Kentucky Equine Research®
World Leaders In Equine Nutrition

Advances in Equine Nutrition

Volume I

Edited by

J.D. Pagan

FAT SOLUBLE VITAMINS AND THE PERFORMANCE HORSE

KATHLEEN CRANDELL
Kentucky Equine Research, Inc., Middleburg, Virginia, USA

Introduction

Feeding vitamin supplements to horses has become commonplace. There are questions about the necessity of this practice, and the dangers involved. Fat soluble vitamins (A, D, E, and K) in particular can be a problem because of their ability to build up toxic levels in the body. In the horse, the amounts of these vitamins needed to prevent deficiency or produce toxicity have been established, but very little work has been done on the effect of exercise on these requirements. This paper addresses functions, forms and requirements of fat-soluble vitamins in the horse.

Fat-soluble vitamins

Vitamins are complex organic compounds, present in minute amounts in natural foodstuffs, that are essential to normal metabolism. Lack of said vitamins in the diet results in deficiency disease. Vitamins are a mixed group of compounds that are not similar to each other but are grouped by function, and are differentiated from trace elements by their organic nature. Although there are two general categories of vitamins, fat-soluble and water-soluble, this article focuses on those that are fat soluble.

Fat soluble vitamins are those that occur in nature in association with lipids and are absorbed along with dietary fats; conditions favorable to fat absorption would also be favorable to absorption of fat-soluble vitamins. Because of their lipid nature, fat-soluble vitamins can be stored in appreciable amounts in the body and are excreted in the feces via the bile. However, the relative ease of accumulating fat-soluble vitamins makes them more likely to cause problems in excessive amounts (particularly A and D) .

Level of fat in the diet may affect absorption of the fat-soluble vitamin A, D, E, and K, as well as the requirement for vitamin E. Fat-soluble vitamins may fail to be absorbed if digestion of fat is impaired.

Many vitamins are delicate substances that can suffer loss of activity due to unfavorable circumstances encountered during processing or storage of premixes and feeds. Stress factors for vitamins include humidity, pressure (pelleting), heat, light, oxidation-reduction, rancidity, trace minerals, pH, and interactions with other

vitamins, carriers, enzymes, and feed additives (NRC, 1973). Rancid fats destroy vitamins A, D and E. Dicumerol, found in certain plants, interferes with blood clotting by blocking the action of vitamin K. Certain sulfonamides may reduce intestinal absorption of vitamin K.

Vitamins represent only a minute fraction of animal feeds, amounting to less than 0.1% by weight and about 1-2% of the cost (depending on the diet used and the level of supplementation required). What many consumers do not understand is that a balanced vitamin fortification for meeting requirements of the animal will more than offset the cost of adding vitamins in the health and vitality of the animal.

Vitamin A

FUNCTIONS

Vitamin A is perhaps the most important of the vitamins and is indispensable for support of growth and health of a horse. Deficiency causes loss of vision, defects in bone growth, defects in reproduction, defects in growth and differentiation of epithelial tissues, and lowered resistance to disease and infection.

FORMS OF THE VITAMIN

Active

Vitamin A is a generic term describing several forms of retinoids. The most abundant forms which are found in use in the body are retinol, retinal, and retinoic acid. These forms of vitamin A will bind easily to other compounds. Transported forms commonly found in the corporal circulation are retinol bound to a binding protein and esters such as retinyl palmitate and retinyl acetate. Esterification makes vitamin A more stable. For that reason, synthetic or animal sources of vitamin A typically are in the form of esters.

Inactive

Although vitamin A does not occur in plant products, its precursor, carotene, does in several forms. However, only about 20 of the over 600 carotenes have provitamin A activity. These compounds are commonly referred to as provitamin A because the body can transform them into the active vitamin. Conversion usually occurs in the intestinal mucosa, but can occur to a lesser extent in the liver and adipose tissue as well. The most biologically active of the carotenes is ß-carotene, which theoretically could be split by the enzyme ‘ß-carotene 15, 15’-dioxygenase’ enzyme to form 2 vitamin A. Unfortunately, the actual conversion rate is much lower. From one mg of

ß-carotene the horse can only get approximately 0.01-0.05 mg of vitamin A. Rate of conversion is affected by many factors: age, activity, level of intake, environmental temperature, and the individual's vitamin A intake and status.

DIETARY SOURCES

Natural

Naturally occurring vitamin A can be found in fish oils, milk fat, egg yolk and liver. Typically these are not feeds eaten by a horse. Carotenes are the natural source of vitamin A for the horse, since they are found in abundance in green forages. Unfortunately, much of the carotene content is destroyed by oxidation in the process of field curing. Horses are not able to absorb sufficient quantities of ß-carotene from hay to meet their requirement, except possibly well made early bloom alfalfa hay (Fonnesbeck and Symons, 1967).

Synthetic

Sources of supplemental vitamin A are derived primarily from fish liver oil and from industrial chemical synthesis. Forms of vitamin A typically used as feed additives are all-trans retinyl palmitate and acetate. Since vitamin A is easily destroyed by oxidation they are stabilized by coating the vitamin in fat or gelatin and adding antioxidants.

REQUIREMENT

National Research Council (NRC) recommendations for vitamins are usually close to the minimal amount necessary to prevent deficiency signs, but not necessarily the optimal amount needed by the animal. Feeding at the NRC recommended level may lead to suboptimum performance even though the animal appears normal. In a 40 week study, growing ponies fed the NRC recommended level appeared normal, but upon postmortem examination exhibited lesions suggestive of vitamin A deficiency (Donoghue and Kronfeld, 1980). Optimal supplementation of vitamin A for the horse has been hypothesized to be more than twice (Greiwe-Crandell *et al*., 1997) and possibly as much as 5 times (Donoghue *et al*., 1981) the NRC recommendation.

Maintenance

Horse grazing high quality pastures during the growing season are easily receiving their full vitamin A requirement (in the form of carotene). Likewise, if the horse is consuming large quantities of very green alfalfa hay, vitamin A might not be a problem. If the horse subsists on hay and grains, then it will need supplementation. The NRC

recommends that diets for all horses should provide 30 to 60 IU of vitamin A activity and retinol or equivalent per kg of body weight per day (NRC, 1989). Even if the horse is grazing, additional vitamin A is not harmful as long as the amount given is not excessive. Vitamin A supplementation has been found to decrease the amount of carotenes converted in the animal.

Performance

Very little research on vitamin A supplementation of the performance horse has been done. Abrams (1979) supplemented four Thoroughbred race horses in training over a two year period with 50,000 IU/d of Vitamin A to a diet of hay, oats and bran. The supplemented horses were sound for 64% more races than the other four horses on the study not receiving the Vitamin A. In addition, by the second year of training, all horses not receiving the vitamin A were plagued with lameness, particularly with tendon problems. The horses receiving the vitamin A were all sound and fit for racing. Butler and Blackmore (1982) sampled 71 two and three year old Thoroughbreds in race training in England monthly for 6 months. Mean plasma level were 16.5 ug/dl, which were low/normal values. Since these are horses in intense training, the low plasma levels may be indicative of an increased utilization of vitamin A with exercise. Further work is needed to define vitamin A requirements for exercise in the horse.

TOXICITY

Vitamin A

Oversupplementation of vitamin A appears to be more of a problem than under-supplementation because vitamin A can be very toxic in excessive amounts (Donoghue *et al*., 1981). Symptoms of mild toxicity (100 times the NRC recommended levels) are difficult to define in the attitude of the horse, but the obvious signs are the same as mildly deficient horses: slowed growth, dull hair coat and poor muscle tone. Severe toxicity (up to 1000 times the NRC recommended level) results in depression, alopecia, ataxia, severe bone deformation and eventually death. Blood values of intoxicated horses may increase to up to 6 times the normal range (normal = 17-50 g/dl). In addition, low red blood cell count and low levels of cholesterol and albumin may be indicative of vitamin A intoxication (Donoghue *et al*., 1981). The NRC has proposed an upper safe limit of 16,000 IU of vitamin A per kg of dry diet per day for chronic administration (NRC, 1989).

Carotenes

Carotenes have not been found to be toxic at any level of intake because the body appears to down-regulate the activity of the enzyme responsible for converting the carotene into vitamin A (McDowell, 1989). The only symptom of excessive carotene

intake may be a yellowing of the skin due to an increase in storage of carotenes in adipose tissue, but this does not appear to have any effect on the overall health of the animal.

Vitamin D

FUNCTIONS

Vitamin D can be considered a vitamin only in the sense that, under modern farming conditions, many animals are raised in total confinement with little or no exposure to natural sunlight. Vitamin D is actually a hormone and adequate sunlight results in the production of sufficient vitamin D from 7-dehydrocholesterol in the skin. Hence vitamin D is not required in the diet if sufficient amounts of sunlight are received. Sufficient vitamin D must be present for calcium and phosphorus to be absorbed; a vitamin D deficiency markedly reduces absorption of both minerals. Lack of adequate photoproduction of vitamin D_3 or inadequate dietary supplementation of vitamin D leads to the failure of bones to calcify normally. This metabolic disease is known as rickets in the young and osteomalacia in adults.

FORMS OF THE VITAMIN

Plant and animal

The two major natural sources of vitamin D are cholecalciferol (vitamin D_3, which occurs in animals) or ergocalciferol (D_2, which occurs predominantly in plants). Vitamin D_2 and D_3 are not equally utilized by the horse; it appears that D_3 may be many times more potent a source than D_2 (Harrington and Page, 1983).

Activation

Vitamin D_3 produced in the skin goes to the liver. Dietary vitamins D_2 and D_3 are absorbed in the distal small intestine. The absorbed vitamin D is transported via the lymph-blood system to the liver where it is hydroxylated to 25-hydroxycholecalciferol. This form is then secreted back into the blood stream where it is further hydroxylated in the kidney to either 1,25-dihydroxycholecalciferol or 24,25-dihydroxycholecalciferol. The conversions of these vitamin D metabolites are under hormonal control. Low blood calcium stimulates the secretion of parathyroid hormone (PTH), which in turn stimulates the of release of these metabolites. Vitamin D metabolites have a three-fold effect: an increase in the absorption of calcium, phosphorus, and magnesium in the intestines, an increase in the release of calcium and phosphorus from the bone, and an increase in the resorption of calcium in the kidney.

DIETARY SOURCES

Intracorporal

Vitamin D_3 (cholecalciferol) results primarily from ultraviolet irradiation of 7-dehydrocholesterol synthesized by the tissues of the horse and present in the skin. The vitamin is then transported to the liver for utilization.

Sun cured feeds

Vitamin D_2 (ergocalciferol) results from ultraviolet irradiation of ergosterol, which is synthesized by plants. This process does not occur until the plant has been cut and is exposed to sunlight. Leaves have a higher concentration of D_2 than stems.

Other sources

Foods naturally rich in vitamin D_3 include fish oils and egg yolks; in vitamin D_2, sun cured forages. Many commercial horse feeds include vitamin D in the formulation in case the animals are not receiving enough vitamin D from other sources.

REQUIREMENT

Modern horsemanship practices may predispose the horse to suboptimal intakes of vitamin D. Restricting the amount of turnout for horses to less than 2 hours a day prevents their bodies from having time to convert sufficient vitamin D in the skin. In addition, during winter, conversion is less efficient because of the lower intensity of light and is further compounded by the use of blankets covering much of their bodies. During summer, horses tend to be turned out at night when it is cool and kept inside when the sun is shining. Furthermore, hays that have been dried artificially with little exposure to sunlight will not contain much vitamin D.

Maintenance

No absolute requirement has yet been established for the horse. However, supplementation with 800 to 1000 IU of Vitamin D per kg of dry diet has been found to be adequate to prevent deficiency symptoms in growing horses. For the adult, 500 IU of vitamin D per kg of dry diet may be sufficient (NRC, 1989). Supplementation at this level with no exposure to sunlight does not produce bones as strong as those in a horse that is maintained outside (Shorafa *et al*., 1979). Optimal supplementation for horses with limited access to sunlight is not yet known.

Performance

Extensive bone remodeling was found in young horses undergoing race training resulting in changes in calcium, phosphorus and vitamin D levels in serum and bone (Nielsen *et al*., 1995). Logically, anything that stresses the bone structure of the animal (such as carrying the weight of the rider, jumping or intensive training) will increase the amount of bone remodeling, and therefore, may increase the need for vitamin D. An increase in vitamin D intake has been found to increase efficiency of calcium and phosphorus absorption in the intestinal tract (Cromwell, 1996) which would increase the supply of these nutrients as needed in times of stress due to exercise. Much more work needs to be done on vitamin D supplementation in the exercising horse.

TOXICITY

Outward signs of vitamin D toxicosis are depression , decreased appetite with weight loss, and limb stiffness. When young ponies were fed 14,000 IU vitamin D per kg body weight daily, acute toxicity and severe calcification of the lungs, heart, kidney and other organs occurred within 10 days. Chronic toxicity (calcification of kidneys, rarefication of bone, severe weight loss and death after 3 to 4 months) occurred when 3500 IU vitamin D per kg of body weight per day was fed (Hintz *et al*., 1973).

The tolerable dose varies with the intake of calcium and possibly other nutrients which influence calcium metabolism, such as phosphorus, magnesium, protein and vitamin A. The NRC suggests that a level of 50 times the requirements may be harmful to horses.

Vitamin E

FUNCTIONS

Vitamin E has numerous functions in the body, many of which are still not completely understood. Vitamin E is essential for the integrity and optimum function of reproductive, muscular, circulatory, nervous and immune systems. Its action as a natural antioxidant is seen as the underlying factor of most vitamin E functions. Vitamin E is considered to be the most effective natural lipid-soluble chain-breaking antioxidant. The action of vitamin E is very important in cell membranes, protecting them from peroxidative damage. This is especially important in the mitochondrial membrane, where enzymes involved in respiratory chain energy production are located. Selenium , like vitamin E, also acts to prevent lipid peroxidation, however with differing mechanisms. The two complement each other, one appearing to be able to compensate

for the absence of the other. Signs of deficiency, such as nutritional muscular dystrophy, are often the same as those seen with selenium deficiency. Other diseases associated with low serum vitamin E include degenerative myelopathy and degenerative myeloencephalopathy.

FORMS OF THE VITAMIN

Natural

There are at least eight forms of vitamin E found widely distributed in nature: four tocopherols (α, ß, δ, and γ) and four tocotrienols (α, ß, δ, and γ). The majority of vitamin E activity in animal tissues is generally assumed to be α-tocopherol and when it is present it is used preferentially (Ullrey, 1981). Tocopherols are extremely resistant to heat but readily oxidized. Natural vitamin E is subject to destruction by oxidation, accelerated by heat, moisture, rancid fat, and certain trace minerals (McDowell, 1989). Horses have the capability to store much less vitamin E than vitamin A; nevertheless, stores are thought to be able to compensate for about 4 months of inadequate vitamin E intake. An increase in the requirement for vitamin E that usually occurs with increasing intakes of oils high in polyunsaturated fatty acids has not been observed in the horse (Siciliano and Wood, 1993).

Synthetic

Since esterification stabilizes vitamin E, commercial supplements usually contain tocopheryl acetates.

DIETARY SOURCES

Found in plants

Vitamin E is abundant in green growing pastures (45 to >400 IU/kg), particularly in alfalfa (McDowell, 1989). The content diminishes with maturation, especially after going to seed. Harvesting the forage diminishes the quantity of vitamin E present (10-200 IU/kg). Storage of the hay further decreases the amount of vitamin A as much as 50% in the first month of storage. Vitamin E is abundant in the germ of grains and oils pressed from the germ (wheat germ oil, 1330 IU/kg). Vegetable oils such as corn and soybean oil are relatively high in vitamin E (50-300 IU/kg). In practice, the vitamin E content of other feedstuffs is variable and not readily predictable because of handling and storage time. Therefore, it is common practice to supplement animal feeds with vitamin E.

Added to feeds

No differences were found in the utilization of natural versus synthetic forms of vitamin E added into the feeds of exercising horses (Gansen *et al*., 1995). However, the natural vitamin E was fed at one-third the amount of the synthetic vitamin E. This would suggest that the natural form is more available than the synthetic form.

REQUIREMENT

Maintenance

Significant confusion of actual vitamin E requirement exists because of the difficulty in determining the amount needed to prevent deficiency symptoms and the amount needed to produce optimal immune function. The NRC (1989) recommends 50 IU of vitamin E per kg of dietary dry matter in the total diet for the maintenance horse, and 80 to 100 IU in foals, pregnant and lactating mares, and working horses.

Performance

Vitamin E appears to be the most researched vitamin at this time in production animals, and in particular, its effects on improving performance. Since the main function of vitamin E is to protect the cell against peroxidative damage, lipid peroxidation as a result of exercise may be influenced by the concentration of vitamin E present in the diet. Several studies have reported evidence of oxidative stress occurring with exercise in both humans and rodents (Siciliano and Lawrence, 1996). Serum vitamin E decreased in horses undergoing exercise conditioning and fed a diet low in vitamin E (< 18 IU/kg dry matter) over a period of four months. However, no improvement in performance of a standardized exercise test following vitamin E supplementation for one month was reported (Petersson *et al*., 1991). Total diet concentrations of 44 IU per kg dry matter increased blood levels of vitamin E but did not increase performance (McNeniman and Hintz, 1992). Because of the nature of vitamin E, benefits of supplementation during exercise would more likely be seen in long duration exercise, such as endurance riding. Unfortunately, little if any work has been done with horses in this field.

TOXICITY

Signs of vitamin E toxicity in the horse have not been produced. However, in other animals, very high intakes have been found to interfere with utilization of other fat-soluble vitamins. The NRC (1989), therefore, takes a conservative upper limit for

vitamin E supplementation of 100 IU/kg of dry diet or 20 IU/kg of body weight per day.

Vitamin K

FUNCTIONS

The major function of vitamin K is in blood coagulation. In fact, the Danish discoverer of the vitamin named it after its function "koagulation" (Danish spelling). The vitamin is required for the activation of the four plasma clotting factors. Recently, vitamin K has also been found to have a role in the activation of a number of other proteins throughout the body, some specifically identified in the skin and bone.

FORMS OF THE VITAMIN

Microbial form

Vitamin K_2 (menaquinone) is the compound synthesized by intestinal microbes, being absorbed from the small intestine by a passive, noncarrier-mediated process.

Plant form

Vitamin K_1 (phylloquinone) is the compound found in green plants. It is absorbed in the proximal small intestine by an energy-requiring process. Phylloquinone also appears to be the form that is stored for a limited amount of time (< 24 hours) in the liver.

Synthetic

Vitamin K_3 (menadione) is a synthetic product and appears to be absorbed from both the colon and small intestine by passive processes.

DIETARY SOURCES

Natural

It is generally assumed that vitamin K is synthesized by microorganisms of the cecum and colon in sufficient quantities to meet requirements. Coprophagy (eating feces) will reintroduce highly available synthesized vitamin K to the intestines. This practice has made defining absolute requirements difficult to quantify.

Green leaves are the richest natural source of vitamin K, and the vitamin remains

present even after the green has diminished. The nature of the diet of the horse, pasture and/or hay, should provide more than adequate quantities of vitamin K.

Added to feeds

Phylloquinone is the safest form for supplementation of vitamin K in the animal but also the most expensive. The water soluble forms of menadione are less expensive and are used commonly in animal feeds.

REQUIREMENT

Maintenance

Dietary requirements of vitamin K have not been determined for the horse (NRC, 1989). Phylloquinone in pasture or in good quality hay and menaquinones synthesized by intestinal bacteria presumably meet those requirements in all but the most unusual of circumstances. High calcium diets fed to pigs have been found to greatly increase the vitamin K requirement (Hall *et al*., 1991). In horses, high calcium diets are usually a result of high alfalfa intakes, and alfalfa is an excellent source of vitamin K. Conditions which interfere with vitamin K function are impaired fat absorption, gastric ulcers, mycotoxins in the feed, long term antibiotic treatment, dicumarol in the feed (found in spoiled sweet clover hay) and warfarin (rat poison).

Performance

No research has been done on the requirement for vitamin K during exercise. Clinical responses of some "bleeders" to vitamin K therapy suggest that some performance horses may require additional supplies of vitamin K (Wakeman *et al*., 1975).

TOXICITY

No problems have been found with excessive intakes of phylloquinone. However, menaquinones administered orally have been found to be toxic at 1000 times the dietary requirement. Phylloquinone injectables appear safer than menadione injectables, as parenteral administration of menadione bisulfite has been found to cause acute renal failure in horses (Rebhun *et al*., 1984).

Summary

While research has shown the importance of the fat-soluble vitamins for normal body function, absolute requirements in the horse are less well defined. Factors which complicate quantifying requirements of fat-soluble vitamins are body stores and intracorporal production of the vitamins. More extensive research is needed on the vitamin requirements of the performance horse. Modern equine management practices may not always allow the horse to obtain sufficient quantities of the vitamins from the sources that nature intended. Addition of vitamins to the diet of the horse supplies a sense of security that the animal is receiving at least the minimum that is necessary.

References

Abrams, J.T. 1979. The effect of dietary vitamin A supplements on the clinical condition and track performance of racehorses. Biblthca Nutr. Dieta. 27:113.

Cromwell, G.L. 1996. Metabolism and role of phosphorus, calcium and vitamin D3 in swine nutrition. In: M.B. Coelho and E.T. Kornegay (Ed.) Phytase in animal Nutrition and Waste Management. BASF Reference Manual p.101.

Donoghue, S., and D.S. Kronfeld. 1980. Vitamin-mineral supplements for horses with emphasis on vitamin A. Compendium Cont. Edu. 8:S121.

Donoghue, S., D.S. Kronfeld, S.J. Berkowitz, and R.L. Copp. 1981. Vitamin A nutrition of the equine: growth, serum biochemistry and hematology. J. Nutr. 111:365.

Fonnesbeck, P.V. and L.D. Symons. Utilization of the carotene of hay by horses. J. Anim. Sci. 26:1030.

Gansen, S., A. Lindner, and A. Wagener. 1995. Influence of a supplementation with natural and synthetic vitamin E on serum alpha tocopherol content and V_4 of Thoroughbred horses. In: Proc. 14th Equine Nutr. And Physiol. Soc. Symp. p. 68. Ontario, CA.

Hall, D.D., G.L. Cromwell, and T.S. Stahly. 1991. Effects of dietary calcium, phosphorus, calcium: Phosphorus ration and vitamin K on performance, bone strength and blood clotting status of pigs. J. Anim. Sci. 69:646.

Harrington, D.D. and E.H. Page. 1983. Acute vitamin D_3 toxicosis in horses: case reports and experimental studies of the comparative toxicity of vitamins D_2 and D_3. JAVMA 182:1358.

Hintz, H.F., H.F. Schryver, and J.D. Lowe. 1973. Effect of vitamin D on calcium and phosphorus metabolism in ponies. J. Anim. Sci. 37:282.

McDowell, L.R. 1989. Vitamins in Animal Nutrition: Comparative Aspects to Human Nutrition. Academic Press, Inc. San Diego, CA.

McNeniman, N.P., and H.F. Hintz. 1992. Effect of vitamin E status on lipid peroxidation in exercised horses. Equine Vet. J. 24(6):482.

Neilsen, B.D., G.D. Potter, L.W. Greene, E.L. Morris, M. Murray-Gerzik, W.B. Smith, and M.T. Martin. 1995. Does the onset of training alter mineral requirements in the young racing Quarter Horses? In: Proc. 14th Equine Nutr. And Physiol. Soc. Symp. p. 70. Ontario, CA.

NRC. 1973. Effect of Processing on the Nutritional Value of Feeds. National Academy of Sciences, Washington, D.C.

NRC. 1987. Vitamin Tolerance of Animals. National Academy Press, Washington D.C.

NRC. 1989. Nutrient requirements of horses. (5th Ed.) National Academy Press, Washington D.C.

Petersson, K.H., H.F. Hintz, H.F. Schryver, and G.F. Combs, Jr. 1991. The effect of vitamin E on membrane integrity during submaximal exercise. In: G.B. Persson, A. Linkholm, and L.B. Jeffcott (Ed.) Equine Exercise Physiology 3. ICEEP Publications, Davis, California, p. 315.

Rebhun, W.C., B.C. Tennant, S.G. Dill, and J.M. King. 1984. Vitamin K_3-induced renal toxicosis in the horse. J. Am. Vet. Med. Assoc. 184:1237.

Shorafa, W.M., J.P. Feaster, E.A. Ott and R.L Asquith. 1979. Effect of vitamin D and sunlight on growth and bone development of young ponies. J. Anim. Sci. 48:882.

Siciliano, P.D., and C.H. Wood. 1993. The effect of added dietary soybean oil on vitamin E status of the horse. J. Anim. Sci. 71:3399.

Siciliano, P.D. and L.M. Lawrence. 1996. The role of vitamin E nutrition in the exercised horse. In: . M.B. Coelho (Ed.) Vitamin E in Animal Nutrition and Management. BASF Reference Manual p. 461.

Ullrey, D.E. 1981. Vitamin E for swine. J. Anim. Sci. 53:1039.

Wakeman, D.L., E.A. Ott, B.H. Crawford, and T.J. Cunha. 1975. Horse Production in Florida. Bulletin No. 188, Florida Dept. of Agriculture and Consumer Services, p.75, Gainesville, FL.


Kentucky
Equine
Research®