How to Feed Horses With Endocrine Disorders

Nicholas Frank, DVM, PhD, Diplomate ACVIM

Horses with pituitary pars intermedia dysfunction or equine metabolic syndrome require special consideration because insulin resistance can be associated with these conditions. Sufficient calories must be provided to these patients without exacerbating insulin resistance or triggering laminitis in susceptible animals. Author’s address: 2407 River Drive, Knoxville, TN 37996; and University of Nottingham School of Veterinary Medicine and Science, Nottingham, United Kingdom; e-mail: nfrank@utk.edu. © 2007 AAEP.

1. Which Endocrine Disorders Require Dietary Management?
The endocrine disorders of interest for this discussion are pituitary pars intermedia dysfunction (PPID; also called equine Cushing’s disease) and equine metabolic syndrome (EMS), which is a collection of problems that includes insulin resistance (IR), obesity and/or regional adiposity, and laminitis. IR plays an important role in both PPID and EMS, so we will focus on this disturbance.

2. What Is IR?
IR can be broadly defined as a decrease in tissue responses to circulating insulin, which causes a decrease in insulin-mediated glucose uptake into skeletal muscle, adipose, and liver tissues. When using the combined glucose-insulin test (CGIT), IR is recognized by the slower decline in blood glucose concentrations after exogenous insulin administration, indicating that insulin-sensitive tissues are less responsive to the hormone. Compensated IR is the most common glucose and insulin metabolism abnormality identified in horses and ponies. In this situation, serum insulin concentrations are higher than normal because more insulin is secreted from the pancreas to compensate for lower tissue responses. Compensated IR is thought to lead to uncompensated IR if pancreatic insufficiency develops. This is sometimes also referred to as β-cell exhaustion and is recognized when serum insulin concentrations are lower than expected for the blood glucose concentrations detected. Uncompensated IR can lead to type 2 diabetes mellitus if blood glucose levels rise above the renal threshold and glucosuria develops. In horses that we have evaluated, compensated IR is the most common abnormality detected, whereas uncompensated IR is rare and is usually associated with advanced PPID. Diabetes mellitus is very rare in horses.

IR is associated with PPID because glucocorticoids decrease glucose uptake into insulin-sensitive tissues such as skeletal muscle and adipose tissue and increase gluconeogenesis within the liver. This causes hyperglycemia, which preserves glucose delivery to tissues to high-priority tissues such as the brain during times of stress or danger. It is presumed that PPID induces chronic IR through increased cortisol production, but studies have not been performed to determine the relative importance of adrenocorticotropic hormone (ACTH) itself and other pituitary products, such as cortico-
trophin-like intermediate peptide (CLIP), in horses with PPID.

Physiological Versus Pathological IR
Known physiological causes of IR include stress, administration of exogenous corticosteroids, and gestation. Administration of dexamethasone induces IR in both humans and horses, and it has been shown that exogenous epinephrine inhibits glucose uptake after dextrose administration. Dexamethasone-induced IR may result from alterations in both insulin signal transduction and glucose transporters. Pregnancy enhances the pancreatic response to glucose and lowers insulin sensitivity in mares. Fowden et al. detected exaggerated pancreatic insulin responses to exogenous glucose and feeding when pregnant mares were compared with non-pregnant animals. Insulin sensitivity decreases during pregnancy to ensure adequate delivery of glucose to the placenta and fetus. Glucose delivery to the placenta occurs along a concentration gradient that is not affected by insulin, so reductions in insulin sensitivity lower maternal glucose use. This ensures that sufficient glucose is available for the developing fetus, which gains 45% of its final weight after 270 days of gestation.

Obesity and IR
These conditions seem to be associated in horses, but a direct cause-and-effect relationship has yet been established. In a recent study of 300 mature (4–20 yr of age) client-owned horses in Virginia, the prevalence of hyperinsulinemia (>30 mU/l) was 10%, and 18 of 30 affected horses were judged to be obese on the basis of body condition score.a Results of this study suggest that obesity and IR are associated but also show that IR can occur in leaner horses.

Relationships between obesity and IR need further study, and dynamic testing may be needed to thoroughly study this association. Mild or early IR may not have been detected in the aforementioned study because only resting glucose and insulin concentrations were examined. More direct measures of insulin sensitivity may be needed to determine whether insulin sensitivity declines with the accumulation of body fat or if it is the duration of obesity that determines the magnitude of IR. If the duration of obesity proves to be important, weight management should be instituted sooner rather than later to prevent the development of IR.

IR in Leaner Horses With Regional Adiposity
These patients are more challenging to understand because IR is not accompanied by obesity per se. Horses may be leaner but exhibit regional adiposity in the form of a cresty neck, bulging supraorbital fat, and enlarged fat pads close to the tail base, or have subcutaneous adipose tissue masses randomly distributed throughout the trunk region. It is well established in human medicine that the accumulation of fat within the abdomen, which is sometimes called abdominal, omental, or central obesity, is a risk factor for IR and coronary heart disease. Increased 11β-hydroxysteroid dehydrogenase 1 (11β-HSD1) activity within these tissues is thought to raise local cortisol concentrations and alter adipocyte physiology. This condition has been referred to as peripheral Cushing’s disease in humans, and it has been suggested that 11β-HSD1 activity may also be higher in the adipose tissues of horses with IR. Expansion of adipose tissues within the neck region may indicate that adipocytes found at this location are more active in horses. Enlargement of the neck region ("cresty neck") was negatively associated with insulin sensitivity in a small group of obese IR horses that we studied. At present, it is equally likely that alterations in 11β-HSD1 activity are a consequence, rather than a cause, of IR, so further studies are needed to examine this question in horses.

3. Dietary Management of IR in Horses
Two important questions must be addressed before selecting feeds for insulin resistant horses—is the feed likely to exacerbate IR and will it increase the risk of laminitis?

These questions are related because it is now recognized that pasture-associated laminitis is triggered by gastrointestinal disturbances arising from alterations in bacterial flora that occur after consumption of pasture grass that is rich in fermentable carbohydrate. Insulin sensitivity therefore seems to affect the threshold for laminitis, because IR animals are more susceptible to the disease, whereas horses with normal insulin sensitivity are less likely to develop the condition.

Goals for managing IR are therefore to (1) reduce body fat mass in obese animals to improve insulin sensitivity, (2) avoid feeds that will exacerbate IR, (3) lower the risk of laminitis by improving insulin sensitivity through weight loss, diet, and exercise, and (4) avoid sudden changes in bacterial flora that might trigger laminitis.

Inducing Weight Loss in Obese IR Horses
Individual horses should be fed according to their metabolic needs. Obese horses that are easy keepers can be placed on a simple diet of hay and a vitamin/mineral supplement. Concentrates are not necessary for these obese horses, and weight loss should be promoted by restricting the horse’s caloric intake until its ideal weight and body condition are achieved. This ideal set-point differs between individual horses and breeds because the physical stature of the animal varies considerably. The horse must be taken out of an obese state, but it is not necessary for every horse to assume an underweight condition.

Weight loss strategies include dietary management and exercise. Obese horses should be fed
enough hay to meet their energy needs, which is usually equivalent to 1.5–2.0% of body weight (15–20 lb hay for a 1000-lb horse). Clients should be asked to weigh their hay so that the correct amount is fed. Horses that are lame because of laminitis should not be exercised until the hoof structures have stabilized, but other affected horses should be exercised as often as possible. The horse can be walked on a lead rope, exercised on a lunge line, or ridden. It is ideal for the horse to be exercised every day, and it is likely that this intervention also improves insulin sensitivity.

Avoiding Feeds That Exacerbate IR

In addition to exercise, care must be taken to avoid feeds that exacerbate IR. The horse with EMS or PPID is similar to a person with diabetes, so excessive sugar should be avoided. Unfortunately, it is very difficult to control sugar intake when horses are grazing freely on pasture. Pasture grass is one of the largest sources of sugar in the horse’s diet, and the carbohydrate content varies between regions and depends on soil type, climate, hours of sunlight, and grass species. It also varies according to season and time of day. This creates large fluctuations in carbohydrate intake, which can exacerbate IR and potentially alter the bacterial flora of the large intestine.

Access to pasture must therefore be restricted or eliminated when managing IR horses and ponies. Sometimes this is only necessary for a few months until weight loss is achieved or PPID becomes better controlled with pergolide therapy. However, there are some IR horses that have to be permanently housed in dirt paddocks because they are extremely sensitive to changes in pasture grass nutrient content. Thankfully, most horses and ponies with PPID or EMS can be managed by limiting grazing time to 1–2 h/d, housing in a grass paddock, strip grazing using an electric fence, or application of a grazing muzzle.

Feeds that are lower in sugar should be selected, and treats such as sugar cubes must be avoided. Hay fed to obese horses should have a lower (<12%) total non-structural carbohydrate (NSC) content, and samples can be tested by commercial laboratories.

Feeding More Calories Without Exacerbating IR

Some IR horses have a leaner overall body condition, but exhibit enlarged fat deposits (regional adiposity). Young horses with this appearance and a genetic predisposition towards obesity are EMS patients, whereas older horses are more likely to suffer from PPID. Some of these horses may still be exercising strenuously or competing, so they need more calories. If hay is not sufficient to provide these calories, a concentrate must be selected.

Thinner IR horses can usually be safely fed concentrates, but care must be taken to provide calories without exacerbating IR. There are three considerations when evaluating feeds for IR patients: (1) the carbohydrate composition of the feed, (2) the glycemic response that will follow ingestion, and (3) the feeding strategies that will be used. For instance, many sweet feeds have a high hydrolyzable NSC content, so the resulting glycemic response is likely to exacerbate IR when these feeds are provided as large meals. Feeds that contain less starch (and total NSC) are appropriate in these situations, and it is advisable to feed smaller amounts more frequently and give hay before concentrates.

We use several strategies when managing leaner IR horses that need additional calories, and the strategy selected depends largely on the appetite of the individual patient. Our strategies include the following:

1. A diet consisting of hay with a low (<12%) NSC content, soaked molasses-free sugar beet pulp, and 0.5 cup (equal to 125 ml; contains ~100 g fat) rice bran oil or corn oil added to the feed twice daily.
2. The same diet with a commercial low-starch specialty feed substituted for beet pulp (discussed in greater detail in Table 1).
3. Either diet with rice bran substituted for oil. Rice bran contains ~20% fat, and we usually recommend that 1 lb (~90 g fat) be fed twice daily.
4. Pelleted feed for geriatric horses in old patients with PPID or in horses with dental problems that need a complete feed.

Horses with finicky appetites sometimes refuse to eat beet pulp or pelleted specialty feeds. Clients may be forced to try several varieties of specialty feed until an acceptable one is found. These horses may also need a transition period as they adapt to beet pulp or the specialty feed. We are sometimes forced to feed oats with added rice bran or oil (in addition to hay) to horses that refuse lower NSC feeds. This is not an ideal diet for IR horses because oats contain more hydrolyzable NSC (e.g., starch) than most pelleted feeds (Table 1), but we attempt to lower the glycemic response by feeding smaller meals more frequently (three to four times daily) and request that hay be fed 15–30 min before the concentrates.

Beet pulp is recommended for the thinner IR horse, but it should be recognized that this feed provides calories, so it is not appropriate for obese horses. Obese IR horses are sometimes fed 0.5–1 cup (before soaking) of beet pulp to help with the delivery of supplements or as a treat, but no more should be fed if the horse is on a weight loss program. Sugar beet pulp is rich in rapidly fermentable carbohydrate, so addition of this ingredient to pelleted feeds lowers the hydrolyzable carbohydrate (e.g., starch) content. It also expands with water when soaked, so beet pulp provides bulk to satisfy the horse’s appetite. Care should be taken to soak...
beet pulp to avoid esophageal obstruction (choke) and remove molasses if this sugar has been added to the feed.

4. Lowering the Risk of Laminitis

Laminitis can develop when triggering factors are released from bacteria within the large intestine after sudden changes in diet. In the case of a grain founder, this occurs when the horse breaks into the feed room and eats too much grain. There is a rapid increase in the amount of sugar arriving in the large intestine, the bacterial flora of the colon becomes altered, intestinal permeability increases, and triggering factors are released into the blood. Pasture-associated laminitis develops in a similar way, except that the sugar comes from grass consumed on pasture. Laminitis is triggered by changes in bacterial flora, but the individual horse’s threshold for laminitis seems to determine whether disease develops. We hypothesize that insulin sensitivity plays an important role in determining the laminitis threshold.

Avoiding Sudden Changes in Bacterial Flora That Could Trigger Laminitis

The decision to allow access to pasture is the most important issue to be addressed when managing the IR horse with a previous history of laminitis. As mentioned previously, there are some horses that are very sensitive to alterations in pasture grass composition, so these animals must be permanently held off pasture. In general, we discourage owners from turning horses out on pasture if they have suffered from repeated episodes of laminitis, and special farrier care is needed. However, there are other horses that have a history of laminitis, yet this problem has resolved after the implementation of weight loss, diet, and exercise programs. These patients can return to limited grazing on pasture. This usually begins with 1–2 h of grazing once or twice a day or turnout with a grazing muzzle.

Pasture grass can also be submitted for analysis, and some clients monitor their pastures to identify low-risk times of the year for grazing. Pasture

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**Table 1. Non-Structural Carbohydrate Composition of Specialty Feeds (Randomly Selected)**

<table>
<thead>
<tr>
<th>Feed Name (Listed Alphabetically)</th>
<th>Manufacturer’s Recommendation (for 1000-lb Horse)</th>
<th>Digestible Energy (kcal/lb)*</th>
<th>NSC†‡ (%)</th>
<th>Starch‡ (%)</th>
<th>Sugar‡ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue Seal</td>
<td>2.5–7.5 lb/d</td>
<td>Max 11%</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CARB-GUARD</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Buckeye Feeds</td>
<td>4.5–6.0 lb/d</td>
<td>1250</td>
<td>11.9%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Safe ‘n Easy Pelleted</td>
<td>Fed with hay</td>
<td>1420</td>
<td>11.9%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Buckeye Feeds</td>
<td>4.5–6.0 lb/d</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Safe ‘n Easy Textured</td>
<td>Fed with hay</td>
<td>1030</td>
<td>Range 8–11%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LMF Low Non-Structural</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Carbohydrate Stage 1</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LMF Low Non-Structural</td>
<td>Fed with hay</td>
<td>1030</td>
<td>Range 8–11%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Carbohydrate Complete</td>
<td>Complete feed</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>McCauley Bros.</td>
<td>2.5–20 lb</td>
<td>1225</td>
<td>Max 15%</td>
<td>Max 9% (calc)</td>
<td>Max 5% (calc)</td>
</tr>
<tr>
<td>Alam</td>
<td>Fed with hay or complete</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Nutrena</td>
<td>2.5–5.0 lb</td>
<td>1000</td>
<td>Range 20–26%</td>
<td>Min 11%</td>
<td>—</td>
</tr>
<tr>
<td>Lite Balance</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Nutrena</td>
<td>2.5–5.0 lb</td>
<td>1360</td>
<td>Range 28–32%</td>
<td>~17%</td>
<td>—</td>
</tr>
<tr>
<td>SafeChoice</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Oats§</td>
<td>—</td>
<td>1500</td>
<td>49.7%</td>
<td>44.2%</td>
<td>3.6%</td>
</tr>
<tr>
<td>Purina</td>
<td>In development</td>
<td>1150</td>
<td>Range 10–13%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>WellSolve L/S</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Purina</td>
<td>In development</td>
<td>1000</td>
<td>Range 10–13%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>WellSolve W/M</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Triple Crown</td>
<td>20 lb initially</td>
<td>1100</td>
<td>8.8%</td>
<td>3.7%</td>
<td>5.0%</td>
</tr>
<tr>
<td>Safe Starch Forage</td>
<td>Complete feed</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Triple Crown</td>
<td>As needed</td>
<td>1428</td>
<td>15%</td>
<td>9.1%</td>
<td>5.9%</td>
</tr>
<tr>
<td>Low Starch</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Triple Crown</td>
<td>As needed</td>
<td>1150</td>
<td>15.9%</td>
<td>9.5%</td>
<td>6.4%</td>
</tr>
<tr>
<td>Lite</td>
<td>Fed with hay</td>
<td></td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Information available to the public by telephone inquiry or company web sites in January 2007.
*As-fed or dry matter basis (not specified).
†Non-structural carbohydrate.
‡Note that the methods used to provide NSC, starch, and sugar values were not specified or compared.
§Source: dry oats, Equi-Analytical Laboratories web site (http://www.equi-analytical.com/CommonFeedProfiles).
Classification of Carbohydrates by Method of Digestion

Hydrolyzable carbohydrates (CHO–H) primarily include simple sugars (monosaccharides and disaccharides) and most starches. These are all NSCs. Digestion of these carbohydrates may begin in the stomach but primarily occurs within the small intestine. However, it should be noted that overfeeding or rapid gastrointestinal transit results in hydrolyzable carbohydrates entering the large intestine, where they will be rapidly fermented.

Hydrolyzable carbohydrates are a major concern with respect to exacerbation of IR because small intestinal digestion results in a glycemic response that may be more difficult for the IR horse to cope with. This situation has not been studied extensively in IR horses, but it is reasonable to assume that repeated glycemic responses would exacerbate IR.

Carbohydrates that are easily hydrolyzed are also a concern because overfeeding can result in excessive quantities of sugar and starch within the large intestine, which will be rapidly fermented and might cause alterations in gastrointestinal flora that precipitate episodes of gas colic, colitis, or laminitis. Starch overload has been used as an experimental model for acute laminitis in horses.16

Fermentable carbohydrates (CHO–F) are primarily digested in the large intestine by the microflora that reside there.17 These carbohydrates include the following:

- Rapidly fermentable carbohydrates (CHO–FR): This group is composed of NSCs such as starches (those that are not hydrolyzed in the small intestine), oligosaccharides including oligofructose (fructan), and soluble fiber (gums, mucilages, and pectins). Digestion of these carbohydrates within the large intestine generates the volatile fatty acid propionate, which can be converted into glucose through gluconeogenesis. Beet pulp is rich in rapidly fermentable carbohydrate.

- Slowly fermentable carbohydrates (CHO–FS): This group is composed of structural carbohydrates such as cellulose, hemicelluloses, and ligno-cellulose (lignin is undigestible). Digestion of these carbohydrates within the large intestine provides the volatile fatty acid acetate, which is converted into acetyl CoA and used to provide energy via the TCA cycle, or converted into fat for energy storage.

Relevance to IR

Because some NSC measurements include both hydrolyzable and fermentable carbohydrates, it is important for us to know how NSC content is calculated. Ideally, the hydrolyzable carbohydrate content of the feed would be evaluated by examining starch and sugar concentrations, which give some insight into the expected glycemic response.
However, it should be recognized that the glycemic response is potentially influenced by the structure of the carbohydrate, presence of other ingredients in the feed (e.g., fat), and method of feeding (small versus large meals). Ideally, we would measure and compare the glycemic responses of each commercial feed, and assess these responses in IR horses and healthy animals. Based on our experiences with IR horses, it is likely that some individuals are much more sensitive to certain carbohydrates than others. These patients are particularly challenging to manage.

Classification of Carbohydrates by Solubility

Water-soluble carbohydrates (WSCs) include simple sugars (monosaccharides and disaccharides) and oligofructoses (fructans). A small amount of WSCs may be extracted using this method, but starches are generally excluded.

Ethanol-soluble carbohydrates (ESCs) include only simple sugars, and this measurement has recently been made available by a commercial laboratory.

Starches are generally excluded from water or ethanol extracts, so this is a separate measurement provided by enzymatic assay.

Relevance to IR

Examination of ESCs (simple sugars) and starch content allows better assessment of the hydrolyzable carbohydrate load and expected glycemic response after small intestinal digestion. Levels of other water soluble carbohydrates (e.g., fructans) are more relevant to laminitis.

Conclusions About NSCs

It is important to recognize that definitions of NSCs vary according to the methods used to measure carbohydrates, the formulae used by laboratories, and the current scientific literature. Here are some guidelines to follow when comparing feeds:

- Find out whether the company has evaluated the glycemic response to their feed. Also examine the other ingredients in the feed and the amounts recommended for feeding. Remember that the physiological response to the feed can only be crudely approximated by examining the nutrient content of the feed.

6. New Information About Carbohydrates

Watching for Changes in the Definition of NSCs

Commercial laboratories sometimes improve testing procedures. Dairy One Forage Laboratory previously provided an NSC measurement calculated using the following formula:

\[
NSC = \text{starch and WSC (simple sugars and fructans)}
\]

However, this laboratory has recently adopted a new formula for calculating NSC:

\[
NSC = \text{starch and ESC (simple sugars).}
\]

This new formula therefore excludes fermentable carbohydrates including fructans.

Fructans Contribute to the Glycemic Response

Coenen et al. reported in the Journal of Nutrition that horses fed an oligofructose (fructan) called inulin exhibited a rise in blood glucose concentrations after feeding. This information should make us re-evaluate some of the classifications described above. Gastric and small intestinal digestion of fermentable carbohydrates such as fructans may contribute to the glycemic response in horses. Hay or concentrations that contain high levels of fructans may therefore challenge the IR horse.

Fructan Digestion and Absorption May Differ Between Individual Ponies

Bailey and Harris presented an abstract at the 2006 American College of Veterinary Internal Medicine Forum that described the effects of dietary inulin on plasma insulin concentrations. Ponies with a history of laminitis exhibited a significantly greater plasma insulin response to inulin compared with unaffected ponies in the control group. These findings suggest that fructan digestion and absorption can differ between individual horses, and this might therefore play a role in IR and laminitis susceptibility.

Excessive Grain Feeding Can Be Associated With Starch Fermentation Within the Large Intestine

As stated above, starches that are not hydrolyzed within the stomach or small intestine are fermented when they reach the large intestine. Overfeeding of grain can therefore increase the amount of starch found within the large intestine, lower intra-luminal pH, and potentially alter the bacterial flora. In a
recent study of client-owned Thoroughbreds in Australia, Richards et al. reported that horses were being fed ~7 kg of grain on average per day, and feeding at this level resulted in starch fermentation within the large intestine.

References and Footnotes

12. Dairy One Forage Laboratory, Ithaca, NY 14850.