Insulin resistance is a condition in which there is a decrease in the physiologic response to insulin.\textsuperscript{1} Insulin resistance has been implicated in the pathogenesis of equine diseases such as hyperlipemia\textsuperscript{2} and laminitis\textsuperscript{3–5} and is a component of equine metabolic syndrome.\textsuperscript{4–7} Furthermore, there is evidence that environmental and managerial factors such as obesity, diet, and physical activity\textsuperscript{8–11} affect insulin sensitivity and thus may modify the risk for development of these disorders. Recognition of the relationship between altered insulin sensitivity and disease states has prompted interest in methods for quantification of insulin sensitivity for use in both research and clinical situations.

In human medicine, 2 methods are recommended for accurate estimation of in vivo insulin action: the EHC and FSIGTT.\textsuperscript{12,13} During the EHC, insulin is infused systematically at a constant rate and glucose is infused at a variable rate to clamp blood glucose concentration at baseline values (isoglycemia) or the accepted fasting concentration of 5 mmol/L (euglycemia).\textsuperscript{14} Under such steady-state conditions of euglycemia and hyperinsulinemia, the glucose infusion rate is an index of glucose uptake by tissues and is thus a measure of tissue sensitivity to insulin. This method is considered the gold standard for assessment of insulin sensitivity because it directly measures the effect of insulin on glucose use under steady-state conditions.\textsuperscript{15} The EHC has been criticized because it may not provide a truly physiologic assessment of insulin-mediated glucose uptake.\textsuperscript{16–18}

Comparison among the euglycemic-hyperinsulinemic clamp, insulin-modified frequently sampled intravenous glucose tolerance test, and oral glucose tolerance test for assessment of insulin sensitivity in healthy Standardbreds

**Objective**

To compare, in horses, estimates of insulin sensitivity obtained from minimal model analysis (MMA) of a frequently sampled IV glucose tolerance test (FSIGTT) with estimates from the euglycemic-hyperinsulinemic clamp (EHC) and to evaluate the validity of surrogate estimates of insulin sensitivity derived from an oral glucose tolerance test (OGTT).

**Animals**

18 mature Standardbreds (mean ± SD body weight, 428.9 ± 35.9 kg; mean ± SD body condition score, 4.4 ± 1.0 [on a scale of 1 to 9]).

**Procedures**

All horses underwent at least 2 of the 3 procedures (EHC [n = 15], insulin-modified FSIGTT [18], and OGTT [18]) within a 10-day time frame to evaluate insulin sensitivity.

**Results**

Insulin sensitivity variables derived from the EHC and FSIGTT were strongly correlated ($r = 0.88$). When standardized to the same units of measure, these measures were still strongly correlated ($r = 0.86$) but were not equivalent. Area under the curve, peak insulin concentration, insulin concentration at 120 minutes, and 2 calculated indices from glucose and insulin data from the OGTT were significantly correlated with the EHC- and FSIGTT-derived estimates of insulin sensitivity.

**Conclusions and Clinical Relevance**

In healthy Standardbreds with moderate body condition score, insulin sensitivities from the EHC and FSIGTT were strongly correlated but not equivalent. Estimates derived from an OGTT also may be useful to estimate insulin sensitivity. (Am J Vet Res 2015;76:84–91)
disposal.13 Nonetheless, the EHC has proven to be repeatable in horses10 and has been used extensively in equine research.9,17,18

The standard FSIGTT evaluates glucose and insulin dynamics after an IV bolus of glucose, whereas the insulin-modified FSIGTT involves the administration of a bolus of exogenous insulin 20 minutes after glucose administration. Mathematical modeling of the glucose and insulin responses (ie, MMA) provides estimates of insulin sensitivity (ie, SI_{MMA}), glucose effectiveness, AIRg, and DI (product of AIRg and SI_{MMA}).12,15,20 The FSIGTT has been used to assess insulin sensitivity in horses,20,21 and recently, optimal doses of both glucose and insulin have been established for the FSIGTT in horses to minimize urinary glucose spillover and to improve accuracy of the technique.22

Glucose and insulin data from an OGTT also can be used to calculate indices of insulin sensitivity,23,24 although interpretation of these data may be confounded by differences in gastric emptying and glucose absorption. Furthermore, the data derived from an OGTT are not a specific quantification of tissue sensitivity to insulin. In horses, an oral sugar test with corn syrup elicited insulin responses that were well correlated to those from an IV glucose tolerance test; however, high variance in insulin response was also reported.23 Other tests used to assess insulin sensitivity include the combined IV glucose and insulin test, which was designed to be more practical for clinical settings26; a 2-step insulin response test27; and insulin tolerance tests.2

Several studies19,28–30 in other species (humans, dogs, and pigs) have been conducted to determine whether results from the different methods for quantification of insulin sensitivity are well correlated and provide equivalent measures of insulin sensitivity when expressed in the same units. In several human studies,23,24,31 insulin sensitivity measurements from the EHC and FSIGTT were strongly correlated (r ≥ 0.75), whereas measures from an OGTT also were well correlated with those of the EHC. Other studies19,29,30 have found equivalence between the EHC and the FSIGTT when insulin sensitivity measurements are expressed in the same units.

To the authors' knowledge, there have been no studies in horses comparing SI_{MMA} with estimates of insulin sensitivity derived from the EHC. Furthermore, no previous study has compared indices of insulin sensitivity derived from an OGTT with values obtained by use of the gold standard EHC. Therefore, the objectives of the study reported here were to compare estimates of insulin sensitivity in horses obtained from MMA of an FSIGTT with estimates derived from the EHC and to evaluate the validity of surrogate estimates of insulin sensitivity derived from an OGTT with the EHC as the criterion measure.

**Materials and Methods**

Animals were owned by the University of Guelph. Animal care and use procedures were approved by the University of Guelph Animal Care Committee and performed in accordance with the guidelines of the Canadian Council on Animal Care.

**HORSES**

Eighteen mature Standardbreds (7 geldings and 11 mares; age range, 3 to 8 years) with a mean ± SD body weight of 428.9 ± 35.9 kg and body condition score22 of 4.4 ± 1.0 (on a scale of 1 to 9) that had not undergone any forced exercise for at least 3 months were used in this study. Diet consisted of forage cubes (mixed alfalfa and timothy hay) fed at 2% to 2.5% of body weight with free access to water and a salt block. Horses were housed in 12 X 12-foot box stalls and turned out in a drylot for 2 to 3 h/d. Fifteen of the horses underwent an EHC, an insulin-modified FSIGTT, and an OGTT. The remaining 3 horses were unavailable for the EHC, and only the FSIGTT and OGTT were administered. Tests were administered in random order to ensure there was no pattern to the test order, with a minimum of 3 days between procedures, and all procedures were completed within a 10-day period. Feed was withheld before (approx 10 hours) and during the tests, with procedures beginning at 7:30 AM. Horses had access to water during the pretest period of feed withholding but were denied access to water during the testing protocols.

**EHC**

The 180-minute EHC procedure was performed according to methods described previously.18 After aseptic preparation and desensitization of the overlying skin, catheters (14-gauge, 5.25 inches in length) were inserted into the left and right jugular veins for blood sample collection and infusion, respectively. After collection of a baseline blood sample for glucose and insulin determinations, a constant rate insulin infusion of insulin (3 mU/min/kg) was started and continued for the subsequent 180 minutes. Blood glucose was clamped at approximately 90 mg/dL by a variable rate dextrose infusion.8 Blood samples (< 1 mL) were collected from the left jugular catheter every 5 minutes for the determination of glucose concentrations by an enzymatic method.7 The glucose infusion rate was adjusted if blood glucose deviated from the target concentration by > 4 mg/dL. Blood samples (approx 10 mL) were also collected every 15 minutes for the subsequent determination of serum immunoreactive insulin concentration.

Insulin-stimulated glucose uptake (M) was calculated during the last 90 minutes of the procedure, as previously described.15,16 In brief, insulin-stimulated glucose uptake (M; mg/kg/min) was calculated from the following equation:

\[
M = GIR - SC
\]

where GIR is the glucose infusion rate (mg/kg/min) and SC is the space correction (mg/kg/min). Space correction is a computation that accounts for any glucose that is added or removed from the glucose space
other than by metabolism. Space correction (mg/kg/min) was calculated as follows:

\[ SC = (G_1 - G_0) \times 0.19/\text{time interval} \]

where \( G_0 \) and \( G_1 \) are glucose concentrations from the previous and present time intervals, respectively. The mean space correction was 0.000 ± 0.001 mg/kg/min.

The steady-state insulin concentration was calculated as the mean of serum insulin concentrations measured during the final 90 minutes of the procedure. The change in insulin concentration was the steady-state insulin concentration minus the basal insulin concentration obtained prior to beginning the EHC. Insulin sensitivity during the EHC (SI\text{CLAMP}) was calculated as follows:

\[ SI_{\text{CLAMP}} = M/(\Delta I \times G) \]

where \( M \) is the insulin-stimulated glucose uptake, \( \Delta I \) is change in insulin concentration, and \( G \) is the steady-state glucose concentration during the last 90 minutes of the procedure. Whole-body insulin sensitivity (SI\text{CLAMP [whole body]}) \(^9\) was then calculated as follows\(^{1,19,29}\):

\[ SI_{\text{CLAMP}}(\text{whole body}) = SI_{\text{CLAMP}} \times BW \]

where \( BW \) is body weight

**INSULIN-MODIFIED FSIGTT**

Catheters were placed IV as already described. Five minutes after collection of a blood sample for determination of basal insulin and glucose concentrations, glucose (50% wt/vol dextrose, 0.3 g/kg) was administered IV. Subsequent blood samples for the determination of plasma glucose concentrations were collected at the following time points: 0, 1, 2, 3, 4, 5, 6, 7, 8, 10, 12, 14, 16, 19, 22, 23, 24, 25, 27, 30, 40, 50, 60, 70, 80, 90, 100, 120, 150, and 180 minutes after glucose administration. Blood samples for the subsequent determination of serum insulin were obtained at the following time points: 0, 2, 4, 6, 8, 10, 14, 19, 22, 24, 30, 40, 60, 90, 120, 150, and 180 minutes. Insulin (0.015 U/kg) was injected IV at 20 minutes. Horses stood in stocks and a catheter was inserted into the left jugular vein. Five minutes after the collection of a baseline blood sample, glucose (1 g/kg; 10% wt/vol solution) was administered via gastric gavage. Additional blood samples (17 mL) were collected every 15 minutes for the first hour, then every 30 minutes for the next 4 hours for measurement of plasma glucose and serum insulin concentrations.

The AUCs for glucose and insulin were determined by use of the trapezoidal method. The peak insulin (defined as the highest measured insulin concentration) concentration and insulin concentration at 120 minutes were also recorded.

An insulin sensitivity index developed for use in human subjects (composite whole-body insulin sensitivity index [ISI\text{CLAMP}]) was also calculated. This index uses baseline glucose (mmol/L) and insulin (µU/mL) concentrations and the mean insulin and glucose response during the OGTT:

\[ \text{ISI}_{\text{CLAMP}} = 10,000/\sqrt{(G \times I) \times (MG \times MI)} \]

where \( G \) and \( I \) are the baseline glucose (mmol/L) and insulin (µU/mL) concentrations, respectively, and \( MG \) and \( MI \) are the mean glucose and insulin concentrations obtained during the OGTT. The numerator value of 10,000 is used to bring the values between 0 and 12.\(^{24}\)

Another insulin sensitivity index was obtained from the OGTT on the basis of the method described by Soonthornpun et al. Briefly, the postloading glucose concentration without insulin (PLGCWI; mmol/L) was estimated from the baseline glucose concentration (G; mmol/L), the glucose load (body weight \( BW \) [kg]) divided by 180 to convert to millimoles, assuming 75% absorption of glucose from the intestine (on the basis of human data), and a glucose space of 0.19 L of body weight.

\[ \text{PLGCWI} = G + [0.75 \times (BW/180)]/(BW \times 0.19) \]

Because the AUC for glucose (AUC\text{GLU}) represents hepatic glucose production and unused glucose from the oral load, the area above the glucose curve (AAGC; mmol) represents peripheral glucose use:

\[ \text{AAGC} = [\text{Time} \times \text{PLGCWI}] - \text{AUC}_{\text{GLU}} \times (BW \times 0.19) \]

where Time is the time of OGTT in minutes. Then, the glucose disposal rate (M; µmol/kg/min) was calculated:

\[ M = (\text{AAGC}) \times 1,000/(BW \times \text{Time}) \]

The insulin sensitivity index obtained from the OGTT (ISI\text{OGTT}) was then calculated by dividing the

where \( G_2 \) is the glucose concentration at 2 minutes after injection and \( G \) is the baseline glucose concentration prior to injection of the glucose bolus.\(^{28,30}\)
glucose disposal rate by the AUC for insulin (AUCINS; pmol) and the resulting value was multiplied by 100 for convenience.

\[ ISIOGTT = \frac{[M]}{AUCINS} \times 100 \]

**DETERMINATION OF PLASMA GLUCOSE AND SERUM IMMUNOREACTIVE INSULIN CONCENTRATIONS**

Blood samples were placed into tubes containing no additive or K₂EDTA and kept on ice until centrifugation (within 20 minutes). The harvested serum and plasma were stored at −20°C until analysis. Serum immunoreactive insulin was measured in duplicate by use of a commercially available solid-phase radioimmunoassay kit validated for use in horses. Intra- and interassay coefficient of variations were 5.5% and 10.5%, respectively (lower limit of detection, 3.0 µU/mL). Plasma glucose concentrations were analyzed in triplicate by use of the spectrophotometric method and a commercial kit. The within-assay CV for the glucose assay was < 3%. Plasma glucose concentrations are reported as mmol/L or mg/dL (glucose concentration in mmol/L was multiplied by a factor of 18).

**STATISTICAL ANALYSIS**

Descriptive statistics of continuous variables are expressed as mean ± SD or mean and 95% CI. Normality of distribution of the data was tested by means of the Shapiro-Wilk test, where a value of \( P > 0.05 \) indicated that the observed distribution of a variable is not statistically different from the normal distribution. Non-Gaussian distributed variables (ie, AIRg) were log transformed to achieve normality. Univariate Pearson correlation analyses were performed to examine the relationship between insulin sensitivity variables derived from the EHC and indices from the MMA and OGTT. The correlations between SIMM and OGTT-derived indices were also assessed. Linear regression was applied to SIMM × VdG and whole-body SI CLAMP to determine whether these values were equivalent. Concordance was defined as an intercept of 0.00 and a slope of 1.00. Lin concordance coefficient was also calculated. Significance was set at \( P \leq 0.05 \). Statistical computations were performed with software packages.

**Results**

Mean plasma glucose and serum insulin concentrations during the EHC (n = 15), FSIGTT (18), and OGTT (18) procedures are shown (Figure 1), and mean and 95% CIs of the calculated indices were summarized (Table 1). Baseline (pretest) plasma glucose (EHC, 86.7 ± 5.5 mg/dL; FSIGTT, 87.3 ± 4.5 mg/dL; OGTT, 84.9 ± 8.9 mg/dL) and serum insulin (EHC, 5.0 ± 5.5 µU/mL; FSIGTT, 7.0 ± 4.3 µU/mL; OGTT, 3.9 ± 2.0 µU/mL) concentrations did not differ among the 3 tests in the 15 horses that completed all procedures. During the EHC, steady-state blood glucose and serum insulin concentrations were achieved at 83.2 ± 9.5 minutes after the start of the insulin and dextrose infusions. Mean insulin concentration, as determined on the basis of 6 measurements/horse during the final 90 minutes of the EHC, was 303.9 ± 56.9 µU/mL. Mean blood glucose concentration during the same period was 90.4 ± 1.9 mg/dL.

In the FSIGTT, glucose concentration increased to a peak of 346.4 ± 46.6 mg/dL by approximately 2 minutes after injection. Thereafter, there was a biphasic decline in plasma glucose concentration, and mean concentrations were not different from baseline between 60 and 180 minutes. Mean serum insulin concentration increased from 7.0 ± 4.3 µU/mL at baseline to 31.9 ± 10.7 µU/mL at 2 minutes after glucose administration, and mean concentrations remained at this level between 4 and 19 minutes (Figure 1). One minute after the administration of exogenous insulin, mean serum insulin concentration was 653.4 ± 140.9 µU/mL. Thereafter, there was a curvilinear decrease in

![Figure 1](https://example.com/figure1.png)

**Figure 1**—Plasma glucose (solid lines) and serum immunoreactive insulin (dotted lines) concentrations in Standardbreds during EHC (A; n = 15), insulin-modified FSIGTT (B; 18), and OGTT (C; 18).
serum insulin concentration with a return to baseline by 150 minutes after glucose injection. During the OGTT, mean plasma glucose concentration increased from a baseline value of 84.9 ± 8.9 mg/dL to a peak of 148 ± 21.5 mg/dL at 90 minutes after glucose administration and had returned to baseline after 360 minutes. Mean serum insulin concentration increased from 3.9 ± 2.0 μU/mL at baseline to 34.5 ± 11.0 μU/mL at 120 minutes. Thereafter, there was a steady decrease in serum insulin concentration with a mean of 7.0 ± 4.9 μU/mL at 360 minutes (Figure 1).

Results of the univariate correlation analysis that compared indices from the EHC, MMA, and OGTT were summarized (Table 2). There was a significant \( P < 0.001 \) positive correlation between whole-body \( SI_{CLAMP} \) and \( SI_{MM} \times V_dG \), with a linear regression equation (Figure 2) of \( SI_{MM} \times V_dG = 2.26 \times SI_{CLAMP} \) (whole body) – 0.06. The \( SI_{MM} \) and \( SI_{CLAMP} \) also were significantly \( P = 0.001 \) correlated. Concordance was defined as an intercept of 0.00 and a slope of 1.00,19 and even though the intercept was close to 0 (0.06), the slope was not equal to 1.0. Lin concordance coefficient was 0.245, which is considered poor.17 Other indices from the MMA, including DI and glucose effectiveness, were not correlated with \( SI_{CLAMP} \) whereas AIRg was significantly correlated with \( SI_{CLAMP} \).

Among the indices derived from the OGTT, AUC was for insulin, peak insulin concentration, and insulin concentration at 120 minutes were significantly correlated with \( SI_{CLAMP} \) (Table 2). These indices were also significantly correlated with \( SI_{MM} \). Further, both composite whole-body insulin sensitivity index and insulin sensitivity index obtained from the OGTT were also significantly correlated with the \( SI_{CLAMP} \) and \( SI_{MM} \).

### Discussion

Although previous equine studies have applied the EHC or insulin-modified FSIGTT for estimation of insulin sensitivity, the present study was the first to directly compare these methods. Furthermore, our study appears to be the first to examine the relationship of OGTT-derived indices of insulin sensitivity with estimates derived from the EHC and the MMA of the FSIGTT in horses. The primary findings were that measures of insulin sensitivity from the EHC and MMA of an insulin-modified FSIGTT are well correlated but not equivalent, and indices derived from an OGTT, including AUC for insulin, peak insulin concentration,
and insulin concentration at 120 minutes, also may be useful for estimation of insulin sensitivity.

There was a strong correlation between SI\textsubscript{MM} and SI\textsubscript{CLAMP}, a finding consistent with results of studies in dogs,\textsuperscript{28} humans,\textsuperscript{38,39} and cats\textsuperscript{40} that have compared these methods. Initial human studies\textsuperscript{38,39} found a poor ($r = 0.44$) correlation between insulin sensitivity from the MMA and that from the EHC. However, subsequent studies\textsuperscript{38,39} that used a modified FSIGTT protocol in which tolbutamide (an insulin secretagogue) or insulin was injected 20 minutes after glucose demonstrated a stronger correlation ($r > 0.8$) between SI\textsubscript{MM} and glucose uptake during the EHC. Similarly, the present study revealed a strong correlation ($r > 0.85$) between insulin sensitivity measures derived from MMA and the EHC, and this is consistent with previous studies in dogs\textsuperscript{28} and cats\textsuperscript{40} in which the insulin-modified FSIGTT was used.

A strong correlation also was evident when SI\textsubscript{MM} and SI\textsubscript{CLAMP} were transformed to the same units, SI\textsubscript{MM} X Vd\textsubscript{e} and whole-body SI\textsubscript{CLAMP}, respectively. However, these values did not meet the criteria for equivalence as outlined by Bergman et al\textsuperscript{19} in which the linear regression line would need to have an intercept of 0.00 and the slope should not be significantly different from 1.00. Furthermore, the low Lin concordance coefficient indicated that whole-body SI\textsubscript{CLAMP} and SI\textsubscript{MM} X Vd\textsubscript{e} were not equivalent.\textsuperscript{17} Therefore, although the estimates of insulin sensitivity from the EHC and FSIGTT were well correlated in the study reported here, these tests did not yield equivalent values for insulin sensitivity. Equivalence between the values of insulin sensitivity from the EHC and the FSIGTT has been reported in human studies\textsuperscript{18,19} but was found neither in the present study, nor in a study of miniature pigs.\textsuperscript{30} Christoffersen et al\textsuperscript{30} suggested a lack of equivalence with these tests in pigs might be the result of physiologic differences between species or differences between testing methods, and these reasons may also explain our findings.

We also reported moderate correlations between EHC and FSIGTT measures and the several indices derived from the OGTT including the AUC for insulin, peak insulin concentration, composite whole-body insulin sensitivity index, and insulin sensitivity index obtained from the OGTT. These findings are consistent with observations made in human subjects, wherein there were poor to moderate correlations between simple OGTT-derived indices, such as AUC for insulin, and direct measurements of insulin sensitivity.\textsuperscript{21,24} Other studies in horses have reported conflicting information on the association between indices of insulin sensitivity derived from oral and IV testing protocols. Schuver et al\textsuperscript{19} reported positive correlations for AUC for glucose (Spearman $r = 0.58$) and AUC for insulin (Spearman $r = 0.90$) when comparing responses to IV glucose administration with responses to oral administration of a commercial corn syrup product (0.15 g of sugars/kg; oral sugar test) in a cohort of healthy Quarter Horses and horses meeting the criteria for equine metabolic syndrome. On the other hand, Banse and McFarlane\textsuperscript{41} found no correlation (Spearman $r = 0.13$; $P = 0.75$) between insulin sensitivity derived from an EHC and insulin concentration measured at 75 minutes during an oral sugar test in 15 light breed horses of varied body condition score. Differences in experimental design, including testing methods, breed and diversity of horses, and insulin sensitivity indices, could account for discrepancy among studies that have compared oral and IV tests.

During an OGTT, glucose tolerance reflects the efficiency with which homeostatic mechanisms, including insulin-dependent processes, restore blood glucose concentrations to basal levels. Therefore, in principle, the OGTT should yield a true physiologic estimate of insulin sensitivity. However, compared with an IV glucose tolerance test, the OGTT is complicated by factors such as the rates of gastric emptying and splanchnic glucose uptake, both of which affect the rate of glucose entry into circulation.\textsuperscript{15} In addition, the insulin response to an oral glucose load differs from that induced by IV glucose administration owing to the effects of gut-derived hormones (enteroinsular axis). Some or all of these factors are potential explanations for the lower correlations between the OGTT-derived indices and SI\textsubscript{CLAMP} and SI\textsubscript{MM} observed in the present study.

Plasma glucose concentrations following an oral glucose load also reflect hepatic glucose production, which is inhibited by the insulin infusion of the EHC method. The composite whole-body insulin sensitivity index represents a composite of both hepatic and peripheral insulin sensitivity on the basis of glucose and insulin concentrations from baseline blood samples and following the glucose load of the OGTT.\textsuperscript{24} The insulin sensitivity index obtained from the OGTT accounts for peripheral glucose use only by estimating the area above the glucose curve, and research in humans has found stronger correlations with the EHC measure, accordingly.\textsuperscript{24} We did not collect urine to account for urinary glucose losses as outlined by the methods of Soonthornpun et al.,\textsuperscript{23} perhaps limiting the value of the insulin sensitivity index obtained from the OGTT in this study. Regardless, compared with the EHC measure, the insulin sensitivity indices calculated in this study (composite whole-body insulin sensitivity index and insulin sensitivity index obtained from the OGTT) did not provide substantial benefit over peak insulin concentration or insulin concentration at 120 minutes for estimating insulin sensitivity with an OGTT.

Selection of either the FSIGTT or EHC in a research setting should be made on the basis of the information hoped to be gained. For example, a benefit of the EHC is the ability to establish a steady state, in which tissue samples (eg, muscle) may be obtained during the steady-state period of the EHC for determination of insulin-stimulated changes to signaling pathways.\textsuperscript{15} Alternatively, an advantage of the FSIGTT with MMA is the additional information that can be
obtained, such as the glucose effectiveness, AIRg, and DI that are derived from this method.

In the present study, a single breed (Standardbred) was represented and insulin sensitivity in all horses would have been considered normal. In addition, this study examined a relatively narrow population of horses: all horses were lean (body condition score < 5.5) and did not have evidence of prior laminitis. Therefore, it is recommended that future studies that compare different methods include horses with a broader range of body condition scores and insulin sensitivity.

Footnotes

References


