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Advances in Equine Nutrition

Volume I

Edited by

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SOME ASPECTS OF FEEDING THE ENDURANCE HORSE

PER SPÅNGFORS
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Feed the hindgut

The low enzyme activity in the saliva and in the small intestine for the digestion of starch and sugars tells us that the horse's main energy source is not supposed to be sugars. Instead, the primary energy source should be volatile fatty acids (VFAs) from microbial fermentation in the large intestine. These VFAs are either used directly as fuel in the cells or they are transformed to other energy molecules, such as glucose. Longer chain fatty acids can also be derived from dietary fat, which is absorbed mainly from the small intestine.

The horse which is best prepared for demanding tasks, such as endurance races, is the horse with a high microbial activity from beneficial microbes in the hindgut. Ideally, 60-75% of the horse's energy requirement should come from hindgut fermentation. The lower value is sufficient for a racehorse, and the higher value for the endurance horse. You can regulate the proportion of energy from the hindgut by the fibre content in the feed. If you have less than 15% crude fibre in the diet, the horse's digestive tract functions like a pig or a human. With a high fibre content, the horse becomes more dependent on microbial fermentation and VFAs from the hindgut.

Water and electrolyte reservoir

High fibre content in the feed can also have a microbe-sparing effect in the hindgut. When the ingesta passes from the large colon to the small colon, fluid with a high microbe content is squeezed from the solids and re-enters the large colon. This effect is more pronounced when the fibre content in the feed is high. When the fluid is separated from the solids, peptidase, water soluble molecules and small undigested particles are also recycled into the large intestine for further digestion. In that way the fecal losses of valuable nutrients is much less in horses fed fibre-rich feeds. Indigestible solids will leave the horse quickly and in this way stimulate the appetite.

This fluid-barrier also prevents water and electrolyte losses via the faeces. If properly fed, the large intestine serves as a big reservoir for water and electrolytes. If the diet is rich in starch that escapes enzymatic digestion in the small intestine, starch particles will hold the water in the large intestine due to osmosis. Thus it will be

difficult to use the water in the large intestine to compensate for the dehydration that always occurs during an endurance race. In an experiment at my laboratory, 4 horses received equal amounts of energy, by either grass hay (1.5 kg/100 kg BW) or grass hay (0.7 kg/100 kg BW) and oats (0.5 kg/100 kg BW). We collected blood, faeces and urine for analysis. As seen in figure 1, the retained sodium (in percent of the intake) is significantly higher for horses getting the high fibre feed. The retention of potassium, as shown in figure 2, is the same between the two diets. Where sodium goes, water goes. Therefore, we can conclude that a high fibre diet also increases the water reservoir in the large intestine.

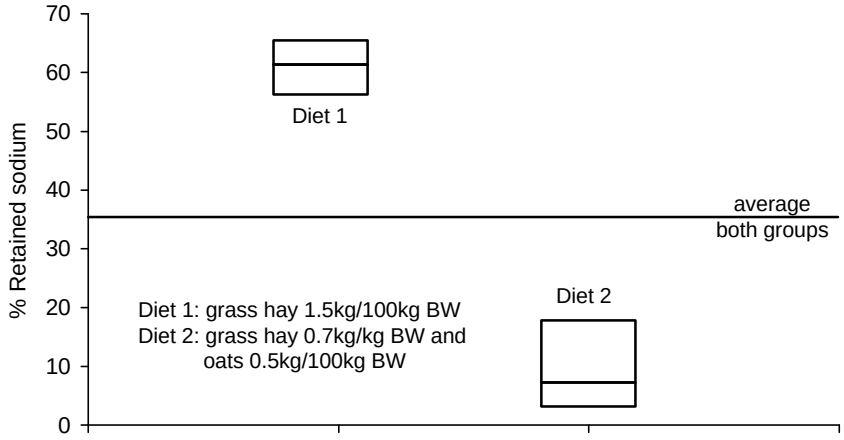

Figure 1. Retained Sodium as a percent of intake

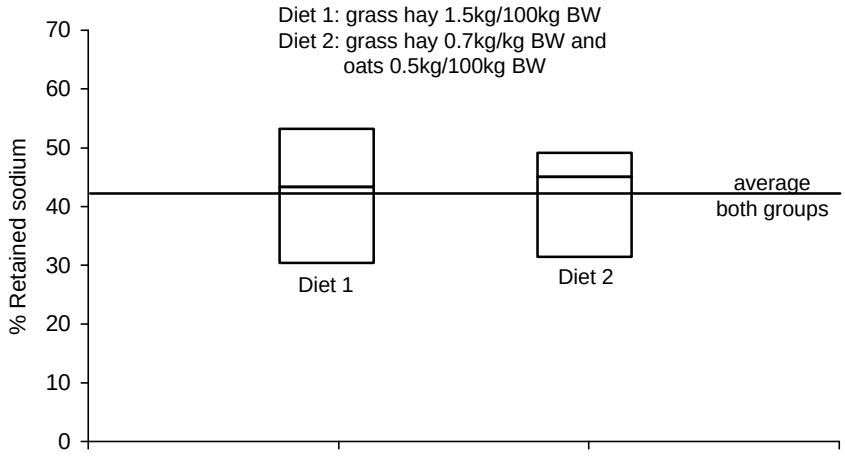

Figure 2. Retained Potassium as a percent of intake

The blood samples showed a postfeeding increase in serum-sodium when the horses were fed oats as shown in figure 3. As the sodium peak occurred 1 hour after feeding, we can assume that the sodium was absorbed in the small intestine. This did not happen when the horses were fed hay. The oats have a higher concentration of starch, that is digested to glucose by enzymes in the small intestine. From research conducted with pigs, we know of a glucose-sodium coupled uptake. This means that when glucose is absorbed through the intestinal wall, sodium is carried along with the glucose molecule. Because of this coupling, which is positive in a piglet with diarrhoea, sodium is absorbed and enters the blood. Unfortunately this sodium, if it is not needed in the metabolism, is immediately excreted in the urine. Orally given electrolytes between meals probably suffer the same fate. At least we can assume this if the electrolyte mixture also contains glucose.

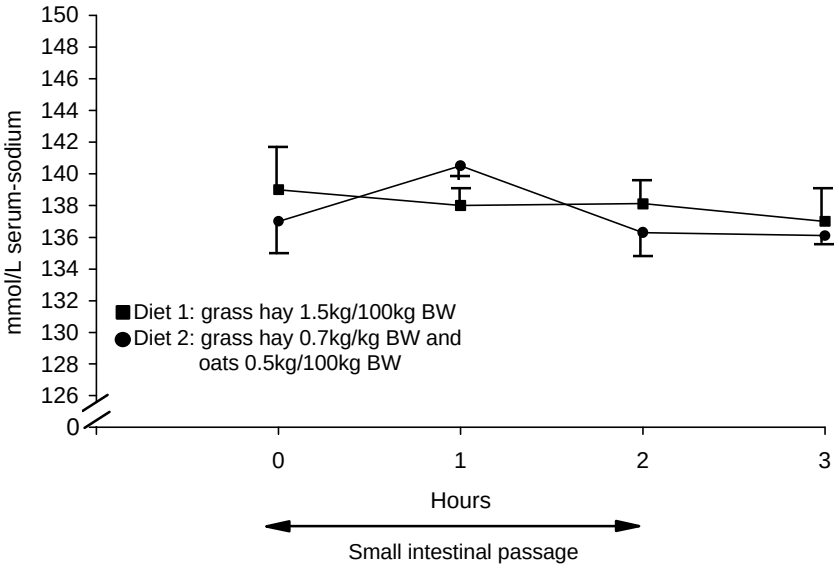

Figure 3. Changes in serum sodium after feeding

If the sodium escapes uptake in the small intestine, as with the hay diet, we create a buffer of electrolytes in the large intestine, which can be used in times of electrolyte depression. This is why the retained sodium is higher when horses get a high fibre diet.

Ideal endurance feeds

From the discussion above it is easy to understand the importance of a good quality hay. In Sweden we use grass hay with a high percentage of timothy and other meadow grasses. We harvest the hay shortly after blooming, in order to increase the

fibre content. We don't want more than 80-90 g crude protein per kg in the hay when fed to competition horses. This will be discussed below. We use metabolizable energy in Sweden when we calculate energy needs and the best hay for horses is a hay with no more that 8.5 MJ metabolizable energy per kg as fed (1.0 Mcal DE/lb).

Fat is a popular source of energy for endurance horses. We use a fluid vegetable fat with 30% linoleic acid and 5-6% in the ration is common. We have found that horses suffering from muscle problems like tying up improve on a high fat/fibre diet. The serum enzymes used for diagnosing the muscle breakdown decrease rapidly and remain low if the horses are kept on this type of diet. This feed holds 11% of the fluid fat mentioned above, and 22% crude fibre with high digestibility. We also add 2000 mg vitamin E per kg. We are now evaluating this feed on a scientific basis.

Fat is not only a source of energy, but should also be regarded as a carrier for fat soluble vitamins and as a source of essential fatty acids such as linoleic acid.

Avoid protein excess

The protein need in an adult horse is very low. The excess nitrogen is a poison and must be made harmless. This energy consuming process takes place in the liver via the formation of urea which is excreted in the urine. When the urea is excreted, water goes with it. Horses on a high protein diet usually have a higher water demand. When urea hits the floor in the box, bacteria convert the nitrogen to ammonia. If horses are kept indoors over night, as is common in Sweden, you can smell the difference between a stable with a high protein diet and one with a low protein diet, when you open the stable door in the morning. This is detrimental for an endurance horse who has to have his lungs in good shape.

During an endurance race, the filtration rate in the kidney is lowered in order to save valuable water. For this reason we always see a linear increase in urea in the blood during endurance competitions. If you start the race with a high urea concentration, soon the level in the blood will become dangerously high which may lead to a number of negative metabolic events.

It is not only the protein level in the feed that affects the urea concentration in the blood. The small intestinal digestibility of the protein source is also important. Protein which escapes enzymatic digestion in the small intestine is transformed by bacteria into ammonia when it enters the large intestine. The ammonia produced in the hind gut eventually reaches the liver where it is converted to urea.

The balance between energy and protein in the feed is also important in order to lower the urea in the blood. We see the lowest urea concentration when we have 5-6 g digestible protein per MJ metabolizable energy.

Fatty acids and glycerol

In a properly fed hindgut, there are nutrients to keep a horse going for days without feed, if you take it easy. But the more speed you demand from your horse, the more

important will the glucose metabolism from glycogen be. Glucose is stored in muscles and liver in the form of glycogen. When you push on and the horse moves more rapidly it gets more dependent on glucose coming from glycogen. The glycogen stores are constantly being refilled, even during work, but it is a slow process, so if the speed is very high, the breakdown and formation of glycogen doesn't keep pace.

If the duration of the work is long, as in endurance riding, you can't feed the horse properly during the race. So the horse must use other sources for the formation of glucose in order to save the glycogen stores. Propionic acid from the hindgut fermentation of fibre is probably the most important precursor for glucose but glycerol from the breakdown of body fat is also important. It seems that the horse has an enormous capacity to form glucose from propionic acid and glycerol.

When we measure volatile fatty acids in plasma after feeding, we never detect propionic acid in venous blood in the resting horse, because it is immediately transformed to glucose in the liver. Postprandial increases of acetic and butyric acid are always detected. As we also measure the hormonal changes in the blood after feeding, we can assume that the postprandial peaks of acetic acid are derived from the feed, and not from endogenous formation in the liver, as seen in the pig. Acetic acid from the hindgut fermentation of fibre and from body fat is also an excellent fuel for muscle cells.

The conditioned horse is also capable of using long chain fatty acids as energy in muscle cells after degradation to shorter carbon units. Sources of long chain fatty acid are dietary fat and body fat.

Feeding strategy is a question of hormones

The most important hormone that regulates energy coming from the small intestine is insulin. Insulin has three important functions. It promotes glycogen formation in the liver, fat storage and protein synthesis in muscles. The net result from these three tasks is that insulin promotes storage and not usage of energy and this makes the horse work intolerant as long as the insulin level is high. Insulin is always elevated after a meal as long as the feed passes the small intestines. The more starch and sugars in the feed, the higher is the insulin peak.

The other important hormone involved in energy metabolism is thyroxine. Thyroxine has many functions in the body, but three of them are very important for the endurance horse. Thyroxine promotes breakdown of glycogen in the liver, the formation of glucose from volatile fatty acids and glycerol, and it increases oxygen consumption in the muscle cells. There are also indications that thyroxine actually promotes the uptake of volatile fatty acids from the large intestine. All of these effects are beneficial for the endurance horse because they promote usage of stored energy.

Thyroxine is therefore an insulin antagonist. We always see a postprandial decline in thyroxine. If the horse is constantly on a high starch diet, the basal insulin level is

high and consequently the thyroxine concentration is low. It is not difficult to realize the detrimental effect if an endurance horse constantly would have low thyroxine levels. To avoid this, the endurance horse must be kept on a high fibre/low starch diet.

The hormonal and energy substrate shift that occurs after feeding, when the feed passes the small intestine, also tells us that you should not feed the endurance horse too close to the start of the race. This is because the feed must enter the large intestine so that the hormonal levels shift back to large intestinal uptake levels, with normal insulin and thyroxine levels.

If we give horses feeds where the main energy extraction occurs in the large intestine, we want the passage time in the small intestine to be as short as possible. Pellets or cubes are the structure of choice because these are consumed faster, and have a faster stomach and intestine passage time. In this way we reduce the postfeeding work intolerance phase. The horse can be fed more often and soon after feeding be ready for training. If you use these feeds it is even possible to feed a tired horse during a long race, rest him for 1-2 hours, and then finish with a fresh horse.

Macro- and micro-minerals

The mineral most discussed in the endurance field is calcium. Nature regards calcium as a very important ion. We can presume this since all mammals have been equipped with two hormones and one vitamin with the mission to regulate the calcium level in the tissues. No other mineral is so thoroughly regulated.

This reminds us of a time in the Precambrian ocean where life began, and calcium was a rare ion. When life came up from the sea it enclosed the ocean inside a cell membrane, and started to look for a way to move that cell. When the vertebrate animals were formed, the calcium ion became the most important extracellular ion. The Precambrian ocean is still, in a sense, in every living cell today and the ions in the cells are called intracellular ions. One of the most important intracellular ions is magnesium.

For a long time the mineral levels have been more or less constant in the soil and hence in the plants growing in that soil. Local variations have been known for generations by the farmers cultivating the fields. But we live in a variable time with heavy industrial influence on the environment. In Sweden the rain-clouds from northern Europe pour acid rain over our meadows. This has changed the mineral concentrations in the fields. Magnesium and many micro-minerals such as copper, zinc, cobalt and molybdenum seem to be the most influenced ions, and their concentrations in the soil are rapidly decreasing while aluminum and cadmium are increasing.

Today we can see deficiency diseases never seen before. As an example, we have just diagnosed a horse with both molybdenum and copper deficiency, which is very contradictory, since in ruminants a deficiency in one of these ions always is associated with excess in the other. This gives us new problems to cope with when formulating new feeds. In this paper, though, we will focus on magnesium.

Magnesium deficiency - a common problem?

Magnesium deficiency is perhaps the oldest known deficiency in animals. Chinese researchers have found eggshells from dinosaurs, 70 million years old. These eggshells contain 30 times less magnesium than normal. These scientists have perhaps found the reason why these animals suddenly disappeared off the face of the earth. An environmental change is postulated as a probable cause.

Magnesium deficiency has been known to appear in horses for a long time, but always together with calcium deficiency. Five years ago we diagnosed the first horse with magnesium deficiency as a solitary finding. Since then a lot of horses have been diagnosed with this deficiency. Unlike earlier finds, the calcium levels in these horses are high.

Magnesium is an intracellular ion. 60% of the magnesium in the body is bound to the apatite crystal in the skeleton while 20% is in the muscles. The muscle store seems to be the primary tissue affected in times of low magnesium intake. Because most of the magnesium in the body is tightly bound to apatite, this pool of magnesium can't be used in times of low intake. Therefore, the horse must meet its daily need of magnesium from the feed. But because calcium and magnesium are chemically alike, both ions use the same uptake and transport mechanisms in the body. We call it a competitive action. But as said before, calcium is regulated by hormones and vitamin D, so magnesium always comes off as a loser. From this we can postulate two theses. Magnesium deficiency can appear when magnesium intake is low or when calcium intake is high and magnesium intake is marginal.

The clinical picture is very variable but one symptom is always present. The horse is work intolerant because of muscle tiredness. Other symptoms that usually appear include hypersensitivity in the skin, muscle tremor, hot temperament when trained, intermittent severe hind leg lameness and paresis with the horse standing up but not able to move, or lying down unable to get up.

The diagnosis of magnesium deficiency is difficult, because serum magnesium only reflects recent magnesium intake, and does not tell us anything about the magnesium content in the muscles. When low magnesium intake has been present for a while, the body tries to compensate for the low intake and shuts off the urinary excretion of magnesium. If that has happened, the serum magnesium is normal to high despite low muscle content and low daily intake. Urine magnesium is a slightly better indicator of magnesium defeciency because low magnesium in the urine tells us that the low intake has been going on for a while. The best way for an accurate diagnosis is via muscle biopsy and analyses of magnesium concentration in the muscle tissue.

There are two ways to improve magnesium content in the muscles. First, of course, you have to increase the daily intake of magnesium. We do this by adding a few grams of magnesium oxide to the diet. At the same time, if possible, we lower the calcium intake if it is excessive. We have found that a calcium/magnesium ratio of 1.5-2.0:1 is ideal for the daily intake, with the higher value for maintenance and the

lower for intense work. The quotient recommended by the NRC is 2.6-2.7:1. Comparisons between recommended daily intake levels of calcium and magnesium in Sweden and the US are shown in figures 4 and 5.

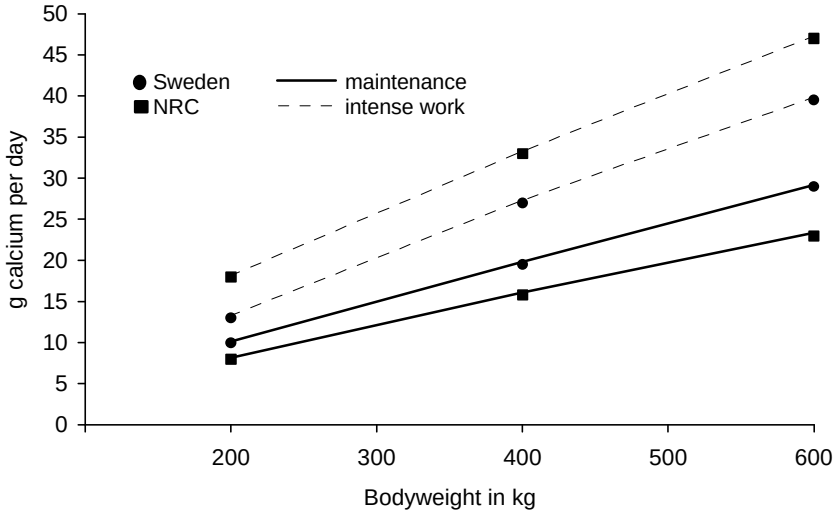

Figure 4. Comparison of recommended daily intake of calcium in the US and Sweden

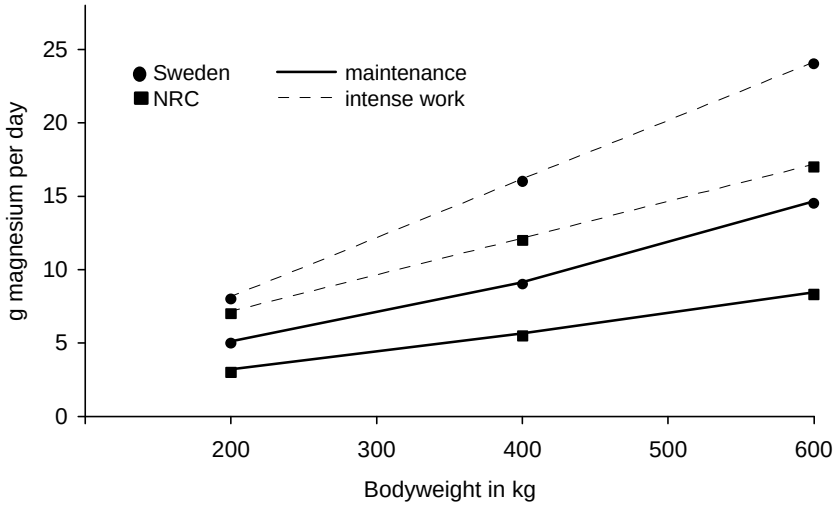

Figure 5. Comparison between recommended daily intake of magnesium in the US and Sweden

Excessive magnesium intake is not recommended. It alters the pH in the intestines and can cause serious problems with metabolic disorders and intestinal formation of concretions. For this reason also, magnesium phosphate should not be used as a feed additive.

Conclusions

Endurance horses are extreme athletes. As in all athletic activity with a long duration in either humans or animals, the feed is the difference between failure and success.

The duration of the energy sources is the vital point. Long time work also means large losses of sweat, and the water balance disturbance that goes with it. Waste products are created in large amounts and must be disposed of. Despite all this, there is a natural way to cope with all these postulations.

Try to provide the hindgut with fuel to keep the microbe activity at an optimum. Avoid large amounts of small intestinal energy, and use moderate protein levels. The protein must have a high small intestinal digestibility. Electrolytes are best provided as a natural part of the feed, in order to build a reservoir in the hindgut. Water must be provided unlimited. Use a structure and composition of the feed to make it pass the small intestines quickly, and feed the horse small amounts as often as possible to avoid large hormone and energy peaks.


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