

## **Feeding the Atypical Horse**

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### INTRODUCTION

Although the majority of horses can be managed using methods that group them based on age, activity level, or stage of production (i.e., pregnant mares or weanlings), some horses fall outside of the “norm.” For these atypical horses, special consideration must be given to ensure their nutritional needs are adequately met. Underweight senior horses, ailing horses, and postsurgical horses are a few of the animals that may temporarily or perpetually require specialized nutritional management in order to maintain optimal health and productivity.

Emaciation results from numerous etiologies. Refeeding the emaciated horse requires careful attention to potentially life-threatening metabolic disorders. Often just putting condition on an underweight horse (e.g., moving a horse from a condition score of 3 to 4) also presents nutritional and management challenges beyond the capabilities of the average horse owner. A thorough approach to nutritional management of the thin horse will be presented.

Excessive loss of condition can result from the effects of the aging process, malnutrition, neglect, disease, parasites, surgery, environmental stress, adrenal insufficiencies, dental problems, and temperament. In cases of extreme emaciation, a careful and complete veterinary medical examination is critical. Geor (2000) reviewed the metabolic effects of starvation and disease, two conditions that must be treated separately.

### **Starvation**

Fasting and starvation as the result of neglect have vastly different metabolic effects as the same conditions brought about by severe illness. In the fasting or malnourished horse, metabolism slows. The body reduces energy expenditure and directs nutrients to functions necessary for survival. Initially, stored liver glycogen and glucose derived from amino acids are sources of energy. Liver glycogen stores are depleted within 24 to 36 hours of the onset of the fast. The central nervous system and red blood cells require glucose in order to support their essential functions. The body’s effort to maintain blood glucose levels results in gluconeogenesis in the liver.

Glucose is synthesized in the liver from the carbon skeletons of glycerol, lactate, and some amino acids (alanine). The demand for glucose is so critical that amino acids are obtained from muscle tissue catabolism. In an effort to spare blood glucose, an increase in mobilization of fatty acids from adipose tissue occurs, as does an increased dependency on fatty acids for energy for all tissues except for the central nervous system and red blood cells. It is estimated that during a prolonged fast the body may be using fatty acids for 80-85% of its energy needs.

The driving mechanism of the metabolic response to fasting is an altered hormone profile. The most prominent change is decreased insulin production, a condition that goes hand in hand with an increased synthesis of counter-regulatory hormones such as glucagon and cortisol. These hormones promote fatty acid mobilization and the breakdown of muscle protein. In addition, the synthesis of the thyroid hormone triiodothyronine ( $T_3$ ) decreases, resulting in a lowered metabolic rate and energy requirement.

### **Disease and Emaciation**

Severe stress such as disease, injury, major surgery, and sepsis affects metabolic systems much differently than starvation. While the body adapts to starvation by decreasing its metabolic rate, severe stress induces a hypermetabolic state that results in rapid breakdown of the body's reserves of carbohydrate, protein, and fat. Hyperglycemia with insulin resistance, hyperlipidemia, negative nitrogen balance, and diversion of protein (particularly glutamine) from skeletal muscle to the liver are prominent features. Protein catabolism results in severe wasting of lean body mass. Dramatic increases in the "stress hormones," including glucocorticoids, epinephrine, and glucagons along with inflammatory cytokines such as interleukin-1 and tumor necrosis factor, creates a catabolic state that results in malnutrition. These processes lead to delayed tissue repair, immune insufficiency, and declining survivability (Geor, 2000).

#### *Effects of Malnutrition and Hyperlipidemia*

Hyperlipidemia is a disease syndrome in ponies, miniature horses, and more rarely in horses. The disease results in a marked increase in plasma triglycerides (7,000 mg/dl), cloudy serum or plasma, and fatty infiltration of the liver and kidneys. Starved horses not suffering from hyperlipidemia have increased plasma free fatty acids and glycerol. Plasma free fatty acids and glycerol consistently increase more dramatically than triglycerides. Hyperlipidemia is often precipitated in ponies by a primary illness that results in reduced feed intake over a period of days to weeks. Increased metabolic needs due to the illness increase fat mobilization. In addition, pregnancy, lactation, and obesity increase plasma triglycerides. Under conditions of rapid and sustained lipolysis, some of the fat is reesterified in the liver and transported to tissues as very low-density lipoproteins (VLDL). When the liver's capacity for VLDL synthesis is overwhelmed, fat is deposited in the liver. Affected animals require aggressive nutritional support and recovery is difficult.

#### *Low Plasma Albumin*

Long periods of reduced feeding, renal disease, liver disease, and enteropathy can reduce plasma albumin. Regardless of the cause, low plasma albumin should be corrected by refeeding energy and protein. In severe cases (albumin is less than 2 g/dl), blood transfusions may be necessary (Lewis, 1995).

#### *Intestinal Tract*

The gastrointestinal tract (GI) is lined with enterocytes, which have high metabolic rates. These cells also have fairly quick turnover rates, with cell replacement occurring every three days. Enterocytes pre-

vent harmful bacteria from entering the bloodstream, while at the same time providing the mechanism for nutrient absorption. Enterocytes depend on nutrients absorbed from the GI tract for DNA transcription and synthesis of replacement cells. Periods of nutrient deprivation rapidly result in impaired digestion as the result of mucosal atrophy (Geor, 2000). Breakdown of the gut lining increases the risk of bacteria entering the bloodstream, which may set the stage for sepsis.

### *Immune System*

Naylor and Kenyon (1981) report a severely compromised cellular and nonspecific immune function after three to five days of complete feed deprivation in horses. This decrease in immune function increases susceptibility to infection and compromises intestinal mucosal barriers, a fatal combination in horses.

### *Hypothermia*

Anorexic horses are susceptible to hypothermia (Naylor, 1999). These horses do not have insulating fat or produce heat from the process of fiber digestion, nor do they have energy reserves. During rehabilitation, keep these horses warm, dry, and out of the wind. Provide deep bedding and blankets.

## **Nutritional Requirements for Emaciated and/or Sick Horses**

Numerous factors affect the energy needs of compromised horses, and there is limited information available specifically for horses. Ousey et al. (1996) reported the metabolic rate of foals with neonatal maladjustment syndrome (NMS) was 50% of healthy age-matched controls. The authors state that foals with NMS were recumbent and inactive during measurement periods. Even though these foals had very low metabolism, they were in negative energy balance because of low milk intakes. Foals with hypermetabolic stress such as septicemia, diarrhea, and pneumonia will have greater energy deficits.

Data from human and animal studies have demonstrated severe trauma increases energy expenditure by a factor of 1.3 to 1.4, and expenditure in animals with sepsis or a major burn can be up to 1.4 to 1.7 times higher when compared to resting healthy humans (Geor, 2000). Pagan and Hintz (1986) reported that the resting energy expenditure (REE) of horses could be estimated from the formula:  $REE = 21 \text{ kcal (BW kg)} + 975 \text{ kcal}$ . Thus, for a 500-kg horse, REE would be approximately 11.5 Mcal/d, 30% lower than the requirement for maintenance under normal conditions (16.4 Mcal/d for a 500-kg horse). No data are available on energy requirements of sick horses. However, if regression equations for human medicine are applied, a stalled 500-kg horse with an infection or postsurgical condition would have energy requirements of 16 to 20 Mcal/d (1.5 to 1.8 x 11.5 Mcal/d).

Increased protein requirements in sick, debilitated, or injured horses due to protein catabolism should be taken into consideration. Rooney (1998) suggested that 5 g of protein be provided per 100 kcal (i.e., 800 g) of crude protein for a diet containing 16 Mcal digestible energy. This is a 25% increase over the NRC (1989) maintenance protein requirement. There are probably increased requirements for essential vitamins and minerals due to debilitation. However, without knowing specifics, meeting maintenance requirements is a reasonable goal. The main concern is to limit tissue breakdown and weight loss. Realistic goals of providing 60 to 70% of maintenance requirements should help the sick or injured horse.

Recent research in human medicine points to the therapeutic effects of certain nutrients. Arginine, glutamine, omega-3 fatty acids, and ribonucleic acid are reported to upregulate immune function in critically ill humans (Beale et al., 1999). Glutamine is a nonessential amino acid that is important in the growth and repair of the small intestinal mucosa. Glutamine also helps maintain intestinal immune function (Nappert et al., 1997). Routledge et al. (1999) reported plasma glutamine concentrations decrease following viral infection. Further research is needed on glutamine and the immune system of horses. Omega-3 fatty acids have also received a great deal of attention because of their effects on the immune system.

Determining body condition scores and evaluating horses for potential disease or injury are important in identifying those horses in need of nutritional support (Doneghue, 1992). Well-fed adult horses that are not pregnant (last trimester) or lactating can withstand up to four days of partial or complete starvation without long-term effects. However, thin horses with condition scores between 1 to 3 that have experienced a dramatic loss in weight (10% or more) need emergency nutritional support. Rapid weight loss is commonly observed in horses with sepsis, endotoxemia, pulmonary abscess, pleuropneumonia, abdominal abscesses, diarrhea, severe trauma, surgery, or intestinal disorders. Overweight horses, those with condition scores of 7 to 9, quickly develop hypertriglyceridemia (7,500 mg/dl) after even short periods of anorexia.

Foals, particularly during the first weeks of life, have limited energy stores. Conditions compromising nutritional support of foals can quickly result in hypoglycemia, weakness, and death.

## **Disease and Nutrition**

Certain diseases result in weight loss and require specific nutritional adjustments. These include respiratory disease, laminitis, Cushing's syndrome, renal failure, hepatic disease, chronic diarrhea, and small intestinal malabsorption syndromes. Resection of the GI tract requires special feeding regimes. For performance horses that have viral or bacterial infections, minor injuries, or orthopedic surgery, the primary nutritional goal is often energy reduction. While on stall rest these horses should be fed a diet of hay at a rate of 1.5-2.0% of body weight, a vitamin/mineral supplement, and salt.

### *Respiratory Disease*

Chronic obstructive pulmonary disease (COPD), or heaves, in horses is caused by hypersensitivity to fungal spores present in hay or bedding, dust, molds, and occasionally grass pollen. The hypersensitivity results in bronchoconstriction, excess mucus, and inflammatory thickening of the alveoli. Often there is a prior infectious respiratory disease that increases the likelihood of the condition. COPD usually subsides when the allergen is identified and eliminated in the early stages.

Housing in barns usually makes the condition worse. Maintaining horses at pasture is usually the best management system. If horses have to be stabled, ventilation and dust-free bedding must be used. Hay is the primary source of allergens. If hay is included in the diet, it must be soaked by submersion in

water for at least five minutes. Hay cubes and complete diets are good alternatives to hay. If hay cubes are used, they too must be soaked prior to feeding.

### *Hepatic Disease*

Liver dysfunction causes the plasma concentration of branched-chain amino acids (leucine, isoleucine, and valine) to decrease and aromatic amino acids (phenylalanine, tyrosine, and tryptophan) as well as ammonia to increase. These changes may be responsible for hepatoencephalopathy. Feeding diets that meet energy needs without excess protein helps minimize neurologic signs. Feeding adequate energy reduces mobilization of body glycogen, fat, and protein. Excess fat is deposited in the liver and further limits hepatic function. Readily available soluble carbohydrates are essential to the diet. In addition, the protein source should have a high branched-chain to aromatic amino acid ratio. A diet consisting of 50% steam-flaked corn or milo and 50% grass hay should be fed. Molasses can be added to provide glucose. Multiple feedings will help maintain glucose homeostasis and prevent bacterial ammonia surges. Legume hays, oats, and soybeans should be avoided because of high levels of aromatic amino acids.

### *Renal Failure*

Chronic renal failure occurs most frequently in older horses. Depression and anorexia are signs of chronic renal failure. Polyuria and polydipsia are due to an inability to concentrate urine. Renal failure in horses is also associated with hypercalcemia and elevated urea. Phosphorus excretion can also be impaired and sodium deficits might develop.

Feedstuffs high in protein (legumes, soybeans), phosphorus (wheat bran), and calcium (legumes, calcium-containing supplements) should be avoided. In some cases, hypoproteinemia can develop with chronic renal failure. In these cases, the level of protein should be increased. Horses with hypoproteinemia benefit from fat supplementation.

### *Gastrointestinal Disorders*

Difficult or painful swallowing can reduce intake in horses. Some of the causes of difficult swallowing include obstruction from abscesses or strangles; nerve damage from equine protozoal myeloencephalitis (EPM) and muscle weakness caused by hyperkalemic periodic paralysis (HYPP); or botulism. Scar tissue from an episode of choking can cause difficulty swallowing. If the esophagus is narrowed due to scarring from choking, a hay cube and grain slurry may need to be fed.

Gastric ulcers can cause a reduction in appetite. The incidence of ulcers in horses is high. Surveys put the incidence for racehorses near 90%. In stalled show horses, the occurrence approaches 65%. Horses maintained on pasture rarely have ulcers, but when they are confined to stalls and fed grain meals, ulcers can develop within four days. Damage to the nonglandular portion of the stomach by hydrochloric acid produced in the glandular region causes painful lesions that can reduce intake. Signs associated with gastric ulcers are irritability, chronic colic, diarrhea, weight loss, and dull hair coat.

Gastrointestinal disorders can be further divided into those associated with the small intestine including enteropathies, malabsorption syndromes, and small intestine resection, and those involving the large intestine such as colitis, diarrhea, chronic impaction, and colon resection. With small intestinal disease, the primary goal is to optimize the large bowel's digestive function. This is achieved by feeding highly digestible fiber sources such as leafy alfalfa, beet pulp, or soybean hulls while reducing grain. Feeding small meals of highly fermentable fibers (beet pulp and alfalfa), fat (rice bran), and vegetable oil has maintained body condition in horses following resection of 50% of the small intestine. Complete pelleted feeds have been fed in small amounts with positive results even after 70% of distal small intestine resection (Lewis, 1995).

Large intestinal dysfunction usually results in diarrhea. In the acute phase, many affected horses are hypophagic and need enteral nutritional support. Small intestinal function is maintained with large colon resection. Diets that are low in fiber and high in digestible energy and protein should be fed. As the horse's appetite improves, fermentable fiber should be added. Probiotics and yeast are recommended to help reestablish the gut microflora. Vitamin K and B vitamins should be supplemented because of decreased production in the hindgut. Nutritional support can be provided by parenteral (intravenous) or enteral feeding. Voluntary feeding is dependent on the condition of the horse and its appetite. Appetite stimulation is often accomplished by "cafeteria style" feeding. Lush, green grass is often the most appealing feed for horses. However, mashes, leafy alfalfa, grains, sweet feeds, and fresh fruit may also appeal to a horse when trying to encourage feed consumption.

There are a variety of enteral diets that have been developed for feeding via nasogastric tube. The Naylor diet is one of the most widely accepted (Naylor, 1977). The Naylor diet is composed of 454 g alfalfa meal, 204 g casein, 204 g dextrose, 52 g electrolyte mixture, and 5 L of water. The digestible energy (DE) is 2.77 Mcal for this mixture, and this should be fed via a nasal tube in six batches to meet the maintenance energy needs of a 500-kg adult horse (16.4 Mcal).

Once horses are eating, a simple refeeding program for starved horses has been reported by Stull (2003). The researchers at UC Davis (Stull, 2003) recommended the following program:

- Days 1-3: Feed one pound leafy alfalfa every four hours (total of 6 lb/d in 6 feedings).
- Days 4-10: Slowly increase the amount of alfalfa and decrease the number of feedings so that by day six you are feeding just over four pounds of hay every eight hours (total of 13 lb/d in three feedings).
- Days 10-several months: Feed as much alfalfa as the horse will eat and decrease feeding to twice per day. Provide access to a salt block. Do not feed grain or supplemental material until the horse returns to normal.

When rehabilitating a thin horse, sudden change from a poor-quality diet to a highly digestible diet can cause death within three to five days. Laminitis and diarrhea are common problems, but the most serious concern is when phosphorus, potassium, or magnesium are depleted and the depletion is made worse by sequestration of these minerals in newly formed cells (Whitam and Stull, 1998).

## Senior Horses

Senior horses can be a challenge. After the diseases discussed above are ruled out, a checklist of management priorities for senior horses must be reviewed. More often than not, an older horse in poor condition has a dental problem. A thorough dental exam and correction of sharp teeth, wavy mouth, and/or gum or tooth infection may allow older horses to more properly grind feeds and improve nutrient digestion and absorption. If the teeth are worn and the horse is unable to chew well, pelleted or extruded feeds fed in a slurry will help improve the condition of the horse.

### *Environmental Effects*

Herd dynamics and pecking order change as horses age and can result in older horses not receiving adequate nutrition in group-feeding situations. Older horses often do better when separated from the group at feeding time. Underweight horses are very susceptible to hypothermia. Table 1 illustrates the increased energy requirements of horses in inclement weather versus the normal horse.

*Table 1. Effects of wind and rain on digestible energy requirements for horses at maintenance.\**

Average Temperature	Wind/Rain	Additional Mcal/day	Additional Hay
32° F	10-15 mph wind	4-8 Mcal/day	4-8 lb/day
32° F	Rain	6 Mcal/day	6 lb/day
32° F	Rain and wind	10-14 Mcal/day**	10-14 lb/day

*\*Adapted from Anderson, 2003.*

*\*\*May not be able to consume enough hay to meet requirements.*

Internal parasites can cause damage to the lining of the GI tract and reduce the efficiency of digestion. Reducing parasite loads in older neglected horses should be done under veterinary supervision to avoid a rapid kill-off of parasites, which may cause impaction.

Chronic pain as a result of arthritis can interfere with grazing and affect an older horse's appetite. Depending on the seriousness of joint pain, chondroitin sulfates and glucosamine may be helpful. In more serious cases, nonsteroidal anti-inflammatory drugs and/or injectable cartilage-building drugs may be necessary.

### *Senior Nutrition*

A number of feed companies now offer feeds for aged horses that are supplemented with water-soluble vitamins and contain 12-16% protein, <1.0% calcium, and 0.45-0.6% phosphorus (Ralston, 1999). Also, these feeds usually contain at least 12% crude fiber. These feeds are typically extruded thereby increasing digestibility for the older horse. Alternative sources of forage such as hay cubes can be used if

the horse has a dental problem that may hinder proper chewing of long-stem hay. The hay cubes should be a mixture of grass and alfalfa, rather than straight alfalfa due to the high calcium content of alfalfa.

### Conclusion

Managing the atypical horse requires an individualized approach to housing, training, and nutrition. Whether horses are unusual due to illness, neglect, temperament, age, or a combination of these factors, they can be productive and healthy once they are properly diagnosed and their particular requirements are met.

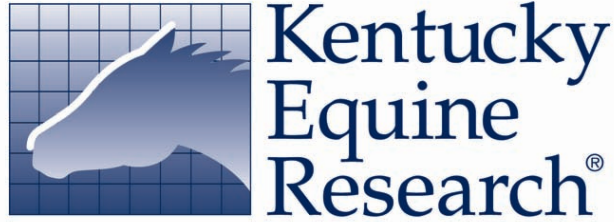
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