Nutrition and Claw Health

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Abstract

The health and function of the bovine claw is dependent upon sound nutrition and feeding practices. In this context, the avoidance of rumen acidosis, which is considered to be the predominant predisposing cause of laminitis, is believed to be of paramount importance. Acidosis in its acute form is a life threatening disease. In its subclinical form, acidosis contributes to decreased performance, poor body condition, and lameness most often due to laminitis and related claw disorders. In addition to being the single largest component of the dairy cow's diet, the one most often incriminated in rumen acidosis and laminitis is carbohydrate. The rapid fermentation rates of certain non-structural carbohydrates place desirable rumen microbes in jeopardy. Therefore, rations must be carefully formulated and fed to avoid potential problems. Not all studies reported in the literature have been able to demonstrate an association between rumen acidosis and laminitis. These inconsistencies substantiate the view of most people that laminitis is multi-factorial and likely complicated by many other factors. Rumen pH is a balance between the acid produced by carbohydrate fermentation and rumen buffering from saliva. Heat stress contributes to rumen acidosis by altering feeding behavior (encouraging slug feeding) and reducing salivary buffering. Although occasionally questioned as a cause of laminitis, the effect of elevated levels of dietary protein in dairy cattle diets has not shown conclusive evidence of contributing to laminitis. Research into the role of vitamins, particularly biotin, suggests significant benefits to claw health. Similar information exists on the role of minerals and trace minerals in dairy cattle diets. A claw healthy diet should include appropriate supplementation of both vitamins and minerals to support the proper growth and development of claw horn. Laminitis results from disrupted blood flow in the corium that leads to damage of the dermal-epidermal junction and the underlying connective tissue matrix of the corium. Inflammation predisposes to the activation of matrix metalloproteinases which break down the strong collagen fiber bundles of the suspensory apparatus of 3rd phalanx **(P3)**. This permits sinking and rotation of P3 and predisposes it to the ulcers of the toe, sole, and heel. There are, however, alternate theories that suggest hormonal changes associated with calving may be major contributors to weakening of the suspensory apparatus. If these observations are correct, it may help to explain those inconsistencies in the literature and those observed clinically that do not show a clear relationship between laminitis and nutrition.

Introduction

The management of feeding and nutrition are the primary areas of interest when attempting to reduce lameness problems. This may or may not be the correct approach depending upon the specific types of lameness experienced. For example, it would be hard to influence the incidence of infectious foot diseases (foot rot, interdigital dermatitis, or

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digital dermatitis) by manipulation of the diet alone. Laminitis and claw disorders share a closer relationship to metabolic disease disorders which are often linked to nutrition and/or feeding issues. Cow comfort considerations are also critical factors in sorting out lameness problems and must be evaluated in herd problem situations as well. However, for the purposes of this discussion, our attention will be on nutrition and claw health.

Rumen Acidosis

Acidosis is generally associated with the ingestion of large amounts of highly fermentable carbohydrate-rich feeds which ultimately result in the excessive production and accumulation of lactic acid in the rumen. In it's acute form, the disease is characterized by severe toxemia, ataxia, incoordination, dehydration, ruminal stasis, weakness, and recumbency. The mortality rate is high. The subclinical form of rumen acidosis (better known as **SARA**, for Sub-Acute Rumen Acidosis) is far more common than the acute form of this disease. Major clinical manifestations would include variable feed intake, depressed fat test, poor body condition despite sufficient energy intake, mild to moderate diarrhea, and occasional cases of epistaxis (nose-bleed) or hemoptysis (the expectoration of blood from the mouth). Conditions such as laminitis or undefined lameness, abomasal disorders, and liver abscesses are generally secondary observations (Nocek, 1997; Nordlund, 2002).

Although few studies have been able to establish a direct link between rumen acidosis and laminitis, most assume that the feeding program is a major underlying factor. In reality, much of the information ascribed to cattle is based on information from studies of starch overloading models in horses (Garner et al., 1975; Vermunt and Greenough, 1994). Recent work suggests that an oligofructose overload model may be appropriate for the study of acute bovine laminitis. Researchers were able to successfully create classical symptoms of rumen acidosis and laminitis in cows treated with an alimentary oligofructose overload (Thoefner et al., 2004). The following is an attempt to identify some of the more important predisposing factors relative to nutrition and feeding of dairy cattle.

Nutrition and feeding considerations

Rumen fermentation disorders that result in acidosis are typically traced to diets with excessive levels of highly fermentable carbohydrates and inadequate levels of effective fiber (Nocek, 1997). Even with high quality ingredients and proper formulation of the diet, what ends up in front of the cow is still at the mercy of those responsible for mixing, delivery, and management of the feed bunk. Add to these selective eating or feed sorting behavior of cows (Leonardi and Armentano, 2000) and it's easy to see that there's ample room for error. Equally important are dietary changes that naturally occur during the cow's lactation cycle. In recent years, nutritionist's have concentrated their attention on feeding programs during the transition period in an attempt to ease the adjustment of cows to higher energy rations necessary to sustain milk production. A Florida study concluded that large differences in the fiber and net energy content of closeup and early lactation diets can contribute to an increase in the incidence of rumen acidosis and subclinical laminitis (Donovan et al., 2004).

Carbohydrate

Feeding rations high in non-structural carbohydrates to animals that are not sufficiently adapted has the potential to result in a lowered rumen pH. Lowered rumen pH is associated with a change in the rumen microflora from predominantly gram-negative to predominantly gram-positive lactic acid-producing bacteria. Coincident with this change in rumen pH and microflora is the release of endotoxin from the outer cell walls of dying and disintegrating gram-negative bacteria. Aided by a damaged and dysfunctional rumen mucosa, lactic acid, endotoxin,

and possibly histamine, are absorbed into the blood stream. These products are rapidly dispersed to the micro-circulation of the corium, where directly or indirectly (through vaso-active mediators), blood flow is disrupted leading to the lesions observed in laminitis (Nocek, 1997; Vermunt and Greenough, 1994).

While there is little dispute that rumen acidosis may occur as described above, it's not clear that laminitis will inevitably occur as a consequence. Three studies observed no correlation between laminitis and the feeding of rations high in carbohydrate (Smit et al., 1986; Frankena et al.,1992; Momcilovic et al., 2000). Despite conflicting information in the literature, one would still have to conclude that there seems to be an association (albeit complex) between carbohydrate nutrition, rumen acidosis, and laminitis, but more research is needed to sort out the details of these relationships.

Protein

Feeding high levels of protein in the diet of dairy cows and the potential to cause laminitis or lameness is surely less well understood. Outbreaks of laminitis in calves fed milk replacer and starter rations containing 18% digestible protein are reported from Israel (Bargai et al., 1992). Calves affected were 4 to 6 months of age and had lesions in their claws consistent with severe acute laminitis. Although this is an interesting observation, most would view the suggestion that high protein was the cause of this problem with significant skepticism, since milk replacers and rations containing 18% protein (or higher) are commonly fed to calves and young stock throughout North America without incident. On the other hand, results of a Canadian study found no relationship between the level of protein fed and lesions associated with laminitis (Greenough et al., 1990; Greenough, 1990). In consideration of the above information, one must conclude that there is simply insufficient information to know what effects, if any, protein may have on foot health.

Vitamins

Vitamin deficiencies sufficient to cause obvious disease are relatively rare under modern feeding conditions. More common are those conditions where vitamin levels are sufficient to prevent the occurrence of clinical disease but possibly insufficient to support optimum growth and performance. For example, rickets from a deficiency of Vitamin D is extremely uncommon since hay and exposure to sunlight normally provide the cow with ample quantities of this vitamin. On the other hand, sporadic instances of white muscle disease associated with Vitamin E and selenium deficiency occur in un-supplemented animals raised in areas where soils are normally deficient in selenium. Sudden death or calves exhibiting a generalized weakness or stiffness of the legs may be observed in animals affected. Vitamin A has important roles in the maintenance of epithelial tissues, including claw horn.

The B-Vitamins are synthesized by rumen micro-flora, and therefore, until recently, rarely fed to dairy cattle. The one exception in recent time is biotin. Biotin is essential for keratin-protein synthesis and the formation of long-chain fatty acids that make up the intercellular matrix of claw horn (Mulling et al.,1999). Canadian research suggested that cattle fed high grain diets are subject to potential biotin deficiency since the rumen microbes responsible for biotin synthesis are sensitive to low rumen pH (Girard, 1998). Since then, several feeding trials with biotin supplemented at a rate of 20 mg/day have shown benefits to claw health, including: an improvement in the healing rate of sole ulcers (Lischer et al., 1996; Koller et al., 1998), a decrease in the occurrence of vertical wall cracks in beef cattle (Campbell et al., 1996), an improvement in white line health (Midla et al., 1998), a decrease in the incidence of lameness in pastured dairy cattle in

tropical Australia (Fitzgerald et al., 2000), reduced the incidence of sole hemorrhages and increased milk production in biotin-supplemented cows (Bergsten et al., 2002), improved horn quality and strength (Koster et al., 2002), and improved white line health (Hoblet et al., 2002). While cost and a lack of scientific information were once reasons to question the value of biotin supplementation, current cost and a growing body of scientific information suggests that biotin is worthy of consideration in the diets of lactating dairy cattle.

Minerals (including trace minerals)

Minerals have at least 3 broad functions in the animal's body: 1) as structural components of body organs and tissues, 2) as constituents of body fluids and tissues where they function to maintain proper osmotic pressure, acid-base balance, and membrane permeability, and 3) as catalysts in enzyme and hormone systems (Underwood, 1981). The specific role of minerals with respect to foot health have been reviewed previously (Socha, et al., 2002; Tomlinson et al., 2004).

One of the macro-minerals of greatest interest relative to claw horn integrity is calcium **(Ca)**. The differentiation of keratinocytes in claw horn epithelium requires Ca for the proper function of enzymes in biochemical pathways that ultimately result in the proper keratinization of horn cells (Nocek, 1997). Any deficiency that may occur, such as with hypocalcemia during the peripartum period, would have the potential to negatively influence normal maturation of keratinocytes and thus affect the integrity of horn produced during this period (Mulling et al., 1999). In view of the fact that hypocalcemia and lameness are both common disorders in dairy cattle, this would seem an area of interest for further research.

The trace minerals zinc and copper play important roles as enzyme catalysts in keratin synthesis. At least 2 studies have reported an improvement in foot health from the feeding of a zinc methionine complex or organic zinc in a corn and grass silage-based diet (Moore et al., 1988; Reiling et al., 1992). The role of copper in keratin synthesis is through the enzyme thiol oxidase, a key enzyme in the biochemical pathways necessary for the cross-linking of keratin filaments within the keratinocyte. Cross-linking of keratin filaments impart strength to the cell, making it more resistant to mechanical and physical forces (Socha et al., 2002).

Selenium and Vitamin E are known to have important functions with respect to the resistance of animals to infectious diseases. Selenium functions within the cytosol of the cell as a co-factor for the enzyme glutathione peroxidase to protect cells and tissues from oxidative damage. Vitamin E serves as a specific lipid-soluble antioxidant in the membrane of the cell where it protects the cell from chain reactive auto-oxidation of membrane lipids. While specific data on foot health and selenium supplementation are lacking, one might expect increased resistance to infectious foot diseases in animals whose selenium and Vitamin E requirements are met.

Heat stress and rumen acidosis

The primary avenues for heat loss during periods of hot weather are sweating and panting. In severe heat, panting progresses to open-mouth breathing, characterized by a lower respiratory rate and greater tidal volume. The result is respiratory alkalosis caused by the increased loss of carbon dioxide. The cow compensates by increasing urinary output of bicarbonate **(HCO_3)**. Simultaneously, the salivary HCO_3 pool for rumen buffering is decreased by the loss of saliva from drooling in severely stressed cows. The end result is rumen acidosis because of reduced rumen buffering and an overall reduction in total buffering capacity (Dale et al., 1954).

The effect of ambient air temperature on rumen pH was evaluated in lactating Holstein cows fed either a high roughage or high concentrate diet in both a cool (65°F with 50% relative humidity) and a hot (85°F with 85% relative humidity) environment. Rumen pH was lower in cows exposed to the higher temperatures and those fed the higher concentrate diets (Mishra et al., 1970). These observations have been corroborated by others (Bandaranayaka and Holmes, 1976; Niles et al., 1998), supporting the current view that increasing the energy density of rations to compensate for reduced dry matter intake during periods of hot weather is not without significant risk.

Connection Between Rumen Acidosis, Laminitis, and Lameness

The dermal-epidermal junction is a highly specialized region within the claw that serves as the interface between the vascular and non-vascular tissue. It is also the specific site of the lesion of laminitis characterized by sinking and rotation of the P3 and the accelerated production of poorer quality claw horn. For the purposes of understanding the lesions as they occur at the cellular level, it is important to have at least a mental picture of this region.

Corium (or dermis) and epithelium

The corium consists of connective tissue with a rich supply of blood vessels and nerves. Adjacent to the corium (moving in the direction of the claw horn surface) is the basement membrane, germinal epithelium, stratum spinosum, and finally, the stratum corneum, otherwise known as the horn layer. Although they have no direct blood supply, cells within the lower layers of the epithelium (germinal epithelium and lower layers of the stratum spinosum) are "living cells" by virtue of nutrients and oxygen received from the underlying corium by diffusion across the basement membrane. The germinal layer is an active region of cellular proliferation and differentiation. Cells within this layer that differentiate into keratinocytes (cells capable of producing and accumulating keratin) will gradually move outward into the stratum spinosum. As they do, they continue to produce and accumulate keratin proteins. Eventually, cells migrate sufficiently away from the corium that they no longer receive an adequate supply of nutrients and oxygen. At this stage, they begin to undergo the process of death and cornification (formation of cells into horn). Clearly, any condition resulting in a disruption in the flow of blood to the corium not only affects the corium, but also the epithelium and thus, the integrity of claw horn.

Laminitis - lesions at the cellular level

The pathogenesis of laminitis is believed to be associated with a disturbance in the micro-circulation of blood in the corium which leads to breakdown of the dermal-epidermal junction between the wall and P3. As described earlier, rumen acidosis is considered to be a major predisposing cause of laminitis and presumably mediates its destructive effects through various vasoactive substances (endotoxins, lactate, and possibly histamine) that are released into the blood stream in coincidence with the development of rumen acidosis and the subsequent death of rumen microbes. These vasoactive substances initiate a cascade of events in the vasculature of the corium, including a decrease in blood flow caused by veno-constriction, thrombosis, ischemia, hypoxia, and arterio-venous shunting. The end result is edema, hemorrhage, and necrosis of corium tissues, leading to functional disturbances including the activation of matrix-metalloproteinases **(MMP)** that degrade the collagen fiber bundles of the suspensory apparatus of the P3 (Boosman et al., 1989). This is complicated still further by the activation of horn growth and necrosis factors that result in structural alterations involving the basement membrane and capillary walls (Mulling and Lischer, 2002).

Changes occurring in the epidermis are secondary to vascular disturbances that result in reduced diffusion of nutrients from the dermis to the living layers of the epidermis. This interrupts cellular differentiation and proliferation in the germinal layer, and the keratinization of epithelial cells in the stratum spinosum. The quality of claw horn is dependent upon keratinization which gives the horn cell structural rigidity and strength. In conditions resulting in vascular compromise, such as laminitis, the keratinocyte may become injured or inflamed from being deprived of nutrients. The end result is the production of poorly keratinized (weak or inferior) horn that weakens the claw horn capsule's resistance to mechanical, chemical, and possibly even microbial invasion. Thus, the term *claw horn disruption* has been proposed as possibly a more appropriate term for laminitis and particularly subclinical laminitis (Logue et al., 1998).

Laminitis - sinking and rotation of the third phalanx

The weakest link between the attachment of P3 to the basement membrane of the epidermis (referred to as "the locus minoris resistentiae") is at the dermo-epidermal junction (Mulling and Lischer, 2002). This region is also referred to as the "suspensory apparatus" and includes all structures between the surface of the bone and the inner aspect of the cornified claw capsule (that is, the inner layers of the epithelium up to and including inner portions of the stratum corneum). The interface between the dermal and epidermal components of the suspensory apparatus are the interdigitating dermal and epidermal laminae. The crucial part of this suspensory apparatus is the series of collagen fiber bundles that run from the surface of P3 to the basement membrane. It is the weakening of this tissue that is believed to be responsible for the displacement of P3 which predisposes the claw to disorders in cattle.

The "supporting tissues" within the claw capsule are made up of 3 parts: 1) connective tissue, a part of which encloses the digital cushions and extends into, and becomes part of, the interdigital ligaments which support the axial side of P3, 2) vascular tissue, and 3) adipose tissue which comprise the digital cushion. Collagen fiber bundles which comprise connective tissue in the supporting structure of the claw are believed to be affected similarly to those in the suspensory apparatus during bouts of laminitis.

Destruction of the dermal-epidermal junction has particular consequences in cattle as it permits weakening of the suspensory apparatus within the claw. As the suspensory apparatus weakens, P3 begins to "sink" or "rotate" within the claw. The result is compression of the corium and supporting tissues that lie between P3 and the sole. When this "P3 sinking phenomenon" involves severe rotation of the toe portion of P3 downward toward the sole, a toe ulcer may develop. If, on the other hand, sinking of the P3 is such that the rear portion sinks furthest, compression and thus a sole ulcer is more likely to develop in the area of the heel-sole junction (known as the "typical site" or the site most commonly associated with the development of sole ulcers). Sole ulcers are very common claw lesions in dairy cattle and constitute one of the most costly of lameness conditions (Mulling and Lischer, 2002; Lischer et al., 2002).

Alternative mechanisms responsible for damage and/or weakness of the suspensory apparatus

Researchers from the United Kingdom suggest there may be a combination of biochemical and biomechanical mechanisms responsible for weakening of the dermal-epidermal segment between the wall and P3 (Tarleton and Webster, 2002). Their work suggests that weakening of the suspensory tissue at the time of calving may be a result of the activation of matrix metalloproteinases

by a gelatinolytic protease they refer to as "hoofase". Levels of this enzyme were highest in the claws of heifers from 2 weeks pre-calving to 4 to 6 weeks post-calving. These researchers also propose another factor responsible for weakening of the suspensory apparatus that is not associated with the inflammatory changes normally observed with laminitis. Hormones, responsible for relaxation (such as relaxin) of the pelvic musculature, tendons, and ligaments around the time of calving, may have a similar effect on the suspensory tissue of P3 as well. Their data further suggest that although this weakening of the suspensory apparatus may be a natural occurrence, housing of animals on soft surfaces during the transition period may be sufficient to reduce or alleviate the potential for permanent damage to these tissues (Webster, 2002). Others suggest that sinking and rotation of P3 is associated with unexplained structural alterations occurring on the surface of P3 where the suspensory tissues are anchored (Mulling, 2002). Regardless of the actual mechanism, the result is a predisposition to claw disorders that often result in permanent damage to the suspensory and supporting tissues within the claw and Lischer, and a higher risk of lameness. These studies also support the view that laminitis is complex and multi-factorial.

Summary

Nutrition has significant influences on claw health in dairy cattle. Damage to the dermal-epidermal junction as occurs with laminitis interferes with the diffusion of nutrients across the basement membrane into the living layers of the epidermis. Furthermore, disruption of the basement membrane and germinal epithelium restricts normal differentiation and proliferation of keratinocytes destined to become claw horn. The end result is weaker, less resistant claw horn. Rumen acidosis predisposes to laminitis. It is most often associated with the ingestion of large amounts of highly fermentable carbohydrate-rich feeds in combination with fiber sources low in effective fiber. Some degree of acidosis seems unavoidable since what ends up in the cow's rumen is not totally determined by the ration formulation, mixing, or delivery to the feed bunk, but to some extent by the cow and what she elects to consume. The levels of protein in rations are often questioned relative to their potential for causing laminitis-like problems. To date, there is no convincing evidence that high levels of protein are responsible for laminitis. Vitamins and minerals have important roles in claw health as they support keratinocyte proliferation and differentiation. They are also necessary for proper keratinization within horn cells. There is strong evidence of a relationship between rumen acidosis and laminitis; however, this has not been documented by all studies reported in the literature. Recent development of a bovine research model may help to establish a clearer understanding of this relationship in the future. Current information suggests that laminitis is a disease affecting tissues at the cellular level. "Claw horn disruption" is the phase preferred by some who believe that this more accurately describes the lesion of laminitis. Reduced keratinization is a major complication of laminitis and results in the production of soft weak horn that is less resistant to physical or mechanical forces. Sinking and rotation of P3 is a secondary consequence of the damage caused by metalloproteinase enzymes released during the course of the disease. These enzymes are responsible for degradation of the collagen fiber bundles in the suspensory apparatus of P3 which creates laxity in this support system and permits sinking and rotation of P3. Recent work suggests that a novel enzyme termed "hoofase" may also play an important role in the activation of metalloproteinase enzymesl. Hoofase was found to increase significantly in animals at or near the time of calving. A second mechanism is believed to be associated with the hormonal changes that occur around the time of calving. It is proposed that the same hormones responsible for relaxation of the pelvic musculature (e.g., relaxin) near the time of calving have a similar effect on the suspensory apparatus of P3. These researchers have also found

that housing of animals on soft surfaces throughout the transition period permitted recovery of these tissues, thus preventing permanent damage.

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