NUTRITION OF THE HORSE

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CONTENTS

INTRODUCTION				 	 		 ٠.	٠.			٠.				٠.	 							
DIGESTIVE TRACT Anatomy	·			 • •	 	•	 	٠.	•		٠.		٠.				٠.			•	•	•	•
Microbial Activity	,			 	 		٠.			٠.	٠.	٠			٠.	 	٠.						
Site of Absorption																							
ENERGY		 	• • •	 • •	 		 									 		 					
Function of the H Environmental Te	orse mperatur	 !		 • •	 • • •		 		•					:		 			• •				
PROTEINS				 	 		 											 					
MINERALS				 	 		 ٠.											 					
VITAMINS				 	 		 						٠.					 					
CONCLUSIONS				 	 		 											 					

INTRODUCTION

The horse was domesticated around 2500 BC and has been used for work, pleasure, and companionship since that time. However, the percentage of time devoted to these endeavors has changed greatly. The number of horses in the US peaked at more than 26 million in 1918. Thereafter, the increasing use of the internal combustion engine caused a steady decline in the number of horses. By 1960, the US Department of Agriculture (USDA) estimated that there were only about 3 million horses in the US. In the 1960s, however, the number of

horses dramatically increased because of a renewed interest in the horse for recreational activities. The USDA no longer conducts a census of horses, but the horse population was estimated at 10.5 million in the mid-1980s. Although the number of horses has decreased recently owing to a general economic decline, the horse industry still remains a vital part of US culture.

The decreasing importance of the horse in agriculture starting in 1918 led to a decline in equine research at universities and agricultural experiment stations. By the late 1950s, almost no equine nutritional research was conducted, and few land grant colleges owned horses or taught horse production courses. The resurgence of the horse in the 1960s led to increased equine research, teaching, and extension activities. Today, many universities and colleges, both public and private, have equine programs. Of course, the amount of money and scientists devoted to equine nutrition is still much less than that devoted to other farm animals.

Much remains to be learned about the horse. In fact, the most common phrase in equine research is "further research is needed," a phrase that appears frequently in this review as well. Nevertheless, much progress has been made in the discipline of equine nutrition. Many studies have been conducted, and a comprehensive review would exceed the allotted length. Thus this review focuses on several areas of progress and identifies areas where research is needed.

DIGESTIVE TRACT

Anatomy

The horse is a nonruminant herbivore with significant fermentation in the hind gut (cecum and colon). According to a study conducted by Colin in 1871, the stomach accounts for only 8% of the capacity of the total digestive tract compared with 30% for the small intestine, 16% for the cecum, and 46% for the colon (144). More recent data should be collected, but the horse obviously has a relatively small stomach for such a large animal. The stomach capacity of the mature horse (average wt. 500 kg) is only about twice that of the mature pig (average wt. 165 kg) (144). The horse's small stomach and its inability to vomit explain why colic, gastric distension, and/or gastric rupture occur in horses that overeat.

A horse's small intestine is ~ 20 m long and accounts for $\sim 75\%$ of the total length of the gut. The remaining gut length is comprised of the cecum (4%) and the colon (21%) (144). These values were also adapted from Colin's studies in 1871. Measurement of intestinal tracts in 103 fetal and adult horses revealed that the absolute length of the gut of a mature horse was slightly shorter than Colin's estimates, but the proportions were similar, i.e. the small intestine accounted for 73.6%, the cecum for 3.5%, and the colon for 22.9% of total gut length. The gut proportions varied with age, but not as much as might be expected. The small intestine comprised 76, 91, and 80% of total gut length in horses aged 150–329, 330–346, and 347–505 days, respectively, compared with 73.6% for horses older than 505 days of age (138).

The small intestine of the horse is not unusual compared with other domestic animals. However, its hindgut has several distinguishing characteristics. It is much larger than that of cattle, sheep, or swine and contributes more to the relative capacity of the equine tract. The colon has sacculations with narrow and broad compartments demarcated by discrete bands of smooth muscle and connective tissue called teniae (19). The teniae are formed by longitudinal muscle and provide intestinal support but yield to intestinal distension caused by fermentation. The sacculations probably reduce the rate of digesta passage and thus enhance microbial fermentation and digestion because the digesta is exposed to the bacteria for a longer period of time. This arrangement likely evolved during the years when horses spent most of their time grazing. Therefore, the microbial population of the hindgut received a slow and steady supply of fermentable material. Today's horse is often confined to a stable for most of the day and fed large meals low in fiber. Clarke et al recently reviewed the physiologic responses by horses fed a concentrated meal (20). They concluded that twice daily feeding of a high-energy diet promotes unnatural digestive functions in horses that predispose them to spontaneous gastrointestinal disturbances. Gastric fill and pH may be influenced by feed type. Ponies fed mixed feed had a 37% incidence of gastric ulcers compared with zero occurrence in ponies fed hay (21). Any gut dysfunction in horses is alarming because it can precede colic. Colic, a general term for abdominal pain, is the main cause of equine mortality.

Significant advances have been made in the prevention and treatment of colic. Approximately 30 years ago, intestinal surgery of the horse had a low rate of success, and often even simple colics were fatal. Currently, surgery of the equine intestinal tract, although difficult, is less risky and has a much higher success rate (5, 55). Improved surgical techniques have also helped clarify nutritional function of segments of the intestinal tract. Cannulas can now be implanted in many parts of the intestine (41, 136) and have thus enabled studies on many sites of absorption and fermentation of gut microflora. The cecum has been removed without great nutritional consequence (126). In horses in which 95% of the large colon was removed surgically, plant cell wall digestibility decreased relative to presurgical values and values for sham-operated horses, but body weight in surgically altered horses was maintained because organic matter digestibility remained effective (8, 10). One year after large-

colon resection, all horses exhibited elevated alkaline phosphatase activity in the cecum and remaining colon, which implied a compensatory response for the reduction in phosphorus utilization by horses with colon resections (9).

Microbial Activity

The principal microbial inhabitants of the equine hindgut are bacteria, protozoa, and fungi (71). The majority of bacteria are gram-negative rods (51-64%) or gram-negative cocci (8-33%). Approximately half of this population is anaerobic. Studies on how diet affects microbial activity in the equine hindgut are limited. Some inferences can be made from studies with ruminants because the types and responses by microflora in the equine hindgut are similar to those in the rumen (2). However, extrapolations about microbial activity must be drawn cautiously because of the dissimilarity in the relative location of the rumen and the hindgut in the respective intestinal tracts. The rumen is located anterior to the small intestine, which is the primary site of absorption of many nutrients. Thus, bacterial end products can be digested in the small intestine of the ruminant. The hindgut of horses is posterior to the small intestine, and many nutrients produced by the equine microflora are not effectively absorbed.

As in the rumen, fermentation in the hindgut produces carbon dioxide, methane, and most importantly, the volatile fatty acids (VFA) acetate, propionate, and butyrate. Methane production in the rumen has recently attracted interest because (a) \sim 8% and as much as 10% of the animal's total energy intake can be lost as methane (62, 74); and (b) methane from ruminants is wrongly perceived by some to be a major contributor to global warming (24). Thus considerable effort has been made to measure methane production by ruminants and to develop methods to lower methane production. Recently, it was calculated that methane loss by ruminants accounts for only a very low percentage of total global methane, even less than that contributed by termites (62), and the total amount of methane from horses is only a small fraction of that produced by ruminants (24). Methane production by horses has been shown to peak 6-8 h after feeding and is greater for horses fed concentrates or with a high feed intake, but methane loss from horses is still less than 3% of the total energy intake (111, 149, 153). Methane from horses has little impact on global warming.

VFA are readily absorbed from the hindgut and can contribute up to 30% of the energy utilized by the horse (7). Acetate:propionate:butyrate ratios normally range from 70:20:10 to 75:15:10 (7), but diet manipulation can alter these ratios. Higher acetate and lower propionate values were obtained for horses fed grass (85). Increasing starch or grain intake reduces the relative amount of acetate and increases the amount of propionate (58), presumably by changing the bacterial population (100). The ratio of acetate to propionate is important because although propionate can be a significant source of glucose,

acetate is not. Approximately 7% of total glucose production in the horse arises from propionate produced in the cecum (41). Simmons & Ford (136) concluded that the propionate produced in the large colon accounts for 57–68% of the glucose from propionate by ponies fed roughage.

Would manipulation of the gut microflora to produce more propionate (and therefore more glucose) benefit working horses that may be limited by glucose availability, or is it simpler to supply glucose directly for absorption from the small intestine? Are such manipulations likely to lead to digestive disturbances? Rapid changes in diet can be harmful when the amount of soluble carbohydrate reaching the hindgut is abruptly increased, resulting in a decrease in gut pH and a shift in bacterial population, which in turns causes diarrhea, colic, and/or laminitis. Feeding a diet containing 90% concentrate resulted in a marked elevation in lipopolysaccharides (a component of gram-negative bacteria), which points to endotoxin production (72). Hence the age-old axiom that dietary changes for horses must be made gradually is valid. However, few studies have characterized the longitudinal changes in microbial population in response to diet manipulation. The thumb rule that changes should be made over a four or five day period is based only on empirical evidence.

Microbial activity of the hindgut has been reported to be enhanced by probiotics. Yeast culture in the diet increased digestibility of dry matter, neutral detergent fiber, hemicellulose, acid detergent fiber, cellulose, and nitrogen; enhanced urea recycling (48, 73); and increased the efficiency of phosphorus utilization (109). Nevertheless, yeast addition did not aid in the digestion of diets containing 50% grass and 50% grains or rice hulls (53, 150).

Site of Absorption

Nutritional knowledge of the horse would progress even further if the sites of absorption and the factors that modify absorption kinetics at these sites were elucidated. Soluble carbohydrates are primarily digested and absorbed in the small intestine (59). However, high intakes of starch allow some starch to bypass the small intestine and accumulate in the hindgut. High concentrations of soluble carbohydrates acutely deposited in the hindgut cause alteration of the microbial population, resulting in colic and laminitis (143). Smaller amounts of soluble carbohydrate reaching the hindgut may not cause the above-mentioned clinical disorders but can nonetheless result in reduced efficiency of energy utilization because glucose is utilized more efficiently than are VFA.

Fiber is digested in the hindgut by the gut microflora. One of the major factors that influences fiber digestion is the rate of passage. Longer retention of ingesta by large-bodied ruminants and hindgut fermenters increases digestive efficiency compared with small animals and permits them to survive on lower-quality feeds (66). The horse digests fiber less efficiently than the cow, presumably because it has a faster rate of passage of digesta (78). However, this faster rate of passage in

the horse may be an advantage that outweighs the poorer digestive efficiency when feed resources are not limited (32, 66). That is, total energy intake by the horse is greater because the horse can eat more than the ruminant owing to the faster rate of passage. Conversely, if feed is limited, the ruminant has the advantage. However, this theory may only hold when grasses and not hays are ingested. Comparative studies with cattle and horses fed various hays revealed that despite similar voluntary dry matter intakes, horses consumed 17–37% less digestible energy than did cattle because of a lower digestive efficiency (27). Donkeys, on the other hand, digested poor quality forages almost as effectively as ruminants. This finding appears to refute the generalization that all equids utilize poor-quality forages less well than ruminants (68).

The site of protein digestion and absorption should be considered when evaluating amino acid nutrition of the horse. Dietary protein digested and absorbed from the small intestine contributes to the amino acid pool. Dietary protein that escapes digestion in the small intestine to reach the hindgut is degraded to ammonia. Ammonia can be absorbed and excreted as urea, a process that adds nothing to the nutritional usefulness of the nitrogen, or it can be incorporated into bacterial protein. The value of hindgut-produced microbial protein for the horse has been debated for years. Recently, support for the position that microbial protein contributes little to the amino acid supply of horses increased (93). Thus scientists in France have proposed that the protein values assigned to feedstuffs account for the amount of absorbable amino acids. The value is called Matières Azotées Digestible Cheval (MADC) and considers site of digestion, i.e. small vs large intestine. The theory is sound, but reported values for precedul protein digestion vary from as low as 2% to as high as 55% of the total amount digested (46, 59). Why such a wide range of values was obtained despite the variety of methods and feedstuffs used is difficult to understand. More information is needed about the factors influencing the sites of protein digestion before MADC can be universally applied.

If the horse practiced coprophagy as do rodents and lagomorphs (140), the bacterial protein would be utilized. The foal eats some feces, particularly from its dam, but coprophagy does not appear to be extensive in the horse (23). Adult horses eat more feces when they are fed protein-deficient or high-concentrate diets (131, 151). Horses fed whole corn plant pellets containing only 6.2% protein practiced coprophagy, which nonetheless ceased after soybean meal was added to the diet (131).

ENERGY

Energy requirements are primarily influenced by body size, the function of the animal, and environmental temperature. The digestible energy (DE) concen-

tration of the diet is usually regulated by adjusting the forage:concentrate ratio. Forage may contain 1.8-2.3 Mcal of DE/kg of dry matter (104). Energy concentrations in grains vary from 3.2 Mcal of DE/kg in oats to 3.8 in corn (104). Fats and oils may contain 8-9 Mcal of DE/kg. The function of the horse determines the energy density that is needed. Mature horses at maintenance do not require a high energy density and therefore usually do not need grain. Mares in late gestation may be expected to consume 1-1.5% of their body weight in forage and 0.5-1% in concentrate. Mares in early lactation may consume 1-2% of body weight in forage and 1-2% in concentrate. Horses at intense work may consume 0.75-1.5% body weight in forage and 1-2% in concentrate. Weanlings may consume 0.5-1% of body weight in forage and 2-3.5% in concentrate (104).

Body Size

The National Research Council (NRC) defined maintenance as the energy required for zero body weight change plus normal activity (104). In past editions, the NRC subcommittee on horse nutrition used metabolic body size to estimate maintenance energy requirements. For the 1989 edition, the 0.75 exponent was not used because the results obtained with horses and ponies (111) correlated with studies in other species (146), which demonstrated no benefit in using an exponent when comparing animals from the same species unless large differences (severalfold) in body size were present. The equation for calculating the DE needed for maintenance energy was (Mcal/day) = 1.4+ 0.03W, where W = weight of the horse in kg. Reasonable agreement was obtained between calculated estimates for maintenance energy and actual data obtained with ponies and light horses weighing up to 600 kg. However, the equation seemed to overestimate the energy requirements for horses heavier than 600 kg. In the above study (111), all horses were confined to metabolism stalls during the measurement of oxygen consumption. The activity factor was estimated for horses weighing less than 600 kg. The discrepancy between the calculation for the larger horses and the observed requirements presumably arose because the large horses had a much lower voluntary activity than the light horses. To accommodate the lower activity of heavy horses, the equation $DE \times (Mcal/dav) = 1.82 + 0.0383W - 0.000015W^2$ was developed, where W = weight in kg. Unfortunately, few data are available on the energy needs of horses heavier than 600 kg, and the hypothesis that heavier horses are less active has never been adequately tested. Moreover, a breed-size interaction may occur in voluntary activity. Horses of some of the large breeds appear to be more animated than others. Therefore, the energy requirements for horses weighing more than 600 kg need further study.

Function of the Horse

The functions of horses are classified as maintenance, growth, pregnancy, lactation, breeding (stallion), and work. Energy requirements for maintenance are discussed above. Energy requirements for growth are important because the rate of growth (and hence of energy intake) may influence the incidence of skeletal problems involved in developmental orthopedic disease (DOD). DOD is a name given to a complex of disorders, including osteochondrosis, flexural deformities, and epiphysitis. Many other factors, such as genetics and mineral nutrition, may also be involved in the development of DOD. Genetic manipulation may ultimately be needed to decrease the incidence of DOD. Although the mode of inheritance has not been determined, heritability for osteochondrosis is moderately high (up to 35%) (52, 63, 115, 129). It has been suggested that DOD is related to a genetic capacity for rapid growth. Certainly the incidence of DOD appears to be greater in foals growing rapidly (30, 125). However, foals fed a diet with a high energy density, achieved by adding vegetable oil, had a higher incidence of dyschondroplasia unrelated to a more rapid growth rate than did those fed a normal energy intake (127). Conversely, a diet of 10% fat was shown to be a suitable substitute for grain without causing adverse effects on the skeleton in growing horses (31). Moreover, high energy intakes do not consistently produce leg abnormalities or dyschondroplasia in foals. One might expect a diet for a weanling horse to be approximately one third forage and two thirds concentrates and to contain 3 Mcal of DE/kg, 31% neutral detergent fiber, 16% protein, and 3.5% ether extract (dry matter basis) (104). Adding 8% vegetable oil to the concentrate could increase the DE concentration of the total diet to 3.3 Mcal/kg and the ether extract to 9%. Thus a "high-fat" diet for horses would still be much lower in DE and in fat content than a typical diet for humans. Yearlings consuming 60% more energy than recommended for normal growth had no abnormalities in bone development (108). In fact, Ott & Asquith observed a reduction in bone mineral deposition in foals fed at normal 1983 NRC energy levels. No simple answers explain the energetics of growth in horses or the energy values that represent normal or excessive intakes.

NRC (104) energy requirements for gestation are primarily based on studies of fetal growth rate and fetal body composition (95, 116). DE requirements were increased by 11, 13, and 20% above maintenance during the ninth, tenth, and eleventh month of gestation, respectively, in accordance with the pattern of energy deposition in the fetus. Such calculations are valid, assuming that the mare does not have any body fat reserves. Thus most textbooks state that energy does not need to be increased until the last third of gestation. Mares kept on pasture gained weight during the second third of gestation and plateaued during the last third (82). Condition score and estimated body fat also

increased during the second trimester, but body fat percentage decreased during the last trimester, which suggests that body reserves were mobilized to support fetal foal growth during the last third of gestation. Over three years, mares in good body condition kept in a university herd had healthy foals despite minimal (2%) increases in body weight during the last two months of gestation (79, 84). Accumulation of body fat reserves during early gestation of the undomesticated horse ensures adequate energy for rapid fetal growth and presents advantages for survival in free-roaming horses. The studies listed above suggest that domestic mares may still respond in this manner. During early and midgestation, mares should not be restricted to maintenance energy intakes. Rather, they should be given the opportunity to accumulate body reserves as early as the sixth month of gestation.

The 1989 NRC (104) energy requirements for the lactating mare are based on studies of milk yield, milk composition, and the assumption that 792 kcal of DE are needed to produce a kilogram of milk. Subsequent studies, particularly those conducted in France, have clarified some of the requirements needed for pregnant and lactating mares (39). These studies used the French energy system (Unité Fourragère Cheval, UFC) (89, 90, 148).

Direct comparisons of the NRC and the Institut National de la Recherche Agronomique (INRA) systems cannot be made easily or without several assumptions. Ironically, however, calculations based on either system when expressed in terms of feed rather than DE or UFC units yield similar values.

For example, using the INRA system, a pregnant, 500 kg mare should be fed 12-15, 10-15, and 8-12.5 kg of feed daily during the first, second, and third months of lactation, respectively (88). NRC classifies the lactation period as early (foaling to three months) and late (three months to weaning). The NRC estimates that a 500 kg mare in early lactation requires 28 Mcal of DE day⁻¹ or ~ 10-15 kg feed day⁻¹. Although different energy units are used, the final calculations in amount of feed are similar. Other papers from France have reviewed milk composition and yield by mares (35). The effect of stage of lactation on milk yield indicates that energy requirements are more accurately defined on a monthly basis (36) than as early or late lactation as suggested by NRC (104). Voluntary feed intake and milk yield and composition were measured in mares fed diets containing 95% hay and 5% concentrates or 50% hay and 50% concentrates during the first two months of lactation (37). The mares consumed more of the high-forage diet (-7%) than the high-concentrate diet, but voluntary feed intake for both diets plateaued after reaching a peak intake at two weeks postlactation at intakes representing ~ 2.5-3.2% of body weight. Similarly, milk yield increased from one to eight weeks postpartum, regardless of dietary manipulation. Although fat, crude protein, lactose, and mineral concentrations differed between treatment groups, only the absolute output of fat, lactose, and magnesium secretion were modified by dietary treatment. In a previous study, feed intakes of 3.4 and 3.3% live body weight were obtained in primiparous and multiparous mares, respectively, fed a mixed diet of grain and hay (38). Foals born to primiparous mares grew more slowly in the first month of life, a result that was correlated to milk energy output.

Establishing energy requirements for stallions was a new venture for the NRC in 1989 since previous editions did not address energy needs for breeding males. The recommendation for stallions was increased ~ 25% above maintenance needs for nonbreeding males. The French recommendation was to increase energy above maintenance by 12, 25, and 57% for light, medium, and intense service, respectively (88). A survey of energy intake by 33 stallions on a central Kentucky thoroughbred breeding farm was conducted that divided the stallions into four groups according to the number of services per season: < 80, 80–100, 101–120 and > 120 services (135). Stallions with the fewest services were fed 25.4 Mcal of DE daily, whereas stallions with more than 120 services were fed 27.7 Mcal DE daily. Body weights were not provided, but assuming weights of 600 kg, energy intakes were slightly above the 24.3 Mcal DE suggested by NRC. Thus the data from France and Kentucky confirm that NRC estimates approach the energy needs of stallions, although the precise energy requirements must be refined.

Many factors, including condition and training of the horse, ability of rider or driver, and environmental temperature, can influence the efficiency of energy utilization. Difficulties arise in describing work activity by horses. Few owners can detail the number of minutes their horses spend at various gaits, such as trotting, galloping, or walking. Therefore, the NRC (104) decided to provide general guidelines. Light work such as pleasure riding and bridle path hacking, moderate work such as ranch work and jumping, and intense work such as racing and polo were estimated to elevate energy needs above maintenance by 25, 50, and 100%, respectively. Obviously such a system has flaws. A horse doing ranch work for 1 h daily may need fewer calories than a horse performing light work 6 h daily. Nevertheless, several surveys indicate that the 1989 NRC (104) requirements for energy are much more accurate than the 1983 (103) estimates (44, 45, 47, 128, 141, 155). As mentioned above, maintenance energy requirements for horses weighing 600 kg or less were similar per unit of body weight (i.e. W1). Recently, energy requirements of ponies and horses working at submaximal speeds were also shown to be linearly related to body weight (40). This finding suggests that the energy guidelines given above will also apply to ponies.

Although much remains to be learned about energy requirements of horses, the determination of precise energy requirements for various activities may not be the best use of resources during this period of financial restraint in research. More useful data and more effective use of resources might be

achieved by studying the effects of dietary manipulation on energy utilization. That is, studies on factors that influence energy metabolism are needed more than studies on energy requirements per se.

Dietary recommendations for human athletes have focused on selection of specific energy sources (carbohydrate, fat, and proteins) for use in various activities. Feed guidelines for equine athletes are limited. Like the human athlete, the horse preferentially uses glucose for anaerobic activity (e.g. flat racing) compared with fat metabolism for aerobic activity (e.g. endurance racing). General guidelines for performance have been proposed: Dietary soluble carbohydrate is needed to maintain glycogen (110); fiber is needed to maintain normal gut and digestive function; and excessive protein intake increases water demand and reduces efficiency of energy utilization because energy is used to excrete the nitrogen (95).

Few studies have specifically examined feed composition and its effect on energy utilization in athletic activity. Recently, however, the use of high-fat diets for athletic horses has become popular. The value of fat in horse diets has been debated since 1975, when Slade et al reported that dietary fat enhanced endurance (137). Indeed, fat may improve muscular activity by sparing glucose. Horses fed fat had increased mobilization of free fatty acids (54, 60). Dietary fat may also reduce thermal stress in horses working in a hot environment (131, 133) and may spare or enhance glycogen storage (70, 105, 106, 149). Other metabolic effects have been noted as well. The composition of dietary fat significantly affects serum fatty acid composition in ponies (92). Inedible tallow diets elevated serum oleic acid concentrations, whereas blended oil diets reduced serum oleic acid content (92). Triglyceride infused into horses had an average half-life of 269 min-10 times longer than in other animals (102). Moser et al noted great variability among individual horses in the utilization of triglyceride and suggested that breed may have an effect on triglyceride clearance. As opposed to results of other studies, however, dietary fat did not alter plasma glucose or free fatty acid concentrations in intensely worked horses (102). Similarly, horses fed diets with 10% added soybean meal showed no difference in blood glucose (112). Lawrence (81) concluded that, "To date the information supporting a positive effect of supplemental fat on fuel metabolism is inconclusive," and Pagan et al (112) wrote, "The results of dietary manipulations on equine performance have not been conclusive." The statements of these authors indicate that most studies evaluating dietary fat have been of short duration and that until long-term effects are quantitated, the results of fat trials should be applied cautiously. Even in studies in which performance was enhanced, authors have advocated caution. Harkins et al (56) reported that the addition of fat improved times in horses racing 1600 m, but they nevertheless concluded, "More research is needed before an ergogenic benefit from fat can be claimed." Other possible benefits from fat have been

suggested. Increased energy density and greater energy intake and decreased gut fill have also been proposed as factors that might improve performance.

Environmental Temperature

Extremes of ambient temperature aggravate energy metabolism in the horse. The thermoneutral zone (TNZ) is the temperature range in which metabolic heat production (Hp) does not need to be increased to maintain thermostability. The lower critical temperature (LCT) is the lower limit of the thermoneutral zone and is the temperature below which an increase in metabolic heat production is needed to conserve body temperature. The upper critical temperature (UCT) is the upper end of the TNZ and is the temperature at which evaporative heat loss must be increased to reduce body temperature. The values for LCT and UCT depend on the ambient temperature to which the horse is currently accustomed; the horse's body condition, body size, breed, and function; the horse's energy intake; and (perhaps) the composition of the diet. Adaptation to cold weather occurs gradually over 10–11 days (134). Thus the LCT is a management guideline for determining when energy intakes must be elevated.

Adult horses fed at maintenance had a LCT of -15° C (91). Metabolic rate increased linearly in response to cold temperatures below -20° C, and it was predicted that at an effective temperature lower than -15° C, an adult 500-kg horse would require 367 kcal metabolizable energy (ME) (407 kcal DE) per degree decrease in ambient temperature. An adult horse of this size requires 16.4 Mcal DE for maintenance. Therefore, cold temperatures increase DE requirements by 2.44% per degree decrease below LCT. Weanling colts exposed to average barn temperatures below -5° C gained weight at a rate 23% slower than that of foals kept at 10° C and fed exactly the same diet (26). Using a linear model based on energy intake, gain, and ambient temperature, a LCT of 0° C was obtained, and the maintenance component of the equation used to determine energy requirements for growth was calculated to increase 1.3% per degree decrease below 0° C. In cold-housed foals, fiber digestibility increased, but phosphorus digestibility decreased. This reduction could be related to changes in hindgut motility. Recent studies confirmed that the LCT for growing draft horses was at least 0° C and may be even lower (29). Nutrient digestibility was unaffected by ambient temperature. In contrast, French studies reported a 9% increase in maintenance energy requirement in summer (19.4° C) compared with winter (7.5° C) (89). Interestingly, variation among horses was almost as great (8%) as differences between seasons, and no differences were observed between 4-year-old and 11-year-old horses (89). However, horses of these ages are metabolically mature animals. Thus, additional data are required to determine the effects of cold on maintenance energy for young growing horses less than one year of age.

In hot weather, the energetic equation is affected by the need to dissipate

body heat. This dissipation is particularly important in the exercising horse, whose metabolic rate can increase 40-60 times that at maintenance. In hot weather, the heat increment of feeds is likely more important than in cold weather. Heat production associated with digestion of feed must be dissipated. Therefore, diets that can effectively increase metabolizable energy without increasing heat production are useful during hot weather conditions. High-fat diets fed to horses provided an elevated metabolizable energy without increasing heat production (92). Similarly, horses exercised in hot weather conditions required less digestible energy when fed 10% fat diets than when fed a control hay/grain diet. This outcome was attributed to lower heat of fermentation and lower energy need for thermoregulation (117).

PROTEIN

The horse receives little amino acid nutrition from protein contributed by hindgut microflora. Thus the amino acids must be supplied by the diet. Studies defining the amino acid requirements of mature horses are scarce. Solutions of amino acids have been given to racehorses in attempts to improve their speed. A mixture of amino acids (leucine, isoleucine, valine, glutamine, and carnitine) administered 30 min pre-exercise lowered plasma lactate response and improved oxidative capacity (49). Further studies are needed to fully evaluate the use of amino acids. Carnitine is not a true amino acid but rather a low molecular weight quaternary amine involved in the transfer of long-chain fatty acids. Muscle carnitine concentrations increase with horse age and training (42). Investigators concluded that supplemental carnitine is unlikely to improve performances of horses because muscle stores cannot be increased by feeding carnitine (139). Carnosine (β-alanyl-L-histidine) is an important cellular buffer in horses with a muscle content approximately four- to sevenfold greater than in humans or dogs (57). However, increasing dietary histidine did not increase muscle carnosine content in horses (99).

Most amino acid studies in horses use growing foals. Lysine is considered the first limiting amino acid for young horses. The daily lysine requirement is 2.1 and 1.9 g Mcal DE⁻¹ for weanlings and yearlings, respectively (104). In conventional diets, this translates to 0.6 and 0.5% lysine in diets for weanlings and yearlings, respectively, and corresponds to 32–36 g lysine day⁻¹ for growing horses expected to attain a mature weight of 500 kg (104). Finnish studies (123) concluded that weanlings require ~ 31 g lysine day⁻¹, a figure that correlates closely with NRC (104) values. Insufficient data are available to determine adequacy levels of other amino acids. However, threonine requirement was recently investigated in two experiments using a total of 39 yearling horses fed a basal ration of corn, oats, and soybean meal with coastal Bermudagrass hay (51). Either 0.2% lysine or 0.2% lysine and 0.1% threonine were

added to the grain. The addition of lysine alone increased protein synthesis and promoted muscle gain, but the addition of threonine and lysine caused an even greater increase. Therefore, it was concluded that threonine was the second limiting amino acid. Unfortunately, the amino acid composition of the basal diet used was not provided. Arginine has been suggested as a limiting amino acid in commercial milk replacers because blood arginine decreased when the replacers were fed (18). However, blood amino acid concentrations in foals must be interpreted critically, given the apparently profound age effect on plasma amino acid content in horses (154). Foals born to mares supplemented with 10 g of carnitine for two weeks prepartum had higher plasma carnitine levels, but their growth rate remained unaffected (6).

The dearth of specific studies on amino acid metabolism in horses has made it necessary to base requirements on protein content (104). Are the NRC (104) values for protein needs reasonable? Growing draft horse colts fed diets containing 12% protein grew less well (88 kg) from 6-8 months of age than those fed 16% protein supplied as either soybean meal or canola meal (99 or 96 kg, respectively) (28). The average daily lysine intake was 25 g for colts fed the basal diet and 43 g for colts fed protein-supplemented diets. No other amino acids were controlled in the study. Therefore, although these data do not help establish lysine requirements, they support the NRC (104) contention that more than 12% dietary protein is needed for foals aged six months. The exact amount of dietary protein required depends on the dietary energy content. NRC (104) proposed that 50 g of crude protein is needed per Mcal DE. In the above study, the basal diet supplied 47 g protein Mcal DE⁻¹, whereas the added protein diets provided 55-58 g protein Mcal DE⁻¹. However, ratios of 39-42 g protein Mcal DE⁻¹ supported maximal growth when energy intakes were high (60%) above normal) (108).

Protein intake by performance horses has important metabolic implications. Dietary protein is not an efficient energy source for exercise (97). Nevertheless, feeding high-protein diets (18%) to horses did not influence liver or muscle glycogen stores (98). Meyer (95) concluded that protein intakes greater than 2 g digestible protein kg BW^{-1} daily should be avoided because (a) water intake increases to promote urinary nitrogen excretion; (b) plasma urea levels increase, resulting in greater urea energy into the alimentary tract, which may increase incidence of enterotoxemia; (c) energy cost increases due to nitrogen excretion; and (d) barn ammonia concentrations increase secondary to elevated urinary nitrogen excretion, which can contribute to and intensify respiratory disorders. However, no adverse effects of high protein intakes other than increased water intakes were observed in endurance horses (61, 118).

Exercise can increase protein needs because nitrogen is lost in sweat and (perhaps) through muscle hypertrophy. To ensure adequate protein intakes for exercise, NRC (104) recommended that the crude protein:energy ratio remain

constant in diets of equine athletes and suggested a value of 40 g crude protein Mcal DE⁻¹. Because exercise requires increased feed intake to provide the needed energy, protein intakes are also elevated. A 500 kg horse doing intense work may ingest 1312 g or more of crude protein daily (2.6 g/kg BW⁻¹). Assuming that the dietary protein is 80% digestible, the intake of digestible protein would exceed 2 g/kg BW⁻¹, a value that Meyer (1987) suggests should be avoided.

Surveys indicate that horses at racetracks are often fed more than 2 g digestible protein kg BW⁻¹ (44, 45, 47, 141), as are endurance horses (118). Would the performance of these horses benefit from a reduced protein intake? Is the value of 40 g crude protein Mcal DE⁻¹ proposed by NRC (104) too high? The trend in diets for human athletes has been to decrease protein and increase carbohydrates. A better understanding of energy metabolism in horses and the influence of the ratio of carbohydrate:protein:fat could play a key role in improving the nutrition of the performance horse.

MINERALS

Investigations in trace minerals, particularly copper, zinc, and selenium, have been spurred by reports that copper and zinc supplementation could decrease the incidence of DOD (75, 77). Diets containing 20–30 ppm copper and 70–80 ppm zinc were recommended for weanlings, and 20–25 ppm copper and 60–70 ppm zinc were suggested for yearlings (43). NRC (104) found the data supporting increased copper and zinc requirements inconclusive and proposed that diets containing 10 copper ppm and 40 ppm zinc were adequate. The quantity of trace minerals required to decrease the incidence of DOD remains a point of controversy.

Copper deficiency can cause skeletal abnormalities. Severe lesions developed in foals fed diets containing low levels of copper (1.5 ppm) aggravated by high dietary zinc (15, 17). Defective collagen cross-linking and altered matrix remodeling were reported in bones of foals fed 7 ppm copper (65). The value of copper has been clearly established, but debates continue over the level required to prevent DOD.

Many studies in which copper has been fed at marginal intakes have revealed no clinical or histological bone abnormalities in growing horses. No lesions developed in foals fed 7 ppm copper (16), and no gross skeletal abnormalities or decreases in bone mineral were noted in foals fed diets containing 9 ppm copper and 20 ppm zinc or 20 ppm copper and 61 ppm zinc (107). Furthermore, most feed companies in the US have significantly increased the copper content of their feeds since 1987, but developmental orthopedic disease continues to afflict growing foals.

Copper supplementation to the lactating mare does not appear to be an

effective means of increasing copper intake by the foal. Milk copper did not increase significantly in mares given a supplement of 90 mg of copper daily (124) or in mares whose dietary copper was increased from 4 to 12 ppm (4). Mares fed diets containing either 7 or 13 ppm copper had similar milk copper concentrations (14). Knight et al (76) fed higher dietary copper concentrations than those in the above studies. They concluded that dietary copper supplementation can affect milk copper concentration but stated, "whether this [increase] is of physiological significance to the foals remains to be determined."

Horse milk contains three times more copper than cow milk but not as much as guinea pig or human milk (1). The zinc content of horse milk, however, is only about half that of cow milk and is less than that found in human or guinea pig milk. The zinc:copper (Zn:Cu) ratio in horse milk was 12:1 compared with 76:1, 8:1, and 7:1 for cow, guinea pig, and human milk, respectively. Other studies have found much lower Zn:Cu ratios in mares' milk, with variable values from 4:1 to 8:1 over the course of lactation (130). NRC (104) summarized data from several studies and found milk Zn:Cu ratios of 5.6:1 during weeks 1-4, 7.7:1 during weeks 5-8, and 9:1 during weeks 9-21. The significance of the milk Zn:Cu ratio remains to be determined, but the significant differences between the cow and horse are intriguing.

The role of calcium and phosphorus in DOD needs to be reexamined. Dietary calcium deficiency causes nutritional secondary hyperparathyroidism in horses, particularly when combined with high intakes of phosphorus (80). Recently, foals fed either high-phosphorus diets or high-calcium and high-energy diets had increased incidences of dyschondroplasia (127). High calcium did not induce dyschondroplasia in foals fed normal-energy diets. Thus, the high energy intake rather than the high calcium intake may be the inciting factor.

Macromineral and energy intake seem to exercise a confounding influence on bone strength in growing horses. A longitudinal study in which 41 foals were fed diets either high in grain or high in forage and dietary phosphorus at low (0.24–0.35%), adequate (0.68%), or high (0.97–1.06%) concentrations but with calcium concentrations greater than 1.4% revealed that foals fed the high-phosphorus diet, which had a calcium:phosphorus (Ca:P) ratio of 1.2–1.5, had a higher load at yield point and higher rectangular and principal moments of inertia (83). These results suggest that calcium and phosphorus intake or Ca:P ratio may affect bone strength in growing horses in a yet undefined manner.

Hyperkalemic periodic paralysis (HYPP) in horses has an interesting genetic-nutritional interaction. Clinical signs of weakness, muscular fasciculations, and (in some instances) recumbency resembling nonspecific colic occur in susceptible horses (121, 142). Attacks are precipitated by exercise and can be induced by administration of oral or intravenous potassium. This condition has been identified as an autosomal dominant disease in man, and recent studies

in horses have confirmed that it is an autosomal dominant disease with incomplete penetrance in horses as well (121). In fact, in horses all cases of HYPP can be traced to one blood line. The incidence of attacks in affected horses can be reduced by feeding diets low in potassium. Forages high in potassium and feeds containing molasses must therefore be avoided.

VITAMINS

Renewed interest in fat-soluble vitamins has led to their reassessment in horses. Serum retinol (average 200 μg liter⁻¹) and α-tocopherol (average 2.3 μg ml⁻¹) concentrations in horses are one third of those in humans (86). Serum 25hydroxyvitamin D₃ (2.2 ng ml⁻¹) was also lower in horses than in humans. Vitamin D and E were ~ 30% higher in summer than winter (87). Approximately 20% of the horses sampled were considered vitamin E deficient in winter. Plasma vitamin A (0.55 μmol liter⁻¹) and vitamin E (5.6 μmol liter⁻¹) concentrations are lower in weanlings than in adult horses (0.72 µmol liter⁻¹ vs 8.8 µmol liter⁻¹) (11). However, liver vitamin A content of newborn presuckle foals is significantly higher than values reported for other newborns (67). Earlier studies found that the low values of both vitamin D₂ and D₃ were unresponsive to seasonal effects, which implied a limited ability of the horse to synthesize vitamin D in the skin or to utilize dietary vitamin D. Ironically, a common problem associated with fat-soluble vitamins is not deficiency but toxicity due to exuberant supplementation, particularly of injectable fat-soluble vitamins.

Numerous investigations in equine vitamin E utilization have been conducted. These were motivated by several reports that (a) immune responses are enhanced by antioxidants; (b) vitamin E may protect against exercise-induced oxidative damage in other species; and (c) at least two clinical conditions in horses may involve abnormalities in vitamin E metabolism.

NRC (104) increased minimum vitamin E requirements of the horse at maintenance to 50 IU kg⁻¹ feed and to 80 IU kg⁻¹ feed for pregnant and lactating mares, growing horses, and working horses. The previous estimate (103) was 15 IU kg⁻¹ feed. The increase was based on studies that higher levels improved immune responses in horses (3) and were needed to maintain plasma vitamin E levels (87, 119). Furthermore, the muscle soreness and lameness observed in zebras and in Przewalskii's horses after capture and restraint when the diet contained 50 IU kg⁻¹ feed did not occur when the diet contained 100 IU vitamin kg⁻¹ (104).

We are not aware of any recent studies on the effect of vitamin E on the immune response in horses, although many such studies continue to be conducted in other species. Vitamin E supplementation caused a response in plasma vitamin E concentrations (122), but concentrations of vitamin E as high

as 80 IU kg⁻¹ feed were needed to maintain plasma levels. Plasma vitamin E concentrations, however, are variable. Serum α -tocopherol concentrations varied significantly among horses fed the same diet and from sample to sample from the same horse (101). Age of horse may also be a factor in plasma vitamin E concentration. Adult horses had sufficient vitamin E reserves to make serum α -tocopherol a relatively insensitive measure of vitamin E status, but yearling horses seemed to be more sensitive to dietary changes (101). Values for plasma vitamin E varied greatly from sample to sample, and a minimum of three blood samples were required to establish a baseline for each animal (22). The form of vitamin E may also influence plasma response to dietary vitamin E intakes. D- α -tocopherol polyethylene glycol 1000 succinate (TPBS) produced a greater plasma response than an equivalent amount of dl- α -tocopherol acetate in some studies (113) but not in others (64).

In horses, the effects of exercise on vitamin E status do not appear to be as dramatic as might be expected from reports in other species. For example, Witt et al speculated that exercise may induce free radical formation in muscle and liver, which results in oxidative damage due to lipid peroxidation in rats (152). These authors state that studies in several species demonstrated that the damage can be reduced by dietary antioxidants such as vitamin E.

Exercise did not affect serum vitamin E concentration in horses exercised on a treadmill at a fast walk and trot for up to 12 min/day (101). Horses fed diets containing 13 IU vitamin E kg⁻¹ feed and exercised on a high-speed treadmill for 30 min/day 3 times a week at a rate of 7 m/s over a 4-month period showed no decrease in plasma vitamin E with exercise. No clinical signs of vitamin E depletion were noted (114). Exercise did not appear to greatly influence vitamin E status of ponies fed a diet containing 42 IU kg⁻¹ for 10 months (94). These studies indicate that vitamin E may not be as critical for exercise as once thought, which led to the suggestion that some other antioxidant is utilized when vitamin E is limiting. Tiidius & Houston (147) recently indicated that the role of exercise in vitamin E nutrition needs further study. They reported that rats fed a vitamin E-free diet for eight weeks while performing either acute or chronic exercise did not exhibit greater peroxidation than controls given vitamin E. Similarly, Goldfarb (50) concluded that limited information is available concerning the effects of vitamin E on exercise-induced oxidative stress. He stated that the viability of vitamins E and C alone and in conjunction with each other in preventing exercise-induced lipid peroxidation requires further investigation

Vitamin E may be involved in equine motor neuron disease (EMND). EMND was first reported in 1990 by Cornell researchers (25). Since then, many other cases have been reported, not only in the US, but also in Europe (145). Affected horses experience generalized weakness, muscle fasciculations, muscle atrophy, and weight loss. Motor neurons in the spinal cord and

brain stem degenerate, resulting in axonal degeneration in the ventral roots and peripheral and cranial nerves. EMND is similar to amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, in humans. Almost all of the horses with EMND diagnosed at Cornell have been without access to pasture and had very low plasma and adipose vitamin E levels (34). Further studies are needed to determine the relationship, if any, between vitamin E nutrition and EMND. The recent suggestion by Rosen et al (120) that an antioxidant deficiency may be involved in the development of ALS in humans lends support to the theory that vitamin E may play a role in EMND.

Equine degenerative myeloencephalopathy (EDM) is a diffuse degenerative disease of the spinal cord and brain stem. Affected animals show various signs: clumsiness, inability to execute complicated movements, malpositioning of limbs at rest, or obvious ataxia (12). Blythe & Craig (12) proposed that EDM is a familial disease that involves a deficiency of vitamin E. They recommended supplementation with 6000 IU of vitamin E day⁻¹ to reduce the incidence of EDM in young horses from affected families or to improve the condition of some affected animals. The reason for the response to high vitamin E intakes in certain families of horses is unknown, but it has been suggested that such horses are more susceptible to antioxidant deficiencies or have an increased antioxidant need during the first year of life (13). Conversely, no differences were found in serum vitamin E or glutathione peroxidase activity of 40 horses with confirmed EDM vs 49 age-matched controls (33).

CONCLUSIONS

Considerable progress has been made in the field of equine nutrition, but much remains to be learned. For example, studies on the effect of dietary carbohydrate:fat:protein ratios in the diets of performing horses could prove very useful. Effects of nutrition of the mare and nutrition of the weanling on skeletal development in the growing horse also merit further study. Finally, nutrition/reproductive interactions should receive more attention.

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Literature Cited

- Anderson RR. 1992. Comparison of trace elements in milk of four species. J. Dairy Sci. 75(11):3050-55
- Argenzio RA. 1990. Physiology of digestive secretory and absorptive processes. In The Equine Acute Abdomen.
- ed. NA White, pp. 25-35. Malvern, PA: Lea & Febeger
- Baalsrud KJ, Overness G. 1986. The influence of vitamin E and selenium supplements on antibody production in horses. Equine Vet. J. 18:472-74

- Baucus KL, Ralston SL, Rich G, Squires EL. 1987. The effect of dietary copper and zinc supplementation on composition of mares milk. Proc. 10th Equine Nutr. Physiol. Soc. Symp., pp. 179-84 Fort Collins, Colo.: Colo. State Univ.
- Beard WL, Bertone AL. 1992. Surgery of the equine small intestine. Comp. Contin. Educ. Pract. Vet. 14:1508-13
- Benamou AE, Harris RC. 1993. Effect of carnitine supplementation to the dam on plasma carnitine concentration in the suckling foal. Equine Vet. J. 25:49-52
- Bergman EN. 1990. Energy contributions of volatile fatty acids from the gastrointestinal tract in various species. *Physiol. Rev.* 70:567-90
- Bertone AL, Ralston SL, Stashak TS. 1989. Fiber digestion and voluntary intake in horses after adaptation to extensive large-colon resection. Am. J. Vet. Res 50:1628-32
- Bertone AL, Toofanian F, Stashak TS. 1990. Alteration of intestinal enzyme activities associated with extensive large-colon resection in horses. J. Am. Vet. Med. Assoc. 51:1329-34
- Bertone AL, Van Soest PJ, Stashak TS. 1989. Digestion, fecal and blood variables associated with extensive large colon resection in the horse. Am. J. Vet. Res. 50:253-58
- Blakley BR, Bell RJ. 1994. The vitamin A and vitamin E status of horses raised in Alberta and Saskatchewan. Can. Vet. J. Vol. 35. In press
- Blythe LD, Craig AM. 1992. Equine degenerative myeloencephalopathy. 1. Clinical signs and pathogenesis. Comp. Contin. Educ. Pract. Vet. 14:1215-21
- Blythe LD, Craig AM. 1992. Equine degenerative myeloencephalopathy. 2. Diagnosis and treatment. Comp. Contin. Educ. Pract. Vet. 14:1633-37
- Breedveld L, Jackson SG, Baker JP. 1987. The determination of a relationship between copper, zinc, and selenium levels in mares and those in the foals. See Ref. 4, pp. 159-64.
- Bridges CH, Harris ED. 1988. Cartilaginous fractures ("osteochondritis dissccans") induced experimentally in foals with low copper diets. J. Am. Med. Assoc. 193:215-21
- Bridges CH, Moffitt PG. 1990. Influence of variable content of dietary zinc on copper metabolism of weanling foals. Am. J. Vet. Res. 51:275-80
- Bridges CH, Womak JE, Harris ED. 1984. Considerations of copper metabolism in osteochondrosis of suckling foals. J. Am. Vet. Med. Assoc. 185:173-78

- Buffington CAT, Knight DA, Kohn CW, Madigan JE, Scaman PA. 1992. Effect of protein source in liquid formula diets on food intake, physiologic values and growth of equine neonates. Am. J. Vet. Res. 53:1941-46
- Burns GA. 1992. The teniae of the equine intestinal tract. Cornell Vet. 82: 187-212
- Clarke LL, Roberts MC, Argenzio RA. 1990. Feeding and digestive problems in horses: physiologic responses to a concentrated meal. In Veterinary Clinics N. America, Equine Practice, ed. HF Hintz, 6:433-50. Philadelphia: Saunders
- Coenen M. 1990. Observations on the occurrence of feed related gastric ulcers in ponies. Schweiz. Arch. Tierheilk. 132: 121-26
- Craig AM, Blythe LL, Lassen ED, Rowe KE, Barrington R, Slizeski M. 1989. Variations of serum vitamin E, cholesterol, and total serum lipid concentrations in horses during a 72-hour period. Am. J. Vet. Res. 50:1527-31
- Crowell-Davis SL, Caudle AB. 1989. Coprophagy by foals: recognition of maternal feces. Appl. Anim. Behav. Sci. 24:267-72
- Crutzen PJ, Aselmann I, Seiler W. 1986. Methane production by domestic animals, wild ruminants, other herbivores, fauna and humans. Tellus Ser. B Chem. Phys. Metorol. 38:271-84
- Cummings JF, DeLahunta A, George C, Fuhrer L, Valentine BA, et al. 1990. Equine motor neuron disease: a preliminary report. Cornell Vet. 80:357-80
- Cymbaluk NF. 1990. Cold housing effects on growth and nutrient demand of young horses. J. Anim. Sci. 68:3152– 62
- Cymbaluk NF. 1990. Comparison of forage digestion by cattle and horses. Can. J. Anim. Sci. 70:601-10
- Cymbaluk NF. 1990. Using canola meal in growing draft horse diets. Equine Pract. 12(4):13-19
- Cymbaluk NF, Christison GI. 1993. Cold weather—does it affect foal growth? Proc. 4th Int. Livest. Env. Symp., Am. Soc. Agric. Eng., St. Joseph, pp. 23-30
- Cymbaluk NF, Christison GI, Leach DH. 1990. Longitudinal growth analysis of horses following limited and ad libitum feeding. Equine Vet. J. 22(3):198– 204
- Davison KE, Potter GD, Evans JW, Greene LW, Hargis PS, et al. 1991. Growth, nutrient utilization, radiographic bone characteristics and postprandial thyroid hormone concentrations

- in weanling horses fed added dietary fat. J. Equine Vet. Sci. 11:119-25
- Demment MW, Van Soest PJ. 1985. A nutritional explanation for body-size patterns of ruminant and nonruminant herbivores. Am. Nat. 125(5):641-72
- Dill SG, Kallfelz FA, deLuhunta A, Waldron CH. 1989. Serum vitamin E and blood glutathione peroxidase values of horses with degenerative myeloencephalopathy. Am. J. Vet. Res. 50: 166-68
- Divers T. 1993. Vitamin deficiency considered in equine form of Lou Gehrig's disease. Equine Vet. Data 141(3):341
- Doreau M, Boulot S. 1989. Recent knowledge on mare milk production—a review. Livest. Prod. Sci. 22(3-4):213– 36
- Doreau M, Boulot S, Barlet JP, Patureau-Mirand P. 1990. Yield and composition of milk from lactating mares, effect of lactation stage and individual differences. J. Dairy Res. 57(4): 449-54
- Doreau M, Boulot S, Bauchart D, Barlet JP, Martin-Rosset W. 1992. Voluntary intake milk production and plasma metabolites in nursing mares fed two different diets. J. Nutr. 122(4):992-99
- Doreau M, Boulot S, Martin-Rosset W. 1991. Effect of parity and physiological state on intake milk production and blood parameters in lactating mares differing in body size. Anim. Prod. 53(1): 111-18
- Doreau M, Martin-Rosset W, Boulot S. 1988. Energy requirements and the feeding of mares during lactation—a review. Livest. Prod. Sci. 20(1):53-68
- Eaton MD, Evans DL, Rose RL. 1991. Comparison of the energy cost of treadmill exercise in horses and ponies. Proc. Nutr. Soc. Aust. 16:26 (Abstr.)
- Ford EJH, Simmons HA. 1985. Gluconeogenesis for caecal propionate in the horse. Br. J. Nutr. 53:55-60
- Foster CVL, Harris RC. 1992. Total carnitine content of the middle gluteal muscle of Thoroughbred horses: normal values, variability and effect of acute exercise. Equine Vet. J. 24:52-57
- Gabel AA, Knight DA, Reed SM, Pultz JA, Powers JD, et al. 1987. Comparison of incidence and severity of developmental orthopedic disease on 17 farms before and after adjustment of ration. Proc. Am. Assoc. Equine Pract. 33:163-69
- Gallagher K, Leech J, Stowe H. 1992. Protein, energy and dry matter consumption by racing Thoroughbreds: a field survey. J. Equine Vet. Sci. 12:43-48

- Gallag er K, Leech J, Stowe H. 1992. Protein, energy and dry matter consumption by racing Standardbreds: a field survey. J. Equine Vet. Sci. 12:382–88
- Gibbs PG, Potter GD, Kreider JL, Boyd CL. 1988. Digestion of hay protein in different segments of the equine digestive tract. J. Anim. Sci. 66:400-6
- Glade MJ. 1983. Nutrition and performance of racing Thoroughbreds. Equine Vet. J. 15:31-36
- Glade MJ. 1988. Dietary yeast culture supplementation enhances urea recycling in the equine large intestine. Nutr. Rep. Int. 37:11-18
- Glade MJ. 1991. Timed administration of leucine, isoleucine, valine, glutamine and carnitine to enhance athletic performance. The Equine Athl. 4(5):1, 5-10
- Goldfarb AH. 1993, Antioxidants: role of supplementation to prevent exerciseinduced oxidative stress. Med. Sci. Sports Exerc. 25:232-36
- Graham PM, Ott EA, Brendemuhl JH, Ten'Broeck SH. 1993. The effect of supplemental lysine and theonine on growth and development of yearling horses. Proc. 13th Equine Nutr. Physiol. Symp., pp. 80-81. Gainesville: Univ. Fla.
- Gröndahl AM, Dolvik NI. 1993. Heritability estimations of osteochondrosis in the tibiotarsal joint and of bony fragments in the palmar-plaantar portion of the metacarpo and metatarsophalangeal joint of horses. J. Am. Vet. Med. Assoc. 203(1):101-4
- Hall RR, Jackson SG, Baker JP, Lowry SR. 1990. Influence of yeast culture supplementation on ration digestion by horses. J. Equine Vet. Sci. 10:130-34
- Hambleton PL, Slade LM, Hamar DW, Kienholz EW, Lewis LD. 1980. Dietary fat and exercise conditioning effect on metabolic parameters in the horse. J. Anim. Sci. 51:1330-39
- Hardy J, Bertone AL. 1992. Surgery of the equine large colon. Comp. Contin. Educ. Pract. Vet. 14:1501-7
- Harkins JD, Morris GS, Tulley RT, Nelson AG, Kamerling SG. 1992. Effect of added fat on racing performance in Thoroughbred horses. J. Equine Vet. Sci. 12:123-29
- Harris RC, Marlin DJ, Dunnett M, Snow DH, Hultman E. 1990. Muscle buffering capacity and dipeptide content in the Thoroughbred horse, greyhound dog and man. Comp. Biochem. Physiol. A 97: 249-51
- Hintz, HF, Argenzio RA, Schryver HF. 1971. Digestion coefficients, blood glucose levels, and molar percentage of

- volatile acids in intestinal fluid of ponies fed varying forage-grain ratios. J. Anim. Sci. 33:992
- Hintz HF, Hogue DE, Lowe JE, Schryver HF. 1971. Apparent digestibility in various segments of the digestive tract of ponies fed diets with varying roughage-grain ratios. J. Anim. Sci. 32: 245-48
- Hintz HF, Ross MW, Lesser FR, Leids PE, White KK, et al. 1978. The value of dietary fat for working horses. J. Equine Med. Surg. 2:483-88
- Hintz HF, White KK, Short CE, Lowe JE, Ross M. 1980. Effects of protein levels on endurance horses. Proc. 72nd Annu. Meet. Am. Soc. Anim. Sci., pp. 202-3. Ithaca, NY: Am. Soc. Anim. Sci.
- Holter JB, Young AJ. 1992. Methane prediction in dry and lactating Holstein cows. J. Dairy Sci. 75:2165-75
- Hoppe F, Philipsson J. 1985. A genetic study of Osteochondrosis Dissecans in Swedish horses. Equine Prac. 7:7-15
- 64. Howard KA, Moore SA, Radecki SV, Schmitt SM, Shelle JE, Ullrey DE. 1992. Relative bioavailability of various sources of vitamin E for white-tailed deer, swine and horses. Res. Rep. MI SU Agric. Exp. Stn. 520:191-98
- Hurtig MB, Green SL, Dobson H, Mikuni-Takagaki, Choi J. 1993. Correlative study of defective cartilage and bone growth in copper-deficient foals. Equine Vet. J. 16:27-28 (Suppl.)
- Illius AW, Gordon IJ. 1992. Modelling the nutritional ecology of ungulate herbivores evolution of body size and competitive interactions. *Oecologia* 89: 428-34
- Irwin LN, Robberson JA, Crouch G. 1991. Hepatic vitamin A and carotene levels in the newborn foal. J. Equine Vet. Sci. 11:278-80
- Izaely H, Choshniak I, Stevens CE, Demment MW, Shkolnik A. 1989. Factors determining the digestive efficiency of the domesticated donkey (Equus asinus asinus). Q. J. Exp. Physiol. 74:1-6
- Deleted in proof
 Jones DL, Potter GD, Greene LW, Odom TW. 1992. Muscle glycogen in exercised miniature horses at various body conditions and fed a control or fat-supplemented diet. J. Equine Vet. Sci. 12:287-91
- Julliand V. 1992. Microbiology of the equine hindgut. Pferdeheilkunde 1. Eur. Konf. Ernahrung Pferdes. Inst. Tiernahrung Tieraerztl. Hochsche Hannover, pp. 42-47
- 72. Kamphues J, Denell S, Radicke S. 1992. Lipopolysaccharides in the alimentary

- tract of ponies fed hay or a concentrate ration. Pferdeheilkunde I. Eur. Konf. Ernahrung Pferdes. Inst. Tiernahrung Tieraerztl. Hochsche Hannover, pp. 59-62
- Kim SM, Kim CM, Lee HK, Park WP, Lim YJ, et al. 1991. Evaluation of nutrient values of some feedstuffs and the effects of yeast culture supplementation on digestibilities of nutrients and blood parameter in horse. J. Anim. Nutr. Feedst. (Korea) 15(5):272-80
- 74. Kleiber M. 1961. The Fire of Life, p. 262. New York: Wiley
- Knight DA, Gabel AA, Reed SM, Embertson RM, Tyznik WJ. 1985. Correlation of dietary mineral to incidence and severity of metabolic bone disease in Ohio and Kentucky. Proc. Am. Assoc. Equine Pract. 31:445-49
- Knight DA, Schmall LM, Reed SM, Bamlage LR, Gabel AA. 1992. Effects of dietary copper on trace element concentrations of mares' milk and the serum and tissues of their foals. Proc. Equine Osteochondrosis in the '90s, pp. 23-24. Cambridge, Engl: Univ. Cambridge. (Abstr.)
- Knight DA, Weisbrode SE, Schmall LM, Reed SM, Gabel AA, et al. 1990. The effects of copper supplementation on the prevalence of cartilage lesions in foals. Equine Vet. J. 22(6):426-32
- Koller BL, Hintz HF, Robertson JB, Van Soest PJ. 1978. Comparative cell wall and dry matter digestion in the cecum of the pony and the rumen of the cow using in vitro and nylon bag techniques. J. Anim. Sci. 47:209-15
- Kowalski J, Williams J, Hintz HF. 1990.
 Weight gains of mares during the last trimester of gestation. Equine Pract. 12(7):6-10
- Krook L, Lowe JE. 1964. Nutritional secondary hyperparathyroidism in the horse. *Pathol. Vet.* 1:44-87
- Lawrence LM. 1990. Nutrition and fuel utilization in the athletic horse. ed. HF Hintz, In Veterinary Clinics N. America, Equine Practice, 6:393-418. Philadelphia: Saunders
- Lawrence LM, DiPietro J, Evert K, Parrett D, Moser L, Powell D. 1992. Changes in body weight and condition of gestating mares. J. Equine Vet. Sci. 12:355-58
- Leach DH, Cymbaluk NF, Hendrix A, Williams K. 1993. Mechanical properties of metacarpal bones of foals fed diets with different energy and phosphorus levels. 2nd Int. Workshop Anim. Locomot., Fallbrook, CA. pp. 14
- 84. Liu P, Collyer C, Hintz HF. 1994. Body

- weights of mares during late gestation. Equine Pract. 16(3):5-6
- Mackie RI, Wilkins CA. 1988. Enumeration of anerobic bacterial microflora of the equine gastrointestinal tract. Appl. Environ. Microbiol. 54:2155-60
- Maenpaa PH, Koskinen T, Koskinen E. 1988. Serum profiles of vitamin A, E and D in mares and foals during different seasons. J. Anim. Sci. 66:1418 23
- Maenpaa PH, Lappetelainen R, Vikkunen J. 1987. Serum retinol, 25-hydroxyvitamin D and α-tocopherol of racing Trotters in Finland. Equine Vet. J. 19:237-40
- Martin-Rosset W. 1990. L'alimentation des chevaux. INRA. Paris, France
- Martin-Rosset W, Vermorel M. 1991.
 Maintenance energy requirement variations determined by indirect calorimetry and feeding trials in hight horses. J. Equine Vet. Sci. 11:42-45
- Martin-Rosset W, Vermorel M, Tisserand JL. 1990. The new French energy (UFC) and nitrogen (MADC) system. Proc. 41st Annu. Meet. Eur. Assoc. Anim. Prod. 2:384 (Abstr.) Toulouse. France: Eur. Assoc. Anim. Prod.
- McBride GE, Christopherson RJ, Sauer W. 1985. Metabolic rate and plasma thyroid hormone concentrations of mature horses in response to changes in ambient temperature. Can. J. Anim. Sci. 65:375-82
- McCann JS, Meacham TN, Fontenot JP. 1987. Energy utilization and blood traits of ponies fed fat-supplemented diets. J. Anim. Sci. 65:1019-26
- McMeniman NP, Elliott R, Groenendyk S, Dowsett KF. 1987. Synthesis and absorption of cysteine from the hindgut of the horse. Equine Vet. J. 19:192-94
- McMeniman NP, Hintz HF. 1992. Effect of vitamin E status on lipid peroxidation in exercised horses. Equine Vet. J. 24: 482-84
- Meyer H. 1987. Nutrition of the equine athlete. In Equine Exercise Physiology, Vol. 2 ed. J Gillespie, N Robinson, pp. 644-773. Ann Arbor, MI: ICEEP Publ.
- Meyer H, Ablswede L. 1976. Über das intrauterine Wachstrum und die Korpuzusammenketzung von Fohlen sowie den Nahrstoffkedarf tragender Stuten. Übers. Tiernahig. 4:263 90
- Miller PA, Lawrence LM. 1988. The effect of dietary protein level on exercising horses. J. Anim. Sci. 66:2185-92
- Miller-Graber PA, Lawrence LM, Foreman JH, Bump KD, Fisher MG, Kurcz EV. 1991. Dietary protein level and energy metabolism during treadmill exercise in horses. J. Nutr. 121:1462-69

- Miller-Graber PA, Syers M. 1993. Effect of dietary histidine level on selected blood and muscle amino acids and metabolites in the mature horse. Proc. 13th Equine Nutr. Physiol. Symp., pp. 56-57. Gainesville: Univ. Fla.
- Moore BE, Dehority BA. 1992. Effects of diet and protozoa on total and celluloytic bacterial and fungal concentrations in the cecum and colon of the equine. J. Anim. Sci. 70(Suppl. 1):240 (Abstr.)
- Moore SA, Shelle JE, Ullrey DE. 1992. Factors affecting serum vitamin E in the horse. J. Anim. Sci. 70(Suppl. 1):244 (Abstr.)
- Moser LR, Lawrence LM, Novakofski J, Powell DM. 1993. Clearance of infused triglyceride by resting horses. Comp. Biochem. Physiol. A 104:361-63
- National Research Council. 1983. Nutrient requirements of horses. Washington, DC: NRC-NAS. 4th ed.
- National Research Council. 1989. Nutrient requirements of horses. Washington, DC: NRC-NAS. 5th ed.
- 105. Oldham SL, Potter GD, Evans JW, Smith SB, Taylor TS, Barnes SW. 1989. Storage and mobilization of muscle glycogen in racehorses fed a control and high-fat diet. Proc. 11th Equine Nutr. Physiol. Symp., pp. 57-62. Stillwater: Oklahoma State Univ.
- Oldham SL, Potter GD, Evans JW, Smith SB, Taylor TS, Barnes SW. 1990. Storage and mobilization of muscle glycogen in exercising horses fed a fat-supplement diet. J. Equine Vet. Sci. 10: 353-59
- Ott EA, Asquith RL. 1993. Trace mineral supplementation of weanling foals. J. Anim. Sci. 72(Suppl. 1):174 (Abstr.)
- Ott EA, Asquith RL. 1986. Influence of level of feeding and nutrient content of the concentrate on growth and development of yearling horses. J. Anim. Sci. 62:290-99
- Pagan JD. 1990. Effect of yeast culture supplementation on nutrient digestibility in mature horses. J. Anim. Sci. 68(Suppl. 1):371 (Abstr.)
- Pagan JD. 1992. Correct mixture of energy sources key in horses diet. Feedstuffs 64(23):11, 12, 22
- Pagan JD, Hintz HF. 1986. Equine energetics. I. Relationship between body weight and energy requirements of horses. J. Anim. Sci. 63:815-21
- Pagan JD, Tiegs W, Jackson SG, Murphy HQ. 1993. The effect of different fat sources on exercise performance in Thoroughbred racing horses. Proc. 13th

- Equine Nutr. Physiol. Symp., pp. 125-29. Gainesville: Univ. Fla.
- 113. Papas AM, Camre RC, Citino SB, Baer DJ, Wooden GR. 1990. Species differences in the utilization of various forms of vitamin E. Proc. Annu. Meet. Am. Assoc. Zool. Vet., pp. 186-90. S. Padre Isl., Tex.: Am. Assoc. Zool. Vet.
- 114. Fetersson K, Hintz HF, Schryver HF, Combs GF Jr. 1990. The effect of vitamin E on membrane integrity during submaximal exercise. In Equine Exercise Physiology, Vol. 3 ed. SGB Persson, A Lindholm, LB Jeffcolt, pp. 315-22. Davis, CA: ICEEP Publ.
- Philipsson J, Andreasson E, Sandgren B, Galin G, Carlsten J. 1993. Osteochondrosis in the tarsocrual joint and osteochondral fragments in the fetlock joints in Standardbred trotters. Equine Vet. J. Suppl. 16:38-41
- 116. Platt H. 1984. Growth of the equine foctus. Equine Vet. J. 16:247
- Potter GD, Webb SP, Evans JW, Webb GW. 1990. Digestible energy requirements for work and maintenance of horses fed conventional and fat-supplemented diets. J. Equine Vet. Sci. 10:214– 18
- Ralston SL. 1988. Nutritional management of horses competing in 160 km races. Cornell Vet. 78:53
- Roneus BO, Lindholm CA, Tyopponen JJ. 1986. Vitamin E requirements of adult Standardbred horses evaluated by tissue depletion and repletion. *Equine* Vet. J. 18:50-58
- Rosen DR, Siddique T, Patterson D, Figlewicz DA, Sapp P, et al. 1993. Mutations in Cu/Zn superoxide dismutase gene are associated with familial amyotrophic lateral sclerosis. Nature 362:59-62
- 121. Rudolph JA, Spier SJ, Byrns G, Rojas CV, Bernoco D, Hoffman EP. 1992. Periodic paralysis in Quarter horses: a sodium channel mutation disseminated by selective breeding. Nature Genet. 2: 144-47
- Saastamoinen MT, Juusela T. 1993. Serum vitamin E concentrations of horses on different vitamin E supplementation levels. Acta Agric. Scand. A 43:53-57
- 123. Saastomoinen MT, Kostinen E. 1993. Influence of quality of dietary protein supplement and anabolic steroids on muscular and skeletal growth of foals. Anim. Prod. 56(1):135-44
- Saastamoinen MT, Lahdekorpi M, Hyyppa S. 1990. Copper and zinc levels in the diet of pregnant and lactating

- mares. Eur. Assoc. Anim. Prod. Publ. 41(II):450 (Abstr.)
- 125. Sandgren B. 1993. Osteochondrosis in the tarsocrural joint and osteochondral fragments in the metacarpo/ metatarsophalangeal joints in young Standardbreds. PhD thesis. Fac. Vet. Med., Sweden Univ. Agric. Sci., Uppsala
- Sauer WS, Devlin TJ, Parker RJ. 1979.
 Effect of cecectomy on digestibility coefficients and nitrogen balance in ponies. Can. J. Anim. Sci. 59:145-51
- Savage CJ, McCarthy RN, Jeffcott LB. 1993. I. Effects of dietary energy and protein on induction of dyschondroplasia in foals. Equine Vet. J. 16:80-83. (Suppl.)
- Schils S, Jordan RM. 1989. Nutrition practices and philosophies of racehorse trainers: survey. Proc. 11th Equine Nutr. Physiol. Soc. Symp., pp. 238-39. Stillwater: Oklahoma State Univ.
- Schougaard, H, Falk-Rönne J, Philipsson J. 1990. A radio-graphic survey of tibiotarsal osteochondrosis in a selected population of trotting horses in Denmark and its possible genetic significance. Equine Vet. J. 22:288-89
- Schryver HF, Oftedal OT, Williams J, Soderholm LV, Hintz HF. 1986. Lactation in the horse: the mineral composition of mare milk. J. Nutr. 116:2142-47
- Schurg WA, Frei DL, Cheeke PR, Holtan DW. 1977. Utilization of whole corn plant pellets by horses and rabbits. J. Anim. Sci. 45:1317-21
- 132. Scott BD, Potter GD, Greene LW, Hargis PS, Anderson JG. 1992. Efficacy of a fat-supplemented diet on muscle glycogen concentrations in exercising Thoroughbred horses maintained in varying body conditions. J. Equine Vet. Sci. 12:109-13
- 133. Scott BD, Potter GD, Greene LW, Vogelsang MM, Anderson JG. 1993. Efficacy of a fat-supplemented diet to reduce thermal stress in exercising Thoroughbred horses. Proc. 13th Equine Nutr. Physiol. Symp., pp. 66-71. Gainesville: Univ. Fla.
- Senft RL, Rittenhouse LR. 1985. A model of thermal acclimation in cattle. J. Anim. Sci. 61:297-306
- 135. Siciliano PD, Wood CH, Lawrence LM, Duren SD. 1993. Utilization of a field study to evaluate digestible energy requirements of breeding stallions. Proc. 13th Equine Nutr. Physiol. Symp., pp. 293-98. Gainesville: Univ. Fla.
- Simmons HA, Ford EJ. 1991. Gluconeogenesis from propionate produced in the colon of the horse. Br. Vet. J. 147:340-45

- Slade LM, Lewis LD, Quinn CR. 1975. Nutritional adaptation of horses for endurance type performance. Proc. 4th Equine Nutr. Physiol. Symp., pp. 114– 28. Pomona: Calif. State Polytech. Univ.
- Smyth GB. 1988. Effects of age, sex and postmortem interval on intestinal lengths of horses during development. Equine Vet. J. 20:104-8
- Snow DH. 1992. A review of nutritional aids to energy production for athletic performance. The Equine Athl. 5(5):1, 6-9
- Soave O, Brand CD. 1991. Coprophagy in animals: A review. Cornell Vet. 81: 357-64
- Southwood LL, Evans DL, Bryden WL, Rose RJ. 1991. Feeding practices at Standardbred and Thoroughbred stables. Proc. Nutr. Soc. Aust. 16:217 (Abstr.)
- Spier SJ, Carlson JP, Holliday TA, Cardinet GH, Pickar JG. 1990. Hyperkalemic periodic paralysis in horses. J. Am. Vet. Med. Assoc. 197:1009-17
- Sprouse RF, Garner HE, Green EM. 1987. Plasma endotoxin levels in horses subjected to carbohydrate induced laminitis. Equine Vet. J. 19:25-28
- Stevens CE. 1977. Comparative physiology of the digestive system. In Duke's Physiology of Domestic Animals, ed. JM Swensen, pp. 216-32. Ithaca, NY: Comstock Publ. Cornell Univ. 9th ed.
- 145. Sustronck B, Deprez P, van Roy M, Muylle E, Roels S, Thoonen E. 1993. Equine motor neuron disease: the first confirmed cases in Europe. Vlaans Diergeneeskd. Tijdschr. 62:40-44
- Thonney ML, Touchbury RW, Goodrich RD, Meiske JC. 1976 Intraspecies relationship between fasting heat production and body weight: a re-evaluation of W.75. J. Anim. Sci. 43:692-704

- Tiidus PM, Houston ME. 1993. Vitamin
 E status does not affect the responses
 to exercise training and acute exercise
 in female rats. J. Nutr. 124:834-40
- Vermorel M, Martin-Rosset W, Vernet J. 1991. Energy utilization of two diets for maintenance by horses; agreement with the new French net energy system. J. Equine Vet. Sci. 11:33-35
- 149. Webb SP, Potter GD, Evans JW. 1987. Physiologic and metabolic response of race and cutting horses to added dietary fat. Proc. 10th Equine Nutr. Physiol. Symp., pp. 115-20. Fort Collins: Colo. State Univ.
- Webb SP, Potter GD, Massey KJ. 1985.
 Digestion of energy and protein by mature horses fed yeast culture. Proc. Equine Nutr. Physiol. Soc., pp. 64-67.
 East Lansing: Mich. State Univ.
- East Lansing: Mich. State Umv.
 151. Willard JG, Willard JC, Wolfram SA,
 Baker JP. 1977. Effect of diet on cecal
 pH and feeding behavior of horses. J.
 Anim. Sci. 45:87-93
- Witt EH, Reznick AZ, Viguie CA, Starke-Reed P, Packer L. 1992. Exercise, oxidative damage and effects of antioxidant manipulation. J. Nutr. 122: 766-73
- 153. Zentek J, Nyari A, Meyer H. 1992. Investigations on postprandal H₂ and CH₄ exhalation in the horse. Pferdheilkunde 8:64-66
- Zicker SC, Spensley MS, Rogers QR, Willits NR. 1991. Effect of age on the concentration of amino acids in the plasma of healthy foals. Am. J. Vet. Res. 52:1014-18
- Zmija G, Kienzle E, Meyer H. 1991.
 Feeds and feeding in German training stables of racehorses. Proc. 12th Equine Nutr. Physiol. Soc. Symp.,pp. 85-90.
 Alberta, Can: Univ. Calgary