Hyperglycemia/hyperinsulinemia after feeding a meal of grain to young horses with osteochondritis dissecans (OCD) lesions

S. L. Ralston

Summary
To test the hypothesis that hyperinsulinemia/hyperglycemia may be correlated with OCD, plasma glucose and insulin responses to feeding high grain rations were evaluated in 15 young Standardbred horses. Four horses had osteochondritis dissecans (OCD), the other horses were normal (NL). Horses with OCD had higher (p<.01) postprandial glucose and insulin responses to feeding than did NL horses. Age differences in responses were also observed. Postprandial hyperglycemia and/or hyperinsulinemia may be correlated with the development of OCD lesions in young Standardbred horses.

Keywords: osteochondritis dissecans, glucose, insulin, age

Introduction
Osteochondritis dissecans (OCD) is a major problem in the equine industry (Grondahl 1991; Jeffcott 1991). Osteochondrotic lesions may cause lameness and reduce the perceived potential for optimal performance (Gaustad et al. 1995; Laws et al. 1993; McIlwraith et al. 1991). In addition, OCD has been incriminated as a contributing factor in catastrophic breakdowns in racehorses (Krook and Maylin 1988). Osteochondritis dissecans is common in Standardbred horses, primarily affecting the hock (Hoppe 1984; McIlwraith et al. 1991). Lesions usually develop in horses between 3 to 12 months of age and new lesions rarely appear in horses over a year old (Jeffcott 1991).

High grain rations are frequently cited as a potential cause of OCD (Glade and Belling 1986; Lewis 1995), though not all young horses fed large amounts of grain develop the problem. Ingestion of grain concentrate results in significant elevations in blood glucose which stimulate insulin release (Freestone et al. 1992; Rodiek et al. 1991; Ralston and Baile 1982; Argenzio and Hintz 1972). However glucose/insulin responses to a standard grain meal may differ markedly between individuals and may be affected by diet (Ralston 1995; Ralston et al. 1993; Jacobs and Bolton 1982). Insulin is known to affect cartilage growth through its influence on growth hormone and somatomedin release (Glade 1986). It has been hypothesized that postprandial hyperglycemia and hyperinsulinemia induced by high grain intake cause changes in thyroxine and growth hormone release which in turn cause OCD (Glade 1986; Glade and Belling 1986).

Genetic predisposition to OCD has been well documented in the Standardbred horse (Gaustad et al. 1995; Grondahl and Dolvik 1993, Philsson et al. 1993). However the nature of the hereditary defect which increases the risk of OCD in horses has not been determined.

We hypothesized that grain-induced hyperglycemia/hyperinsulinemia would be greater in young horses that had radiographic evidence of OCD than in animals of the same age that were radiographically normal.

Materials and methods
Data from three two-year-old geldings, four yearling (10–14 months old) geldings and eight weanling (3–10 months old) colts were used. All horses were registered Standardbreds. Four horses (one two-year-old, two yearlings and one weanling) had radiographic evidence of OCD at the time of testing (OCD), the other eleven horses (NL) had no radiographic abnormalities. The horses were fed 50% textured sweet grain mix (Omolene 300, Purina Mills, St. Louis, MO, USA), 50% alfalfa/grass mix hay in amounts that met or exceeded NRC (1989) nutrient recommendations for rapid growth. Rations were divided into two equal feedings. Glucose and insulin responses to feeding were measured in 14 horses four times at four week intervals. One NL weanling was only tested twice though on the same protocol as the others. Blood for glucose
and insulin analyses was drawn from preplaced indwelling venous catheters before feeding grain (1.7 to 2.5 kg, depending on age and body weight) and hay (2 to 3 kg) at 0800 h, then hourly for 6 h. The blood was drawn into heparinized tubes (Vacutainer, Becton Dickinson, Inc., Rutherford, NJ), centrifuged immediately and plasma drawn off and stored at <0 °C pending analysis. Plasma glucose concentrations were determined by automated dry chemistry analysis (VetTest 8000 autoanalyzer, IDEXX Inc, Westbrook, ME). Insulin concentrations were determined by radioimmunoassay (Kit TKIN1, Diagnostic Products Corp, Los Angeles, CA) previously validated for equine insulin analysis (Freeston et al. 1991).

Rates of change in glucose and insulin and glucose/insulin ratios were calculated from the data. Glucose and insulin data were subjected to stepwise regression analysis (Analytical Software, 1994) factoring the effects of trial, presence or absence of OCD, age and, where appropriate, time after feeding. For parameters that were identified by regression as contributing significantly (p<.05) to the variability observed, means were compared between groups by two tailed T-test. Data were subjected to stepwise regression analysis (Analytical Software, 1994). Glucose and insulin ratios were calculated from the data. Glucose and insulin concentration-age and body weight) and hay (2 to 3 kg) at 0800 h, then immediately and plasma drawn off and stored at <0 °C (Vacutainer, Becton Dickinson, Inc., Rutherford, NJ), centrifuged before feeding grain (1.7 to 2.5 kg, depending on the animal). Plasma glucose concentrations were determined by automated dry chemistry analysis (VetTest 8000 autoanalyzer, IDEXX Inc, Westbrook, ME). Insulin concentrations were determined by radioimmunoassay (Kit TKIN1, Diagnostic Products Corp, Los Angeles, CA) previously validated for equine insulin analysis (Freeston et al. 1991).

Results and discussion

Young horses that had radiographic evidence of OCD had greater (>0.02) postprandial changes in glucose and insulin when fed high grain rations than did NL horses (n=11) (Tab. 1 and 2). Mean postprandial plasma glucose and insulin were higher (>0.02) in OCD horses (glucose: 8.59±2.69 mmol/L; insulin: 43.7±3.1 IU/dl) than in NL horses (glucose: 7.89±0.80 mmol/L; insulin: 27.8±1.0 IU/dl). Mean rate of glucose increase was higher (<0.05) in OCD horses than NL horses at 1 hour (NL: 0.37±0.03 mmol/min; OCD: 0.68±0.09 mmol/min, p<.01) and 2 hours (NL: -0.05±0.02 mmol/min; OCD: -0.18±0.07 mmol/min, p<.01) after feeding. Concomitant insulin rates of increase also differed between groups.

Tab. 1: Plasma glucose (mmol/L) in normal versus OCD horses after feeding meals of sweet grain mix plus hay.

<table>
<thead>
<tr>
<th>Time after feeding (hours)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>SEM²</th>
</tr>
</thead>
<tbody>
<tr>
<td>NL¹</td>
<td>8.89±(1.13)</td>
<td>9.12±(2.03)</td>
<td>8.86±(2.63)</td>
<td>8.01±(1.63)</td>
<td>7.78±(0.12)</td>
<td>7.29±(0.12)</td>
<td>7.04±(0.09)</td>
<td>1.17</td>
</tr>
<tr>
<td>OCD²</td>
<td>6.22±(1.17)</td>
<td>10.33±(2.52)</td>
<td>11.44±(0.06)</td>
<td>10.18±(1.52)</td>
<td>8.59±(0.50)</td>
<td>7.01±(0.36)</td>
<td>6.35±(0.27)</td>
<td>0.49</td>
</tr>
</tbody>
</table>

1 Values are means (±SE) for 4 replicates for 10 horses and two replicates for one horse.
2 Values are means (±SE) for 4 replicates for four horses.
3 SEM=Standard error of mean.
4 OCD differs from NL within time period (p<.05).
5 OCD differs from NL within time period (p<.01).

OCD (0.018±.007 mmol/min, p<.01) after feeding. Concomitant glucose/insulin remained equal, but significantly increased in OCD horses (p<.01) compared to NL horses (p<.001; Two hours: NL: -.03±.02 IU/min, OCD: -.29±.07 IU/min, p<.01). Glucose/insulin ratios however did not differ (NL: -.40±.02; OCD: -.37±.04, p>.1).

Tab. 2: Plasma insulin (IU/dl) in normal versus OCD horses after feeding meals of sweet grain mix plus hay.

<table>
<thead>
<tr>
<th>Time after feeding (hours)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>SEM²</th>
</tr>
</thead>
<tbody>
<tr>
<td>NL¹</td>
<td>10.2±(2.0)</td>
<td>35.8±(2.3)</td>
<td>37.4±(2.7)</td>
<td>36.9±(3.1)</td>
<td>34.2±(1.9)</td>
<td>25.4±(1.9)</td>
<td>18.9±(1.4)</td>
<td>2.3</td>
</tr>
<tr>
<td>OCD²</td>
<td>8.8±(1.7)</td>
<td>48.1±(3.1)</td>
<td>65.5±(6.7)</td>
<td>66.1±(9.0)</td>
<td>61.7±(10.0)</td>
<td>35.1±(5.4)</td>
<td>20.5±(3.6)</td>
<td>6.3</td>
</tr>
</tbody>
</table>

1 Values are means (±SE) for 4 replicates for 10 horses and two replicates for one horse.
2 Values are means (±SE) for 4 replicates for four horses.
3 SEM=Standard error of mean.
4 OCD differs from NL within time period (p<.05).
5 OCD differs from NL within time period (p<.01).

Tab. 3: Plasma glucose (mmol/L) in NL colts 3 to 14 months old colts and geldings versus NL two-year-old geldings after feeding meals of sweet grain mix plus hay.

Plasmaglucose (mmol/L) bei 3 bis 14 Monate alten NL-Hengsten und -Wallachen sowie zweijährigen Wallachen nach Fütterung einer Getreidemischung mit Heu.

<table>
<thead>
<tr>
<th>Time after feeding (hours)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>SEM²</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-14 month olds</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>NL: 7.17±(0.11)</td>
<td>9.50±(0.19)</td>
<td>9.98±(0.25)</td>
<td>8.56±(0.19)</td>
<td>8.00±(0.12)</td>
<td>7.49±(0.11)</td>
<td>7.21±(0.11)</td>
<td>1.17</td>
<td></td>
</tr>
<tr>
<td>Two-year-olds</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>NL: 5.72±(0.32)</td>
<td>7.53±(0.30)</td>
<td>6.71±(0.23)</td>
<td>7.14±(0.21)</td>
<td>6.86±(0.15)</td>
<td>6.45±(0.15)</td>
<td>6.06±(0.16)</td>
<td>0.23</td>
<td></td>
</tr>
</tbody>
</table>

1 Values are means (±SE) for 4 replicates for 8 horses and two replicates for one horse.
2 Values are means (±SE) for 4 replicates for four horses.
3 SEM=Standard error of mean.
4 OCD differs from NL within time period (p<.05) by Two way T-test.
5 OCD differs from NL within time period (P<.01) by Two way T-test.

Weanling colts and yearling geldings without radiographic evidence of OCD (n=9) had greater (<0.001) postprandial changes in glucose than did NL horses >16 months of age (n=2) (Tab. 3). However insulin responses did not differ between the age groups (p>.50) (Tab. 1). Mean postprandial plasma glucose and insulin were higher (<0.001) after feeding. Concomitant glucose/insulin ratios however did not differ (NL: -.40±.02; OCD: -.37±.04, p>.1).

(One hour: NL: .42±.04 IU/min, OCD: .65±.06 IU/min; p<.001; Two hours: NL: .03±.02 IU/min, OCD: .29±.07 IU/min, p<.01). Glucose/insulin ratios however did not differ (NL: .40±.02; OCD: .37±.04, p>.1).

Weanling colts and yearling geldings without radiographic evidence of OCD (n=9) had greater (<0.001) postprandial changes in glucose than did NL horses >16 months of age (n=2) (Tab. 3). However insulin responses did not differ between the age groups (p>.50) (Tab. 4). Mean postprandial plasma glucose and insulin were higher (<0.001) after feeding. Concomitant glucose/insulin ratios however did not differ (NL: .40±.02; OCD: .37±.04, p>.1).
Conclusions

Young horses with OCD lesions may have greater postprandial hyperglycemia/hyperinsulinemia than those that do not have lesions. The existence of glucose intolerance and apparent insulin resistance in horses <14 months old relative to horses >16 months old strengthens the hypothesis that hyperglycemia and/or hyperinsulinemia may be correlated with the development of OCD in young horses.

References


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Tab. 4: Plasma insulin (IU/dl) in NL colts 3 to 14 months old colts and geldings versus NL two-year-old geldings after feeding meals of sweet grain mix plus hay.

Time after feeding (hours)

1 2 3 4 5 6

SEM

3-14 month

old 4

0.9 ± 0.1

3.3 ± 0.4

3.6 ± 0.4

3.7 ± 0.4

3.4 ± 0.4

25.1 ± 0.4

19.4 ± 0.4

2.6

Two-year-

old 5

11.5 ± 0.1

4.4 ± 0.2

3.9 ± 0.2

4.1 ± 0.2

3.4 ± 0.2

26.5 ± 0.2

16.6 ± 0.2

4.9

1 Values are means (±SE) for 4 replicates for 8 horses and two replicates for one horse.

2 Values are means (±SE) for 4 replicates for 4 horses.

3 SEM = standard error of mean

4 OCD differs from NL within time period (P<.05) by Two way T-test.

5 OCD differs from NL within time period (P<.01) by Two way T-test.