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Equine laminitis: What have we learned so far?
Normal structure and function

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The tough hoof capsule protects sensitive structures within and allows the natural horse to gallop over dry, rocky terrain with apparent impunity. Equids are normally mobile and athletic but when they develop laminitis and become crippled we realise, belatedly, how dependent they were on an intact, functional, pain-free suspensory apparatus of the distal phalanx (SADP).

We have evaluated the effect of cooling the distal limb of horses by continuously applying very cold water to the lower limbs of horses (Pollitt and van Eps 2004; Van Eps and Pollitt 2009). Horses were stood in a circulating cold water bath for 72 h. Thus all 4 limbs were subjected to continuous cryotherapy and when removed from the water bath the horses were observed for 4 days. There was mild transient oedema of the distal limbs but no lameness or skin damage. Histological examination of hoof wall lamellae, taken 7 days after cryotherapy commenced, showed no lesions. No adverse clinical effects were noted in the cooled limbs up to one year following cessation of the cryotherapy.

We have obtained information on the physiological control of the foot circulation by measuring hoof temperature in cold environments. Six mature horses, with clinically normal feet and acclimatised to winter in Norway were kept in outside yards and fed ad libitum hay. Forelimb hoof temperature and ambient temperature were logged continuously and successful continuous recordings were made over periods ranging from 2–3 days. Ambient temperatures ranged from 0°C to -12°C. Hoof temperatures fluctuated continuously over a remarkably wide range. Sometimes they were close to zero and at other times they were 30°C.

Horses appear able to exert a fine degree of control over the temperature of their feet probably by a central mechanism. This confirms a conclusion from our distal limb cryotherapy studies that severe cold is not noxious to the horse. Frequent warming of the foot no matter what the ambient temperature appears to be a feature of normal foot physiology perhaps to ensure that metabolism and growth are never compromised. Practitioners palpating or thermal imaging either warm or cold horse’s feet should not be surprised; both are manifestations of normal foot physiologic control.

Despite much speculation regarding insulin resistance, glucose metabolism and their links to laminitis there is little information regarding, if, where and how glucose is consumed by the living foot. Equids are normally mobile and athletic but when they develop laminitis and become crippled we realise, belatedly, how dependent they were on an intact, functional, pain-free suspensory apparatus of the distal phalanx (SADP).

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Despite much speculation regarding insulin resistance, glucose metabolism and their links to laminitis there is little information regarding, if, where and how glucose is consumed by the living cells of the equine hoof. To address this we investigated if glucose is consumed by the equine hoof and if glucose uptake into hoof epidermal cells is insulin dependent (Wattie and Pollitt 2004). Blood glucose concentrations were recorded in 7 adult horses by simultaneously taking samples from 3 blood vessels; an artery, the jugular vein, and a digital vein at the level of the pastern of one of the front legs. Antibody towards glucose transport proteins (GLUTs) and the insulin receptor was used for immunolocalisation of these proteins in the gluteus muscle and in the feet of 7 horses. We found that the foot of a horse consumes more glucose than its head. GLUT1 was the major epidermal cell glucose transporter of the hoof. In contrast to the gluteus muscle, hoof lamellae do not rely on insulin for glucose uptake. Insulin receptors were absent from cells of the epidermal lamellae and only present in vascular tissue.

Microdialysis (MD) was used to monitor the lamellar extracellular fluid (ECF) of standing horses. Gentamicin was chosen as an indicator drug and after intravenous injection, reached the lamellar region in quantifiable concentrations thus showing a circulatory relationship between blood and the lamellar ECF (Nourian et al. 2009a). Thus, MD was validated as a method for monitoring the concentration of intravenously injected drugs at the lamellar interface. Next MD was used to measure the lamellar concentration of intraosseously (i.o.) injected gentamycin. Utilising the MD sampling method already developed the indicator compound, gentamycin was slowly perfused (20 µl/min or 1.2 m/h) through a custom designed, hollow, bone screw implanted in the dorsal cortex of the distal phalanx. The i.o. bone screw was remarkably well tolerated, after the local anaesthetic, regional nerve blockade, that was used to implant it, had worn off. Gentamycin was present in lamellar tissue at much higher concentrations in lamellar ECF than in blood (Nourian et al. 2009b). Thus, an efficacious, i.o. drug delivery system to the lamellar region in the standing, conscious horse was validated. Numerous fine foramina, in the dorsal (but not the palmar) cortex of the bone connected the i.o. and lamellar compartments and appeared to participate in the distal phalanx-lamellar circulation. Both arteries and veins passed through the foramina suggesting roles in pressure modulation and thermoregulation of the distal foot. The discovery of a distal phalangeal-lamellar vascular relationship suggested that the lamellar circulation could be accessed via the i.o. route.

References


Equine laminitis: What have we learned so far? Laminitis histopathology and ultrastructure

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The sequence of microscopic events that lead to clinical laminitis follow a consistent temporal pattern (Croser and Pollitt 2006), and the stages of histological laminitis can be identified by the degree of severity of these changes. Making the lamellar basement membrane clearly visible is important and requires staining lamellar tissues with periodic acid-Schiff (PAS) stain or with immunohistochemical methods using basement membrane-specific antibodies (Pollitt 1996; Pollitt and Daradka 1998).

During the developmental phase, loss of shape and normal arrangement of the lamellar basal and parabasal cells occurs. The basal cell nuclei become rounded instead of oval and take an abnormal position in the cytoplasm of the cell. The secondary epidermal lamellae become stretched, long, and thin, with tapering instead of club-shaped tips. These changes were present at 12 h in serial lamellar biopsies taken after oligofructose dosing (Croser and Pollitt 2006).

While the former takes place, the basement membrane of the secondary epidermal lamellae loses its attachment to the basal cells. This is first noticeable at the tips of the secondary epidermal lamellae where teat-shaped bubbles of loose basement membrane form. PAS staining shows this best.

Examination of laminitis tissues with the electron microscope confirms lysis and separation of the lamellar basement membrane 24 h after oligofructose dosing (Nourian et al. 2007). Importantly, the greater magnification shows widespread loss of basal cell attachment plaques (hemidesmosomes) and contraction of the basal cell cytoskeleton away from the inner cell surface. Electron microscopy shows why the basement membrane separates from the feet of the basal cells. The filaments that anchor hemidesmosomes to the lamina densa of the basement membrane no longer bridge the dermal/epidermal interface (French and Pollitt 2004a,b; Nourian et al. 2009).

Because the basement membrane is no longer completely tethered to the basal cells, it slips farther away with each cycle of weight bearing by the horse. Portions of the lamellar basement membrane are lysed initially between the bases of the secondary epidermal lamellae. The basement membrane retracts from the tips of secondary epidermal lamellae and taking with it the dermal connective tissue. The basement membrane-free epidermal cells appear not to be undergoing necrosis, at least initially, and clump together to form amorphous, basement membrane-free masses on either side of the lamellar axis (Pollitt 1996).

In laminitis the worst-case scenario is a rapid and near-total basement membrane separation from all the epidermal lamellae of the hoof toe, quarters, heels and bars. Sheets of basement membrane peel away to form aggregations of loose, isolated basement membrane in the connective tissue adjoining the lamellae. The epidermal lamellar cells are left as isolated columns with little viable connection to the dermal connective tissue still attached to the distal phalanx. The hoof lamellar tips slide away from the basement membrane connective tissue attachments, at first microscopically, but as the degree of separation increases, the distance between hoof and distal phalanx becomes measurable in millimetres and can be detected radiographically (van Eps and Pollitt 2009). This manifests clinically as the sinker. Because the basement membrane is the key structure bridging the epidermis of the hoof to the connective tissue of the distal phalanx, wholesale loss and disorganisation of the lamellar basement membrane follows and inexorably leads to the pathology of hoof and bone that characterises the chronic stage of laminitis.

The laminitis process also affects the lamellar capillaries. As the basement membrane and the connective tissue between the secondary epidermal lamellae disappear, so do the capillaries. They become obliterated, compressed against the edges of the primary dermal lamellae. Without a full complement of capillaries in the lamellar circulation, blood probably bypasses the capillary bed through dilated arteriovenous shunts (Pollitt and Molyneux 1990) changing the nature of the foot circulation. A bounding pulse becomes detectable by finger palpation of the digital arteries. Furthermore, epidermal cell necrosis, intravascular coagulation, and oedema are not universally present in sections made from tissues in the early stages of laminitis. The vessels in the primary dermal lamella, even the smallest, are predominantly open, without evidence of microvascular thrombi. The gross anatomical appearance of freshly dissected laminitis tissue is dryness. Sometimes the lamellae just peel apart.

**References**


Thursday 10th September 2009

15.00-15.20

Epidemiology and clinical perspective of endocrinopathic laminitis

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Endocrinopathic laminitis

Endocrinopathic laminitis has been defined as laminitis developing from hormonal influences rather than in association with pro-inflammatory and intestinal conditions (Johnson et al. 2004). Conditions associated with endocrinopathic laminitis include equine metabolic syndrome (EMS), equine Cushing’s syndrome (ECS) also called pituitary pars intermedia dysfunction (PPID) and iatrogenic corticosteroid administration. Common to all these conditions are disturbed glucose and insulin regulation and, most importantly, the development of insulin resistance and hyperinsulinaemia.

Laminitis associated with EMS has been linked to insulin resistance and hyperinsulinaemia both in field (Treiber et al. 2006) and experimental studies (Asplin et al. 2007).

Laminitis associated with ECS has also been attributed to the effects of hyperadrenocorticism and the development of insulin resistance. Between 50 and 80% of horses with ECS will have clinical laminitis. Supporting the importance of the development of insulin resistance in the pathogenesis of laminitis is the importance of insulin as a prognostic indicator for survival in horses with ECS. Horses with abnormally high levels of insulin (>188 μIU/ml) were much more likely to develop laminitis and survive less than 2 years after diagnosis, than those with only moderate elevations or normal insulin levels (<62 μIU/ml) (McGowan et al. 2004).

Laminitis associated with iatrogenic corticosteroid administration is rare in normal horses. The common link between both ECS and iatrogenic corticosteroid administration is the ability of cortisol to induce insulin resistance. Insulin and cortisol have opposing actions on glucose metabolism such that hyperinsulinaemia and insulin resistance are potential consequences of corticosteroid treatment. A single dose of triamcinolone at 0.05 mg/kg bw i.m. induced marked hyperglycaemia for a period of 3 days and a higher dose of 0.2 mg/kg bw i.m. induced marked hyperglycaemia and hyperinsulinaemia for over 6 days (French et al. 2000). Three weeks of dexamethasone treatment at 0.08 mg/kg bw every 48 h resulted in a 10-fold increase in basal insulin and insulin resistance demonstrated by the euglycaemic-hyperinsulinaemic clamp technique (Tiley et al. 2008).

Epidemiology

Endocrinopathic laminitis is the most prevalent form of laminitis. In a large survey of horse operations across the USA, the causes of laminitis as reported by the property manager were recorded. It was found that approximately 50% of all laminitis cases were associated with grazing lush pasture, while a further 27% were associated with ‘other known’ problems including feed problems, pregnancy and obesity (Anon 2000). The cause was unknown in 15%. In epidemiological research from Australia >80% horses with a history of laminitis had evidence of an endocrinopathy based on elevated ACTH, alpha MSH or insulin. Similarly, in hospital based research from Finland, a diagnosis of endocrinopathic laminitis accounted for >80% laminitis cases seen in an 18 month period with risk factors including evidence of laminic rings and phenotypic indicators of EMS.

Both ECS and EMS are prevalent conditions. The prevalence of ECS in horses aged 15 years and older is 15% (McGowan et al. 2007) and age is a significant risk factor with the risk of ECS increasing by 20% for every year of age over the age of 15. The prevalence of insulin resistance in the general population probably depends on the breed sampled. Research from the USA showed a prevalence of hyperinsulinaemia in a random sample of 300 horses of 10%, with a relationship with age and body condition score (Geor et al. 2007). Research from Australia in ponies has shown the prevalence of hyperinsulinaemia to be as high as 28% in pony breeds, including the Welsh Mountain pony and cob, Shetland, Connemara ponies. Risk factors for the condition included increasing age, management factors and a history of laminitis (McGowan and McGowan 2008).

Based on the high prevalence of both ECD and EMS it is very likely that in an aged population that there will be concurrent diagnoses in the same horses. This is especially likely in the UK due to the popularity of EMS predisposed ponies and cobs. This does not necessarily imply a causal relationship and in a study from Australia where the Thoroughbred and cross was the most common breed of horse kept, the prevalence of concurrent diagnosis was very low.

References

Research in endocrinopathic laminitis: New model and its implications

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Introduction

Recently, significant advances have been made by scientists researching endocrinopathic laminitis. In 2007, Asplin and associates described a new model for inducing laminitis experimentally in clinically normal ponies using a prolonged-euglycaemic hyperinsulinaemic clamp technique (Asplin et al. 2007).

Insulin and laminitis

The implications of this new model for researching insulin-induced laminitis are substantial. This research has been vital in linking laminitis directly with hyperinsulinaemia and associated endocrinopathic conditions such as equine metabolic syndrome and equine Cushing’s disease. Furthermore, previous studies on clinical cases of endocrinopathic laminitis have been limited to being retrospective or linear in nature, as the disease cannot be detected clinically until it has reached the acute phase. With a reliable, well tolerated method for inducing laminitis secondary to hyperinsulinaemia, the prodromal stages of the disease process can now be studied in detail.

Advancing the research

To progress from the initial research using ponies, it was necessary to determine if hyperinsulinaemia has a laminogenetic effect in horses, as horses are intrinsically more insulin sensitive than ponies (Jeffcott et al. 1986). In 2008, laminitis was induced in healthy Standardbred racehorses within 48 h of the onset of hyperinsulinaemia (de Laat et al., unpublished data). The model consisted of a controlled, paired experiment during which insulin was infused at a fixed-rate to a treatment group to promote very high serum insulin concentrations, while euglycaemia was maintained (blood glucose: 5 ± 1 mmol/l) with a variable rate infusion of 50% dextrose solution. A control group received the equivalent volume of a balanced electrolyte solution. Monitoring of the subjects during the experiment included heart and respiratory rate, body temperature, ambient temperature, hoof wall surface temperature (HWST), regular blood sampling and urinalysis, pre- and post radiography and lameness assessments. The experiment was ceased at the onset of Obel grade 2 laminitis (Obel 1948) in treated animals, and lamellar tissue was harvested for analysis. Tissue was processed for electron and light microscopy, molecular studies and immunohistochemistry.

Current findings

Currently, data have been gathered on histopathological and ultra-structural changes, vascular pathologies, degradative enzyme activity and inflammatory and oxidative markers of disease in lamellar tissues at end-time points (48 h and 72 h) of disease. Significant findings to date include persistently elevated HWST and increased digital pulses in treated horses compared to insignificant digital pulses and variable HWST in the control horses. Consistent with the vasodilatory actions of insulin, the development of endocrinopathic laminitis appears to be associated with increased blood flow to the hoof. Light microscopical examination of lamellar samples from affected horses shows significant histopathological changes including marked lamellar lengthening and attenuation, basement membrane separation at the tips of the secondary epidermal lamellae (SEL) and rounding of the basal cell nuclei. Radiographic evidence of laminitis as a result of the study has not been recorded; however, further investigation is warranted. There appears to be a relationship between bodyweight and the severity of laminitis, heavier horses progressed to Obel grade 2 laminitis more rapidly than lighter horses and ponies.

Future directions

Further application of the model will involve the analysis of lamellar tissue samples from earlier time points in the disease process as has been done in other research models of laminitis (Belknap et al. 2007). This will allow detailed analysis of the pathophysiological processes occurring in the early, mid and late stages of disease, and will provide the best opportunity for elucidation of the pathogenic pathways involved in insulin-induced laminitis. Future refinement of this research model will focus on more accurate quantification of the degree of hyperinsulinaemia required for the induction of laminitis, and the potential contribution of an individual animal’s genetic make-up to its susceptibility to disease. Using this research model to study endocrinopathic laminitis will facilitate rapid advances in our understanding of the disease resulting in improved prevention and therapeutic strategies.

References

Equine laminitis: What have we learned so far?

Pathophysiology

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Pathological deformation of the hoof lamellar distal phalanx suspensory begins during the developmental phase of laminitis. Tightly controlled, metabolic processes are targeted, causing lamellar specific pathology. We are investigating the timeline of the basement membrane zone (BMZ) degradation sequence using serial biopsies. Accumulating evidence suggests that during developmental laminitis, compromise of the BMZ is associated with production and increased activation of constituent lamellar enzymes. The enzymes involved are metalloproteinase-2 (MMP-2), metalloproteinase-9 (MMP-9), membrane-type metalloproteinase (MMP-14) and aggreganase (ADAMTS-4) and singly or together they destroy key components of the lamellar suspensory apparatus (Loftus et al. 2006; 2008; Budak et al. 2009; Coyne et al. 2009). A second, newly discovered, lamellar deformation mechanism is seen in hyperinsulinaemic horses. In laminitis induced by dosing horses with extract of black walnut (Juglans nigra) heart wood there was little evidence of basement membrane breakdown (Belknap 2007; Faleiros et al. 2009).

It is rare to detect leucocytes in the lamellar tissues of normal horses (Faleiros et al. 2009). However, extravasation of leucocytes into the perivascular lamellar dermis occurs in carbohydrate (Pollitt 1996), Black Walnut extract (Black et al. 2006; Faleiros et al. 2009) and the hyperinsulinaemic (Nourian et al. 2009) induced forms of laminitis. Since leucocytic infiltration of tissues is common to most, if not all, forms of laminitis, this has re-emphasised an inflammatory pathway to laminitis development. Indeed, there is molecular evidence that inflammatory mediators may activate many of the processes known to damage the lamellar interface (Belknap et al. 2007; Loftus et al. 2007). Polymorphonuclear leucocytes are rich in (MMP-9) and their presence within lamellar epidermal compartments in grade 3 histopathology (Pollitt 1996) suggests that this basement membrane degrading enzyme may have a pathological role in disease development. Also neutrophils produce reactive oxygen species and proinflammatory cytokines that probably contribute to cellular damage within the lamellar milieu (Belknap 2007). However, carbohydrate-induced laminitis biopsies at the 12 h post dosing time point show basement membrane degradation occurring in advance of leucocytic infiltration thus downplaying an initiating role for leucocytes in lesion development (Visser 2009).

Lamellar BMZ disintegration occurs well before clinical signs. The molecular conformation of the lamellar basement membrane is altered 12 h after dosing with oligofructose and a major constituent of the basement membrane, collagen IV, begins to disappear. Previously, damage to the lamellar basement membrane was attributed to MMP release and activation but new evidence places MMP activation many hours later than other molecular events (Visser 2009). An enzyme capable of modifying the proteoglycan components of lamellar basement membrane is ADAMTS-4 (a disintegrin and metalloproteinase with thrombospondin motifs) the gene for which has the greatest fold increase of any so far discovered in laminitis development (Budak et al. 2009; Coyne et al. 2009; Visser 2009). ADAMTS-4 gene expression occurs early in laminitis development and its gene product may play a central role in the pathophysiology of the disease.

During carbohydrate overload rapidly proliferating species of hindgut streptococci, predominantly S. bovis (now S. lutetiensis), ferment carbohydrate and produce large quantities of lactic acid (Milinovich et al. 2006, 2007, 2008). In the presence of virtually unlimited substrate, the population of S. bovis increases exponentially and then die and lyse en masse. The liberated cellular components of lysed hindgut streptococci may cross the synovial barrier of the damaged hindgut and reach the hoof lamellae haematogenously to initiate laminitis.

References


Equine laminitis: What have we learned so far?
Chronic laminitis

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The disintegration of the inner hoof wall accompanying acute laminitis is initially invisible to the naked eye. With the passage of time, however, the hoof begins to display the effects wrought upon it by the pathology of the acute phase. If lameness persists and worsens there is likely to be on-going displacement of the distal phalanx within the hoof capsule and lysis of its cortex. To care for foundered horses effectively the chronic phase needs to be better understood.

Ultimately it is the strength of the lamellar interface that determines prognosis. Many horses relapse after the initial laminitis episode despite early signs of improvement. We studied laminitis affected tissue, 7 days after the initiating episode, to assess the healing response to the disease (van Eps and Pollitt 2009a). Surprisingly, 7 days after laminitis, the destructive processes that caused lamellar basement membrane (BM) dysadhesion and lysis, basal cell dislocation and lamellar attenuation had abated. All epidermal compartments were enveloped in normal appearing BM and the majority of epidermal basal cells (EBCs) were of normal shape and orientation. However, lamellar anatomy was in disarray. The rows of organised, symmetrical hoof wall lamellae that characterise normal lamellae had been replaced with epidermal strands and islands, many no longer connected to their respective primary hoof wall lamellae. The stretched elongated lamellae allow the distal phalanx to sink into the hoof capsule. Initially this results in a small but significant increase in distance between the outer hoof wall and the dorsal distal phalanx. This may be measurable on good quality radiographs (van Eps and Pollitt 2009b) and venograms (Baldwin and Pollitt 2008). Early radiographs are a yardstick against which to measure subsequent exacerbation. The formation of a lamellar wedge is often described as a hallmark of chronic laminitis (Roberts et al. 1980) but there was no sign that one was forming at the 7 day stage of the disease. Presumably the wedge forms as a consequence of unstable lamellar attachments. Laminitis, observed 7 days after induction, had reduced the surface area of the lamellar attachment apparatus and weakened it. Premature resumption of athletic exercise and thus greater foot break-over strain, particularly in the fore feet, could rupture surviving lamellar attachments. This is the likely mechanism behind recrudescent laminitis in apparently recovered horses.

Horses with severe laminitis develop progressively greater venographic changes (especially filling deficits). Serial venography gives important information into otherwise nonvisible vascular pathology. Venographic changes occur in virtually all cases of clinical laminitis often when subtle plain radiographic changes are inconclusive. Insignificant initially, filling deficits become more complete as the weeks go by. At 7 weeks there are usually prominent deficits in the coronary band and toe venous circulations. These progressively worsen until, in some cases, the entire dorsal wall appears to have no venous circulation. Histological studies show the reason for the venous filling deficits. The veins in the region of the deficits are compressed. The neighbouring arteries appear relatively unaffected. Why are the veins flattened and nonfunctional at the coronary? When the lamellar attachments fail the distal phalanx descends and drags with it the growth zone of the proximal hoof wall. The growth zone continues proliferation of new hoof wall but now in a kinked pattern. In severe cases the growth zone sinks below the prelaminitis hoof wall and in this trapped situation produces hoof wall that grows inwards. The ingrowing coronet of chronic laminitis feet compresses the coronary cushion producing the venographic filling deficits.

Coronary profiles of chronic laminitis feet are abnormal. Normal coronary profiles are broad and triangular with hoof tubules that are straight and parallel. Feet affected by chronic laminitis however have flattened compressed coronary profiles, kinked hoof wall tubules and growth zones that are below the level of the prelaminitis wall.

Similar events occur at the toe. When the distal tip of the distal phalanx descends into the hoof capsule it not only crushes the sole corium but drags downwards the growth zone of the dorsal sole and white line. As with the coronet this growth zone continues to proliferate but instead of the normal downwards direction it grows inwards towards the tip of the distal phalanx. Here the pathological changes are more obvious. Not only are veins compressed the pressure is sufficient to lyse the tip of the distal phalanx.

The zones of hard, inward growing toe and terminal wall tubules approach the front half of the foot and may contribute to inexorable rotation of the distal phalanx; the lamellar wedge slowly pushes the tip of the bone backwards and downwards. This is contrary to the widely held belief that it is the pull of the deep flexor tendon that causes distal phalanx rotation. Soft tissue and vascular compression and bone lysis is likely painful and contributes to the lameness of chronically foundered horses. If left unchecked the inward growing hoof of the proximal and distal hoof wall progressively destroys foot architecture eventually leading to incurable pathology. Early resection of the hoof wall in a zone corresponding to the in growing coronet and toe appears to release compression of the tissues beneath and, depending on the extent of the soft tissue and bone pathology, restore a semblance of normal hoof growth.

References


