Nutritional Diseases of Farm Animals

BY LOUIS L. MADSSEN

AN UP-TO-DATE SURVEY of the whole field of nutritional deficiencies and the diseases they produce in various classes of livestock. Further details regarding cattle, swine, poultry, and dogs and cats are given in other articles in the Yearbook.

ANIMAL PRODUCTION has kept pace with the general trend toward intensive production in modern agriculture and methods practiced for high production of animal products or rapid growth and fattening for meat have created many problems in animal nutrition. In many large dairies and other feeding establishments the animals are stall- or lot-fed throughout the year, mostly on purchased feeds, and little or no pasture is available. An unwise choice or a poor quality of feed may lead to a shortage of some dietary essentials, or other factors may cause the development of serious nutritional diseases. Overfeeding may be as disastrous as underfeeding.

Although the cause, prevention, and treatment of many nutritional diseases are now understood, such diseases continue to reduce profits in the livestock industry because the knowledge is not put into practice.

The importance of specific treatment or specific means of preventing nutritional diseases cannot be overemphasized. As in diseases due to micro-organisms, parasites, or other causes, prevention is more satisfactory than cure because the disease may result in damage to the animal body that is difficult or impossible to repair. As Russell (55) points out: "The simplest and most fundamental fact that can be stated with regard to the relation of vitamins to disease is that a given vitamin will cure only the disease condition caused by a lack of that vitamin." The same principle holds for

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2 Italic numbers in parentheses refer to Literature Cited, p. 350.
nutritional diseases caused by an insufficiency or excess of any of the many other dietary constituents. General tonics, which are often thought of as cure-alls, should not be relied on when a condition that may be of nutritional origin develops in animals.

Methods for the prevention and control of nutritional diseases are better understood if the nature and functions of the essential factors that must be supplied in the feed are known. Knowledge of the effects of a nutritional deficiency or disease on the body is also helpful. Many laboratory methods for the detection of dietary diseases have been perfected, but recognition of the observable symptoms is one of the most useful means of diagnosis.

THE ESSENTIAL MINERALS

The mineral elements recognized as essential in the diet of farm animals are calcium, phosphorus, magnesium, sodium, chlorine, potassium, iodine, manganese, iron, copper, cobalt, and sulfur. Experiments with rats have shown that zinc is also essential to some animals. A continued deficiency or imbalance of these minerals in the diet will often lead to the development of characteristic symptoms, such as disturbances of metabolism, injury to vital tissues of the body, an abnormal composition of the blood or other body fluids, and depraved appetite.

Ordinarily the essential mineral elements can be supplied in ample quantities by careful selection of common feedstuffs. Sometimes, however, special mineral supplements are needed and should be used, but only the specific minerals that are deficient, in the quantities indicated, need be supplied. Feeding an excessive amount of minerals or a complex mineral mixture when it is not required is expensive and wasteful and may be harmful to the animals. An excess of some mineral salts in the diet interferes with the assimilation of phosphorus, and others, such as selenium and fluorine salts, are directly toxic even in very small quantities.

THE MINERAL-DEFICIENCY DISEASES

Diseases of Calcium and Phosphorus Metabolism and the Interrelationship of Vitamin D

Approximately 70 percent of the ash, or mineral content, of the animal body consists of calcium and phosphorus. About 99 percent of the calcium and over 80 percent of the phosphorus are found in the bones and teeth. The soft tissues, the blood and other body fluids, and some secretions also contain these minerals. Lactating animals utilize larger quantities of these two minerals than nonlactating animals, since milk contains considerable calcium and phosphorus. Disturbances of metabolism caused by an insufficient supply of calcium or phosphorus or an unbalanced proportion of these minerals, with or without an adequate amount of vitamin D in the ration, are largely responsible for a series of related diseases—rickets, osteomalacia, osteoporosis, and osteofibrosis—involving not only the skeletal system but also other tissues and the metabolism of the body as a whole.
Rickets (Rachitis)

Rickets is a disease of young animals—calves, foals, pigs, lambs, kids, pups, and chicks—which is characterized by a failure of growing bone to calcify, or harden, properly.

Bones that are rapidly increasing in length, such as the long bones of the legs and the ribs, are the most likely to be visibly affected. At the epiphysis (the region near the ends of bones where growth in length takes place) cartilage continues to be produced, but normal bone is not formed. The epiphyseal region thus becomes wider than it is normally, and because it is soft from lack of ossification (bone formation) it becomes more or less curved and bulges out, causing irregular enlargements, due presumably to the weight of the animal body and the normal tension or strain of the muscles. The result is a noticeable enlargement of the joints, particularly the knees and hocks, with an abnormal straightening or curving of the pasterns which interferes with normal walking. In addition the joint surfaces (articular cartilages) may become eroded and roughened so that movement of the joint is painful. This results in a typical stilted gait, and the animal lies down often. The costo-chondral junction (union of the rib with the breastbone cartilage) also bulges irregularly, and the result is what is known as the rachitic rosary, or beaded ribs, which, though it can be detected in the living animal, is often noticed only in the carcass. These malformed bones may cause paralysis or pain by pressing on nerves, and they are very easily fractured because of their fragility.

Bones also become softened because of partial resorption of salts from previously formed bone when there is a deficiency of the normal quantity of mineral salts to be deposited in the newly forming bone. The shafts of the leg bones may bend, presumably owing to the weight of the body and to muscle tension. The spinal column also may bend sidewise, a hump may form, or the back may sway. The bones of the head may become distorted, with a tendency towards shortening and an increase in width. Teeth that grow in during a rachitic period may be malformed.

The skeletal abnormalities described are symptoms of the advanced stage of the disease, but they may be the first ones a stockman notices or recognizes. Symptoms that often precede the more severe clinical manifestations are loss of appetite, slowing down of growth or even loss of weight, digestive disturbances, and tetany (convulsions, rigid muscles, stiff legs). A decline in blood calcium or inorganic phosphorus or both usually precedes all other clinical symptoms. Where laboratory facilities are available to make such determinations, the loss of these minerals provides an excellent means of detecting vitamin D deficiency or other abnormalities affecting calcium and phosphorus metabolism.

CAUSE, PREVENTION, AND TREATMENT

Swine and poultry that are normally fed heavily on grains may not receive an adequate supply of calcium unless a mineral supplement or a feed source of calcium is provided. The supply of phosphorus is usually ample in a heavy grain ration. Swine are sensi-
tive to a calcium deficiency, and colts cannot tolerate a high proportion of phosphorus to calcium. Maynard (40) writes, "The normal calcium-phosphorus ratio has been defined as lying between 2:1 and 1:2, but adequate nutrition is possible outside of these limits." The general statement may be made that the harmful effects of vitamin D deficiency do not develop so soon when the calcium-phosphorus ratio is between 1:2 and 2:1. This explains the difficulties resulting from feeding horses a heavy ration of grains with only a small allowance of hay. Timothy hay is usually even lower in calcium than clover or alfalfa hay. Calves and lambs rarely suffer from calcium undernutrition on ordinary rations and are not so sensitive as colts to an abnormal calcium-phosphorus ratio, but they are affected if the amount of phosphorus is inadequate.

Rickets may or may not be due to a simple vitamin D deficiency. An adequate supply of vitamin D cannot compensate for a faulty proportion or inadequate amounts of calcium and phosphorus in the diet. How vitamin D acts is not entirely clear, but in cases of rickets where vitamin D deficiency is the limiting factor the results of administering it are very specific and dramatic. Almost immediately there is an increase in the retention of calcium and phosphorus in the body, probably involving both increased absorption and reduced excretion. Blood phosphorus and calcium return to normal, and new deposits of bone salts can be demonstrated within a few hours.

Direct exposure of the body to sunlight has the same effect on calcium and phosphorus metabolism as vitamin D in the ration, but the effectiveness of sunlight in curing and preventing rickets depends on the intensity of the ultraviolet part of the light and the length and regularity of exposure. Winter sunlight is normally lower in ultraviolet rays than spring or summer sunshine. Ordinary window glass filters out the ultraviolet rays of the sun and makes the light that passes through it ineffective as an antirachitic.

Successful treatment of rickets depends on supplying adequate amounts of vitamin D and adjusting the intake and the ratio of calcium and phosphorus. Providing calves, colts, and lambs with liberal amounts of sun-cured hay of good quality and allowing them daily exposure to direct sunlight are the cheapest assurances against vitamin D deficiency. For swine, also, sun-cured alfalfa or some other legume hay of good quality is a good source of vitamin D and calcium. Roughages that are artificially dried immediately after cutting and allowed little or no ultraviolet irradiation from the sun are poor sources of this vitamin.

Commercial sources of vitamin D, such as fish oils, fish-oil concentrates, irradiated ergosterol, activated animal sterols, and irradiated yeast are also available for animal feeding when extra vitamin D is needed. The various forms of vitamin D from activated ergosterol and cholesterol are in general equally valuable in the nutri-
tion of all farm animals except poultry, which do not utilize irradiated ergosterol or calciferol economically.

Sunshine, cod-liver oil, and other fish oils containing vitamin D are effective in preventing and curing rickets in swine (30), colts, and other farm animals, but little is known about the actual vitamin D requirements of the animals. Increasing the calcium content of rations containing a large proportion of grain may also be helpful. The prevention and treatment of rickets in calves are discussed in the article on Nutritional Diseases of Cattle, page 665.

Prompt treatment of rickets is imperative. If the disease is allowed to progress until marked joint enlargement and bending of the bones has taken place, treatment is less successful. Bony malformations usually calcify and become permanent. Stiffness may largely disappear, and bowed legs may straighten considerably, but badly eroded joint cartilages are practically irreparable. Rachitic females, after successful treatment, may reproduce normally (fig. 1).

**Osteomalacia, Osteoporosis, and Osteofibrosis in Relation to Phosphorus and Calcium Deficiency**

Three abnormal bone conditions are characterized by rather definite alterations in the conformation of the skeleton. Several factors, among them phosphorus deficiency, calcium deficiency, an abnormal proportion of these two minerals in the ration, vitamin D deficiency, and an altered rate of secretion of certain endocrine glands such as the parathyroids, may be involved in the production of these changes. A coexisting protein and vitamin A deficiency may also be involved in some cases.

Osteomalacia is a disease of adult animals named from one of its characteristic symptoms—softening and replacement of bone with osteoid tissue, which resembles uncalcified bone. In some respects this condition is similar to rickets and has been referred to as adult rickets, but it occurs after growth in the length of the bones has largely ceased. When the condition is caused by a deficiency of phosphorus in the animal's ration, it is usually called phosphorus deficiency. The disease is common in pregnant or lactating cows, particularly on phosphorus-deficient range or during periods of drought. It is also seen in sheep, goats, swine, horses, and mules. Calcium and vitamin D deficiency may also be involved.

Osteoporosis is a similar condition resulting from faulty bone metabolism, except that the changes in the bone are those of atrophy, producing a thin porous structure and a failure of normal bone regeneration, but without the osteoid tissue seen in osteomalacia.

Osteofibrosis is characterized by enlargement and partial replacement of the bones with soft, poorly calcified fibrous tissue which may also occupy the marrow cavity. The bones of the face and jaws especially become enlarged, and the condition in horses is commonly known as bighead. Goats and swine are similarly affected. The animals are easily fatigued and may have a snuffing respiration, and they often have enlargements of the leg bones and show varying degrees of lameness.
Lesions or tissue changes, resembling osteomalacia, osteoporosis, and osteofibrosis may occur to some extent in animals as a secondary condition resulting from rickets, whether caused by vitamin D deficiency or faulty calcium and phosphorus nutrition, or as the result of some endocrine disturbance. Such conditions can be identified by the changes they produce in the skeleton, but changes in blood calcium and phosphorus are not always characteristic. A reduction in total inorganic phosphorus or in total calcium may, however, precede extensive bone malformations and other symptoms.

Phosphorus-deficient animals have a depraved appetite (fig. 2) and fail to breed regularly, and their milk production is markedly decreased. Growth and development are slow, and the animals become emaciated and fail to reach normal adult size.

In horses phosphorus deficiency causes a reduction in blood phosphorus and considerable rarefaction of bone before the effects are evident in their general condition. Preliminary results from experi-
FiguRe 2.—These sheep are eating a dried sheep carcass. The supply of phosphorus and protein was low on the natural range where they had been grazing, and phosphorus-deficient animals often chew bones and other nonfood materials apparently in an effort to remedy the deficiency. (Courtesy of G. H. Hart, California Agricultural Experiment Station.

mental work in the Bureau of Animal Industry indicate that X-ray photographs of the tail bones of horses can be used to determine changes in density and structure of bone due to phosphorus deficiency.

Ringbones, splints and bone spavins, and other unsound conditions of the bones of horses are suspected of being symptoms of abnormal bone metabolism due to a deficiency or imbalance of calcium and phosphorus in the ration. Lameness, stiffness of gait, fragility of bone, and enlargement of the bones of the face and jaw may occur in horses, swine, and goats. Bony enlargements are frequently due to osteofibrosis.

Animals receiving an insufficient supply of calcium may also develop fragile bones, and fail to reproduce and lactate normally. This condition is very rare, however, and does not usually occur if good hay or pasture forage of normal calcium content is available.

CAUSE, PREVENTION, AND TREATMENT

A deficiency of phosphorus in the forage in many areas of the world is largely responsible for the losses from phosphorus deficiency in cattle, sheep, and goats. A feed shortage due to drought or other causes may also be responsible. Calcium-deficient areas, though apparently not as widespread as those deficient in phosphorus, have been reported in parts of Florida, Louisiana, Nebraska, Virginia, and West
Virginia. Workers in the Department of Agriculture and in some State experiment stations are attempting to locate all the soil areas in the United States that produce mineral-deficient forages.

As noted in the discussion of rickets, heavy grain feeding may lead to calcium deficiency unless the ration contains liberal quantities of other calcium-rich feeds. Calcium deficiency is very rare in cattle and sheep. There is little experimental evidence to indicate the necessity of adding calcium-rich supplements to the ration of growing calves or lactating dairy cows when they are fed ordinary rations of hay and grain, though the evidence is still incomplete in the case of cows producing large quantities of milk.

Diseases caused by abnormal phosphorus and calcium metabolism due to errors in diet can be prevented or treated in any one of three ways: (1) By using natural feeds that contain sufficient quantities of calcium and phosphorus; (2) by increasing the calcium and phosphorus content or the yield of pasturage or hay by fertilizing the soil on which the crops are grown; and (3) by feeding a specific mineral supplement.

Feeds low in phosphorus and requiring supplementation if they make up a large portion of the ration are beet pulp, molasses, black grama hay, kafir or corn fodder, prairie hay, sorgo hay and fodder, and legumes and grasses grown on phosphorus-deficient soils, especially during dry seasons. Feeds rich in phosphorus are wheat bran; whole cereal grains; tankage; cottonseed, linseed, peanut, and soybean meals; and hays or other herbage from phosphorus-rich soils.

Feeds rich in both calcium and phosphorus are steamed bonemeal, skim milk, and buttermilk. When they contain bone, tankage, meat scrap, and fish meal also supply considerable calcium as well as phosphorus. High-quality ground limestone (calcium carbonate) and oyster-shell are excellent sources of calcium. Phosphatic limestone, purified to remove fluorine, may also be used as a source of calcium and phosphorus. Legume hays (alfalfa, lespedeza, soybean, clover, etc.) are generally rich in calcium, and timothy hay and other grass hays, though containing less, usually furnish adequate amounts even for lactating dairy cows if the hay is of good quality and is eaten in liberal quantities.

Meigs and his coworkers, who studied the effects of feeding low-calcium rations for long periods on calcium and phosphorus metabolism in dairy cows, writes:

> It appears that, when the calcium content of a ration is reduced by substituting timothy hay or straw for alfalfa, the vitamin A content is also likely to be reduced, and that the failures in reproduction, which have occurred on rations in which the roughage was timothy hay or straw, are to be attributed to a vitamin A deficiency rather than to calcium deficiency. The physiological effects of rations which are deficient in calcium, though adequate in vitamin A, are in need of further investigation.

Hart and Miller point out the necessity of correcting protein deficiency, which may be coexistent with a low phosphorus intake.

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4 A survey of deficient and excess minerals in forage in the United States was made by Hartman in 1939 (19). Reported phosphorus-deficient areas and areas of similar parent soil materials where natural feeds under certain conditions may be low enough in phosphorus to produce phosphorus deficiency in livestock were also outlined in Technology on the Farm (63).
MAGNESIUM DEFICIENCY

It is unlikely that a deficiency of magnesium will occur in ordinary farm rations. The deficiency has been produced, however, by feeding calves for extended periods on milk without hay or a grain supplement, as well as by feeding purified diets low in magnesium. Symptoms, prevention, and treatment are discussed in the article on Nutritional Diseases of Cattle, page 651. Huffman and Duncan (22, 23) have demonstrated that magnesium improves calcium and phosphorus metabolism and calcification of bone in dairy calves, the action being described as a "vitamin D-sparing" effect. It is most evident on rations low but not entirely lacking in vitamin D.

Magnesium is needed by the body in relatively small amounts, but it is very important physiologically. About 70 percent of the magnesium in the body is in the bones, where it occurs in combination with calcium and phosphorus compounds, forming complex bone salts. The muscles contain more magnesium than calcium, but the function of magnesium in muscles is not completely understood. Magnesium is closely related to calcium metabolism, and is also known to function as a coenzyme in various reactions involving carbohydrate and phosphorus metabolism. Magnesium is also present in the blood, organs, and tissue fluids of the body.

SALT (SODIUM CHLORIDE) DEFICIENCY

Salt is one of the essential mineral compounds most likely to be lacking in the diet of herbivorous (grass-eating) animals, but fortunately it is one of the easiest and cheapest to provide as a supplement. Both sodium and chlorine, which combine in common salt, are essential in animal nutrition. Wild herbivores, such as deer and buffalo, will travel long distances and risk danger to lick salt from natural deposits. Swine and poultry also need salt. Such animals as horses, which work hard and perspire profusely, as well as lactating animals such as heavy-producing dairy cows, need more salt than usual to compensate for that lost in perspiration and in milk.

Animals deprived of salt develop a ravenous appetite for it, and if suddenly given free access to salt may consume too much, with symptoms of poisoning and even death as a result. (See the article on Salt Tolerance and Salt Poisoning of Swine, p. 803.) Other symptoms of salt deficiency are loss of appetite for ordinary foods, loss of weight, a rough coat, and a drop in milk yield. Salt deficiency is easily avoided by giving animals free access to salt regularly. It is important, however, to hand-feed small amounts to salt-hungry animals for several days before allowing them access to unlimited quantities.

Salt has several important functions in the body. It helps to maintain osmotic-pressure relationships in body cells and fluids, aids in the regulation of water metabolism, and is an essential constituent of milk, eggs, and all body cells. Sodium is the largest in quantity of the base minerals in the body cells, fluids, and alkaline digestive juices (bile and pancreatic juice), and chlorine has a part in the formation of hydrochloric acid in gastric juice.
Chemical and spectrographic analyses of tissues and fluids of animal bodies reveal that a large number of mineral elements are present in very small quantities. Some of these so-called trace minerals, including iodine, manganese, iron, copper, cobalt, and zinc, are now known to be indispensable in animal nutrition, while the essential nature of others, including silicon, nickel, boron, bromine, aluminum, strontium, vanadium, and silver, has not been established. Some may be present in the body merely because they were contained in the food; on the other hand, they may prove to be essential to certain physiological functions. A deficiency of some essential trace minerals is now known to produce typical symptoms. Small quantities of other minerals, including fluorine, selenium, arsenic, molybdenum, and lead, may be harmful to animals.

**Iodine Deficiency**

Approximately half of the small amount of iodine in the body is located in the thyroid gland. Iodine was the first trace element demonstrated to be essential for normal nutrition, probably because a deficiency of this mineral causes such conditions in man as goiter and cretinism. As early as 1820 the chemist Dumas and the physician Coidet in France studied the use of iodine in the treatment of goiter. Iodine therapy was hailed for a time, but it gradually fell into disrepute for several reasons. It failed to cure all types of goiter, there were some toxic results from overdosing, and the rise of Pasteur’s germ theory of disease (1873–95) caused the older chemical theories to be neglected; medical men and bacteriologists were looking for a microbe as a cause of goiter.

In 1895 Baumann (1) discovered iodine in organic combination in the thyroid gland, and in 1915 Kendall (28) isolated from thyroids an iodine-containing compound in crystalline form which had the same properties as the whole gland. Later Harrington and Barger (16) synthesized this compound—thyroxin—in the laboratory. The essential facts concerning thyroxin are that it contains 65 percent of iodine by weight, is the principal hormone produced by the thyroid gland, has a regulating effect on body metabolism, and is also concerned in growth, development, and the reproductive processes.

**SIMPLE GOITER IN FARM ANIMALS**

Enlargement of the thyroid gland is a common manifestation of iodine deficiency in domestic animals. It is an advanced symptom, however, and the chief loss from iodine deficiency is from interference with reproductive processes and the birth of weak, deformed offspring which often fail to survive (27, 65). The gestation period of mares receiving insufficient iodine is frequently longer than normal, and the foal is either still-born or so weak that it is unable to get up and nurse normally. Most such colts die within a few days, although some survive and make a complete recovery.
Cows may also give birth to weak, goitrous (often called big-neck) calves. Most such calves are alive at birth, although a few may be still-born; some are weak and die within a few days; others have approximately normal vigor and are not noticeably affected except for enlargement of the thyroids, which may or may not cause difficult breathing because of pressure on the trachea (windpipe). If the calf is able to take nourishment, the goiter frequently diminishes in size until it is no longer noticeable, but sometimes it remains throughout adult life. In severe cases the hair of the calf may be thinner than normal or the animal may be almost completely hairless.

Sows may give birth to weak pigs which are often more or less hairless and may be still-born or die within a few hours. Some of the pigs in a litter may be more seriously affected than others. Thyroid enlargement may be present but is often overlooked unless a dissection is made. The skin of the abnormal pigs is often thick and pulpy, especially over the shoulder and neck region, owing to a watery swelling (edema) similar to that seen in human beings suffering from hypothyroidism.

Ewes receiving insufficient iodine also give birth to weak lambs which often show thyroid enlargement (big neck—fig. 3) and may be partially woolless. The death rate among such lambs is very high.

**Figure 3.**—A lamb with the skin removed to show an enlarged thyroid gland (goiter), a typical result of iodine deficiency.
Iodine deficiency in domestic animals usually results from a deficiency of this mineral in the soil and hence in the feed and water. Other factors such as diets high in calcium or fat may be antagonistic to iodine absorption. Iodine-deficient areas are known to exist in Montana, Idaho, Oregon, Washington, and parts of Utah, Wyoming, North Dakota, Minnesota, Wisconsin, and Michigan. Iodine deficiency has also been reported in certain sections of California, Nevada, Colorado, Nebraska, Iowa, and Texas (19, 44). The exact boundaries of the deficient areas are not known, and some sections are more deficient than others.

PREVENTION AND TREATMENT OF GOITER

The effectiveness of supplementary feeding of iodine to prevent goiter and associated symptoms due to iodine deficiency has been conclusively demonstrated for all classes of farm animals, but the exact minimum requirement of iodine has not been established. In a recent publication Welch (66), of Montana, confirms the finding that the control of iodine deficiency in farm animals is most easily accomplished by the use of iodized salt containing 0.02 percent of potassium iodide. Salt of this iodine content can be purchased from feed dealers, or salt containing approximately the same iodine content can be prepared by thoroughly mixing 1 ounce of powdered potassium iodide with 300 pounds of granulated stock salt. The iodized salt should be made available to sows during at least 3 months of the pregnancy period, to ewes for 3 to 4 months, and to cows and mares for 5 months or slightly more. Iodine deficiency is usually most prevalent in animals born early in the spring. If goiter appears in animals born during midsummer or fall, iodized salt in place of ordinary salt should be fed, at least to breeding animals, throughout the year. The need of feeding iodine to other than pregnant animals in iodine-deficient areas is questionable. Crampton (7) has recently summarized the recommendations of many investigators which have presumably been found satisfactory in the control of iodine deficiency.

Iodine compounds are expensive, however, and they have toxic effects on animals if fed in large amounts. Where extra iodine is needed, the amounts required are so small that the increase in feed costs is not appreciable. Salts of iodine readily disintegrate, with loss of iodine, if the mineral mixture is stored for long periods or exposed to sunshine and rain. Methods of stabilizing iodine in mineral feeds have recently been perfected.

Control of goiter and related conditions in animals—at one time a serious threat to the livestock industry in some areas—by feeding iodine compounds is so successful that economic losses from this condition should be very small if proper precautions are taken.

Manganese Deficiency

The essential function of manganese in the animal body was first established in laboratory experiments on rats. Female rats on low manganese rations failed to suckle their young, while male animals developed testicular atrophy. Chickens also require this min-
eral for normal growth of bone, normal development of embryos, and hatchability of eggs. Experiments with farm animals have been less successful in establishing symptoms of manganese deficiency, although recent work by Miller and coworkers (43) indicates that a lameness developing in pigs fed a ration consisting of yellow corn, tankage, soybean meal, ground alfalfa, and salt could be effectively prevented, but not cured, by adding 50 to 60 parts per million of manganese to the ration.

**Iron, Copper, and Cobalt**

The essential functions of iron, copper, and cobalt are in some respects interrelated, and prolonged deficiency of one or more of them causes rather characteristic symptoms. The importance of iron as a constituent of hemoglobin is widely recognized. Iron also functions in other compounds that play an important part in cellular oxidation reactions everywhere in the body. The relation of copper to iron metabolism in hemoglobin formation was discovered in 1928. Recently cobalt has been demonstrated to be indispensable in animal nutrition, functioning in an unknown way in blood formation and in the utilization of iron. A deficiency of this element may occur alone or in combination with copper or iron deficiency.

Hemoglobin formation is a continuous process throughout life, and whenever the body reserve of iron becomes depleted because of an inadequate intake of iron or copper, nutritional anemia (decrease in red blood cells and hemoglobin) develops. Such a condition is common in suckling pigs kept in indoor pens, and it may also occur in calves and lambs if they are kept on a diet of milk, exclusively, too long. Cattle, sheep, goats, and adult swine also become anemic when there is a natural deficiency of iron, copper, or cobalt in forage and grains caused by a deficiency of these minerals in the soil. In Florida this condition is known as “salt sick,” and it can be successfully controlled by supplemental feeding as described later. Intestinal parasitism frequently results in anemia, and this condition may be confused with anemia due to a primary nutritional deficiency.

Cobalt feeding was known to have the effect of producing an increase in red blood cells in rats before its significance in the nutrition of farm animals was discovered. For many years a group of somewhat similar diseases of sheep and cattle have been recognized under the names “enzootic marasmus” or “coast disease” in Australia, “bush sickness” or “Morton Mains disease” in New Zealand, “naku-riris” in Kenya, “pine” or “pinning” in Scotland, “salt sick” or “hill sick” in Florida, and “Grand Traverse” or “lake-shore disease” in Michigan.

The conditions named are characterized by anemia, progressive emaciation, and death. Moving the animals from the “affected” pastures of the coastal regions to pasture farther inland or feeding certain crude iron salts occasionally resulted in cures, and this led to a hypothesis that the condition was due to iron deficiency. Discouraging results frequently followed dosing with large amounts of various iron compounds, however, and it was also shown by Under-
wood (61) that the liver and spleen of sheep suffering with the disease often contained extra stores of iron. Filmer and Underwood (12) discovered that an iron-free extract of limonite, a curative iron compound, was just as effective in curing the condition as limonite itself. Further work by these investigators demonstrated that the active substance of the iron-free extract was cobalt (62) and that normal growth and health of sheep resulted from the feeding of cobalt chloride in the small amounts of 0.1 milligram of cobalt daily for sheep and 0.3 milligram daily for cattle. Dixon (9) obtained excellent results in the control of Morton Mains disease of lambs by providing free access to cobaltized salt lick, which was made by spraying a solution of 4 ounces of cobalt chloride on 1 ton of salt.

In coast disease of sheep in South Australia (39) the iron content of the liver is greatly increased above normal, and there are smaller increases in the spleen and kidneys, but the copper content of the liver is usually greatly reduced. Supplements of copper and cobalt are more effective than either alone in treating this condition.

**COPPER DEFICIENCY AND COPPER POISONING**

It is evident from the previous discussion of iron, copper, and cobalt that a deficiency of these minerals may occur in combination or separately. An apparently primary copper deficiency is now known to exist in Great Britain and western Australia, causing swayback, warfa, or enzootic ataxia in lambs. A similar condition has also been reported from South Africa, Sweden, and Peru. The full extent to which such a deficiency exists in the United States is not known, although sections of Florida are known to be copper-deficient, which suggests also a possible deficiency in other areas of the Coastal Plain region.

The condition called swayback usually affects lambs during the first month of life, although some lambs are unable to rise and suckle at birth or shortly afterwards. Death usually results from starvation. Degeneration of the myelin (sheath) of nerve tracts, particularly in the spinal cord, is usually found. When pregnant ewes are given access to salt licks containing 1 percent of copper as copper sulfate, the lambs are usually free from swayback (10), although the relation between copper and myelin metabolism is still obscure. Copper sulfate is essential in small quantities in cases of copper deficiency, but it is poisonous when fed in large amounts, and this should be remembered in all cases where copper therapy is indicated. Rietz (54) found that giving sheep free access to a mixture of 1 pound of copper sulfate to 30 pounds of salt while on pasture proved ineffective in controlling parasites yet caused death from copper poisoning. Losses on the range and experimental production of copper poisoning (so-called ictohemoglobinuria) in sheep by feeding copper containing commercial mineral mixtures are also reported by Boughton and Hardy (4), of the Texas Agricultural Experiment Station.

**Sulfur**

Sulfur, in the form of sulfur-containing protein or amino acids, is an essential dietary constituent for animals. Inorganic sources,
such as the sulfates or flowers of sulfur, have not definitely proved to be of value when added directly to the ration or used as a constituent of mineral supplements. Prevention of sulfur deficiency therefore involves providing an adequate supply of sulfur-containing proteins.

**TOXIC MATERIALS**

**Selenium Poisoning**

In 1856 T. C. Madison, stationed at Fort Randall, Territory of Nebraska (now in Gregory County, S. D.), accurately described a new disease of army horses at that midwestern outpost. The question whether the disease was caused by the horses' eating poisonous plants or was due to contagion was not settled, however. Many reports followed in later years from ranchers, homesteaders, and veterinarians concerning losses among livestock in the north-central Great Plains area from this disease. The disease became known as alkali disease owing to a mistaken idea that it was caused by drinking alkali water, but this was finally ruled out, and attention was centered on plant poisons as the cause. In 1931 H. G. Knight, Chief of the Bureau of Chemistry and Soils of the Department of Agriculture, suggested that the disease might be due to selenium poisoning resulting from the consumption of plants that had absorbed traces of selenium from the soil. Cooperative work by the South Dakota experiment station and several bureaus of the Department of Agriculture provided evidence that this was true. A preliminary field survey report of this cooperative effort (13) describes the symptoms of alkali disease, which has also been called blind stagger and bob-tailed disease, as follows:

In horses, cattle, and swine, the malady manifests itself clinically by an alteration in the growth of the horn, of the hoofs, and a loss of hair from the mane and tail of horses, and switch of cattle, and the hair of swine. There are various gradations of these conditions, from mild cases in which there may be only subnormal growth or a loss of hair, to severe cases in which a break in the continuity of the growth of the walls of the hoof develops, followed eventually by a sloughing-off of the old hoofs. When this happens, the animals are more or less lame for months. In the more severe cases, the animals move about with great difficulty, and unless given careful attention may die of thirst or starvation. Many of the most severely affected animals recover, but it is the belief of many that such animals are never quite as valuable as before an attack.

In swine, particularly in young pigs, in addition to lesions produced in the feet and loss of hair, death also may occur. After being badly "alkalied," many of the animals make but little, if any, gain in weight in spite of plenty of nutritious feed.

"Alkali" disease due to selenium poisoning has been reported primarily from areas in South Dakota, Montana, Wyoming, Nebraska, and Kansas, although other States in the Great Plains and Rocky Mountains have areas of seleniferous soils which produce plants of increased selenium content. Control measures for the disease are largely based on prevention. Affected animals should be transferred to areas where the disease is not prevalent and fed selenium-free
grains and forage. "Alkalied" (selenium-containing) grain, hay, or grass should not be used for livestock feed or sold on the market.

In the rat, diets high in casein and lactalbumin (both contained in milk) tend to protect against chronic selenium poisoning (14).

**Fluorine Poisoning (Fluorosis)**

Fluorine is widely distributed in nature in soil, rocks, water, and plants, but only in certain areas is the concentration high enough in the water or food supply to interfere with animal and human nutrition. All animals also contain traces of fluorine in their bodies, concentrated chiefly in the bones and teeth, but there is little evidence that it is an essential constituent of these structures.

Fluorine has a marked affinity for calcium, and this undoubtedly accounts for its interference with normal calcification when the intake becomes excessive. As little as 1 part per million of fluorine in the drinking water when used during the period of tooth formation will cause mottled enamel in human teeth, and severe mottling follows the continuous drinking of water containing 6 to 16 parts per million (56). Livestock are apparently not affected to any great extent by the low concentrations of fluorine that cause trouble in human beings. In the survey conducted by Hartman (19), reports were obtained that feed produced in some parts of Arkansas contained excess fluorine and that water from certain warm springs of California contained enough fluorine to cause trouble with the teeth of cattle. Mottled enamel in the teeth of cattle has also been reported in South Carolina and western Texas (8).

Fluorine is a cumulative poison, and long-continued consumption of relatively small quantities produces chronic fluorosis in all farm animals and poultry (51). By far the greatest danger from fluorine poisoning to livestock comes from the use in mineral mixtures of natural phosphatic limestone or rock phosphates, which are usually high in fluorine.

The general symptoms of chronic fluorine poisoning are abnormal teeth and bones, stiffness of joints, loss of appetite, emaciation, reduction in milk flow, diarrhea, salt hunger, kidney damage, and injury to other organs such as the liver, heart, adrenal glands, testes, and thyroid.

Prevention and treatment of fluorosis depends on eliminating any excessive fluorine intake by careful selection of feeds (particularly constituents of mineral supplements) and discontinuing the use of water with a high fluorine content. Animals suffering from marked injuries to teeth, bones, and organs due to fluorine poisoning may improve to some extent if the source of excessive fluorine is eliminated, but the damage may be permanent.

**THE VITAMINS**

The most important vitamins in the nutrition of cattle, sheep, goats, and horses are A and D, while in swine nutrition, in addition to these two vitamins practically all of the members of the vitamin
B complex must be considered. A general knowledge of the vitamins, including an understanding of the conditions under which deficiencies may occur and how to prevent them by the use of available feeds whenever possible, is necessary for continued success in feeding farm animals.

**Vitamin A**

Vitamin A is distinctive in that it is formed nowhere in nature except as a product of animal metabolism, but it is synthesized from carotenoids (alpha, beta, and gamma carotene and cryptoxanthin), which are formed only by plants. Beta carotene is the most important source of vitamin A, since it is capable of yielding two molecules of this essential substance, while the other carotenes form only one.

The carotenes are yellow to orange red in color, depending on their concentration, and are responsible for the yellow color of common varieties of carrots, corn, sweetpotatoes, etc. Carotene is also abundant in green forage of all kinds. The solution of the mystery of why both the yellow carotene and the nearly colorless vitamin A, as found in fish oils, can cure and prevent symptoms of vitamin A deficiency was one of the first triumphs of vitamin chemistry.

Farm animals of all ages may suffer from vitamin A deficiency, but young animals are particularly susceptible. The symptoms in calves are like those in older animals (45, 46). Details of the symptoms in cattle and hogs are given in the articles on nutritional diseases of these animals, pages 658 and 818.

Sheep also develop night blindness from vitamin A deficiency, and pregnant ewes may abort or give birth to weak offspring which usually die shortly afterwards (18). Other symptoms in sheep are loss of appetite, poor condition, and weakness. Hale (15) reports that sows on vitamin-A-deficient rations may fail completely in reproduction or give birth to blind or eyeless pigs.

According to work by Howell, Hart, and Ittner (21), horses on a vitamin-A-deficient diet develop typical symptoms of night blindness, excessive lachrymation, keratinization of the cornea, difficult breathing, reproductive failure, poor appetite, scaly hoofs, and progressive weakness. Death may follow. Characteristic rarefying lesions of the joint cartilages and bones also were present in animals suffering from vitamin A deficiency, but positive evidence that the bone condition was due only to this deficiency was not obtained.

In work at the Beltsville Research Center, Beltsville, Md., microscopic examination of a urine sample obtained with a catheter from the bladder of a yearling Belgian filly on a very low carotene diet revealed a large proportion of crystalline sediment and a considerable number of large keratinized epithelial, or lining, cells. A diagnosis of vitamin A deficiency based on the abnormal cellular debris and the response of the animal to vitamin A therapy appeared to be correct (fig. 4). During the period of deficiency, there was also faulty hoof growth (fig. 5).
A lack of sufficient vitamin A or its precursor, carotene, in the diet is the chief cause of vitamin A deficiency in farm animals.

Figure 4.—Horses develop vitamin A deficiency if their diet is low in carotene. A, A yearling Belgian filly suffering from vitamin A deficiency. Note posture, rough coat, large area of scaly skin outlined in white, and poor condition. B, The same animal apparently normal again after vitamin A therapy.
Chronic intestinal disturbances, such as diarrhea or constipation, and insufficient secretion of bile are factors that interfere with the absorption of carotene. Liver injury may also interfere with the conversion of absorbed carotene into vitamin A, which is believed to take place largely in this organ. Charcoal added to a diet may destroy vitamin A and certain other vitamins, presumably by their removal or inactivation, and this raises the question of the advisa-

![Image of horse hooves showing hoof growth before and after vitamin A therapy.](image-url)

Figure 5.—Hoof growth is abnormal in horses during a period of vitamin A deficiency, but normal growth will probably be resumed if the deficiency is corrected. These photographs show the right front foot of the filly shown in figure 4 during the deficiency period and at two intervals during recovery.  

*A*, Note the band of rough, scaly hoof formed while the animal was suffering from vitamin A deficiency.  

*B* and *C*, Side and front views of the hoof after 5 months of vitamin A therapy. The contracted band of defective hoof is growing out and normal hoof growth is proceeding.  

*D*, About 11 months after the deficiency was corrected the area of defective hoof has completely grown out, and the newly formed hoof is apparently normal.
bility of using charcoal in grain or mineral mixtures. Iron salts and rancid fats in a ration also destroy vitamin A and carotene.

Carotene in hay decreases rapidly in amount under ordinary storage conditions; hence animals fed old hay may not receive enough carotene. Moldy hay or forage that has been rained on or sun-bleached during the curing process is also low in carotene content. During periods of prolonged drought, range or pasture becomes deficient in carotene as well as other essential nutrients, such as protein and phosphorus. Cattle or other animals grazing on such forage may show deficiency symptoms while on the range or go into the winter feeding period with an abnormally low storage reserve so that deficiencies develop before fresh green feed is available again.

**Prevention and Treatment of Vitamin A Deficiency**

An abundance of green grass or legume pasture in the summer and of properly cured, good-quality legume or grass hay or silage of the current season's crop in the winter or during periods of dry-lot feeding will prevent or cure this deficiency in cattle, sheep, horses, and goats. Swine also obtain considerable carotene and other essential factors from good pasture. Leafy legume hay for sows or 5 to 10 percent of high-grade alfalfa meal in the grain mixture for fattening swine will supply an abundance of carotene. Yellow corn furnishes some carotene, but it should not be depended on to meet the entire vitamin A requirement of animals.

The minimum requirements of vitamin A and carotene needed to cure night blindness in various species of animals have been determined by Hart (17) and are given in table 1.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Vitamin A</th>
<th>Carotene</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Micro-grams</td>
<td>International units</td>
</tr>
<tr>
<td>Cattle</td>
<td>5.1-6.4</td>
<td>21-27</td>
</tr>
<tr>
<td>Sheep</td>
<td>4.3-6.3</td>
<td>17-26</td>
</tr>
<tr>
<td>Swine</td>
<td>4.4-5.7</td>
<td>18-24</td>
</tr>
<tr>
<td>Horses</td>
<td>4.2-5.3</td>
<td>17-22</td>
</tr>
<tr>
<td>Rats</td>
<td>4.6-5.3</td>
<td>16-23</td>
</tr>
<tr>
<td></td>
<td>3.8-4.6</td>
<td>16-22</td>
</tr>
</tbody>
</table>

1 Data based on cod-liver oil used in cattle, sheep, swine, and horse experiments.
2 Data based on U. S. P. reference (standardized) cod-liver oil.

Hart recommends that at least 5 to 10 times the minimum level of carotene or vitamin A should be furnished under practical conditions, since little or no storage occurs at the minimum level needed to prevent night blindness. The carotene requirements of growing calves and fattening steers and those for reproduction in adult cattle are considered in detail in the article, Nutritional Diseases of Cattle, page 662. As a practical recommendation, Meigs (41), from work in
The Bureau of Dairy Industry at the Beltsville Research Center, Beltsville, Md., states:

The experiments so far carried out indicate that dairy cows will receive about the minimum carotene required for successful reproduction when the dry matter of their rations contains 10 p.p.m. [parts per million] of carotene. Approximately this quantity is supplied when the rations consist of equal parts of grain and hay, if the hay is U. S. No. 1 clover or U. S. No. 1 timothy.

If hays of lower grade and little or no silage or other source of carotene are fed, vitamin-A-deficient calves may be born after the cows have been on the ration for about 5 months following the pasture season. Less is known about the minimum vitamin A requirements for successful reproduction in other farm animals, but it is advisable to avoid a possible deficiency of this factor in breeding animals at all times.

Typical feeds that are deficient in carotene are cottonseed meal and hulls, linseed and soybean meals, wheat, oats, barley, white corn and corn stover, white sorghum grains, beet pulp and molasses, cereal straw, and low-grade hays. These feeds should not be fed alone or in combination with other carotene-deficient feeds over an extended period unless the ration is supplemented with carotene-rich feeds.

Feeds that are good sources of carotene are green grass or legume pasture, properly made silage, high-grade grass or legume hay of a good green color, and yellow varieties of carrots, sweetpotatoes, squash, etc. Cod-liver oil or other vitamin-A-rich fish oils and concentrates, or carotene concentrates in oil solution, may be fed animals to prevent or cure vitamin A deficiency when the regular feed source of carotene is inadequate. All oil solutions of vitamin A and carotene are relatively unstable, and if they are mixed with other feeds, it is not safe to make up more of the mixture at one time than will be used within a few weeks.

**THE VITAMIN B COMPLEX**

The number of recognized components of the vitamin B complex has steadily increased since 1926, when Smith and Hendrick (57) first demonstrated that the so-called water-soluble factor of McColllum and Davis (31) consisted of two fractions, one heat-stable and the other destroyed by heat. Nine so-called B vitamins are now recognized, and most of them have been synthesized in the laboratory. The vitamins of this complex that have been identified as separate entities are thiamin (B₁), riboflavin (growth factor of B-G complex), nicotinic acid (antipellagric factor of B-G complex), pyridoxine (B₆), pantothenic acid, choline, biotin, para-aminobenzoic acid, and inositol. The older method of alphabetical designation and descriptive nomenclature for these factors is rapidly being displaced by their chemical names.

Much is known concerning the essential nature and symptoms of a deficiency of these vitamins in the diet of laboratory animals, such as the rat, and of poultry. Recent studies have also shown that most of these factors are essential for the normal growth and health of swine and that a deficiency may produce typical disease symptoms. Under practical conditions, however, usually only border-line de-
ficiencies occur which seldom manifest specific symptoms other than slow growth, digestive disturbances, unthrifty appearance, and lameness. When these symptoms appear, the possibility of a deficiency of the B vitamins should be investigated. (See the article on Nutritional Diseases of Swine, p. 820.)

A definite dietary requirement for the individual B vitamins has not been established for cattle, sheep, and goats but the existing evidence is not entirely complete. Some workers (6, 47) have reported good results from feeding a concentrate of B vitamins or yeast to horses on very poor rations.

**Synthesis of B Vitamins by Ruminants**

Bacteria growing on the contents of the rumen (paunch) of cattle and sheep have the ability to synthesize practically all of the known B vitamins, an unusual relationship that has been the subject of several recent studies. It is generally believed that vitamins thus synthesized in the rumen become available to the animal as the mass of ingesta and bacteria are digested.

Recently McElroy and Goss have made a quantitative study of the formation of riboflavin and vitamin K (33), vitamin B₆ (pyridoxine) (34), and thiamin (35) in the rumen of cattle and sheep fed diets low in these vitamins. It has also been demonstrated that pantothenic acid, nicotinic acid, and biotin (anti-egg-white-injury factor) are also formed in the rumen (32). Wegner and coworkers (64) made the interesting observation that when thiamin was added to the ration there was an apparent stimulation of synthesis of the other factors except nicotinic acid. Hunt and coworkers (24) found that riboflavin was synthesized in the rumen of steers fed yellow corn, alfalfa, and a protein supplement, although when a diet of alfalfa alone was fed, the rumen contents contained less of this material than did the hay. They point out that the synthesis of riboflavin may depend on the reaction of the rumen, since Kick and associates (39) had previously reported that the rumen contents of steers fed only on alfalfa were alkaline in reaction while those fed grain and a protein supplement had rumen contents that were acid in reaction.

It is impossible to say at present whether rumen synthesis of B vitamins can be depended on to furnish cattle, sheep, and goats with an adequate supply of these factors under all conditions. Work with synthetic or purified diets for herbivorous animals has led to some interesting results. Madsen and coworkers (38) were unable to raise young kids or lambs on purified diets low in B vitamins unless yeast or an alcoholic extract of yeast was added. Pearson and associates (50), however, raised lambs on a diet which was capable of producing blacktongue in dogs and pellagralike symptoms in swine. All of the lambs made good growth for 4 to 5 weeks, but then went off feed and declined in body weight, in some cases as much as 24 percent. Lambs treated with thiamin and nicotinic acid, as well as untreated lambs, subsequently recovered. These workers remark:
Whether this temporary failure reflects a period during which the animal organism was making an adjustment to the deficiency, perhaps through an increase in microorganisms in the rumen that might synthesize nicotinic acid or to some other factor, cannot be answered at present.

Johnson, Loosli, and Maynard (26) fed young calves on rations low in the B vitamins and were unable to demonstrate that the grass-juice factor (as yet unidentified), vitamin C, wheat-germ oil, thiamin, or riboflavin were limiting factors in the ration. However, the performance of the calves was far from normal. Most of them were 30 to 50 percent of normal in rate of growth and had frequent attacks of scours, some failed to grow during the first month of the experiment, and there was often a leveling off of appetite with periodic variations in gain and food intake after about 100 days. These findings emphasize the complexity of the dietary requirements of herbivorous animals.

**Vitamin C (Ascorbic Acid)**

Ascorbic acid, the vitamin that prevents and cures scurvy in human beings, monkeys, and guinea pigs, is receiving new attention in the nutrition and reproductive physiology of farm animals (52, 53). Further details are given in the article on Nutritional Diseases of Cattle, page 664. Ascorbic acid is rapidly destroyed in the rumen of cattle, so that treatment of the type of breeding failure which responds to vitamin C therapy can be accomplished, according to present knowledge, only by injecting the vitamin subcutaneously.

**Vitamin D (Activated Ergosterol and Cholesterol)**

The role of vitamin D in the nutrition of farm animals and the principal sources of the vitamin have been discussed earlier in this article in relation to its function in calcium and phosphorus metabolism and in bone formation.

**Vitamin E (Alpha Tocopherol)**

Twenty years ago it was demonstrated that rats fed certain purified or restricted diets often seemed to grow normally and appeared healthy but frequently failed to reproduce. The missing factor necessary for reproduction was shown to be distinct from any of the vitamins recognized at that time. Later studies have indicated that this vitamin is also concerned in growth, the metabolism of the muscular and nervous systems, and possibly the activity of certain endocrine glands, such as the pituitary and thyroid.

Vitamin E has been isolated from natural sources and synthesized. In the pure synthetic form this vitamin is a light yellow, viscous, oily fluid. It is sold in sealed air-evacuated glass vials. The presence of rancid fats, the oxidation of other substances such as cod-liver oil in a feed mixture, or the presence of iron salts tend to destroy the vitamin.

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5 Several closely related compounds known as tocopherols have vitamin E activity, but alpha-tocopherol or 5-, 7-, 8-trimethyltocol is probably the most potent of these compounds.
Natural foods that are good sources of vitamin E are whole cereal grains, fresh or well-cured roughages, leafy vegetables, and vegetable oils, such as wheat-germ, cottonseed, corn, and soybean.

**Vitamin E and Reproduction in Farm Animals**

Little is known concerning the vitamin E requirements of farm animals except poultry. This is due to the widespread occurrence of the vitamin in common livestock feeds as well as to the fact that it is very expensive to prepare vitamin-E-free diets for the larger animals for experimental purposes. Wheat-germ-oil therapy for the treatment of sterility in cattle has led to conflicting results (see the article on Nutritional Diseases of Cattle, p. 666).

Attempts to determine whether farm animals require vitamin E have led to some interesting findings. Thomas and coworkers at the Iowa Experiment Station have fed goats on a ration of chopped alfalfa hay and ground grains which had been treated with ethereal ferric chloride to destroy or inactivate vitamin E. Rats fed this ration failed to reproduce, showing that it was deficient in vitamin E, but the goats continued to reproduce normally through several generations. Ewes given the same vitamin-E-deficient ration as the goats in preliminary breeding experiments failed to give birth to living young, while of two groups of 10 breeding ewes the group receiving weekly supplements of wheat-germ oil produced a larger lamb crop. The latter experiment was repeated with about 120 ewes, and “this time the feeding of tested wheat-germ oil appeared to be neither beneficial nor harmful to reproduction” (60). Similar work with dairy cattle has been discontinued because of the technical difficulties and expense involved in preparing sufficient amounts of vitamin-E-free rations.

Madsen and coworkers produced muscular dystrophy (a paralysis; the word “dystrophy” means “faulty nutrition”) in goats, sheep, guinea pigs, and rabbits by feeding purified rations. The cause of this disease is now generally accepted to be vitamin E deficiency, although specific evidence for goats and sheep is lacking. No difficulties due to muscular dystrophy were mentioned in the work by Thomas and associates previously cited.

The possible role of vitamin E deficiency in the development of a degeneration of the skeletal muscles in other animals—for example, the stiff-lamb disease, so-called white-muscle disease of calves and pigs, and paralytic myoglobinemia (azoturia, or Monday-morning sickness) of horses—is worthy of investigation. It is now plainly evident, however, that a number of species of animals other than the rat require vitamin E and that this factor has important roles in other vital functions besides reproduction.

The question whether farm animals other than poultry require vitamin E needs further study before recommendations for supplying extra sources of this factor can be made. Ordinary farm rations are usually rich in vitamin E. Good sources include wheat, bran, shorts, linseed meal, hominy feed, corn, cottonseed meal, kafir, alfalfa, white clover, and pasture grasses.
Nutritional Diseases of Farm Animals

MISCELLANEOUS NUTRITIONAL DISEASES

URINARY CALCULI (UROLITHIASIS) AND DIETARY IMBALANCE

Urinary calculi (sand, grit, or stones) formed by the precipitation of salts from the urine in the kidney or bladder are quite common, particularly in cattle, sheep, and horses. Calculi may cause no apparent discomfort to the animal, or they may result in serious symptoms or death by obstructing the flow of urine. The symptoms depend, of course, on the size, location, number, and rate of increase of the calculi and whether or not infection is present. Symptoms caused by urinary calculi are similar in the various species of animals and include frequent attempts to urinate, dribbling or stoppage of urine, pain or renal colic, restlessness in the early stages, and later walking with a characteristic straddling gait. Rupture of the bladder may follow chronic obstruction.

It is known that diet is definitely a factor in the development of calculi, but the problem is very complex and few of the factors involved have been definitely proved. Vitamin A deficiency is thought to favor calculi formation. The case is not so clear-cut for farm animals, however, as it is for laboratory animals such as the rat. Newsom (48) states that vitamin A is quite generally deficient in the rations in the dry lands of eastern Colorado, where calculi are so prevalent, but the disease has also been observed in cattle and sheep on good alfalfa rations.

Calculi occur in animals fed certain rations more frequently than in others. In a recent progress report from the Nebraska station an experiment is described in which an attempt was made to compare Early kalo and Sooner milo with corn as a grain for fattening lambs, and to determine the advisability of adding a small amount of alfalfa hay to such rations when Atlas silage was the only roughage fed. In the experiment there were 30 lambs in each of several lots. In the group receiving Early kalo, cottonseed meal, bonemeal, and Atlas silage, and in another lot receiving Sooner milo as the grain with the other three ingredients, 42 and 58 percent of the wether lambs, respectively, developed severe cases of urinary calculi. Lambs of other lots which received one-half pound of alfalfa in addition to the rations mentioned were largely free of calculi. Lambs receiving yellow corn, alfalfa, or both, were also largely protected from calculi. The results are in agreement with findings of Newsom (48), who states that changing the ration of cattle or sheep to include corn and alfalfa has usually resulted in a cessation of the outbreaks of urinary calculi in fattening animals.

In work by Johnson, Palmer, and Nelson (25), high dietary magnesium was suspected of forming calculi in a group of 2,600 wether lambs in which 10 percent of the animals were affected, but feeding experimental rations containing as much as 1.5 percent of magnesium

to some of these lambs did not produce calculi, although the blood magnesium of the animals was increased threefold.

Specific preventive and curative measures for severe urolithiasis have not been completely worked out. Some veterinary surgeons have become skilled in removing calculi obstructing the urethra of the male, thus giving temporary relief and making it possible for the animal to finish an approximately normal feeding period. Precautionary measures include supplying animals with plenty of drinking water, succulent feed, and an adequate amount of vitamin A; attempting to establish a normal calcium, phosphorus, and magnesium intake; and providing plenty of exercise. The fact that calculi are somewhat more prevalent in animals on full feed indicates the desirability of reducing the grain intake if cases develop. As previously pointed out, changing the ration to corn and alfalfa when possible may also prove beneficial. The value of dietary treatment when calculi are well developed is doubtful. Response may be slow at best.

**OVERFEEDING**

Effects of overfeeding were outlined in the Yearbook of Agriculture for 1939 (36), as follows:

Overfeeding of farm animals can result in heavy financial losses by causing temporary or permanent disability and even death of animals. * * * Livestock will frequently overeat if given an opportunity. The consequences may be only temporary digestive disturbances or they may be so serious as to cause death. * * * Such symptoms as diarrhea, constipation, bloat, colic, and failure to eat the usual quantities of food are danger signals, which if observed in time may be corrected. Digestive troubles from overeating may occur very suddenly, and even if the services of a veterinarian are immediately available a fatal outcome may not be prevented.

Newsom and Thorp (49) describe the effects of overeating in lambs and state that this condition causes more losses in the feed lots of Colorado than all other troubles combined. Lambs sick from overeating, particularly on grain, may have nervous symptoms such as head retractions, staggering gait, walking in circles and rapidly progressing weakness followed by prostration, coma, convulsions, and death within a few hours. The mortality is high, but some animals may recover. Reducing the grain intake to less than a pound daily and distributing the daily grain allotment equally in sufficient feeder space so that all lambs have an opportunity to eat may be helpful. A puzzling feature of the trouble is its sporadic occurrence and the frequent failure of attempts to produce it experimentally. Losses are usually reduced to a minimum if the lambs are started on feed slowly, with gradual increases in the grain allowance as the feeding period progresses and the appetite of the animals increases. Self-feeding of lambs with a mixture of grains and ground roughage, the proportion of grain being gradually increased, is often successful. Overeating may also occur when lambs or other animals are turned into grain or corn fields. The use of temporary fences to rotate the fields and restrict the animals to small areas that can be cleaned up in a relatively short time, thus eliminating the possibility of eating too much grain, will reduce losses.
Fattening cattle, dairy cows fed for high production, and calves also suffer from overeating. (See the article on Nutritional Diseases of Cattle, p. 669.)

Horses often overeat if given an opportunity. Various digestive disturbances known collectively as colic are frequently due to overeating or the eating of spoiled feed. Severe attacks may result in disability or even death.

Animals that are kept too fat by heavy feeding do not usually breed normally.

Effects of Undernutrition

Undernutrition results either from a deficiency of essential nutrients in the diet or from a lack of sufficient total feed to maintain body processes. Undernutrition of the first type involves the so-called nutritional diseases previously described, while that of the second type is frequently spoken of as starvation. A deficiency of a single essential factor such as phosphorus, however, often produces clinical results very similar to complete starvation. Starvation is in fact involved, because the animal voluntarily refuses to eat sufficient food and as a result multiple deficiencies develop.

The effect of undernutrition on the body depends on a number of factors, such as the nature of the deficiency; age and kind of animals and whether they are working, lactating, or pregnant; duration of the deficiency; and whether or not the available body stores of essential nutrients have been depleted. Undernutrition with respect to some factors may lead to permanent injury to the body, whereas a moderate energy or protein deficiency, for instance, may slow up growth or cause a loss of weight and yet not be particularly harmful unless continued too long. Animals can never continue to produce economically if the supply of food is insufficient or if specific nutrients are lacking. In such cases, most of the food consumed is expended in maintenance of the body, and little or none is left over for conversion into useful products or growth.

Breeding animals are also injured by undernutrition and often fail to breed regularly. Pregnant cows or ewes about to lamb die from starvation sooner than nonpregnant animals. A high death loss due to weakness of the young animals at birth or to failure of the mother to lactate normally, may also occur among calves, lambs, or colts born to mothers in poor condition. Esplin, Madsen, and Phillips (11) have reported studies on feeding ewe lambs during their first winter as compared with the usual system of grazing on the open range during the entire year. The ewe lambs that had been fed during the winter produced more wool, showed a lower death loss, and had a much higher percentage of lambs at 2 years of age than the range group.

Animals suffering from malnutrition are usually more susceptible to certain infectious diseases and parasitic infestations than well-fed animals, and for this reason, as well as because of the economic factors involved, faulty nutrition should be avoided and corrected whenever possible.
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