Chapter 3

Equine nutrition and metabolic diseases

P. A. Harris (Consultant Editor), M. Coenen, D. Frape, L. B. Jeffcott and H. Meyer

Chapter contents

INTRODUCTION 152
GUIDE TO TYPICAL FEEDSTUFFS 153
Cereals, sugar beet and oil seeds as feedstuffs 154
Major cereals fed to horses 154
Oil seeds 156
Vegetable oils 156
Forages and other fiber sources 158
Hay 158
Soaking hay before feeding 159
Semi-wilted forages 159
Silage 159
Straw 160
Hydroponics 160
Soya hulls and sugar beet pulp 161
Methods of feed processing 161
Feed storage 162
Feedstuffs analysis 162
THE DIGESTIVE TRACT 164
Physiology and pathology of digestion 164
Mastication 164
Stomach and small intestine 166
Large intestine 167
Water and electrolytes 168
The importance of microbial fermentation in the gut 168
Normal fermentation in meal-fed horses 169
Dysfermentation 169
The importance of fiber 170
Gastric ulceration 170
NUTRIENT REQUIREMENTS 171
Introduction 171
Body weight estimations 173
Water 173
Sweat production 174
Energy 175
Energy requirements 176
Energy deficiency and excess 177
Protein 177
Protein sources 178
Dietary protein requirements 178
Protein deficiency 178
Amino acid balance 178
Protein excess 179
Minerals and trace elements 179
Utilization of dietary sources 180
Interactions 180
Mineral requirements 180
Calcium, phosphorus and magnesium 180
Sodium, potassium and chloride 183
Possible clinical signs associated with deficiencies 183
Possible clinical signs associated with excess 183
Micro-(trace-) mineral requirements 183
Vitamins 185
Introduction 185
Vitamin requirements 185
Fat-soluble vitamins: sources and possible clinical signs of deficiency and excess 186
Water-soluble vitamins: sources and possible clinical signs of deficiency and excess 188
PRACTICAL NUTRITION AND PRACTICAL RATION FORMULATION 190
General aspects 190
Appetite 190
Number of meals 190
When to feed and water 191
Forage: concentrate ratios 191
Pasture 192
Concentrate feed design and selection 192
Compound nuts, coarse mixes and extruded feeds 193
“Straight” rations 193
Mineral and vitamin supplements 193
Specific micronutrient supplements 193
Items to avoid 194
FEEDING THE PERFORMANCE HORSE 194
Energy 194
Protein 195
Water and electrolytes 196
Trace elements 197
Vitamins 197
Fat-soluble vitamins 197
Water-soluble vitamins 198
Antioxidant supplementation 198
FEEDING THE PREGNANT/LACTATING MARE 199
Introduction 199
Energy requirements 201
Protein requirements 201
Calcium and phosphorus requirements 202
Water requirements 203
Nutrition can be defined as the process of feeding and the subsequent digestion and assimilation of food. It is, therefore, of fundamental importance to the well-being of any animal. However, good nutrition will only help a horse to be able to perform optimally; it cannot improve the intrinsic ability of the animal (nor the horse and rider combination). Poor nutrition, on the other hand, may impose limits on an animal’s ability to perform. In most instances it is only when an adult horse is asked to exert itself that the result of imbalanced or inadequate nutrition becomes apparent.

Many of the nutrients we now take for granted were only recognized a relatively short time ago. Although in the early nineteenth century it was appreciated that calcium and phosphorus were needed for “hard bone”, vitamin B₁₂, for example, was not seen as an essential nutrient until the 1940s and selenium not until the 1950s. The original research into the feeding of horses came mainly from army veterinarians and others involved with cavalry, working and pack horses. Mechanization and the replacement of the horse as a means of transport and a power source resulted in a decrease in equine research. The first National Research Council (NRC) report on equine nutrition was published in 1949. The second, in 1961, stated that little new information had been obtained since 1949 and contained many estimates based on extrapolations from ruminants. The next in 1966 was similar. The last one, carried out in 1989, is well overdue for revision. Since then increasing numbers of equine nutrition studies have been carried out, as the popularity of the horse for recreational purposes has increased.

However, many of the equine nutritional practices currently employed have not changed significantly from those followed hundreds of years ago,
although the nature and composition of the basic feedstuffs has changed in some instances. The most significant change in the twentieth century was probably the introduction of pelleted feeds around 1920. These became popular in the 1960s when competition, increased knowledge, more ethical companies and government regulations resulted in the evolution of good quality, commercial, compound feeds.

Unfortunately, in many areas of equine nutrition good scientific information is still not available. Confusion and controversy often exist, especially where one research paper’s findings directly contradict those of another. There are many reasons for this confusion. Much of the research has been carried out in ponies and then applied to the horse but such extrapolations may not always be applicable. In other instances, the data available have been extrapolated from another species such as a ruminant. Largely for financial reasons, most of the nutritional research in horses has been carried out on relatively few animals and has concentrated on the effects of short-term alterations in intake.

The adaptive changes that occur over longer periods are therefore not well understood. Differing experimental protocols, basal diets, and exercise regimens may all contribute to the conflicting results. In addition, the normal daily nutrient requirements vary according to several factors, depending on the nutrient involved, and include age, body weight, exercise and environmental conditions. The availability of a particular nutrient may vary not only with the nutrient, its nature in that feedstuff (e.g. of organic or inorganic source) and the presence of other nutrients, but also with the individual animal’s absorptive ability. Nutrition cannot be considered to be an exact science. Considerably more information on the dietary requirements and digestive physiology of domestic ruminants is available than for the horse, however, and more basic and applied scientific research is needed into most aspects of equine nutrition.

Optimal feeding of horses uses both art and science. The science provides the information about the digestive and metabolic processes, the nutrient requirements and the principles behind feeding practices. The art is the ability to convert this theory into practice for the individual horse, its needs, likes and dislikes. In this chapter, a general and, it is hoped, practical guide to equine nutrition is given. Wherever possible, recommendations are based on sound research but interpreted practically. It aims to provide a guideline to follow and to highlight some of the reasons behind the basic rules of feeding. Although, wherever possible, discrepancies between various authors’ recommendations have been removed, some remain because in a number of instances “correct” values are simply not known and the best that exists at the present time is a range of acceptable levels.

GUIDE TO TYPICAL FEEDSTUFFS

Domestication and our increasing demand for horses to perform repeatedly have resulted in energy requirements that, for some horses, are above those able to be provided by their more “natural” diet of fresh forage. Cereals provide more net energy than hay, which in turn provides more than twice the...
net or usable energy of straw. However, the upper part of the gastrointestinal tract (GIT) has a relatively small capacity and the horse has digestive and metabolic limitations to high grain, starch and sugar based diets. Large grain meals may overwhelm the digestive capacity of the stomach and small intestine leading to the rapid fermentation of the grain carbohydrate in the hindgut. This potentially can result in one of a number of disorders including colic, diarrhea and laminitis.

Therefore, there has been increasing interest in the use of alternative energy sources for horses, especially alternative fiber sources, which do not cause such marked disturbances in the hindgut and yet provide more energy than typical forages. In addition, because vegetable oils provide proportionally more net energy than the cereals, yet contain no starch or sugar and may provide other advantages, there is an increasing use of supplementary vegetable oils.

Table 3.1 provides figures for the typical composition of some common feedstuffs. Individual samples may vary within a range and, for complete accuracy when evaluating a dietary regimen, individual analyses must be undertaken. Pasture and hay analyses vary according to season, soil type, geographic location and other variables such as harvest date. For accuracy, a number of actual representative forage (fresh or preserved) samples must be analyzed when reliable results are required.

The importance of calculating the actual elemental levels of the required mineral from the selected source cannot be overemphasized. The bioavailability of certain minerals will be affected by many factors such as the levels of any antagonistic minerals present. In addition, there will also be variation in utilization between individual horses.

When assessing the nutrient value of particular feedstuffs, it must be remembered that modern methods of feed production have had two major effects on the composition and feed value of equine rations. First, they have allowed a wider range of ingredients to be used as modern production methods destroy many of the harmful substances that might prevent the feedstuff being used in its raw state. Second, they have affected the nutrient availability and digestibility of certain traditional and recently introduced feedstuffs. Moreover, there are several methods of preparation or treatment of feedstuffs that have differing effects on palatability, digestibility and stability in storage.

CEREALS, SUGAR BEET AND OIL SEEDS AS FEEDSTUFFS

Major cereals fed to horses

Oats are the traditional cereal fed to horses in work. They contain significantly higher fiber and lower starch levels than most other cereals and the nature of their starch particles helps to promote a high pre-cecal starch digestibility in contrast, for example, to corn and barley. As with all cereals and cereal by-products, oats provide a low level of calcium and a moderate level of phosphorus (which may be bound in phytate compounds, reducing its availability), giving a reversed calcium to phosphorus ratio (q.v.). Also, in common with most cereals, the level of the essential amino acid lysine is
# Chapter 3

Equine nutrition and metabolic diseases

## Table 3.1  Guide to the typical composition of common feedstuffs (assuming 880 g DM/kg), in part derived from the NRC\(^1\)

<table>
<thead>
<tr>
<th>Feedstuff</th>
<th>Crude protein (g/kg)</th>
<th>Crude fiber (g/kg)</th>
<th>Ca (g/kg)</th>
<th>P (g/kg)</th>
<th>DE (MJ/kg)</th>
<th>Mg (g/kg)</th>
<th>Lysine (g/kg)</th>
<th>Starch (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dried milk</td>
<td>340</td>
<td>0</td>
<td>10.5</td>
<td>9.8</td>
<td>15.1</td>
<td>1.2</td>
<td>29.0</td>
<td>—</td>
</tr>
<tr>
<td>Oats</td>
<td>96</td>
<td>100</td>
<td>0.7</td>
<td>3.0</td>
<td>10.9–12.1</td>
<td>1.4</td>
<td>3.2</td>
<td>385</td>
</tr>
<tr>
<td>Barley</td>
<td>95</td>
<td>50</td>
<td>0.7</td>
<td>3.3</td>
<td>12.8</td>
<td>1.3</td>
<td>3.1</td>
<td>515</td>
</tr>
<tr>
<td>Extruded soya bean meal</td>
<td>440</td>
<td>62</td>
<td>2.4</td>
<td>6.3</td>
<td>13.3</td>
<td>2.7</td>
<td>26.0</td>
<td>50</td>
</tr>
<tr>
<td>Field beans</td>
<td>255</td>
<td>74</td>
<td>0.8</td>
<td>4.8</td>
<td>13.1</td>
<td>1.5</td>
<td>17.0</td>
<td>360</td>
</tr>
<tr>
<td>High protein grass meal</td>
<td>160</td>
<td>220</td>
<td>6.0</td>
<td>2.3</td>
<td>9.6</td>
<td>2.7</td>
<td>8.0</td>
<td>15</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>155</td>
<td>110</td>
<td>1.0</td>
<td>12.0</td>
<td>10.8</td>
<td>5.6</td>
<td>6.0</td>
<td>165</td>
</tr>
<tr>
<td>Sugar beet pulp</td>
<td>70</td>
<td>174</td>
<td>10.0</td>
<td>11.0</td>
<td>10.5</td>
<td>2.5</td>
<td>4.5</td>
<td>10</td>
</tr>
<tr>
<td>Cane molasses</td>
<td>30</td>
<td>0</td>
<td>7.2</td>
<td>1.0</td>
<td>11.4</td>
<td>4.0</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Limestone flour</td>
<td>—</td>
<td>0</td>
<td>365</td>
<td>0</td>
<td>0</td>
<td>20.6</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>—</td>
<td>0</td>
<td>238</td>
<td>187</td>
<td>0</td>
<td>5.9</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Steamed bone flour</td>
<td>—</td>
<td>0</td>
<td>323</td>
<td>133</td>
<td>0</td>
<td>3.3</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Grass hay</td>
<td>45–90</td>
<td>330</td>
<td>2.9</td>
<td>1.7</td>
<td>7.6</td>
<td>1.1</td>
<td>2–4</td>
<td>V</td>
</tr>
<tr>
<td>Grass/clover hay</td>
<td>60–110</td>
<td>330</td>
<td>4.0</td>
<td>1.7</td>
<td>7.8</td>
<td>3.0</td>
<td>3–5</td>
<td>—</td>
</tr>
<tr>
<td>Full fat soya beans</td>
<td>360</td>
<td>44</td>
<td>1.7</td>
<td>4.7</td>
<td>15.5</td>
<td>2.9</td>
<td>24</td>
<td>45</td>
</tr>
<tr>
<td>Linseeds</td>
<td>320</td>
<td>90</td>
<td>4</td>
<td>7.4</td>
<td>14.6</td>
<td>3.7</td>
<td>12.9</td>
<td>36</td>
</tr>
<tr>
<td>Flaked maize</td>
<td>80</td>
<td>15</td>
<td>0.2</td>
<td>2.9</td>
<td>15.2</td>
<td>1.0</td>
<td>2.6</td>
<td>610</td>
</tr>
<tr>
<td>English alfalfa</td>
<td>180</td>
<td>260</td>
<td>12</td>
<td>3.8</td>
<td>11.5</td>
<td>2.6</td>
<td>7.3</td>
<td>30</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Feedstuff</th>
<th>Oil (g/kg)</th>
<th>Cu (mg/kg)</th>
<th>Zn (mg/kg)</th>
<th>Mn (mg/kg)</th>
<th>Fe (mg/kg)</th>
<th>Na (g/kg)</th>
<th>K (g/kg)</th>
<th>Cl (g/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dried milk</td>
<td>10</td>
<td>10</td>
<td>41</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>Oats</td>
<td>45</td>
<td>4</td>
<td>35</td>
<td>38</td>
<td>65</td>
<td>0.2</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Barley</td>
<td>17</td>
<td>4</td>
<td>15</td>
<td>16</td>
<td>16</td>
<td>7.3</td>
<td>0.1</td>
<td>4</td>
</tr>
<tr>
<td>Extruded soya bean meal</td>
<td>18</td>
<td>13</td>
<td>50</td>
<td>31</td>
<td>160</td>
<td>0.2</td>
<td>17</td>
<td>0.1</td>
</tr>
<tr>
<td>Field beans</td>
<td>1.5</td>
<td>9</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>0.1</td>
<td>12</td>
<td>0.7</td>
</tr>
<tr>
<td>High protein grass meal</td>
<td>30</td>
<td>7</td>
<td>16</td>
<td>30</td>
<td>200</td>
<td>1</td>
<td>22.5</td>
<td>0.8</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>35</td>
<td>12</td>
<td>98</td>
<td>120</td>
<td>145</td>
<td>0.1</td>
<td>4</td>
<td>0.9</td>
</tr>
<tr>
<td>Sugar beet pulp</td>
<td>5</td>
<td>10</td>
<td>1</td>
<td>34</td>
<td>250</td>
<td>3</td>
<td>17.5</td>
<td>2.2</td>
</tr>
<tr>
<td>Cane molasses</td>
<td>—</td>
<td>20</td>
<td>15</td>
<td>44</td>
<td>200</td>
<td>2</td>
<td>26</td>
<td>25</td>
</tr>
<tr>
<td>Limestone flour</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Steamed bone flour</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Grass hay</td>
<td>21</td>
<td>3</td>
<td>12</td>
<td>20</td>
<td>150</td>
<td>—</td>
<td>10–35</td>
<td>—</td>
</tr>
<tr>
<td>Grass/clover hay</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Full fat soya beans</td>
<td>180</td>
<td>12</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>0.1</td>
<td>16</td>
<td>0.4</td>
</tr>
<tr>
<td>Linseeds</td>
<td>340</td>
<td>7</td>
<td>N/A</td>
<td>60</td>
<td>90</td>
<td>0.6</td>
<td>6.8</td>
<td>0.2</td>
</tr>
<tr>
<td>Flaked maize</td>
<td>36</td>
<td>2</td>
<td>19</td>
<td>5</td>
<td>31</td>
<td>0.1</td>
<td>3</td>
<td>0.4</td>
</tr>
<tr>
<td>English alfalfa</td>
<td>25</td>
<td>12</td>
<td>28</td>
<td>25</td>
<td>250</td>
<td>1</td>
<td>21</td>
<td>5</td>
</tr>
</tbody>
</table>

relatively low. There are now varieties of oat produced without husks, colloquially often called *naked oats*. They have a better amino acid profile, higher oil levels, and thus considerably higher energy levels than the husked varieties.

**Barley** has a higher energy level than oats and is generally fed rolled or cooked (cooking increases pre-cecal starch digestibility; *q.v.*). However, the amino acid profile, low calcium level, and phytate-bound phosphorus need correcting when barley is the major ingredient in horse rations.

**Maize** tends to have the lowest crude protein (CP) of the common cereals fed to horses and the highest energy value. In the UK it is generally micronized or steam flaked before feeding but in the USA and mainland Europe it is often simply cracked to open the outer husk.

**Wheat** has traditionally not been fed to horses. However, with the introduction of efficient cooking methods, the resultant alteration in the structure of the starch has made it a useful high energy feed for inclusion in commercial coarse mixes and home-mixed cereal rations. Large amounts per meal should be avoided, however, as they may lead to a sticky consistency in the stomach which may favor *dysfermentation* (*q.v.*).

**Triticale** is a hybrid resulting from crossing wheat and rye. In a ground form it should have a feeding value slightly greater than that of barley and evidence indicates that triticale may be better digested by the horse than is wheat. Both rye and triticale are subject to *ergot* infestation (*q.v.*), so clean samples should be sought.

Regardless of how triticale, wheat, barley or maize are mechanically processed, the starch will be less well digested in the small intestine than when cooked grains are fed.

**Oil seeds**

The *soya bean* is an excellent source of protein in an equine diet, providing good levels of essential amino acids. It must be properly cooked before feeding to help destroy the protease inhibitors as well as potential allergenic, goitrogenic and anticoagulant factors. However, in practice, most soya available in retail outlets has been suitably treated.

Soya is available as an *expelled meal*, with the oil removed; as the *full fat meal*, which has added advantages of providing good levels of essential fatty acids; and as the *full fat flake*, which has usually been micronized. This latter form is the most common to add as a top dressing, as the physical consistency of the meals can be unpalatable to some horses.

**Linseed** must also be cooked before feeding to destroy the glycoside, which, after soaking, could potentially release *hydrocyanic acid*. Linseed also has a good amino acid profile and in some methods of processing, where the oil is not extracted, also provides good levels of certain key fatty acids.

**Vegetable oils**

Horses have been shown to be able to digest and utilize high amounts of oil, but for practical purposes approximately 0.75–1 g of oil/kg BW/day may be considered to be the upper limit. Levels of 5–8% in the total diet are more
common in some high performing horses and many performance horses can be fed up to 100 mL/100 kg BW daily in divided doses without any problems provided that it has been introduced gradually, is not rancid, the horse requires such an energy intake, additional vitamin E is provided and the overall diet is re-balanced.

Adding oil to existing feed has the potential to create multiple imbalances (including an inadequate vitamin E intake and a calcium imbalance) and therefore could be considered less safe than feeding a diet where the oil has been balanced in relation to all of the essential nutrients in the feed. Any supplemental oil or oil-supplemented feed should be introduced slowly.

Supplemental fat or oil diets can be supplied in four main ways:

1. As an oil supplemented, manufactured diet. The advantage here is that such diets should be balanced with respect to the protein, vitamin and mineral intake that they provide when fed with forage (and salt as required). This can be a simple, practical and convenient way to feed high oil diets.

2. High oil supplemental feedstuffs, such as rice bran, which are also high in fiber and usually low in starch. However, many of the rice brans available have the same disadvantages as wheat bran in that they have a very imbalanced calcium-phosphorus content.

3. Supplemental animal fat. Many horses find most animal fats to be unpalatable and these fats seem often to be more likely to cause digestive upsets. Their use is not to be recommended.

4. Supplemental vegetable oils. The exact type of oil that may be preferred will depend on the individual horse and the nature of the processing to which the oil has been subjected. Corn oil and soy oil are probably the most commonly used vegetable oils in Europe.

It has been suggested that feeding oil supplemented diets, with appropriate training, can result in a range of effects on a variety of physiologic, metabolic parameters as well as on performance. These include:

1. Increased mobilization of free fatty acids (FFA) and increased speed of mobilization; increased speed of uptake of FFA into muscle—often considered to be rate limiting.

2. A glycogen sparing effect so that fatigue is delayed and performance improved (this could be especially important in endurance activities).

3. Increased high intensity exercise capacity.

4. In horses with a high energy demand, helping to reduce feed volume so that the roughage intake can be maintained (oils have also been suggested to help reduce the extent of bacterial dysfermentations in the stomach and small intestine).

5. Behavioral advantages over high cereal starch diets.

In order to have the potential to obtain metabolic benefits from the feeding of oil or oil supplemented diets, in addition to those associated with its high energy density and lack of starch content, the oil needs to be fed for several months.
Linoleic acid and alpha-linolenic acid are considered dietary essential fatty acids. However, no evidence of deficiency has been described for the horse and it must be assumed that a normal diet of cereal grains and natural forage, or of pasture herbage, will provide the dietary requirements.

FORAGES AND OTHER FIBER SOURCES

The choice of fiber source is now much wider with the advent of new methods of grass preservation, improved grass and other forage plant species as well as other technological advances. There are two equally important aspects when selecting the most appropriate fiber source:

1. The nutrient levels in the selected forage, and thus the percentage of the horse’s total daily nutrient requirements that will be met through the fiber source.

2. The effect that forage may have on other aspects of the horse’s health, especially the respiratory system.

Hay

Hay is the most commonly used long fiber source and may be divided into several types.

Meadow hay is generally made from permanent pasture and has a great diversity of species, including several different grasses, herbs and other plants. This hay is often termed “soft”, as the diversity of plants results in different rates of growth and stages of maturity at the time of cutting.

Meadow hay will, on average, also provide higher protein (typically 8–12% dry matter [DM]) and digestible energy levels (typically 9–11 MJ/kg DM) than most seed hays. Meadow hays also often have higher mineral levels than seed hay, as they contain a greater proportion of deeper rooting herbaceous species.

Seed hay is produced from specially seeded leys, usually 1–3 yr old, and contains predominantly one or two grass species (often rye grass or timothy). This hay is more uniform and, due to the growth rate and cutting time, is usually fairly mature, with a lower proportion of leafy material and higher proportion of stalk than meadow hay. Typically, seed hay has lower energy (for example approximately 8–10 MJ/kg DM) and protein levels (approximately 4–8% DM) and a lower digestibility than meadow hay. The mineral levels in seed hay also tend to be lower than for meadow hay, although this will depend on the soil upon which the hay was grown.

Legume hay (usually alfalfa or lucerne, clover or sainfoin) has, due to nitrogen-fixing properties, higher protein levels than seed or meadow hay. Legume hay may be grown as a pure stand, but can be difficult to dry in cool wet climates as the stalks are very moist and thick so that cutting is often left until later in the season, when temperatures increase. However, by then the hay may have become very mature and stalky, with low energy and digestibility values. An exception to this is alfalfa, which can be cut earlier, barn dried and packaged in a short chopped form in plastic-covered bales. Clover and sainfoin are commonly mixed with grass species such as timothy to make
conventional hay, but care must be taken with the leys, as competition between the species will alter the relative proportions over a number of years.

Barn-dried hay is wilted in the field for 2–3 days when weather conditions are good and loose packed into special buildings; air of a particular temperature and humidity is then blown through the stored grass for 7–10 days before baling. When correctly practiced, this results in hay with a high DM, thus limiting the development of fungal spores.

Sometimes hay is baled with too high a moisture content due to unsuitable weather conditions at cutting time, insufficient turning in the rows, or other factors. A high moisture content will allow the development of large quantities of fungal spores (q.v.), which can be inhaled by the horse. Hay with a DM ≤87% should always be assessed for spore level.

Soaking hay before feeding

The practice of soaking hay with the aim of reducing the number of airborne particles is popular in some areas. It has been reported that soaking a 2.5 kg hay net for 30 min reduces respirable particle numbers by approximately 90%. The water used for soaking must be fresh and increasingly it is recommended that soaking for >30 min may not be advisable due to the potentially negative effect of prolonged soaking on the soluble carbohydrate and nitrogenous content of hay and the pollutant nature of the effluent. Alternative solutions include the practice of steaming hay or the use of semi-wilted forages, silages or vacuum packed dust-free fodder.

Semi-wilted forages

In an attempt to reduce the level of fungal spores in forage for horses, methods of preserving grass have diversified. “Dust-free” grass and other forage plants such as alfalfa are wilted to approximately 50–60% DM and packaged in semi-permeable plastic, where a mild lactic fermentation occurs owing to the limited amounts of oxygen present. This lactic fermentation stabilizes at approximately pH 4.5–5.5, depending upon the amount of oxygen and substrate available for the microbial fermentation and helps to inhibit the proliferation of fungal spores. (Water activities <0.985 combined with a low pH may help to control in particular the sporulation of Clostridium botulinum, q.v.).

These forages exert a lower challenge to the horse’s respiratory system and may be a valuable way to provide long fiber to a horse suffering from allergic respiratory disease (q.v.).

Damage to packaging allows the influx of oxygen, permitting further microbial activity to occur. This can be identified as a patch of mold, which is limited by the extent of the oxygen diffusion through the hole in the packaging. There may also be an increase in temperature as a result of the microbial activity. Bags in which this has occurred should be discarded.

Silage

Some larger horse keepers now find it economical to use silage. The rapid pH drop, the greater water activity, the decrease in available oxygen and soluble
carbohydrates as fermentation progresses all can help inhibit the development of fungal spores. There are, however, several important factors to be considered before using silage.

First, the DM content is very important to help inhibit clostridial activity. Horses also find very low DM material unpalatable, therefore it is recommended that silage for horses should have a DM >35% and preferably >40%.

Second, pH should ideally be <6.0 in order to inhibit undesirable microbial activity. At pH values between 5.0 and 6.0, the DM should exceed 40% as an added inhibitory factor to clostridial activity. Horses may find very low pH material unpalatable, and silage with a pH <4.5 may be rejected by some horses. There are also anecdotal reports that low pH silage may be extremely unsuitable for donkeys.

Third, it should be appreciated that if the silage was made to meet the nutrient requirements of dairy cattle the nutrient levels may be unsuitable for horses: for example, lactating dairy cattle require a higher daily protein intake than most horses. Finally, once a “big bale” of silage has been opened it should be used within 2–3 days in order to prevent secondary microbial activity from occurring.

Where large amounts of silage or haylage are used, supplementary vitamins D and E are necessary as ensiling destroys vitamin E and vitamin D₂ is not synthesized during the ensiling process. It is always advisable to get professional help when considering producing silage for horse feeding.

Straw

Straw provides a low nutrient level forage, which may be used to provide a portion of the daily forage intake for some horses, although problems with spores and dust must be taken into consideration. In addition, due to the high silica and indigestible fiber content there is a risk of impactions (q.v.) especially in Thoroughbreds and Thoroughbred crosses.

Chemical treatment of straw with sodium hydroxide and ammonia may increase nutrient value but this requires specialized equipment and expertise and the practice has not gained widespread popularity. Straw may be sprayed with molasses to increase its palatability but the nutrient values are still very low. There have been some concerns expressed regarding the various chemicals that may be used in grain production (e.g. to restrict the growth in height and to prolong the vegetative stage). It has been thought, for example, that they may leave unwanted residues on the straw; however, currently there is no evidence to support this concern.

Significant intakes of straw should also be avoided in young animals, where the hindgut microflora may not be fully established and the highly indigestible straw may lead to impactions.

Hydroponics

A hydroponic culture is the practice of germinating seeds (usually barley) in water-filled trays in a humidity- and temperature-controlled, enclosed environment.

Correct maintenance of the hydroponic unit is vital to ensure optimal conditions for germination—usually 20/24 h of light and a temperature of
19–20°C. **Routine hygiene** is important to prevent the build-up of mold spores. The barley seeds used should be of the highest quality and must not be treated with mercurial or other seed dressings. The feeding of hydroponic barley is not commonly practiced today.

**Soya hulls and sugar beet pulp**

It has been suggested that certain fiber sources (sometimes referred to as **highly digestible fibers**)) such as sugar beet pulp practically provide more digestible energy to the horse than their traditional crude fiber, protein, fat, etc., analysis would suggest. This is in part because sugar beet pulp contains major fractions of pectins, arabinans and galactans, etc., which are lost during the crude fiber analysis, yet these carbohydrates can be fermented and thereby utilized by the horse. In addition, the fiber or more specifically the non-starch polysaccharide (NSP) in beet pulp is highly digestible over the total tract with a significant proportion being degraded in the small intestine during transit to the hindgut.

Various digestibility studies suggest that not only is sugar beet pulp well fermented in the horse (>60% digestibility of organic matter) but that this degradation occurs to a large extent within the time period that such a feed-stuff would remain within the gut. This explains why sugar beet pulp and a similar feedstuff, **soybean hulls**, are increasingly being used as fiber-based energy sources in modern horse feeds.

In the UK, **sugar beet** is usually molassed and presented as **dehydrated shreds** of compressed pellets. When rehydrated, there is a considerable increase in volume, and thus the practice of soaking sugar beet shreds or pellets in at least twice their dehydrated volume of water is essential before feeding. Small quantities of extruded unmolassed sugar beet in compound mixes need not be soaked before feeding, as the extrusion process has already expanded the material.

**METHODS OF FEED PROCESSING**

**Mechanical rolling, bruising or grinding** of cereal grains aims to break open the outer husk, thereby releasing the floury kernel for enzymic digestion. The disadvantages include a greater predisposition for oxidation and an increase in dust. In addition, **mold growth** occurs more readily in grains where the kernel has been broken.

**Micronizing** cereal grains and vegetable protein seeds is a rapid method of cooking and rolling the grains to gelatinize the starch and improve enzymic digestibility within the small intestine.

**Steam flaking** of cereal grains is a method of mechanically and biochemically altering the structure of the starch molecules in order to improve digestibility.

**Extrusion** of cereal grains and vegetable protein seeds involves grinding and increasing the moisture of the cereal grains and “cooking” the resultant slurry at very high temperatures and pressures before forcing the cooked material through a die, where the resultant drop in pressure forces a rapid expansion of the material as air enters the mixture. Typically this process leads to a moisture content of 8–10% (or DM of 90–92%).
Oil extraction and “roasting” of high oil seeds is an industrial process. The resultant oils extracted are then available for use in human or animal feeds, depending upon the degree of refinement.

Grinding and steam pelleting: individual ingredients are ground, mixed, steamed and forced under mechanical pressure.

Coarse mixing combines feedstuffs, often processed by one of the above methods, usually mixing them with a sugary syrup such as molasses or glucose.

FEED STORAGE

Correct storage of feedstuffs is essential to preserve their nutrient value, ensure palatability is retained, and help prevent fungal or bacterial contamination, which may affect the horse’s health. In order to help prevent fungal growth, feedstuffs should be of the correct moisture level; cereals with a moisture content >16% must be considered suspect. The treatment of high moisture cereal grains with propionic acid to act as a mold inhibitor and preservative has been practiced, although the effects of this compound on horses have not been widely studied.

Oxidation is a potential problem in rolled, cracked or bruised cereals. This rancidity will clearly affect palatability and may also affect nutrient availability, especially of certain vitamins. There may also be metabolic implications such as an increased level of peroxides. Storing feed at low humidity and low temperatures will reduce the rate at which oxidation occurs. The importance of maintaining feedstuffs at a suitable temperature, humidity and with adequate ventilation must therefore be strongly emphasized.

Infestation with mites is another common cause of feed spoilage. The practice of keeping feeds in galvanized bins can be useful, but care must be taken to ensure the bins are fully emptied and cleaned of all the previous material before a new bag or load is tipped in. In hot weather, high moisture feeds may cause condensation in the galvanized bins, so caution should be exercised.

FEEDSTUFFS ANALYSIS

From time to time it may be necessary to undertake more detailed analyses of specific feedstuffs. Such costly analysis will only be of value if representative samples, especially of pastures or roughage, are taken. Sampling may be required because of a suspected nutritional involvement in a particular disease or disorder where typical, tabulated analyses do not provide the necessary degree of detail or accuracy. Analysis may also be required in order to confirm that dietary ingredients meet the required or designated specification, or to assist with future purchases. If there is any suspicion of a feed related problem the samples should obviously be taken from the feedstuffs that have been fed.

Caution should be exercised as to whether the analyses are given on the fresh as fed material, or on a dry matter basis. “Dry matter” basis refers to the feed or forage after the moisture has been taken out, whereas the term “as fed” refers to a feed as it would be fed to a horse. Most concentrate feeds such as
cereals, cubes, pellets, etc., contain approximately 10% moisture with a dry matter content of 88–92% and fresh forage from 20% to 60%. It is important to realize that only the dry matter contains nutrients, so more feed will need to be fed, on an as fed basis, to match requirements if the feed contains more water.

Box 3.1 gives suggestions for routine analyses in order to assess general feed quality and major nutrient levels. In special circumstances, more detailed analyses will be required.

**Box 3.1 Suggestions for routine analysis in assessing general feed quality**

Cereal grains and by-products, vegetable and animal proteins

1. Dry matter.
2. Crude protein (CP).
3. Digestible crude protein (often based on ruminant data so caution needed with interpretation); or, as a guide,
   \[
   \text{DCP (g/kg DM)} = -27.2 + 0.917 \times \text{CP (g/kg DM)}.
   \]
4. NDF (and ADF).
5. Ash (as a possible guide to soil contamination).
6. Oil (various methods available, ether extract is usually adequate).
7. Energy (check if given in terms of ruminant ME [metabolizable energy], and conversion coefficients to horse DE should be used—current suggested conversions: divide by 0.9 for high fiber material, divide by 0.85 for low fiber material—very approximate). Many equations are available to estimate DE (i.e. there is no definitive equation) e.g.:
   \[
   \text{DE (MJ/kg DM)} = -3.54 + 0.0209 \times \text{CP} + 0.042 \times \text{Oil} + 0.0001 \times \text{CF} + 0.0185 \times \text{NfE}.
   \]
8. Micronutrient levels for these ingredients may be obtained from published tables.
9. Certain feedstuffs in the above category contain anti-metabolites in their raw state, and where toxic effects are suspected, analyses for the presence of the anti-metabolites should be made.
10. Results should be interpreted in terms of units per day intake (e.g. g/day protein, or MJ/day DE), rather than percentages as, unless intake quantity is known, the figures are meaningless in terms of effect upon the animal.

Forages (including hay, haylage, silage, straw)

1. The level of fungal contamination in hay should be assessed before purchase.
2. Dry matter.
3. Crude protein.
4. Digestible crude protein (is likely to be based on ruminant data so caution on interpretation).
5. Modified acid detergent (MAD) fiber (other more accurate methods may soon be available). NDF and ADF may also be of value.

*Box 3.1 continues on page 164*
PHYSIOLOGY AND PATHOLOGY OF DIGESTION

The empty alimentary tract accounts for about 5% of the total BW of a horse. The weight of the gut contents varies according to feeding, from 5% (concentrate) to 10% (roughage). In the small intestine, digestion is primarily by the body’s own enzymes, while in the voluminous large intestine the feed components are fermented by microorganisms. It is important to appreciate, however, that some fermentation will occur in the stomach and the small intestine. The approximate sizes of the various sections of the gastrointestinal tract are shown in Table 3.2.

Mastication

The duration of feed intake (Table 3.3) depends on the type of feed and the size of the animal. By feeding mainly concentrates, the time taken to ingest the feed and the number of chewing movements are greatly reduced. This may lead to a change in behavioral patterns: for example, animals may bite
and lick objects within their reach. Horses should be fed sufficient amounts of roughage (preferably long fiber or chop) daily in order to help prevent such abnormal behavior.

During chewing, the feed is thoroughly ground by the molar teeth; at the same time the secretion of saliva is stimulated. The grinding of whole grains is necessary for their optimal digestion in the small intestine. The intensity of the grinding of the roughage may be important for the passage of digesta through the ileocecal orifice and the large intestine. Short chopped straw or hay (≤20 mm) as well as very fine grasses (e.g. wind bent grass [or silky bent-grass], *Agrostis spica-venti*) may be swallowed without intensive chewing and grinding. This increases the risk of obstructions as well as increasing the risk of dental enamel points and hooks developing due to restricted chewing movements. The ingestion of lawn mower cuttings increases the risk of colic either due to an obstruction (q.v.) or dysfermentation (q.v.).

The ground feed is mixed with varying amounts of saliva depending on the duration of the feed intake (Table 3.4). This means that the DM of the boluses swallowed is higher after feeding concentrates than roughage. The occurrence of esophageal obstructions depends not only on the swelling capacity of the feedstuff (for example, dried sugar beet pulp), but also the speed of the feed intake and the size and DM content of the boluses swallowed.

### Table 3.2 Guide to the size of various parts of the gastrointestinal tract in horses as well as the duration of ingesta passage (500 kg BW)

<table>
<thead>
<tr>
<th>Length (m)</th>
<th>Fill (kg/100 kg BW) when feeding</th>
<th>Duration of passage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hay</td>
<td>Concentrates</td>
</tr>
<tr>
<td>Esophagus</td>
<td>Up to 1.5</td>
<td>—</td>
</tr>
<tr>
<td>Stomach</td>
<td>2.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Small intestine</td>
<td>16–24</td>
<td>2.2</td>
</tr>
<tr>
<td>Cecum</td>
<td>1</td>
<td>3.5</td>
</tr>
<tr>
<td>Colon</td>
<td>6–8</td>
<td>12</td>
</tr>
<tr>
<td>Rectum</td>
<td>0.2–0.3</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>20.2</td>
<td>11.5</td>
</tr>
</tbody>
</table>

### Table 3.3 Guide to the duration of feed intake in horses and ponies (min/kg feed)

<table>
<thead>
<tr>
<th>Approximate BW (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horse 500</td>
</tr>
<tr>
<td>Hay</td>
</tr>
<tr>
<td>Straw</td>
</tr>
<tr>
<td>Milled hay, pelleted</td>
</tr>
<tr>
<td>Oats</td>
</tr>
<tr>
<td>Pelleted feed (diameter 4–8 mm)</td>
</tr>
<tr>
<td>Chewing movements/min</td>
</tr>
<tr>
<td>Chewing movements/kg hay</td>
</tr>
<tr>
<td>Chewing movements/kg oats</td>
</tr>
</tbody>
</table>
Stomach and small intestine

The horse has a small, simple stomach, which is suited to the intake of rather small quantities of feed per meal. The cranial region of the stomach is non-glandular and is lined by stratified squamous epithelium similar to the esophagus. As this region fills, bacterial fermentation of the feed starts. This principally involves lactobacteria, which convert easily soluble carbohydrates to lactic acid.

Microbial activity and degradation is stopped when the gastric contents reach the fundic gland region and mix with the acid stomach juice containing pepsinogen.

Large quantities of digestive fluids are secreted into the small intestine, in particular from the liver (bile) and the pancreas into the duodenum. The main functions of the pancreatic secretion are to neutralize the acid chyme and to provide proteolytic, amylolytic and lipolytic enzymes. Bile also helps to alkalize the digesta, and the bile acids are required for emulsification and digestion of lipids.

Although the pancreatic enzyme amylase hydrolyses starch to disaccharides and trisaccharides, these have to be further digested by the mucosal enzymes before the resultant hexoses can be absorbed. Mucosal enzymes are also important for protein digestion and absorption.

In the small intestine of the adult horse, the digestive processes (i.e. the enzymatic degradation of proteins, fats, starch and sugar) are similar to those of other monogastric animals. However, the activity of most of the enzymes in the chyme, especially amylase, is lower than in other monogastric animals. The type of feedstuff affects the amount of soluble carbohydrate absorbed as glucose: up to 85% of the starch content of whole oats will be digested by the end of the ileum but only 30% of the starch from whole maize (for heat processed maize grains digestibility increases to approximately 90%).

Adult horses (500 kg BW) secrete >100 L fluid/day into the pre-cecal gut at approximately 70–100 mL/min. The DM content of the small intestine is about 5% so that even indigestible fibrous particles can be easily passed to the

Table 3.4 Principal differences between feeding roughage and concentrate feed

<table>
<thead>
<tr>
<th></th>
<th>Roughage</th>
<th>Concentrate feed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of feed intake</td>
<td>Long</td>
<td>Short</td>
</tr>
<tr>
<td>Salivation</td>
<td>Heavy</td>
<td>Less intensive</td>
</tr>
<tr>
<td>Dry matter of swallowed boluses (%)</td>
<td>&lt;15</td>
<td>&gt;25</td>
</tr>
<tr>
<td>Filling of the stomach</td>
<td>Slowly</td>
<td>Quickly</td>
</tr>
<tr>
<td>Content of the stomach (temporary)</td>
<td>Moderate</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>pH reduction in region of pylorus</td>
<td>Normal</td>
<td>Retarded</td>
</tr>
<tr>
<td>Microbial activity in stomach and small intestine</td>
<td>Moderate</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Production of organic acids in the large intestine</td>
<td>Continuously</td>
<td>Discontinuously with the risk of low pH in the cecum</td>
</tr>
<tr>
<td>Feces dry matter (%)</td>
<td>20</td>
<td>20–45</td>
</tr>
</tbody>
</table>

1 According to the amount of feed offered per meal.

The digestive tract

Roughage Concentrate feed

166

Table 3.4 Principal differences between feeding roughage and concentrate feed

Duration of feed intake | Long | Short |
Salivation | Heavy | Less intensive |
Dry matter of swallowed boluses (%) | <15 | >25 |
Filling of the stomach | Slowly | Quickly |
Content of the stomach (temporary) | Moderate | Moderate to high |
Dry matter of stomach content (%) | 20 | 30–40 |
pH reduction in region of pylorus | Normal | Retarded |
Microbial activity in stomach and small intestine | Moderate | Moderate to high |
Production of organic acids in the large intestine | Continuously | Discontinuously with the risk of low pH in the cecum |
Feces dry matter (%) | 20 | 20–45 |

1 According to the amount of feed offered per meal.

Stomach and small intestine

The horse has a small, simple stomach, which is suited to the intake of rather small quantities of feed per meal. The cranial region of the stomach is non-glandular and is lined by stratified squamous epithelium similar to the esophagus. As this region fills, bacterial fermentation of the feed starts. This principally involves lactobacteria, which convert easily soluble carbohydrates to lactic acid.

Microbial activity and degradation is stopped when the gastric contents reach the fundic gland region and mix with the acid stomach juice containing pepsinogen.

Large quantities of digestive fluids are secreted into the small intestine, in particular from the liver (bile) and the pancreas into the duodenum. The main functions of the pancreatic secretion are to neutralize the acid chyme and to provide proteolytic, amylolytic and lipolytic enzymes. Bile also helps to alkalize the digesta, and the bile acids are required for emulsification and digestion of lipids.

Although the pancreatic enzyme amylase hydrolyses starch to disaccharides and trisaccharides, these have to be further digested by the mucosal enzymes before the resultant hexoses can be absorbed. Mucosal enzymes are also important for protein digestion and absorption.

In the small intestine of the adult horse, the digestive processes (i.e. the enzymatic degradation of proteins, fats, starch and sugar) are similar to those of other monogastric animals. However, the activity of most of the enzymes in the chyme, especially amylase, is lower than in other monogastric animals. The type of feedstuff affects the amount of soluble carbohydrate absorbed as glucose: up to 85% of the starch content of whole oats will be digested by the end of the ileum but only 30% of the starch from whole maize (for heat processed maize grains digestibility increases to approximately 90%).

Adult horses (500 kg BW) secrete >100 L fluid/day into the pre-cecal gut at approximately 70–100 mL/min. The DM content of the small intestine is about 5% so that even indigestible fibrous particles can be easily passed to the
cecum. At the ileocecal junction, the chyme flow is stopped and the contents are discontinuously pressed into the cecum (5–7 times/h; up to 1 L at a time), which means that obstruction is a potential risk.

For the commonly fed diets consisting of hay and oats, approximately two thirds of the completely digestible parts of the feed will have been broken down and absorbed by the time the ingesta reaches the large intestine.

**Large intestine**

The large intestine does not possess mucosal enzymes and does not have active transport mechanisms for hexoses and amino acids. Digestion and absorption of residual carbohydrates and proteins relies instead on microbial action and absorption of the end products of microbial fermentation. The intensity of this process depends on the amount and the temporal influx of fermentable material arriving from the small intestine.

This bacterial degradation mainly produces volatile fatty acids (VFA), i.e. acetate, propionate and butyrate, plus amino acids, ammonia, sulfides, etc., and, after a high influx of easily fermentable carbohydrates, lactic acid. The rate of VFA absorption increases with decreasing pH. Disturbances of the digestive processes in the large intestine are marked on the one hand by insufficient microbial activity and on the other hand by accelerated degradation rates, particularly in the cecum.

Excluding damage to the flora (e.g. by antibiotics or mycotoxins) low microbial activity in the large intestine occurs when animals are fed rations consisting mainly of poorly fermentable components such as straw or late harvested hay. If large amounts of these feeds, which are difficult to break down, are ingested, obstruction of the colon (q.v.) may occur due to slow and incomplete microbial activity. This will be aggravated by any factor that decreases the rate of the passage of ingesta, such as lack of water, little exercise, parasites, and intake of soil and toxins which influence the flora of the large intestine, as well as gastrointestinal motility. On the other hand, if large amounts of easily fermentable substances that escaped digestion in the small intestine flow into the cecum, abnormal fermentation in the cecum may result in digestive disturbances and acidosis. This may happen with large amounts of mixed feed per meal or if carbohydrates such as maize starch or lactose are fed. Disturbances are especially likely if the animal has not adapted to a high grain diet.

Undigested proteins and urea that enter the large intestine are broken down by microbial enzymes. The main end product is ammonia, which is absorbed particularly at alkaline pH. Microbial protein, which is synthesized in the large intestine, fundamentally cannot be utilized by the horse. Animals with a high demand for protein (e.g. foals or lactating mares) must therefore be fed high quality protein that can be broken down and absorbed primarily in the pre-cecal section of the gut.

Most water-soluble vitamins as well as the fat-soluble vitamin K are synthesized in the large intestine. The horse appears to be able to utilize these so that oral supply is only necessary under certain circumstances (q.v.).
There is a large water and electrolyte turnover in the gastrointestinal tract. While considerable amounts of water, sodium (Na) and chloride (Cl) enter the small intestine via the saliva, stomach juices, pancreatic juice and bile, only about 50% of water, 35% Na and 80% Cl will be absorbed by the end of the ileum. Therefore a large ileocecal flow of water and Na (and to a lesser extent chloride) takes place (Table 3.5), but most of the water and electrolytes that enter the large intestine will be absorbed.

The large water and electrolyte turnover in the intestine has two consequences:

1. With an ileus (*q.v.*) in the small intestine, the intestine will fill up very quickly proximal to the blockage. The liquor may possibly reach back to the stomach while the animal becomes dehydrated.

2. Diarrhea (*q.v.*) in adult horses is mainly related to dysfunction of the large intestine because an elevated flow of water from the small intestine can usually be reabsorbed in an intact functional large intestine.

### The importance of microbial fermentation in the gut

Several types of microorganisms are present within the gut contents; the exact make-up is influenced by a number of factors including substrates that have been fed in the past and are currently being fed, the passage time, the pH and organic acid composition at the various sites as well as extent of the various interactions with the horse’s own digestive secretions (such as the gastric juices), etc. There are marked individual differences in the amount and type of the organisms throughout the GIT but the lowest numbers are found in the fundus region of the stomach.

---

**Table 3.5** Guide to the daily intake and turnover of water (L/100 kg BW) and electrolytes (g/100 kg BW)\(^1\)

<table>
<thead>
<tr>
<th></th>
<th>Intake</th>
<th>Secretion(s)</th>
<th>Flow from ileum into the cecum</th>
<th>Absorption</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water</td>
<td>3–5</td>
<td>20–25</td>
<td>10–14</td>
<td>9–12</td>
</tr>
<tr>
<td>Sodium</td>
<td>5</td>
<td>~50</td>
<td>30–42</td>
<td>28–35</td>
</tr>
<tr>
<td>Chloride</td>
<td>15</td>
<td>~60</td>
<td>10–14</td>
<td>10–14</td>
</tr>
<tr>
<td>Potassium</td>
<td>10</td>
<td>~9</td>
<td>5–7</td>
<td>2–3</td>
</tr>
</tbody>
</table>

Normal fermentation in meal-fed horses

Ingesta is propelled quite rapidly through the small intestine in a fluid form in the adult horse, some appearing in the cecum within 45 min and much of it reaching that point within 3 h of eating. Protein that escapes digestion in the small intestine is degraded to ammonia by bacteria in the ileum and to a much larger extent in the large intestine. The carbon skeletons are utilized as energy sources by the bacteria yielding acetic, propionic and butyric VFA as by-products. Starch that escapes digestion in the small intestine and most structural carbohydrates are subjected to fermentation by the large gut bacteria, again yielding VFA.

Bacterial fermentation also produces some longer chain fatty acids, as well as lactic acid. These acids and some of the ammonium ions are absorbed from the large intestine and enter the systemic circulation. Much of the ammonia, however, is reutilized by bacteria in the synthesis of bacterial protein, stimulating rapid bacterial growth. The bacterial population therefore ebbs and flows between the surges of ingesta reaching the large intestine.

It is important to note that there is a fluctuation in the hindgut bacterial populations in any meal-fed horse. The microflora population within the hindgut does adapt to a certain extent to the type of feed being fed. However, if the dietary fluctuations are too marked, or excessive starch or rapidly fermentable carbohydrates reach the hindgut even in the concentrate adapted horse, this will result in a significant change in the microbial population, which may have clinical consequences.

Dysfermentation

As well as obstructions, conditions associated with dysfermentation are the main cause of digestive upsets. Mistakes in feeding technique, selecting incompatible feeds or insufficient preparation of some feeds may induce inappropriate microbial growth and/or dysfermentation (q.v.), potentially with severe consequences for the health of the horse.

After ingestion of large meals of starch or sugar, fermentation is more extensive in the first part of the stomach, because the stomach contents have a higher DM content and the mixing of feed and gastric juice is therefore slower. This means that either the normal reduction in pH is delayed or the pH remains >4.5. Large amounts of lactic acid will be produced. The same process can occur with feeds that have a sticky consistency or potentially whenever gastric acid secretion is reduced. A further risk comes from feeds that are contaminated, especially with yeasts, when gas production may be so extensive that there is a risk that a stomach rupture (q.v.) may occur.

Mistakes in feeding technique and the selection of incompatible feeds may also have consequences in the small intestine. Again, inappropriate microbial fermentation (e.g. of easily digestible starch provided in large amounts) may produce high amounts of organic acids, resulting in a reduction in the pH and disruption of normal digestion. Spasmodic colic (q.v.) may be the final clinical result. All factors that reduce passage in the small intestine (excitement, stress, parasites, etc.) favor microbial activity and potentially increase the risk of colic.
As described above, large amounts of starch and, to a lesser extent, protein, ingested in a single meal may have an effect on digestion both in the stomach and the small intestine. In addition, significant amounts reach the large intestine, stimulating an almost explosive growth of microorganisms. Gas production can exceed the rate at which the methane, hydrogen and carbon dioxide are normally absorbed into the blood and expelled through the lungs so that the lumen of the large intestine becomes distended. Moreover, the rapid production of VFA and lactic acid in particular causes a rapid decrease in pH of the fluid, increases the permeability of the mucosa and upsets the microbial balance. This favors the growth of organisms that can withstand a lower pH, stimulating more lactic acid production and causing the death of certain bacteria that cannot survive under such conditions, thereby releasing endotoxins (non-protein lipopolysaccharide fragments of the cell wall of Gram-negative bacteria) and other compounds. These endotoxins, together with the other unwanted compounds produced as a consequence of this change in the conditions of the hindgut, may be absorbed into the blood and have further adverse effects. The blood flow to the feet, for example, may be particularly sensitive to some of these factors that may in turn trigger the development of laminitis (q.v.).

The importance of fiber

In addition to functioning as a source of energy, the fibrous components of feed have other values. In the long form (pieces in excess of 2–3 cm in length), fiber occupies the stabled horse’s time in chewing, so that it is less inclined to what are often referred to as “boredom-related” stable vices. The gastric contents have a higher moisture content and are more friable, allowing more immediate penetration of gastric juices, including HCl, and promoting better digestion further down the digestive tract. Also the microbial fermentation of fiber (fed as short or long material) proceeds at a slower pace than does the fermentation of starch or protein. This in turn has two advantages:

1. The ebb and flow of the microbial population of the hindgut, in numbers of organisms and in their species distribution between meals, is less marked than when starch and protein are the principal substrates.

2. By diluting readily fermentable material, wild fluctuations in the pH of the hindgut are prevented and thus the likelihood of acidosis is reduced.

Gastric ulceration

Modern management practices that include meal feeding, low fiber/high concentrate diets, early weaning and intensive training programs help to produce a poorly buffered, acidic environment in the stomach. This has been linked to the high prevalence of gastrointestinal ulcers (q.v.) particularly in intensively managed horses such as performance horses.

Foals are highly susceptible to ulceration because they start secreting gastrin just after birth before the gastric mucosa has fully developed. In addition, stressful weaning programs may act as an exacerbating factor in the development of gastrointestinal tract ulcers. There is a strong correlation between the
diet fed and the pH of the stomach. Concentrate diets have always been implicated but ensuring that these are fed in small amounts, possibly in combination with forages such as alfalfa hay, may help. A high forage intake, which encourages chewing and stimulates salivation, may also be advantageous. Turning out to pasture can be very beneficial for those that are affected. Medical treatment is often required.

NUTRIENT REQUIREMENTS

INTRODUCTION

The principal function of feed is to provide the nutrient requirements of the horse and its symbiotic gastrointestinal microorganisms. Maintenance requirements can be defined as the daily intake that maintains constant body weight (BW) and body composition as well as the health of a healthy adult horse with zero energy retention at a defined level of low activity in comfortable surroundings.

Nutrient requirements are typically stated as an amount per kg of feed or amount per kg BW daily. These amounts are the minimum needed to sustain normal health, production and performance of an average healthy animal. The amounts vary widely amongst horse groups with differing physiologic demands, i.e. growth, age, lactation, physical activity and workload (rider weight and ability, terrain and intensity of activity). They will also be affected by other factors including the environmental conditions. For example, energy requirements increase in animals exposed to very low temperatures, particularly where there is considerable air movement, and rain decreases the insulation properties of the coat.

As well as varying according to breed, body composition, stage of training, etc., it is also very important to remember that horses are individuals and differ in their metabolic efficiency (e.g. some horses are “good doers”), temperament, health status (including level of parasitic burden), appetite, likes and dislikes and other variables. It should be noted that there can be a difference between what a horse can eat and what it might need for maintenance, which in practice means that many mature horses will gain weight if fed free choice hay and not exercised.

Guidelines to requirements only can be provided; these then need to be tailored to the individual circumstances. It is important to note that one of the main reference materials used and referred to are the National Research Council (NRC) requirements, which recommend minimal rather than optimal requirements.

Imprecision in the assessment of nutrient requirements is compounded by uncertainty in feed analysis. The bioavailability of nutrients also varies between feed sources. The values given in Table 3.6 assume a high bioavailability from the ration.

Requirements can be given in a variety of ways. Two of the most common are per kg DM feed intake, and amounts per day on a dry matter or as fed basis. It is important to check what units are being used. Either can be suitable but obviously the DM intake guidelines rely on horses being fed appropriate amounts of feed for their workload, age, reproductive status, etc.
<table>
<thead>
<tr>
<th>Nutrient Requirements</th>
<th>Digestible energy (MJ)</th>
<th>Crude protein (g)</th>
<th>Lysine (g)</th>
<th>Ca (g)</th>
<th>P (g)</th>
<th>Mg² (g)</th>
<th>K (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Mature weight 500 kg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maintenance (adult)</td>
<td>69</td>
<td>656</td>
<td>23</td>
<td>21</td>
<td>14</td>
<td>7.5</td>
<td>25.0</td>
</tr>
<tr>
<td>Stallions (breeding)</td>
<td>86</td>
<td>820</td>
<td>29</td>
<td>26</td>
<td>18</td>
<td>9.4</td>
<td>31.2</td>
</tr>
<tr>
<td>Pregnant mares</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 mo</td>
<td>76</td>
<td>801</td>
<td>28</td>
<td>36</td>
<td>26</td>
<td>8.7</td>
<td>29.1</td>
</tr>
<tr>
<td>10 mo</td>
<td>77</td>
<td>815</td>
<td>29</td>
<td>36</td>
<td>27</td>
<td>8.9</td>
<td>29.7</td>
</tr>
<tr>
<td>11 mo</td>
<td>82</td>
<td>866</td>
<td>30</td>
<td>38</td>
<td>28</td>
<td>9.4</td>
<td>31.5</td>
</tr>
<tr>
<td>Lactating mares</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foaling to 3 mo</td>
<td>118</td>
<td>1427</td>
<td>50</td>
<td>57</td>
<td>36</td>
<td>10.9</td>
<td>46.0</td>
</tr>
<tr>
<td>3 mo to weaning</td>
<td>102</td>
<td>1048</td>
<td>37</td>
<td>38</td>
<td>22</td>
<td>8.6</td>
<td>33.0</td>
</tr>
<tr>
<td><strong>Working²</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light</td>
<td>86</td>
<td>820</td>
<td>29</td>
<td>29</td>
<td>18</td>
<td>4.3</td>
<td>14.1</td>
</tr>
<tr>
<td>Moderate</td>
<td>103</td>
<td>880</td>
<td>31</td>
<td>32</td>
<td>21</td>
<td>5.1</td>
<td>16.9</td>
</tr>
<tr>
<td>Intense</td>
<td>137</td>
<td>1050</td>
<td>37</td>
<td>40</td>
<td>29</td>
<td>6.8</td>
<td>22.5</td>
</tr>
<tr>
<td>Foal growing at 1.0 kg/day³</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo</td>
<td>60</td>
<td>730</td>
<td>30</td>
<td>37</td>
<td>19</td>
<td>4.0</td>
<td>11.3</td>
</tr>
<tr>
<td>6 mo</td>
<td>71</td>
<td>864</td>
<td>36</td>
<td>40</td>
<td>20</td>
<td>4.6</td>
<td>13.3</td>
</tr>
<tr>
<td><strong>Yearling</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Growing at 0.6–0.8 kg/day</td>
<td>87</td>
<td>956</td>
<td>40</td>
<td>40</td>
<td>20</td>
<td>5.7</td>
<td>18.2</td>
</tr>
<tr>
<td>24 mo old</td>
<td>79</td>
<td>820</td>
<td>32</td>
<td>28</td>
<td>14</td>
<td>7.0</td>
<td>23.1</td>
</tr>
<tr>
<td>Not in training</td>
<td>110</td>
<td>1050</td>
<td>41</td>
<td>35</td>
<td>19</td>
<td>9.8</td>
<td>32.2</td>
</tr>
<tr>
<td><strong>2. Mature weight 200 kg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maintenance</td>
<td>31</td>
<td>296</td>
<td>10</td>
<td>10</td>
<td>6</td>
<td>3.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Stallions (breeding)</td>
<td>39</td>
<td>370</td>
<td>13</td>
<td>11</td>
<td>8</td>
<td>4.3</td>
<td>14.1</td>
</tr>
<tr>
<td>Pregnant mares</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 mo</td>
<td>34</td>
<td>361</td>
<td>13</td>
<td>16</td>
<td>12</td>
<td>3.9</td>
<td>13.1</td>
</tr>
<tr>
<td>10 mo</td>
<td>35</td>
<td>368</td>
<td>13</td>
<td>16</td>
<td>12</td>
<td>4.0</td>
<td>13.4</td>
</tr>
<tr>
<td>11 mo</td>
<td>37</td>
<td>391</td>
<td>14</td>
<td>17</td>
<td>13</td>
<td>4.4</td>
<td>14.2</td>
</tr>
<tr>
<td>Lactating mares</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foaling to 3 mo</td>
<td>57</td>
<td>688</td>
<td>24</td>
<td>27</td>
<td>18</td>
<td>5.0</td>
<td>21.2</td>
</tr>
<tr>
<td>3 mo to weaning</td>
<td>51</td>
<td>528</td>
<td>18</td>
<td>18</td>
<td>11</td>
<td>4.0</td>
<td>14.8</td>
</tr>
<tr>
<td><strong>Working⁶</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Light</td>
<td>39</td>
<td>370</td>
<td>13</td>
<td>11</td>
<td>8</td>
<td>4.3</td>
<td>14.1</td>
</tr>
<tr>
<td>Moderate</td>
<td>46</td>
<td>410</td>
<td>14</td>
<td>14</td>
<td>10</td>
<td>5.1</td>
<td>16.9</td>
</tr>
<tr>
<td>Intense</td>
<td>62</td>
<td>450</td>
<td>16</td>
<td>18</td>
<td>13</td>
<td>6.8</td>
<td>22.5</td>
</tr>
<tr>
<td>Foal growing at approx 0.5 kg/day</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo</td>
<td>31</td>
<td>365</td>
<td>15</td>
<td>16</td>
<td>9</td>
<td>1.6</td>
<td>5.0</td>
</tr>
<tr>
<td>Yearling growing at 0.2–0.3 kg/day</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 mo old</td>
<td>33</td>
<td>337</td>
<td>13</td>
<td>9</td>
<td>5</td>
<td>2.8</td>
<td>9.4</td>
</tr>
<tr>
<td>Not in work</td>
<td>48</td>
<td>485</td>
<td>16</td>
<td>13</td>
<td>7</td>
<td>4.1</td>
<td>13.5</td>
</tr>
</tbody>
</table>

2 Assumes availability of 35%.
3 See also Tables 3.15 and 3.16.
4 See also Tables 3.17 and 3.18.
BODY WEIGHT ESTIMATIONS

Many nutrient requirements are proportional to body weight. Unfortunately, judgment by eye can be inaccurate; calibrated weighbridges are the most accurate but are not commonly available. Weigh tapes and estimations based on linear measurements provide an approximation but these can only be taken as a guide or used for monitoring purposes where standardized procedures are followed.

There are a number of equations available such as that to estimate body weight in kg \( w \) from heart girth in cm \( hg \) and length of the body in cm \( l \) from the point of the shoulder to the point of buttock for the adult horse only:

\[
w = \frac{(hg)^2 \times l}{11877}
\]

This will tend to overestimate the weight, for example, of those horses with reduced gut fill in hard work such as the fit racehorse and is not reliable for the pregnant mare in late gestation or for young, growing animals, etc.

WATER

The requirements for water are given in Table 3.7. The water content of the body should remain within the approximate limits of 68–72% of fat-free mass. Values below this represent a dehydrated state. Water is lost by excretion in urine, feces and sweat, as well as by evaporation from the lungs and in milk. Lactation can increase needs by 50–70% above maintenance.

The requirement for supplementary water is influenced both by the amount of DM consumed and by the moisture content of feeds available. Cereals and hay contain approximately 10% moisture whereas pasture herbage contains 40–80% moisture depending on season and rainfall. These sources affect the supplementary amounts of water required (Tables 3.7 and 3.8). Dry mares (or...
other non-lactating horses) on lush pastures with shade, undertaking no work, can thrive without additional water, although it is always advisable that a clean supply be made available. Pregnant and lactating mares should be provided with supplementary water at all times (see Tables 3.7 and 3.8). Foals should have adequate access from around 2 weeks of age. The requirements of the breeding stallion are similar to those of the pregnant mare.

Maintenance needs are usually met by providing 2–3.5 L/kg DM intake on a mixed diet of grain and hay in temperate conditions. For all hay diets, 3.5–4.0 L/kg DM may be needed. Environmental temperature has a large effect on the amount consumed. Values of 2 L/kg DM at 18°C and 8 L/kg DM at 38°C have been reported.

Sweating involves losses in particular of sodium, potassium and chloride in addition to water so that severe sweating also necessitates the replacement of these electrolytes.

**Sweat production**

Unfortunately, the conversion of chemical energy provided by the feed to mechanical energy in the form of ATP that can be used by the muscles is not very efficient, and the “waste” heat that is produced has to be removed from the body. One of the main mechanisms for heat removal is via the evaporation of sweat. The amount of sweat produced depends on the environmental conditions, nature of the work (which in turn will depend on the rider’s ability and the terrain) and the animal’s fitness. Under favorable climate conditions, sweat loss can be around 2–5 L/h if the work is a run at a low pace (approximately 2–4 m/s) or in the order of 7–8 L/h in long distance rides at a faster pace or in difficult terrain (Table 3.9).

In hot humid conditions where sweating is partially ineffective, production can be as high as 10–15 L/h. Sweat production seems to decrease only after extreme water loss; although there may be some changes in sweat composition with time, basically sweat production is accompanied by an obligate loss of electrolytes. When the sweat loss is low, much of the loss can be made up by absorption of water contained in the large intestine, but if water losses are greater (3–4% BW) a decrease in circulatory volume as well as loss of skin elasticity occurs.

### Table 3.8 Guide to the minimum supplementary water requirements of mares (liters daily per mare)—refer to Table 3.7 for maintenance levels

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>200</th>
<th>400</th>
<th>500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Last 90 days of gestation&lt;sup&gt;2&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In stable</td>
<td>13</td>
<td>22</td>
<td>26</td>
</tr>
<tr>
<td>On pasture</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>First 3 months of lactation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In stable</td>
<td>28</td>
<td>42</td>
<td>50</td>
</tr>
<tr>
<td>On pasture</td>
<td>7</td>
<td>11</td>
<td>12</td>
</tr>
</tbody>
</table>

<sup>1</sup> These amounts are minimum. They assume ideal conditions and will be insufficient for mares on parched pasture with environmental temperatures >30°C.

<sup>2</sup> Needs of the breeding stallion are similar to those of the pregnant mare.
Horses participating in endurance rides over distances of 50–200 km typically lose 3–7% of their body weight during the competition—some horses may lose 10% or more. These losses are only partially compensated for during overnight stops, perhaps due to persistent loss in the GIT content, which takes longer than an overnight period to recover to pre-race levels.

Sweat contains relatively low levels of calcium (approximately 0.12 g/L), magnesium (approximately 0.05 g/L) and phosphate (0.01 g/L) but relatively high levels of sodium, potassium and chloride as shown in Table 3.10. There are also small amounts of various trace elements, e.g. iron at approximately 4.3 mg/L and Zn at 11.4 mg/L. However, the main electrolytes lost with sweat are sodium, potassium and chloride.

**ENERGY**

Energy is supplied to the horse via its diet, but fundamentally energy is not a nutrient. The chemical energy or gross energy contained within feeds needs to be converted into a form of energy that the cells can use for work or movement (usable or net energy). Dietary energy is provided by the four principal dietary energy sources.

1. **Hydrolyzable carbohydrates**, e.g. simple sugars and starch. These can be digested by mammalian enzymes to hexoses that are absorbed from the

<table>
<thead>
<tr>
<th>Electrolyte/element</th>
<th>Amount lost per L sweat</th>
<th>Approximate amount needed to be ingested to replace amount lost per L sweat (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>3.1 g/L</td>
<td>3.45</td>
</tr>
<tr>
<td>Potassium</td>
<td>1.6 g/L</td>
<td>2</td>
</tr>
<tr>
<td>Chloride</td>
<td>5.3 g/L</td>
<td>5.5</td>
</tr>
<tr>
<td>Calcium</td>
<td>0.12 g/L</td>
<td></td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.05 g/L</td>
<td></td>
</tr>
<tr>
<td>Phosphorus</td>
<td>&lt;10 mg/L</td>
<td></td>
</tr>
<tr>
<td>Zinc</td>
<td>11 mg/L</td>
<td></td>
</tr>
<tr>
<td>Iron</td>
<td>5 mg/L</td>
<td></td>
</tr>
<tr>
<td>Copper</td>
<td>0.3 mg/L</td>
<td></td>
</tr>
<tr>
<td>Selenium</td>
<td>Traces</td>
<td></td>
</tr>
</tbody>
</table>

Table 3.10 Guide to the composition of sweat

Table 3.9 Sweat production

<table>
<thead>
<tr>
<th>Work</th>
<th>Amount of sweat (L/100 kg BW)</th>
<th>Average</th>
<th>Average water requirement (L/100 kg BW/day [including maintenance])</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td>0.5–1</td>
<td>0.75</td>
<td>5</td>
</tr>
<tr>
<td>Moderate</td>
<td>1–2</td>
<td>1.50</td>
<td>5–7</td>
</tr>
<tr>
<td>Heavy</td>
<td>2–5</td>
<td>3.50</td>
<td>7–10</td>
</tr>
<tr>
<td>Very heavy</td>
<td>&gt;5</td>
<td>5.00</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>

1 Depending on duration and intensity of activity and environmental temperature. Rule of thumb: 1 L/100 kg BW/h with light trotting at 18–20°C.

**Chapter 3  Equine nutrition and metabolic diseases**

- 175
small intestine (SI) or, if they “escape” digestion in the SI, are rapidly fermented in the hindgut.

2. **Fermentable fibers**: component of dietary fiber—cellulose, pectins, hemicelluloses, etc. These are not digestible by mammalian enzymes but can be fermented by the microorganisms predominantly located in the hindgut. Speed of fermentation as well as site may play an important role in the energy value to the horse.

3. **Oils/fats**, utilized as fatty acids and glycerol. Despite their more “evolutionary traditional” diet containing relatively low concentrations of oils, horses in general appear to be able to digest and utilize up to 20% of their diet as oil if suitably introduced.

4. **Proteins**. Although much of the constituent amino acids will be utilized in protein synthesis, depending on the dietary level and the balance of the amino acids, a significant proportion of the carbon skeletons of many amino acids are combusted as energy sources or converted into fat or glucose. Not an efficient energy source (see below).

With the exception of feeds that contain a lot of fats or ash, the gross energy content of feeds tends to be similar. Differences arise mainly from differences in digestibility. Hay has a lower digestibility than cereals; it produces much more spare “lost” heat so is much more “internally heating” and is therefore especially useful in winter. In addition, the efficiency of conversion of digestible to usable or net energy differs widely.

Cereals have more net energy than hay, which in turn contains more than twice the net or usable energy of straw. Vegetable oils contain proportionally more net energy than the cereals and 2.25–3 times the amount of digestible energy. Replacing forage with cereals and/or oil decreases the amount of feed the animal has to eat in order to obtain the required amount of energy (important as horses have a finite appetite).

**Energy requirements**

Critical to feeding any horse for health and vitality is the appropriate and adequate supply of energy, especially during the **training** phases (q.v.). If a horse is fed too little energy for its needs it will tend to become **dull and lethargic**, lose weight and/or become clinically ill. If a horse is fed too much energy or inappropriate energy it may become **hyperactive**, gain weight and/or become ill.

Adult horses in particular tend to be fed primarily for energy; the diet is then balanced for protein, vitamins and minerals.

At present, the energy potential of a horse feed is described in two main ways: digestible energy (DE) and net energy (NE). Each of these has been determined in a number of ways over the years. The DE system is the most commonly used in UK, Germany and the USA; the NE system and its variants are gaining popularity in the rest of mainland Europe.

The French (who pioneered the value of the NE system for horses), for example, base their system on the horse feed unit (UFC), a net energy system of values relative to the NE values of barley. Net energy is considered to be
that portion of the feed energy that is actually available for use—be that movement, production of milk, fetal growth, etc. However, the efficiency of conversion of the feed energy to available energy varies according to use: for example, approximately 25% efficiency when converted to kinetic energy, approximately 70% for milk production, but only approximately 20% for fetal growth. Figures that have been derived from one use may therefore not be appropriate for another. It is also important to realize that the DE and NE systems are not easily interchangeable and therefore one system should be used throughout to calculate both requirements and the rations to fulfill such requirements.

Two units of energy are in common use in the horse industry: the SI unit of energy, the joule (J where kilojoule [kJ] = \(10^3\) J, and the megajoule [MJ] = \(10^6\) J), a unit of electrical work used predominantly in Europe, and the calorie in the USA (4.184 J approximates to 1 calorie).

Requirements and intake are given either as the amount per kg of DM (zero moisture) or the amount in air-dried feed (assumed to contain 12% moisture). Depending on the system and the country, recommendations are given for estimated metabolic body weight and others for actual body weight. Various equations have been used around the world to predict energy requirements. The NRC (1989), for example, uses for maintenance (up to 600 kg BW):

\[
\text{Maintenance} = 4.184 \times (1.4 + 0.03 \times \text{Body weight}) \text{ MJ DE/day}
\]

An adult horse not doing any work typically requires, very approximately, about 13–15 MJ DE/100 kg body weight. The amount of total feed in the daily ration should be adjusted according to the amount of work performed and the condition of the individual horse. It is therefore important to monitor body condition and weight regularly. Obese or underweight horses are unlikely to perform optimally, and certain clinical conditions may result in a loss of weight.

As energy is stored by the horse there is no requirement for energy needs to be fully met on a daily basis. Indeed it is unlikely that they will, or should be. The immediate energy requirements are met almost entirely from energy stored as glycogen, fat, circulating glucose and high energy phosphates.

**Energy deficiency and excess**

A protracted deficiency of dietary energy causes a loss of depot fat and of muscle mass, whereas excess dietary energy leads to fat deposition at subcutaneous abdominal and intra- and inter-muscular sites. Both can result in clinical signs as well as changes in temperament.

**PROTEIN**

Dietary protein is normally measured as crude protein (CP: nitrogen \(\times 6.25\), for most common feedstuffs). Digestible crude protein (DCP) is defined as the proportion of dietary CP that is apparently digested. Frequently this is a calculated value rather than a value derived from digestibility studies in horses, and it varies considerably from approximately 40% to 80% of CP. The digestibility appears to vary in relation to the protein content of the feed.
(e.g. low protein grass hay will have a lower protein digestibility than high protein alfalfa hay) and to the concentrate–roughage ratio (increasing the concentrate–hay ratio from 1:1 up to 3:1 will tend to increase digestibility; above this range the digestibility may decrease).

**Protein sources**

Most feedstuffs contain some protein but, as indicated in Table 3.1, there is considerable variability in the amount of protein and amino acids, especially lysine (limiting to the rate of tissue protein synthesis). The oil seed meals are generally good sources of protein whereas cereal grains contain less protein and are also generally poorer in their amino acid profile. Grass hay typically provides less digestible protein than cereals, whereas the protein value of legume roughages depends considerably on the care that has been taken to preserve plant leaf.

**Dietary protein requirements**

Requirements vary in proportion to tissue demands for protein synthesis and are, therefore, much greater during lactation or during growth than for adult working horses. The requirements are set (see Table 3.6) as those that meet the minimum needs of approximately 95% of healthy horses, accepting that individual differences exist. During ill health, such as recovery from trauma and infection, protein needs increase considerably. Again, recommendations vary according to the various systems. The NRC recommends approximately 10 g CP/MJ of DE per day for horses at rest and in work.

**Protein deficiency**

Insufficient dietary protein causes a negative nitrogen balance, loss of protein from tissues, restrictions in milk production of lactating mares and growth failure in foals. There are no specific symptoms of deficiency other than a gradual loss of muscle and liver mass, with a decline in blood plasma albumin concentration, failure of suckling foals, or of weaned foals, to grow and some loss of appetite. A protein deficiency is possible especially where poor roughages constitute the majority of the diet, when energy will also be limiting. This partly results from a decline in the ability of large intestine microorganisms to degrade dietary fiber when nitrogen intake is severely restricted.

**Amino acid balance**

A protein deficiency implies an amino acid deficiency. Few natural protein sources contain the optimal proportion of dietary essential to non-essential amino acids, and few potential feed proteins contain an ideal distribution of the 10 dietary essential amino acids (based on experimental work on growth in the rat). Thus, in biologic terms, a protein deficiency can be most efficiently rectified by giving initially supplements containing the amino acid(s) that
is/are the principal limiting amino acid to tissue protein synthesis (including growth, milk production, etc.).

In the horse, **lysine** has been established as the first limiting amino acid for most diets. Of the other dietary essential amino acids, only six of the 10 have been demonstrated to change in respect of their blood plasma concentration when a protein deficiency is induced. These are isoleucine, leucine, phenylalanine, threonine, tryptophan and valine. Few of these have been adequately examined, although it is believed that for growing horses **threonine** may be the second limiting amino acid.

The practical solution for supplying additional limiting amino acids is to add **soya bean meal**, or other good quality protein, to the diet. It could then be assumed that the need for the limiting amino acids would be met (providing extensive synthetic sources alone have not been used, see below).

**Protein excess**

A moderate excess of dietary protein (up to approximately 50% above the requirements of the individual) has no observable adverse effect in healthy horses. More than this may cause a slight reduction in the performance of racehorses and possibly some reduction in appetite. It has been recommended not to feed >2g DCP/kg BW to exercising horses, especially endurance horses.

Protein, however, is not a nutritionally preferred option as an energy source as it is inefficiently converted to usable energy with proportionally higher amounts of waste energy (heat) produced; the nitrogen must be removed, as excess protein is not stored. Excess protein is degraded in the liver with the formation of urea. This is excreted by the kidneys (urine) and is secreted back into the lumen of the gut. There are potentially higher **ammonia levels** in the stable as the urea in the urine is converted by bacteria in the environment to ammonia.

The process of degradation and excretion of the products requires the expenditure of energy and increases basal water requirements. Where there is loss of either hepatic or renal function, large excesses of dietary protein may cause an accumulation of protein degradation products and even cause **ammonia toxicity**, although under normal circumstances this is unlikely.

Protein excess may result in increased renal losses of Ca and P, although evidence regarding this is conflicting. Despite a link in many horse breeders’ minds, there have not been any conclusive studies demonstrating adverse effects of high or low protein intakes on the incidence of **developmental orthopedic disease (DOD)** (q.v.) in the horse.

Deficiencies and excesses can also occur where total protein intake is within the normal range, but where dietary amino acid imbalances exist. The most likely situation is when growing horses receive diets providing poor quality proteins deficient in lysine. Another possibility exists where synthetic amino acid supplementation occurs of some amino acids only or in the incorrect proportions.

**MINERAL AND TRACE ELEMENTS**

The macro- and micro- (or trace) elements required in the diet for normal cellular function can be provided inorganically or organically. **Macroelements**
are required in quantities in the range of several grams per day and include sodium, potassium, chloride, calcium, phosphorus and magnesium. Sulfur is normally considered a macromineral and is provided organically from proteins containing sulfated amino acids such as methionine and cystine. The electrolytes (Na, K, Cl, Ca, Mg—substances that exist as positively or negatively charged particles in aqueous solution) principally affect intra- and extracellular ion and acid-base balance. Microelements are required in milligrams per day and include iodine, iron, manganese, zinc, selenium, cobalt and copper.

The elements that are most frequently deficient in natural diets are calcium, sodium, chloride, copper, zinc, iodine and selenium. Others that could be deficient in some areas of the world include cobalt and chromium. It is also possible that some of these elements could be present in toxic quantities.

Utilization of dietary sources

The availability, i.e. the proportion of an element that can be absorbed (true digestibility), differs considerably amongst sources of the same element, and also amongst elements; for example, the availability of common calcium sources may vary from 35% to 60% whereas the extent to which some trace elements are absorbed may be <5% of that consumed. In addition, there may be marked individual variability in the availability from different sources. Thus it is often not possible to give exact values for required dietary content.

Interactions

There are many interrelationships amongst the macro- and micronutrients. Few of the possible interactions have been thoroughly investigated in the horse and in practice only a few of the possible interactive effects appear to require practical measures to avoid adverse consequences. For example, while little evidence exists that moderate excesses of dietary Ca have any deleterious effects on P or Zn utilization, an excessive dietary concentration of P with Ca:P ratios ≤1.0 may depress calcium absorption and may cause skeletal problems.

Mineral requirements

The minimal maintenance requirements of minerals depend on their endogenous (inevitable) fecal, renal and cutaneous losses and their availability (Table 3.11).

Calcium, phosphorus and magnesium

In adults, the rate of calcium absorption seems not to be regulated at the gut wall, so that the amount of calcium absorbed increases proportionately with intake. Renal excretion therefore increases with intake. Calcium absorption appears to be higher in younger than in older animals. It is, however, reduced with high intakes of oxalic acid and phytate. Greater than 0.5% oxalic acid in
the feed may reduce calcium absorption when the calcium–oxalate ratio is <0.5 (weight to weight basis). With a ratio ≥1, higher oxalic acid levels (up to 0.87%) may be tolerated. High phosphorus intake (especially in the form of phytate) may disturb calcium absorption, especially when the Ca:P ratio is ≤1.

Major natural sources of calcium are leafy forages, particularly legumes, while supplementation may be achieved for example with limestone flour, calcium gluconate or dicalcium phosphate.

The greatest requirements for calcium arise in the young foal where bone mineral is being accreted. Inadequate calcium intakes by developing foals are characterized by poor mineralization of the osteoid tissue (q.v.). Mares at the peak of lactation use considerable quantities of calcium for secretion in milk and therefore their requirements are higher than those of other adult horses. At peak lactation they are usually in negative calcium balance, drawing on bone calcium reserves. In working horses, low calcium intake or high oxalate or phosphorus content in feeds can lead to nutritional secondary hyperparathyroidism (“big head disease”) (q.v.).

Although there is no conclusive evidence that excessive calcium intakes are harmful to skeletal development in the horse, it is considered inadvisable to exceed 3–4 times the requirements as shown in Table 3.12.

Phosphorus absorption, which mainly takes place in the large intestine, is not affected significantly by high calcium intakes. Phytate P of plant origin is less well absorbed than inorganic P, but it may be partially available because there is some phytase activity in the large intestine.

Cereal grains and their by-products are the principal sources of phosphorus. Phosphorus from cereal sources is approximately half to two thirds as available as that in dicalcium phosphate, the common supplementary source.

Protracted and inadequate intake of phosphorus may produce skeletal abnormalities in growing horses similar to those produced by inadequacies of calcium and vitamin D. Deficiency may also cause abnormal appetite.

Excessive phosphorus reduces calcium absorption and potentially leads to nutritional secondary hyperparathyroidism (q.v.). Dietary phosphorus should conform to the amounts indicated in Tables 3.6 and 3.12, and the calcium–phosphorus ratio should lie between 1:1 and 2:1.

Magnesium absorption is slightly higher from organic compounds (e.g. magnesium aspartate) than from inorganic sources (e.g. MgO). No negative effects on absorption have been reported with oxalates. Hay feeding has

---

**Table 3.11** Guide to endogenous mineral losses by horses, average availability and the minimum mineral requirements for maintenance per kg BW/day

<table>
<thead>
<tr>
<th></th>
<th>Ca</th>
<th>P</th>
<th>Mg</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endogenous losses (mg/kg BW/day)</td>
<td>30</td>
<td>12</td>
<td>5</td>
<td>18</td>
<td>40</td>
<td>5–10</td>
</tr>
<tr>
<td>Assumed availability (%)</td>
<td>60</td>
<td>40</td>
<td>35</td>
<td>90</td>
<td>80</td>
<td>95–100</td>
</tr>
<tr>
<td>Requirements for maintenance (mg/kg BW/day)</td>
<td>50</td>
<td>30</td>
<td>15</td>
<td>20</td>
<td>50</td>
<td>80</td>
</tr>
</tbody>
</table>

The endogenous losses include a safety margin to allow for variations resulting from the type of feed, the individual animal and the amount supplied.

1 According to influence on acid-base balance.
been said to favor calcium and magnesium absorption (in contrast to pure concentrate feeding). Major sources are oil seeds, rice grains, legume forages, sugar beet pulp and sugar beet molasses. Most good quality mixed natural diets contain at least 0.1% magnesium. This will meet the requirements of the majority of horses. Nevertheless many manufacturers provide supplements of magnesium as calcined magnesite (MgO), in commercial feeds. The availability of magnesium in MgO of various origins has been shown to differ for the ruminant, but no equivalent studies have been undertaken for horses.

Muscular tremors and ataxia resulting from acute hypomagnesemia are unlikely to occur in horses given good quality natural feedstuffs, but may occur in horses receiving magnesium-deficient feedstuffs although not proven to date.

No evidence has been produced that excessive intakes of magnesium are harmful, except sulfates, which are purgative. Although there is a potential risk of magnesium toxicity where dolomite limestone is used, the lack of evidence for this may indicate low availability of magnesium from this source.
Sodium, potassium and chloride

With a low sodium or potassium intake the endogenous losses will be reduced to <10 mg/kg BW/day for sodium and around 5 mg/kg BW/day for potassium. However, this does not appear to be the case for chloride. Low intakes may increase the true absorption rate for sodium and potassium. In horses with diarrhea or dysfermentation in the intestine, the fecal losses and, therefore, the total requirement will increase.

The maintenance requirement of chloride is not only related to the Cl balance but also to the acid-base metabolism. Intakes of <80 mg/kg BW/day may result in lower plasma Cl concentrations (≤100 mmol/L), alkaline urine, and a tendency for the development of metabolic alkalosis (q.v.).

Sodium and chloride are commonly provided by common salt, milk products and sugar beet molasses. Nutritionally improved straw is also a source. The main sources of potassium are leafy forages, sugar beet molasses and milk products.

The provision of forage and water in the last meal before extended exercise increases the fluid and electrolyte contents of the large intestine, which it is believed can act as a reservoir of these nutrients.

Possible clinical signs associated with deficiencies

Forages are generally rich in potassium, and diets containing adequate forage of good quality are unlikely to be potassium deficient. Acute water and potassium depletion may occur in foals with chronic diarrhea, or in adult horses subjected to extended exercise, especially when being fed low forage intakes and/or in hot weather. In both cases, water and electrolyte supplements are required to bring about immediate rectification.

Whereas most good quality rations provide sufficient potassium for normal maintenance, supplementary sodium in the form of common salt is generally advised, and is necessary for horses given rations based on cereals and undertaking extended exercise in hot weather. Sodium, potassium and chloride are major constituents of sweat, and depletion of these electrolytes by heavy sweating suppresses thirst and the desire to drink despite dehydration.

Possible clinical signs associated with excess

The horse has little capacity to store water or potassium and excessive dietary intakes are rapidly excreted, although in the case of potassium this assumes that water intake is adequate. Potassium toxicity is unlikely except when given parenterally in excess, when it can cause cardiac arrest (q.v.) or hyperkalemic periodic paralysis (q.v.). Sodium toxicity is unlikely under normal circumstances, except where a dehydrated horse is given access to salt water ad libitum.

Micro- (trace–) mineral requirements

Principal sources of microminerals

The adequacy of most natural plant materials as sources of microminerals depends to a large extent upon the soil on which the plants are grown. However,
soil content and plant content, especially for certain trace elements such as selenium, do not always closely correlate. It is common practice to supplement feeds with inorganic sources. These should be prepared by competent organizations, as there is a risk of providing excess. Synthetic sources are the most practical alternative for copper, manganese, zinc and selenium. Seaweed meal or potassium iodide can be used as sources of iodine, although toxic intakes have occurred where users have not been aware of the risks of excess.

The most critical trace elements in typical horse diets are iodine, selenium and copper (see Table 3.12 for information on requirements).

**Deficiencies and excesses of microminerals**

Signs of *iodine* deficiency and excess are similar, and are observed under practical conditions only in foals. Diets for the dam that provide <0.5 mg or >50 mg iodine per day, with a daily dry feed consumption of 10 kg, are likely to cause goiter. Some reports suggest that even lower intakes may increase the risk of goiter (*q.v.*) and therefore it has been recommended that pregnant mares are in fact fed <1 mg iodine/kg DM intake and preferably not >1 mg of iodine per 100 kg BW. Excess iodine is also a possible cause of bony abnormalities in foals. Excesses are commonly caused by feeding large amounts of *seaweed meal* or supplementation with excessive potassium iodide or iodate.

Horses apparently can tolerate high levels of dietary *copper*. Dietary deficiencies of copper, including *copper-deficient pastures*, especially for the dam during gestation, are thought to be a factor in the pathophysiology of certain developmental orthopedic diseases (*q.v.*). It has been suggested that diets containing approximately 20 mg/kg DM may be preferable both for the pregnant and lactating mare as well as the growing foal. Moreover, copper availability in pastures may be adversely influenced by heavy metals.

*Zinc* deficiency in foals is accompanied by inappetence, reduced growth rate and parakeratosis (especially of the lower limbs). Zinc deficiency and excess have been linked to DOD, although there are limited data to support this assertion. Excessive intake can depress plasma copper concentration.

*Manganese* is required in the synthesis of chondroitin sulfate and deficiencies in other species have led to abnormalities in *cartilage development*. *Cobalt* is required by intestinal bacteria in the synthesis of vitamin B₁₂ and therefore is likely to be of most importance to breeding mares and growing foals. *Iron* is essential for the synthesis of hemoglobin, myoglobin and the cytochromes. Natural diets containing 50 mg/kg iron should be adequate for growing foals, and therefore a deficiency should not occur where good quality natural diets are given. There is a risk of toxicity (*nutritional siderosis*) where excessive supplementation occurs.

*Selenium* deficiency is seen particularly in foals of mares grazing selenium-deficient pastures. This may lead to nutritionally associated myopathy (*white muscle disease*) (*q.v.*), which occurs in both skeletal and cardiac muscle. Selenium deficiency may also lead to depressed immune responses and lowered thyroid hormone availability. Diets containing <0.05 mg/kg DM are likely to depress blood glutathione peroxidase (GSH-px) activity below an acceptable limit.
The margin of safety for selenium is relatively narrow and selenosis can occur. While acute toxicosis (blind staggers) \( (q.v.) \) tends to result in blindness, as well as gastrointestinal, cardiovascular and respiratory signs, it is the more chronic form of toxicity (alkali disease) \( (q.v.) \) that is more common and causes problems for the feet. The signs include hair loss, especially in the mane and tail, and sloughing of the hooves. This is seen in particular in areas of seleniferous soils/plants and when supplementation is miscalculated. It has also been suggested that moderately high intakes of selenium, which are not toxic enough to cause the signs described above, may affect the frog horn and be a factor in some cases of persistent thrush.

There is a current legal European limitation to the amount of selenium that can be fed to horses (maximum of 0.5 mg/kg diet at 88% DM) which approximates to around 1 mg/100 kg BW as fed.

Horses are more tolerant than are cattle to excess dietary fluoride and adults seem to be able to tolerate 50 mg fluoride per kg DM for extended periods of time.

**VITAMINS**

**Introduction**

Vitamins are a heterogeneous group of organic substances, essential for life, that have metabolic functions and are not degraded as sources of energy. They are arbitrarily divided into those soluble in fats and fat solvents, and those soluble in water. Most vitamins are represented by more than one compound, which may have different potencies. Certain pro-vitamins exist, e.g. \( \beta \)-carotene and 7-dehydrocholesterol, that do not possess vitamin activity but which can be converted into active vitamins by the body.

The ability of the body to store reserves of vitamins varies. For example, enough vitamin A can be stored by the liver to last for 2–6 mo and reserves of vitamin B\(_{12}\) can last for a year through enterohepatic cycling, whereas reserves of thiamin may be sufficient for only 1–2 wk. The healthy horse, however, requires no dietary source of ascorbic acid (vitamin C) as it is synthesized in adequate quantities from glucose in the liver.

Each vitamin may have many functions with differing demands on reserves. It is possible that greater dietary amounts of certain vitamins than are necessary to sustain life may, therefore, have metabolic advantages under certain circumstances.

**Vitamin requirements (see Table 3.12)**

Similar to other nutrients, vitamin requirements can be affected by a number of factors including age, amount of exercise and reproductive status. However, many are also influenced by the health status of the individual and in particular that of the kidney and the gastrointestinal tract. The need for vitamin supplementation can also depend on external factors such as the type and quality of the diet and the amount of access to sunlight. Today many horses do not have access to good quality green forage all year round and therefore vitamin supplementation is often required. Relatively little research has been carried out into the vitamin requirements of the horse, in particular the amounts needed.
not just to prevent or correct deficiency symptoms but to promote optimal growth rate, health and performance.

**Fat-soluble vitamins: sources and possible clinical signs of deficiency and excess**

**Vitamin A**
The principal source of vitamin A is β-carotene, contained in green herbage and to a lesser extent in carrots, yellow maize and legume seeds. Principal supplementary sources are the partly water-soluble retinyl esters of palmitate and acetate (synthetic). During the winter, herbage contains less pigment and may be deficient in β-carotene. Research has suggested that the use of certain synthetic β-carotenes as the sole source of vitamin A cannot meet vitamin A requirements of horses and is not to be recommended (always check the bioavailability). Heat, light and oxidation can destroy β-carotene content of feed and therefore the content is significantly reduced with storage.

Possible clinical signs associated with deficiency include night blindness, poor growth, excessive lacrimation, reduction in disease resistance and reduced appetite. Clinical signs associated with excess may include bone fragility and malformations, plus there have been suggested teratogenic effects. In adults there is unthriftiness, poor muscle tone, ataxia and loss of hair. Hypervitaminosis A is unlikely to result from the provision of rich sources of β-carotene, such as artificially dried alfalfa, as the horse is thought to be able to reduce the conversion of β-carotene to vitamin A.

**Vitamin D**
Vitamin D is provided as vitamins D₃, through ultraviolet (UV) irradiation of 7-dehydrocholesterol in the skin, and D₂ through exposure of ergosterol, found in plants, to the action of UV light during desiccation after cutting. It occurs in significant amounts only in naturally cured hays. Ergosterol also occurs in other plant forms, e.g. fungi, yeasts, etc. Irradiated yeast is used as a commercial source of vitamin D₂ (ergocalciferol). Commercial supplements of vitamin D₃ and D₂ are widely available. Vitamin D is not believed to be essential for the absorption of calcium in the horse. Vitamin D may be destroyed by heavy metals and alkaline components of feeds.

The dietary requirement for vitamin D is considerably influenced by the extent of exposure to direct sunlight and its angle of incidence. In winter there is almost no UV light at latitudes above 50°. The requirement is also influenced by the degree to which dietary minerals (Ca, P and Mg) are balanced and the extent to which the current year’s sun-cured hay is given.

Possible clinical signs associated with deficiency include skeletal abnormalities in foals and osteomalacia (q.v.) in adult horses. Potential signs of a deficiency can include a decrease in appetite, food intake and growth rate as well as impaired mineralization of growing bone with a reduction in bone ash content, bone cortical area and bone breaking strength. True ‘rickets’ and osteomalacia as seen in other species have not been confirmed in foals or adult horses.

Possible clinical signs associated with excess vitamin D include hypercalcemia, hyperphosphatemia, bone resorption, soft tissue calcification, anorexia
and poor performance. When given in large oral doses, vitamin D₃ is approximately twice as toxic as vitamin D₂.

**Vitamin E**

Vitamin E (α-tocopherol) occurs naturally as several isomers of tocopherol and tocotrienol. The highest potency, as a vitamin, is possessed by the α-isomer of tocopherol. Research has suggested that the d-alpha form may result in higher plasma concentrations than the equivalent amounts of dl-forms. The richer sources are oils of green plants. Wheat germ oil is the richest source, containing 0.85–1.28 mg/g α-tocopherol. Synthetic supplements of α-tocopherol acetate are widely available. Natural sources are believed to be more bioavailable than the synthetic.

Whether athletic performance is improved by high dietary concentrations of α-tocopherol is unproven. However, there is some evidence that exercising animals may benefit from additional supplementation. Large doses accumulate in muscle and liver tissue, and the stability of polyunsaturated fatty acids of tissues is affected by their vitamin E content. Vitamin E is believed to be important for the immune system in general, and intakes of 160 IU/kg DM in gestating mares have been shown to specifically increase the immunoglobulin concentrations of the colostrum and subsequently in the suckling foals.

The requirement is currently considered to be between 1.5 and 4.4 mg/kg BW daily and even up to 6 mg/kg BW in the intensively exercising animal. Therefore, levels of 160–250 IU/kg DM feed have been recommended for the performance horse with an additional 100 IU/100 mL of any supplementary oil. This is higher than the level of supplementation of many rations and may be higher than the total vitamin E content, i.e. natural plus supplementary, of some rations. In particular, supplementation needs to be considered carefully for horses in work, gestating mares, sufferers of the equine rhabdomyolysis syndrome (q.v.), those being fed supplementary oil and those without access to green pasture.

Possible clinical signs of deficiency include vitamin E and selenium responsive myodegeneration “white muscle disease” (q.v.)—a common manifestation of vitamin E and/or selenium deficiency in young foals in particular. Possibly more important potential signs of deficiency are depressed immune status, poor performance, reduced fertility and fetal death. Equine motor neuron disease (q.v.) is believed to be caused by a lack of vitamin E, which may predispose the type 1 oxidative neurons to oxidative injury and death. It tends to occur in horses that are either stabled or have access to dirt paddocks and are only fed grass hay with a high grain ration. However, this is not always the case.

Possible clinical signs of excess have not been produced and are likely to require very high intakes. However, it has been suggested that very high doses may interfere with absorption or utilization of other fat-soluble vitamins.

**Vitamin K**

The horse appears to have no need for supplementary sources as the intestinal bacteria seem to synthesize the menaquinones (vitamin K₂), which are absorbed in adequate quantities. If there is chronic use of sulfonamides, or of other antibiotics, in amounts adequate to disrupt this synthesis then supplements may become necessary. Vitamin K status affects bone metabolism as
well as **blood coagulation** and it has been suggested that the requirement may increase during times of increased bone metabolism, but there is little work to support this in the horse.

It has been suggested that an increased risk of prolonged bleeding may be seen with the ingestion of certain plants, or if hindgut fermentation is severely depressed, or if liver function is compromised (as vitamin K is converted to its active form in the liver).

The plant form is not thought to be well absorbed and is therefore unlikely to cause toxicity; however, grossly excessive intakes of synthetic vitamin K₃ may be toxic and cause fatal anemia and jaundice. Injectable forms may be toxic and have been suggested to have the potential to result in depression, kidney failure, loss of appetite and laminitis.

**Water–soluble vitamins: sources and possible clinical signs of deficiency or excess**

**Vitamin C (ascorbic acid)**

Ascorbic acid is synthesized in adequate amounts by the liver and other tissues from glucose under normal circumstances. It has been suggested that the requirement may increase in times of stress and disease when natural production may not meet demand. Recently it has been shown that ascorbic acid is a key antioxidant in the fluid lining the lungs and that levels are reduced in horses with recurrent airway obstruction (RAO; formally known as COPD, or heaves) (q.v.) as well as with lung inflammation in general. Additional supplementation may be of value under such circumstances. Synthetic forms are sometimes used as supplements but the efficiency of intestinal absorption is very poor for some forms and their stability through processing varies.

The needs of the healthy horse for ascorbic acid are met by tissue synthesis, but horses that have been subjected to trauma, disease (especially of the respiratory system) or major surgery may require additional supplementation.

**Vitamin B₁ (thiamin)**

Thiamin is synthesized in the intestinal tract and is present in natural diets. The richest source is brewer’s and baker’s yeast, containing 150–160 mg/kg. The cereal grains are good sources, thiamin being contained in the scutellum and germ, so that cereal by-products contain 10–15 mg/kg. Synthetic thiamin salts are widely available.

Some feeds may contain insufficient amounts of thiamin, and supplementation to bring the dietary concentration to 3 mg/kg air-dried feed has been suggested to increase growth rate in young stock. Amongst exercising horses 4 mg/kg air-dried feed may be insufficient and it may be prudent to ensure that the level is approximately 5 mg/kg air-dried feed.

Signs suggested to be associated with deficiency include a reduced growth rate and appetite, leading to anorexia and loss of weight, ataxia, bradycardia, missing heartbeats, muscular fasciculations and decreased erythrocyte transketolase activity. Bracken fern poisoning (q.v.) (bracken contains thiaminase) causes nervousness.

As to excess, there have been unconfirmed reports that parenteral doses ≥5 mg/kg BW may have a **tranquilizing** effect.
**Biotin**

Biotin sources differ in bioavailability. Reasonably good sources include brewer’s yeast, oil seed meals (especially rapeseed meal), alfalfa meal and maize grain, although availability in other cereal grains is low. Synthetic supplements are widely used. Biotin is synthesized by gut microorganisms in amounts adequate to supplement the diet for most horses. There is some evidence that certain types of equine hoof defect may be aided by biotin supplementation over many months or years. Biotin in wheat, barley, sorghum and bran is said not to be very available due to phytate binding.

Biotin has been identified as a key component of the metabolic pathways of cornification, and in cattle biotin has been shown to be specifically required for the normal production and cornification of hoof horn tissue. A deficiency of biotin, therefore, has often been related to poor skin, poor coat condition and/or poor hoof quality in other species.

Although evidence of biotin deficiency per se has not been published in the horse (and depressed activity of biotin-dependent carboxylase has not been investigated), there is some scientific research to suggest that when supplemental biotin is added to the diet of horses with poor hoof health, an improvement may be seen in some but not all horses. However, other defects do not respond to biotin and require adequate calcium, protein and possibly zinc. As it is not possible by eye to determine which horses have which type of defect, it may be that a good balanced diet with additional biotin is the initial route forward for horses with problem hooves.

The amounts of biotin that are recommended to be of value vary: levels of 3–4 mg/100 kg BW/day have been recommended in the literature (for a minimum of 6–9 mo). It is, therefore, important to recognize that biotin is not a panacea for all horses with poor hooves but that it may be worth considering for some.

Currently no recommended upper daily intake limit has been set for biotin but it has been suggested that horses are not given >3 times the levels recommended above.

**Folic acid and vitamin B₁₂**

Reasonably good sources of folic acid include brewer’s yeast (15 mg/kg), green forages (alfalfa meal 1–5 mg/kg), wheat bran (0.8 mg/kg), oilseed meals and whole wheat (0.5 mg/kg). Synthetic supplements are widely available. Vitamin B₁₂ is found only in animal products. For foals, fishmeal, milk products and synthetic supplements are used. The foal has the greatest need for dietary sources of folic acid and vitamin B₁₂ as it does not have a fully functioning hindgut for some time after birth. Some evidence indicates a need for supplementation of horses in training with folic acid.

Synthetic supplements of folic acid providing 20–200 mg daily have been used, but there is little scientific evidence as to their efficacy. Suggested levels for folic acid are 0.55 mg/kg DM feed for adult horses at rest and in light to moderate work, approximately 1.1 mg/kg DM in pregnant and lactating mares, and approximately 1.7 mg/kg DM for those in intensive work and young growing horses with the greatest demands. Vitamin B₁₂ levels of 5 μg/kg feed as fed for performance horses and 15 μg/kg feed for young growing horses have also been suggested.
Possible clinical signs of folic acid deficiency include **megaloblastic anemia with macrocytosis**. However, this has not been confirmed for folic acid in the horse. In other species differentiated from vitamin B₁₂ deficiency by the absence of methyl malonyl CoA accumulation but with homocysteine accumulation in the blood.

Signs due to excesses of folic acid and vitamin B₁₂ have not been produced in the horse.

No dietary supplementary requirement appears to exist for other water-soluble vitamins.

---

**PRACTICAL NUTRITION AND PRACTICAL RATION FORMULATION**

**GENERAL ASPECTS**

General information about rations and feeding technique is provided in several specialist books about horse feeding; in this chapter only an overview is given.

Horses are individuals and vary in many of the areas that influence their needs with respect to nutrients and energy. Owners and riders also have individual requirements with respect to the condition of their horse and the nature of the ride, some of which may be influenced by the diet fed. Some horses seem to be “extremely lively” whatever they are fed, others remain stoical, but what might make one horse fat or excitable may be ideal for its stable mate.

Although BW can be a useful guide to requirements, **feed conversion efficiency rates** vary significantly. Daily feed intakes determined as a percentage of BW must be used as a guide only, and adapted to suit the individual’s metabolism. Regular monitoring of BW and condition scoring can be a very valuable tool.

**Appetite**

Horses have a **finite appetite**, which influences what they can be fed in order to meet their energy requirements. This ranges from approximately 1.5% to 3.0% of BW on an as fed basis for most adult horses, although nursing and weaning foals may eat significantly more. On average, most horses eat approximately 2–2.5% BW/day (at 88% dry matter), i.e. 10–12.5 kg for a 500 kg horse (corresponding to approximately 2% of BW on a dry matter basis, i.e. 10 kg DM for 500 kg BW).

**Number of meals**

Feeding rations based on roughage or silage in the stable causes few problems because the ingestion time is long (see Table 3.4). Feeding roughage twice a day is satisfactory. On the other hand, **horses eat concentrate feed very quickly** (see Table 3.3). To prevent disturbances in the stomach (gas production, ulcers) and/or in the cecum (acidosis) and to have the highest energetic utilization of the feed, horses should, ideally, not be fed >0.5 kg concentrate...
feed/100 kg BW/meal. This means that horses (500 kg BW) that need >5 kg concentrate feed/day should be fed three times a day and performance horses with requirements for 8–10 kg concentrate feed/day should be fed four times a day.

**When to feed and water**

There has been considerable debate over the years about when and what to feed horses before they are exercised or at a competition. Should they be fed or fasted and when should the hay be fed in relation to the grain or exercise? Some studies have concluded that, at least in horses undertaking event type work, feeding large amounts of hay along with grain may result in a lowered plasma volume and an increase in body weight, which may be detrimental to performance. Recently it has been recommended that grain should be withheld from many types of performance horses for at least 3 h before exercise, but repeated small quantities of hay should be fed to ensure proper gastrointestinal tract function and psychological well-being.

After physical activity and sweating, horses should have water (before feeding) to restore their water losses. Initially small quantities (not too cold) should be offered soon after intensive exercise and during cooling out. Water may be offered ad libitum to a fully recovered horse. At other times it is not necessary to provide water only before feeding—it should be available constantly.

Protein digestibility may be improved by feeding a small amount of roughage a few hours before feeding concentrates.

**Forage: concentrate ratios**

Forage (fresh or preserved) should be the foundation of any horse’s diet, even those in hard work. Many horses and ponies do not require any other core feed, and those animals with maintenance or low energy needs may be satisfied by feeding plenty of forage at 1.5–2% BW. As energy needs increase, hays with higher energy levels and greater digestibilities should be considered. If this is not sufficient then additional feeds will be needed to meet the energy needs. Protein, vitamin and mineral supplements are likely to be needed to supply the essential nutrients not contained in the forage, or the forage and additional energy source(s), especially if manufactured compound feeds are not being fed.

Those horses in little or no work or those that are extremely efficient feed converters may benefit from being fed lower energy containing roughages but care must be taken that this does not increase the risk of impactions (q.v.).

For the majority of horses, even those in work, at least 50% of the diet, on a dry matter basis, should be suitable forage (approximately 1 kg DM/100 kg BW). Even fit, very intensively working horses should be fed at least 35%, and preferably 40%, of DM intake as forage. This may be as “long” fiber, such as hay, or may be chopped or ground and pelleted. Under most circumstances it appears to be beneficial to provide a significant part of the fiber intake as either “long” fiber or as a long chop. Depending upon the season and land available for grazing, pasture may provide a significant proportion of the
forage requirements. Forage type should not be changed rapidly and poor quality forage should be avoided.

For accuracy of formulation, and particularly where nutritional problems are suspected, the forage must be analyzed. In practical situations where hay is purchased in small quantities, visual assessment of the leaf–stalk ratio coupled with knowledge of the typical analysis for that type of hay may provide a rough guide to nutrient content.

During certain times of the year the fiber levels in grass and other fresh herbage are low, the moisture content high, and the soluble carbohydrate level high. During these periods, restricted access to pasture is necessary for some animals (especially those prone to the equine rhabdomyolysis syndrome and laminitis) if such a nutrient composition is undesirable (q.v.). Where access to pasture is restricted, or animals are being kept on “starvation paddocks” for example, alternative sources of forage of an appropriate nutritional content (plus vitamins and minerals) must be provided to maintain key nutrient intake and to help avoid disorders such as hyperlipemia (q.v.).

Pasture

It must not be assumed that abundant pasture will meet all nutrient requirements. Pastures over the winter months are generally poorer sources of protein, energy and phosphorus. Moreover, with wet winter weather the calcium content can be low. Caution should be exercised where the pasture appears to meet energy, crude protein and fiber requirements (as judged by the animal’s bodily condition) as the mineral and amino acid intakes may in fact be deficient. This is particularly important in brood mares, weanlings and yearlings turned out on abundant pasture. Other minerals, such as copper, zinc, and in some areas manganese and/or selenium, can also be very low.

As well as nutrient deficiencies some further risks should be taken into account, for example very short grass (golf grass) may lead to ileal obstructions while high fructan contents, especially in spring and harvest, may induce laminitis (q.v.).

For horses at risk, such as brood mares, young stock and laminitis cases, herbage analysis should ideally be used to provide the necessary data to indicate whether supplementary minerals and amino acids are required. Additional protein and energy from concentrate feed may not, however, be required.

CONCENTRATE FEED DESIGN AND SELECTION

There are two main options when selecting a concentrate regimen:

1. To use a manufactured compound feed (either cubes, coarse mixes or extruded feeds), or
2. To mix “straights” or individual raw materials and mineral and vitamin supplements to the desired specification.

The latter includes the practice of feeding cereals and a cereal “balancer”.

Compound nuts, coarse mixes and extruded feeds

If a compound feed is used, it is vital that it is chosen to meet the requirements of the horse or pony in question. In most cases, these feeds are designed to be fed as virtually the sole concentrate feed, with hay, water and sometimes additional salt or other electrolytes. Additions may therefore cause significant imbalances. Owners should be discouraged from adding to the selected compound feed, in particular micronutrient supplements, as minerals and vitamins can be fed in excess. Reduced performance and possibly clinical toxicosis may result.

Most compound feeds contain a broad-spectrum mineral and vitamin supplement designed to meet the requirements of a normal, healthy horse for which the feed was designed, when fed at the minimum specified level. If higher levels of feed are required, larger amounts of micronutrients will also be consumed. These amounts are unlikely to be toxic, but this depends on their concentration and what other supplements are being fed. It is usually advisable to change to a feed with a higher energy density that is more suitable to that individual’s requirements.

When the level of compound feed drops below the required minimum, either a lower energy and protein specification ration should be offered (at the required minimum level), or additional micronutrients should be added to the reduced concentrate intake.

“Straight” rations

Many owners still like to mix their own materials. This is often referred to as “traditional feeding”. Unfortunately, much of the “tradition” has been lost or distorted. In addition, the nutrient composition of traditional feedstuffs may have altered as a result of changing agricultural methods. However, it is of course possible to use home mix rations very satisfactorily. Tables 3.6 and 3.12 can be used to help to determine the nutrient requirements of the horse in question. With knowledge of the raw materials, a mixture of these ingredients may then be selected to meet these nutrient requirements. It is likely that a broad-spectrum mineral and vitamin supplement will be required; these are readily available but must be assessed for their efficacy.

Mineral and vitamin supplements

Several mineral and vitamin supplementary feeds are available. The correct dose depends on ration calculation and the requirements of the horse. Most times they are only necessary to complete rations of roughage or roughage and grain. Industrially produced feeds from reputable manufacturers usually contain sufficient amounts of minerals and vitamins (when fed as recommended). Consequently there is normally no need for additive feeding of single minerals or vitamins unless for a specific reason, e.g. biotin and poor hooves.

Specific micronutrient supplements

Many supplements target specific potential deficiency areas or cases where limited research indicates that a higher level of a certain micronutrient may
enhance performance. These supplements include those said to provide higher levels of the antioxidant micronutrients vitamin E and selenium; the so-called “hematonics” which claim to provide elevated levels of the B complex vitamins, and perhaps iron and copper, for their roles in the hemoglobin molecule; or elevated levels of certain microminerals known to be important in musculoskeletal development such as copper, zinc and manganese. Although there may be cases where the use of these supplements is indicated (e.g. biotin for horses with poor hooves, vitamin E and selenium in gestating mares), in most horses they are not necessary.

**Items to avoid**

Items such as lawn clippings, large amounts of **rapidly fermentable feeds** such as apples, or feeds designed for other types of animals should not be given to horses.

**FEEDING THE PERFORMANCE HORSE**

The main specific problems in working horses result from their high energy, water and electrolyte demands.

**ENERGY**

Energy is supplied to the horse via its diet but fundamentally **energy is not a nutrient**. The chemical energy or gross energy contained within feeds needs to be converted into a form of energy that the cells can use for work or movement (usable or **net energy**).

The energy demand, above that of maintenance, required for various physical activities is suggested in Table 3.13. The average estimated requirements for working horses of energy and digestible protein are presented in Table 3.14.

The energy turnover in muscle during maximum exercise is high. According to recent figures, racehorses need 40–60 MJ digestible energy (DE) above maintenance during training or racing. Assuming the maintenance requirement of such animals is similar to that of other animals (see Table 3.14), the total requirement of DE is in the range of 100–130 MJ DE/day. However, individuals can vary considerably in how much energy they require to maintain condition and provide the rider with the type of ride they require, and all diets should be adjusted to the individual.

Tables 3.13 and 3.14 may be useful for estimating the specific energy demands of horses maintaining submaximal speeds over longer distances. For example, horses of 500 kg BW require for maintenance approximately 65 MJ DE; for 1 h walking (including tack and rider of 75 kg: total BW = 575 kg) = 5.75 MJ DE; for 1 h slow trotting, 14.4 MJ DE; for 10 min galloping 14.4 MJ DE, giving a total requirement of approximately 100 MJ DE/day. Cold weather may increase energy needs by 10–15% to maintain warmth. Exercising up hills or under heavy wet or deep soil conditions may also increase energy needs.
The daily DE intake of 3-day event horses (average 600 kg BW) during intense training (2.9 h/day) will be in the range of 150–170 MJ DE, but again many individual animals will not need anywhere near as much energy as this. When horses work very hard for several days the energy requirements often cannot be covered by the energy ingested because their feed intake may be reduced and requirements are high. Such horses mobilize energy from the fat stores in the body. Therefore, before starting long distance rides, horses must be in good condition.

Recent work has suggested that at least in the more difficult endurance rides, thin horses with a condition score (CS) <3 (on a 1–9 scale) might be at a disadvantage because of lower energy reserves, whereas over-conditioned horses could have problems, for example due to the insulating effect of a thicker fat cover.

### PROTEIN

If the energy intake or energy stores are adequate, exercising horses need only small amounts of protein above the maintenance requirements. An additional

---

Table 3.13  Guide to the energy requirements above maintenance for various activities (kJ DE/kg BW)¹

<table>
<thead>
<tr>
<th>Activity</th>
<th>Speed (km/h)</th>
<th>Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Per km</td>
</tr>
<tr>
<td>Walking</td>
<td>4</td>
<td>2.5</td>
</tr>
<tr>
<td>Slow trotting</td>
<td>10</td>
<td>2.5</td>
</tr>
<tr>
<td>Fast trotting/cantering</td>
<td>15</td>
<td>3.4</td>
</tr>
<tr>
<td>Galloping</td>
<td>25</td>
<td>6.0</td>
</tr>
<tr>
<td>Top speed</td>
<td>40–50</td>
<td>Up to 40</td>
</tr>
</tbody>
</table>

¹ Horse and rider.
² Per min.

Table 3.14  Guide to the recommendations for the supply of working horses with digestible energy (DE) and digestible crude protein (DCP). Note that this excludes rider and tack weight. (DCP intake from a ration will be approximately 40–80% of the CP content depending on the diet.)

<table>
<thead>
<tr>
<th>Degree of work</th>
<th>Additional requirements in % of maintenance requirements (MJ)</th>
<th>Kg body weight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>200</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DE (MJ)</td>
</tr>
<tr>
<td>Maintenance</td>
<td></td>
<td>31.9</td>
</tr>
<tr>
<td>Light</td>
<td>Up to 25</td>
<td>38</td>
</tr>
<tr>
<td>Moderate</td>
<td>25–50</td>
<td>44</td>
</tr>
<tr>
<td>Heavy</td>
<td>50–100</td>
<td>56</td>
</tr>
<tr>
<td>Very heavy</td>
<td>~100</td>
<td>64</td>
</tr>
</tbody>
</table>

The daily DE intake of 3-day event horses (average 600 kg BW) during intense training (2.9 h/day) will be in the range of 150–170 MJ DE, but again many individual animals will not need anywhere near as much energy as this.

When horses work very hard for several days the energy requirements often cannot be covered by the energy ingested because their feed intake may be reduced and requirements are high. Such horses mobilize energy from the fat stores in the body. Therefore, before starting long distance rides, horses must be in good condition.

Recent work has suggested that at least in the more difficult endurance rides, thin horses with a condition score (CS) <3 (on a 1–9 scale) might be at a disadvantage because of lower energy reserves, whereas over-conditioned horses could have problems, for example due to the insulating effect of a thicker fat cover.
protein supply should, however, be provided to cover the endogenous fecal nitrogen losses (which are about 3.6 g N/kg DM intake), the nitrogen loss in sweat (1–1.5 g N/kg sweat) and the building of new muscle mass after a period with low activity (bodybuilding).

In exercising horses, the protein intake (digestible crude protein: DCP) per energy unit (MJ DE) can be the same as in horses at maintenance metabolism, approximately 5 g DCP/MJ DE. With increasing energy demand the proportional increase in protein intake will fulfill requirements and maintain protein stores in muscle and organs and, by keeping the ratio of protein to energy at 5:1, even with a very high energy intake (150 MJ DE/day), an excess of protein does not occur (750 g DCP/day).

In exercising horses, especially endurance horses, an intake of >2 g DCP/kg BW/day should be avoided.

WATER AND ELECTROLYTES

As discussed above, sweat contains relatively low levels of calcium (approximately 0.12 g/L), magnesium (approximately 0.05 g/L) and phosphate (<0.01 g/L) but relatively high levels of sodium, potassium and chloride as shown in Table 3.10. There are also small amounts of various trace elements, e.g. iron at approximately 4.3 mg/L and zinc at 11.4 mg/L. However, the main electrolytes lost with sweat are sodium, potassium and chloride.

The water and electrolyte requirements of working horses are mainly related to losses in sweat and via the respiratory tract (water). Some figures for sweat production are presented in Table 3.9.

Horses do not reduce their sweat production even with extreme water deficiency. At maintenance level, horses need approximately 4–5 L water/100 kg BW/day (depending on diet and environment; see also Tables 3.7 and 3.8). The amount required increases proportionally with activity and sweat production (Table 3.9).

The total requirement of electrolytes depends on the amount of sweat produced and its composition. The composition of sweat does not change even with low intakes of electrolytes. Therefore the electrolyte requirement increases proportionally with higher activity and sweat production (Table 3.15).

Losses of calcium and phosphorus in sweat are small (see Table 3.9). Therefore the requirements of exercising horses are only slightly higher than maintenance (see Table 3.15). After a prolonged period without activity, calcium and phosphorus supply should be increased to approximately 20% above the recommended figures to compensate for the losses from the skeleton during inactivity. An excessive calcium intake (more than three times the recommended figures) should be avoided because the absorption of other nutrients (e.g. zinc, copper and iron) may be reduced. Excessive intakes have to be excreted by the kidney and the large intestine.

The magnesium requirement of performance horses increases because magnesium is lost in sweat (see Table 3.15), although it has been suggested that the magnesium concentration of sweat decreases approximately 30–60 min after the onset of exercise. Higher supplies of magnesium compounds have been recommended to improve performance but this measure is open for discussion.
TRACE ELEMENTS

Iron is important for synthesis of hemoglobin and for replacing iron lost in sweat (see Tables 3.10, 3.12 and 3.16). With most rations, the iron intake of horses is adequate. Suboptimal hemoglobin values are usually not due to marginal iron intake. There are possible risks of inducing problems due to excessive iron given either nutritionally or via injection.

In exercising horses, the requirements for copper and zinc increase only slightly because only small amounts are excreted with sweat (see Tables 3.10 and 3.16). Because iodine requirements increase with heat production, and some iodine is lost in sweat, the supply of exercising horses should be higher than during maintenance.

Traces of selenium have been found in sweat and it has been suggested that there may be increased losses via the urine in exercising horses (perhaps dependent on selenium source). The supply of selenium to exercising horses should therefore be higher than for maintenance. Selenium in combination with vitamin E is important for integrity of muscle tissue.

VITAMINS

Fat-soluble vitamins

Vitamin E is important for exercise capacity. The tissue levels of vitamin E should be sufficiently high to prevent production of lipid peroxidation products. To obtain maximal tocopherol contents in various tissues, exercising horses need approximately 4–6 mg α-tocopherol/kg BW/day. Intakes from around 160 IU to 250 IU/kg DM intake are currently typically recommended for performance horses.

Vitamins A and D are not involved in energy turnover in muscle. Intakes higher than those needed for maintenance are therefore not justified (see Table 3.16). Since a moderate excess of vitamins A and D might affect performance negatively, intakes of >5–10 times the maintenance requirement should be avoided.
Vitamin K is sometimes used to prevent exercise-induced pulmonary hemorrhage. There is no evidence to support this practice.

**Water-soluble vitamins**

Racehorses, in comparison with other types of horse, appear to be more susceptible to marginal thiamin deficiency (*q.v.*). It is thought that this occurs as thiamin is required for energy utilization and the racehorse requires such a large amount of energy. Furthermore, the gut synthesis may be lower in racehorses fed low roughage diets. Thus the recommendation of 3 mg thiamin/kg feed for maintenance may not be adequate for performance horses, and approximately 5 mg/kg air-dried feed has been suggested.

It has been suggested that animals in hard work that go “off feed” may be beneficially treated with a thiamin supplement (e.g. brewer’s yeast); however, this is anecdotal. Stabled racehorses may require a folate supplement, but horses on pasture usually do not. Pasture is an excellent source of folate, although weathered pasture may have lower levels. High intakes of biotin (up to 20 mg/day for a 500 kg horse) might only be useful in horses that have a disposition for poor hooves. Doubt must be placed on the practice of giving regular doses of vitamin B₁₂ to horses before racing.

**ANTIOXIDANT SUPPLEMENTATION**

Free radicals are independent yet unstable molecules that contain one or more unpaired electrons and are formed as the result of normal body processes. In particular they are formed as a result of aerobic metabolism, therefore their production is increased with exercise, but they may also be created following injury, disease or exposure to certain environmental factors such as allergens, pollutants or radiation.

---

**Table 3.16** Guide to the recommendations for the daily supply of working horses with trace elements and vitamins (based on allowing between 10 and 12.5 kg DM intake for a 500 kg horse)

<table>
<thead>
<tr>
<th>Trace elements</th>
<th>Per kg total feed DM</th>
<th>Per kg BW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron</td>
<td>50–70 mg</td>
<td>1.0 mg</td>
</tr>
<tr>
<td>Copper</td>
<td>10–20 mg</td>
<td>0.2–0.5 mg</td>
</tr>
<tr>
<td>Zinc</td>
<td>40–60 mg</td>
<td>0.8–1.5 mg</td>
</tr>
<tr>
<td>Manganese</td>
<td>40–60 mg</td>
<td>0.8–1.5 mg</td>
</tr>
<tr>
<td>Cobalt</td>
<td>0.1–0.15 mg</td>
<td>2–4 μg</td>
</tr>
<tr>
<td>Selenium</td>
<td>0.2 mg</td>
<td>4–5 μg</td>
</tr>
<tr>
<td>Iodine</td>
<td>0.15 mg</td>
<td>3–4 μg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamins</th>
<th>Per kg total feed DM</th>
<th>Per kg BW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>4000–6000 IU</td>
<td>80–150 IU</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>400–600 IU</td>
<td>8–15 IU</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>150–250 mg</td>
<td>3–6 mg</td>
</tr>
<tr>
<td>Vitamin B₁</td>
<td>3–5 mg</td>
<td>0.06–0.125 mg</td>
</tr>
<tr>
<td>Biotin¹</td>
<td>0.05 mg</td>
<td>0.001–0.00125 mg</td>
</tr>
</tbody>
</table>

¹ Note that this not the level that would be recommended for specific hoof support, see text.
The phrase “reactive oxygen species” (or ROS) *(q.v.*) is often used to describe all those free radicals and other molecules that are capable of causing oxidative damage and contain one or more oxygen atoms. “Oxidative stress” occurs when the antioxidant defense system cannot cope with the rate of free radical production. Left uncontrolled, the self-perpetuating “chain reaction” production of free radicals and the consequent damage they cause can lead to alterations in the structure of cell membranes, cell DNA and other cellular components, and cause disruption of normal physiologic processes (some of which may be irreversible), i.e. “oxidative damage”.

In recent years, there has been growing awareness of the role of oxidative damage, at least in humans, in both the aging process and the development of a number of diseases or conditions such as asthma, cancer, skin disease, arthritis, muscular atrophy, cardiovascular disease, liver disease and autoimmune disease. It is thought that oxidative damage may also be implicated in similar diseases in animals. Antioxidants, often referred to as “free radical scavengers” (although not all antioxidants act by scavenging free radicals), are the body’s natural defense against oxidative damage. The main dietary antioxidants for the horse are vitamins C, E and β-carotene as well as selenium. It is therefore important to ensure that adequate amounts are provided to all horses, in particular the performance horse.

There is now clear evidence, both in humans and horses, that a mixture of antioxidants (especially from natural sources) may be more beneficial than a single antioxidant in helping to support antioxidant defenses. Feeding a single antioxidant or unbalanced mixtures of antioxidants may lead to antioxidant depletion, imbalance and a diminished antioxidant capacity; if fed in excessive amounts, some antioxidants may act as pro-oxidants.

**FEEDING THE PREGNANT/LACTATING MARE**

**INTRODUCTION**

It is increasingly recognized in the horse, as well as in humans, that nutrition of the mother may have a very important role in the subsequent health of any offspring. The gestation period of the mare commonly lasts 335–345 days but a considerable variation can exist. The pregnant mare should be kept fit throughout pregnancy, but not fat. Even before pregnancy, a lean but healthy barren, or maiden, mare is better where early conception is required. Individuals that are increasing in body weight are thought more likely to conceive.

By the seventh month of gestation the fetus will be approximately 17% of its birth weight. The majority of studies have suggested that embryonic development and fetal growth require no additional nutrients throughout the first 8 mo of pregnancy. Feeding a good balanced diet during this period is still very important. However, fetal growth obviously accelerates markedly during the last three months of gestation and this is a crucial time in the nutrition of the mare. Nearly half the copper, zinc, and manganese accumulation, for example, occurs in the 10th month.

Postnatally, pre-weaning is the period of most rapid growth (approximately 110 kg gain in the first 3 mo in Thoroughbred foals). Maternal effects (birth
weight, milk yield, etc.) appear to be more important than direct genetic effects during the early postnatal period. However, maternal influence decreases later in life and direct genetic effects become the primary influencing factors for growth.

**During early to mid gestation** the mare’s nutrient needs for maintenance and any change in body condition or her own growth as well as any exercise should be met. Good quality pasture may meet energy and protein needs but it is unlikely to meet all the mineral requirements. Acute malnutrition with maternal weight loss greater than 50 kg has been associated with early embryo loss and it has been suggested that levels of nutrition of either half or double maintenance may have adverse effects on early embryo survival.

**Late gestation** is a crucial period in the nutrition of the mare. Mares have varying reserves that they can draw on if intakes during pregnancy are inadequate, but at what stage nutritional imbalances might influence the health of the foal or its health as an adult horse is currently unknown. There is an increased risk of inducing hyperlipemia (*q.v.*) if a pregnant mare is starved. It is recommended that the intake of feed be gradually increased during the last few weeks of gestation so that there is no sudden change in feed intake when the mare starts to lactate. The dam ideally should be kept in the foaling environment for at least 2 wk prior to foaling so that the antibodies produced and passed to the foal via the colostrum are appropriate.

**Birth weight** is important because of neonatal viability. In general, the mature number of cells in many tissues will have been achieved by birth or shortly afterwards, so to a large extent mature weight (apart from fat deposition) is determined by birth weight. It depends much more on the size of the mare than upon the size of the stallion or upon the amount of feed given during gestation. The amount of feed given to the mare during the latter part of gestation will, nevertheless, influence the flow of milk in early lactation.

Abundant good quality pasture can meet the energy, protein, calcium and phosphorus needs of lactation, even though the minimum dietary protein requirement will have risen to 125 g/kg of dry feed. Increases in milk yield have been obtained by providing a third more than this, but it is unlikely that this is desirable. As many horse pastures contain poor quality herbage species, the yield of good quality forage can be short lived and stocking density may be such that the provision of concentrates and hay is necessary.

Lactating mares will consume large quantities of roughage, but if the quality of this is poor, milk yield will suffer. This could be desirable around weaning to encourage the foal in the search of supplementary feeds and more grazing. A deficiency of water, energy, protein, calcium or phosphorus will ultimately bring about a decrease in milk output without altering its composition, so that within limits the foal will remain healthy but small and of slightly abnormal proportions, as for example, physes in the limb extremities may close early.

**During lactation** the concentrate proportion of the feed required will vary according to the individual and the quality of the feed being provided. A very heavy milker may need 1.75% BW as concentrates (non-forage portion) although more typical may be 0.75–1.25% BW, especially if the mare is being kept on good pasture. Typical feed intakes during lactation are approximately 2–3.0% BW on an as fed basis (approximately 90% DM). According to the NRC, mares produce approximately 3% of their BW as milk for the first 12 wk
of lactation and then an average of 2% BW for the next 12 wk. Other studies have suggested that the rates are higher—at around 4% reducing to 3%. Because of this discrepancy it has been suggested that the energy recommendations of the NRC are approximately 10–12% lower than those actually required.

A guide to the nutrient requirements of the breeding mare is given in Tables 3.6 and 3.12. The requirements of mares of other weights can be approximately gauged by interpolation (or within small limits of extrapolation). If the mare is lactating during the early part of gestation then her requirements during that period will exceed those of later gestation. The most crucial nutrients for mares given traditional diets are sources of energy, protein, copper, vitamin E, selenium, calcium and phosphorus.

ENERGY REQUIREMENTS

The first 8 mo of gestation have no practical impact on nutrient needs over those for maintenance unless the mare is growing, exercising or needs to gain condition. Most of the fetal growth occurs during the last 90 days of gestation. Even so, the nutrient drain incurred to sustain normal fetal growth is much less than that for lactation. Nevertheless, as the fetus occupies an increasing proportion of the mare’s abdominal cavity, her capacity for bulky feeds declines during the period in which nutrient requirements increase. This may correspond to an increase in the quality of grazing but, if not, mares should be given forage and concentrates of higher quality than hitherto.

In late pregnancy some allowance for the energy needs of the fetus is needed. It has been recommended that mares in the 9th, 10th and 11th months of pregnancy (3rd trimester) should receive 1.11, 1.13 and 1.2 times the maintenance requirements respectively (NRC). Certain breeds may require more to maintain condition and optimal fetal development.

For a mare in the first 3 mo of lactation typically producing around 3% BW of milk according to the NRC:

\[
\text{DE MJ/day} = \text{Maintenance} + \text{allowance for the energy content in milk, i.e. } [4.184 \times (1.4 + 0.03 \times \text{Body weight})] + [(0.03 \times \text{BW}^* \times 0.792) \times 4.184]
\]

PROTEIN REQUIREMENTS

The protein requirements of the pregnant mare are relatively low; nevertheless, when grass hays form a major part of the diet, supplementary protein is required as such hays typically contain only 5–7% protein. If they constitute 70% of the diet, then the concentrate should contain 16–17% protein for the latter part of gestation. The protein requirements for milk production are considerably higher, but vary with the potential yield at each stage of lactation (see Table 3.6). Where the intention is to reduce, or limit, milk secretion this is best achieved by restricting the total concentrate intake, rather than by lowering the protein content of that concentration.

The ratio of protein and calcium requirements to energy intake are not identical in pregnancy and lactation to those at maintenance, so one cannot

*NB increase to 0.04 × BW if the mare is a heavy milker.
just increase the amount of the basal diet to match energy needs. For many mares a compound, manufactured, appropriately fortified feed (complementary feedstuff) specifically designed for the purpose can be advantageous in the last third of pregnancy and during lactation.

The NRC protein requirements are taken as crude protein intake and calculated for pregnant mares at a fixed proportion of the energy intake, i.e.:

Requirements g/day ca.10.5 g/MJ DE/day or approximately 1.6–1.75 g of CP per kg BW (dependent on the type of ration being fed)

For the lactating mare this is calculated by allowing for the milk protein content and the amount of milk produced (and therefore should vary during the lactating period), but as a guide, in early lactation (up to 3 mo) for a 400–900 kg horse producing 3% BW in kg each day of milk (which is taken to contain 2.1% protein) plus allowing for the fact that CP is used for milk production at around 65% efficiency and that a reasonable quality ration is being fed with a protein digestibility of around 55%:

\[
\text{Protein for lactating mares} = \text{Digestible protein for maintenance} + \text{DP for milk production divided by the estimated CP digestibility} = \text{ca. 2.85 g CP per day/kg BW}
\]

For heavy milkers this will increase to around \(3.45 \text{ g CP per day/kg BW}\).

In late lactation, with a milk yield of approximately 2% BW and a milk protein content of approximately 1.8%, the requirements will be approximately \(2.10 \text{ g CP per day/kg BW}\).

The digestibilities will vary with the diet, being higher than the estimated CP digestibility of 55% allowed for here if more concentrates are fed or high quality protein feedstuffs are used.

**CALCIUM AND PHOSPHORUS REQUIREMENTS**

During the last 90 days of gestation, mares kept entirely on reasonably good quality pasture or on high quality conserved forage containing some 30–40% leafy clover, alfalfa or sainfoin require no other source of calcium and, if the forage contains 10% protein per unit DM, no supplementary protein. The phosphorus requirements, however, amount to approximately 3 g/kg of the total dry diet. Although this would be provided by good pasture, it is frequently not provided by the hay typically given to horses in the UK. This hay normally contains \(<2 \text{ g phosphorus/kg} and a supplement may be needed. However, there is a need to ensure that the overall intake of both calcium and phosphorus is adequate; the ratio is ideally between 1.5:1 and 2:1. Table 3.6 gives a guide to requirements. Full tables are available in textbooks but if the data are based on NRC recommendations it is important to remember that these are minimal requirements.

During lactation, calcium and phosphorus requirements depend on the actual amount of milk produced and the amount deposited within the milk as well as the availability of the nutrient from the diet. For example, heavy milkers produce approximately 4% BW as milk; moderate milkers approximately 3%, and low or late lactating mares approximately 2%. The amount of calcium
deposited in early and late lactation is approximately 1.2 g and 0.8 g/kg milk respectively. Assuming an absorption efficiency of 50%, this would mean a minimum requirement of approximately $0.11 \times \text{BW g Ca/day}$ for a moderately milking mare in early lactation. However, 50% may not be valid for all individuals and all diets, therefore it has been recommended that more optimal levels may be approximately $0.14 \times \text{BW g Ca/day}$.

**WATER REQUIREMENTS**

The requirements of the mare for supplementary water during gestation and lactation are given in Table 3.8.

**SUPPLEMENTARY FEED DURING PREGNANCY AND LACTATION**

Supplemental vegetable fat or oil in the diet of pregnant and gestating mares can be beneficially used as an energy source, allowing for the fact that it provides no additional protein, vitamins (available vitamin E content is variable) or minerals.

Pasture and forage vary considerably in their nutritional content and yet in most pregnant and lactating horses these provide the bulk of the diet. These should ideally be monitored regularly so that the diet can be appropriately fortified for the individual circumstances.

Recent research has suggested that dietary supplementation of vitamin E (total intake of 160 IU/kg DM) and selenium (total intake approximately 0.2–0.3 mg/kg DM) to the mare during the peri-parturient period may beneficially influence colostrum concentrations of immunoglobulins.

It is also suggested that providing adequate copper supplementation during gestation in particular may be advantageous (approximately 15–20 mg/kg DM intake).

**FEEDING THE FOAL AND WEANLING REQUIREMENTS**

Foals of many breeds have the capacity to grow relatively fast, but the rate of growth per 100 kg BW declines continuously from about 2 wk of age. The rate of growth of long bones and of skeletal muscle declines at an even faster rate and an increasing proportion of the gain constitutes fat, which has much higher demands for feed energy. Thus, the dietary requirements for protein, calcium and phosphorus, in particular, decline fairly rapidly as a proportion of the total diet with increasing age (see Tables 3.6, 3.12, 3.17 and 3.18).

By 12 mo of age, Thoroughbred foals reach approximately 60–70% mature weight, 90% mature height and 95% of eventual bone growth. This can be delayed somewhat, without incurring an ultimate reduction, by slightly reducing the rate of feeding. Foals growing at a rapid rate deposit greater quantities of bone, muscle and fat than their slower growing counterparts and therefore need more calcium, phosphorus, lysine, etc.
Energy

The large intestine of the foal is relatively small and so it depends to a greater extent on digestible starch and sugars as sources of energy. Nevertheless it is prudent to stimulate the development of the large intestine and a mature digestive function. Thus high quality leafy hay may be provided in small and increasing quantities as a stimulant.

It is important to allow for maintenance plus growth and exercise, if appropriate, which means, based on the NRC (1989):

\[
\text{DE MJ/day} = \text{DE for Maintenance} + \text{Allowance for growth: where}
\]

\[
X = \text{age in mo and ADG is average daily gain in kg}
\]

\[
= 4.184 \times ([1.4 + 0.03BW] + [4.81 + 1.17 - 0.023X^2] \times \text{ADG})
\]

There is increasing evidence to suggest that the way energy is provided to the growing foal may be important, especially in relation to DOD (q.v.).

---

**Table 3.17** Recommended requirements (based on the NRC, 1989)\(^1\) of DE and CP on a daily basis for growing horses (500 kg mature weight) with varying average daily gains (ADG)

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>Body weight (kg)</th>
<th>ADG (kg/day)</th>
<th>DE (MJ)</th>
<th>CP (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weanling</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>175</td>
<td>0.85</td>
<td>60.0 (14.4 Mcal)</td>
<td>720</td>
</tr>
<tr>
<td>6</td>
<td>215</td>
<td>0.65</td>
<td>63.0 (15.0 Mcal)</td>
<td>750</td>
</tr>
<tr>
<td>6</td>
<td>215</td>
<td>0.75</td>
<td>67.0 (16.1 Mcal)</td>
<td>805</td>
</tr>
<tr>
<td>6</td>
<td>215</td>
<td>0.85</td>
<td>72.0 (17.2 Mcal)</td>
<td>860</td>
</tr>
<tr>
<td>Yearling</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>325</td>
<td>0.50</td>
<td>79.0 (18.9 Mcal)</td>
<td>851</td>
</tr>
<tr>
<td>12</td>
<td>325</td>
<td>0.65</td>
<td>89.0 (21.3 Mcal)</td>
<td>956</td>
</tr>
</tbody>
</table>


**Table 3.18** Recommended minimum requirements (based on the NRC, 1989\(^1\)) of calcium, phosphorus, magnesium, potassium, vitamin A and copper on a daily basis for growing horses (500 kg mature weight)

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>ADG (kg/day)</th>
<th>Calcium (g)</th>
<th>Phosphorus (g)</th>
<th>Magnesium (g)(^2)</th>
<th>Potassium (g)</th>
<th>Vitamin A ((\times 10^3) IU)</th>
<th>Copper (mg/kg)(^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weanling</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>0.85</td>
<td>34</td>
<td>19</td>
<td>3.7</td>
<td>11.3</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>0.65</td>
<td>29</td>
<td>16</td>
<td>4.0</td>
<td>12.7</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>0.75</td>
<td>33</td>
<td>18</td>
<td>4.2</td>
<td>13.0</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>0.85</td>
<td>36</td>
<td>20</td>
<td>4.3</td>
<td>13.3</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Yearling</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>0.50</td>
<td>29</td>
<td>16</td>
<td>5.5</td>
<td>17.8</td>
<td>15</td>
<td>10</td>
</tr>
<tr>
<td>12</td>
<td>0.65</td>
<td>34</td>
<td>19</td>
<td>5.7</td>
<td>18.2</td>
<td>15</td>
<td>10</td>
</tr>
</tbody>
</table>


\(^2\) Higher magnesium levels have been suggested by other workers (see Table 3.10).

\(^3\) mg/kg Cu in the feed on a DM basis. Higher copper levels have been recommended by other workers (see Table 3.9 and text).

ADG, average daily gain.
Replacing traditional diets high in starches and hydrolyzable sugars with ones high in fat and fiber has been suggested to have a number of potential advantages. The feeding of appropriately fortified fat and fiber based diets may help to reduce the incidence of certain digestive and metabolic disturbances as well as resulting in a reduced risk of DOD.

**Protein**

Protein requirements for growth in horses depend to a large extent on the amino acid requirements of the foal, the amino acid content of the feed ingredients and the digestibility of these amino acids. Lysine, as for the mare, is believed to be the first limiting amino acid in typical horse rations for growth and threonine is believed to be the second. The mare’s milk has this quality, as do fishmeal and properly processed soya bean meal.

**Minerals**

The foal has particularly high requirements for calcium and phosphorus and, to a lesser extent, for magnesium, reflecting its skeletal growth (see Tables 3.6, 3.12 and 3.18).

**Vitamins**

The absence of a fully functioning hindgut necessitates a dietary source of B vitamins. Supplements of vitamins A, D₃ and E often should also be provided.

**COLOSTRUM AND NEONATAL FEEDING**

It is essential that the foal receives colostrum during the first 12 h of life, and the absorption of the immunoglobulins depends upon it not receiving other sources of food during that time. Where colostrum is not available from the dam the foal should receive it from another source. The first factor to be determined with the sick newborn foal (q.v.) is its immune status. If immunoglobulin levels are ≤400 mg/dL and the foal is <12 h old, then provision of equine colostrum should be considered and be administered by stomach tube (approximately 2 L colostrum or until immunoglobulin level reaches 800 mg/dL). If the foal is >12 h old, septicemic and/or hypothermic, the specialized absorptive cells lining the intestinal tract will have a decreased absorptive capacity and consideration should be given to providing equine plasma (2–4 L at ≥1600 mg globulins/dL) from a suitable donor or plasma bank. Immunoglobulin levels should be monitored and plasma administration can be stopped once levels are approximately 800 mg/dL.

Foals have low energy reserves and therefore can rapidly become hypoglycemic if undernourished. This is especially the case if they are premature or compromised by infection. The mare should be kept in contact with the foal when possible, even if it is too sick to nurse, so there is a greater chance of
the dam accepting the foal at a later date. There are two advantages if the dam can be milked:

1. The foal receives equine milk/colostrum from its dam.
2. The mare’s milk supply will not dry up.

At birth, much of the large intestine contains the meconium, which is normally completely voided within the first 2–3 days of life. Sucking usually sets up a reflex promoting defecation of this material. If this does not occur, the normal passage of colostrum and milk may become blocked. The foal may then go off suck for an extended period and should be given fluid feed by stomach tube or appropriate IV solutions of glucose and an isotonic electrolyte solution.

SUPPLEMENTARY FEED

Normal foals start to nibble hay and concentrates at 10–20 days of age. If the supply of milk or the amount of grass is inadequate, the provision of a creep feed from this time may well enable a normal growth rate to be achieved. A month or so before the intended weaning time the foal should be given increasing amounts of a dry feed to compensate for a decline in pasture quality and in milk production of the dam and to accustom the foal to the dietary regimen it must expect upon weaning. This should reduce weaning stress. Any product should be adequately fortified with minerals, trace elements and vitamins and it should contain high quality protein.

WEANING

Age at weaning does not appear to influence significantly mature height and weight but nutrition around this time can be crucial. More gradual techniques of weaning seem to be beneficial. If foals are not used to eating solid feed before they are weaned they will show a more prolonged decrease in ADG post weaning which is often followed by an undesirable compensatory growth spurt. NRC states that 200–230 kg weanlings consume up to 3.5% BW but practical work suggests that intakes of approximately 2.5–2.9% BW are more likely. The NRC recommends 30% hay or forage in the ration regardless of the growth rate. Others suggest varying the forage–concentrate ratio depending on the desired ADG; however, it would be recommended that at least 30% of the ration was forage or forage equivalents.

Weaning (q.v.) should entail reducing the supplementary feed of the mare, or removing her from high quality pasture some days before weaning. Foals should have company subsequent to weaning but should be out of sight and earshot of the dam. As a proportion of the diet, requirements for protein and calcium are high at weaning, owing to rapid growth of muscles and bones and their mineralization (see Tables 3.6, 3.12, 3.18 and 3.19). The requirement for these declines markedly over the first year of life as the skeleton matures and as fat deposition replaces rapid muscle growth. Clean water should always be available to the foal and provided in an accessible manner from around 2 wk of age (see Table 3.7).
The yearling’s appetite tends to be approximately 2–3% BW as fed. The maximum non-forage recommended is 60%. On good, lush pastures, only an appropriately fortified vitamin and mineral supplement may be required. Levels of approximately 1–1.7 kg complementary concentrate feed per 100 kg BW may, however, be required, especially for foals receiving grass hay based rations and those on poor quality pasture.

The long yearling’s appetite tends typically to be approximately 2–2.75% BW as fed. It is recommended that the non-forage feed proportion should be a maximum of 50–60% of the total intake, i.e. approximately 1.0–1.35 kg complementary concentrate feed/100 kg BW.

SODIUM, POTASSIUM AND CHLORIDE REQUIREMENTS FOR PREGNANT, LACTATING AND GROWING HORSES

Pregnant, lactating and growing horses probably need approximately 0.1–0.2% of total diet as sodium (on an “as fed” basis). An average of 0.15% may be most useful.

Potassium intakes for the pregnant mare have been given as 0.38 g × MJ intake per day or approximately 0.35% potassium in the total diet at the end of gestation. Early lactating mares require slightly more potassium, depending again on the amount of milk being produced, the level of deposition in the milk and the digestibility, e.g. for early lactation in a moderate milker (=0.05 × BW + (0.03 × BW × 0.7)/0.5) = ca. 0.092 BW. These are minimal requirements from the NRC; more optimal requirements may be approximately 1.3 times these levels.

Recently, it has been recommended that the chloride intakes for pregnancy are 80–82 mg/kg BW/day; early lactation, 89–93 mg/kg BW/day; growth until 6 mo, 93 mg/kg BW/day; and growth 6–12 mo, 85 mg/kg BW/day.
EFFECTS OF DIETARY EXCESSES AND DEFICIENCIES

The young foal has higher requirements for nutrients per unit of feed than other horses.

Excesses

Concern exists that an excess of nutrients, and too rapid a rate of growth, may predispose the foal to abnormalities in skeletal development. Present evidence suggests that amounts of dietary protein sufficient for the foal to attain its potential growth rate have no significant adverse effect as long as the remainder of the diet is adequately supplemented. A moderate excess of calcium, trace minerals or vitamins also does not appear to have consistent adverse effects. Excess dietary phosphorus can, however, contribute to skeletal problems, and an excessive intake of soluble carbohydrate may contribute to the development of DOD (q.v.). High dietary concentrations of soluble carbohydrate should be avoided.

Deficiencies

The most obvious nutritional deficiencies seen in foals at birth are those of selenium, associated with vitamin E and selenium responsive myopathy, and potentially of copper, associated with bony abnormalities. These deficiencies reflect deficiencies in the mare’s diet. In foals receiving inadequate milk, or in poorly fed weaned foals, general condition and conformation are adversely affected by an insufficient intake of protein and energy.

FEEDING THE AGED HORSE

INTRODUCTION

There is little reliable published information on nutritional needs specific to the aged horse. Nevertheless, the old horse is generally less able to assimilate feeds than are healthy younger animals. Badly worn teeth make it difficult to chew and ingest poor quality roughage, and body weight and condition can be difficult to maintain, especially in winter. Ground roughage contained in high fiber pelleted feeds can make a useful contribution.

Maintenance of normal body weight may require some concentrated feedstuffs. Elderly horses, and especially elderly ponies on good pasture, are subject to laminitis and colic. Therefore concentrated feedstuffs should be split amongst several daily feeds, and a feed of good roughage should be given, and eaten, before old horses are first introduced to pasture. Pelleted feeds are often helpful in avoiding colic, especially if they contain adequate and appropriate fiber (approximately 15%), 10–20% grain and approximately 12% good quality protein; vitamins and minerals are as required by other adult horses (see Tables 3.6 and 3.12). It is important to ensure that good quality protein is fed and that an adequate calcium and phosphorus intake is maintained.

The inclusion of cooked cereal grains as well as highly digestible fiber sources can be of benefit. Several high fiber pellets are available which, when
moistened and fed as mashes with additional salt and oil if required (and no contraindications), can be used as part or all of the diet of older horses with poor or limited dentition.

PROBIOTICS

Probiotics are **live microbial feed supplements** fed with the aim of improving the intestinal microbial balance. Much more work is needed in this area since the evidence for their use is currently conflicting. Certainly, **lactobacilli** have been administered to scouring foals for hundreds of years through the practice of allowing milk to sour in the manger. Similarly, fecal material collected from healthy horses has been used as a drench in cases of diarrhea (*q.v.*), as a way of recolonizing the intestinal flora.

When selecting a probiotic product, it is helpful to choose one that most closely resembles the normal range of flora in the intestine, although the use of selective strains may be beneficial under some circumstances. The product needs to reach its target area of the GIT in sufficient numbers to enable it to function effectively.

In recent years, certain **yeasts** have been included in probiotic preparations for their putative role in improving digestibility of fiber and other nutrients, notably phosphorus—in particular when added to cereal (starch) based rations. The apparent increase in digestibility has been suggested to arise at least in part from the yeasts stimulating the required enzyme-producing bacteria rather than from a direct effect of the yeasts themselves. Yeasts do not appear to be maintained in the established flora, and presumably need to be fed continuously to maintain their effect. Recently a yeast culture has obtained EU registration for use in equine diets and there is some evidence for beneficial effects when fed with high starch diets.

NUTRITION AND MANAGEMENT IN THE FIELD

The horse is less efficient at digesting fiber than domesticated ruminants and it therefore tends to seek out **younger herbage**. The capacity of pastures to maintain horses in good condition will depend on the amount of herbage in the pre-flowering state of growth. Studies that have been undertaken with horses weighing approximately 500 kg show that, with access to good quality pasture for 7 h daily, they can consume an average of 5.5 kg organic DM, sufficient to maintain their body weight. The intake of herbage diminishes with increasing maturity of that herbage and horses therefore eat reduced quantities of less digestible material, rather than more to compensate for its lower digestibility. Consequently the milk production of mares and the growth rate of foals and yearlings decline rapidly as pasture herbage matures to the flowering stage.

One hectare (approximately 2.5 acres) of high quality grassland could provide pasture and hay for 3–4 light horses of approximately 400 kg, or 4–6 ponies. Low quality permanent pasture, however, may support only one horse per hectare, and where rainfall is low many hectares may be required to supply the needs of a single horse throughout the year. Where the annual rainfall is
of the order of 61–64 cm, average quality grassland can produce sufficient growth for 2 horses and, when fertilized, for 3 horses per ha, or when just being used as summer pasture potentially up to double the number.

The productivity and feeding value of all pastures varies considerably with season (see Table 3.19), rainfall, soil fertility and ambient temperature. Pasture grasses that are going to seed, and brown-colored summer or winter (frost-affected) pastures tend to be of low feeding value for horses.

**NUTRITIONAL ASPECTS OF METABOLIC DISEASES**

**INTRODUCTION**

A proper assessment of the nutritional status of healthy working or breeding horses is always a difficult matter, but it is far more complex when there is a superimposed medical problem. The relationship of diet to growth and skeletal development of foals and horses, and a series of metabolic problems that commonly affect horses are described below.

**DISTURBANCES OF GROWTH AND SKELETAL DEVELOPMENT**

The necessity for correct nutrition of foals, weanlings and yearlings cannot be overemphasized, both in terms of productivity and longevity of competitive horses. Despite this necessity, few specific details of nutritional requirements are available and optimal growth rates of young horses have not been precisely established. This may be why problems of overnutrition in young animals appear to be much more prevalent than those associated with undernutrition.

The term developmental orthopedic disease (DOD) \( \text{(q.v.)} \) was first coined in 1986 to encompass all orthopedic problems seen in the growing horse and therefore encompasses all general growth disturbances of horses. It is non-specific and the definitions are not uniformly agreed. However, as defined by some, DOD may be taken to include osteochondrosis \( \text{(OC)} \) \( \text{(q.v.)} \), i.e. “a defect in endochondral ossification that can result in a number of different manifestations, depending on the site of the endochondral ossification defect”, one manifestation of which is osteochondritis dissecans \( \text{(OCD)} \) \( \text{(q.v.)} \) of cartilaginous origin; physitis \( \text{(q.v.)} \) acquired angular limb deformities; flexural deformities; tarsal collapse; juvenile arthritis or juvenile degenerative joint disease; bony fragments of the palmar/plantar surface of the first phalanx of Standardbred horses (believed to be traumatic lesions); and wobbler disease or cervical vertebral malformation \( \text{(q.v.)} \).

It has been suggested that the clinical signs occur only after a progression of events that begin with a disturbance in the normal development of the cartilage (sometimes referred to as dyschondroplasia or DYP) leading to OC. At this point physical (i.e. biomechanical) stresses are superimposed, leading to clinical signs. It is also thought possible that the initial defects or lesions may heal or develop into OCD or SBC.

Due to the multifactorial nature of DOD, no single cause is likely to result in expression. Factors that may contribute include genetic predisposition,
biomechanical trauma, and mechanical stress through inappropriate exercise, obesity, rapid growth and inappropriate or imbalanced nutrition. Different combinations may be involved in different cases. Environmental or management factors most likely determine whether expression occurs, i.e. provide the final triggering factor(s).

Various nutrients have been highlighted over the years, in particular energy, calcium, phosphorus, copper and zinc and protein. Many studies have been undertaken and suggested to either support or refute the role of a particular nutrient. However, caution needs to be taken when evaluating and extrapolating from these studies. In a survey in Germany, for example, there was no apparent significant link between the feeding management of foals and the incidence of OC, and no relationship was found between the nutritional status of the mares (in relation to digestible crude protein and digestible energy) and the incidence of OC. However, for a number of reasons (e.g. when stabled, foals were not fed separately from the mares, estimations were used to determine milk and pasture intakes, etc.), the nutritional status of the animals could not be determined accurately.

OVERFEEDING FOALS

Energy

A number of studies have suggested that a high intake of energy may result in an increased incidence of DOD. A significant increase in incidence was for example found when diets provided approximately 130% of NRC energy intake (see Tables 3.6 and 3.17). However, it should be noted that there has been concern that the lesions produced by some of these studies are not directly comparable to those found in the field and many field studies have reported foals being fed much higher energy intakes without an apparent increase in clinical incidence. This may be linked perhaps to the background level of predisposition within the individuals, the nature of the energy being provided and the balance of the diet. It has been suggested for example that diets which intrinsically produce high glycemic peaks, or individual horses that respond to certain diets to produce high glycemic peaks (and subsequent effects on insulin and other hormones) may have an increased risk of developing DOD. Such diets have the potential to establish a feeding–fasting cycle, which is a perturbation from the hormonal patterns likely seen in grazing animals. This in turn may adversely influence bone development as the cyclical changes in glucose and or insulin may influence bone maturation via effects on the somatotropic axis.

In other species, such as the pig, dog and sheep, evidence of increased osteochondral lesions has been found in breeds with rapid growth rates compared with the slower growing breeds. Growth rate depends on both genetics and nutrition. A number of studies have linked rapid growth rates (and often, by inference, excessive feed/energy intake—see above) with an increased incidence of DOD. The connection, however, lacks strength because the chain of events between rapid growth and the molecular events that initiate DOD is a long one. Also, the multifactorial nature of this syndrome means that the connection is not always direct; for example, in some animals a rapid growth rate could be a predisposing factor but expression might only
occur when a number of other factors are superimposed (e.g. as a hypothetical example, those foals genetically predisposed to rapid growth whose dams were fed inadequate copper during gestation and who were subsequently fed an unbalanced diet as weanlings).

Rapid growth may affect bone maturation directly or indirectly due to mechanical overload or disturbances in nutritional supply and hormonal balance. These and other factors associated with rapid growth rates therefore may be involved to a greater or lesser extent in individual cases. However, it is generally recommended that rapid growth rates with high energy intakes should be avoided, especially in animals or breeds prone to DOD.

Despite proposals that excessive protein in the diet causes DOD, this has not been confirmed as a causative factor, nor does it appear to have deleterious effects on growth. However, diets with excessive high quality protein are expensive and therefore wasteful. Whether certain diets that contain higher amounts of insulinogenic amino acids are more likely to adversely affect the incidence of DOD is currently unknown.

In a study of Dutch Warmbloods, the incidence of OC in foals fed high intake with high exercise (HH), high intake with low exercise (HL), low intake with high exercise (LH) or low intake with low exercise (LL) was compared. The researchers found that the HL and LH groups had a significantly higher incidence of OC than the HH or LL groups, the highest incidence being found in the HL group. This suggests that it is likely to be a combination of factors that influence incidence and that neither low activity nor high intakes will always result in an increased incidence. However, the lowest incidence was in fact seen in the HH group, suggesting that, if matched by intake, appropriate exercise may be of value in reducing the incidence of OC.

The nature and type of exercise that should be taken is still under discussion. However, it has been suggested that paddock exercise may be preferable rather than just forced exercise. Several studies have suggested that the time of birth may influence the incidence of particular forms of DOD (positively or negatively) perhaps due to influence of time of year on nature of the pasture, access to land for exercise and the relative hardness of such ground.

Mineral imbalances

Imbalance of mineral homeostasis is another important facet of the pathogenesis of osteochondrosis (q.v.). Foals fed excessive amounts of phosphorus (e.g. four times NRC recommendations, with limited exercise) tended to show lesions of dyschondroplasia in one study, although there may be no clinical signs of nutritional secondary hyperparathyroidism (q.v.) provided adequate calcium levels are fed.

Excessive dietary calcium has been proposed as a cause of hypercalcitoninism leading to osteochondrosis and osteosclerosis (q.v.) but there is no evidence to support this theory in foals. The levels of calcium recommended by NRC appear to be adequate (see Tables 3.6 and 3.18) and supplementation (e.g. three to four times NRC recommendations) does not appear to be detrimental to the skeletal development of foals. Increasing the calcium–calorie ratio to foals fed excessive energy (e.g. 129% DE and 340% calcium of
NRC recommendations) does not appear to be protective as in one study lesions of dyschondroplasia still developed.

No adverse effects of magnesium have been reported in relation to the incidence of DOD. However, little direct work has in fact been done on this in the horse despite the fact that magnesium may compete for copper binding sites and excessive magnesium may inhibit parathyroid hormone (PTH) secretion.

A copper-containing enzyme—lysyl oxidase—is involved in the cross-linking of protein chains in elastin and collagen of cartilage. Disruption of these cross-links due to copper deficiency may result in biomechanically weakened cartilage and increase the risk of DOD. A number of studies have suggested a relationship between copper and DOD. In discussing the possible inter-relationship it is important to consider that:

1. Milk has a low copper content, which does not fulfill the copper requirements of the suckling foal. Normally the foal is born with high copper concentrations in the liver, which it is thought will compensate for the milk. If the mare does not get sufficient copper during pregnancy (15–20 mg/kg BW/day) then the foal is more likely to be born with low copper reserves in the liver which in turn makes it less likely to be able to compensate for the low intake from the milk.

2. If foals are grazing in an area with low copper values in grass (<5 mg/kg DM) without supplementation, then a copper deficiency may potentially occur.

Feeding high levels of copper does not automatically entail a reduction in the incidence of DOD. In a number of studies it has been suggested that early signs of DOD may resolve, although there may be a time component to this. In fact, recently it has been suggested that rather than low copper being a causative factor, an increased availability of copper may support various degrees of reparation. A study in Holland correlated the liver copper status of mares and foals with OC scores. All animals had the same availability of feed but their estimated intakes of copper ranged from 95 to 153 mg/day and resulted in mean foal liver copper levels of 372 mg/kg at birth. No direct link between liver copper status of mares and foals at birth or radiographic evidence of OC development in foals at 5 or 11 mo was established. However, foals with higher liver copper status at birth showed increased recession of lesions before reaching the threshold age of 8 mo. It could be suggested that a slightly better placental supply and greater liver stores at birth helped in the lesion repair process and that a good copper supply during gestation may be advantageous. This conclusion is supported by work carried out in New Zealand that suggested possible advantages of providing supplements to the gestating mare, although further research on a much larger scale is needed in this area.

In conclusion, more recent work confirms that copper supplementation may not be the “magic bullet” suggested by some in the past as far as DOD is concerned. However, reduced copper intake or absorption, especially during gestation, could possibly either be permissive to the development of DOD under certain conditions or reduce the ability to repair lesions.
Horses appear to have a high resistance to **chronic copper toxicity** and it seems they can withstand levels of <800 mg/kg; therefore it seems very unlikely that levels of approximately 20 mg/kg copper in the feed for the mare and foal will have any deleterious effect. No evidence exists that molybdenum interferes with copper metabolism in the horse since the persistent, protein-bound thiomolybdates that occur in ruminants have not been identified.

There have been several reports that industrial contamination of grassland with either **zinc** or **cadmium** has resulted in an increased incidence of DOD (sometimes not confirmed histologically) in foals and young stock, suggesting that increased exposure to zinc and possibly cadmium may result in the development of OCD. However, more research is needed to confirm any possible relationship, to determine what the zinc–copper ratio should be and to ascertain what influence this may or may not have on the incidence of DOD.

There has also been some discussion regarding the nature and distribution of lesions caused by chronic **zinc** or **zinc/cadmium toxicosis** and whether such atypical patterns should be considered in the same way as the more **typical** DOD found routinely in the field. It has been suggested that in fact they should be specifically referred to as “zinc induced osteochondrosis” rather than being “combined under the more general term of DOD”.

**ROLE OF NUTRITION AND EXERCISE**

At present the interrelationship between exercise and feed intake in relation to DOD is far from clear, although there is an indication that there needs to be a balance between these two factors and extremes must be avoided. There also seems to be a “time window” in which the development of lesions may be influenced by both exercise and nutrition either positively or negatively. This may vary with the different joints but is most likely to range from in utero to approximately 9 mo of age.

**NUTRITIONAL MANAGEMENT OF FOALS WITH DOD**

Foals identified as having lesions of DOD must be carefully managed to prevent any exacerbation and to allow healing to be promoted. Three factors affecting foals with DOD have been identified:

1. **Dietary digestible energy level.** Although few experimental data exist, it appears to be beneficial to decrease the DE level to approximately 85% of that recommended by NRC, with 100% levels of crude protein, calcium, phosphorus and other nutrients. Excessive levels of calcium in combination with high levels of DE have not been effective in protecting foals from osteochondrosis and it is unlikely that high levels of calcium assist in the healing of DOD.

2. **Dietary copper level.** Collagen cross-linking may be enhanced when dietary dry matter (DM) contains higher levels of copper (e.g. 30–45 mg/kg). However, collagen cross-linking is probably adequate at lower levels (e.g. 10 mg/kg). In cases of physitis it has been suggested by some that levels of approximately 30 ppm copper in the dietary DM may be beneficial. Since
horses have a high threshold level for copper toxicity, the level can be increased with relative impunity.

3. **Exercise and feeding.** If a foal is diagnosed with osteochondrosis and/or physitis, then the level of DE fed should be decreased and any joint trauma minimized by confining the foal and allowing only hand-walking exercise. However, the lack of exercise will further decrease the foal’s DE requirement and 70–75% NRC DE may then be adequate.

### NUTRITIONALLY ASSOCIATED LAMINITIS

Laminitis is the local manifestation of a serious systemic metabolic disturbance (*q.v*). Laminitis is perhaps most commonly associated with certain feeding and management factors that will increase the likelihood of a potential attack whatever the type or breed of horse. **Grain overload** for example, whether by accident or deliberately induced, increases the risk of developing laminitis.

Administration of a suitably large 85% carbohydrate and 15% **fiber meal** can result in clinical signs of laminitis within 40h; another potential iatrogenic cause is **constant tube feeding** with a high energy–protein ration. Turning certain ponies out onto **lush pastures** in the spring and autumn/fall is a common triggering factor for the development of laminitis. Currently it is thought that the high levels of water-soluble carbohydrates (which include the simple sugars as well as the more complex storage carbohydrate fructans) may be involved in this process. It has recently been shown that giving relatively large amounts of a particular fructan (*q.v*.) can induce laminitis in the horse.

It is thought that, as in other mammals, the horse does not have the necessary enzymes to digest fructans directly within the small intestine. The bulk of the fructans therefore passes into the hindgut where they are readily fermented, in a similar manner to starch that escapes digestion in the small intestine, triggering off abnormal fermentation as discussed above. Under these circumstances any **endotoxins**, together with various other compounds that may be absorbed into the blood, may have further effects, in particular within the feet (not necessarily directly), triggering the development of laminitis. The degree of fermentation within the small intestine when other more rapidly fermentable feeds are fed possibly is also of some importance in this condition.

### Reducing fructan intake

The fructan content of grass varies and is largely dependent upon multiple factors including fertilization, light intensity, ambient temperature, stage of growth, residual fructan accumulation from the previous day, and past and present pasture management regimens. It is usually when energy demands upon the plant are high (e.g. during growth) that the fructan concentration is at its lowest (as it is being utilized to provide energy for growth) and vice versa. It is therefore thought that the safest time to turn out the horse may be late at night, bringing it in by no later than mid morning.
Fructan levels tend to be higher in the stem than the leaf due to the role of storage carbohydrate and therefore **grass management** is important. The amount and nature of the fructan stored vary between grasses; for example, timothy and cocksfoot tend to accumulate larger molecules which may be more slowly fermented in the hindgut than the smaller molecules found in perennial rye grass. The fructan content of hay is likely to be lower than that of fresh grass, and is lower still in haylage due to the fermentation process. Overall, the fructan content of grass is likely to be higher during spring, before the development of the flower, when the fructans are being stored.

Current recommendations to help reduce the risk of laminitis due to high fructan intake include:

1. Turn horses out to pasture when fructan levels are likely to be at the lowest, such as late at night to early morning, removing them from the pasture by mid morning.

2. Do not graze horses on pastures that have not been properly managed by regular grazing or cutting. Try to maintain a young leafy sward approximately 4 cm high. Mature, stemmy grasses may contain high levels of stored fructans.

3. Avoid or restrict turning out in spring (before flower development) and autumn/fall when fructan levels are accumulating and plants generally contain relatively high levels of water-soluble carbohydrates.

4. Do not allow horses to graze on pastures that have been exposed to low temperatures (e.g. frosts) in warm, bright, sunny weather.

5. Avoid grazing horses on freshly cut stubble, e.g. after hay harvest, as plant stems are the storage organs for fructans.

6. Consider zero grazing (while providing the horse with suitable forage) if it is essential for the horse to ingest minimal levels of fructans.

7. Graze horses on pastures containing species that produce low levels of fructan, e.g. timothy.

**Ponies with laminitis**

Prevention is preferred: feed in a similar way to that described for equine rhabdomyolysis syndrome (ERS) (*q.v.*) based on forage with minimal cereals, utilizing oil as an energy source if required (providing there are no contraindications because of liver function). Minimize the intake of fructans as outlined above. As soon as signs of laminitis are noted the animal should be removed immediately from the pasture and treated medically. The hoof should be radiographed for evidence of pedal bone rotation (*q.v.*) and appropriate treatment and support provided. Feed should not be totally restricted as reduced feed intake and stress may promote **hyperlipidemia** (*q.v.*). Pregnant or stressed ponies should probably be fed approximately 80% of their maintenance requirements to decrease the chance of hyperlipidemia.
High levels of carbohydrate in the feed should be avoided; consequently roughage diets are most suitable.

Ponies with laminitis are frequently obese and glucose intolerant. As a result severe vasoconstriction and local ischemia may exacerbate the laminitis. Due to the pain involved there is an increased output of glucocorticoids, which potentiates the digital vasoconstrictive effects of sympathetic catecholamines in blood vessels and further compromises vasodilatation.

**Horses with laminitis**

Horses with carbohydrate (i.e. grain) overload and endotoxemia (q.v.) always have a poor prognosis, which may be further complicated by the onset of laminitis. There is controversy over the feeding of horses suffering laminitis secondary to endotoxemia. Although some veterinarians maintain that the affected horse should be fed minimal amounts of grain and alfalfa, with the predominant ingredient in any diet being grass hay, others suggest that endotoxemic horses with laminitis should be fed in a different manner to obese laminitic ponies, in which feed intake and DE should be dramatically reduced.

Horses suffering laminitis secondary to endotoxic episodes may therefore require a diet providing approximately 100–130% of the normal maintenance rations. This can be incrementally implemented over the course of week. Sick horses that are in a catabolic state may benefit from feeding at 130% of maintenance.

Severely affected horses commonly become potassium depleted, when renal loss of potassium is assessed using creatinine clearance (urinary fractional electrolyte excretion [FE]) ratios. Potassium has a vasodilatory effect and dietary replacement may be beneficial. Grain has a low potassium content, but levels may be boosted by feeding alfalfa hay, molasses or supplemental potassium chloride in the acute stages. Serum electrolyte levels should be monitored every second day. Oat-based diets are also low in sodium and a complete fiber-based pellet/cubed diet may be more rational. If this is not possible a vitamin–mineral supplement may be required. If voluntary feed intake is still low due to pyrexia or painful episodes, then parenteral nutrition should be considered.

Horses with endotoxemia need to be supported medically (e.g. IV fluid therapy, antibiotics, flunixin meglumine) and may benefit from the vasodilatory effects of acepromazine and also from appropriate foot support.

**EQUINE RHABDOMYOLYSIS SYNDROME**

The equine rhabdomyolysis syndrome (ERS: also known as tying up, azoturia, exertional rhabdomyolysis etc.) affects primarily the muscles of horses of apparently any age, breed or gender and results in the partial or complete inability to move. Death can result. Recurrent sufferers are likely to have an underlying susceptibility to the condition, which may then be triggered by one or more factors, usually including exercise, resulting in the clinical signs. The underlying predispositions as well as the triggering factors are likely to differ between groups of sufferers, so the measures that are successful in one individual may not be so successful in another.
Two subgroups with specific and different causes have been identified and others will exist. One subgroup involves a disorder in muscle contractility or excitation–contraction coupling and the other involves a defect in carbohydrate storage and/or utilization (polysaccharide storage myopathy, PSSM).

There is no single procedure or set of procedures (including diet and management) that can guarantee against further episodes of ERS (q.v.). However, appropriate management procedures and nutrition of susceptible animals may help to reduce the likelihood or frequency of future episodes. The actual diet that will be the most beneficial to an individual sufferer will depend on the horse as an individual and what it is being used for (as influences energy needs), as well as its history with respect to tying up.

**Nutritional advice for a horse prone to ERS**

The major proportion, if not all, of the daily intake of feed should be forage—either fresh (pasture) or preserved (hay). If the energy needs of the individual horse can be met by forage alone then it should be fed approximately 100% forage (non-legume based). An appropriate general vitamin and mineral supplement normally is required to ensure adequate overall nutrition.

Horses prone to ERS should not be turned out onto lush, fast growing pastures but prolonged daily periods outside in a sparse paddock are often beneficial. If the horse’s energy needs cannot be met by forage alone then add in some highly digestible fiber sources such as soya hulls or unmolassed sugar beet pulp (appropriately prepared). In addition, either a fiber based complementary manufactured feed (i.e. high fiber, low cereal—a low oat—feed; these feeds tend to be “low energy” feeds and if the horse requires either more energy then consider adding additional supplementary oil) or supplementary oil may be given, remembering to balance protein, vitamins, mineral and trace element intake. It is advisable in all cases to try to ensure that the diet provides a sufficient intake of electrolytes in an adequate and balanced manner. Ensure that sufficient salt is given and that an appropriate electrolyte supplement is given following or during prolonged exercise, especially in hot weather to compensate for sweat losses.

The addition of wheat bran to the horse’s diet should be avoided wherever possible, and large amounts should always be avoided (unbalanced calcium to phosphorus ratio). Certain animals might have an individual alteration in their ability to utilize or absorb certain electrolytes and may benefit from having their electrolyte status evaluated using the fractional electrolyte excretion test (q.v.) followed by appropriate supplementation.

Vitamin E and selenium deficiencies or imbalances (q.v.) are unlikely to be the cause of ERS but they may be permissive or contributory. It is therefore advisable to ensure that all horses, especially those in hard work, are fed adequate vitamin E (at least 160IU/kg DM) and selenium (approximately 0.2mg/kg DM). Energy intake should be reduced during periods of decreased exercise and sufferers should not be fed in anticipation of an increase in workload.
HYPERLIPEMIA

Hyperlipidemia and hyperlipemia (q.v.) are the terms used respectively for the subclinical and clinical disorders associated with an elevation in blood triglycerides and lipids. Hyperlipemia describes the clinical syndrome characterized by depression, anorexia and hepatic failure and is associated with dramatic elevations in blood lipid and fatty infiltration of the liver and other parenchymatous organs.

The syndromes are generally precipitated by stress (i.e. transport, alteration of diet, pregnancy, lactation, change of paddock/stabling and infection). Most hyperlipemic episodes are associated with reduced dietary intake and, in large horses, an azotemic state, whether pre-renal (most common), renal or post-renal, which exacerbates the condition by inhibiting the peripheral removal of lipoproteins. During fasting, the triglycerides of adipose tissue are broken down into free fatty acids and glycerol, which are then released into the blood.

In horses, fatty acids taken up by the liver are oxidized completely to provide energy or are re-esterified to form triglycerides and phospholipids. These either remain in the liver or are released into the plasma as very low density lipoproteins (VLDLs). In horses, ketone formation is limited, therefore lipemia occurs in response to aphagia rather than ketosis as in ruminants.

Fat, unconditioned ponies are much more susceptible to hyperlipemia than large horses because of the innate insensitivity of ponies to insulin. Insulin normally potentiates the peripheral removal of VLDLs and decreases the mobilization of adipose tissue lipid. The condition arises when the animal is in negative energy balance because of depression of food intake or digestion and absorption. This may be associated with a scarcity of feed (e.g. in winter in the northern hemisphere and summer in the southern hemisphere) or inciting stresses such as pregnancy, lactation, parasitism and transportation.

Affected ponies appear dull and anorexic and show rapid loss of bodily condition. The mortality rate can be high if treatment is not initiated immediately. An uncommon presentation of hyperlipemia is related to hormonal imbalance due to a pituitary tumor (q.v.).

The initial treatment regimen involves the reduction of the negative energy balance by improving feed intake. If the animal will not voluntarily increase its intake of palatable feed and pasture, then tube feeding of a slurry of readily utilizable carbohydrates (Table 3.20) or a slurry of a complete pellet must be implemented. An indwelling nasogastric tube is best if the animal will tolerate it. Concurrently, drugs should be used to remove the lipids from the blood. Insulin and glucose/carbohydrate therapy (with supportive amino acids) may be used to curtail lipid mobilization and heparin administration may accomplish an acceleration of triglyceride removal from the blood.

Antipyretics and analgesics should be used if the animal is febrile or in pain. The hydration status should also be carefully monitored to avoid pre-renal azotemia (q.v.), which greatly exacerbates the hyperlipidemic condition. Sodium bicarbonate may be necessary to correct base deficits, which must be assessed by monitoring blood gases and acid-base status. Food intake produces waves of exogenous glycerides which decline so that the delivery of
dietary glucose is reduced and there is increased movement of free fatty acids from the adipose tissue.

**RENAL FAILURE**

Although it is preferable to provide adequate nutrition without excessive protein, it is better to maintain energy intake than to over-restrict protein (dietary crude protein should ideally be <10% DM and of good quality). Palatable food should be offered with options as it is important to maintain appetite. Good quality grass hay, possibly with 1–2 kg of oats or corn, is one suggestion. Calcium should not be over supplemented; therefore alfalfa should be avoided but, again, if this is the only forage that the animal will eat, it is preferable to starvation.

**Vegetable oil** can be added (gradually) if weight cannot be maintained and the serum is not lipemic (e.g. up to 100 mL of corn oil in grain twice a day) but it is necessary to watch out for lipemia (*q.v.*). The blood urea nitrogen (BUN) to creatinine ratios (mg/dL) (*q.v.*) should ideally be maintained at between 10:1 and 15:1 (<10:1 suggests inadequate dietary protein; >15:1 suggests that dietary protein may be excessive and possibly will aggravate the degree of uremia; *q.v.*).

**LIVER DYSFUNCTION**

The most useful form of treatment for acute liver damage (*q.v.*) is good support and feeding. This involves the provision of oral or IV glucose to combat hypoglycemia. In acute hepatitis, fluid therapy may also be indicated. The value of protein-rich diets to control the faulty production of plasma proteins is controversial. However, some have used amino acids, especially mixtures containing leucine and valine, with apparently good results. B group vitamins are also commonly used.

In less acute cases the aim is to maximize small intestinal starch digestion in order to increase glucose availability. Oats or cooked grains should therefore be considered. It has been recommended that high protein diets (especially high methionine and other aromatic amino acids diets) should be avoided but intake levels of lysine and other key amino acids, including the branched chain amino acids, should be maintained. Micronized or steam flaked corn has a relatively low protein level but a good branched chain amino acid

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water (L)</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Dextrose (g)</td>
<td>120</td>
<td>160</td>
<td>200</td>
<td>240</td>
<td>320</td>
<td>320</td>
<td>360</td>
</tr>
<tr>
<td>Casein (g) or dehydrated cottage cheese</td>
<td>120</td>
<td>120</td>
<td>180</td>
<td>240</td>
<td>240</td>
<td>300</td>
<td>360</td>
</tr>
<tr>
<td>Dehydrated lucerne meal (g)</td>
<td>600</td>
<td>600</td>
<td>600</td>
<td>700</td>
<td>700</td>
<td>800</td>
<td>800</td>
</tr>
<tr>
<td>Electrolyte and vitamin mixture (g)</td>
<td>80</td>
<td>80</td>
<td>80</td>
<td>80</td>
<td>80</td>
<td>80</td>
<td>80</td>
</tr>
</tbody>
</table>

Table 3.20 Example of a daily tube feeding schedule that has been used for a 200 kg pony with hyperlipemia. The total diet should be divided into four tube feedings per day.
to aromatic amino acid ratio and a high starch availability. Sugar beet has
been suggested as a potential source of branched chain amino acids.

Alfalfa also has a better ratio than grass hay but it has a higher overall
protein content so the intake should be restricted to perhaps 0.75% of body
weight with supplemental corn to maintain body weight. Also, avoid early
cut hay and haylages, clover in pasture, dried grass products, soya bean meal
and high protein compound feeds. It is necessary to feed little and often
to reduce the metabolic load on the liver. A low fat diet should be fed wherever
possible. It is not known whether providing choline daily is advantageous (it
is a methyl donor and may help moderate hyperlipemia) in the horse. However,
additional B vitamins and vitamin C have been suggested to be of value.

WASTING CONDITIONS

Protein-losing syndromes

Loss of bodily condition with or without depression of appetite is common
in horses and results from many different causes. Significant amounts of plasma
proteins may be lost in urine (i.e. protein-losing nephropathy \( q.v. \)), pleural
and peritoneal fluids, and perhaps most commonly from the gastrointestinal
tract (protein-losing gastroenteropathy [PLGE]; \( q.v. \)). Diets should contain
high amounts of quality protein to assist in replacement, although in cases of
protein-losing nephropathy it is usually best not to increase protein concen-
tration in the diet as it can exacerbate the azotemic condition.

Protein-losing gastroenteropathy

Protein can be lost from the gastrointestinal tract through loss of mucosal
integrity or lymphangiectasia associated with increased lymphatic pressure.
In acute cases a plasma transfusion \( (q.v.) \) is often necessary to provide protein
(although a low concentration) to improve osmotic pressure transiently and
restore vascular volume. The protein requirement is increased in these horses
and if total parenteral nutrition is necessary then the maximum percentage of
protein energy used in solution should be approximately 12%.

In chronic cases, diets with a high quality (i.e. digestibility, amino acid
spectrum and balance) and quantity of protein should be fed (e.g. soya bean
meal, sunflower meal, fishmeal, and alfalfa hay as roughage), unless the horse
is concurrently azotemic. Anthelmintics should also be administered.

Diarrhea/malabsorption syndromes

Diarrhea \( (q.v.) \) results from a multitude of different causes including the
so-called “malabsorption syndromes”. Requirements therefore depend on the
cause, and the optimal ration also depends on the likely underlying cause.
Frequent small meals are usually preferable, however, and probiotics \( (q.v.) \) may
be worth considering. Horses with chronic diarrhea often have PLGE as well,
usually because of decreased mucosal integrity. They therefore require addi-
tional protein. Usually they are also in a state of energy, mineral and vitamin
malnutrition.

One possible diet worth considering is ad libitum alfalfa hay, as well as
a high protein–energy concentrate, plus a vitamin and mineral supplement.
Horses with chronic salmonellosis (q.v.) usually have impaired fiber digestion, and a diet with an increased proportion of grain/concentrate may help maintain body weight. In some refractory cases of chronic diarrhea the best solution is to turn the horse out onto pasture, which may promote normalization of gastrointestinal flora. In other cases of diarrhea removal from pasture may be of benefit.

If the diarrhea is due primarily to small intestinal dysfunction then little or no grain should be fed. Highly digestible fiber is essential in all cases as the volatile fatty acids produced (together with glutamine and aspartate) are primary energy sources for the enterocytes. Sugar beet pulp is a good source of fiber for horses, especially those with an intact large intestine; immature alfalfa may be better if only the small intestine is intact. Avoid overly mature, poorly digestible, hays. If small intestinal fat digestion is impaired for >1–2 wk the fat-soluble vitamins A, D, E and possibly K should be given parenterally.

If the condition is mainly a large bowel dysfunction grain could be beneficial. However, the exact site and extent of pathology is often difficult to ascertain in the live horse making precise recommendations difficult. It is necessary to start slowly and build up to 50% of the ration if this does not exacerbate the diarrhea. A mineral supplement (particularly if not feeding a legume hay) with added oil (up to 20% if introduced gradually and if small intestinal function is adequate) and water-soluble vitamins may also be indicated. Horses with colitis frequently have severe electrolyte imbalances. Monitoring is therefore advisable.

**Hyponatremia and hypochloremia (q.v.)** occur primarily owing to dilution caused by replacement of fluid loss with water. Severe hyponatremia usually occurs in cases of acute salmonelloses. Hypokalemia may occur in horses suffering colitis (q.v.) because of decreased intake and gastrointestinal loss. These horses are also acidotic and require intensive corrective fluid therapy (e.g. isotonic bicarbonate [1.2% solution]). The horses should be offered fresh water and a separate electrolyte drink. Feed should be restricted to grass hay for approximately 2 wk in convalescence as the normal cecal and colonic flora are likely to be at decreased levels. Grain/concentrate may then be slowly and incrementally introduced (e.g. 0.5 kg/wk). A vitamin B complex supplement may also be used for 2 wk.

**Hyperkalemic periodic paralysis**

The aim in hyperkalemic periodic paralysis is to control potassium intake, maintaining it at <1% potassium in total diet (and being consistent in feeding amounts and time). Forage is the biggest source of potassium but as forages vary tremendously according to type, region, irrigation, stage of cutting etc., it is recommended that likely sources be analyzed. In general, grass type forages have approximately half (0.8–1.7% on a DM basis) the potassium content of the legumes (1.5–3%) and the more mature forages have a lower content than those that were rapidly growing.

Satisfactory diets can be made by mixing unmolassed sugar beet pulp, which is low in potassium, with grass hay and a vitamin–mineral supplement. It is best to avoid lush grass, early harvest hay, legumes, alfalfa and soybean meal and sugar beet molasses.