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May 20th, 12:30 PM


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Amy S. Biddle
University of Massachusetts Amherst

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Biddle AS, Black SJ, Blanchard JL. (2014). An In Vitro Model of the Horse Gut Microbiome Enables Identification of Lactate-Utilizing Bacteria That Differentially Respond to Starch Induction. UMass Center for Clinical and Translational Science Research Retreat. Retrieved from https://escholarship.umassmed.edu/cts_retreat/2014/posters/23

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An In Vitro Model of the Horse Gut Microbiome Enables Identification of Lactate-Utilizing Bacteria That Differentially Respond to Starch Induction

Amy S. Biddle¹, Samuel J. Black², Jeffrey L. Blanchard^{3,4,5}

¹Department of Microbiology; ²Department of Veterinary and Animal Science; ³Graduate Program in Molecular and Cellular Biology; ⁴Department of Biology; ⁵Graduate Program in Organismal and Evolutionary Biology, University of Massachusetts, Amherst, Massachusetts

Contact:

Jeffrey Blanchard

email: jeffb@bio.umass.edu

phone: 413-77-2130

Abstract

Laminitis is a chronic, crippling disease triggered by the sudden influx of dietary starch. Starch reaches the hindgut resulting in enrichment of lactic acid bacteria, lactate accumulation, and acidification of the gut contents. Bacterial products enter the bloodstream and precipitate systemic inflammation. Hindgut lactate levels are normally low because specific bacterial groups convert lactate to short chain fatty acids. Why this mechanism fails when lactate levels rapidly rise, and why some hindgut communities can recover is unknown. Fecal samples from three adult horses eating identical diets provided bacterial communities for this *in vitro* study. Triplicate microcosms of fecal slurries were enriched with lactate and/or starch. Metabolic products (short chain fatty acids, headspace gases, and hydrogen sulfide) were measured and microbial community compositions determined using Illumina 16S rRNA sequencing over 12-hour intervals. We report that patterns of change in short chain fatty acid levels and pH in our *in vitro* system are similar to those seen in *in vivo* laminitis induction models. Community differences between microcosms with disparate abilities to clear excess lactate suggest profiles conferring resistance of starch-induction conditions. Where lactate levels recover following starch induction conditions, propionate and acetate levels rise correspondingly and taxa related to *Megasphaera elsdenii* reach levels exceeding 70% relative abundance. In lactate and control cultures, taxa related to *Veillonella montpellierensis* are enriched as lactate levels fall. Understanding the microbial dynamics underlying lactic acidosis and laminitis will lead to better informed models of health and the development of a probiotic treatment to prevent acidosis