

## Laminitis: New Study on Sugar and Starch as a Cause

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A breakthrough in laminitis research by a team of scientists at University of Queensland, Australia, and colleagues, was published in August 2007 *The Veterinary Journal*. The study explains an important link in sugar and starches as causes for laminitis.

Laminitis was induced in healthy, lean, young ponies by prolonged (up to 72 hours) administration of insulin via euglycemic clamp. This procedure maintains blood glucose at a set level, eliminating involvement of glucose toxicity. Prior to the study, all ponies' insulin levels were in normal range. In addition to clinical observation of lameness, examination of hoof tissues after euthanasia confirmed laminitis in all four feet. There was no evidence of gastrointestinal involvement.



**This is the author's 11-year-old mixed breed pony mare taken the day of her last insulin test, after grazing for only two hours. Her insulin tested 3.5 times normal. One month earlier on a diet of only low sugar hay, her insulin was within normal levels. She has had laminitis several times in the past, although she was sound throughout this period. Note she is not overweight.**

While insulin resistance has long been associated with laminitis, researchers have only speculated as to the causative agent. Some have suggested that inflammatory substances released as a consequence of obesity were to blame. This new research points the finger at the toxic aspects of insulin, although the mechanism by which it causes laminitis is still not

understood. The subject ponies were not obese. While obesity is sometimes (but not always) a consequence of insulin resistance, this study shows it is not necessary to trigger laminitis.

This study puts new importance on the role of sugar and starch as the cause of laminitis. These are the carbohydrates that induce a glycemic response that includes release of insulin. Much of the data being used to justify the fructan theory of laminitis are actually water soluble carbohydrates, which include sugar and fructan in cool season grasses.

This study now explains why horses get laminitis from eating warm season grasses such as Bermuda, or legumes such as clover, that don't have fructan. Sugar concentration increases in both warm and cool season grasses under stress, such as cold, drought, or low nutrient supply. This stress triggers the formation of storage carbohydrates to be used later after conditions for better growth resume. In cool season grasses, the storage form of carbohydrate is fructan, and in warm season grasses it is starch. When fructan concentration in grass is high, sugars are nearly always fairly high as well.

Previous focus on maintaining stable populations of fermentative micro-organisms in the hind gut should be re-evaluated in light of this new research.

Owners concerned with laminitis prevention should look toward minimizing sugar and starch levels in horses' diets and maintaining a regular program of exercise. To identify an animal at high risk for laminitis, ask your veterinarian to pull a blood sample for insulin levels and blood glucose. High insulin or a very low glucose:insulin ratio should trigger a pro-active program of laminitis prevention.

As with humans, diet and exercise are the only way that insulin resistance, which causes high levels of circulating insulin, can be managed.

The study was titled: "Induction of Laminitis by Prolonged Hyperinsulinaemia in Clinically Normal Ponies." Authors were Katie E. Asplina, Christopher C. Pollitt, and Catherine M. McGowana of the School of Veterinary Science, The University of Queensland, Australia, and Martin N. Sillenceb, School of Agricultural and Veterinary Sciences, Charles Sturt University, Wagga Wagga, Australia.

### **Abstract**

"The purpose of this study was to determine the effects of prolonged administration of insulin, whilst maintaining normal glucose concentrations, on hoof lamellar integrity in vivo on healthy ponies with no known history of laminitis or insulin resistance. Nine clinically healthy, unrelated ponies were randomly allocated to either a treatment group (n = 5; 5.9 ± 1.7 years) or control group (n = 4; 7.0 ± 2.8 years). The treatment group received insulin via a euglycaemic hyperinsulinaemic clamp technique modified and prolonged for up to 72 hours. Control ponies were infused with an equivalent volume of 0.9% saline. Ponies were euthanased at the Obel Grade 2 stage of clinical laminitis, and hoof lamellar tissues were harvested and examined for histopathological evidence of laminitis.

"Basal serum insulin and blood glucose concentrations were 15.7 ± 1.8 µU/mL and 5.2 ± 0.1 mmol/L, respectively (mean ± SE) and were not significantly different between groups. Mean

serum insulin concentration in treatment ponies was  $1036 \pm 55$   $\mu$ U/mL vs.  $14.6$   $\mu$ U/mL in controls. All ponies in the treatment group developed clinical and histological laminitis (Obel Grade 2) in all four feet within 72 h ( $55.4 \pm 5.5$  h), whereas none of the control ponies developed laminitis. There was no clinical evidence of gastrointestinal involvement, and the ponies showed no signs of systemic illness throughout the experiment.

"The data show that laminitis can be induced in healthy young ponies, with no prior history of laminitis, by maintaining prolonged hyperinsulinaemia with euglycaemia. This suggests a role for insulin in the pathogenesis of laminitis, independent of hyperglycaemia, or alterations in hind-gut fermentation. For the clinician, early detection and control of hyperinsulinaemia may facilitate management of endocrinopathic laminitis."

