

Relationship between adiponectin and insulin sensitivity



Plasma concentrations of adiponectin and insulin sensitivity change during oligofructose induced equine laminitis development.

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Summary

REASON FOR PERFORMING STUDY: The roles and the relationship between adiponectin and insulin sensitivity during oligofructose induced equine laminitis development are incompletely understood.

OBJECTIVES: To determine the temporal roles and relationship between adiponectin and insulin sensitivity during oligofructose induced laminitis development.

METHODS: Horses (n= 18) were divided into 2 groups. A oligofructose (OF; n= 9) group that received 10g/kg bwt oligofructose dissolved in 4L water via nasogastric intubation, a normal saline (NS; n= 9) group that received 4L normal saline. Blood collected at 4 h intervals over a 72 h study period. Blood samples were analysed by ELISA kit and Portable Blood Glucose Meter. The fasting insulin resistance index (FIRI) = fasting glucose × fasting insulin/25.

RESULTS: Horses receiving oligofructose decreased the level of adiponectin and increased the FIRI significantly (P < 0.05), There was a significant negative correlation between the level of adiponectin and the FIRI over time, but responses did not differ significantly in group.

CONCLUSIONS: Adiponectin play an important role in oligofructose induced equine laminitis development by affect insulin sensitivity.

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POTENTIAL RELEVANCE: Adiponectin have a role in laminitis pathology and control of its level may prevent or treatment laminitis.

1. Introduction

Laminitis, according to Wikipedia, is ‘ a disease that affects the feet of hooved animals (ungulates) and it is best known in horses. Clinical signs include foot tenderness progressing to inability to walk, inflammation, and increased temperature in the hooves. Laminitis is characterized by pain of the digital laminae of the hoof, and severe cases with outwardly visible clinical signs are known by the colloquial term founder. Insulin is a major regulatory hormone in glucose and fat metabolism, vascular function, inflammation, tissue remodeling, and the somatotropic axis of growth. Insulin resistance alters insulin signaling by decreasing insulin action in certain resistant pathways while increasing insulin signaling in other unaffected pathways via compensatory hyperinsulinemia ^[2] . Insulin resistance has been identified as a risk factor for equine laminitis in some reports ^[1, 2] , it is thought that equids experience alimentary carbohydrate overload when grazing on pasture after large amounts of grass are consumed during periods of rapid plant growth ^[3, 4] . Laminitis can also develop after horses accidentally consume too much grain and then develop alimentary carbohydrate overload ^[1] . However, a recent study using the oligofructose model showed that experimentally induced laminitis was not associated with a loss of insulin sensitivity ^[5] .

The adiponectin high molecular weight isoform (HMW-adp) and its relation with the other adiponectin isoforms (adiponectin index, SA) is an adipose

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tissue-secreted endogenous insulin sensitizer, which plays a key role as a mediator of peroxisome proliferator-activated receptor γ action. Adiponectin alters glucose metabolism and insulin sensitivity [6, 7]. In skeletal muscle, adiponectin increased expression of molecules involved in fatty-acid transport such as CD36, in combustion of fatty-acid such as acylcoenzyme A oxidase, and in energy dissipation such as uncoupling protein 2. These changes led to decreased tissue TG content in skeletal muscle [8]. Increased tissue TG content has been reported to interfere with insulin-stimulated phosphatidylinositol (PI) 3-kinase activation and subsequent glucose transporter 4 translocation and glucose uptake, leading to insulin resistance [9].

Laminitis related to equine sepsis has enjoyed the most attention of any type of laminitis from researchers over the past few decades, because the most commonly used models to study laminitis are models of equine sepsis. In the carbohydrate-overload models, including the traditional corn starch/wood flour and the more recent oligofructose models [10, 11]. The investigators reported that oligofructose reliably induced laminitis and a link is established between laminitis of oligofructose models and pasture fructan content [11]. Primarily, the oligofructose-induction model the best available means of experimentally inducing laminitis to mimic that resulting from exposure to excess carbohydrates, because the reliability of this model, combined with the reduced incidence of colic and low mortality rate compared with the starch-induction model [12].

It was hypothesised that plasma concentrations of adiponectin and insulin sensitivity would significantly change during oligofructose induced equine laminitis development. Specific aims of this study were to: 1) use oligofructose induced equine laminitis; 2) determine whether adiponectin play an important role in oligofructose induced equine laminitis development by affect insulin sensitivity; and 3) investigate whether insulin resistance was present in oligofructose induced equine laminitis.

References

1. Toth, F., Frank, N., Chameroy, K. A., & Boston, R. C. (2009). Effects of endotoxaemia and carbohydrate overload on glucose and insulin dynamics and the development of laminitis in horses. *Equine veterinary journal* , 41 (9), 852-858.
2. Treiber, K. H., Kronfeld, D. S., & Geor, R. J. (2006). Insulin resistance in equids: possible role in laminitis. *The Journal of nutrition* , 136 (7), 2094S-2098S.
3. Elliott, J., & Bailey, S. R. (2006). Gastrointestinal derived factors are potential triggers for the development of acute equine laminitis. *The Journal of nutrition* , 136 (7), 2103S-2107S.
4. Longland, A. C., & Byrd, B. M. (2006). Pasture nonstructural carbohydrates and equine laminitis. *The Journal of nutrition* , 136 (7), 2099S-2102S.
5. Kalck, K. A., Frank, N., Elliott, S. B., & Boston, R. C. (2009). Effects of low-dose oligofructose treatment administered via nasogastric intubation on induction of laminitis and associated alterations in

- glucose and insulin dynamics in horses. *American journal of veterinary research* , 70 (5), 624-632.
6. Ziemke, F., & Mantzoros, C. S. (2010). Adiponectin in insulin resistance: lessons from translational research. *The American journal of clinical nutrition* , 91 (1), 258S-261S.
 7. Pita, J., Panadero, A., Soriano-Guillén, L., Rodríguez, E., & Rovira, A. (2012). The insulin sensitizing effects of PPAR- γ agonist are associated to changes in adiponectin index and adiponectin receptors in Zucker fatty rats. *Regulatory peptides* , 174 (1), 18-25.
 8. Yamauchi, T., Kamon, J., Waki, H., Terauchi, Y., Kubota, N., Hara, K., & Kadowaki, T. (2001). The fat-derived hormone adiponectin reverses insulin resistance associated with both lipodystrophy and obesity. *Nature medicine* , 7 (8), 941-946.
 9. Shulman, G. I. (2000). Cellular mechanisms of insulin resistance. *Journal of Clinical Investigation* , 106 (2), 171-176.
 10. Garner, H. E., Coffman, J. R., Hahn, A. W., Hutcheson, D. P., & Tumbleson, M. E. (1975). Equine laminitis of alimentary origin: an experimental model. *American Journal of Veterinary Research* , 36 (4 Pt. 1), 441.
 11. Eps, A. V., & Pollitt, C. C. (2006). Equine laminitis induced with oligofructose. *Equine veterinary journal* , 38 (3), 203-208.
 12. Milinovich, G. J., Klieve, A. V., Pollitt, C. C., & Trott, D. J. (2010). Microbial events in the hindgut during carbohydrate-induced equine laminitis. *Veterinary Clinics of North America : Equine Practice* , 26 (1), 79-94.