Glucose uptake in the equine hoof
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Background - Laminitis is a crippling and potentially fatal condition of the equine hoof, associated with many causes, including carbohydrate overload, overfeeding, dietary oligofructans, metabolic syndrome, and Cushing’s Syndrome.1 The mechanism of laminitis is unknown, but could be associated with impaired glucose uptake into the hoof.

Objectives - To characterise the physiological regulation of glucose transport in normal equine lamellae.

Design - Equine lamellar explants were incubated for up to 48 h in a cell culture medium. Glucose uptake was measured using 2-deoxy-D-[2,6-3H] glucose. Separation force was determined by tension testing. β1-Adrenoceptors were measured by radioligand binding using [3H]-CGP-12177.

Outcomes - Lamellar explants incubated in the presence of glucose remained intact at forces of up to 916 g, whereas without glucose, explants became separated from the hoof wall at 416 ± 36 g. Insulin (300µU/mL) had no effect on glucose uptake, but Isoprenaline (40 nM) reduced glucose uptake to 60% of basal levels after 24 h and 48 h. Explants contained 90 ± 2.6% β2-adrenoceptors and 10 ± 2.6% β1-adrenoceptors.

Conclusion - Healthy lamellar tissue is dependent on glucose for structural integrity, Glucose uptake is impaired by catecholamines, which are associated with various stress conditions that can cause laminitis. Therefore, the data presented are consistent with the hypothesis that glucose uptake plays a role in certain types of laminitis.

References

Do commercial tryptophan pastes elevate plasma tryptophan concentrations in horses?
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Background - Tryptophan (Trp) is an essential amino acid that is a precursor of serotonin, a neurotransmitter implicated in sedation, inhibition of aggression, fear and stress in various animal species and humans. Preparations containing Trp are marketed worldwide as calmative agents to treat excitable horses, but there is no evidence of their efficacy. Coupled with this, it has been shown that high oral doses can be toxic. No work has been done to establish that commercial pastes actually increase Trp concentrations in the blood of horses, or how long this elevation lasts.

Objectives - To determine if a commercial Trp oral paste increases plasma Trp concentrations and to establish whether the pharmacokinetics of Trp are altered by different feeding regimens.

Design - Eleven mature geldings (BW 507 ± 12.9 kg) were fed a meal of 2.7 kg lucerne hay at 0900 h each day, supplemented in the afternoon with sufficient hay to meet maintenance energy requirements. At 0900 h the horses were also given either 3 doses of a commercial Trp oral paste (total 6.3 g L-Trp) or an equivalent volume of water in a cross-over design. Blood samples were collected every 30 min for 6 h and analysed for total Trp using gas chromatography. The study was repeated after 3 weeks using the same horses fed a morning meal of 1.5 kg oats.

Outcomes - Baseline Trp concentrations measured 2 h after feeding were 71.8 ± 3.7 µM and 36.7 ± 3.4 µM for horses fed lucerne hay or oats, respectively (P < 0.001). Peak Trp concentration in response to Trp paste was also higher in horses fed hay (167.6 ± 8.0 µM at 1130 h) versus oats (149.4 ± 13.7 µM at 1030 h; P <0.001), as was the area under a plot of Typ concentration versus time (227.8 ± 8.7 µMh versus 179.1 ± 14.9 µMh; P<0.01). Tryptophan concentrations returned to baseline after 6 hours regardless of the ration fed.

Conclusion - No adverse signs were seen in this study although the effect of such doses fed over a more prolonged period is unknown. Tryptophan paste fed at this level does increase plasma Trp concentrations in horses. The amount and duration of this elevation is dependent on the type of diet fed. Further work is needed to determine if the Trp paste fed at lower doses has an effect and whether in fact an increase in Trp concentrations has any effect on equine behaviour.