Equine Nutrition Update

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I greatly appreciate the opportunity to present a Frank J. Milne lecture on equine nutrition.   I first met Dr. Milne in 1974 at the famous "First International Equine Veterinary Conference" held in Kruger Park, South Africa.   I enjoyed our interactions over the years.   Furthermore, it is always a pleasure to discuss nutrition with equine veterinarians.   Several surveys have demonstrated that the equine veterinarian is the primary source of professional nutritional information for horse owners.

The National Animal Health Monitoring System (NAHMS) of the USDA conducts surveys or compilations of data "that provide a forum for discussions of impact of animal demographics, emerging disease agents, and conditions, mortality and management practices."   In 1998 it was reported that 85% of equine operations surveyed replied that the veterinarian was a very important or somewhat important source of equine nutrition information.[1]   Feed store personnel were listed by 72% of the operations and 64% mentioned the farrier.   In contrast, only about 30% of the operations considered the extension agent, university instructor, or nutritionist to be a very important or important source of nutrition information. Such results should not be unexpected because other surveys have shown that veterinarians are the professionals most respected by the horse owner, and the horse owner is likely to have more frequent contact with the veterinarian than with an extension agent or university faculty.

Historically, veterinarians have been involved with horse nutrition research.   Meyer reported that with the founding of European veterinary schools in the 18th century, veterinary teachers started gathering equine nutrition information based on field experiences.[2]   He suggested the first book entirely dedicated to horse nutrition was written in 1887 by the Dutch veterinarian, H. Mars, and was entitled *De Voeding en de Voedsels van het Paard in Neder-landisch-India.*   The translated English title is *The Feeding and Feedstuffs of Horses in Dutch India.*

Meyer also reported that in Europe at the end of the 19th century, when scientific experiments on horse nutrition began, veterinarians remained leaders in the field of horse nutrition but many agricultural leaders had also begun to conduct horse nutrition experiments.   Meyer concluded "If one looks back over the last 100 years of scientific research and practice in the feeding of horses, one realizes, paradoxically, that the horse, a peaceful creature which tends to flee rather than fight, has profited largely from the activities of military vets.   It has not been until the second half of the 20th century that new promoters have appeared in the research of nutrition for horses.   However, on a long-term basis, horse owners and friends of the horse have to determine for themselves the direction

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of nutritional research.   The only aim must be the horse's welfare.   Our challenge is to develop species-specific, health-promoting nutrition to ensure a long, well-fed life."

The need for the veterinarian to be involved with equine nutrition may depend on the type of client. It has been suggested that 80% or more of the horses in the United States could be considered recreational and that the typical recreational owner may have horses for only five years.   Thus, many horse owners may not have a great amount of horse experience and could likely need basic help with horse nutrition.

Background Information

My plan is to first discuss some of my philosophy about feeding horses and then discuss several areas of recent research, particularly those relating to digestive function and nutrient requirements.   It was written in Ecclesiastes 1:9–10, "what has been is what will be, and what has been done is what will be done; and there is nothing new under the sun. Is there a thing of which it is said, 'see, this is new?' It has been already in the ages before us."

Some interpret the above verses to mean that many things seem to be new simply because the past is easily and quickly forgotten.   Certainly, knowledge of the importance of roughage and concern associated with overuse of concentrates is not new. But it seems to me that recently there has been greater emphasis in educational material on the importance of roughage for horses, and rightly so. Perhaps fiber has been rediscovered.

Dr. Dean Scoggins of the University of Illinois suggested that horse feeding can be simple and should be left that way.[a]   Certainly for the maintenance horse this is true.   He suggested that horses should have fresh air, high quality forage, trace mineral salt, fresh clean water and grain as needed, with a footnote that many horses only need what grain it takes to catch them.   I would add that the horse must be free of dental and parasite problems for the diet to be effective.   Of course, the nutrition of the lactating broodmare, foal, and performance horse would be more complicated.

Today, feeding mismanagement probably causes more problems than improper dietary formulation. The use of commercial rations that are fortified with vitamins and minerals has significantly increased over the last few decades in many parts of the world.

Harris wrote, "One of the biggest changes in the last part of this century has been the increased use of manufactured commercial feeds for horses.   The impetus for such feeds came initially in the 19th century from the military and those involved with using horses for transportation.   At the start of this century 'compressed food cakes consisting of a mix of oats, beans, corn, bran and sometimes chaff' were mentioned as possible feeds but it was recommended that they be broken up before being fed damp. The few manufactured feeds available tended to be expensive and contained poor quality ingredients such as ground corn stalks and sawdust.   By the late 1920s, for convenience, 'prepared chop' could be purchased which was reported to contain mainly cereal straw, bruised oats (of indeterminate quality), broken maize, kibbled locust beans, and other beans, but it was recommended that 'horse owners should buy and mix the food for the horses themselves, then they know exactly what their animals are getting and that the food is of good quality.'   Pelleted feeds were being fed as early as 1917 but their popularity did not increase dramatically until the 1960s.   Increased competition and improved knowledge, as well as more ethical companies and government regulations have resulted in a large industry producing mainly high quality pelleted and coarse mix feeds (sweet feeds) often tailored for the different types or uses of the horse."[3]

The results of NAHMS survey of 1998 indicated that 87% of the grain/concentrate fed to horses was purchased from a retail source in bags, 5% was purchased as bulk delivery and only 6% was home grown.[1]   Unpelleted sweet feed (grain mixed with molasses and usually fortified with protein, vitamins, and minerals) was fed on 57% of all equid operations.   Complete feed pellets or grain mix with pellets was fed on 40% of the operations.   The commercial feeds are particularly useful in the United States because, according to the NAHMS survey, 79% of all equine operations have five or fewer horses.   These operations are not likely to have significant feed mixing and storage facilities. It is much more convenient to purchase a feed and not be concerned about the need to add proper amounts of micronutrients or find storage for the basic ingredients. (However, about one-third of horse owners still purchase vitamin and mineral supplements.)   Commercial feeds also provide diets specifically designed for various classes of horses such as weanlings, broodmares, performance, and older horses, and even supply special diets for horses with conditions such as hyperkalemic periodic paralysis (HYPP) or equine polysaccharide storage myopathy (EPSSM).

Good quality pasture is still an excellent basis for a feeding program.   The old saying that "Dr. Green is an excellent veterinarian" is still true.   Proper use of pasture provides a much higher level of antioxidants such as vitamin E and carotene than are present in hay.   Dr. David Pugh of Auburn University stated in a presentation at the North American Veterinary Conferences in 2000 that "grass is magic but hay is dead."[4]   Certainly much of the antioxidant content is lost during drying and storage. Pasture can reduce the incidence of colic, ulcers, signs of respiratory diseases and abnormal behaviors.

It is frequently suggested that horses evolved as grazers eating numerous meals and therefore should be fed in that manner.   Of course not all early horses were grazers.   MacFadden et al. re-

ported that skeletons of horses of 5000 years ago (late Hemphillian) found in Florida were grazers, mixed feeders, and browsers.[5]   However, the more immediate ancestors of modern day horses were grazers.

Of course pasture is not a perfect diet.   Excessive intake of lush pasture can cause founder and colic. Pasture may be lacking in certain minerals depending on the content of the soil.   Toxic plants might be present or harmful products such as endophytes may be present in the grass.   The USDA recently tested 888 samples of fescue. Nationally, they found a total of 61.6% of the samples tested positive for endophyte, *Neotyphodium coenophalium.*[6]   The endophyte was found in 56.3%, 76.4%, 61.9%, and 61.2% of the pastures in the Northeast, Southern, Central, and Western regions respectively.   It was suggested that less than half of the stems tested in most infected pastures were test positive for endophyte, but that without intervention, low initial pasture infection rates will increase over time due to the infected fescue grasses' ability to out-compete non-infected fescue grasses.

It was stated "The level of toxin in pasture at which disease will occur in horses is dependent on the amount of pasture consumed and the horse's status (late in pregnancy vs. not pregnant).   However, any detectable level of toxin may be harmful to horses.   Horses in all regions of the U.S. are at risk of developing problems associated with endophyte-infected pastures as all regions had pastures that were positive for both the endophyte and the toxin. Disease potentially caused by the toxin on participating operations was not evaluated."[6]

Pasture can also be a source of parasite infestation.   Prompt removal of feces will greatly reduce the parasite load and improve pasture utilization. Horses will not normally graze on pasture near fecal piles.   However, they will graze near piles if the pasture is in short supply.

Many parasites or other life forms that need to be in the digestive tract are not proactive.   They wait until an animal ingests them at the proper stage of life.   But there are some forms that take a proactive stance to increase their chances of ingestion.   One of them is *Pilobolus,* which appears to be a favorite of many mycologists.

Pilobolus, a fungus, has an interesting method of increasing the chances of surviving a life-cycle. *Pilobolus* lives in animal feces and is useful for the decomposition of feces.   However to complete the life cycle, the spores of the fungus must pass through the digestive system.   Therefore, the horse or cow must graze on grass containing spores. *Pilobolus* has evolved a way to shoot its spores on to the grass that animals will ingest.   "Its shotgun is a stalk swollen with cell sap, bearing a black mass of spores on the top.   Below the swollen tip is a light-sensitive area.   The light sensing region affects the growth of *Pilobolus* by causing it to face toward the sun.   As the fungus matures, water pressure builds

in the stalk until the tip explodes, shooting the spores into the daylight."[7]   Buller reported that "the spores fly away at 35 feet per second (10.8 m per second), to a height of six feet (2 m) and horizontally as far away as 8.5 feet (2.8 m)."[8]   *Pilobolus* is Latin for "hat thrower." Buller wrote about the "great violence in the discharge of the projectile."[8]   After the herbivore eats the grass contaminated with the spores, the spores pass through the digestive tract of the host animal.   Buller further wrote, "And so the spores are deposited in the solid excreta which the animals drop to the ground.   In these excreta the spores germinate and the mycelium so produced ultimately gives rise to new gun-producing fruit bodies."   Buller concluded that the guns and projectiles of *Pilobolus* "are beautifully adapted by their structure and functions to bring about dispersion of the spores."[8]

What does *Pilobolus* have to do with the nutrition of the horse?   Probably nothing, but it may influence the health of the horse.   *Pilobolus* has been reported to transfer infective *Dictyocaulus arnfieldi* larvae from feces to grass and thus spread lungworm in cattle[9,10] and elk.[11]   Jorgensen and Andersen suggested that the *Pilobolus* mechanism may also play an important part in the spread of equine lungworm.[12]

For many horse owners good quality pasture is not an option due to climate, lack of land or other reasons.   However, the NAHMS survey of 1998 indicated that the majority of operations in 1997 had some pasture available during the summer months.[1]

Surprisingly, although many countries have increasingly become urbanized, the percentage of the horses in those countries with access to pasture may not differ from some other earlier periods as much as one might expect.

The large cities, in the late 1800s and early 1900s, required a great number of horses for hauling of goods and transportation.   I discussed the following recently.[13]   Michael Crichton, author, has written many interesting novels such as *Disclosure, Rising Sun, The Andromeda Strain,* and *Jurassic Park.* The novel written by Mr. Crichton that I enjoyed the most is *The Great Train Robbery.*   In addition to an excellent plot, Mr. Crichton also provided insights into life in London in the 1800s.   He pointed out that there was great concern over traffic jams caused by horse-drawn vehicles.   He wrote, "In the midst of this [traffic jam] the street sweepers began their day's labours.   In the ammonia-rich air, they collected the first droppings of horse dung, dashing among the carts and omnibuses." And they were busy: "an ordinary London horse, deposited six tons of dung in the streets each year and there were at least a million horses in the city" according to Henry Mayhew (cited by Crichton).

It is unlikely these horses had significant exposure to pasture.   The large numbers of horses were housed in other major cities such as New York and Chicago.   They were often housed in poorly venti-

lated, filthy stalls with no access to pasture. Many of these horses developed calcium and other nutrient deficiencies. The absence of good quality pasture necessitates that greater attention be paid to the feeding program to ensure that the supply of nutrients is adequate.

In the past, pasture research probably did not receive the priority it deserved. But in recent years interest has increased in this research. For example, important pasture projects are being conducted at universities such as University of Florida, Texas A&M, and University of Kentucky. Dr. David Kronfeld and his associates at Virginia Tech have focused on identification of nutrient shortage in pasture and methods for supplementation. Their studies have led to the development of a fortified high fat, high fiber product that has great potential.

It was concluded that pastures (at least in the eastern U.S.) need to be supplemented with phosphorus, selenium, copper, zinc, and vitamin A.[14] They also stated "providing a fat and fiber supplement designed to complement the pasture avoided the spring slump in growth of yearlings and increased immunoglobulin-G and linoleic acid in mares' milk."

Of course pasture can be great, but it is not essential. A principle of feeding is that there is no one best ration. Many different ingredients can be used in horses if the proper amount of nutrients are provided. The ingredients used are determined, in part, by which ingredients are readily available. For example, ponies in Iceland are fed fish. Australian horse rations may contain lupins, tick beans, rice, or copra (coconut meal). Triticale (a cross of wheat and rye) and extruded feeds are increasing in popularity in Australia.[15] English horses are more likely to be fed carrots and herbs than are American horses.[16]

Many developments in horse feeding seem to be stimulated by developments in the feeding of other species. For example, extruded feed was first fed to horses by Dr. John Knight in the early 1980s at the Armstrong Brothers Farms because he saw that it worked well for dogs and he thought that extruded feed could reduce the incidence of colic in young horses that were group-fed. Feeds for older horses were introduced after foods for older dogs were manufactured. Soon after herbs were added to dog foods, they were added to horse feeds. The interest in herbs for horses is likely to increase. A recent Zogby America poll indicated that 40% of Americans have used herbal remedies for various illnesses.[17] There is considerable interest in complete feeds for horses. Of course several complete feeds are currently on the market but my opinion is that future innovations will make complete feeds even more popular.

Recent Research—Digestive Function

"The present is too close at hand for recent advances to be seen in perspective. Current fashion can so easily be given to great significance."[18]

Following is a discussion about the sites of nutrient digestion and absorption and also the potential manipulation of the environment in the digestive tract in order to improve the health and nutrition of the horse.

Colic is still considered to be the most common cause of death of horses even though new deworming compounds have been more effective in the control of parasites than previous products. Feeding mistakes and mismanagement are now probably the number one cause of colic. The guide for horse owners on prevention of colic[19] lists 16 recommendations to reduce the incidence of colic. At least 13 of the recommendations deal with factors that influence the ecology of the intestine. In spite of the fact that the importance of the ecology of the intestine is well known, relatively little research has been conducted on the manipulation of environment. Furthermore, owners often appear to forget a fundamental principle of equine nutrition. Feeding the microflora properly is critical to the health of the horse. That is, when you are feeding horses, you must make certain the microflora are happy.

A. Ecology of the Hind Gut

Dr. K. A. Dawson, in a keynote lecture at the Equine Nutrition and Physiology Symposium in 1999, stressed that the ecology of microflora in the hind gut is important for the digestive process, to stimulate normal gut development, to detoxify some compounds and to prevent build-up of harmful bacteria.[d]

The hind gut has three major groups of microflora. 1) The bacteria. Dawson pointed out that there are more than 50 significant species.[d] They are present in a high concentration (8–10 billion/ml of fluid). They can utilize fiber, starch, and protein. Perhaps it may come as a surprise to some that only 23% of the bacterial species in the hind gut of the horse utilize ammonia compared to almost 100% of the species in the rumen. 2) The protozoa. Dawson reported there are fewer than 50 species of protozoa. They have a low concentration; (only 10,000–100,000/ml of fluid). They are not essential for normal function and significant amount of protozoa are only found in 50% of the horses. 3) Anaerobic fungi. Dawson reported the fungi were identified only about 20 years ago. Fewer than 30 species are known. They probably have a function in fiber digestion but the significance is not well known.

Dawson listed several methods for the manipulation of the microflora. One of the primary methods is the hay to grain ratio. The control of pH is essential. Acidosis in the hind gut can cause founder and colic.

Chronic alkalosis could increase the incidence of enteroliths. Several factors such as the intakes of magnesium, phosphorous and nitrogen, pH of the hind gut, presence of a nidus, breed of horse, and alfalfa intake have been associated with enteroliths. The intakes of magnesium, phosphorous and nitro-

gen are of interest because the majority of en- teroliths are struvite, a material composed of the above three elements.   Struvite is more likely to be formed in a pH of 6.5 or higher, therefore pH of the hind gut can be a factor.   A nidus is needed to start the precipitation of the struvite.   In most studies, Arabians are the horses most likely to have the highest incidence of enteroliths.   The reason for the high incidence is unknown.   I think the Arabian horses in California have a higher incidence than breeds such as Thoroughbreds, in part, because Ara- bians are fed less grain and therefore are more likely to have a higher pH in the hind gut.   But other factors may also be involved.   Results of several studies in regions where enteroliths are not common have suggested an association between Arabian horses and colic.   Reeves et al.[21] studied data sup- plied by veterinary hospitals at the University of Guelph, The Ohio State University, University of Pennsylvania, and Tufts University.   Arabian horses were more than twice as likely to be colic cases.   Cohen and Peloso[22] reported that Arabians were more likely to have colic than other breeds based on data from cases treated by Texas veteri- narians.   In their study, the Arabian breed was not associated with any particular type of colic.   Dart et al.[23] studied data from Veterinary Medical Teaching Hospital, University of California at Davis.   They found that Arabian, Morgan, and Appaloosa horses were more at risk for cecal impaction than other breeds.

White and Edwards[24] suggested the association of colic and Arabian horses may be related to differing management practices for Arabian horses, a greater concern about colic and its management by owners of Arabian horses, or perhaps there is a genetic predisposition to gastrointestinal disorders among Arabian horses.   Cohen and Peloso[22] suggested that perhaps Arabians "may be more likely to man- ifest signs of pain than other horses."   But not all studies show that Arabians have a higher incidence of colic.   A prospective study with horse owners in Virginia and Maryland found that the incidence of colic was almost one-third lower in Arabian horses.[25]

Studies in California indicate most horses with enteroliths have been fed alfalfa (of course most horses in California are fed alfalfa).   However, grass hay is likely to produce a lower pH in the hind gut of horses because of the buffering capacity of alfalfa.[26] Furthermore, alfalfa provides the hind gut with significant amounts of nitrogen, and alfalfa raised in some parts of California contains a higher magnesium content than found in alfalfa raised in the Midwest and East.   Also, alfalfa usually has a higher digestible energy content than grass hay and therefore less grain is needed when feeding alfalfa. Less grain could lead to higher pH.

The addition of vinegar has been reported to decrease the pH in the hind gut of ponies.[27] Therefore, 2 cups of vinegar per day has been rec-

ommended as a preventative method.   No clinical trials have been conducted to prove that vinegar would be of value, but I have had California horse owners tell me that the incidence of enteroliths has decreased since they have been using vinegar. Hassel et al.[28] conducted an excellent evaluation of 900 cases of enteroliths from the medical records at the University of California Veterinary Medical Teaching Hospital at Davis.   They found that 7.7% of the study population had a recurrence of en- teroliths.   Horses with recurrent enterolithasis had less reduction of alfalfa in the diet ($p = 0.1$) and less dietary vinegar ($p = 0.09$).   The authors rightly concluded that "conclusions cannot be made regard- ing the effectiveness of vinegar supplementation in the prevention of enterolithiasis on the basis of results of this retrospective study."[28]   However I think the anecdotal reports and the association ob- served in the Hassel et al. report could be a basis for justification of clinical studies on the use of vinegar to prevent enterolith formation.

Another approach to reduce pH in the hind gut might be to use fructans.[b] As discussed elsewhere in this article, fructans are not digested in the small intestine but pass into the hind gut where they are fermented by the microflora and the pH of the hind gut is lowered.   Of course excessive fructans intake could lower pH such that colic and/or laminitis might result.   I think the potential benefits for fruc- tans are significant, but obviously, clinical studies must be conducted before any recommendations can be made.   As mentioned elsewhere, fructans are available commercially and also can be found in significant amounts at certain times of the day in certain grasses.

Excessive grain intake can create a more acidic environment than desired.   For example, Radicke et al.[29] reported that seven hours after a meal of hay, the pH of cecal fluid was 7.15 compared to 6.65 when a meal of corn was fed.   Thus, fiber is valu- able for the regulation of pH and prevention of aci- dosis.   The type of starch can be critical.   The effect of the type of starch on pH may be determined by site of digestion.   This topic will be covered in more detail in the section on site of digestion, but basically starch such as that in oats is more effec- tively digested in the small intestine than starch from barley.   The more starch reaches the intestine, the more acidic the pH becomes.

How much fiber is needed to maintain gut func- tion?   The National Research Council (NRC) does not provide a requirement for fiber.[30]   It is stated by NRC that all diets for horses should contain adequate amounts of roughage with at least 1 kg of roughage dry matter per 100 kg of body weight. But the concentration of fiber in the roughage is not specified.   Perhaps in the next edition of NRC more information about fiber will be supplied.   It would be useful if types and amounts of various fibers that maintain normal intestinal function were specified. However, few studies have been conducted to estab-

lish such values. We found that diets providing as little as 24% NDF or 14% ADF apparently maintained gut function. Wolter[31] recommended that diets for horses should contain at least 17% cellulose, 20% NDF, or 12% ADF. Ferrante et al.[32] fed a diet of dried shelled corn and ground alfalfa hay in a 2:1 ratio with 10% molasses for binding and palatability. The final ration contained 13% crude fiber and 16% ADF. The horses performed low-intensity submaximal exercise. No clinical abnormalities were observed.

Although the horses in the above studies appeared to have no digestive upsets, I would be reluctant to recommend that such low fiber diets be fed routinely. I think 1.5 to 2 kg of hay or hay equivalent per 100 kg of body weight would be more reasonable. Further studies are needed to define the amount of fiber needed when the fiber is supplied by products such as soybean hulls, beet pulp, and citrus pulp. All fibers are not equal.

Dawson reported that parasiticides can influence the ecology of the gut. This could explain the colic observed after horses have been dewormed.[33] Probiotics have been defined as live microorganisms that confer health effects on the host when consumed in adequate amounts.[34] This is of great interest for the potential to improve the health of humans.

It has been suggested that probiotics in humans might influence the incidence of colon cancer, have a mediating antihypertensive effect, decrease urogenital infections, lower cholesterol and influence *Helicobacter pylone* infections in the stomach.[34]

Rolfe wrote, "Probiotics represent a potentially significant therapeutic advance. In an effort to decrease reliance on antimicrobials, the time has clearly come to increase the exploration of the therapeutic applications of probiotics. Probiotics offer dietary means to support the balance of the intestinal flora [in humans]."[35]

DiRienzo wrote, "In Japan and Europe, a large number of products that contain probiotics, prebiotics and synbiotics to promote health and well being [of humans] are available."[36]

What is the value of probiotics for horses? It has been suggested that a yeast culture preparation could stimulate the fibrolytic activity of the large intestine fluid contents when horses are fed diets containing high levels of fiber. Moore et al. found that yeast cultures could modify microbial populations and VFA production in the hind gut.[37] Yeast cultures have also been reported to improve nitrogen retention,[38] to increase utilization of fiber,[39] and phytate phosphorus[40] for the horse. However, yeast culture did not aid in the digestion of diets containing 50% grass hay and 50% grain[41] or rice hulls.[42]

Antibiotics can have helpful or harmful effects. Excessive amounts of antibiotics can destroy the bacterial population. Others can be very useful. Viginiamycin as Founderguard® has been reported

Table 1. Estimates of LD_{50} Dose of Ionophores to Horses, Cattle, and Chickens (mg/kg BW)

	Horse	Cattle	Chickens
Monensin	2–3	20–80	200
Salinomycin	0.6	n/a	44
Lasalocid	21.5	50–150	72

Adapted from Roder and Stair.[47]

to have the potential to decrease the incidence of founder when high intakes of grains are fed because the antibiotic reduces fermentative acidosis in the hind gut.[43] Johnson et al. reported that the addition of Founderguard® reduced the incidence of abnormal oral behaviors, such as wood chewing, eating of bedding and stall licking, in horses fed a diet containing 3:1 grain to forage.[44] The feces of horses fed the diet without Virginiamycin had a lower pH than when Virginiamycin was fed. Earlier, Willard et al. reported that horses fed a high concentrate ration had a reduced cecal pH and spent more time chewing wood and engaged in coprophagy than those horses fed hay.[45]

Ionophores are used to alter fermentation in the rumen, to increase efficiency of feed utilization and as coccidiostats. Ionophores increase the permeability of cell membranes to a specific ion. The ionophores used in animal feeds are produced by saprophytic fungi. Blomme et al. stressed the extreme sensitivity of horses to the toxic effects of ionophores.[46] The LD_{50} of monensin for chicken (mg/kg of body weight) is 70–200 times that for horses (Table 1). Many ionophores are marketed (Table 2) primarily for poultry and beef cattle but also for turkey and sheep. Blomme et al. pointed out that "the greater sensitivity of the equine species is of particular concern because of the closeness of habitat and similar dietary needs that commonly prevail between horses and cattle."[46] Furthermore,

Table 2. Ionophores Used in Animal Feeds

Ionophore	Trade Name	Target Species	Benefit
Monensin	Rumensin	Beef cattle	Improved feed efficiency and rate of gain
Monensin	Colban	Chickens	Coccidiostat
Salinomycin	Sacox	Chickens	Coccidiostat
Salinomycin	Bio-Cox	Chickens	Coccidiostat
Lasalocid	Avatec	Chickens	Coccidiostat
Lasalocid	Bovatec	Beef cattle	Improved feed efficiency and rate of gain
Narasen	Monteban	Chickens	Coccidiostat
Semduramicin	Aviax	Chickens	Coccidiostat
Laidlomycin propionate potassium	Cattlyst	Beef cattle	Improved feed efficiency and rate of gain

Adapted from Roder and Stair.[47]

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no specific antidote is available. Because of the widespread use of ionophores, horse owners should be made aware of the dangers of ionophores and become familiar with which products are ionophores or contain ionophores. Monensin, salinomycin, and lasolocid are probably the three most well-known ionophores but, as shown in Table 2, several other ionophores exist.

Clinical signs of toxicosis include anorexia, profuse sweating, colic, depression, incoordination, hyperventilation, tachycardia, and EKG alteration, prostration, and death; the heart becomes the primary target organ. Signs of chronic toxicity that often may not be noted until weeks or months after withdrawal of the drug include unthriftiness, poor performance, or sporadic sudden death.

Unfortunately, toxicity levels have yet to be determined for many ionophores, but estimates of LD_{50} for monensin, salinomycin, and lasolocid, respectively, can be seen in Table 1. In the past, ionophore toxicosis has resulted from overdose due to mixing errors, misuse in nontarget species, and failure to properly clean feed-mixing or delivery facilities when using feed containing ionophores prior to their use for horse-feed. In feed mills that prepare feeds for a variety of species, including those more tolerant of ionophores than horses, faulty equipment formerly allowed ionophores to be added to horse feeds.

Ionophores are added to ruminant rations to increase feed efficiency by altering microbial activity in the rumen. Blomme et al. concluded the exact mechanism of ionophore toxicity is not known but suggested that interference with membrane cation transport could cause cell death by destabilizing biological membranes.[46] Myocardial tissues may be affected by the excessive cardiac myocyte calcium concentration. Roder and Stair concluded the transport of specific ions (Na^+, K^+, and Ca^{++}) across membranes accounts for the pharmacologic effects of ionophores.[47]

The dangers of ionophores for the horse were first recognized in the 1970s, but episodes still occur in this country and elsewhere. For example, Bezerra et al. reported three outbreaks of monensin toxicosis in Brazil.[48] Twelve horses died after eating a commercial ration labeled for feeder calves. The feed contained 180 ppm sodium monensin. As mentioned earlier, there is a constant flow of new horse owners that needs to be educated.

Conditions listed in the reviews by Blomme et al.[46] and Roder and Stair[47] that should be considered in the differential diagnosis include: exertional myopathy, myodegeneration induced by cardiotoxic plants, myocardial alternations from calcinogenic plants, blister beetle intoxication, nutritional myopathy, neurogenic cardiomyopathy (brain-heart syndrome), ischemic myocardial necrosis due to thrombotic disease, myocardial degeneration, and fibrosis of aged horses.

Prebiotic has been defined as a nondigestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of one or a limited number of bacterial species of the colon, and thus improves the health of the host. Prebiotics range in size from small sugar alcohols and disaccharides, to oligosaccharides and large polysaccharides.

Fructans, nonstarch storage polysaccharides of plants, are examples of prebiotics. They have received a great deal of attention from human nutritionists in recent years because of their potential to promote health. The fructans, inulin (a term applied to a blend of fructopolymers) and fructo-oligosaccharides, also called oligofructose, have received the most attention.[49] Fructans are not digested in the small intestine but are fermented by bacteria in the large intestine. Studies in rats indicate that the fermentation of fructo-oligosaccharides decreases intracolonic pH and increases butyrate production.[50] In humans, fructo-oligosaccharides have been suggested to reduce detrimental bacteria, reduce toxic metabolites, prevent the development of pathogenic bacteria, prevent constipation, reduce serum cholesterol, and have an anti-cancer effect. Furthermore, fructo-oligosaccharide provides about 30 to 50% of the sweetness of sucrose. It could be used to improve the flavor and sweetness of low calorie food and, because it does not have a glycemic effect, could be used in diets of diabetic people.

Niness reported that most of inulin and fructo-oligosaccharide commercially available on the food ingredient market today is either synthesized from sucrose and extracted from chicory roots which contain 15 to 20% inulin and 5 to 10% fructo-oligosaccharide.[49] Foods that make significant contributions to the fructan intake of humans are wheat (71%), onions (24%), bananas (2%), and garlic (2%).[51]

Horses are not likely to eat large amounts of the above foods but they can get too much fructan intake from grasses such as ryegrass. As mentioned above, fructans are rapidly fermented by the microflora of the hind gut. Excessive intakes of fructans and rapid fermentation could lead to acidosis and laminitis. Horse operations reported to NAHMS that 2.1% of their horses had laminitis during the previous 12 months.[52] About 13% of all horse operations in the survey had one or more resident horses with laminitis during the previous 12 months.

Grazing of lush pasture was the single most commonly perceived cause of laminitis. Other causes are shown in Table 3. Lush pasture could likely have a high fructan content. The perception that lush grass was the most common cause seems to agree with the reported seasonal effects. In the Northeast, Western and Central regions, the average of horses with laminitis was 6.0% in the spring season whereas the average of the three areas was only 2% in the winter. In the Southern region, the

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Table 3. Percent of Laminitis Cases by Perceived Cause[52]

Perceived cause	Percent Laminitis Cases	Standard Error
Grazed lush pasture	45.6	(11.5)
Other known (misc.)	26.9	(11.1)
Unknown	15.4	(6.4)
Grain overload	7.4	(3.7)
Colic or diarrhea	2.7	(2.7)
Retained placenta	2.0	(1.6)

highest incidence was in the summer (6.0%) versus 2.6% in the spring and 1.1% in the winter.

Several methods have been suggested to decrease the incidence of laminitis from lush pasture, such as limiting time on pasture, adjusting the horses by gradually increasing time allowed on pasture, feeding hay before turning the horse out to pasture, and providing hay in the pasture. One of the most novel methods might be to pasture horses at night. Researchers at the Grasslands Research Institute in Wales found that ryegrass can contain a high concentration of fructan during the day, but the fructan content rapidly decreases when the sun goes down.[53] Studies on fructan contents of other grasses are needed.

Synbiotics are combinations of probiotics and prebiotics that beneficially affect the host by improving the survival and the implantation of live microbial dietary supplements in the gastrointestinal tract by selectively stimulating the growth and/or by activating the metabolism of one or a limited number of health promoting bacteria.[36] No studies were found in which synbiotics were fed to horses.

B. Site of Digestion

The rate of digestion and absorption of nutrients such as protein, carbohydrates, and minerals can have a significant effect on efficiency of utilization and environment of the intestine. Many studies on the site of digestion have used markers such as chromium oxide in slaughtered animals or in fistulated animals. More recently several studies have been reported in which nylon bags were used.[54]

Protein

Protein that is digested in the small intestine and absorbed as amino acids is much more effectively utilized and valuable than protein that passes through the small intestine. Although some of the nitrogen is utilized by bacteria in the large intestine, amino acids in microbial protein are not effectively absorbed from the large intestine. Some of the absorbed nitrogen can be used in the synthesis of non-essential amino acids or other products. A significant amount of the nitrogen released from protein by bacterial action is absorbed and is excreted in the urine.

The NRC described protein requirement as crude protein.[30] It was thought there was little to gain from using apparent digestible protein because such values are greatly influenced by level of protein intake. The true digestibility of protein in most traditional horse feeds is high.

In France, a more interesting approach is used.[55] Not only is digestibility considered, but also the site of digestion and therefore an estimate of the amount of absorbable amino acids is obtainable. The unit, called matières azotées digestible cheval (MADC) or, in English, horse digestible crude protein, was introduced in the mid 1980s.

Such a system could be superior to the crude protein system used by NRC. The relative value of feed sources of amino acids could more accurately be evaluated. More importantly, application of the system would permit feeding methods that could decrease the amount of nitrogen returned to the environment. Nitrogen and phosphorus disposal on horse farms will very likely receive greater attention from governmental agencies in the future. My concern with the MADC system is the variation in the reported estimates of the percentage of the nitrogen that was absorbed from the small intestine in a given feed.

Martin-Rosset suggested that relative value of grains and protein concentrates was similar when using digestible protein or MADC.[52] I made some comparisons using crude protein and found similar results (Table 4). Martin-Rosset found, however, that forages should be discounted based on the estimate of amounts of nitrogen that reached the hind gut (Table 5).[55] The values used to estimate the discounts were primarily developed from studies in France, Germany, and Texas A&M. I think the MADC is an excellent start, but further steps are needed. The amino acid requirements of the horse need to be better understood as well as the absorption of individual acids determined. For example, soybean meal is a reasonable source of lysine but gluten feed is not. Thus the relative ranking of gluten feed to soybean meal should be even less than was determined by crude protein and MADC standards.

Table 4. Value of Grains and Protein Feeds When Evaluated by Crude Protein and MADC Standards[a]

	Crude Protein (g/kg)	Relative Value	MADC (g/kg)	Relative Value
Grains				
Barley	117	100	92	100
Corn	103	88	79	86
Oats	132	113	98	106
Protein Feeds				
Soybean meal	545	100	496	100
Linseed meal	380	70	323	65
Gluten feed	233	43	191	39

[a]Values taken from Martin-Rosset.[106]

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Table 5. Factors for the Calculation of MADC Values of Feedstuffs from Digestible Protein Content (MADC = DP × k)[55]

Factor	Type of Feed
k = 1	Concentrate
k = 0.9	Green forage
k = 0.85	Hays and dehydrated forages
k = 0.80	Straws and high lignin byproducts
k = 0.70	Silage

I feel that eventually an evaluation system that includes amino acid absorption will be developed. Ration balancing for efficient utilization of amino acids could significantly decrease nitrogen waste. The amount of protein concentrates fed to horses could be greatly reduced.

Starch

The site of digestion of starch, as mentioned earlier, can influence the incidence of digestive problems in the horse because starch reaching the hind gut can increase the incidence of acidosis. Cuddeford reported that the rate and extent of starch digestion in the small intestine is determined by:[56]

1. The properties of the starch granule
2. The effect of processing
3. Plant cell walls
4. Transit time through the small intestine
5. The availability and concentration of enzymes

Total tract digestibility of starch in grains is usually almost complete. That is, significant amounts of starch are seldom found in the feces. The properties of the granule can greatly influence site of digestion.

Cuddeford reported that starch in peas (retrograded amylose) is entirely resistant to amylose digestion in the small intestine.[56] Starch granules that have a B or C crystalline structure is highly resistant to digestion. Studies at Texas A&M indicated that 78, 91 and 94% of the starch in corn, oats, and sorghum was digested in the small intestine.[57] Meyer et al. measured the pre-ileal digestibility of oats, corn, barley, potatoes and manioc.[58] The grains were fed whole or processed. The starch intake was about 2 g/kg body weight per meal. The starch of whole or rolled oats was 80–90% digested prior to the ileum. In contrast, the starch in whole or cracked corn was only 30% digested. Grinding the corn increased pre-ileal digestibility to 51% and micronizing increased digestibility to 90%. Barley starch had a pre-ileal digestibility of only 26%. Previous to these studies I thought that oats was a safe feed because of the higher fiber content. These data indicate that type of starch may be even more important than the fiber. Oats is less likely to cause colic or founder because less starch reaches the hind gut. I remember years ago reading an article written by a veterinarian in the late 1800s. Unfortunately I cannot remember the reference. But I remember he stated that it was his experience that barley was much more likely to cause founder than oats and that barley was as likely as corn to cause founder. At the time, I thought he may have been mistaken. Barley has more fiber than corn and therefore should be "safer". The work of Dr. Meyer demonstrates that the veterinarian was correct. Barley starch would be as likely to influence the ecology of the hind gut as the starch in corn, even though more fiber reached the hind gut.

Meyer et al. concluded that since rolling of oats had no effect on small intestinal digestion, oats needed no processing when fed to horses with intact teeth.[59] It was recommended that corn and oats should be processed before feeding. Feeding of hay about the same time as feeding grain could depress digestion of starch in the small intestine. Therefore, Meyer et al. recommended that hay be fed before feeding concentrates or that hay be fed considerably later than concentrates.[59]

Cuddeford studied the effects of the processing of barley starch.[56] The apparent total tract digestibility and therefore estimated energy contents of rolled, micronized, and extruded barley were similar for all three forms. However, based on in situ, in vivo and mobile nylon bag studies, it was concluded that the feeding of micronized barley could result in less undigested starch entering the large intestine than when rolled barley was fed.

Cuddeford concluded, "It is apparent that horse feeders must minimize the flow of fermentable polysaccharide to the large intestine of the horse to maintain health and to maximize substrate (glucose) availability for the performance horse."[56] He stated the goal could be achieved with starch "simply by feeding highly degradable micronized cereals little and often." The processing of oats apparently would have minimal effect, but the effect on barley and corn would be significant.

The studies mentioned above usually involved fistulated animals and were laborious and time consuming.

Pagan et al. reasoned that relative starch digestion could be evaluated by measurement of the blood glucose response to a feed.[60] That is, the blood glucose response would be greater in feeds that had more starch digested in the small intestine. Steam-flaked, cracked, and ground corn were compared. Steam-flaked corn produced a greater glycemic response than cracked or ground corn and therefore probably had a higher percentage of starch digestion in the small intestine.[61]

Whole oats had a higher glycemic response than cracked corn. Higher intakes of oats actually decreased glycemic response, indicating less absorption from the small intestine, perhaps because of an increased transit time through the small intestine.[62] As Pagan et al. indicated, "more research is needed to determine the relevance of glycemic response in

predicting the effects of different feed ingredients on a horse's performance or behavior."[60]

Economic studies would also be useful. How much of a response is required to make processing such as micronizing practical? Of course, that would depend on the type of horse. The response of a backyard horse might be difficult to measure, whereas the performance of a racehorse could be dramatic. The difference between being first or second could mean significant amounts of money and glory.

Nutrient Requirements of the Horse

The standard for nutrient requirements in the United States has been the NRC's publication, *Nutrient Requirements of the Horse.* The most recent edition was published in 1989. It is expected that a new committee will be formed before this article is published and that a new edition of NRC will be published within 2 to 3 years. What changes might be considered?

Energy

Lawrence recently reviewed the digestible energy requirements of horses.[63] For growing horses, she compared the predicted values with actual results from five studies conducted in the 1990s. She calculated the energy requirements of growing horses as predicted by the 1989 NRC equation. The equation includes a requirement for maintenance and factors for average daily gain and age. The predicted values were similar to actual values for horses 12 months of age and under. The predicted values exceeded the actual values for yearlings. She concluded that the energy requirements of long yearlings and two-year-olds in training need further study.

She pointed out that the 1989 NRC publication does not include recommendations for nursing foals and that such information should be included. She further concluded that information generated in the last decade about the growth rate of nursing foals and information on milk composition could be used to estimate the energy requirements of foals from birth through 2 months of age.

The 1989 NRC recommendations suggest that maintenance digestible energy levels are adequate during the first 8 months of gestation and that energy requirements should be increased 11, 13, and 20% above maintenance in the 9th, 10th, and 11th months, respectively. These estimates were based on studies, conducted in Germany, of the composition and growth of the fetus.

Lawrence pointed out that some studies indicate that the mare is likely to gain significant weight during the second trimester.[63] Therefore, perhaps an increase in energy intake probably takes place earlier than previously expected. Thus the energy requirements might not be as great as suggested by NRC during the last trimester and greater than expected during the second trimester. This would

Table 6. Average Body Scores for Equine Athletes[a]

Function	Average	Range
Racehorses	5.0	(4.0–7.0)
Endurance	4.6	(3.5–5.5)
Three-day event	4.9	(3.6–6.0)

[a]Based on review conducted by Lawrence.[63]

mean the pregnant mare uses energy the body stores to help support the growth of the fetus during the last month of gestation.

The NRC requirements for lactating mares were based on the assumption that mares were likely to produce milk equivalent to 3% of body weight during early lactation and 2% of body weight during late lactation. Of course, such a stepwise effect would not be expected in nature. Perhaps there is now adequate data to develop a lactation curve for mares that would be more realistic. Lawrence suggested that some studies in the 1990s indicated that the NRC values for milk production may be too low and therefore energy requirements for lactating mares may be too low.[63] Lawrence suggested the 1989 energy recommendations for breeding stallions are realistic but further studies are needed to verify the optimal body condition and energy intakes. Lawrence suggested the 1989 NRC recommendations for moderate and intense work are relatively realistic. Although equations such as produced by NRC are useful as a starting point, the ultimate answer as to the energy needs depends on the body condition of the horse. Body condition is of particular importance for the performance horse. Excess weight would decrease performance, whereas horses that lack body condition or are too thin could lack stamina. Lawrence reviewed several studies and reported the body condition scores shown in Table 6 for horses of various activities. She also calculated the estimated increase in digestible energy intake needed to change a body condition score (Table 7). This table can be very useful to indicate expected weight gains to owners and to encourage them not to feed for excessively rapid changes in body condition.

Table 7. Estimated Increase in DE Intake Necessary to Change the Condition Score of a 500 kg Horse from 4 to 5[a][63]

Time to Accomplish Gain	Daily DE Above Maintenance (Mcal/d)	% Increase in DE Above Maintenance
60 d	5.3 to 6.7 Mcal	32 to 41%
90 d	3.6 to 4.4 Mcal	22 to 27%
120 d	2.7 to 3.3 Mcal	16 to 21%
150 d	2.1 to 2.7 Mcal	13 to 16%
180 d	1.8 to 2.2 Mcal	11 to 14%

[a]Assumptions: 1 unit of change of condition score requires 16 to 20 kg of gain and 1 kg gain requires 20 Mcal DE above maintenance.

Protein

Ott reviewed protein and amino acid requirements.[64] He concluded that there is little new information on the protein and amino acid requirements of horses at maintenance. Likewise, he concluded that few studies are available to verify or modify the protein recommendations for gestating or lactating mares. He reported that some studies indicate that the lactating mare will respond to amino acid availability by altering milk composition and that the foal will respond to improvements in amino acid balance by increasing rate of growth.

Lysine was the only amino acid for which a requirement was estimated for growing horses in the 1989 NRC. Ott concluded that studies conducted since 1990 verify the 1989 NRC requirements for protein and lysine. He referred to two studies that indicated threonine is likely to be the second limiting amino acid.[64] Perhaps the next NRC publication will be able to include estimates of the requirement for threonine.

Graham-Thiers et al. demonstrated that high protein diets are not essential for working horses if the proper amino acids are available.[65] They compared a diet containing 7.5% crude protein and supplemented with lysine and threonine to a diet containing 14% crude protein. The low protein diet did not appear to cause any harmful effects and, in fact, was not as acidogenic and resulted in less nitrogen excretion and therefore less water loss than the 14% crude protein diet.

The importance of branched chain amino acid (leucine, isoleucine, and valine) remains to be determined. It has been suggested that these amino acids could decrease plasma lactate and heart rate in horses working on a treadmill. However, not all studies have shown a benefit from branched chain amino acid supplementation and it is suggested that a high quality protein could meet the requirement for branched chain amino acid without need for supplementation.[64]

Calcium

The requirements for calcium have been reviewed by Ott[64] and Hintz.[66] Both concluded that the NRC (1989) calcium requirements for horses at maintenance, growing horses, and pregnant and lactating mares are reasonable. The new data developed since 1989 (although limited) generally agrees with the 1989 values. However, more studies need to be conducted on the effect of the calcium intake on bone integrity. The studies that were the basis for the 1989 values were primarily based on estimates of endogenous losses and balance studies.

There is, however, some concern about the calcium requirements for young horses in training. Nielsen et al. studied changes in the third metacarpal bone in Quarter Horses put into race training at 18 months of age.[67] Bone density began to decrease at the onset of training and continued to do so until day 62 of training, remaining low through day 104

when it began to increase to day 224. Horses with greater cortical mass in the lateral and medial aspects of the third metacarpal relative to the palmar aspect at the commencement of training had fewer injuries. In a subsequent study, it was demonstrated in balance studies that horses in typical race training at 24 months of age retained 4 to 6 more grams of calcium per day when fed 36 g of calcium per day, than when 30 g of calcium were fed per day. The NRC estimated requirement is 34 g of calcium per day. Coenen concluded that observations on the changes in bones in young horses during the first weeks of training have stimulated discussion of the mineral supply, particularly of calcium. But he stated, "However, at present it seems that elevated intake alone is ineffective in minimizing the risk of injury in early training."[68] Porr et al. used 12 unconditioned Arabian horses to study the effect of conditioning and intake of calcium and chloride on bone mineral content (BMC).[69] The horses were exercised on a treadmill for 12 weeks and fed one of four diets: 1 (0.7% Ca, 1.6% Cl), 2 (0.7% Ca, 0.9% Cl), 3 (0.35% Ca, 1.6% Cl), or 4 (0.35% Ca, 0.9% Cl). Bone mineral content of the left third metacarpal bone increased about 5% by 12 weeks in horses fed the high calcium diets but not in the horses fed the low calcium diet. No difference in BMC was associated with dietary cation-anion difference. It was concluded that the level of 0.35% Ca recommended by NRC (1989) for working horses did not allow adequate bone remodeling. In any case, the recommendation by Nielsen et al. that diets for young horses in training should contain 0.4% calcium does not seem to be unreasonable and further studies are warranted.[70]

Phosphorus

Phosphorus requirements were also reviewed by Ott[64] and Hintz.[66] Again both concluded that NRC requirements for horses at maintenance, growing horses, pregnant and lactating mares are reasonable. But the effect of long-term feeding of various levels of phosphorus on bone integrity needs further study.

Magnesium

The magnesium requirements of NRC (1989) were also based on estimates of endogenous or balance studies. Pagan suggested the magnesium requirements for horses at maintenance were less than suggested by NRC.[71] Grace et al. reported a requirement of magnesium for growing horses which is similar to that of NRC.[72]

Electrolytes

NRC estimated that the sodium requirement was 0.1% of the diet (dry matter basis) for maintenance, pregnant and lactating mares, and growing horses but 0.3% for working horses.[30] Thus a 500 kg horse at maintenance would require about 8 to 10 g of sodium per day. A 500 kg horse at intense work

Table 8. Estimated Sweat Fluid Losses and Sodium, Chloride, and Potassium Requirements (in grams) for a 500 kg Horse at Different Levels of Work[73]

Sweat losses (liters per day):	Non-exercised	Low workload	Moderate workload	Strenuous and/or prolonged exercise program
Sweat losses (liters per day):	0.5	5	10	20
Electrolyte requirements (grams) for a 500 kg horse				
Na^+	10–15	25	45	90
Cl	30–35	40	80	160
K^+	15–20	30	45	80

would require 40 to 45 g of sodium per day. McCutcheon recommended 10 to 15 g of sodium for a 500 kg horse at maintenance and 90 g of sodium for a horse at intense work.[73] Coenen recommended 10 g of sodium at maintenance and 57 g of sodium for a horse exercising 60 minutes at a rate of 250 meters/minute plus 15 minutes at 400 meters/minute and 5 minutes at 600 meters/minute.[68]

NRC concluded there were not sufficient data to estimate a requirement for chloride.[30] Fortunately there have been several studies conducted since 1989, particularly in Germany, so the new NRC committee should be able to estimate a requirement. McCutcheon recommended 30 to 35 g of chloride per day for a 500 kg horse at maintenance.[73] Coenen recommended 40 g of chloride per day for the 500 kg horse.[68] McCutcheon recommended 160 g of chloride per day for a horse at intense exercise and Coenen recommended 115 g per day for horses doing the exercise described in the above paragraph.

Potassium requirements were estimated by NRC to be 0.3% for maintenance and 0.35 to 0.43% for brood mares and horses performing intense work.[30] A 500 kg horse at maintenance would need 25 g of potassium and 50 g at intense work. McCutcheon recommended a 500 kg horse at maintenance would need 15 to 20 g of potassium and a horse at intense work would need 80 g of potassium.[73] Coenen rec-

ommended 25 g of potassium at rest and 52 g for horses doing the exercise described above.[68]

Of course, the requirements at work depend on how much sweat is produced. Sweat production would depend on condition of the horse and temperature. It would be useful if these conditions could be factored into the equation used by NRC to formulate estimates. In any case, the new NRC will need to closely reexamine the requirements for sodium, potassium, and chloride. McCutcheon concluded, "While access to salt should be provided at all times, dietary supplementation is a more accurate method of ensuring intake, as voluntary consumption of salt can vary significantly and loss of sodium, potassium and chloride as a result of sweating will increase in hot weather and the intensity and duration of exercise required by the horse. Therefore, supplementation of the minerals should be adjusted according to work required and ambient conditions."[73] Her recommendations of electrolytes for changes in the level of work are shown in Table 8 and electrolyte losses under different ambient conditions are shown in Table 9. The recommendations of Meyer, which are similar to those of McCutcheon, are shown in Table 10.[74]

Copper

I am certain that the new NRC committee will have long discussions concerning the requirements for copper. Copper is required for many enzymes, the synthesis and maintenance of elastic connective tissue, the mobilization of iron stores, preservation of the integrity of mitochondria, melanin synthesis, and the detoxification of superoxide.[30] Copper has

Table 9. Calculated Total Sweat Fluid and Ion Losses Under Different Ambient Conditions During Training and a Treadmill-Simulated Speed and Endurance Exercise Test[73]

	Daily Training		Speed and Endurance Test	
	Cool	Hot	Cool	Hot
Total fluid losses (liters)	4.6	11.3	11.7	19.2
Total ion (Na^+, K^+ and Cl) losses (g)	43.5	115.2	112.3	200.6

Cool = 20–22°C, 45–55% relative humidity; Hot = 33–35°C, 45–55% relative humidity; Daily training = 1 hr of submaximal exercise.

Table 10. Daily Requirements of Sodium, Potassium, and Chloride of Working Horses[a] (500 kg BW)[74]

	Work		
	Slight	Moderate	Heavy
Sodium (g)	20	50	125
Potassium (g)	30	44	75
Chloride (g)	25	70	175

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received a great amount of attention since Knight et al. reported a negative correlation between copper concentrations of weanling diets and degree of metabolic bone disease.[75] Gabel et al. recommended that the rations of weanling foals contain 25 to 30 ppm copper or a daily intake of 150 to 175 mg of copper.[76] NRC, however, reviewed other studies in which foals were fed lower levels of copper and did not develop bone abnormalities.[30] The data to increase the requirement from 10 ppm to 25 to 30 ppm were considered to be inconclusive by the NRC committee.

Are the results still inconclusive? There is no doubt that feeding diets containing low levels of copper can result in skeletal abnormalities in foals. Bridges et al. fed ultra-low copper diets (1.7 ppm) to foals and produced osteochondritis dissecans (OCD).[77] Hurtig et al. fed foals diets containing 4 to 6 ppm copper (which is less than NRC requirements) and 25 ppm which is more than NRC requirements.[78] It was suggested that there was a relationship between low copper intakes in fast-growing horses, inferior collagen quality, biomechanically weak cartilage, and OCD lesions. But of interest, although all foals fed the low copper diet were affected, only five of the nine developed clinically significant lesions. Furthermore, the authors reported that "the experiment created lesions in multiple sites throughout the body and that this is not typical of naturally occurring OCD. The lesions created in this study appear to be more similar to copper deficiency in cattle than OCD in horses."

Pearce et al. suggested that the copper nutrition of the pregnant mare was more important than the copper nutrition of the growing foal for the prevention of skeletal problems.[79] The effect of supplementation was perhaps on liver storage of the fetus because several studies have indicated that milk copper is not increased by feeding copper to the mare. They divided 24 pregnant mares into two groups, controlled or copper supplemented. Live foals within each group were then also divided into controlled or copper supplemented groups. Thus there were four groups: 1) both mares and foals supplemented with copper; 2) neither mares nor foals received copper supplements; 3) mares but not foals received copper supplement; and 4) foals but not mares received copper supplement. Foals from copper supplemented mares had a significant reduction in physitis score, regardless of copper intake by the foal. No significant decrease in physitis score was noted when the foals were supplemented with copper but the mares were not. Similar results were noted with the incidence of articular cartilage lesions.

Firth reported the effects of supplementing mares with copper during the last 13 to 25 weeks of pregnancy.[80] The mares were grazing on pasture containing 4.5 ppm copper (dry matter basis). Supplementation of mares with copper did not significantly affect liver copper of mares, plasma copper concentration or ceruloplasmin activity of foals. Liver copper of foals was significantly increased by supplementation. Bone and cartilage were monitored clinically and radiologically. Fewer cartilage lesions were found in foals from copper supplemented mares but the articular cartilage lesions were minute in all foals. There was no evidence of clinical OCD. It was concluded that the higher copper intake in pregnant mares and foals did not abolish developmental orthopedic disease and thus it was emphasized that copper may play a role, but that OCD has a multifactorial nature.

What is the copper requirement of foals? Is a level of 25 to 30 ppm of copper needed? Apparently not in all situations. But what conditions define when higher levels are needed? Feeding foals a diet containing 25 to 30 ppm copper would not be harmful to foals. It could be harmful to any sheep that happened to eat such a diet. Several cases of copper poisoning in sheep caused by consumption of horse feed have been treated at Cornell's Veterinary Clinic in recent years.

Excessive supplementation of copper would also add to environmental contamination. High intakes would also decrease efficiency of retention. Hudson et al. fed diets containing 50%, 100% and 200% of NRC copper concentrations to mature, sedentary horses.[81] Copper supplementation at 200% NRC resulted in only a small increase in copper retention compared to 100% NRC (103 vs. 90 mg of retention, respectively) because the true digestibility of copper greatly decreased at the higher level.

Balance studies with mature horses by Pagan[82] and Hudson et al.[81] resulted in estimates of values similar to the NRC value of copper for maintenance.

Selenium

Selenium nutrition of the horse has been recently reviewed by Pagan[82] and Hintz.[13]

I think selenium is the most interesting of all minerals. First known for its toxicity, it was then determined to be an essential nutrient, and now widely acclaimed to be important for the immune response and to be an anticarcinogen. Reilly reported that selenium has been claimed to have a protective role in at least 50 diseases of humans.[83] He stressed that many of the claims are not yet supported by convincing evidence. Casey concluded that many of the claims of selenophiles might be exaggerated.[84] Nevertheless, evidence for the health benefits appears to be increasing. Clark et al. reported significant decreases in diseases of lung, prostate and colorectal cancer in human subjects given 200 mg of selenium per day in a selenium enriched brewer's yeast tablet.[85]

The toxic effects of selenium were first identified in the United States in the 1930s. Chronic selenium toxicosis was reported to cause hair loss from mane and tail, sloughing of hooves, joint erosion, and lameness. The condition had previously been called alkali disease. Blind staggers characterized

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by ataxia, blindness, head pressing, and respiratory failure were also thought to be caused by selenium toxicosis.   Raisbeck et al., however, suggested blind staggers in cattle originally attributed to selenium toxicity was really polioencephalomalacia.[86]   Perhaps blind staggers in horses was not caused by selenium excess but to ingestion of plants that contained selenium and also a toxin such as swainsonine.   Swainsonine could cause the clinical signs of blind staggers.   Blind staggers has never been produced in horses by feeding high intakes of selenium.

Selenium deficiency was first reported in the 1950s by Klaus Schwarz.[87]   White muscle disease (WMD) of foals was shortly thereafter attributed to selenium deficiency.   WMD is characterized by myopathy resulting in weakness, impaired locomotion, difficulty in suckling and swallowing, respiratory distress, and impaired cardiac function.   Tying-up disease was also associated with selenium deficiency but selenium deficiency does not seem to be a major factor in cases of clinical cases today.   Selenium has also been associated with reduced reproductive efficiency.   Furthermore, Lavoie reported that oral supplementation with 1 mg of selenium increased blood selenium and decreased the number of abortions and neonatal mortalities in mares fed a diet containing a low level of selenium.[88]

Selenium deficiency has been suggested to be involved in some cases of flexural deformities because of the importance of selenium for normal muscle. It is tempting to speculate that selenium deficiency may also be involved in some cases of osteochondrosis in foals.

Kaschin-Beck disease, a form of chondrodystrophy in people, has been associated with regions in China where the foods contain low levels of selenium. However the disease is also associated with fusarium.   Selenium was able to prevent the disruption of collagen and the development of dyschondroplasia in chicks fed diets containing the mold fusarium. Could a combination of low selenium intake and ingestion of grains containing fusarium have an influence on chondroplasia in foals?   Probably not, but the USDA reported that the grain/concentrate fed to 5.3% of horse operations had fumonisin (the toxin produced by fusarium) levels above that considered safe for horses (2 ppm).[89]   According to the USDA, horses are the most sensitive of all domestic species to the effect of fumonisins.   Of the grains sampled in the central states (WI, MN, MI, IL, IN, MO, and KS) 10.5% had levels greater than 2 ppm.

Fumonisin is more likely to be associated with corn than the other grains and corn is more likely to be fed in Central regions than in the West or Northeast regions which had 0.1% and 3.7% of horse operations with grains containing more than 2 ppm fumonisin.   The grains on 6.7% of the horse operations in the Southern region had fumonisin levels higher than 2 ppm.

The NRC requirement for selenium was estimated to be 0.1 ppm.[30]   Stowe recently suggested that 0.3 ppm would be a more appropriate concentration in the total diet of horses.[90]   Pagan also suggested 0.3 ppm.[82]   Janicki et al. reported that foals from mares fed 3 mg per day had higher concentrations of IgG at 2, 4 and 8 weeks of age than foals from mares fed 1 mg of selenium per day.[91]   An intake of 3 mg per day could correspond to 0.3 ppm.   The form of selenium and interaction with other nutrients can influence the amount needed in the ration.   Sodium selenite and sodium selenate appear to have similar potency but organic forms may be more effective. Pagan et al.[62] reported the apparent digestibility of yeast selenium was greater than that for inorganic selenium when fed to exercised horses.   Perhaps organic selenium would be less negatively influenced by interactions with other nutrients than would inorganic selenium.   If so, organic selenium could be useful for those areas in which veterinarians report requirements higher than normal selenium supplementation.

Adaptation might also be a factor in determining the requirement of selenium.   Coenen suggested that Icelandic ponies could tolerate diets containing selenium levels lower than the NRC recommendations.[68]   Perhaps because Icelandic ponies have been exposed to low selenium for centuries and new genes have not been introduced, there was a natural selection for tolerance of low selenium intake.

Vervuert et al. reported that there is high variability in selenium status as measured by plasma levels among horses given the same feed.[92]   They also found that performance horses with plasma levels below the plasma level of 100 to 200 μg/l had no signs of myopathy.   They questioned the current methods of assessment of selenium status in horses.

Vitamin A

The vitamin A requirement as estimated by NRC appears to be too low.[30]   Crandell reviewed several studies and concluded that perhaps the requirement should be doubled.[93]   She suggested that horses without access to pasture would have low liver stores if fed according to the 1989 NRC standards. Griewe-Crandell et al. reported that a relative dose response method was a more sensitive measure of vitamin A status than serum vitamin A.[94]   Beta-carotene in pasture is an excellent source of vitamin A activity but several studies indicated that synthetic beta-carotene is not readily absorbed and should not be recommended as a source of vitamin A activity.[95]

Vitamin D

Crandell suggested that horses with adequate access to sunshine may always be able to meet their vitamin D requirement, but not the horses which are doing intense work or those confined indoors for the majority of the day.[93]   According to Crandell, such horses may need a supplement and at a higher intake than suggested by NRC (1989).   She explained

that young horses in training would be particularly in need of vitamin D.

Unfortunately, few studies have been conducted on vitamin D nutrition of the horse, and those that have been conducted provide contradictory results. Saastamoinen concluded that "deficiencies [of vitamin D] in practical diets are not likely."[96] Coenen concluded that it is doubtful if there is a remarkable vitamin D requirement for the horse and the 1989 NRC requirements are probably higher than necessary.[68] Because of the relationship of vitamin D, calcium, and phosphorous for bone development, studies on the requirement of vitamin D could be useful, although clinical causes of vitamin D insufficiency are very rare.

Vitamin D toxicosis is much more frequent than a deficiency. Common causes are over-supplementation and toxic plants such as cestrum diurnum, trisetum flavescens and solanum malacoxylon. Excessive vitamin D intake can cause calcification of blood vessels, heart, and other soft tissues and bone abnormalities. The maximum tolerance level was suggested by NRC to be 2,200 IU of vitamin D_3 per kg of diet or 44 IU/kg of body weight per day.[30]

Vitamin E

Vitamin E nutrition of horses and humans has received considerable attention recently. Vitamin E is the general term for several derivatives that have the biological activity of alphatocopherol. As an antioxidant, vitamin E reduces free radical activity and the deleterious reactions of these highly oxidizing compounds. Therefore vitamin E helps maintain membrane integrity in virtually all cells of the body. It also enhances immune response.

The NRC in 1989 increased their estimate of the vitamin E requirement for maintenance from 15 IU per kg of feed to 50 IU. For pregnant or lactating mares, growing horses and working horses an increase of 80 IU/kg was suggested because of reports that higher levels were needed to improve immune response and to maintain plasma vitamin E levels. Few feeds other than pasture provide 80 IU or more per kg. Therefore fortified grains or supplements are usually needed to meet the NRC requirement. It has been suggested that the requirement is even higher.[93] Hoffman et al. reported that mares fed 160 IU per kg of feed during the periparturient period had higher serum immunoglobulin G concentrations (IgG) than mares fed 80 IU.[97] Suckling foals of mares fed the higher concentration of vitamin E had higher serum concentrations of IgG than foals of mares fed 80 IU. The value of the higher level of IgG remains to be determined as foals from both groups appeared to be healthy, however, perhaps the higher IgG could have been more protective if the animals had been challenged.

Some studies with other species have indicated that vitamin E may reduce free radical formation in muscle during exercise. However, such a benefit was not detected in two other studies with horses doing light work. Siciliano and Lawrence suggested that the current NRC recommendations are probably adequate under most circumstances.[98] However, it is not unusual for racehorses to be given supplements of 1000 IU of vitamin E in addition to that in feed.[c] Saastamoninen recommended that horses in training and racing receive 3 to 5 mg/kg body weight (1500 to 2500 IU for a 500 kg horse).[96] On the other hand, field studies have indicated increased performance at vitamin E intakes of 1000 mg/day. Therefore, because of the value of vitamin E for the promotion of the immune response and because of possible benefits during exercise, it is recommended that the athlete be fed at least 1000 IU of vitamin E/day which is similar to the value suggested by NRC.

Of course the dietary intake needed depends on the efficiency of utilization. Several factors such as form of vitamin E and level of dietary unsaturated fatty acids can influence utilization. The d-forms of vitamin E are more effective than dl-forms. Natural vitamin E is more effective than synthetic forms.[93] Craig et al. reported that vitamin E mixed with grain was more effectively absorbed than when given as oral paste or by stomach tube.[99] Although vitamin E and vitamin A interactions have been reported in some species, Gück found no evidence that a 10 fold increase of dietary vitamin A influenced vitamin E status in ponies.[100]

Signs of vitamin E excess have not been reported in the horse but the NRC suggests that the maximum tolerable level is 1000 IU per kg of dry diet (12 times the requirement). Severe vitamin E deficiency in conjunction with selenium deficiency may cause myodegeneration with pale diffuse or linear areas in the skeletal and cardiac muscle (white muscle disease). Vitamin E has been implicated in equine degenerative myeloencephalopathy (EDM). It has been demonstrated that EDM is a familial disease that involves vitamin E. Supplementation with 6000 IU of vitamin E per day (about 6 times the NRC estimate of requirement for working horses) has been reported to improve the condition of some affected animals. Vitamin E may also be involved with equine motor neuron disease (EMND) a neurodegenerative condition which resembles human amyotrophic lateral sclerosis (Lou Gehrig's disease) in humans.[101] EMND in horses was first reported in 1990. The clinical signs are acute onset of trembling, excessive recumbency, almost constant shifting of the weight in the rear legs when standing, and noticeable muscle atrophy. The definitive diagnosis is histological examination of nerves and muscle. Horses with EMND almost always have very low plasma and adipose vitamin E levels. However, not all horses with low levels of vitamin E develop EMND. Further studies are needed to define possible interactions that lead to the development of EMND.

Vitamin E supplements of 2000 to 5000 IU per day are also used in the treatment of equine protozoal

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myeloencephelitis (EPM) to aid in the healing of nerve tissue.[102]

B-complex Vitamins

Thiamin, riboflavin, and biotin have probably received the most attention. The only B-complex vitamins for which NRC (1989) estimated requirements were thiamin and riboflavin. Crandell reported "little work has been done to investigate requirements of these vitamins since the last publication."[93]

Forages may contain a significant amount of thiamin and there appears to be some utilization of the thiamin produced by the microorganisms of the hind gut.   Dried brewer's yeast is a rich source of thiamin and was frequently used in the past to supply thiamin.   Working may increase the requirement of thiamin because of its role in energy metabolism.

Thiamin deficiency can be produced in horses by feeding low quality feeds for prolonged periods. Thus, intestinal synthesis alone is not adequate to meet the requirements.   Plants that contain thiaminase such as horsetail (*Equisetum arvense*) and bracken fern (*Pteridium aquilinum*) or rations with caffeic acid or the coccidiostat amprolium can induce thiamin deficiency.   Signs of deficiency include:

- Loss of body weight
- Ataxia
- Bradycardia
- Periodic hypothermia of hooves, ears and muzzle

Oral toxicity has not been reported in horses.   It has been suggested that 1000 to 2000 mg given by intramuscular or intravenous injection could have a tranquilizing effect, but recent studies could not substantiate such an effect. The NRC suggests that diets of non-working horses contain at least 3 mg of thiamin per kg of dry matter and that the diets of working horses contain 5 mg per kg.

Neither riboflavin deficiency or excess has been reported in horses.   Although riboflavin deficiency was once linked to recurrent uveitis, the linkage has not been substantiated.   NRC suggested that the requirement is probably no more than 2 mg per kg of dry matter.

Biotin is of interest because of the reports that supplementation with 15–20 mg of biotin daily can improve hoof horn quality.[103] Although supplementation may not improve hoof quality of all horses, several studies have reported that in some horses biotin can improve crumbling horn in the stratum medium of the horn wall, softness of the white lines, and decrease degradation of the cells of the sole horn.   It may take as long as six to nine months before signs of improvement are noted.   The addition of biotin (50 mg of biotin per horse per day) when horses were exercised was studied by Lindner et al.[104] Plasma biotin concentrations were increased but exercise performance was not influenced.

Other B Vitamins

NRC has not made estimates of requirements for niacin, pantothenic acid, vitamin B_6, folic acid, vitamin B_{12}, or choline.   It was concluded that signs of deficiency or excess in the horse have not been reported.   Niacin has an important role in the utilization of energy, however, Parker et al. found no benefit of supplementing exercised horses with 3 g of nicotinic acid during a trial of six weeks.[105]

References and Notes

1. USDA. Baseline reference of 1988 equine health and management. USDA:APHIS:VS, CEAH. National Animal Health Monitoring System.   Fort Collins, CO.
2. Meyer H.   Horse nutrition in central Europe: 1000 years of empirical knowledge, 100 years of research.   In: *Guardians of the horse: past, present and future*. Rossdale PD, Greet TRC, Harris PA, Hall S, ed. London and Newmarket: BEVA and Romney Publ., 1999;158–162.
3. Harris P. Changes in feeding practices. In: *Guardians of the horse: past, present and future*. Rossdale PD, Greet TRC, Harris PA, Hall S, ed. London and Newmarket: BEVA and Romney Publ., 1999;149–152.
4. Pugh.   2000 (see footnote e).
5. MacFadden BJ, Solounias N, Cerling TE. Ancient diets, ecology and extinction of 5-million-year-old horses from Florida. *Science* 1999;283:824–827.
6. USDA.   Endophytes in U.S. horse pastures—2000: USDA: APHIS:VS, CEAH. National Animal Health Monitoring System.   Fort Collins, CO.
7. Fogel R.   Pilobolus-Fungal shotgun.   1996;www.herb.lsa. umich.edu/kidpage/pilobfct.htm.
8. Buller AHR.   *Researchers on Fungi V*.   New York: Hafner Publ., 1958.
9. Gronvold J, Jdorgensen RJ. The occurrence of pilobolus fungi in Danish stables. *Prev Vet Med* 1987;5:37–42.
10. Eysker M, DeCoo FAM.   Pilobolus species and rapid translation of dictyocaulus-viviparus from cattle feces. *Res Vet Sci* 1988;44:178–182.
11. Foos KM.   Pilobolus and lungworm disease affecting elk in Yellowstone National Park. *Mycological Res* 1997;101: 1535–1536.
12. Jorgensen RJ, Andersen S. Spread of equine lungworm dictyocaulus-arnfieldi larvae from feces by pilobolus fungi. *Nord Veterinaermed* 1984;36:162–169.
13. Hintz HF. Developments in equine nutrition in the past century. In: *Guardians of the horse: past, present and future*. Rossdale PD, Greet TRC, Harris PA, Hall S, eds. London and Newmarket: BEVA and Romney Publ., 1999; 153–157.
14. Hoffman RM, Kronfeld DS. Nutrient requirements for grazing horses: Development of an optimal pasture supplement. In: *Recent advances in animal nutrition*. Garnsworthy PC, Wiseman J, eds.   Nottingham, UK: Nottingham University Press, 1999;19–37.
15. Huntington P.   Feeding and management practices in Australia in Proceedings.   Equine Nutrition Conference for Feed Manufacturers.   2000;277–288.
16. Harris P.   Feeding and management practices in the UK and Germany, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers, 2000;241–266.
17. Zogby America.   on the internet.
18. Ainsworth GC, Sussman AS.   *The fungi: an advanced treatise*, vol. 1.   New York: Academic Press, 1965;18.
19. AAEP.   Colic—understanding the digestive tract and its function.   *AAEP Guide for Horse Owners* 1996.   AAEP & Bayer; 1–7.
20. Reeves MJ, Salman MD, Smith G.   Risk factors for equine acute abdominal disease (colic): results from a multi-center-case-control study.   *Prev Vet Med* 1996;26:285–301.

MILNE LECTURE: EQUINE NUTRITION UPDATE

21. Cohen ND, Peloso JG. Risk factors for history of previous colic and for chronic intermittent colic in a population of horses. *J Amer Vet Med Assoc* 1996;208:697–703.

22. Dart AJ, Hodgson DR, Snyder JR. Caecal disease in equids. *Aust Vet J* 1997;75:552–557.

23. White NA, Edwards GB. *Handbook of equine colic.* Oxford: Butterworth and Heineman Publ., 1999.

24. Tinker MK, White NA, Lessard P, et al. Prospective study of equine colic incidence and mortality. *Equine Vet J* 1997;29:448–453.

25. Hintz HF, Lowe JE, Livesay-Wilkens P. Studies on equine enterolithiasis, in *Proceedings.* Annu Conv Amer Assoc Equine Practnr 1988;53–59.

26. Hintz HF, Hernandez TM, Soderholm V, et al. Effect of vinegar supplementation on pH of colonic fluid, in *Proceedings.* 11th Equine Nutr Physiol Symp 1989;116–118.

27. Hassel DM, Langer DL, Snyder JR, et al. Evaluation of enterolithiasis in equids: 900 cases. *J Amer Vet Med Assoc* 1999;214:233–237.

28. Radicke S, Kiengle E, Meyer H. Preileal apparent digestibility of oats and corn starch and consequences for cecal metabolism, in *Proceedings.* Equine Nutr Physiol Soc 1991;43–48.

29. National Research Council. *Nutrient requirements of horses.* Washington DC: National Academy Press. 1989.

30. Wolter R. Fibre in the feeding of horses. *Practique Veterinaire Equine* 1993;25:45–59, as abstracted in *Nutrition Abstract Reviews* 1993;63:605.

31. Ferrante PL, Menniger JH, Spencer PA, et al. Metabolic response of horses to a high soluble carbohydrate diet: effects of low-intensity submaximal exercise and sodium bicarbonate supplementation. *Am J Vet Res* 1992;53:321–325.

32. Cohen ND, Gibbs PG, Woods AM. Dietary and other management factors associated with colic in horses. *J Amer Vet Med Assoc* 1999;215:53–60.

33. Sanders ME. Considerations for use of probiotic bacteria to modulate human health. *J Nutr* 2000;130:384S–390S.

34. Rolfe RD. The role of probiotic cultures in the control of gastrointestinal health. *J Nutr* 2000;130:396S–402S.

35. DiRienzo DB. Symposium: Introduction. *Amer Soc Nutr Sci* 2000;130:382S–383S.

36. Moore BE, Newman KE, Spring P, et al. Effect of yeast culture (Yea-Sacc 1026) on microbial populations and digestion in the cecum and colon of the equine. *J Dairy Sci* 1994;77:252.

37. Glade M. Dietary yeast culture supplementation enhances urea recycling in the equine large intestine. *Nutr Rpt Int* 1988;37:11–18.

38. Kim SM, Kim CM, Lee HK, et al. Evaluation of nutrient values of some feedstuffs and the effect of yeast culture supplementation on digestibilities of nutrients and blood parameters in horses. *J Anim Nutr Feedstuffs (Korea)* 1991;15:272–280.

39. Pagan JD. Effect of yeast culture supplementation on nutrient digestibility in mature horses. *J Anim Sci* 1991;68(Suppl 1):371.

40. Webb SP, Potter GD, Massey KJ. Digestion of energy and protein by mature horses fed yeast culture, in *Proceedings.* Equine Nutr Physiol Soc 1985;64–67.

41. Hall RR, Jackson SG, Baker JP, et al. Influence of yeast culture supplementation on ration digestion by horses. *Equine Vet Sci* 1990;10:130–34.

42. Rowe JB, Lees MJ, Pethick DW. Prevention of acidosis and laminitis associated with grain feeding in horses. *J Nutr* 1994;124:2742S–2744S.

43. Johnson KG, Tyrrell J, Rowe JB, et al. Behavioral changes in stable horses given nontherapeutic levels of virginiamycin. *Equine Vet J* 1998;30:139–143.

44. Willard JG, Willard JC, Wolfram SA, et al. Effect of diet on cecal pH and feeding behaviors of horses. *J Am Sci* 1977;45:87–93.

45. Blomme EAG, La Perle KMD, Wilkind PA, et al. Ionophore toxicity in horses. *Equine Vet Educ* 1999;11(3):153–158.

46. Roder JD, Stair EL. Ionophore toxicosis. *Vet Human Toxicol* 1999;41:178–181.

47. Bezerra PS, Driemeier D, Loretti AP, et al. Monensin poisoning in Brazilian horses. *Vet Human Toxicol* 1999;41:383–385.

48. Niness KR. Inulin and oligofructose: What are they? *J Nutr* 1999;129:1402S–1406S.

49. LeBlay G, Michel C, Blottière, et al. Prolonged intake of fructo-oligosaccharides induces a short-term elevation of lactic acid-producing bacteria and a persistent increase in cecal butyrate in rats. *J Nutr* 1999;129:2231–2235.

50. Moshfegh AJ, Friday JE, Goldman JP, et al. Presence of inulin and oligofructose in the diets of Americans. *J Nutr* 1999;129:1407S–1411S.

51. USDA. Lameness and laminitis in US horses—2000. USDA:APHIS:VS, CEAH Animal Health Monitoring System. Fort Collins, CO.

52. Longland AC, Cairns AJ. Fructans and their implications in the aetiology of laminitis, in *Proceedings.* Internat Conf Feeding Horses 2000;53–56.

53. Cuddeford D. Partitioning digestion in horses and ponies, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers. 1999;9–20.

54. Martin-Rosset W. Feeding standards for energy and protein for horses in France, in *Proceedings.* Equine Nutrition Conference for Feed Manufacturers 2000;31–94.

55. Cuddeford D. Starch digestion in the horse, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers. 1999;129–139.

56. Potter GD, Arnold FF, Householder DD, et al. Digestion of starch in the small or large intestine of the equine. *Pferdeheilkunde:* Sonderheft, 1992;107–111.

57. Meyer H, Radicke S, Kienzle, E, et al. Investigations on pre-ileal digestion of starch from grain, potato and manioc in horses. *J Vet Med Series A* 1995;42:371–381.

58. Meyer H, Radicke S, Kienzle E, et al. Investigations on pre-ileal digestion of oats, corn and barley starch in relation to grain processing, in *Proceedings.* Equine Nutr Phys Symp 1993;92–97.

59. Pagan JD, Harris PA, Kennedy MAP, et al. Feedtype and intake affect glycemic response in Thoroughbred horses, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 1999;147–149.

60. Hoekstra KE, Newman K, Kennedy MAP, et al. Effect of corn processing on glycemic response in horses, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 2000;141–146.

61. Pagan JD, Karnezos P, Kennedy MAP, et al. Effect of selenium source on selenium digestibility and retention in exercised Thoroughbreds, in *Proceedings.* Equine Nutr Physiol Soc Symp 1999;135–140.

62. Lawrence LM. Advances and gaps in energy nutrition, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 2000;11–22.

63. Ott EA. Protein and amino acids, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 2000;23–30.

64. Graham-Thiers PM, Kronfeld DS, Kline KA. Dietary protein influences acid-base responses to repeated sprintz. *Equine Vet J* 1999(Supp);30:463–467.

65. Hintz HF. Macrominerals—calcium, phosphorus and mangesium, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 2000;121–132.

66. Nielsen BD, Potter GD, Morris EL, et al. Changes in the third metacarpal bone and frequency of bone injuries in young Quarter Horses during race training: observations and theoretical considerations. *J Equine Vet Sci* 1997;17:541–549.

67. Coenen M. German feeding standards, in *Proceedings.* KER Equine Nutrition Conference for Feed Manufacturers 2000;159–174.

68. Porr CAS, Kronfeld DS, Lawrence LA, et al. Diet and conditioning influence bone development. *Equine Pract* 2000;22(5):18–21.

69. Nielsen BD, Potter GD, Greene LW.   An increased need for calcium in young racehorses beginning training, in *Proceedings*.   Equine Nutr Phys Symp 1997;153–158.
70. Pagan JD.   Nutrient digestibility in horses, in *Proceedings*. KER Equine Nutrition Conference for Feed Manufacturers 1994;127–136.
71. Grace ND, Pearce SG, Firth EC, et al.   Content and distribution of macro- and micro-elements in the body of pasture-fed young horses.   *Australian Vet J* 1999;77:172–176.
72. McCutcheon J.   Sodium, potassium and chloride: in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000;133–146.
73. Meyer H.   Nutrition of the equine athlete, in *Proceedings*. Intl Conf On Equine Exercise Physiology 1987;644–673.
74. Knight DA, Gabel AA, Reed SM, et al.   Correlation of dietary mineral to incidence and severity of metabolic bone disease in Ohio and Kentucky, in *Proceedings*.   *Am Assoc Equine Practnr* 1985;31:445–449.
75. Gabel AA, Knight DA, Reed SM, et al.   Comparison of incidence and severity of developmental orthopedic disease of 17 farms before and after adjustment of ration, in *Proceedings*.   Annu Conv Amer Assoc Equine Practnr 1987; 163–170.
76. Bridges CH, Harris ED.   Cartilaginous fractures ("osteochondritis disseccans") induced experimentally in foals with low copper diets.   *J Am Med Assoc* 1988;193:215–221.
77. Hurtig MB, Green SL, Dobson H, et al.   Correlative study of defective cartilage and bone growth in foals fed a low-copper diet.   *Equine Vet J* 1993;16:66–73.
78. Pearce SG, Firth EC, Grace ND, et al.   Effect of copper supplementation on the evidence of developmental orthopaedic disease in pasture-fed New Zealand Thoroughbreds. *Equine Vet J* 1998;30:211–218.
79. Firth EC, Pearce SG, Gee EK, et al.   Copper status, copper supplementation and the incidence of developmental orthpaedic disease, in *Proceedings*.   Internat Sem Osteochondrosis.   1999;45–46.
80. Hudson CA, Pagan JD, Hoekstra KE, et al.   The effects of intake level on the digestibility and retention of copper, zinc and manganese in sedentary horses, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000; 191–192.
81. Pagan JD.   Micromineral requirements in horses, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000;107–120.
82. Reilly C.   Selenium in Food and Health.   London: Black Academic, 1996.
83. Casey CE.   Selenophilia.   *Proc Nutr Soc* 1988;47:55–62.
84. Clark LC, Combs GF, Turnbull BW, et al.   The nutritional prevention of cancer with selenium: a randomized clinical trial.   *J Amer Med Assoc* 1996;276:1957.
85. Raisbeck MF, Dahl ER, Sanchez A, et al.   Naturally occurring selenosis in Wyoming.   *J Vet Diagn Invest* 1993; 5:84–87.
86. Schwarz, Foltz.   Selenium is an integral part of factor 3 against dietary necrosis degeneration. *J Amer Chem Soc* 1957;79:3292–3295.
87. Lavoie J.   Abortion and neonatal mortality associated with selenium deficiency, in *Proceedings*.   N Amer Vet Conf 2000;14:147–148.
88. USDA.   Fumonisin B$_1$ mycotoxin in horse grain/concentrate on U.S. horse operations—2000.   USDA:APHIS:VS, CEAH Animal Health Monitoring System.   Fort Collins, CO.
89. Stowe HD.   Selenium supplementation for horse feed. In: *Advances in equine nutrition*.   Pagan JD, ed.   UK: Nottingham University Press, 1998;97–103.
90. Janicki KM, Lawrence LM, Barnes T, et al.   The effect of dietary selenium source and level on broodmares and their foals, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000;207–208.
91. Vervuert I, Coenen M, Holtershinken et al.   Assessment of selenium status in horses, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000; 197–198.
92. Crandell K.   Vitamin requirements in horses, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000;96–106.
93. Griewe-Crandell KM, Kronfeld DS, Gay LA, et al.   Seasonal vitamin A depletion in grazing horses is assessed better by the relative dose response test than by serum retinol concentration.   *J Nutr* 1995;125:2711.
94. Griewe-Crandell KM, Kronfeld DS, Gay LA, et al.   Vitamin A repletion in Thoroughbred mares with retinyl palmitate or beta carotene.   *J Animal Science* 1997;75:2684–2698.
95. Saastamoinen MT.   Vitamin requirements and supplementation in athletic horses, in *Proceedings*.   KER Equine Nutrition Conference for Feed Manufacturers 2000;201.
96. Hoffman RM, Morgan KL, Lynch MP, et al.   Dietary vitamin E supplemented in the periparturient period influences immunoglobulins in equine colostrum and passive transfer in foals, in *Proceedings*.   Proc. Equine Nutr Physiol Symp 1999;96–97.
97. Siciliano PD, Lawrence LM.   The role of vitamin E in the exercised horse.   Vitamin E in Animal Nutrition and Management.   Mount Olive, NJ: BASF, 1996.
98. Craig AM, Blythe LL, Rowe KE, et al.   Evaluation of the oral vitamin E absorption test in horses.   *Am J Vet Res* 1991;42:912.
99. Gück T.   Influence of increased vitamin A supplements on the vitamin A and E status in the Shetland pony.   Thesis, Tierärztliche Hochschule Hannover, Germany: 1998;165.
100. Divers TJ, Mohammed HO, Cummings JF.   Equine motor neuron disease.   *Vet Clin North Am: [Equine Practice]* 1997;13:97.
101. AAEP.   Equine protozoal mycloencephalitis (EPM) 1998: *AAEP Guide for Horse Owners*. AAEP & Bayer; 1-7.
102. Josseck H, Zenker W, Geyer H.   Hoof horn abnormalities in Lippizaner horses and the effect of dietary biotin on macroscopic aspects of hoof horn quality.   *Equine Vet J* 1995; 27:175.
103. Lindner A, Von Wittle P, Frigg M.   Effect of biotin supplementation on the V$_{la4}$ of Thoroughbred horses.   *J Equine Vet Sci* 1992;12:149.
104. Parker AL, Lawrence LM, Rokuroda S, et al.   The effects of niacin supplementation on niacin status and exercise metabolism in horses, in *Proceedings*.   Equine Nutr Physiol Symp 1997;19–24.
105. Martin-Rosset W.   L'alimentation des chevaux.   INRA, Paris.   1990

[a]Scoggins D.   Personal communication.   1999.
[b]Duren S.   Personal communication.   1999.
[c]Breuer L.   Personal communication.   2000.
[d]Dawson.   Personal notes.   1999.
[e]Pugh. Personal notes.   2000.

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