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Original Research

Effects of High-Sugar and High-Starch Diets on Postprandial Inflammatory Protein Concentrations in Horses

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ABSTRACT

Mature, nonpregnant, Thoroughbred mares were used to determine the influence of high-starch and high-sugar diets on postprandial inflammation. Plasma samples were obtained hourly from mares (n = 12) consuming one of two treatment diets, either a diet high starch and sugar (STR) or the control (CON) diet that was low in starch and sugar. Plasma was analyzed for concentrations of lipopolysaccharide (LPS) and the inflammatory cytokines interleukin (IL)-1β and IL-6. Hour 0 was included as a covariate in the statistical model, and where interactions between the covariate and other model variables existed, simple effect means were separated at three levels of the covariate: lower 95% confidence limit (CL), mean, and upper 95% CL. For horses with low ($P = .016$) and average ($P = .065$) initial LPS concentrations, LPS was greater or tended to be greater in STR compared with CON at hour 2 after feeding. No other differences were detected for LPS concentrations. For horses with low ($P = .037$), average ($P = .006$), and high ($P = .001$) initial IL-1β concentrations, plasma IL-1β was greater in STR than CON at hour 2 after feeding. For horses with high initial IL-1β concentrations, IL-1β also tended to be greater at hour 3 ($P = .077$). For horses with low ($P = .022$) or average ($P = .063$) initial IL-6 concentrations, IL-6 was greater or tended to be greater at hour 1 than 0. No effect of diet was detected for horses that started with high initial IL-6 concentrations. High-starch and high-sugar diets increase postprandial IL-1β concentrations, and it is likely that this effect is independent of LPS.

1. Introduction

In horses, obesity leads to an increased risk of insulin resistance, and it is possible that consumption of high-glycemic diets (those high in starch and sugar, HSS) exacerbate the onset or degree of this dysfunction [1,2]. Recent research on 300 horses in Virginia indicated that more than half of the studied population was overweight or obese [3]. Furthermore, 70% of the studied population was offered a grain-based concentrate meal every day. Because insulin resistance increases the risk of laminitis [4], an excruciatingly painful disease of the equine hoof, it is important to determine how HSS diets could specifically influence insulin resistance.

It is possible that HSS diets induce insulin resistance by promoting increased plasma concentrations of proinflammatory cytokines. Cytokines such as interleukin 1β (IL-1β) [5], IL-6 [6], and tumor necrosis factor (TNF)-α [7]

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are known to reduce insulin sensitivity in a variety of species and tissue types. Additionally, the acute-phase protein, serum amyloid A (SAA), correlates with obesity and insulin concentration in horses [8] and alters insulin sensitivity in vitro in adipocytes [9]. In ruminants, there is evidence that starch fermentation, particularly after consumption of high-starch diets, promotes increased postprandial inflammation [10]. Similarly, fermentation of starch in the gastrointestinal tract could link HSS diets to increased inflammation in horse, which could be a factor relating HSS diets to insulin resistance.

Although starch is primarily digested in the small intestine, it overwhelms the digestive capacity of the small intestine when ingested in large enough concentrations and enters the cecum and large intestine (hindgut) where it is fermented by bacteria [11,12]. Starch is one of several carbohydrates that on reaching the hindgut are rapidly fermented. Although different from starch in its structure, oligofructose is a rapidly fermented carbohydrate that may have similar effects on the hindgut when it is consumed in large amounts. As soon as 4 hours after consuming a large quantity of oligofructose, cecal concentrations of organic acids (such as lactate) are altered, cecal pH is lowered [13], and blood concentrations of lipopolysaccharide (LPS) are increased [14]. In horses, IV infusion of LPS induces insulin resistance and increases the concentrations of several proinflammatory cytokines in plasma [15–18]. The ability of starch to influence plasma LPS and inflammation has only been shown after experimental overfeeding of starch, but we hypothesize that routine consumption of HSS diets generates a whole-body state of low-grade chronic inflammation that in turn facilitates and promotes insulin resistance and, further, that this inflammation occurs due to routine exposure to increased blood LPS.

2. Materials and Methods

All procedures were approved by Virginia Tech's Institutional Animal Care and Use Committee. Methods and results of a companion study were previously published [19] and are briefly described here.

2.1. Horses

Twelve mature (9–18 years), nonpregnant, Thoroughbred mares were used for this experiment. Mares were housed in drylots for 30 days before the beginning of the study to allow for acclimation to study conditions, including consuming concentrate twice daily in individual stalls. As previously described, mares were blocked by fasting insulin (0.4–16.6 mIU/L), age, and body condition (5–7; [20]), and two horses from each block were randomly assigned to each treatment (n = 6 to a low-starch control [CON] and n = 6 to a high-starch [STR] treatment diet) [19]. Horses were housed in three separate drylots, with two horses from each treatment per drylot. At all times throughout the study, horses had ad libitum water and iodized salt. Horses were brought into stalls for concentrate feeding at 8 AM and 2 PM and then returned to the drylots where they were group-fed hay. Daily observations showed that hay was consumed in entirety within a few hours, and thus horses were likely in a fasted state before consuming concentrate. Horses were not glucose intolerant nor did they have fasting hyperinsulinemia (CON = 2.1 mIU/L; STR = 5.6 mIU/L) before the start of the study [19]. Further, neither treatment induced fasting hyperinsulinemia (CON = 2.7 mIU/L; STR = 6.9 mIU/L) or glucose intolerance after 90 days of continuous feeding.

2.2. Treatments and Experimental Design

The experiment was a randomized complete block design with repeated measures, whereby drylots had staggered start dates of the study to enable intensive blood collections. Daily digestible energy (DE) requirements were estimated using the 2007 National Research Council (NRC) recommendations for horses at maintenance [21]. Treatment diets were formulated to contain either 10% (CON) nonstructural carbohydrates (NSC; sum of starch and ethanol soluble carbohydrate fractions) or 60% NSC (STR). Please see our previous article for the dietary ingredients and analysis [19]. All horses received the CON diet during the 30-day adaptation period, and those assigned to STR were abruptly switched on day 1 of the study. Horses were then fed their treatment diets for the duration of the 90-day study. Concentrate provision was estimated to provide 20% of daily DE requirements, with the remainder of energy coming from group-fed hay.

2.3. Blood Sampling

On days 1 and 90 of the study, blood samples were collected at −1, 60, 120, 180, 240, and 300 minutes relative to concentrate offering. On the day before testing, horses were fitted with indwelling 14-ga jugular venous catheters (Abbocath; Abbott Corp, Abbott Park, IL), using aseptic technique, and after sterilization and desensitization (2% lidocaine) of the overlying skin. Horses were maintained in individual stalls overnight with feed withheld for at least 10 hours. Blood samples were collected into 10-mL heparin-coated evacuated tubes (Vacutainer; Becton-Dickson, Franklin Lakes, NJ) and immediately centrifuged at 2,000*g* for 10 minutes and at 4°C. Plasma was stored in 1.5-mL tubes at −20°C until analysis.

2.4. Plasma Analysis

Plasma samples were analyzed using commercially available kits and previously published methods for concentrations of LPS (Pierce LAL Chromogenic Endotoxin Quantitation Kit; Thermo Scientific, Rockford, IL) [22], SAA (Phase SAA Assay; Tridelta Development Ltd, Maynooth, County Kildare, Ireland) [17], TNF (Equine TNF alpha ELISA Reagent Kit; Thermo Scientific) [23], and IL-1β (Equine IL-1 beta ELISA VetSet; Kingfisher Biotech, Inc, Saint Paul, MN) [8]. Plasma IL-6 concentrations were analyzed using previously published methods [24,25].