnosis: (5/5 initially, now 2/5 after starting treatment) laminitis in both front feet due to poor hoof trim (heels too short, toes too long… leaving soles flat and all 4 feet bleeding) Pasture ice caused bruising on all 4 feet)

Treatment Plan: 1 ½ g Bute daily (able to walk without signs of lameness). Diet change, now getting Bermuda Grass hay (am) with 4 c soaked timothy pellets in (pm)

Within the reference group, the following diseases were reported:

2 cases of lameness – one of which was due to a flesh wound injury (lameness level 3/5), the other was due to a bone splint (lameness level 2/5).
History of “cribbing” and medical colic was reported in one patient.

Cribbing is a condition in which the patient in question swallows air excessively in large gulps which can lead to stomach ulcers and or colic.

Eye ulcers – were reported in one patient.

Within the laminitis patients, the following diseases were reported:

1 case of shoulder trauma – this occurred from a road accident that resulted in a loss of a few inches of shoulder muscle (though range of motion remained).

1 poor farrier job – this occurred after a trimming left one of the patients with hooves that had heels that were excessively short, toes left excessively long and resulted in an unstable hoof conformation that was believed to have induced the laminitis (according to the patient’s veterinarian).

![Age Comparison](image)

**Figure 17. Age Comparison**
**QUIZ: IS YOUR HORSE AT RISK FOR DEVELOPING LAMINITIS?**

1.) Does your horse get turned out daily?
   - Yes
   - No

   If No, go to 2.

2.) Does your horse get exercised for at least 5 hours a week?
   - No
   - Yes

   If No, go to 2.

   If Yes, go to 3.

3.) Does your horse have access to field grass while turned out?
   - Yes
   - No

   If No, go to 2.

   If Yes, go to 4.

4.) During what time of day is your horse out in pasture?
   - Overnight
   - Constant turn out
   - Afternoon / evening

   If Overnight or Constant turn out, add 1 point. If Afternoon / evening, add 2 points.

5.) Does your vet consider your horse overweight/obese?
   - No
   - Yes

   If No, go to 6.

   If Yes, add 2 points.

6.) Do you feed more calories than recommended?
   - No
   - Yes

   If No, add 2 points. If Yes, add 4 points.

7.) Does your horse have a history of endocrine disease? (Cushing's syndrome, Insulin Resistance, Equine Metabolic syndrome or thyroid disease)
   - No
   - Yes

   If No, add 2 points. If Yes, add 3 points.

**Scoring and Recommendations**

**0-2: Low Risk**
Great work! Your horse is not likely to develop laminitis with your current management techniques.

**3-4: Moderate Risk**
Consider making changes to your horses exercise and grazing schedules. Additionally, talk with your vet about dietary needs.

**5-6: High Risk**
If your horse has not developed laminitis thus far, it is CRUCIAL to talk with your vet about prevention techniques.