

VITAMIN NUTRITION OF LIVESTOCK ANIMALS: OVERVIEW FROM VITAMIN DISCOVERY TO TODAY

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The term “vitamin” or “vitamine” was first used in 1912. What later became known as vitamin deficiency diseases—scurvy, beriberi, night blindness and xerophthalmia—had plagued the world from antiquity. From 1900 through the 1930s, experiments with animals helped to advance knowledge of vitamins considerably. There are 15 vitamins of significance for livestock. A number of factors influence vitamin requirements and vitamin utilization, including physiological make-up and production function; confinement rearing without pasture; stress, disease and adverse environmental conditions; vitamin antagonists; use of antimicrobial drugs; and body vitamin reserves. Under commercial livestock and poultry production conditions, vitamin allowances higher than National Research Council (USA) requirements may be needed for optimum performance. Generally, the optimum vitamin supplementation level is the quantity that achieves the best growth rate, feed utilization and health (including immune competency), while also providing adequate body reserves.

Le mot “vitamine” a été utilisé pour la première fois en 1912. Ce qui a finalement été reconnu comme des maladies dues à une carence vitaminique – scorbut, bérubéri, héméralopie (cécité nocturne), xérophtalmie – ont affligé l'humanité depuis l'antiquité. De 1900 aux années 30, l'expérimentation animale a permis d'augmenter considérablement les connaissances sur les vitamines. Il y a 15 vitamines importantes pour la nutrition des animaux d'élevage. Plusieurs facteurs influencent les besoins en vitamines et leur utilisation, incluant le stade physiologique et le type de production; l'élevage en claustration sans pâturage; le stress, les maladies et les conditions environnementales difficiles; les antagonistes des vitamines; l'emploi de substances antimicrobiennes et les réserves corporelles en vitamines. Sous les conditions d'élevage commercial du bétail et de la volaille, des quantités de vitamines plus grandes que celles recommandées par le National Research Council (USA) peuvent être nécessaires pour optimiser les performances zootechniques. Généralement, la supplémentation optimum en vitamines est la quantité qui permet d'obtenir les meilleurs taux de croissance et de conversion alimentaire et un bon état de santé (incluant la compétence immunitaire) tout en maintenant des réserves corporelles adéquates.

Vitamins, a group of complex organic compounds that are present in minute amounts in natural foodstuffs, are essential for normal metabolism, and a lack of these compounds in the diet causes deficiency diseases. The term “vitamin” or “vitamine” was coined less than 100 years ago, in 1912, by Polish biochemist Casimir Funk. To date, 15 vitamins (Table 1) have been identified and classified as either fat- or water-soluble. The number of compounds that can justifiably be classified as vitamins is controversial. The term “vitamin” has

been applied to many substances that do not meet the criteria used to define vitamins (McDowell 2004).

Some vitamins deviate from the above definition in that they do not always need to be constituents of food. Certain substances considered to be vitamins are synthesized by intestinal tract bacteria in quantities that are often adequate for bodily needs. However, a clear distinction is made between vitamins and substances that are synthesized in body tissues. Of the 15 vitamins listed, choline is only tentati-

Table 1. Fat- and water-soluble vitamins with synonym names

Vitamins	Synonyms
Fat soluble	
Vitamin A ₁	Retinol, retinal, retinoic acid
Vitamin A ₂	Dehydroretinol
Vitamin D ₂	Ergocalciferol
Vitamin D ₃	Cholecalciferol
Vitamin E	Tocopherol, tocotrienols
Vitamin K ₁	Phylloquinone
Vitamin K ₂	Menaquinone
Vitamin K ₃ ^z	Menadione
Water soluble	
Thiamin	Vitamin B ₁
Riboflavin	Vitamin B ₂
Niacin	Vitamin PP, Vitamin B ₃
Vitamin B ₆	Pyridoxol, pyridoxal, pyridoxamine
Pantothenic acid	Vitamin B ₅
Biotin	Vitamin H, vitamin B ₈
Folic acid	Folacin, folate, Vitamin M, Vitamin Bc, Vitamin B ₉
Vitamin B ₁₂	Cobalamin
Choline	Gossypine
Vitamin C	Ascorbic acid
Carnitine	

^zThe synthetic form is water-soluble

vely classified as one of the B-complex vitamins. Choline differs from the other B vitamins in that it can be synthesized in the body, it is required in larger amounts and it apparently functions as a structural constituent rather than as a coenzyme. Similarly, carnitine was tentatively classified as a vitamin, but this applies to only a few species under special circumstances.

Vitamin C can be synthesized by most animal species, except when they are young or under stress conditions. Likewise, in most species, niacin can be synthesized from the amino acid tryptophan and vitamin D from the action of ultraviolet light on precursor compounds in the skin. Thus, under certain conditions and for specific

species, vitamin C, niacin and vitamin D do not always fit the classic definition of a vitamin. Vitamin D nonetheless fits the vitamin definition for cats and dogs, which lack skin receptors for this vitamin, as well as for poultry and swine that are raised under management systems that preclude exposure to ultraviolet light (McDowell 2000).

The major functions of the B-vitamins have been known for many years. Now we have a clearer picture of the metabolism and functions of vitamins A, D, E, K and C. For example, vitamins A and D with hormone functions are both considered to play as equally important roles as do thyroid hormones. It has been determined that the functions of vitamins K and D are

not limited to blood clotting and bone formation, respectively. Vitamin C, which is synthesized by most species, may nonetheless be beneficial when provided to animals under stress. Animals with a functional rumen were once thought to synthesize B-vitamins and vitamin K and therefore to have no need for supplementation. However, deficiencies do occur under special conditions and supplementation has proven beneficial in the case of thiamin, niacin, vitamin B₁₂, choline, biotin and vitamin K. Similarly, supplemental carnitine has been shown to be beneficial for several species.

Classical deficiency symptoms and non-specific parameters (e.g. lower production and reproduction rates) are associated with vitamin deficiencies or excesses. Vitamin nutrition should no longer be considered important solely for preventing deficiency signs, as vitamins can play a role in optimizing animal health (e.g. immune function), productivity and product quality. Given the huge scope of this topic, this review will focus on vitamin requirements, vitamin occurrence and the history of vitamins. The concept of optimum vitamin allowances and the supplemental vitamins most needed by livestock will also be covered.

EARLY HISTORY OF VITAMINS

The history of the discovery of the vitamins is an inspirational and exciting account of the ingenuity, dedication and self-sacrifice of many individuals. Excellent reviews of this history and accompanying references can be found in Funk (1922), Harris (1955), McCollum (1957) and Wagner and Folkers (1962). This history section draws on those reviews, sometimes without specific citations, and readers are encouraged to refer to the reviews. The development of the concept of vitamins can be roughly divided into four, broadly overlapping periods: 1) Empirical healing of some diseases through

the administration of certain foods; 2) Development of analytical capabilities to identify classes of nutrients in foods; 3) Experimental induction of dietary diseases in animals; and 4) Administration of synthetic diets to discover essential nutritional factors.

Most of the history of vitamins is linked to efforts to find cures for human diseases such as night blindness, xerophthalmia, scurvy, beriberi, rickets and pellagra. Experiments with animals including rats, mice, chickens, pigeons, guinea pigs and dogs contributed greatly to the advances made in vitamin research between 1900 and the 1930s.

Early observations leading to the vitamin theory

The first phase of developing the concept of vitamins began many centuries ago, and gradually led to the recognition that night blindness, xerophthalmia, scurvy, beriberi and rickets are dietary diseases. These diseases had long plagued humankind and they are mentioned in the earliest written records. Records of medical science from antiquity attest to the fact that researchers had already made a link between certain foods and diseases or infirmities, postulating that food constituents played either a causal or a preventive role. These are considered the nebulous beginnings of the concept of essential nutrients (Wagner and Folkers 1962).

Beriberi is probably the earliest documented deficiency disorder, being recognized in China as early as 2697 B.C. By 1500 B.C, scurvy, night blindness and xerophthalmia were described in Egypt. Two books of the Bible contain accounts that point to vitamin A deficiency (McDowell 2000). Jeremiah 14:6 states: “and the asses did stand in high places, their eyes did fail because there was no grass.” In addition, the Bible mentions that fish bile was used to cure a blind man named Tobias.

In 400 B.C., the Greek physician Hippocrates, known as the Father of

Medicine, reported using raw ox liver dipped in honey to prevent night blindness. He also described soldiers afflicted with scurvy. Scurvy took a heavy toll on the Crusades because the soldiers travelled far from home and their diet was deficient in vitamin C.

During the long sea voyages that took place between 1492 and 1600, scurvy posed a serious threat to the health of sailors and undermined world exploration. For example, Magellan lost 80% of his crew to the disease while sailing around the world. Vasco de Gama, another great explorer, lost 60% of his 160-man crew while mapping the coast of Africa. In 1536, during Jacques Cartier's expedition to Canada, 107 out of 110 men became sick with scurvy. However, the expedition was saved when the Indians shared their knowledge of the curative value of pine needles and bark. In 1593, British Admiral Richard Hawkins wrote: "I have seen some 10,000 seamen die of scurvy, some sailors tried treating themselves by trimming the rotting, putrid black flesh from their gums and washing their teeth in urine."

In 1747, James Lind, a British naval sergeant, carried out the first controlled clinical experiment aboard a ship aimed at finding a cure for scurvy (Lind 1757). Twelve patients with scurvy were divided into six treatment groups. Two sailors received a dietary supplement of oranges and lemons while the other treatment groups were given nutmeg, garlic, vinegar, cider and sea water, respectively. The two men who had received the citrus fruit were cured of scurvy. Where did Lind get the idea that scurvy was related to nutrition? He had been told a story of an English sailor with scurvy who was left to die on a desolate island with no food. Feeling hungry, the man nibbled a few blades of beach grass. The next day, he felt stronger and ate some more grass. After a few weeks on this "diet," he was completely well.

Prior to the beginning of the 20th century, there was a growing body of evidence that nutritional factors, which later became known as vitamins, were implicated in certain diseases. Louis Pasteur was the chief opponent of the "vitamin theory," which held that certain diseases resulted from a shortage of specific nutrients in foods. Pasteur believed there were only three classes of organic nutrients: carbohydrates, fats and proteins. His research showing that microorganisms caused disease made scientists with medical training reluctant to believe the vitamin theory. It has been said that the immensely successful "germ theory" of disease, coupled with toxin theory and the successful use of antisepsis and vaccination, convinced scientists of the day that only a positive agent could cause disease (Guggenheim 1995). Right up to the mid-1930s, the majority of U.S. doctors still believed that pellagra was an infectious disease (McDowell 2000).

In the second half of the 19th century, a different disease from scurvy was killing sailors in the Japanese navy. In 1880, the Japanese navy numbered 4,956; within a year, 1,725 men had died of beriberi. In three years, almost 5,000 Japanese sailors died of beriberi. Patients with beriberi became weak and eventually partially paralysed and they lost weight and died. Doctors tried to find the germ that was causing beriberi. Finally, they listened to Japanese naval sergeant Kamekiro Takaki, who believed that the sailors' diet was causing beriberi. Takaki noted a 60% incidence of beriberi on a ship returning from a one-year voyage during which the sailors' diet had been mostly polished rice and some fish. He sent out a second ship under the same conditions, but this time substituted barley, meat, milk and fresh vegetables for some of the rice. The dietary change eliminated beriberi but Takaki incorrectly concluded that the beriberi was

prevented by the additional protein. Regardless, the Japanese now knew that they could prevent beriberi by not relying on polished rice as a dietary staple.

The vitamin theory takes shape

Beginning in the mid-1850s, German scientists were recognized as leaders in the field of nutrition. In the late 1800s, Professor C. von Bunge, who worked at the German university in Dorpat, Estonia and then at Basel, had some graduate students conduct experiments with purified diets for small animals (Wolf and Carpenter, 1997). In 1881, N. Lunin, a Russian student studying in von Bunge's laboratory, observed that some mice died after 16 to 36 days when fed a diet composed solely of purified fat, protein, carbohydrate, salts and water. Lunin suggested that natural foods such as milk contain small quantities of "unknown substances essential to life."

In 1896, Dutch physician and bacteriologist Christian Eijkman made a historic finding in relation to a cure for beriberi. Eijkman was conducting research in Indonesia in an effort to identify the causal pathogen of beriberi. He astutely observed that a polyneuritis condition in chickens produced symptoms similar to those seen in humans with beriberi. This chance discovery was made when a new head cook at the hospital discontinued the supply of "military" rice (polished rice), and the chickens were fed whole-grain "civilian" rice and recovered from the polyneuritis. Many great advances in science have come about as a result of chance observations like this being made by men and women of inspiration. After extensive experimentation, he proved that both polyneuritis and beriberi were caused by eating polished white rice and that both of these afflictions could be prevented or cured when the outer portions of the rice grain (e.g. rice bran) were eaten. Thus, Eijkman became the first researcher to produce a vitamin deficiency disease in an experimental animal (Eijkman

1890-1897). He also noted that prisoners with beriberi who had been eating polished rice tended to get well when they were fed a less milled product.

In 1901, Grijns, one of Eijkman's colleagues in Indonesia, was the first to come up with a correct interpretation of the connection between the excessive consumption of polished rice and the etiology of beriberi. He concluded that rice contained "an essential nutrient" found in the outer layers of the grain.

In 1902, a Norwegian scientist named Holst conducted some experiments on "ship-beriberi" (actually scurvy) using poultry but the experiments failed and then in 1907 Holst and Frolich produced experimental scurvy in guinea pigs. Later it was learned that poultry can synthesize vitamin C, while guinea pigs cannot.

In 1906, Frederick Hopkins, who was working with rats in England, reported that "no animal can live upon a mixture of pure protein, fat and carbohydrate and even when the necessary inorganic material is supplied, the animal cannot flourish." Hopkins found that small amounts of milk added to purified diets allowed rats to live and thrive. He suggested that unknown nutrients were essential for animal life, calling them "accessory food factors." Hopkins' experiments were similar to those of Lunin; however, they were more in-depth, and he played an important role by recording his views in such memorable terms that they received wide recognition (McCollum 1957). Hopkins also expressed the belief that various disorders were caused by diets deficient in unidentified nutrients (e.g. scurvy and rickets). He was responsible for opening up a new field of discovery that largely depended on the use of experimental rats.

In 1912, Casimir Funk, a Polish biochemist working at the Lister Institute in London, proposed the "vitamin theory" (Funk 1922). He had reviewed the literature and made the

important conclusion that beriberi could be prevented or cured by a protective factor present in natural food, which he successfully isolated from rice by-products. What he had isolated was given the name “beriberi vitamine” in 1912. This term “vitamine” denoted that the substance was vital to life and it was chemically an amine (vital + amine). In 1912 Funk proposed the theory that other “deficiency diseases” in addition to beriberi were caused by a lack of these vital substances, namely scurvy, rickets, sprue and pellagra. He was apparently the first to suggest that pellagra was a nutritive deficiency disease.

In 1907, Elmer McCollum arrived in Wisconsin to work on a project to determine why cows fed wheat or oats (versus yellow corn) gave birth to blind or dead calves. The answer was found to be that wheat and oats lacked the vitamin A precursor carotene. In 1913-1915, McCollum and Davis discovered two growth factors for rats, “fat-soluble A” and “water-soluble B.” By 1922, McCollum had identified vitamin D as a substance independent of vitamin A. He bubbled oxygen through cod liver oil to destroy its vitamin A; the treated oil remained effective against rickets but not against xerophthalmia. Thus, “fat-soluble vitamin A” had to be two vitamins, not just one.

In 1923, Evans and Bishop discovered that vitamin E deficiency caused reproductive failure in rats. Steenbock (1926) showed that irradiation of foods as well as animals with ultraviolet light produced vitamin D. In 1928, Szent-Györgyi isolated hexuronic acid (later renamed “ascorbic acid”) from foods such as orange juice. One year later, Moore proved that the animal body converts carotene to vitamin A, in an experiment that involved feeding one group of rats carotene and finding high levels of vitamin A in their livers compared to controls. By 1928, Joseph Goldberger and Conrad Elvehjem had shown that vitamin B was more than one

substance. After the “vitamin” was heated, it was no longer effective in preventing beriberi (B₁) but it was still good for rat growth (B₂). The 1930s and 1940s were the golden age of vitamin research. During this period, the customary approach was to: 1) study the effects of a deficient diet; 2) find a food source that prevents the deficiency; and 3) gradually concentrate the particular nutrient (vitamin) in a food and test potency. Laboratory animals were used in these procedures.

Henrick Dam of Denmark discovered vitamin K in 1929 when he noted hemorrhages in chicks fed a fat-free diet. Ironically one year earlier, Herman Almquist, working in the United States, had discovered both forms of the vitamin (K₁ and K₂) in studies with chicks. Unfortunately university administrators delayed the review of his paper, and when it was finally submitted to the journal *Science*, it was rejected. Therefore only Henrick Dam received a Nobel prize for the discovery of vitamin K.

Vitamin B₁₂ was the last traditional vitamin to be identified, in 1948. Shortly thereafter it was discovered that cobalt was an essential component of the vitamin. Simple monogastric animals were found to require the vitamin, whereas ruminants and other species with large microbial populations (e.g. horses) require dietary cobalt rather than vitamin B₁₂.

Compared with the situation for night blindness, xerophthalmia, beriberi, scurvy and rickets, there are no records from the ancient past of the disease of pellagra. The disease was found to be caused by niacin deficiency in humans, a problem prevalent mainly in cultures where corn (maize) was a key dietary staple (Harris 1919). Columbus took corn to Spain from America. Pellagra was not recognized until 1735, when Gaspar Casal, physician to King Philip V of Spain, identified it among peasants in northern Spain. The local people called it *mal de la*

rosa, and Casal associated the disease with poverty and spoiled corn. The popularity of corn spread eastward from Spain to southern France, Italy, Russia and Egypt, and so did pellagra. Babcock, of Columbia, South Carolina, who identified pellagra in the United States by establishing a link with the disease in Italy, studied the case records of the South Carolina State Hospital and concluded that the disease had occurred there as early as 1828. Most of the cases occurred in low-income groups, whose diet was limited to inexpensive foodstuffs. Diets characteristically associated with the disease were referred to as the three M's, specifically meal (corn), meat (backfat) and molasses.

The word pellagra means rough skin, which relates to dermatitis. Other descriptive names for the condition were *mal de sol* (illness of the sun) and "corn bread fever." In the early 1900s in the United States, particularly in the South, it was common for 20,000 deaths to occur annually from pellagra. It was estimated that for every death due to pellagra, there were at least 35 cases of the disease. Even as late as 1941—five years after the cause of pellagra was known—2,000 deaths were attributed to the disease. The clinical signs and mortality associated with pellagra can be referred to as the four D's: dermatitis (of areas exposed to the sun), diarrhea, dementia (mental problems) and death. Several mental institutions in the United States, Europe and Egypt were primarily devoted to the care of pellagra sufferers, or pellagrins.

In 1914, Goldberger, a bacteriologist with the United States Public Health Service, was assigned the task of identifying the cause of pellagra. In his studies he observed that the disease was associated with poor diet and poverty and that well-fed persons did not contract the disease (Carpenter, 1981). In orphanages, prisons and mental institutions in South Carolina, Georgia and Mississippi the therapeutic value of good diets was demonstrated. Goldberger, his

wife and 14 volunteers constituted a "filth squad" who ingested and were injected with various biological materials and/or excreta from pellagrins, thus demonstrating the non-infectious nature of pellagra. At the time, researchers and physicians did not want to believe that pellagra resulted from poor nutrition, and they sought to link it to an infection in keeping with the popular "germ theory" of diseases. An important step toward isolating the preventive factor for pellagra involved the discovery of a suitable laboratory animal for testing its potency in various concentrated preparations. It was found that a pellagra-like disease (blacktongue) could be produced in dogs. Elvehjem and his colleagues (1937) isolated nicotinamide from liver and identified it as the factor that could cure blacktongue in dogs. Reports of the dramatic therapeutic effects of niacin in human cases of pellagra quickly followed from several clinics.

In 1824, Combe first discovered fatal anemia (pernicious anemia) and suggested that it was linked to a digestive disorder. Minot and Murphy reported in 1926 that large amounts of raw liver would alleviate the symptoms of pernicious anemia. In 1948, Rickes and his colleagues in the United States and Smith in England isolated vitamin B₁₂ and identified it as the anti-pernicious anemia factor (McDowell 2000).

Much earlier, in 1929, Castle had shown that pernicious anemia resulted from the interaction between a dietary factor (extrinsic) and a mucoprotein substance produced by the stomach (intrinsic factor). Castle used an unusual, but effective, method to relieve the symptoms of pernicious anemia patients. He ate some beef and after allowing enough time for the meat to mix with gastric juices, he regurgitated the food and mixed his vomit with the patients' food. With this treatment, the patients were able to recover because they received both the extrinsic (vitamin B₁₂) and intrinsic (a mucoprotein) factors

from Castle's incompletely digested beef meal.

VITAMIN REQUIREMENTS

While metabolic needs are similar, dietary needs for vitamins differ widely among species. Some vitamins are metabolic essentials, but not dietary essentials for certain species, because they can be synthesized readily from other food or metabolic constituents.

Poultry, swine and other monogastric animals are dependent on dietary sources of vitamins to a much greater degree than are ruminants. Tradition has it that ruminants in which the rumen is fully functioning cannot suffer from a deficiency of B vitamins. It is generally assumed that ruminants can always satisfy their needs from the B vitamins that are naturally present in their feed plus those synthesized by symbiotic microorganisms. However, under specific conditions relating to stress and high productivity, ruminants have more recently been shown to have requirements, particularly for the B vitamins thiamin and niacin. Similarly, vitamin B₁₂ cannot be synthesized in the rumen if the essential building block cobalt is lacking in the diet.

The rumen does not become functional with respect to vitamin synthesis for some time after birth. For the first few days of life, the young ruminant resembles a non ruminant in that it requires dietary sources of the B vitamins. Beginning as early as eight days, and certainly by two months of age, the ruminal flora has developed to the point that it contributes significant amounts of B vitamins. Production of these vitamins at the proximal end of the gastrointestinal tract is indeed fortunate for they become available to the host as they pass down the tract through areas of efficient digestion and absorption.

Intestinal synthesis of B vitamins in non-ruminants occurs in the lower intestinal tract, an area of poor absorption. The horse,

with its high production of B vitamins in the large intestine, is apparently able to meet most of its requirements for these vitamins despite the poor absorption from this area. Intestinally synthesized vitamins are more available to animals (rabbit, rat and others) that habitually practice coprophagy and thus recycle products of the lower gut. This behavior provides significant amounts of B vitamins to the host animal.

VITAMIN OCCURENCE

Vitamins originate primarily in plant tissues and are present in animal tissue only because the animal has ingested plant material, or because it harbours microorganisms that synthesize them. Vitamin B₁₂ is unique in that it occurs in plant tissues as a result of microbial synthesis. Two of the four fat-soluble vitamins, vitamins A and D, differ from the water-soluble B vitamins in that they occur in plant tissue as a provitamin (a precursor of the vitamin), which can be converted to a vitamin in the animal body. No provitamins have been identified for any of the water-soluble vitamins. However, the amino acid tryptophan can be converted to niacin in most species. In addition, fat- and water-soluble vitamins differ in that water-soluble B vitamins are universally distributed in all living tissues, whereas fat-soluble vitamins are completely absent from some tissues.

FACTORS AFFECTING VITAMIN REQUIREMENTS AND VITAMIN UTILIZATION

Physiological make-up and production function

The vitamin needs of animals and humans depend greatly on their physiological make-up, age, health and nutritional status and function, such as producing meat, milk, eggs, hair or wool or developing a fetus (Roche 1979). For example, cows producing greater volumes of milk have higher vitamin requirements. Breeder hens have

higher vitamin requirements for optimum hatchability, since vitamin requirements for egg production are generally less than those for egg hatchability. Higher levels of vitamins A, D₃ and E are needed in breeder hen diets than in feeds for rapidly growing broilers. Selection for a faster growth rate may allow animals to reach higher weights at much younger ages with less feed consumed. Dudley-Cash (1994) concludes that since genetic potential has improved feed conversion at the rate of 0.8% yearly and most of the NRC vitamin requirement data are 20 to 40 years old, the vitamin requirements determined several decades ago may not apply to today's poultry. Selection for faster weight gains in swine and the increased number of litters per year also call for elevated vitamin requirements (Cunha 1984). Different breeds and strains of animals have been shown to vary in their vitamin requirements. Vitamin needs of new strains developed for improved production are higher. Leg problems seen in fast-growing strains of broilers can be corrected in part by higher levels of biotin, folacin, niacin and choline (Roche 1979).

Confinement rearing without access to pasture

The shift to swine and poultry operations involving complete confinement without access to pasture has had a profound effect on vitamin nutrition as well as mineral nutrition. Pasture can be depended on to provide significant quantities of most vitamins, since young, lush, green grasses or legumes are good vitamin sources. More available forms of vitamins A and E are present in pastures and green forages, which contain ample quantities of β -carotene and α -tocopherol compared with the less bioavailable forms in grains. Confinement rearing of poultry in cages and swine on slatted floors has limited animal access to feces (coprophagy), a substance rich in many vitamins. Confinement rearing requires producers to pay more attention to higher

vitamin requirements associated with this type of management system (Cunha 1984).

Stress, disease or adverse environmental conditions

Intensified production increases stress conditions and subclinical disease levels because of the higher animal densities. Stress and disease conditions in animals may increase the basic requirement for certain vitamins. Nutrient levels that are adequate for growth, feed efficiency, gestation and lactation may not be adequate for normal immunity and for maximizing the animals' resistance to disease (Cunha 1985; Nockels et al. 1996). Diseases or parasites affecting the gastrointestinal tract will reduce intestinal absorption of vitamins, both from dietary sources and those synthesized by microorganisms. If the infections cause diarrhea or vomiting, this will also decrease intestinal absorption and increase needs. Vitamin A deficiency is often seen in heavily parasitized animals that supposedly were receiving an adequate amount of the vitamin. Mycotoxins are known to cause digestive disturbances such as vomiting and diarrhea as well as internal bleeding, and they interfere with absorption of dietary vitamins A, D, E and K. In broiler chickens, moldy corn (mycotoxins) has been associated with deficiencies of vitamins D (rickets) and vitamin E (encephalomalacia) despite the fact that these vitamins were supplemented at levels regarded as satisfactory.

Vitamin antagonists and other nutrients

Vitamin antagonists (antimetabolites) interfere with the activity of various vitamins (Oldsfield 1987). The antagonist may cleave the metabolite molecule and render it inactive, which is the effect of thiaminase on thiamin; it may complex with the metabolite, with similar results, as illustrated by avidin and biotin; or its structural similarity may allow it to occupy reaction sites and block the metabolite's access to them, as is the case with dicumarol

and vitamin K. Rancid fats inactivate biotin and destroy vitamins A, D and E and possibly others. Some antimicrobial drugs will increase animals' vitamin needs by altering their intestinal microflora and inhibiting the synthesis of certain vitamins. Certain sulfonamides may increase the requirements for biotin, folacin, vitamin K and possibly other vitamins when intestinal synthesis is reduced. The presence of vitamin antagonists in animal and human diets should be considered in adjusting vitamin allowances, as most vitamins have antagonists that reduce their utilization.

The level of fat in the diet may affect the absorption of the fat-soluble vitamins A, D, E and K, as well as the requirement for vitamin E and possibly other vitamins. Fat-soluble vitamins may fail to be absorbed if the digestion of fat is impaired. Many relationships exist between vitamins and other nutrients and they affect requirements. For example, important relationships exist between vitamin E and selenium, between vitamin D and calcium and phosphorus, between choline and methionine and between niacin and tryptophan.

Body vitamin reserves

The body's stores of vitamins from previous food intake will affect the daily requirements for these nutrients. This is truer for the fat-soluble vitamins A, D and E and for vitamin B₁₂ than for the other water-soluble vitamins and vitamin K. Vitamin A may be stored by an animal in its liver and fatty tissue in sufficient quantities to meet requirements for periods of up to six months or even longer.

OPTIMUM VITAMIN ALLOWANCES

The National Research Council (NRC) and the Agriculture Research Council (ARC) requirements for given vitamins are usually close to the minimum levels required to prevent deficiency signs and for adequate health and performance, provided sufficient amounts of all other nutrients are supplied. Most nutritionists usually consider the NRC

and ARC requirements for vitamins to be close to the minimum requirements that are sufficient to prevent clinical deficiency signs and they may adjust them upward based on industry experience in situations where a higher level of vitamins is needed. Thus, under commercial production conditions, vitamin allowances higher than NRC and ARC requirements may be needed to allow optimum performance (Roche 1979). Generally, the optimum supplementation level is the quantity that achieves the best growth rate, feed utilization, and health (including immune competency), while also providing adequate body reserves (Coelho 1996).

The concept of optimum vitamin nutrition under commercial production conditions is illustrated in Fig. 1 (Roche 1979). The requirement zones are minimum vitamin quantities that are needed to prevent deficiency signs, but they may lead to suboptimum performance even though animals appear normal. The optimum allowances in Fig. 1 permit animals to achieve their full genetic potential for optimum performance. In the excess zone, vitamin levels range from levels still safe, but uneconomical, to concentrations that may produce toxic effects. Under practical feeding conditions, usually only vitamins A and D pose the risk of toxicity problems for livestock.

It should be emphasized that subacute deficiencies can exist although the actual deficiency signs are not present. Such borderline deficiencies are both the most costly and the most difficult to overcome, and they often go unnoticed and untreated, but they may cause poor and expensive gain, impaired reproduction or reduced production.

The optimum animal performance required under modern commercial conditions cannot be obtained by fortifying diets to meet only minimum vitamin requirements. The establishment of adequate

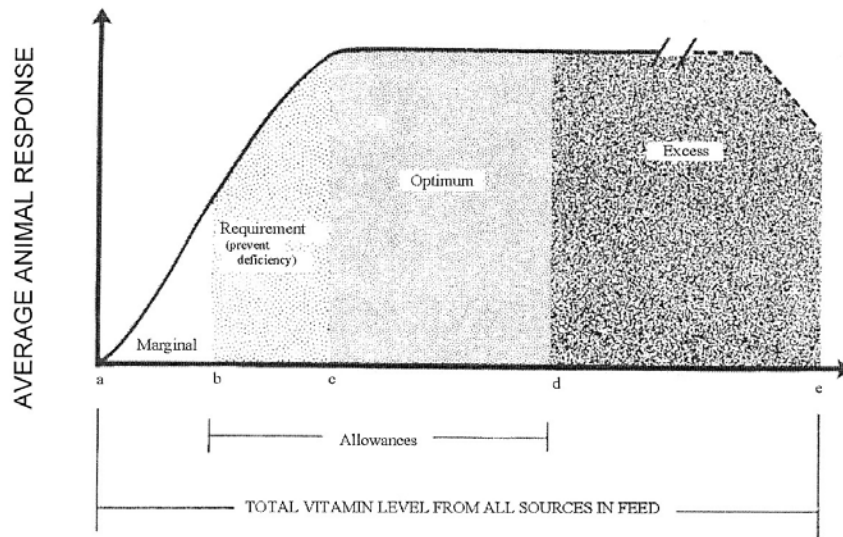


Fig. 1. Optimum vitamin nutrition for animal's under commercial production conditions.

margins of safety must provide for those factors (Fig. 2) that may increase certain dietary vitamin requirements and for variability in inactive vitamin potencies and availability within individual feed ingredients.

The NRC and ARC requirements often do not take into account that certain vitamins have special functions in relation to disease conditions and that higher than recommended levels are needed to obtain a response (Cunha 1985). In pigs artificially infected with *Treponema hyodysenteriae*, a causal agent of diarrhea, high supplementation with vitamin E (200 mg day⁻¹) in combination with selenium (0.2 mg day⁻¹) markedly reduced the number of pigs that became clinically ill (Tiege et al. 1978). In this study, clinical signs and pathological changes were less severe than in vitamin E-deficient pigs. Thus, high doses of vitamin E increase resistance against disease.

For poultry, vitamin E feeding above NRC requirements has been shown to reduce the incidence of whole bird condemnation,

septicemia/toxemia and depression in egg production brought about by heat stress (Ward 2002). In practice, feeds contaminated with mycotoxins increase the requirements for fat-soluble and other vitamins (e.g., biotin, folacin and possibly others), and therefore supplementation should be increased above NRC and ARC minimum requirements. Apart from these fat-soluble vitamins, additions of folacin will also improve performance in pigs fed moldy grain (Purser 1981) and additions of biotin will do the same for pigs given feeds containing certain molds (Cunha 1984). Vitamin supplementation above requirements has been shown to be required for optimum immune responses (Ellis and Vorhies 1976; Cunha 1985).

Vitamin Supplementation Most Needed by Livestock

Vitamin requirements, as previously noted, are highly variable among the various species and classes of animals. Supplementation allowances need to be set at levels that reflect different management systems and

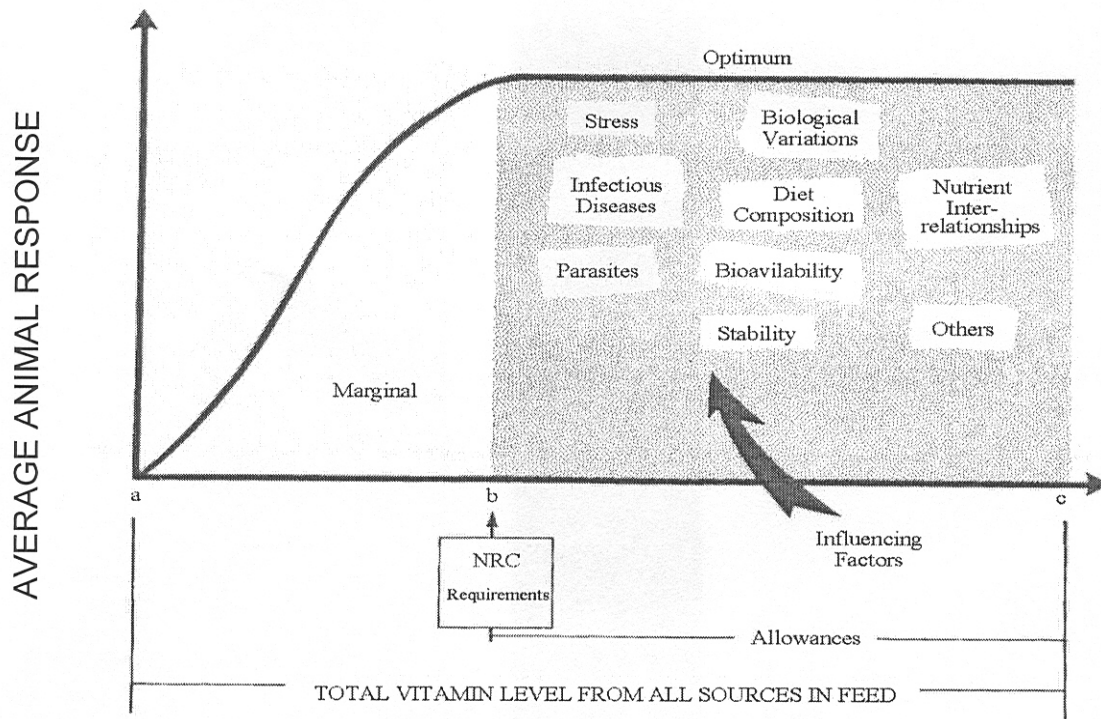


Fig. 2. Optimum vitamin nutrition for animal's under commercial production conditions and influencing factors

that are high enough to take care of fluctuations in environmental temperatures, energy content of feed, or other factors that might influence feed consumption or vitamin requirements in other ways (McGinnis 1986). This section briefly outlines the vitamins that are normally provided in ruminant, poultry, swine and horse diets (McDowell 2004).

Ruminants

Grazing ruminants generally only need supplemental vitamin A if pastures are low in carotene and possibly vitamin E (influenced by selenium status). Vitamin D is provided by ultraviolet light activity on the skin, while all other vitamins are provided by ruminal or intestinal microbial synthesis.

Ruminants housed under more strict confinement conditions generally require vitamins A and E and may require vitamin D

if deprived of sunlight. Additional supplemental vitamin E is needed to stabilize the meat color of finishing animals. Under specific conditions relating to stress and high productivity, ruminants may benefit from supplemental B vitamins, particularly thiamin and niacin. Biotin deficiency has been linked to lameness in cattle (Distl and Schmid 1994). Increased plasma biotin levels have been associated with hardness and positive conformational changes in bovine hooves as well as increased milk production (McDowell 2004). Future research may identify a need for folacin and carnitine supplementation. Adding a complete B-vitamin mixture for cattle entering the feedlot during the first month can reduce stress and increase gains. Under the stressful conditions of feedlots, the microbial population in the rumen apparently does not synthesize certain B

vitamins at adequate levels.

Poultry

Poultry under intensive production systems are particularly susceptible to vitamin deficiencies. The reasons for this susceptibility are as follows: (1) poultry derive little or no benefit from microbial synthesis of vitamins in the gastrointestinal tract; (2) poultry have high requirements for vitamins; and (3) the high density concentration of modern poultry operations places many stresses on the birds that may increase their vitamin requirements. Typical grain-oilseed meal (e.g., corn-soybean meal) poultry diets are generally supplemented with vitamins A, D (D₃), E, K, riboflavin, niacin, pantothenic acid, B₁₂ and choline (Scott et al. 1982). Thiamin, vitamin B₆, biotin and folacin are usually, but not always, present in adequate quantities in the major ingredients such as corn-soybean meal-based diets. Carnitine may be found to be of value in future studies.

Vitamins A, D, riboflavin and B₁₂ are usually present at low levels in poultry diets. However, adding other vitamins to poultry diets is good insurance. Vitamins D and B₁₂ are almost completely absent from diets based on corn and soybean meal. Vitamin K is generally added to poultry diets more than to the diets for other species because of the lower level of intestinal synthesis that occurs in birds owing to their shorter intestinal tract and the faster rate of food passage. Birds in cages require more dietary K and B vitamins than those in floor housing because of the more limited opportunity for coprophagy.

Swine

Vitamin supplementation of swine diets is obviously necessary with vitamin needs having become more critical in recent years as complete confinement feeding has increased. Swine in confinement, without access to vitamin-rich pasture, and housed on slatted floors, which limits vitamins available from feces consumption, have greater needs for supplemental vitamins.

For swine, the vitamins most likely to be marginal or deficient in corn-soybean diets are vitamins A, D, E, riboflavin, niacin, pantothenic acid and B₁₂, and occasionally also vitamin K and choline.

Almost all swine diets in the United States are now fortified with vitamins A, D, E, B₁₂, riboflavin, niacin, pantothenic acid and choline. An increasing number of feed manufacturers are also adding vitamins K, biotin, folacin and B₆ to diets. Diets are fortified with these vitamins even though not all experiments indicate a need for every one of them. Most feed manufacturers add them as a precaution to take care of stress factors, subclinical disease level and other conditions on the average farm that may increase vitamin needs (Cunha 1977). It appears that carnitine supplementation of weaning pigs may have potential.

Horses

There is a lack of experimental information on the level of vitamins required in well-balanced horse diets, as well as on which vitamins need to be added (Cunha 1980). The vitamins most likely deficient for all classes of horses are vitamins A and E, with vitamin D also being deficient for horses in confinement. Inadequate vitamin D may be provided to racehorses that are exercised only briefly in the early morning, when sunlight provides less antirachitic protection. Requirements for vitamins A, D, and E can be met with a high-quality (e.g., green color) sun-cured hay. Deficiencies of vitamin K and the B vitamins appear to be less likely in the mature horse than in other monogastric species because many vitamins are synthesized in the cecum of the horse. It is not known, however, what quantities of the vitamins synthesized in the cecum are absorbed in the large intestine. Since it is difficult to depend on intestinal synthesis, many horse owners use B-vitamin supplementation of diets for young horses and for horses being developed for racing or performance purposes (Cunha 1980).

In recent years, vitamin supplementation has become more critical for horses as the trend toward total confinement has increased. At present, many horses do not have high levels of vitamin intake from consuming a lush, green pasture or from a high-quality, leafy green hay. Typically a vitamin premix for

horses contains vitamins A, D, E, K, thiamin, riboflavin, niacin, B₆, pantothenic acid, biotin, folacin, B₁₂ and choline. Biotin supplementation is recommended for hoof integrity (Comben et al. 1984). Recently, it has been suggested that carnitine supplementation is beneficial for horses.

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