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Racehorse feeding strategies in Norway.

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ABSTRACT

In this thesis, the topic of thoroughbred nutrition was discussed, the goal was to analyse the diet of a Norwegian thoroughbred stable, in order to see whether the trainer who had no previous education in nutrition had managed to provide a suitable diet for his racing horses. First, a literature review was conducted to obtain necessary information for the feed analysis. The topics discussed was the digestive physiology and anatomy of the horse to better understand the reason behind their dietary needs. Next, a chapter about the most important nutrients of an equine diet, namely the carbohydrates, protein, and fats. The energy production of the body was also discussed here. Following this, a review of function and requirements of vitamins and minerals. After this the topic of the special and intense way of feeding thoroughbreds, and the most common nutritional disorders related to such diet. To analyse the feeding regime of the stable in question, a thorough interview was conducted with the trainer. A study of what ingredients were given and in which amounts. After calculating the total daily intake of each ingredient, PChorse was used for calculation of the daily nutrient supply. The results showed an overall reasonable diet, where most nutrients were fed in excess, except form energy which was lower than expected. This can however be explained by the probable lower workload of Norwegian racehorses compared to international top athletes. The conclusion was that even though the trainer did not have any education in nutrition, he has managed to create a suitable diet for his horses.

Table of contents

1. LIST OF ABBREVIATIONS

- Aa= Amino Acid
- ADP = Adenosine Diphosphate
- ATP = Adenosine Triphosphate
- BCS = Body Condition Score
- BW = Body Weight
- Ca = Calcium
- CH4 = Methane gas
- Cl= chloride
- CO2 = carbon dioxide
- CP = crude protein
- DE = Digestible Energy
- DM= dry matter
- ECF = Extra Cellular Fluid
- EGUS = Equine Gastric Ulcer Syndrome
- FA = Fatty Acid
- GE = Gross Energy
- GI = Gastrointestinal
- M = Maintenance
- Mcal = Mega Calorie
- ME = Metabolizable Energy
- MJ = Mega Joule
- NE = Net Energy
- NEFA = Non-Esterified Fatty Acid
- PSSM = Polysaccharide Storage Myopathy
- RER = Recurrent Exertional Rhabdomyolysis
- SCFA = Short Chain Fatty Acid
- SI= Small Intestine
- VFA = Volatile Fatty Acids
- VHW = Very Heavy Work

2. INTRODUCTION

Thoroughbred racing is quite a widespread sport in the world, having an especially illustrious history in The United Kingdom and the USA. In Norway however, the sport has historically attracted less public interest. There is only one racing track in Norway, located in the outskirts of Oslo. Close to the track, there are approximately 10-15 racing stables. Some trainers are also located in other parts of Norway. In total about 20 professional trainers work with thoroughbred horseracing in Norway.

Nutrition is a critical part of racehorse management in order to be able to extract top performance. In the wild, horses can survive on just grazing alone, however, the racing thoroughbred needs high levels of energy to be able to perform as top athletes. The most important source of energy is glycogen which can be stored in the muscle itself. The glycogen stored in muscle cells are vital to energy production during races or high intensity work. Another important source of energy is fat, this is stored in the adipose tissue, this is mainly used as a source of energy during rest or low intensity work. Starch is an important source of glycogen and can be found in high amounts in cereal grains such as oats. although its tempting to feed large amounts of e.g., oats to a racehorse in order to provide as much energy is possible, care must be taken when formulating the diet of racehorses. Too high levels of dietary starch can lead to severe consequences, not only affecting the horse, but also the performance, and hence the income of both trainer, jockey, and owner. High starch meals may not be fully digested in the small intestine, causing some starch to enter the large intestine where it is fermented. These fermentation products can decrease the pH leading to hindgut acidosis. This form of acidosis can have detrimental effects on the horse, both locally and systemically.

Racing in Norway is mostly similar to racing in the rest of the world, however, as it is a small sport in Norway, the workloads may be different from other countries. The distances raced in Norway range form 900 metres to 2700 metres, on both dirt track and turf. Generally, each trainer has 15-30 horses in their stable, and proper management is important. The general training regime for racehorses in Norway is: 1-2 hours in the walker per day, canter/track work 3-4 days, fast speed workouts once a week, approximately once a week they are ridden in the forests for longer trips. On Sundays they are usually not ridden, most horses get 1-2 hours walking and trotting on the walker, and possibly some hours in the paddock. To become a professional trainer in Norway, one must attend a trainer's course

held by the Norwegian Jockey club. The instructors of these courses are usually other experienced trainers and veterinarians. There is however very little teaching of nutrition and dietetics in these courses, so each trainer must figure out a diet based on own experience and knowledge or hire an equine nutritionist to formulate their diets for them. The topic of racehorse nutrition in a Norwegian racing stable will be discussed further in this thesis. The feeding regime of a Norwegian racing stable will be analysed. The aim is to see whether the chosen stables diet can meet the horses´ nutritional needs at such high levels of exercise, or if there is room for improvement.

3. LITERATURE REVIEW

3.1 GENERAL EQUINE DIGESTIVE PHYSIOLOGY AND ANATOMY:

Horses, Equus Caballus, are non-ruminant herbivores which means they require a plant-based diet. Their digestive tract consists of the oral cavity, oesophagus, stomach, small intestine, caecum, and large intestine. Equidae are called hindgut fermenters, due to the highly developed large intestines that serves the purpose of fermenting and utilising the fibre in their diet. The large intestine consists of the caecum, the large and small colon, and the rectum.

3.1.1 THE ROLE OF THE MASTICATION AND THE OESOPHAGUS:

Adult horses have anywhere between 36-42 teeth, depending on the appearance of canine and wolf teeth. The mare has 36-40, and the male has 40-42 as the canines usually appear in male horses more frequently than they do in females. The 6 incisors are made for grasping forage and feed. 12 premolars and 12 molars have the role of grinding the feed into smaller particles before swallowing, making it easier for the gastrointestinal (GI) tract to digest the feed. Canine and wolf teeth do not have an essential role in mastication and digestion. Mastication in horses consists of three main movements, opening, closing and a grinding motion also called power stroke. It is important to maintain proper dental health, as dental issues can cause mastication problems, leading to a decrease in body weight and digestion problems for the horse.[1, 2].

Another important role of the horse's mouth is saliva production. They have three main salivary glands which are the parotid, sublingual, and the mandibular glands. All three glands produce saliva and secretes it into the oral cavity. The amount of secreted saliva depends on

the mastication of the horse, and the dry matter (DM) content of the feed. Prolonged mastication and high DM content stimulate the production of saliva, thereby increasing the secreted amount[1]. Equine saliva contains mostly water, it has an alkalic pH of around 9 and contains high amounts of Cl and Ca. The enzyme activity of the saliva is low, meaning the main role of the saliva is the lubrication of the feed, and functioning as a buffer to protect against the acidity of the stomach. [1–3]

The oesophagus in equines does not have any other major functions other than transporting feed from the oral cavity to the stomach. It consists of striated muscles in the cranial parts, and smooth muscle closer to the stomach. No secretory glands are located in the oesophagus. [1]

3.1.2 THE ROLE OF THE STOMACH AND SMALL INTESTINE:

The stomach is divided into two main parts, the non-glandular, and the glandular part. The oesophagus opens into the non-glandular part, also known as the cardia. The proximal portion of the stomach is known as the saccus caecus and has no glands [1, 2]. The non-glandular part does not produce gastric juice and has a higher pH (5-7) than the rest of the stomach. As there is no mucous production in this area, it has a lower resistance against the low pH of the acids produced in the glandular part of the stomach, and therefore more predisposed to gastric ulceration [1, 3]. The non-glandular and glandular parts of the stomach are divided by a structure known as the margo plicatus, which contains a mix of cardia and fundic glands [1, 2]. There are two main areas in the glandular part, the ventral glandular fundus, and the pylorus, each with different types of secretory glands. This area has a much lower pH (1-3) than the non-glandular part, but the mucous membranes here are much better protected against the acidic environment. The production of mucous from the neck cells of the gastric glands line the stomach surface and acts as a buffer against the acids [1, 2]. The gastric glands also consist of chief cells and parietal cells. The chief cells produce pepsinogen, which is the precursor of pepsin that helps break down proteins, and gastric lipase that break down lipids. The parietal cells produce chloride and hydrogen ions, creating hydrochloric acid (HCl) [1, 2, 4]. HCl is produced continuously, even when the stomach is empty, this can cause an overflow of acid. If this happens it can reach the non-glandular and less protected area of the stomach, and lead to gastric ulcers [1]. This is one of many important aspects concerning proper feeding regimens and meeting nutritional requirements. In addition to the acid and enzymes of the stomach, there is also a population of important

bacteria that helps the breakdown of feed and nutrients. Studies have shown that lactobacillus is the main species in the stomach, while streptococci have a higher population in the small intestine even though both species are present in both the stomach and the small intestine. [1–3, 5, 6]

The small intestine (SI) commences at the pylorus and end in the ileocaecal junction. It consists of three main parts, the duodenum, the jejunum, and the ileum. In the lumen of the SI, we find the villi and crypt cells. They secrete digestive enzymes and absorb nutrients via a network of blood and lymph vessels, transporting the nutrients to the portal vein or the thoracic duct, respectively [1, 2]. The stomach contents will be neutralised by bile and pancreatic juice, which is secreted into the duodenum through the duodenal papilla. After the duodenum comes the longest section of the SI, the jejunum. It is suspended loosely by mesentery making it mobile and a predisposed area for gut displacements, invaginations and torsions which may to colic [1–4]. After the jejunum comes the short ileum which is characterised by its thick muscle layer and Peyer's patches. It opens into the caecum in the ileocecal fold[1, 2]. The absorption of nutrients occurs at different areas of the SI, such as glucose in the duodenum, calcium and phosphorous in the proximal SI, fat mainly in the jejunum and protein in the distal parts of the SI. [1, 2, 7]

Bile has two main roles, neutralising the acidic chyme and acting as an emulsifier for fats, making them more water soluble which allows lipase to break it down[1]. The pancreas secretes a range of substances important for the digestion of nutrients[1]. Bicarbonate is the main buffer substance that helps neutralise the stomach content and has a pH of around 8. It is secreted continuously, independent of the release of chyme from the stomach. In addition, the pancreas also secretes lipase which break down long chain fatty acids (FA). These FAs are absorbed into the lymphatic vessels of the villi and transported to the thoracic duct. Other pancreatic enzymes are amylase which aids in the breakdown of starch, and trypsin which breaks down proteins. However, the equine pancreas produces very low amounts of trypsin and amylase compared to other species, and research show that bicarbonate and lipase are the two main products released by the pancreas[1, 2]. The brush border enzymes are mainly the disaccharidase, sucrase and maltase which aids in the breakdown of starches and sugars. [1–3, 6, 7].

3.1.3 THE ROLE OF THE CAECUM AND LARGE INTESTINE:

Horses are, as mentioned, hindgut fermenters, meaning they can utilise complex carbohydrates such as cellulose for nutrition. The hindgut comprises 60% of the total GI tract volume and is generally larger than most other domestic animals [1, 2]. The hindgut can digest and absorb plant material that the SI cannot do, due to the diverse microbial population of the cecum and colon. In contrast to the SI, the caecum and colon does not have villi and does not produce own enzymes. It is composed of Lieberkühn crypts and goblet cells and a large population of microbes[1–3]. Anaerobe microbes, bacteria, protozoa and fungi for the most part, produce enzymes that aid in breaking down important nutrients for the horse, the two main species of bacteria are *Ruminococcus Flavefaciens* and *Fibrobacter Succinogenes* [1, 5, 6]. The pH of the hindgut varies depending on when the horse is fed, and when digesta enters the caecum and colon, it averages at around 7, but can go as low as 4,1 when processing feedstuff. The pH also greatly depends on the type of feedstuff given to the horse, experiments show that the pH decreases more when fed a concentrate rich diet, versus forage[1]. Fibre degrading microbes turn fibres into volatile fatty acids (VFA), and as a by-product of this degradation gasses such as CO2 and CH4 are produced. Microbes have amylolytic activity, breaking down starches and sugars, which are hydrolysed inside the bacteria, producing large amounts of lactate. In addition, they have lipolytic activity helping in the digestion of fats[1, 5]. Regarding motility of the hindgut, it is much slower than the SI, and it takes about 24-65 hours for digesta to pass from the ileum to the anus[1]. One of the factors that determines the speed of passage is the DM content of the feed, other factors are the peristaltic motions and the water and gas content of the intestines. Too dry digesta can lead to impactions and colic, the same goes for increased amounts of gas. [1–5]

3.2 GENERAL NUTRITIONAL NEEDS OF HORSES:

In order to decide if any horse is fed adequately, we first need to establish a baseline, or a minimum limit of what a horse requires to maintain normal function. This is called maintenance nutrition, and is what a horse who does no work at all would need to survive. When reviewing these parameters from several articles and literature, maintenance nutritional values are mainly based on adult horses of 500 kg body weight[1, 3, 8]. As 500 kg is generally the mean weight of thoroughbreds in training, this paper will also use 500 kg BW as a standard measure. When calculating the nutritional needs of different horses, we need to know what kind of work the horse does, the amount and intensity of work and its weight. In addition, one must always take the horse´s Body Condition Score (BCS) into

account to ensure healthy maintenance of weight. A variety of work intensity levels are used in different literature, ranging from little to no work, all the way up to very high-intensity work, such as horseracing and long-distance racing. In this chapter, both maintenance and very heavy work (VHW) requirements will be mentioned to give a better impression of the high nutritional needs a racing thoroughbred has.

3.2.1 CARBOHYDRATES

Carbohydrates are one of the main nutrients used for producing energy from either glucose or VFA. Plants contain high levels of carbohydrates, and the main sources of this nutrient in horse feeds are grains and forage [3, 8]. As carbohydrates are one of the sources of energy, there is no recommended daily amount for carbohydrates alone, but for daily energy needs. These needs will be discussed under the energy chapter. When we look at the way horses digest the different types of carbohydrates, it becomes apparent that we can divide them into two main groups, the water soluble starches and simple sugars digested in the SI, and the fibre degraded by bacterial fermentation[1].

The simple sugars and water soluble starches are compounds such as monosaccharides (e.g., Glucose), disaccharides (e.g., Sucrose, lactose) and oligosaccharides (e.g., Starch) [1, 3]. The carbohydrates are hydrolysed in the small intestine by enzymes such as alpha-amylase, glucosidases and galactosidase, cleaving starches into smaller sugars. When these compounds, such as maltose, sucrose and lactose reach the brush border cells, the enzymes maltase, sucrase and lactase cleaves them into free sugars, namely glucose and fructose [1]. The sugars are absorbed by the brush border cells and transported to the portal vein where they are transported to the liver and further distributed throughout the body [1, 3]. The pancreas has two major roles in carbohydrate digestion, the first being the production and release of the main enzyme amylase, and secondly, regulating the blood sugar levels by releasing insulin. Blood glucose levels rises after intake of feedstuff, and as a response insulin is released, making the cells of the body able to take the elevated amount of circulating glucose in for storage as glycogen.

The second category of carbohydrates, the bacterially fermented carbohydrates, are largely derived from the structural components of plants, such as plant cell walls [1, 3]. The plant fibres, cellulose and hemicellulose, are a mixture of several sugar molecules, mainly pentoses and hexoses [3]. Due to its complex structure, the horses´ own digestion is not able

to break hemicellulose down into usable nutrients, instead. The microorganisms of the hindgut ferment them and produces VFAs. These anaerobic, cellulolytic bacteria exist mainly in the cecum, but also in smaller amounts in the colon [1, 5]. Some researchers also claim that such bacteria occur as early as in the stomach; this is however not fully confirmed by experiments [1, 3, 6, 8]. The main VFAs produced by these bacteria are acetate, propionate and butyrate, which are absorbed by the lining of both the cecum and colon, before entering the portal vein and transported to the liver [1, 3]. Propionate is transformed in the liver for glucose production, by the process of gluconeogenesis, while acetate and butyrate are utilised for fatty acid synthesis [1, 3, 8, 9].

3.2.2 FAT

Fats and oils are often supplemented in horse feed for many reasons, the most common being to help horses gain weight and improve the shine of the horse coat. Fats, or lipids, are also a very important source of energy for the horse and can easily be stored in the adipose tissue of the body[1, 3, 4, 8]. Lipids are also important carriers of the fat-soluble vitamins, and act as structural components of cells and precursors for several important mechanisms throughout the body such as being the precursor of prostaglandins [8]. Horse´s muscles have a great aerobic capacity, and can easily utilise FAs for energy during high-intensity work, therefore lipid supplementation is crucial in a thoroughbred diet [8, 10]. As fats are also used for energy, there are no agreed-upon numbers for the daily intake of fats, there are however a few fatty acids that the horse cannot produce itself, these are known as essential fatty acids. Omega 3 and omega 6 are the two groups of essential fatty acids that must be supplemented in the diet, as they have important roles, such as reducing inflammation and retaining cell wall structure[1, 3]. These essential fatty acids are found in most horse feed, especially in pasture grass and hays, and oils such as linseed or flaxseed. Due to the occurrence of these fatty acids in several feeds, it is assumed that the horses needs are met, and therefore there are no recommendation for a daily minimum intake [1, 3].

Horses digest fat in three phases, first there is a mechanical breakdown of the larger particles into finer molecules by chewing and grinding by the teeth, and physical churning in the stomach. This process makes fat more available for enzymatic digestion by emulsifying it into smaller lipid droplets. The second phase, enzymatic digestion, occur both in the stomach and small intestine. As mentioned in chapter 3.1.2, the chief cells of the stomach produce gastric lipase that begins the breakdown of lipid droplets[1]. When the gastric chyme enters

the small intestine, the hormone cholecystokinin is released which activates the secretion of pancreatic lipase. As horses have no gall bladder, bile is secreted continuously into the lumen of the SI, this helps to emulsify the lipid droplets, making them more accessible for other enzymes[1, 11]. The lipase further breaks down the lipid molecules to release the fatty acids from its carrier molecules, this means that triacylglycerol (TAG) is hydrolysed into two non-esterified fatty acids (NEFA) and one monoacylglycerol, in addition, short-chain fatty acids (SCFA), cholesterol and phospholipids are released [1]. The products, except for SCFAs, of this fat hydrolysis form mixed micelles that can easily travel to the intestinal brush border where they are absorbed. The micelle formation is the third phase of lipid digestion. Inside the enterocyte, these products are re-esterified and rearranged into chylomicrons that are released into the lymphatic vessels, and enter the blood stream in the thoracic duct [1]. The SCFAs can avoid the process of re-esterification and transport via chylomicrons, instead, they are able to be absorbed by enterocytes and directly transported into the blood circulation. In the blood, they are bound to serum albumin and transported to the liver, muscle cells or to adipose tissue for storage. [1, 3, 8].

3.2.3 PROTEIN

Protein is a part of every cell in the body, and therefore an important factor for several vital functions throughout the body. Proteins partake in the growth, repair and renewal of tissues, hormone and enzyme synthesis and the regulation of fluid balance[3, 8]. Proteins are also a part of antibodies, the red blood cells as haemoglobin and connective tissue in form of keratin, making them extremely important components for life[1, 3, 8]. All cells produce proteins, which are comprised of long chains of amino acids, these amino acids (Aa) can be produced endogenously or taken up through the diet[3]. The amino acids that are not produced in sufficient amounts endogenously, are known as essential amino acids and must be supplemented through the diet. For a cell to produce a protein, all necessary Aa´s must be present simultaneously, the Aa that is present in lesser amounts is known as the limiting amino acid. In equine feeds, lysine is generally the first limiting Aa, and therefore special attention must be paid to its amounts when creating an equine diet[1, 3, 8].

Green forages and grains are the two main sources of protein in equine diets. The protein is mainly digested in the foregut, starting in the stomach, and continuing in the SI. The parietal and chief cells of the stomach produce HCl and the zymogen pepsinogen. The HCl both activate the pepsinogen into pepsin and unwinds the complex formation of proteins[1, 3, 8].

When the proteins are unwound, the pepsin cleaves peptide bonds, creating peptide chains. These chains enter the small intestine, where pancreatic peptidases further cleave these chains down to oligo -, tri-, and di-peptide chains. When the short peptide chains arrive at the brush border for absorption, a membrane-associated aminopeptidase breaks them apart to form single AAs which are absorbed through several different mechanisms. After they are absorbed by enterocytes, they are released into the portal vein and transported to the liver. [1, 3, 8].

3.2.4 ENERGY

When discussing the energy need of thoroughbreds, we first need to define it, and where it comes from as it is not an ingredient or a single compound in feedstuff. A simple definition is that "Energy is the ability to do work" [3], and is derived from several nutrients such as carbohydrates, fats and proteins. In the intestines feedstuff such as hay, cereals and concentrates are degraded into smaller molecules, and the three main molecules contributing to energy production are amino acids, volatile fatty acids and glucose which are all absorbed into the portal vein and transported to the liver where they are processed. Adenosine triphosphate, ATP, is the main source of chemical energy in the cells and is the final product of these processes[3, 4, 8]. Energy is produced when the bonds of the ATP are split, resulting in adenosine diphosphate (ADP), one phosphate and energy[3]. For ADP to return to ATP, it must go through the process of oxidative phosphorylation, where ADP gains a new phosphor molecule. The ATP storage of the horse is low, only enough to maintain vital functions at rest. When the horse begins to move, the energy requirement increases, and the production of ATP increases. [1, 3].

The energy content of feeds can be quantified in several different ways. The use of the different systems depends on several factors such as what type of energy is quantified and the country of origin of the feed analysis, as different countries use different systems[1, 8]. The total amount of energy in a feedstuff is described as gross energy, GE, however, the horse's digestive tract may not be able to extract all this energy, and some will be excreted. The GE of the feed that is digestible by the GI tract is therefore named digestible energy, DE. This value is calculated by removing the faecal energy output from the GE. If we also subtract the gas and urinary losses, we get metabolisable energy, ME, which accounts for around 80-90% of the DE, depending on the feed source. The last category is net energy, NE, represents the amount of ATP that can actually be produced from the given feedstuff.

The calculation looks like this: “NE= ME x k, where k represents the efficiency in conversion of energy substrates to ATP” [1]. In general, DE is the most commonly used system and will be used in this thesis. [1, 3, 8]. In addition to using an energy basis, we also need to be able to quantify the energy numerically, in energy units. The two most common energy units are mega calories (Mcal) and mega joule (MJ), where Mcal is more commonly used in America, and MJ is mainly used in Europe. In this thesis, MJ will be used.[1, 3, 8]

Glucose is the main source of energy, especially important for red blood cells and brain cells. The main dietary source of glucose is carbohydrates that can be found in feeds such as grass and hay[3, 8]. Glucose can either be transformed to its storage form – glycogen in the liver or transported to other cells via blood vessels. In the muscle, glucose can be used directly, or again transformed to glycogen for storage. Glycogen is a series of glucose molecules attached together, which is transformed back to glucose when energy supply is needed. The glucose is transformed to pyruvate through a process called glycolysis, from here one of two reactions may occur. In the absence of oxygen an anaerobe reaction occurs in the cytoplasm, reducing the pyruvate to lactic acid and ATP. Contrastingly, if oxygen is present, pyruvate is transported into the mitochondria and is converted to a acetyl Co-A. this compound then enters the Krebs cycle, which produces CO2, water and ATP. [3, 4, 8].

As mentioned, fat is an important source of energy. After absorption, fatty acids, triglycerides and glycerol can be stored in either adipose tissue, myositis or very low-density lipoproteins (VLDL) circulating in the blood [3, 4, 8]. When energy is needed, these compounds can be released and transported to several sites for energy production. Glycerol can be transformed to glucose through gluconeogenesis in the liver [3, 4]. The fate of glucose in energy production is explained in the previous section. The Krebs cycle, which produces ATP in the mitochondria of cells, begins with the substance acetyl CoA [4, 12]. Fatty acids can be transformed to acetyl CoA through beta-oxidation, and therefore serve an important role in the production of energy[4, 13]. Proteins can in certain circumstances also be used for energy, if the horse is in a negative energy balance. Different amino acids can enter the Krebs cycle by transformation directly to acetyl CoA or, similarly to glucose, first transformed to pyruvate and then acetyl CoA. [1, 4, 12, 14].

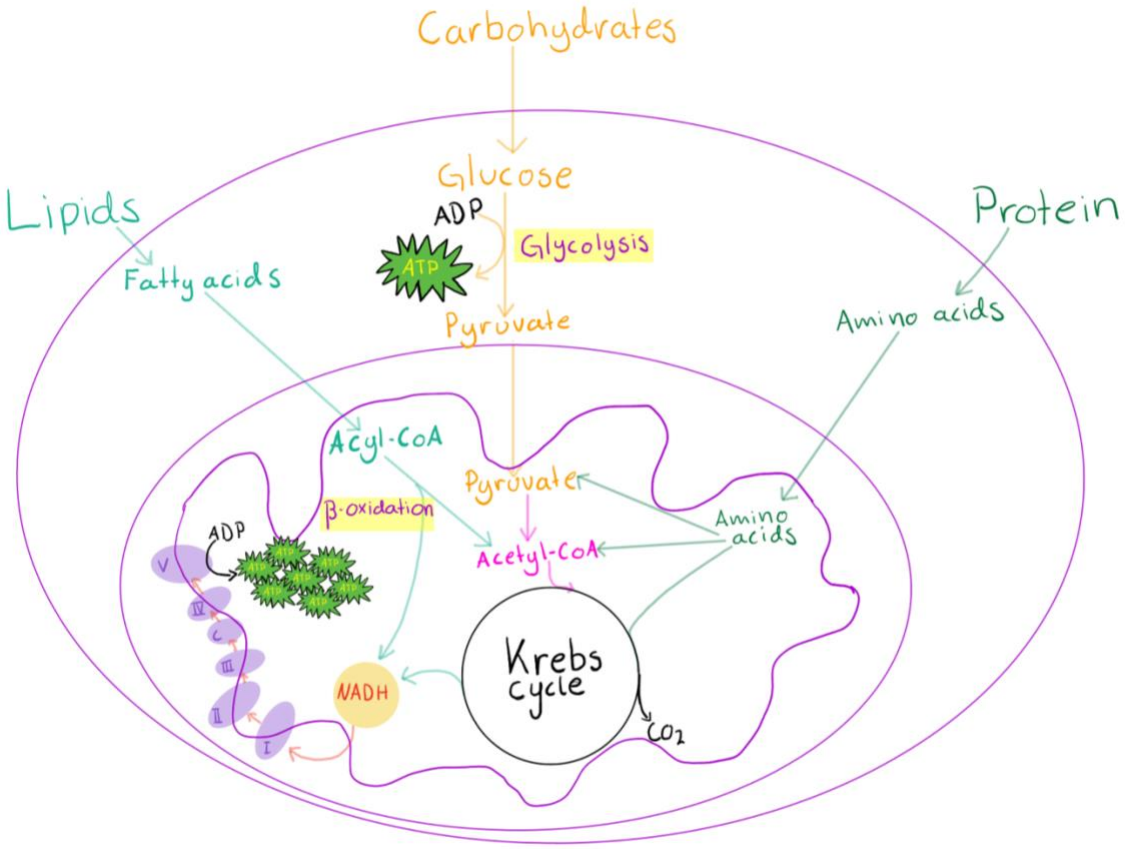


FIGURE 1: Depiction of the energy production in the cell and mitochondria from different nutrients.

Table 1. Energy, protein, and lysine requirements.

	DE (MJ)	**CP (g)**	**Lysine (g)**
Maintenance	76,15	720	31
Very hard work	144,35	1004	43,2

3.2.5 VITAMINS

Vitamins are micronutrients essential for most processes within the body and are an important part of any diet. In horses, the two most important ones to supplement in the diet are vitamins A and E, as horses are not able to produce them themselves. Most other vitamins are produced by microbes in the hindgut, or through other processes in the body. When talking about vitamins, there are several different ways to quantify their amounts. The two most commonly used methods are international units per kilo bodyweight, IU/kg BW, and milligrams per kilo bodyweight, mg/kg BW. International unit is "An international agreed unit of vitamin potency. This is a unit of measurement for the amount of vitamin substance, based on measured biological activity or effect." [3]. Vitamins are divided into two main

groups based on their solubility. The fat-soluble group consist of vitamins A, D, E and K, while the water-soluble vitamins are the various B vitamins and vitamin C.

3.2.5.1 FAT SOLUBLE VITAMINS

Fat soluble vitamins are, as the name implies, dependent on fats or lipids for absorption in the intestines and transport through the body, it is also stored in fatty tissues and the liver, and therefore cannot be excreted through the urine. As they are more difficult to excrete, vitamin toxicity or overdoses are more common with this group.

Vitamin A is one of the vitamins that must be supplemented in a horse's diet, as they cannot produce it endogenously. Carotenoids are a group of compounds found in various feedstuffs such as carrots and greenery, that can be transformed into the active form of vitamin A, retinoic acids. These compounds play an important role in the maintenance of night vision and the integrity of connective tissue. The recommended amount of vitamin A in a horse´s diet for maintenance is 30 IU/kg BW[3, 8, 15].

Vitamin D is one of the vitamins that a horse can produce on its own, this is done in the skin when exposed to sunlight. The vitamin is also found in dried meadow hays. This means that a horse kept on pasture or in a paddock during the daytime should be able to produce enough endogenously. However, stabled horses and horses kept in the northern hemisphere during the fall and winter months may need supplementation. The role of vitamin D in most mammals, is to maintain the calcium homeostasis by controlling the absorption from the intestines and the mobilisation from bones and other tissues [3, 8, 16]. Ca acts as a biologically active substance and is synthesised into its active form by the kidney [1]. However, it has been proven that Ca in horses is not synthesised into its active component to the same extent compared to other mammalian species[1]. There is not a lot of viable research done regarding the maintenance requirements for vitamin D, but the NRC[8] suggests 6,6 IU/kg BW. [3, 8].

Tocopherol, or vitamin E, is the other vitamin that horses depend on getting through their diets. This vitamin acts as an antioxidant mainly in cell membranes, protecting polyunsaturated fatty acids and other sensitive cell wall components from oxidative damage. The antioxidant effect is important for protecting several mechanisms of the body, for example, the immune system due to the immune cells´ large amount of polyunsaturated fatty

acids. It also protects many parts of the reproduction from damage by reactive oxidative species, such as embryo maturation and spermatogenesis. [16, 17]. In racehorses, this vitamin is especially important as it protects the muscle cells against the increased oxidative damage that is related to high workloads. For maintenance, 1IU/kg BW or 500 IU/day is recommended. [3, 8, 18].

The final fat-soluble vitamin is vitamin K, this is a group of compounds such as phylloquinone and menaquinone, who is mainly produced by the microorganisms of the hindgut, in addition it is also found in hay and grass, so supplementation is rarely needed. Vitamin K is transformed to a water-soluble state before storage in the liver and can therefore be excreted by urine, and toxicity is therefore rarely seen. The main function of vitamin K is that it acts as a cofactor in the synthesis of blood clotting factors, it also contributes to bone development and growth [3, 8]. The maintenance value for this vitamin is not determined, as the horses own production and hay intake is likely enough to maintain the horse's needs. [3, 8, 16]

3.2.5.2 WATER SOLUBLE VITAMINS

The water-soluble vitamins are a much less researched group in terms of the supplementation and requirements of the horse. This group consists of the vitamin B complex (B1, B2, B3, B5, B6, B7, B9 and B12), and vitamin C. These vitamins are mainly produced by hindgut fermenting microorganisms or in the liver and exist in most feedstuffs. This may be one of the reasons behind the lack of information on the amounts to be supplemented. As they are water-soluble, they are easily excreted via the urinary system and are not stored in the body like the fat-soluble group is, except for vitamin B12 which is stored in the liver. Therefore, the horse greatly depends on its endogenous production and regular intake of such compounds. A horse with hindgut imbalance, anorexia or treated with antibiotics harming the microorganisms may need additional supplementation. [1, 3, 8, 16]

In the vitamin B complex, there are only a few that have an agreed-upon value for maintenance nutrition. Vitamin B1, or Thiamine, is required by several enzymes involved with carbohydrate metabolism, and aids in the production of ATP. [8, 18]. It is proven that there is an endogenous production of thiamine in the large colon of horses, however studies have shown that the horses own production is not enough to avoid deficiency symptoms [8]. Cereal grains such as oats, wheat and barley contain high levels of the substance, and a horse

fed with grain daily rarely needs B1 supplements.[8, 18]. Research shows that supplementation of 0,06 mg/kg (30mg/day for a 500kg horse) is sufficient for maintenance[3, 8, 18]. Riboflavin, or vitamin B2, is another vitamin where researchers has reached a recommended daily amount of 0,04mg/kg or 20 mg/day/500kg [3, 8]. This vitamin also partakes in the production of ATP due to it being the precursors to the coenzymes flavin adenine dinucleotide and flavin mononucleotide (FAD, FMN), in addition to being an antioxidant [8, 16, 18]. Riboflavin is found in alfalfa, clover and other grass hays[8]. The vitamins B3 and B9, niacin and folate, are both produced in the GI tract by microbial fermentation, and neither have reportedly caused deficiencies [8]. It is therefore reasonable to believe that the horses own production covers the needs for these two vitamins. Niacin is the common name for the two compounds nicotinamide adenine dinucleotide (NAD) and nicotine adenine dinucleotide phosphate (NADP), these are the two forms that naturally occurs in feedstuffs and throughout the body[8, 16]. NAD and NADP has a wide range of functions throughout the body, mainly partaking in oxidation-reduction reactions, and aids in the DNA production [16]. Folate also contributes to the DNA production, and functions involved in rapid cell growth [8, 16]. Vitamin B12, or cyanocobalamin, is synthesised in the GI tract and the vitamin itself does not need supplementation. However, the synthesis of cyanocobalamin requires cobalt, a trace mineral, which cannot be produced by horses, therefore cobalt is an important part of a horse's diet. B12 is an essential part in many mechanisms throughout the body, including the production of red blood cells and in the metabolism of several nutrients [8, 16]. Due to the endogenous production of B12, there are no recommendations for daily supply, the same applies for Biotin, B5 and B6. Vitamin B5 and B6 are both produced by gut fermentation, and both contribute to several enzymatic reactions in the metabolism of various nutrients[8]. Biotin acts as a co-enzyme for several carboxylase enzymes involved in fatty acid synthesis, gluconeogenesis and amino acid metabolism, although it is produced by microorganisms of the gut, deficiencies can cause hoof weakness and nutritional supplementation may be needed in such cases[3, 8].

Vitamin C acts as an antioxidant and is a cofactor for several oxidase enzymes partaking in the production of several compounds, such as collagen and carnitine. Ascorbic acid, another name of vitamin C, also aids the regeneration of vitamin E, red blood cell production and even helps iron absorption. This vitamin is produced endogenously in the liver from glucose and will not need supplementation in a healthy horse. Research shows that horses in

prolonged heavy exercise may benefit from occasional supplementation of 10 g daily, but it shall not be given continuously as the liver may stop production. [3, 8, 16]

Table 2: Vitamin requirements

Fat soluble vitamins	**Daily needs (500 kg horse)**	**Water soluble vitamins**	**Daily needs (500 kg horse)**
Vitamin A	M: 1500 IU VHW: 22500 IU	**Thiamine – B1**	M: 30 mg VHW: 62,5 mg
Vitamin D	M: 3300 IU VHW: 3300 IU	**Riboflavin – B2**	M: 20 mg VHW: 25 mg
Vitamin E	M: 500 IU VHW: 1000 IU	Other B vitamins	Not determined, produced endogenously
Vitamin K	Not determined	Vitamin C	Not determined

3.2.6 MINERALS

Minerals comprise a very small part of a horse's diet, but these inorganic substances are very important, as they have several vital roles in the horse's body. Minerals occur naturally in water, soil and rocks, and grazing horses will mainly get their mineral supply through eating grass roots with soil remnants. However, a stabled horse with little to no access to daily grazing might not get all mineral needs covered by consuming hay or other plant material, therefore supplementation is often needed. There are 22 known minerals that horses need for vital functions, and these are divided into two groups, macro minerals and micro minerals.

Table 3: Macro minerals, function, sources, and requirements of a 500kg horse

Macro mineral	**Function in the body**	**Dietary sources**	**Daily requirements**
Calcium	Constituent of skeleton and teeth Muscle contraction Blood clotting Nerve impulse transmission	Alfalfa Grass Limestone flour	M: 20 g VHW: 40 g
Phosphorous	Constituent of skeleton and teeth	Cereals Grass	M: 14 g VHW: 29 g

	Regulate blood pH Phospholipids for lipid transport ATP, ADP constituent – oxidative phosphorylation Protein synthesis		
Magnesium	Skeleton and muscle constituent Activate enzymes Muscle contraction	Forages, grass, alfalfa Linseed	M: 7,5 g VHW: 15 g
Sodium	Major cation of ECF Regulates osmotic pressure Acid-base balance ECF maintenance – hence regulating blood volume	Salt lick Grasses, hay	M: 10 g VHW: 41 g
Chlorine	Anion of ECF Acid base balance Osmotic pressure Composition of bile and HCl	Salt lick Molasses Grass, hay	M: 40 g VHW: 93 g
Potassium	Important component of skeletal muscle cells Fluid, electrolyte, and acid-base balance Muscle contraction	Forages Oil seed meals	M: 25 g VHW: 53 g
Sulphur	Aa synthesis Hoof and horn production Constituent of enzymes, insulin, and heparin	Grass	M: 15 g VHW: 18,8 g

Information in the above table are found in the following sources: [1, 3, 8] -

Table 4: Microminerals, function, sources, and requirements of a 500 kg horse

Micromineral	Function in body	Dietary sources	Daily requirements
Cobalt	Synthesis of vitamin B12 Blood cell formation	Forage	M: 0,5 mg VHW: 0,6 mg
Copper	Copper dependent enzymes Haemoglobin synthesis Elastin production	Molasses Forage, depend on Cu concentration of soil	M: 100 mg VHW: 125 mg
Iodine	Mainly found in the thyroid gland Essential in the production of thyroid hormones T4 and T3	Most feeds Kelp, seaweed	M: 3,5 mg VHW: 4,4 mg
Iron	Haemoglobin and myoglobin constituents Part of cytochromes and other enzymes Vital for oxygen transport and cellular respiration	Forage Most natural feeds	M: 400 mg VHW: 500 mg
Manganese	Fat and carbohydrate metabolism Cartilage and bone formation	Forages Cereal bran	M: 400 mg VHW: 500 mg
Selenium	Antioxidant, interacts with vitamin E Thyroid hormone metabolism	Varying content in feedstuffs, depending on concentrations in the soil.	M: 1 mg VHW: 1,25 mg
Zinc	Constituent of over 100 enzymes	Yeasts Cereal	M: 400 mg VHW: 500 mg

Table sources [1, 3, 8]

3.3 DIETARY CONSIDERATIONS OF THE RACING THOROUGHBRED

Thoroughbred racing is a sport where horse and jockey competes over a range of distances, on a flat dirt or turf surface, at gallop speeds up to 70 km/h. The speed depends on the distance travelled, which can be anywhere between 900 to 2400 meters. The training regimen for each horse depends on what distances they usually race, and their diets are usually made to fit each individual[1]. As discussed previously, the nutritional requirements of a horse under heavy exercise are quite elevated compared to the maintenance needs. In order to meet those needs, the racing thoroughbred must be fed an energy and protein dense diet, and special care must be taken regarding to the BCS of the animal.

The body condition scoring system is based on the overall condition of the horse, on a scale from 1-9, where 1 is an emaciated horse and 9 would be extreme obesity. The optimum range would be between 4-6 [1]. Keeping a racehorse in a good condition is very important, as it affects both performance and health. An overweight horse will have an increased strain on its legs during training and may be more prone to injuries such as ruptured tendons or fractures. An increased amount of adipose tissue in the thoracic cavity may also decrease lung capacity by limiting the amount of space for the lung to inflate. Fat also serves as insulation, and while during winter a horse in pasture may benefit from this, a racehorse may suffer overheating during training and racing if the BCS is too high. Lastly, obesity may affect performance simply due to the increased weight the horse carries, and therefore tires more easily than its competitors. [1, 19]. On the other end of the scale, the thin racehorse will also suffer consequences of its condition during training and racing. Being underweight can come from a rage of causes such as chronic starvation, malnutrition, or digestive disorders. An underweight thoroughbred kept at a professional racing stable with a proper feeding regime will likely have either a underlying digestive disorder or be in negative energy balance, NEB, as a result of being fed too little energy compared to the workloads[1, 8]. Such horses will have worse performance, due to their reduced glycogen stores, which leads to much faster fatigue much faster than their stable mates with a good BCS. The amount of anaerobic energy production also decreases during short, intense exercises, giving the horse less energy to use during races. When a horse is in NEB, the body's own proteins will be used for energy production, which may affect the integrity of tissues, and other vital body functions. [1, 3, 4, 8]. Due to the decreased performance of either an obese or a thin horse, it is very important to maintain the BCS within a healthy range in order to optimise

racing results. It is also proven that a horse within the moderate BCS range utilises its fat stores much more efficiently than those below or above 4-6[1]. For a trainer to optimise each horse´s diet, regular body condition scoring should be done, to control that the daily rations meet horses´ requirements in terms of keeping a perfect condition, and is a practical way of assessing whether the energy intake is adequate[1, 3].

As racehorses require a lot of energy, the amount of starch in their diets are usually quite high, it is therefore important to spread the feedings evenly throughout the day, to avoid sudden starch overloads. Special care should be taken when creating a ration, to the amount of starch per meal, and the meal frequency. The NRC[8] recommends that no more than 0,2-0,4% of kg BW in starch should be fed per meal. High starch diets have been associated with several diseases, such as equine gastric ulcer syndrome, colic, and laminitis. Too much starch in the diet means that more starch reaches the hindgut, and consequently changes the microbial population[1, 8]. This change affects several functions of the intestines, such as altering the water absorption and motility, and decreasing intestinal pH resulting in hindgut acidosis and decreased performance. These alterations may lead to a build-up of toxins and production of abnormal metabolites, which in turn may be a cause of laminitis, colic and stereotypical behaviours[8].

Equine athletes also have a higher need for protein, due to anabolic changes in the muscles during training, and the repair of muscle fibres after an injury or straining work [1, 4]. The recommended daily amounts of protein intake is only a recommendation, based on horses as a group, and does not take individual needs into account[1, 8, 20]. This must be defined by the trainer or person designing the diets, and very often protein is fed in excess compared to recommendations[1, 21]. While feeding horses excess protein in reasonable amounts is common, feeding extreme amounts of protein may have adverse effects[1, 20, 21]. Sweat is also a source of protein loss and may cause variations in protein needs in different seasons. Other compounds easily lost through sweating are the electrolytes Na, Cl and K. It is therefore important to ensure sufficient intake of these electrolytes, especially during summer when the horses sweat more[1, 8]. This can be ensured through free access to a salt or mineral stone or supplementing the feed with electrolyte products. When using quality hay and feed grain, vitamin deficiencies are unlikely in racehorses, but making sure there is enough vitamin E in the diet is important, as it is a cofactor in energy metabolism. [1].

3.4 NUTRITIONAL DISORDERS

When creating a diet for a horse, knowledge about certain nutrition related diseases and how to avoid them, is essential. Horses have a complex digestive tract, that can easily be upset by the wrong diet and cause disorders such as gastric ulcers and colic. Endocrine and systemic disorders can also be a result of improper nutrition, examples of these can be insulin resistance and hindgut acidosis [1, 3, 4, 8].

Hind gut acidosis can be the culprit behind poor performances in thoroughbreds, as well as laminitis and colic symptoms. This can come from diets rich in cereal and starch, especially when fed in large amounts [1, 3]. When too high amounts of starch are fed at once, the SI cannot digest it all, and some starch reaches the LI where it is rapidly fermented. This causes an increase in VFA´s and lactic acids which lowers the pH [3]. The acidic milieu damages the mucosa and causes inflammation. In addition, the acidic environment is unsuitable for many of the microbes and bacteria of the HG, and dysbiosis may occur. Dead bacteria releases endotoxemia, which is linked to the occurrence of laminitis [1, 3]. The abnormal bacterial flora that occurs in HG acidosis also produce abnormal metabolic products, which affects the ratio of absorbed nutrients, and change certain metabolic pathways. In addition, buffer systems of the body work full time on trying to increase the pH, leading to reduced availability of buffer systems in other parts of the body. When buffer supplies are low, it affects the performance of the horse[1, 3]. As a result of HG acidosis, horses feel pain, show signs of colic and perform less than expected, they can also start to show stereotypical behaviour. [1, 3, 4].

Some horses show unwanted, stereotypical behaviour as a result of intestinal disease or deficiencies. Crib biting or air sucking can be a sign of HG acidosis [1, 3]. When horses are cribbing, they bite down on the crib or other material, sucking in air. This can damage the incisors, and lead to excess gas in the intestines which can lead to gas colic[1, 3]. Other stereotypical behaviours linked to HG acidosis, namely weaving and box walking. Signs of deficiencies can be wood chewing and soil licking. Wood chewing can be an indicator of fibre and salt deficiencies, chewed fences is a common sight in horse paddocks. Soil licking, or geophagia, can indicate mineral deficiencies [1, 3]. A revision of the diet when observing such behaviours can be a helpful tool when trying to reduce the unwanted traits.

Gastric ulcers is a well-known disorder in horses, and has a higher prevalence in horses exercising at top intensity levels [1, 3, 4]. As mentioned in chapter 3.1.2, the stomach is divided into two main parts, the non-glandular and glandular part, between these area runs the margo plicatus. The non-glandular part is more prone to ulceration, as it has less protection than the glandular part. The most common areas of gastric ulcers in the non-glandular part are the areas just above the margo plicatus[1, 3]. Gastric ulcers or equine gastric ulcer syndrome (EGUS) have different severities, and a grading system is developed to determine its severity. The scale goes from 0-4, where 0 is red, irritated but still intact mucous membranes, and 4 is deep ulceration[1, 4, 22]. If left untreated, ulcers can cause perforation of the stomach which can be detrimental[1, 3]. The pathophysiology of EGUS is often linked to high starch content, and low forage intake [1, 4]. Other triggering factors can be poor dentition or feeds that require less chewing, the less a horse chews, the less saliva is produced. This can increase the vulnerability of the mucosal membranes of the stomach as saliva is an important buffer [1, 3, 23]. Results of EGUS can be reduced performance, weight loss, decreased appetite and colic symptoms[4, 23]. Treatment of EGUS can be done with good prognosis, as long as the risk factors are eliminated, and dietary changes are made. Alfalfa is proven to be beneficial to horses with gastric ulcers, as it has a buffering effect, increasing the gastric pH[1, 3, 4]. As gastric ulcers are more frequently seen in horses in intense work, it is important to be aware of the risk factors when designing a diet for a racing thoroughbred [1, 3, 4, 23]

Colic is a well-known disease amongst horse owners and is one of the more common diseases seen in exercising horses. Colic is a collective term for intestinal disorders causing abdominal pain and is often recognised by excessive rolling, kicking or biting towards the abdomen, sweating and increased heart and respiratory rates[1, 3, 4]. Colic can be caused by several factors, one of which being related to feeding and nutrition. Too low water intake may lead to dryer intestinal content, which can cause impactions especially in narrower parts of the GI-tract, another reason can be poor dental health which leads to larger particles of feed swallowed due to less thorough mastication[3]. Abrupt changes in feed composition, or increased starch intake may lead to imbalance or acidosis of the gut, potentially causing colic[1, 3, 8]. Colic can manifest in different areas of the GI tract, and be caused by a range of disorders[3]. It can be a colic located in the stomach due to decreased motility, or in the SI due to an ileal obstruction, strangulated parts of the SI or even caused by a SI enteritis. In the large intestine, the most diagnosed type of colic is an impaction of the pelvic flexure.

The pelvic flexure is a part of the LI that changes direction in the abdomen, and narrows simultaneously, making it a sensitive spot for impactions[1, 3, 4]. The right, and left, dorsal colon are predisposed to displacements as they are very mobile compared to the rest of the GI-tract [3, 24]. Other LI colic's can be torsions or gas build up causing tympany, the latter is often caused by sudden changes in feed or increased starch content of the feed, which increases the amount of gas produced in the colon[1, 3]. Intense exercise can also result in colic, as digestion is slowed down during physical activity, and less water may be released into the GI-tract. [1, 3, 4, 8, 24, 25].

Laminitis is a systemic disease, that affects the horse's foot, and leads to pain and lameness. This foot pain is caused by a failure of the attachment between the dermis and the epidermal lamina, in other words, the connection between the distal phalanx and the hoof wall. Laminitis can be both acute and chronic, whereas increased starch intake is mostly related to the acute form[8]. The pathogenesis behind laminitis is not fully understood, but there are several theories. The theory linked to nutrition is that too much rapidly fermentable carbohydrates are fed, leading to a release of monoamines that negatively affect the blood circulation in the hoof, causing the delamination of the hoof wall. [1, 3, 4, 8].

Exertional rhabdomyolysis is a disease where horses experience pain and muscle cramps in relation to exercise. This disease is also known as tying-up or Monday morning disease as it often appears during or after exercise on the day after a rest day, which is usually on Sundays in the racing industry[1, 3]. This disease has two forms, polysaccharide storage myopathy (PSSM) and recurrent exertional rhabdomyolysis (RER). RER is more commonly seen in race horses and is associated with abnormal Ca regulation and dysfunction of normal contractions in muscle fibre [3, 8]. The intracellular Ca regulation deficit linked to this disease can be hereditary, and causes muscle necrosis during exercise [8]. The lysis of muscle cells cause inflammation which can lead to high levels of pain for the horse[1, 3]. Other symptoms can be observation of dark urine, which occur due to the elevated levels of myoglobin that is excreted, this can, in severe cases lead to kidney damage [1, 3]. This disease can also be subclinical, where the only signs are lower tolerance to exercise and decreased stamina which lowers the overall performance of the horse [1, 3, 8]. Another contributing factor can be electrolyte imbalance as a result from excess sweating during exercise in increased temperatures [1, 3]. It is suggested that decreasing the level of starch

in the diet, and supplementing with fats and oils can help decrease the occurrence of RER[3, 8].

4. OBJECTIVES

The objective of this thesis is to see if the feeding plan of a Norwegian racing stable is adequate in terms of meeting the racing thoroughbred's nutritional needs when performing at such high levels. The Norwegian trainer's education does not contain a course about nutrition, and so the feeding regimes are mostly made from experience and preferences of the individual trainers. The goal is to see if an experienced trainer has gained enough knowledge about nutrition, through lifelong work with thoroughbreds, to be able to put together a diet that suits the horses, or if there are any major deficiencies or surpluses of the most important nutrients in this diet.

5. MATERIALS AND METHODS

Several methods have been used to analyse the topic in question. First, a literature review of several research articles and literature to obtain a clearer picture of equine dietetics and nutritional requirements.

A longer interview was conducted to obtain information about the feeding regime of the racing stable. In addition, watching the feed mixture being made, measuring every ingredient, writing down the recipe, and observing the feeding of the horses, asking questions during every part about how and why he chooses this way of feeding. The questions asked are listed in table 5. After the interview, a list of ingredients, feeding schedule and amounts were made. All ingredients used were measured in kg in order to calculate the actual ration of each horse.

Table 5: Questionnaire of the racing trainer

Question	How long have you been a racehorse trainer?
Answer	Since 1983, for 39 years
Q	How did you become a trainer?
A	A racehorse trainer course is kept by the Norwegian
Q	Do you have any education within the field of nutrition?
A	No, but I have been to seminars about nutrition
Q	Who hosted these seminars?

A	Mainly feed salesmen
Q	How have you gathered information about how to feed your horses?
A	Read articles and books, and learnt from feed companies and other trainers
Q	What books have you read?
A	Introduction to horse nutrition by Zoe Davies, trainers' magazines etc.
Q	Have you always fed you horses the same way?
A	No. The composition of what I feed have changed over the years, but the recipe I use now is probably the one I have stuck to the longest.
Q	Why is that?
A	Many reasons, the feed marked has changed a lot over the years, producing more advanced feedstuffs. A lot of trial and error. Previous history of more horses colicing compared to what they do now.
Q	Why do you think the current recipe is the one you have stuck to the longest?
A	The recipe I use now seems to work very well for my horses, it is also affordable and easy to make, and can be easily adjusted to fit different needs. I also have almost zero occurrence of digestive troubles in the stable with this diet. Horses weight and condition seem to be stable on this type of feed
Q	How do you adjust the recipe to each individual horse?
A	First of all, by the amounts of feed I give each horse, they get more or less depending on their condition, age and stage of training. I can also easily swap out ingredients according to the seasons, during low training season (winter) they get a less energy dense component. It is also easy to add certain supplements into the basis recipe.
Q	What is the prevalence now of nutrition related disorders in your stable?
A	I don't have an exact overview, but as mentioned I have had way less cases of colic, and probably gastric ulcer now than what I did previously.
Q	Do you have any routines for controlling the occurrence of nutritional disorders?
A	No, we don't have a routine for it. We do of course consult a veterinarian when a horse seems to perform worse than usual or shows symptoms of disease.
Q	Have you had any recent cases of colic at all on the current diet?

A	Yes, we had one severe case, but I don't think this was due to the diet alone. The horse had just moved to my stable and the colic symptoms started acutely when the horse was on its way home from work. Nothing can be said for certain, but in my opinion, it was probably an exercise induced colic, where the new diet could have been an inducing factor.
Q	Have you had any cases of gastric ulcers in the previous year?
A	Only one case has been confirmed by a veterinarian, this was resolved quickly by administering gastric protectants, and this horse now gets a type of supplement that helps prevent recurrence. I also treated another horse with similar symptoms shortly after, but this was not a confirmed case by the veterinarian.
Q	Did the horse you treated show any improvement?
A	Yes, it seemed like it responded to the treatment.

5.1 INGREDIENT LIST

Every horse has an automatic drinking trough in their boxes, meaning they get ad libitum water access every day. In addition, horses that drink poorly may get supplemented with water buckets with added taste enhancers such as molasses, treats such as apples or carrots, or products made specifically for increasing drinking in horses, usually with added electrolytes etc. The additives in the water will not be included in this paper.

In addition, they are fed 8 kg/ day with good quality haylage produced by a local farmer. This hay does not have a nutritional analysis, as it is produced by a private person and not a larger company. It is however of very good quality, as the trainer did not notice any differences in his horses when he changed from a top quality haylage from a larger company to this locally sourced haylage. The programme PChorse that is used for controlling the diet in this thesis has several standard data for different forages, the type "Haylage, top quality" will be used.

The concentrates are mixed in a large tub and then divided into portions for each horse, meaning horses get the same type of feed but in different quantities, and possibly other additives if needed. The amount of feed given to each horse is decided individually based on the stage of training, workload and each individual BCS and ability to maintain the optimal

condition. For example, two horses in peak racing season may get different amounts of feed if one is prone to obesity and the other has a harder time keeping optimal racing weight. The general rations fed to a horse in racing season is 3 litres at 06.00, 3 litres at 12.00 and 5 litres at 18.00, in total 11 litre concentrates and 8 kg haylage daily. The fibre beet and Pegus Buildmax is not put into the mix but portioned to each horse individually. Champion fibre beet is soaked before serving, and approximately 500 grams are added on top of the night feed, in dry form this 150 g/horse. Pegus Buildmax is given according to the manufacturers recommended daily dose of 50g. Even though individual variations occur, this is the amount that will be discussed further in this thesis and will be based on a 500 kg adult horse in full exercise and racing.

Table 6: The different ingredients of the feed mixture in the racing stable

Name of feedstuff	**Description of feedstuff**
Champion energi	Pelleted concentrate
Champion oats	Whole oats
Krafft chopped lucerne	Dried, chopped lucerne grass
Pegus chaff	Finely cut straw
Champion betfiber	Fibre beet, fed once a day
Pegus Buildmax	Powdered protein supplement
Champion soyaolje	Soy oil with vitamin E

Table 7: The amounts of feed given as stated by the trainer

Feedstuff	**06.00**	**12.00**	**18.00**	**Total**
Concentrate mix	3 litres	3 litres	5 litres	11 litres
Pegus Buildmax	25 g	0	25 g	50 g
Fibre beet	0	0	200 g	200 g
Haylage	2,5 kg	2,5 kg	3 kg	8 kg

In table 7 we see the feeding schedule as stated by the trainer. The trainer uses am measure cup to ration the feed mix to each horse, and only knew the amounts given in litres. The study was not conducted during a feeding, so control measuring the weight of the amounts

given was not possible. However, the feed mix was premade at the time of the study, so 1 litre mixture was weighed to 492 grams. In table 8 the timing of the feeds, as well as the amounts given is shown. The total amount of feed mix given daily is 5,42 kg, plus the 200 g (dry weight) of fibre beet and the Pegus Buildmax.

Table 8: daily feed intake in kg per meal

Feedstuff	**06.00 (3 L)**	**12.00 (3 L)**	**18.00 (5 L)**	**Total (11L)**
Champion energi	0,9 kg	0,9 kg	1,4 kg	3,2 kg
Champion oats	0,33 kg	0,33 kg	0,55 kg	1,2 kg
Krafft chopped lucerne	0,08 kg	0,08 kg	0,14 kg	0,3 kg
Pegus chaff	0,1 kg	0,1 kg	0,2 kg	0,4 kg
Champion soy oil	0,085 kg	0,085 kg	0,15 kg	0,32 kg
				Total: 5,42 kg

5.2 NUTRITIONAL COMPONENTS OF EACH INGREDIENT

The nutritional components listed underneath are collected from the manufacturer's own websites, directly from the feed bags used during the collection of data in the stable, or from the PChorse programme.

Table 9: Champion Energi.

Analytical constituents %		**Minerals & trace elements**		**Vitamins**	
CP	10	Lysine	4,4 g/kg	A	7000 IU/kg
Digestible CP	7,5	Ca	12 g/kg	D	740 IU/kg
Fat	5,2	P	6 g/kg	E	630 mg/kg
Fibre	6,4	Mg	3,5 g/kg	B1	17,4 mg/kg
Starch	30	Na	10 g/kg	B2	7,5 mg/kg
Sugar	7	NaCl	25 g/kg	B6	4,5 mg/kg
DM %	98	Fe	160 mg/kg	B12	0,05 mg/kg
		Cu	55 mg/kg	Niacin	10 mg/kg
		Mn	100 mg/kg	Folate	5 mg/kg
		Zn	188 mg/kg	Biotin	1 mg/kg
		Se	0,66 mg/kg	C	150 mg/kg

		Co	0,4 mg/kg		
		I	1,2 mg/kg		

Table 10: Champion Oats.

Analytical constituent		**Minerals**		**Vitamins**	
DM %	86	Ca g	0,6	A IU	40
Energy MJ	10,5	P g	3,1	D IU	0
CP g	99	Mg g	1	E mg	15
Lysine g	4,3	Na g	0,25	B1mg	6
Starch g	400	Cl g	1	B2 mg	1,4
Sugar g	15	K g	4,7	B6 mg	2,6
Fat g	35	S g	1,2	B12	0
Fibre g	120	Fe mg	55	Niacin mg	15
		Cu mg	2,6	Folate mg	0,4
		Mn mg	40	Biotin mg	0,3
		Zn mg	30		
		Co mg	0,01		
		I mg	0,2		

Table 11: Chopped Lucerne:

Analytical constituents		**Minerals**	
Dry matter	88%	Lysine	4 g/kg
Metabolisable energy	8,5 MJ/kg	Calcium	1,3 %
Crude protein	15%	Phosphorus	0,3 %
Digestible CP	105 g/kg	Magnesium	0,2 %
Digestible CP/ energy	12,4 g/MJ		
Crude fibre	27%		
Crude oil and fat	3%		
Sugar	11%		

Table 12: Pegus Chaff

Analytical constituents		**Minerals (g)**	
DM %	87	Ca, P, Mg	0
Energy MJ	7	Na	0,87
Cp g	35	Cl	4,3
Lysine	0	K	16
Starch g	4	S	2,2
Sugar g	170		
Fat g	4		
Fibre g	210		

Table 13: Pegus Buildmax

Analytical contents	
DM %	98
Energy MJ	8,5
CP g	570
Lysine g	120
Sugar g	360
Fat g	60
Fibre g	1

Table 14: Fibre beet

Analytical constituents		**Minerals**	
DM %	98	**Ca g/kg**	5,2
CP %	8	**P g/kg**	1
Digestible CP %	6,3	**Mg g/kg**	2,2
Fat %	4,7	**Na g/kg**	9
Fibre %	17,5	**Added NaCl g/kg**	20
Starch %	0		
Sugar %	8		
Lysine g/kg	4,5		

Table 15: Champion soy oil with vitamin E

Fat	**95%**
Vitamin E	1000 mg/kg

Table 16: Haylage data collected from PChorse

Analytical constituents		**Minerals**		**Vitamins**	
DM%	65	Ca g	2,9	A IU	2300
Energy MJ	6,8	P g	1,5	D IU	750
CP g	78	Mg g	0,84	E mg	19
Lysine g	2,6	Na g	1,4	B1 mg	1,5
Sugar g	29	Cl g	7,8	B2 mg	9,3
Fat g	20	K g	16	Niacin mg	77
Fibre g	150	S g	1,3	Folate mg	0,4
		Fe mg	72		
		Cu mg	3,9		
		Mn mg	59		
		Zn mg	16		
		Se mg	0,026		
		Co mg	0,08		
		I mg	0,36		

In the final step to finding the exact nutrient constituents of the ration, the computer programme PChorse was used. PChorse is developed by Hove Software LTD of Norway, and is a tool made to analyse, create, and control equine diets, which can be used by both veterinarians and horse owners. This programme is also available for educational purposes. In this programme, one can create a stable, and add horses with specific breed, gender, age, and bodyweight. This programme can simply calculate the nutrient amounts in a given ration, showing the percentage of feeds given, alerting the user of any deficient or excessively fed nutrient, and helpful comments about how to stabilise these anomalies. It also calculates the different nutrient ratios like Ca:P ratio and forage to concentrate ratio. The programme also allows for veterinarians or nutritionists to create new diets in a simple way and is a good tracking device of horse's diets and weights. For this thesis a 4-year-old thoroughbred gelding/mare of 500 kg was used. The exercise level was adjusted to very

heavy work, or 1,9 times maintenance, as this is normally the level racing thoroughbreds are placed on. After this, the different ingredients were added to a feed list, and the amounts of each ingredient were filled in according to the stables diet.

6. RESULTS

6.1 DAILY INTAKE OF THE HORSE

In table 14 we can see the recommendations compared to the calculated amounts from PC horse, it also shows the deficient or excess amounts. The recommendations used are the ones discussed in the literature review, not the ones PChorse uses, as they differ to some extent. These results show that energy is provided in lower amounts than recommended, this is however the only nutrient fed in too low amounts. The water soluble vitamins are excluded from this analysis as they have no agreed upon recommended value, and are thought to be supplied through either grazing alone or microbial and endogenous production[1, 3, 8]. Another reason for their exclusion is that these vitamins have no proven clinical cases of either deficiency or toxicity[8], which means that they are likely to as important when considering the diet. Cobalamin and the vitamins A, D, E and B2 are fed in high excess, up to 400%, these high values will be discussed further in the discussion. Overall, most nutrients are fed within reasonable ranges, compared to the requirements. This already shows that the diet is likely to be a well-functioning one for the horses.

Table 17: recommended amounts compared to actual intake

Nutrients	Unit	Recommendations	Daily amounts	Difference	%
Energy	MJ	144	131	-13	91
CP	G	1004	1200	196	119,5
Lysine	G	43	47	4	109
Ca	G	40	61	21	152
P	G	29	36	7	124
Mg	G	15	20	5	133
Na	G	41	46	5	112
Fe	Mg	500	880	380	176
Mn	Mg	500	750	250	150
Zn	Mg	500	800	300	160
Se	Mg	1,25	2,4	1,15	192

Co	Mg	0,6	1,5	0,9	250
I	Mg	4,4	6,9	2,5	156
Vitamin A	IU	22500	43000	20500	191
Vitamin D	IU	3300	8500	5200	257
Vitamin E	Mg	670 (1000 IU)	2500 (3732 IU)	1830	373
Vitamin B1	Mg	62,5	83	20,5	132
Vitamin B2	Mg	25	100	75	400

Sources for table 14: PC horse results, [1, 3, 8]

In figure 2 we can see the different ingredients contribution to each nutrient. This provides a good picture of which ingredients are the most significant ones of the diet. It also shows a percentage range different to the one in table 14. This is because PChorse uses different requirement recommendations than what was used in the literature review. This feed analysis will only take the previously discussed data of the literature review into account, due to the lack of sources referred to by the PChorse programme. It is obvious when looking at figure 2 that the two feedstuffs that contribute the most are the haylage and the Champion Energi. Champion Havre (oats) also seems to play an important role in this diet.

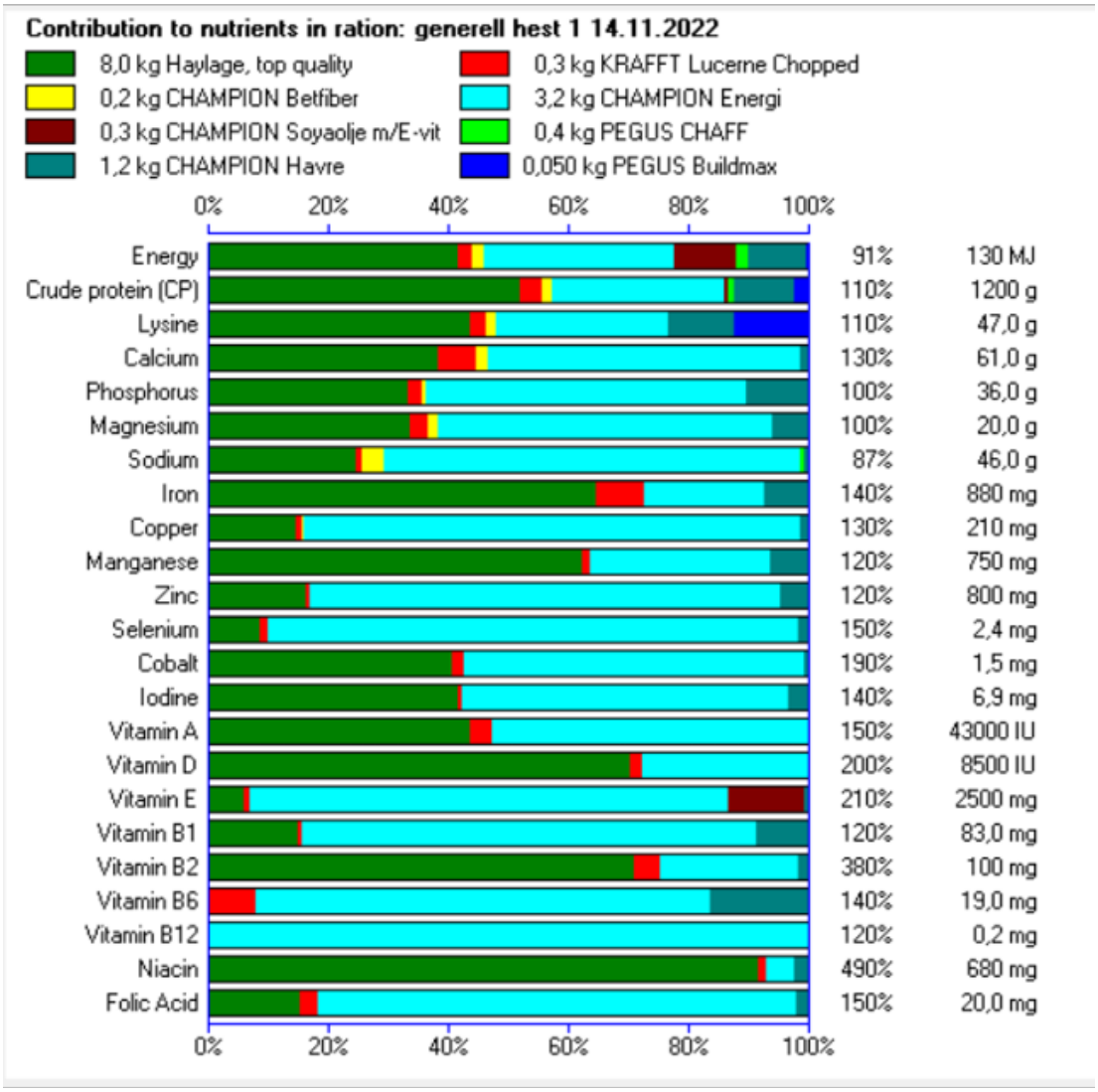


FIGURE 2: PChorse results showing the percentage of each nutrient and the contribution of each ingredient to each nutrient.

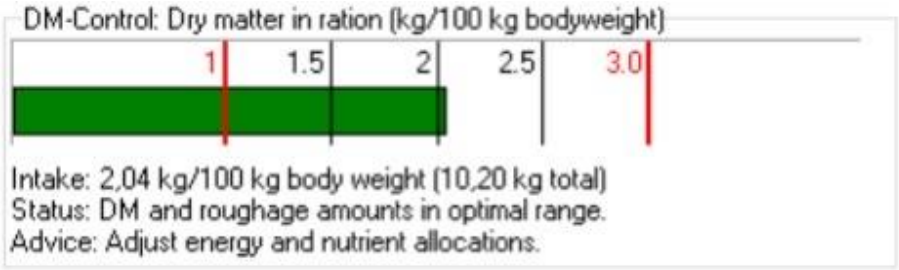
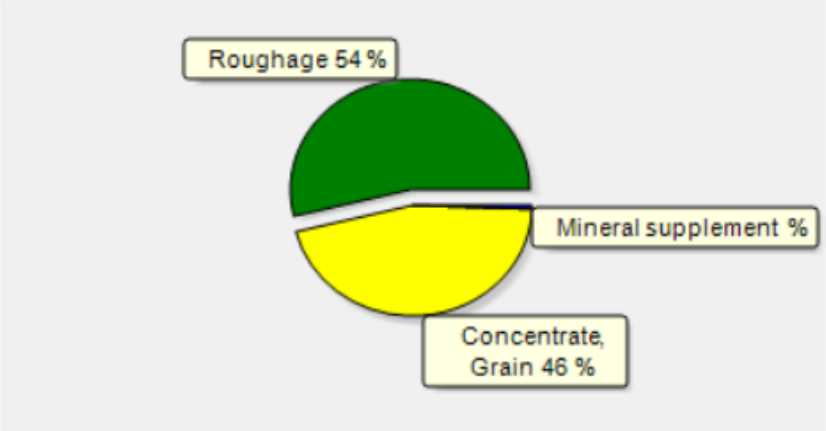


FIGURE 3: Kg dry matter intake per 100 kg BW

10,20 kg Dry matter in ration (maximum 15,00 kg/day)
% on dry matter basis: Roughage 54%



Nutrient ratios

Crude protein (CP)/Energy ratio = 9,1 g/MJ
Calcium/Phosphorus ratio = 1,7 g/g
Calcium/Magnesium ratio = 3,0 g/g
Zinc/Copper ratio = 3,7 mg/mg
Manganese/Copper ratio = 3,5 mg/mg
Iron/Copper ratio = 4,1 mg/mg

FIGURE 4: Ratios given by PChorse

7. DISCUSSION

Based on the low frequency of nutritional disorders the diet seems to be suitable for equine health. It is likely that these horses have lower nutritional requirements than the international thoroughbred top-athletes, who perform on a somewhat higher level compared to the average Norwegian thoroughbred. Overall, the diet given corresponded well with the dietary requirements outlined in the literature review.

We found that the amount of energy given was 9% lower than the expected amount of 144MJ [8]. The horses showed stable weights and did not have a history of a change in BCS, and it is, therefore, unlikely that they are being underfed compared to their actual requirements for their current workload. One possible reason for the amount of energy being lower than expected is that this study was conducted during spring, which is only the beginning of racing season in Norway, as the track is closed during winter. Another reason behind this theoretical shortfall could be the use of the generically designed data for haylage as the actual haylage used did not have a nutritional analysis. One could also argue that these horses don't fit perfectly in the ´very heavy work´ category. Even though racing thoroughbreds are placed in this group in most literature, variations in training regimes may vary internationally. As

racing is a small sport in Norway, the workload may be less than e.g., England where this sport has been part of the culture for many centuries.

Protein is, as shown in table 15, fed in excess of 119%, this is within an acceptable range. The main feedstuffs contributing to the protein supply of the diet are the haylage, Champion Energi and Oats. Although one shall avoid extreme overfeeding of any nutrient, no detrimental effects has been proved from excess protein supply. However, increased protein supply can lead to increased urea which is excreted by the kidneys, this may lead to increased water loss, consequently leading to a higher water demand[8]. If the supply had been lower than the requirements we would have observed weight and muscle loss of the thoroughbreds in the stable[1, 8]. Although lysine is part of the protein and amino acid pool, its supply has to be controlled separately as it is the first limiting amino acid and can indicate a deficiency in amino acid supply if it is lower than recommended[1]. The estimated requirement of a 500 kg horse in very high work is 43 grams, this diet supplies enough lysine of 47 grams[1, 8].

Micronutrients were given in adequate amounts, and some were given in excess e.g., vitamin A. Excess supplementation with fat-soluble vitamins is more concerning than excess supplementation with water-soluble vitamins since they are more difficult to excrete and more readily cause symptoms of toxicity. Nutrients given in excess of theoretical requirement raises the question of potential signs of toxicity. With vitamin A toxicity one would expect anorexia, thick and scaling skin, coat dullness and reduced bone strength[3, 8, 16]. However, no such cases have been identified in the stable under investigation in the past years on this particular diet. Vitamin A toxicity can occur when fed 10 times the requirement [16] or over 20 000 IU per kg feed/day [3]. This diet provides 43 000 IU/day which is within safe limits. Vitamin D was supplied at 257% of the recommendations, meaning 5200 IU more than the requirement of 3300 IU [8]. This is still below the amounts that would cause signs of toxicity, this limit is estimated to be 22 000 IU/day for a 500 kg horse [8]. The signs of toxicity would be calcification of tissues and even death, no such cases have been seen in the stable. Vitamin E is also oversupplied, as 670 mg is recommended and the amount supplied is 2500 mg/ day [1, 8]. No signs of vitamin E toxicity have been reported, but it is assumed that coagulopathies and bone mineral dysfunctions would occur [1]. It seems horses have a high tolerance for excess vitamin E, and as no symptoms of excess is seen in the horses[8], the supplied amount can be regarded as safe.

Most of the minerals in the diet are supplied in tolerated amounts, the only mineral given above 200% is cobalt. This in itself is not a problem as there are no reported findings of clinical signs of excess cobalt intake in clinical cases, nor has it been experimentally induced[8]. However, cobalt can act as a hypoxia inducing factor stabiliser, and if given in too large amounts, regarded as doping in Scandinavian racing [26]. As it is a naturally occurring compound, there is an upper limit of 0,1 microgram/ml in urine [26]. Even though we did not collect urine samples for analysis, it is reasonable to believe that the excess of cobalt does not reach the limits set by the Nordic Equine Medication and Anti-doping Committee, as no horses in the stable has ever tested positive for cobalt during regular doping controls.

This study has several strengths, as a large stable with 20 horses in active training, was studied. These horses have consistent workloads, the only variation in terms of training are the distances, depending on their usual racing distance. The feeding regime is very consistent, making the theoretical calculations more reliable. As most of the ingredients are produced by professional manufacturers, we had access to their specific nutrient constituents. The use of PChorse provided reliable calculations and nutrient distribution tables, making the diet constituent overview much clearer.

This study also had some limitations such as only one measurement timepoint outside of the maximal workload. In optimal conditions, the study would have been performed in peak performance season, however, the trainer stated that he did not make any changes to the diet itself. However, if the energy supply was in fact low, it is reasonable to assume that the amounts would be increased during more intense periods. The lack of exact analysis of the haylage, is a weakness, as the results may have varied to some degree, but it should be mentioned that from the results shown, the generic data is likely to be similar to the actual haylage. Testing for micronutrient deficiencies would have given us a clearer picture of the correlation between the theoretical results and the actual status of each horse. Ideally, one would have analysed each horse separately, taking into account the true body weight and BCS, age, racing distance and individual forage and concentrate rations. Another interesting factor would be knowing the true history of nutritionally related disorders of the stable, both from this diet and previous diets. Comparing the occurrence of e.g., gastric ulcers or colic in relation to different diets could have shown useful in understanding why the incidence of colic has decreased on the current diet, like the trainer stated in the interview.

8. CONCLUSION

We conclude that the current diet could undergo some minor modifications such as a reduction of supplementation of e.g., vitamins A, D, and B1. Another option could be to rebalance the diet as it seems to be below or very close to the recommendations for energy, CP, lysine while the minerals and vitamins are generally above the requirements discussed. Theoretically, the horses should have received more energy, but we think that energy delivery should primarily be guided by day-to-day observations of the individual horse's BCS as well as their performance characteristics rather than rigidly following the prescribed algorithm. Further studies could increase measuring points assessing a change in feeding regimens over the season and take into account the changes in energy requirements potentially seen at the maximal workload. In addition, it would be of value to have exact analysis of all components of the feed mix to ensure exact quantities of nutrients. It would be valuable to add blood analysis for the assessment of micronutrient levels. Lastly, a comparison between multiple stables using different feeding regimens utilising endpoints such as changes in blood analysis and changes of BCS over the course of a season could add insights into which changes in feeding regimens could be most impactful.

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26. List over prohibited substances and withdrawal times Valid from 01.10.22.pdf

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Department: Department of Animal Breeding, Nutrition and Laboratory Animal Science

Thesis title: Racehorse feeding strategies in Norway

Consultation – 1st semester

Timing				Topic / Remarks of the supervisor	Signature of the supervisor
	year	month	day		
1.	2022	3	26	How to collect data	
2.	2022	3	29	Methods	
3.	2022	4	6	Structure and methods	
4.	2022	4	21	Literature and PChorse	
5.	2022	5	6	Progression discussion	

Grade achieved at the end of the first semester: 5

Consultation – 2nd semester

Timing				Topic / Remarks of the supervisor	Signature of the supervisor
	year	month	day		
1.	2022	9	27	Thesis writing guidance	
2.	2022	10	3	PCHorse	
3.	2022	10	24	Thesis writing guidance	

4.	2022	10	25	Thesis writing – introduction and material and methods	
5.	2022	11	17	Final version discussion	

Grade achieved at the end of the second semester: 5

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