6 Minerals

A number of inorganic elements are essential for normal growth and reproduction of animals. Those required in gram quantities are referred to as macrominerals and this group includes calcium, phosphorus, sodium, chlorine, potassium, magnesium, and sulfur. The macrominerals are important structural components of bone and other tissues and serve as important constituents of body fluids. They play vital roles in the maintenance of acid-base balance, osmotic pressure, membrane electric potential and nervous transmission. Those elements required in milligram or microgram amounts are referred to as the trace minerals. This group includes cobalt, copper, iodine, iron, manganese, molybdenum, selenium, zinc, and perhaps chromium and fluorine. Other elements have been suggested to be essential based on studies in other species but these are generally not considered to ever be of practical importance in dairy cattle. The trace minerals are present in body tissues in very low concentrations and often serve as components of metalloenzymes and enzyme cofactors, or as components of hormones of the endocrine system. A factorial approach was used to describe the requirements for both the macro- and trace minerals whenever such an approach could be supported by research data.

Maintenance requirements as described in this model will include the endogenous fecal losses and insensible urinary losses. Though technically not correct, it will also include losses incurred through sweat. The lactation requirement will be defined as the concentration of the mineral in milk multiplied by the 4 percent FCM milk yield. The pregnancy requirement is defined as the amount of mineral retained within the reproductive tract (fetus, uterine contents, and uterus) at each day of gestation. For most minerals, the requirement of the animal pregnant for $<$190 days is small and not considered in the model. The growth requirement is expressed as the amount of mineral retained/kg body weight gained and entered into the model as expected average daily gain (ADG).

The sum of the maintenance, lactation, pregnancy, and growth requirements is the true requirement of the tissues for the mineral, and is referred to as the ''requirement for absorbed mineral.'' The diet must supply this amount to the tissues. Not all the mineral in a diet is available for absorption. Where data permitted, the availability of minerals from forages, concentrates, and inorganic sources was assigned an absorption coefficient. The model evaluates the absorbable mineral content of a diet by determining the available mineral provided by each constituent of the diet and comparing the sum of the amount of mineral available from the diet with the requirement of the animal for absorbed mineral.

For all minerals considered essential, detrimental effects on animal performance can be demonstrated from feeding excessive amounts. Generally, the dietary amount required for optimal performance is well below amounts found to be detrimental to performance. However, toxicity from several of the essential minerals, including fluorine, selenium, molybdenum, and copper are unfortunately problems that can occur under practical feeding conditions. The National Research Council (1980) described signs of toxicosis and the dietary concentrations of minerals that are considered excessive. Certain elements such as lead, cadmium, and mercury are discussed because they should always be considered toxic and are of practical concern because toxicosis from these elements unfortunately occasionally occurs.

Concentrations of mineral elements in both concentrate and forage feedstuffs vary greatly (Adams, 1975; Coppock and Fettman, 1977; Kertz, 1998). Reliable or typical analyses of concentrations of some mineral elements (e.g., chloride and various micromineral elements) in many feedstuffs are unavailable (Henry, 1995c). Also, concentrations among samples of the same feed type may be quite variable depending upon such factors as fertilization and manure application rates, soil type, and plant species (Butler and Jones, 1973). Concentrations in byproducts or coproducts also are variable and influenced by the method of processing to produce the feedstuff. Therefore, laboratory analyses of feeds for macro- and micromineral element content is

critically important for precise and accurate diet formulation to meet requirements at least cost. Laboratory analyses using wet chemistry methods is critical for accurate determination. Near infrared reflectance spectroscopy (NIRS) is not reliable (Shenk and Westerhaus, 1994).

Estimates of mean concentrations and of variation (standard deviations) in mineral element content of many commonly used feedstuffs are given in Table 15-1 of this publication. Compositions of inorganic mineral sources commonly used in diet supplementation are presented in Table 15-3.

MACROMINERALS

Calcium

FUNCTIONS

Extracellular calcium is essential for formation of skeletal tissues, transmission of nervous tissue impulses, excitation of skeletal and cardiac muscle contraction, blood clotting, and as a component of milk. Intracellular calcium, while 1/10,000 of the concentration of extracellular calcium, is involved in the activity of a wide array of enzymes and serves as an important second messenger conveying information from the surface of the cell to the interior of the cell.

About 98 percent of the calcium in the body is located within the skeleton where calcium, along with phosphate anion, serves to provide structural strength and hardness to bone. The other 2 percent of the calcium in the body is found primarily in the extracellular fluids of the body. Normally the concentration of calcium in blood plasma is 2.2 to 2.5 mM (9 to 10 mg/dl, or 4.4 to 5 mEq/L) in the adult cow, with slightly higher values in calves. Between 40 and 45 percent of total calcium in plasma is bound to plasma proteins, primarily albumin, and another 5 percent is bound to organic components of the blood such as citrate or inorganic elements. From 45 to 50 percent of total calcium in plasma exists in the ionized, soluble form; the amount being closer to 50 percent at low blood pH and closer to 45 percent when blood pH is elevated. The ionized calcium concentration of the plasma must be maintained at a relatively constant value of 1 to 1.25 mM to ensure normal nerve membrane and muscle end plate electric potential and conductivity, which has forced vertebrates to evolve an elaborate system to maintain calcium homeostasis. This system attempts to maintain a constant concentration of extracellular calcium concentration by increasing calcium entry into the extracellular fluids whenever there is a loss of calcium from the extracellular compartment. When the loss of calcium exceeds entry, hypocalcemia can occur and this results in loss of nerve and muscle function, which can in some instances lead to recumbency and the clinical condition referred to as milk fever. During vitamin D intoxication, calcium enters the extracellular compartment faster than it leaves resulting in hypercalcemia, which can lead to soft tissue deposition of calcium.

CALCIUM HOMEOSTASIS

Calcium leaves the extracellular fluids during bone formation, in digestive secretions, sweat, and urine. An especially large loss of calcium to milk occurs during lactation in the cow. Calcium lost via these routes can be replaced from dietary calcium, from resorption of calcium stored in bone, or by resorbing a larger portion of the calcium filtered across the renal glomerulus, i.e., reducing urinary calcium loss. Whenever the loss of calcium from the extracellular fluids exceeds the amount of calcium entering the extracellular fluids there is a decrease in the concentration of calcium in plasma. The parathyroid glands monitor the concentration of calcium in carotid arterial blood and secrete parathyroid hormone when they sense a decrease in blood calcium. Parathyroid hormone immediately increases renal reabsorption mechanisms for calcium to reduce the loss of urinary calcium, and will stimulate processes to enhance intestinal absorption of calcium and resorption of calcium from bone.

Ultimately dietary calcium must enter the extracellular fluids to permit optimal performance of the animal. Calcium absorption can occur by passive transport between epithelial cells across any portion of the digestive tract whenever ionized calcium in the digestive fluids directly over the mucosa exceeds 6 mM (Bronner, 1987). These concentrations are reached when calves are fed all milk diets and when cows are given oral calcium drenches for prevention of hypocalcemia (Goff and Horst, 1993). In nonruminant species, studies suggest that as much as 50 percent of dietary calcium absorption can be passive (Nellans, 1988). It is unknown how much passive absorption of calcium occurs from the diets typically fed to dairy cattle but the diluting effect of the rumen would likely reduce the degree to which passive calcium absorption would occur. Active transport of calcium appears to be the major route for calcium absorption in mature ruminants and this process is controlled by 1,25-dihydroxyvitamin D, the hormone derived from vitamin D. By carefully regulating the amount of 1,25-dihydroxyvitamin D produced, the amount of dietary calcium absorbed can be adjusted to maintain a constant concentration of extracellular calcium (DeLuca, 1979; Bronner, 1987; Wasserman, 1981).

When dietary calcium is insufficient to meet the requirements of the animal, calcium will be withdrawn from bone to maintain a normal concentration of extracellular calcium. If dietary calcium is severely deficient for a prolonged period the animal will develop severe osteoporosis to the point of developing fractures—still, because the desire to maintain a normal concentration of extracellular calcium

is so strong, plasma calcium will only be slightly lower than normal. A sudden large increase in loss of calcium from the extracellular pool can result in acute hypocalcemia before the calcium homeostatic mechanisms can act. This is discussed further in the section on milk fever (Chapter 9).

REQUIREMENT FOR ABSORBED CALCIUM

The amount of calcium that must enter the extracellular compartment for maintenance, growth, pregnancy, and lactation is fairly well known and essentially the same equations were used to predict these amounts as were used in the 1989 National Research Council publication *Nutrients Requirements of Dairy Cattle*.

Maintenance For maintenance of nonlactating cattle, the absorbed calcium required is 0.0154 g/kg body weight (Visek et al., 1953; Hansard et al., 1957). For lactating animals the maintenance requirement is increased to 0.031 g/kg live BW (Martz et al., 1990). The increase in lactating cows reflects the impact increased dry matter intake (DMI) has on intestinal secretion of calcium during digestion.

Growth Growth of cattle requires more calcium when animals are young and actively accruing bone and less as they reach mature skeletal size. The Agricultural and Food Research Council (1991) developed an allometric equation to describe the calcium requirement of growing calves which will be adopted in this model. The requirement for absorbed calcium/kg average daily gain is:

$$\text{Ca (g/day)} = (9.83 \times (\text{MW}^{0.22}) \times (\text{BW}^{-0.22})) \times \text{WG}$$

where MW = expected mature live body weight (kg), BW = current body weight, and WG = weight gain.

Pregnancy The developing fetus requires a negligible amount of calcium until the last trimester of pregnancy (after day 190 of pregnancy), when the fetal skeleton begins to become calcified. Fetal skeletal calcification is especially great in the last weeks before parturition. The absorbed calcium required to meet the demands of the uterus and conceptus is best described by the exponential equation of House and Bell (House and Bell, 1993) for any given day of gestation beyond day 190 as:

$$\text{Ca (g/day)} = 0.02456\ e^{(0.05581 - 0.00007\ t)t} - 0.02456\ e^{(0.05581 - 0.00007(t-1))(t-1)}$$

where t = day of gestation.

Lactation The amount of calcium/kg milk produced varies slightly with the amount of protein in the milk which in turn varies with breed. The absorbed calcium required/kg milk produced is 1.22 g for Holstein cows, 1.45 g for Jersey cows, and 1.37 g for other breeds. Cows require about 2.1 g absorbed Ca/kg of colostrum produced.

CALCIUM ABSORPTION COEFFICIENT

The amount of calcium that must be fed to meet the requirement for absorbed calcium is dependent on the availability of calcium from the feedstuffs and inorganic calcium sources in the diet, and the efficiency of intestinal calcium absorption in the animal being fed. The amount of calcium absorbed from the diet will generally equal the requirement of the body for calcium if the diet contains enough available calcium. The proportion of dietary calcium absorbed will decrease as dietary calcium increases above requirement of the tissues for absorbed calcium. To truly determine the efficiency of absorption of calcium from a feedstuff, the animals being tested should be fed less total dietary calcium than the amount of absorbed calcium required to meet their needs. This will ensure that intestinal calcium absorption mechanisms are fully activated so that the animal will absorb all the calcium from the feedstuff that it possibly can. Few studies fulfill this requirement; thus, it is likely that the published data have underestimated the availability of calcium in many cases. Previous National Research Council (1978, 1989) publications have determined a single efficiency of absorption of dietary calcium regardless of the source of calcium. This absorption coefficient was 0.38 in the 1989 *Nutrient Requirements of Dairy Cattle* and 0.45 in the 1978 *Nutrient Requirements of Dairy Cattle* based on the average proportion of calcium absorbed during a variety of trials. The coefficient was reduced in the 1989 *Nutrient Requirements of Dairy Cattle* partly in response to reports that cows in early lactation were less able to utilize dietary calcium (Van't Klooster, 1976; Ramberg, 1974) making use of a lower coefficient for calcium absorption more prudent. The decision to utilize 0.38 as the calcium absorption coefficient was based largely on a summary of 11 experiments with lactating dairy cows in which the average percentage of dietary calcium absorbed was 38 (Hibbs and Conrad, 1983). In the majority of these 11 experiments, the cows were fed diets supplying calcium well in excess of their needs placing the cows in positive calcium balance by as much as 20 to 40 g/day. In 3 of the experiments, the cows were in negative calcium balance and the percentage of dietary calcium absorbed was still below 40 percent. In those experiments, alfalfa and/or brome hay were supplying the dietary calcium. The French Institut National de la Recherche Agronomique (1989) used 30 to 35 percent as an estimate of efficiency of absorption for dietary calcium using similar logic. The 1996 *Nutrient Requirements of Beef Cattle* utilized 50 percent as the calcium absorption coefficient. The 1980 United Kingdom Agricultural Research Council (Agricultural Research Council, 1980) chose 68

percent as the coefficient of absorption for calcium; a coefficient considerably higher than the estimate of other groups that had examined dietary calcium requirements of cattle. This number is based on a model that predicts calcium is absorbed by dairy cattle according to need. Using data from a variety of balance studies in which a substantial number of lactating cows was included, the observed efficiency of absorption of calcium (diets utilizing feedstuff and mineral sources of calcium) reached a plateau of about 68 percent. Concern over the validity of this coefficient prompted the Agricultural Research Council (1980) to form a second committee to review the 1980 recommendation for calcium. This technical committee agreed with the use of 68 percent as an estimate of the absorption coefficient to be used in calculating dietary calcium requirements of cattle (AFRC, 1991).

A single coefficient is inappropriate and in this model the coefficient for calcium absorption will be based on the sources of calcium used in the diet. Unfortunately, our knowledge of the efficiency of absorption of calcium from individual feedstuffs is limited. Martz et al. (1990) fed lactating dairy cows two diets with no added mineral sources of calcium in which alfalfa supplied nearly all of the dietary calcium. One diet was 33 percent alfalfa, 39 percent hominy grits and 21.5 percent corn cobs; the second diet was 24 percent alfalfa, 41.5 percent corn silage and 29 percent hominy grits. The diets contained more calcium than suggested by the 1978 National Research Council *Nutrient Requirements of Dairy Cattle* publication and less than suggested by the 1989 National Research Council *Nutrient Requirements of Dairy Cattle*. True absorption of calcium from alfalfa, corrected for endogenous fecal calcium loss, was 25 percent; whereas, from the alfalfa-corn silage ration 42 percent of calcium was truly absorbed. Ward et al. (1972) estimated the efficiency of absorption of calcium from alfalfa ranged from 31 to 41 percent. About 20 to 30 percent of calcium within plants is bound to oxalate which is relatively unavailable to the ruminant (Ward et al., 1979). Studies of Hibbs and Conrad (1983) where cows were in negative calcium balance and were fed only alfalfa or alfalfa/brome diets fit the criteria of determining calcium absorption in animals that are being fed less calcium than they require and in these studies the efficiency of absorption of calcium from alfalfa ranged from 8 to 37 percent. Because alfalfa is a major contributor of calcium in dairy rations, absorption of calcium from alfalfa is used as an estimate of efficiency of absorption of calcium from forages in general. An efficiency of absorption of 30 percent is used in the model for calcium from forages.

Availability of calcium from grains and concentrates has not been determined in ruminants. In nonruminant animals, the availability of calcium from concentrates generally is less than the availability of calcium from an inorganic source such as calcium carbonate (Soares, 1995). The presence of phytate is felt to be a factor impairing absorption in nonruminants. This is not a factor in ruminants. Because oxalate is not as likely in concentrate feedstuffs the proportion of calcium available should be greater than 30 percent. It is possible that it may be comparable to that of the mineral sources of calcium. However the current model uses 60 percent as a conservative estimate of the proportion of calcium available from concentrate feedstuffs based in part on an assumption that the availability is not as high as from calcium carbonate. Efficiency of absorption of calcium from feedstuffs that are not forages (e.g., concentrates) was set at 60 percent.

Most non-forage feedstuffs will contain only small amounts of calcium. However, a notable exception is the calcium soap of palm oil fatty acids, which can be 7 to 9 percent calcium. The fat of this product is approximately 80 percent digestible, and digestion can only occur following dissociation of the calcium from the palmitate in the small intestine. This also implies that 80 percent of the calcium in this feed ingredient is available for absorption. This is in contrast to the work of Oltjen (1975) which suggested that formation of calcium soaps within the rumen impaired calcium absorption necessitating an increase in diet calcium when fat was added to a ration. No effect of added fat on apparent absorption of calcium was observed in the experiments of Rahnema et al. (1994). The model does not include a factor to increase dietary calcium when fat is added to the diet. There may be a need to increase diet magnesium when fat is added to the diet as magnesium must be soluble in the rumen to be absorbed. Since hypomagnesemia can affect calcium metabolism (see Chapter 9) there is an effect of diet fat on calcium metabolism but it is not overcome by adding calcium to the ration.

Calcium within mineral supplements is generally more available than calcium in forages and common feedstuffs (Hansard et al., 1957). Theoretically the factor limiting mineral calcium absorption is the solubility of the calcium from the mineral source. Calcium chloride represents a source of highly soluble calcium. When $^{45}CaCl$ was used as a source of radioactive tracer for calcium absorption studies it was absorbed with >95 percent efficiency in young calves (Hansard et al., 1954). Calcium chloride is assigned an efficiency of absorption coefficient of 95 percent. Estimates of the efficiency of absorption of calcium from calcium carbonate range from 40 percent or 51 percent (Hansard et al., 1957) or up to 85 percent (Goetsch and Owens, 1985). Unfortunately, these studies were conducted using steers, with very low requirements for absorbed calcium. The studies of Hansard et al. (1957) demonstrate that calcium chloride is between 1.2 and 1.32 times more absorbable than calcium carbonate. Therefore, the efficiency of absorption of calcium from calcium carbonate is designated to be 75 percent. The absorption of calcium from various mineral sources is often compared

to the efficiency of absorption of calcium from calcium carbonate. Table 15-4 lists a number of common mineral sources of calcium (including bone meal) and an estimate of the efficiency of absorption of calcium in each source, using data summarized by Soares (1995a) and based on the efficiency of absorption relative to calcium carbonate. The calcium from limestone generally is slightly less available than from pure calcium carbonate and has been assigned an efficiency of absorption coefficient of 70 percent.

EFFECTS OF PHYSIOLOGIC STATE

The amount of available calcium that will actually be absorbed varies with the physiologic state of the animal. Hansard et al. (1954) and Horst et al. (1978) reported that the efficiency of absorption of calcium decreases as animals age. Young animals absorb calcium very efficiently and very old animals absorb calcium poorly. As animals age, there is a decline in vitamin D receptors in the intestinal tract (Horst et al., 1990), which is thought to reduce the ability to respond to 1,25-dihydroxyvitamin D. From the data of Hansard et al. (1954), the difference in efficiency of calcium absorption in beef steers from 1 to 6 years of age is nearly negligible. Age was not included as a factor to adjust dietary calcium requirement in cattle >200 kg body weight. The absorption coefficient for calcium from diets normally fed to calves is high and will be considered to be 90 percent for all calves <100 kg body weight (see calf section, Chapter 10).

In early lactation nearly all cows are in negative calcium balance (Ellenberger et al., 1931; Ender et al. 1971; Ramberg, 1974). As feed intake increases and calcium intake increases most cows go into positive calcium balance about 6 to 8 weeks into lactation (Hibbs and Conrad, 1983; Ellenberger et al., 1931). Cows in the first 10 days of lactation are at greatest risk of being in negative calcium balance (Ramberg, 1974) and some are subclinically hypocalcemic throughout this period (Goff et al., 1996). Ramberg (1974) reported that the rate of entry of calcium into the extracellular fluid pool from the intestine increased about 1.55-fold from the day before parturition until 10 days in milk. Thereafter, the rate of entry of calcium into the extracellular pool from the intestine was not increased any further. Van't Klooster (1976) demonstrated that calcium absorption increased from 22 percent in late gestation to 36 percent by day 8 of lactation after which it remained relatively constant. This represented a 1.6-fold increase in efficiency of calcium absorption over this 8-day period. Regression analysis of data of Ward et al. (1972) predicted that cows need to be fed 5 g Ca/kg milk in early lactation to avoid negative calcium balance. However, there was no evidence to demonstrate that negative calcium balance in early lactation was detrimental to the cow provided the concentration of calcium in plasma remained normal, i.e., lactational osteoporosis ensures adequate entry of calcium from bone into the extracellular calcium pool. During lactational osteoporosis, data of Ellenberger et al. (1931) suggest 800 to 1300 g of calcium are removed from bone to support milk production during early lactation and this calcium is restored to bone during the last 20 to 30 weeks of lactation and the dry period. This could increase the requirement for absorbed calcium in later lactation by as much as 8 g/d to rebuild bone lost during early lactation. No calcium requirement for rebuilding bone is included in the model.

The effects of calcium-to-phosphorus ratio on absorption of calcium and phosphorus was once felt to be important but recent data suggest that the calcium: phosphorus ratio is not critical, unless the ratio is >7:1 or <1:1 (Miller, 1983a; Agricultural Research Council, 1980).

CALCIUM DEFICIENCY

A deficiency of dietary calcium in young animals leads to a failure to mineralize new bone and contributes to retarded growth. Rickets is more commonly caused by a deficiency of vitamin D or phosphorus but a deficiency of calcium can contribute to rickets as well. In older animals a deficiency of dietary calcium forces the animal to withdraw calcium from bone for homeostasis of the extracellular fluids. This causes osteoporosis and osteomalacia in the bones, which makes the bone prone to spontaneous fractures. The concentration of calcium in milk is not altered even during a severe dietary deficiency of calcium (Becker et al., 1933).

EXCESS DIETARY CALCIUM

Feeding excessive dietary calcium is generally not associated with any specific toxicity. Dietary concentrations of calcium >1 percent have been associated with reduced DMI and lower performance (Miller, 1983a) but diets as high as 1.8 percent calcium have been fed with no apparent problems for nonlactating dairy cows (Beede et al., 1991). Feeding excessive calcium could interfere with trace mineral absorption (especially zinc) and replaces energy or protein the animal might better utilize for increased production. Feeding calcium in excess of requirements has been suggested to improve performance, especially when cows are fed corn silage diets. Because calcium is a strong cation, addition of calcium carbonate to diets above that required to meet absorbed calcium needs may be providing a rumen alkalinizing effect to enhance performance.

Phosphorus

Of all dietary essential mineral elements for dairy animals, phosphorus represents the greatest potential risk if

excess is released into the environment contaminating surface waters and causing eutrophication. Accurate and precise management of phosphorus nutrition is crucial to optimize performance and health of dairy animals, and to minimize phosphorus excretion.

PHYSIOLOGIC ROLES

Phosphorus has more known biologic functions than any other mineral element. About 80 percent of phosphorus in the body is found in bones and teeth. It is present in bone, along with calcium, principally as apatite salts, and as calcium phosphate. It is located in every cell of the body and almost all energy transactions involve formation or breaking of high-energy bonds that link oxides of phosphate to carbon or to carbon-nitrogen compounds (such as adenosine triphosphate, ATP). Phosphorus also is intimately involved in acid-base buffer systems of blood and other bodily fluids, in cell differentiation, and is a component of cell walls and cell contents as phospholipids, phosphoproteins, and nucleic acids.

Phosphorus concentrations in blood plasma normally are 1.3 to 2.6 mmol/L (4 to 8 mg/dl; 6 to 8 mg/dl for growing cattle and 4 to 6 mg/dl for adult animals). About 1 to 2 g circulate as inorganic phosphate in blood plasma of a 600-kg animal. Because of greater concentrations in erythrocytes, whole blood contains 6 to 8 times as much phosphorous as plasma. About 5 to 8 g are present in the extracellular pool of a 600-kg cow. The intracellular concentration of phosphorus is about 25 mmol/L (78 mg/dl), and total intracellular phosphorus is about 155 g in a 600-kg cow (Goff, 1998a).

Phosphorus also is required by ruminal microorganisms for digestion of cellulose (Burroughs et al., 1951) and synthesis of microbial protein (Breves and Schroder, 1991). Durand and Komisarczuk (1988) recommended that available phosphorus (from dietary sources and salivary recycling) within the rumen should be at least 5 g/kg of organic matter digested to optimize degradation of cell walls from feeds by microbes. When cattle were fed 0.12 percent dietary phosphorus, ruminal fluid concentration was over 200 mg phosphorus/L, considerably greater than the 20 to 80 mg of phosphorus/L needed for maximum cellulose digestion *in vitro* (Hall et al., 1961; Chicco et al., 1965). This concentration typically is achieved in cattle by salivary recycling of phosphorus and from diets adequate to meet the animal's requirement.

PHOSPHORUS UTILIZATION AND HOMEOSTASIS

Net absorption of phosphorus occurs mainly in the small intestine (Grace et al., 1974; Reinhardt et al., 1988). Only small amounts are absorbed from the rumen, omasum, and abomasum. However, little is known about mechanisms and regulation of absorption anterior to the small intestine (Breves and Schroder, 1991). Absorption is thought to occur mainly in the duodenum and jejunum (Care et al., 1980; Scott et al., 1984). Unlike absorption of calcium, absorption of phosphorus is in direct relation to supply of potentially absorbable phosphorus in the lumen of the small intestine (Care et al., 1980). Presumably, as in nonruminants, absorption occurs via two distinct mechanisms. A saturable vitamin D-dependent active transport system, separate and distinct from the active transport mechanism for Ca, is operative when animals are fed low phosphorus-containing diets. Synthesis of 1,25-dihydroxyvitamin D can be stimulated when blood phosphorus is very low resulting in more efficient absorption (Horst, 1986). Passive absorption predominates when normal to large amounts of potentially absorbable phosphorus are consumed, and absorption is related directly to the amount in the lumen of the small intestine and to concentrations in blood plasma (Wasserman and Taylor, 1976).

Absorbed phosphorus may be retained or secreted (e.g., in milk) for productive functions or secreted into the lumen of the digestive tract for reabsorption or excretion in feces. Homeostasis of phosphorus is maintained predominantly by salivary recycling and endogenous fecal excretion, which are related directly to the amount of dietary phosphorus consumed and absorbed. Concentration of phosphorus in saliva can be 4 to 5 times of that in blood plasma. In cows, between 30 and 90 g of phosphorus is secreted daily into saliva (Reinhardt et al., 1988; Scott, 1988). Almost all phosphorus in saliva is inorganic (Reinhardt et al., 1988), and the amount secreted appears to be regulated by parathyroid hormone (Wasserman, 1981). Inorganic salivary phosphorus is absorbed across the intestine with equal or greater efficiency than dietary phosphorus (Challa et al., 1989).

REQUIREMENT FOR ABSORBED PHOSPHORUS

For the model, the requirement for absorbed phosphorus was factorially derived by summing estimates of true requirements for maintenance, growth, pregnancy, and lactation.

Maintenance Typically, 95 to 98 percent of total phosphorus excretion is in feces. Three fractions are present—that of dietary origin unavailable for absorption or not absorbed, that of endogenous origin which is inevitably excreted (inevitable fecal loss), and that of endogenous origin which is excreted to maintain homeostasis (representing phosphorus absorbed by the intestine in excess of the need to maintain normal blood phosphorus). By definition, the maintenance requirement of phosphorus is the endogenous fecal loss (inevitable fecal loss) when phosphorus supply is just below or just meets the true requirement. In the past, the maintenance requirement was expressed

as a function of body weight (National Research Council, 1989a), based on fecal phosphorus excretion data extrapolated to zero phosphorus intake (Agricultural Research Council, 1980). This was later determined to be an inappropriate approach (Agricultural and Food Research Council, 1991). Other workers suggested that inevitable fecal loss in ruminants was a function of total fecal dry matter (DM) excretion (Conrad et al., 1956; Preston and Pfander, 1964), which reflects the role of the salivary glands in phosphorus metabolism. It follows, therefore, that inevitable fecal loss of phosphorus also is related to DMI. The Agricultural and Food Research Council (1991) hypothesized that inevitable fecal loss of phosphorus is determined mainly by DMI, and not by live body weight. New research was available with cattle illustrating that a conceptually more sound and repeatable approach than expression as a function of body weight is to express maintenance requirement as a function of DMI when, by definition, dietary phosphorus is fed and absorbed very near the true requirement.

Part of the maintenance requirement for absorbed phosphorus of the animal is the inevitable fecal loss associated with microbial cells of the digestive tract which contain phosphorus and are excreted in feces. It is estimated that about half of the inevitable fecal loss of phosphorus is associated with microbial debris, and purines and pyrimidines of nucleic acids. This fraction can vary depending upon fermentability (fermented organic matter) of the diet. However, sufficient data are lacking to quantify this relationship accurately (Kirchgessner, 1993).

Klosch et al. (1997) fed growing bulls (228 or 435 kg BW) diets low (50 percent) or high (80 percent) in concentrates and total phosphorus balance was determined. Net phosphorus retention was <1 g/animal per day, and fecal phosphorus excretion was not influenced by digestibility of organic matter consumed, or body weight. Total fecal phosphorus excretion (phosphorus of dietary origin not absorbed plus that of endogenous origin that was inevitably excreted) averaged 1.0 g/kg of DMI. The absorption coefficient of total dietary phosphorus was assumed to be about 80 percent in the study of Klosch et al. (1997). Therefore, the absorbed phosphorus requirement for maintenance of growing animals was set at 0.8 g/kg of DMI in the current model. An additional 0.002 g/kg BW (Agricultural Research Council, 1980) of endogenous phosphorus loss from urine was considered part of the maintenance requirement for absorbed phosphorus in the model.

Spiekers et al. (1993) fed a low phosphorus (0.21 percent) diet to two groups of lactating dairy cows of similar BW, but differing in daily milk yield (stage of lactation effect) and feed intake. For the two groups total phosphorus intakes were 37 and 21.5 g/day, respectively; and, phosphorus balance was similar and slightly negative, indicating that animals were fed below or very near the true requirement. Total excretion of fecal phosphorus differed between groups (20.3 versus 13.3 g/cow per day) and was 51 percent greater per kg of body weight for cows at high versus low dietary phosphorus. However, calculated as a function of DMI, excretion of fecal phosphorus was 1.20 and 1.22 g/kg DMI per day for the high and low intake groups, respectively. It is estimated that the absorption coefficient of total dietary phosphorus for cows fed very close to the true requirement is 80 percent. Therefore, in the current model the maintenance requirement for nonlactating pregnant and lactating cows was set at 1.0 g/kg of dietary dry matter consumed. A small amount of endogenous phosphorus is inevitably excreted in urine. To account for this, an additional 0.002 g/kg BW (Agricultural Research Council, 1980) is considered as part of the maintenance requirement for absorbed phosphorus in the model.

Growth The requirement for growth is the sum of the amount of absorbed phosphorus accreted in soft tissues plus that deposited in skeletal tissue. An accretion of 1.2 g of phosphorus/kg soft tissue gain was estimated by Agricultural Research Council (1980) and data of Grace (1983) from lambs confirmed this value. However, the majority of phosphorus deposition in growing animals is associated with new bone (hydroxyapatite) growth. Bone contains 120 g of calcium/kg and the theoretic accretion ratio of calcium-to-phosphorus is about 2.1 g calcium-to-1.0 g phosphorus (1.6 mol per 1.0 mol). Using this relationship and the accretion rate in soft tissues, the Agricultural and Food Research Council (1991) developed an allometric equation from data in the literature with growing cattle to describe the requirement for absorbed phosphorus for growth (g/kg average daily gain):

$$P\ (g/day) = (1.2 + (4.635 \times MW^{0.22})(BW^{-0.22}))) \times WG$$

where MW = expected mature live body weight (kg), BW = current body weight, and WG = weight gain.

Because bone is an early maturing component of the body, the allometric equation reflects declining requirement for absorbed phosphorus for growing animals. This equation was used to define the absorbed phosphorus requirement for growing dairy cattle. For example in the model, for an animal with M = 681 kg, the absorbed phosphorus requirements (g/kg average daily gain) ranges from 8.3 g at 100 kg live BW (C) to 6.2 g at 500 kg.

Pregnancy Quantitatively the requirement for phosphorus for pregnancy is low until the last trimester. New information on accretion of phosphorus in conceptuses (fetus, fetal fluids and membranes, placentomes and uterine tissues) of 18 multiparous Holstein cows slaughtered at varying times from 190 to 270 days of gestation was available (House and Bell, 1993). Changes in fetal mass and phosphorus content across the sampling period were similar

to earlier data (Ellenberger et al., 1950). Therefore, the requirement for absorbed phosphorus to meet demands of the conceptus for any day beyond 190 days of gestation is described in the model by the exponential equation:

$$\text{absorbed phosphorus (g/d)} = 0.02743e^{(0.05527-0.000075\ t)t} - 0.02743e^{(0.05527-0.000075\ (t-1))(t-1)};$$

where t = day of gestation (House and Bell, 1993).

Estimates of rates of phosphorus accretion in conceptuses of Holstein cows increase from 1.9 g/d at 190 to 5.4 g/d at 280 days of gestation, respectively. This equation should not be used to predict phosphorus accretion of the conceptus prior to 190 days of gestation. The phosphorus requirement of the conceptus at <190 days of gestation is very small and was set to zero in the model.

Lactation The requirement for absorbed phosphorus (g per day) for lactation is equal to daily milk yield multiplied by the percentage of phosphorus in milk. The phosphorus content of milk ranged from 0.083 to 0.085 percent (Wu et al., 2000), 0.087 to 0.089 percent (Spiekers et al., 1993), and 0.090 to 0.100 percent (Flynn and Power,1985). The value of 0.090 percent (0.90 g of phosphorus per kg of milk) was used to compute requirements for absorbed phosphorus in the model. This is the same as that used by the working groups in Scotland and the United Kingdom (Agricultural and Food Research Council, 1991), France (Gueguen et al., 1989), and Germany (Kirchgener, 1993). In the last edition of this publication (National Research Council, 1989), the requirement of phosphorus for lactation was adjusted depending upon fat content of milk. However, the phosphorus in cows' milk is distributed as: 20 percent esterified to casein; 40 percent as colloidal inorganic calcium phosphate; 30 percent as phosphate ions in solution; and, only about 10 percent associated with the lipid fraction (Jenness and Patton, 1959; Renner, 1983). Therefore, an adjustment based on milk fat content is not of major quantitative and practical significance in defining the phosphorus requirement for lactation of dairy cows.

DIETARY REQUIREMENT AND EFFICIENCY OF ABSORPTION

The dietary requirement is the sum of the requirements for absorbed phosphorus for maintenance, growth, pregnancy, and lactation divided by the absorption coefficient(s) for phosphorus from the diet. The absorption coefficient in the denominator of the factorial equation potentially has more influence on the final computed dietary requirement than any of the single or combined requirement values for absorbed phosphorus. The smaller the absorption coefficient, the greater will be the calculated dietary requirement. In the last edition, an overall absorption coefficient of 50 percent was used (National Research Council, 1989b). Other working groups established overall values of 58 percent (Agricultural and Food Research Council, 1991), 60 percent (NRLO, 1982), 60 percent (Gueguen et al., 1989), and 70 percent (Kirchgessner, 1993). As with calcium, a single overall absorption coefficient was not considered appropriate for all types of feedstuffs, supplemental mineral sources, or diets fed to various classes of dairy animals because of the known variation in absorption coefficients. The model evaluates the absorbable phosphorus content of the diet by determining the phosphorus available for absorption from each ingredient of the diet and comparing the sum of total phosphorus in the diet with the requirement for absorbed phosphorus of the animal.

To accurately determine the true absorption coefficient from a particular feedstuff or mineral source, phosphorus must be fed in an amount less than the animal's true requirement. This is to insure maximum efficiency of absorption of all potentially absorbable phosphorus from that particular source. Also, especially with phosphorus, the amount of endogenous phosphorus recycled via saliva must be taken into account. This is most appropriately done experimentally by quantifying recycling with a tracer (e.g., P^{32}). Most studies do not satisfy these experimental specifications. Thus, the true absorption coefficient is generally unknown and the value given is an underestimation of true absorption. Apparent absorption of phosphorus (or apparent digestibility) determined in many studies is lower (largely because of copious endogenous fecal excretion) and not equivalent to the true absorption coefficient. If apparent absorption estimates are used to compute a dietary requirement, gross over-estimation results.

Based on available data, absorption coefficients of phosphorus used in the model for most feedstuffs commonly fed to cattle of various physiologic states were: 90 percent for calves consuming milk or milk replacer; 78 percent for young ruminating calves 100 to 200 kg body weight. True absorption coefficients for phosphorus from alfalfa hay or corn silage were 67 percent or 80 percent, respectively, for lactating cows yielding about 33.6 kg of 3.5 percent fat-corrected milk and consuming 21.7 kg DM daily (Martz et al., 1990). Using a tracer technique, Lofgreen and Kleiber (1953, 1954) reported the true absorption coefficient of phosphorus in alfalfa hay fed to lambs ranged from 0.81 to 0.96. In the model, absorption coefficients of 64 percent and 70 percent were used for forages and concentrates, respectively.

More complete data are available to estimate absorption coefficients of various potential supplemental mineral sources (Table 15-4). These values were tabulated from Soares (1995b) and Peeler (1972), and other sources in the literature and used in the model. Those values determined with ruminants, and especially with cattle, were given preference whenever possible in tabulation.

Dicalcium phosphate (calcium phosphate dibasic) with a true absorption coefficient of 75 percent in cattle (Tillman and Brethour, 1958; Challa and Braithwaite, 1988), phosphoric acid with true absorption coefficient of 90 percent in cattle (Tillman and Brethour, 1958), and monosodium phosphate with a true absorption coefficient of 90 percent in sheep (Tillman and Brethour, 1958) were taken as reference standards. The absorption coefficients of phosphorus in other mineral sources were set based on these reference standards and data where relative differences in phosphorus absorption among these and other sources were estimated in various experiments (Soares, 1995b).

Because sufficient studies with appropriate tracers are not available to estimate true absorption coefficients for most feedstuffs fed to lactating dairy cattle, an alternate approach would be useful. One such approach involves utilizing experimentally derived phosphorus balance data and the assumption that an accurate estimate of the maintenance requirement for absorbed phosphorus is 1.0 g/kg of DMI (Spiekers et al., 1993) plus endogenous urine output (0.002 g/kg BW; Agricultural Research Council, 1980). A calculated absorption coefficient can be derived as: [true requirement for maintenance (g per day) plus milk phosphorus output (g per day) plus phosphorus balance (g per day)] divided by total phosphorus intake (g per day). The fecal output value from the actual balance determination is ignored because it represents unabsorbed dietary phosphorus plus excess endogenous phosphorus which has been recycled to the digestive tract via saliva and excreted in feces. Using this approach, the calculated absorption coefficients of phosphorus in mixed diets fed to lactating cows ranged from 67 to 100 percent (Morse et al., 1992b; Spiekers et al., 1993; Brintrup et al., 1993; Wu et al., 2000). In each study, two or three different concentrations of dietary phosphorus were fed. Within each study the calculated absorption coefficient declined as the dietary phosphorus concentration increased, as would be expected (Challa et al., 1989). Also, among three studies in which dietary phosphorus concentrations (0.39 to 0.42 percent) most closely supplied the requirement of lactating cows, the calculated absorption coefficients [67 percent, Brintrup et al. (1993); 74 percent, Morse et al. (1992b); 72 percent, Wu et al. (2000)] were similar to the overall absorption coefficient (70 percent) set by the German working group (Kirchgessner, 1993). In the case of Spiekers et al. (1993), in which lactating cows were fed diets with 0.21 percent phosphorus (phosphorus-deficient diet which resulted in slightly negative phosphorus balance) the calculated absorption coefficient was about 100 percent, as would be expected. This relationship is corroborated by regression of the calculated absorption coefficients on dietary phosphorus concentrations ranging from 15 to 62 percent, dry basis. Regression analysis (adjusted for number of experimental observations per treatment mean) was performed with a data set of 71 treatment means from 20 phosphorus balance trials (Hibbs and Conrad, 1983; Martz et al., 1990; Morse et al., 1992b; Spiekers et al., 1993; Brintrup et al., 1993; Wu et al., 2000; Rodriguez, 1998). The regression equation is: calculated absorption coefficient = 1.86696 − 5.01238 (dietary phosphorus percent) + 5.12286 (dietary phosphorus percent)^2; (r^2 = 0.70). Based on the regression equation, the calculated absorption coefficient was 1.0 with 0.22 percent phosphorus and declined to a minimum absorption coefficient of 0.64 with 0.49 percent dietary phosphorus. All of these calculated absorption coefficients are greater than that (0.5) used by the National Research Council (1989).

Efficiency of absorption of phosphorus depends upon a number of factors: age (or body weight) of the animal; physiologic state (e.g., nonlactating versus lactating); amount of DM or phosphorus intake; calcium-to-phosphorus ratio; dietary concentrations of aluminum, calcium, iron, magnesium, manganese, potassium, and fat; intestinal pH; and, source of phosphorus (e.g., forages, concentrates, inorganic mineral supplements, and salivary phosphorus) (Irving, 1964; Peeler, 1972; Agricultural and Food Research Council, 1991; Soares, 1995b).

EFFECT OF INTAKE OF PHOSPHORUS

Efficiency of absorption of phosphorus declines as intake of phosphorus increases in cattle (Challa et al., 1989) and in sheep (Field et al., 1977). However, over a considerable range of phosphorus intakes within recommended amounts the efficiency of absorption (absorption coefficient) from inorganic sources remained high and relatively constant in cattle (83 percent; Challa et al., 1989) and in sheep (74 percent; Braithwaite, 1986). Because salivary phosphorus typically supplies appreciably more (e.g., at least two-fold greater amounts) phosphorus to the lumen of the small intestine than does dietary phosphorus, the efficiency of absorption of salivary phosphorus is important. Salivary phosphorus is in the form of inorganic phosphate salts with sodium and potassium. Over a considerable range of phosphorus intakes in tracer studies, the absorption coefficient of salivary endogenous phosphorus recycled to the small intestine was 68 percent to 81 percent in bull calves (Challa et al., 1989). Excessive dietary phosphorus relative to the requirement reduced the efficiency of absorption of inorganic or salivary phosphorus (Braithwaite, 1983, 1986; Challa et al., 1989).

EFFECT OF DIETARY CALCIUM

Effect of increasing dietary calcium on phosphorus absorption was investigated where dietary calcium-to-phosphorus ratios ranged from 0.6 to 3.6 (Field et al., 1983). Efficiency of absorption of phosphorus in sheep was reduced by 18 percent with increasing amounts of calcium;

amounts of calcium and phosphorus were within those amounts recommended by Agricultural Research Council (1980). At higher than recommended supplemental calcium, greater depression of phosphorus absorption would be expected (Agricultural and Food Research Council, 1991). Phosphorus deficiency was exacerbated in lambs fed diets supplying 1.5 times daily requirements for calcium (Sevilla and Ternouth, 1981), likely a result of reduced soluble phosphorus in the digestive tract (Wan-Zahari et al., 1990).

PHYTATE PHOSPHORUS

About two-thirds or more of phosphorus in cereal grains, oilseed meals, and grain by-products is bound organically in phytate; stems and leaves of plants contain very little phytate phosphorus (Nelson et al., 1976). Phytate phosphorus is only slightly available or totally unavailable to nonruminants (Soares, 1995b; National Research Council, 1998). However, inherent phytase activity of ruminal microorganisms renders nearly all of the phytate phosphorus available for absorption (Reid et al., 1947; Nelson et al., 1976; Clark et al., 1986; Morse et al., 1992a; Ingalls and Okemo, 1994; Herbein et al., 1996).

VARIATION IN PHOSPHORUS CONTENT OF FEEDS

Phosphorus is the most expensive macromineral element supplemented in diets of dairy cattle. Therefore, laboratory analyses of feeds for phosphorus content is critically important for precise and accurate diet formulation to meet requirements at least cost. There is considerable variation in actual phosphorus content within types of forages and concentrates fed to dairy animals (Adams, 1975; Kertz, 1998). Estimates of variation (standard deviations) in phosphorus content of many commonly used feedstuffs are given in Table 15-1 of this publication.

GROWTH AND MILK YIELD RESPONSES TO VARYING DIETARY PHOSPHORUS CONCENTRATIONS

In addition to the factorial approach for deriving the absorbed and dietary requirements, results of feeding trials in which varying dietary concentrations of phosphorus were fed to growing calves and lactating cows were evaluated.

GROWING CALVES

Huffman et al. (1933) concluded that 0.20 percent dietary phosphorus was not sufficient for growth of dairy heifers from 3 to 18 months of age. Maximum weight gains of dairy calves from 90 to 125 kg BW occurred when dietary phosphorus content was 0.24 percent, dry basis (Wise et al., 1958). However, bone ash content was greater when dietary phosphorus was 0.33 percent compared with 0.24 percent, but greater phosphorus intake did not improve any other performance variables. Noller et al. (1977) found no differences in BW gain, efficiency of converting feed to gain, or concentrations of phosphorus in blood of Holstein heifers gaining between 0.68 to 0.82 kg/head per day when fed diets containing either 0.22 or 0.32 percent phosphorus. In a second trial, 0.32 percent compared with 0.22 percent dietary phosphorus increased concentrations of phosphorus in serum, but no differences in weight gain or efficiency of feed conversion were observed. Increasing dietary phosphorus from 0.24 to 0.31 percent (dry basis) increased DMI, average daily gain, breaking strength of ribs and tibia, and concentrations of inorganic phosphorus in blood plasma of dairy calves (Teh et al., 1982). Langer et al. (1985) compared 0.24, 0.30, and 0.36 percent dietary phosphorus fed to growing calves and found over the 10-week study that 0.30 percent resulted in maximum feed intake, average daily gain, and concentrations of phosphorus in blood plasma; no additional benefits were detected with 0.36 percent phosphorus. Miller et al. (1987) fed diets containing 0.08, 0.14, 0.20, or 0.32 percent phosphorus and concluded, from concentrations of phosphorus in blood plasma and average daily gains, that at least 0.32 percent phosphorus was needed for heifers to gain 0.75 kg per day. Two sources (monoammonium phosphate and dicalcium phosphate) of phosphorus each used to give three dietary phosphorus concentrations (0.26, 0.34, and 0.41 percent, dry basis) were compared with growing dairy calves (Jackson et al., 1988). Increasing dietary phosphorus from 0.26 to 0.34 percent increased feed intake, body weight gain, concentrations of inorganic phosphorus in blood plasma, and bending moment of the tibia and rib. Body weight gain (0.94 kg/head per day) of calves fed 0.34 percent phosphorus was about 13 percent greater than that of calves fed 0.26 percent dietary phosphorus. Only plasma concentration of phosphorus was increased further with 0.41 percent phosphorus compared with lower concentrations. All responses were similar between sources of supplemental phosphorus. Based on all of these studies, 0.30 to 0.34 percent dietary phosphorus was sufficient for normal blood concentrations of phosphorus in blood, maximum average daily gains, and greater bone strength of growing dairy calves.

LACTATING CATTLE

Research literature was reviewed to find all possible results characterizing lactational responses to varying dietary concentrations of phosphorus. Phosphorus is often fed in greater dietary concentrations than needed to meet the requirement established in the current model. Is feeding phosphorus in excess of requirement beneficial?

Nine studies, with dietary phosphorus concentrations ranging from 0.24 to 0.65 percent of dietary dry matter, fed for periods ranging from the first 8 weeks of lactation to as long as three consecutive lactations, with average milk yields ranging from 15 to 40/kg per cow per day were examined to try to answer this question.

Overall, supplying more dietary phosphorus than that calculated to meet the dietary requirement did not increase DMI or milk yield in any of the studies. The study of Kincaid et al. (1981) suggested that increasing dietary phosphorus increased DMI and 3.5 percent fat-corrected milk yield. However, based on the description of the analysis of variance in that paper the data were improperly analyzed, thus invalidating interpretation. In one other study, feed intake and milk yield were lower for cows fed 0.24 versus 0.32 or 0.42 percent phosphorus (Call et al., 1987). Within none of the other studies was DMI or milk yield increased by increasing dietary phosphorus from its lowest concentration to a higher concentration (Stevens et al., 1971; Carstairs et al., 1981; Brodison et al., 1989; Brintrup et al., 1993; Dhiman et al., 1996; Wu and Satter, 2000; Wu et al., 2000).

Milk fat and protein percentages were not affected by concentration of dietary phosphorus in most studies. Milk protein percentage increased as phosphorus increased from 0.32 or 0.42 percent compared with 0.24 percent (Call et al., 1987). Protein content of milk was higher with 0.45 versus 0.35 percent phosphorus in the study of Wu and Satter (2000). Milk fat percentage was higher in year 1 of the study of Brodison et al. (1989) with 0.44 versus 0.35 percent phosphorus, but lower in the study of Brintrup et al. (1993) with 0.33 versus 0.39 percent phosphorus. There were no consistent effects of dietary phosphorus concentration on milk composition among studies.

Concentrations of phosphorus in blood were evaluated in seven of the nine studies. The normal concentrations of inorganic phosphorus in plasma is 4.0 to 6.0 mg/dl for adult cattle (Goff, 1998a). In only one case among all of the studies was phosphorus in blood below the normal range (3.6 mg/dl for cows fed 0.24 percent dietary phosphorus; Call et al., 1987); 0.24 percent did not provide the dietary requirement. In other studies, increasing dietary phosphorus increased the concentration of phosphorus in blood within or above the normal range.

The DMI and milk yield of cows during early lactation were maximized with 0.40 to 0.42 percent dietary phosphorus, and greater concentrations (0.50 to 0.52 percent) did not increase DMI or milk yield (Carstairs et al., 1981; Wu et al., 2000). Milk yield was not affected by the concentration of the phosphorus in the diet during the first month, but from week 5 through 12 of lactation, it tended to be greater with 0.40 percent compared with 0.50 percent phosphorus (Carstairs et al., 1981). For the entire 84-d treatment period, cows fed 0.40 percent phosphorus produced 8 percent more milk than those fed 0.50 percent phosphorus. Feeding 0.42 percent phosphorus to high yielding cows during the first 8 weeks of lactation maximized milk production, and resulted in positive phosphorus balance and normal concentrations of phosphorus concentrations in blood serum (Wu et al., 2000).

Based on results of nine studies, a concentration in the range of 0.32 to 0.42 percent phosphorus for the entire lactation was sufficient, depending upon milk production potential of the cows and nutrition supplied. No benefits on lactational performance of dietary concentrations >0.42 percent phosphorus were reported in any short- or long-term studies which were properly analyzed.

Daily dietary requirement determined by the factorial method is expressed as g per cow per day, and not as a percentage of the diet. Therefore, supplying the requirement requires a reasonably accurate estimate of actual DMI.

FREE-CHOICE PHOSPHORUS

Coppock et al. (1972, 1975) studied the practice of free-choice feeding of phosphorus-containing supplements to dairy heifers and lactating cows to meet requirements when diets were low or marginally deficient in phosphorus or calcium. With heifers there was little relationship between need for the mineral elements and free-choice consumption of dicalcium phosphate or defluorinated phosphate. For lactating cows offered basal diets providing phosphorus and calcium below requirements for 9 and 12 weeks, there was no evidence that cows consumed dicalcium phosphate to correct the deficiency or that appetite for phosphorus and calcium supplements coincided with the animals' nutritional requirements.

PHOSPHORUS DEFICIENCY

Detailed description of occurrence, etiology, clinical pathology, diagnosis, treatment, and prevention of phosphorus deficiency in ruminants has been described by Goff (1998a). Signs of deficiency may occur rather quickly if dietary phosphorus is insufficient. Deficiency is most common in cattle grazing forages on soils low in phosphorus or in animals consuming excessively mature forages or crop residues with low phosphorus content (less than 0.25 percent, dry basis). Nonspecific chronic signs of deficiency include unthriftiness, inappetence, poor growth and lactational performance, and unsatisfactory fertility; but signs are often complicated by coincidental deficiencies of other nutrients such as protein or energy. Animals may be chronically hypophosphatemic (low phosphorus in blood plasma—2 to 3.5 mg/dl), but the concentration of phosphorus in milk remains within the normal range. In severe deficiency cases, bone mineral mass is lost, and bones

become weak. Severe clinical manifestations of phosphorus deficiency include acute hypophosphatemia, rickets in young growing animals, and osteomalacia in adults.

Acute hypophosphatemia (less than 2 mg phosphorus/dl of plasma) may occur if cows are fed marginally low dietary phosphorus and challenged by extra demand for phosphorus in late pregnancy with accelerated fetal growth, especially with twin fetuses, and with colostrum and milk formation during early lactation. The disease usually is complicated with concurrent hypocalcemia, hypomagnasemia, and possibly hypoglycemia.

Concentrations of phosphorus in plasma often fall below the normal range (4 to 6 mg/dl) in the periparturient period. In other mammals, physiologic correction can occur rather rapidly as phosphorus absorption is responsive to renal production of 1,25-dihydroxyvitamin D which is stimulated by very low phosphorus in the blood (Reinhardt et al., 1988; Goff, 1998a). While presumed similar, this response has not been studied in periparturient dairy cows. However, in some cases correction of hypophosphatemia may not occur and may be further complicated if the cow is developing or has severe hypocalcemia because parathyroid hormone is secreted increasing urinary and salivary losses of phosphorus. Secretion of cortisol around parturition also may depress concentrations of phosphorus in plasma. Intravenous calcium to correct hypocalcemia usually results in a rise in phosphorus in plasma because parathyroid hormone secretion is reduced, reducing urinary and salivary loss of phosphorus. It also stimulates resumption of gut motility, recycling of salivary phosphorus, and absorption. Oral or intravenous administration of a soluble form of phosphorus such as sodium monophosphate can help correct hypophosphatemia. In some cows with severe cases of clinical milk fever, protracted hypophosphatemia (phosphorus in plasma <1 mg/dl) occurs with recumbency; even with successful treatment for hypocalcemia, phosphorus in blood remains low. This disorder is not well understood. However, it is unlikely that increasing the amount or concentration of phosphorus in the diet in excess of requirement in late pregnancy or early lactation will correct hypophosphatemia in the periparturient period, as this disorder seems to occur secondary to hypocalcemia.

Rickets results in young growing calves that are fed a deficient diet and have low phosphorus in the blood from a failure of mineralization in osteoid and cartilaginous (growth plate) matrices during bone remodeling. In contrast, osteomalacia occurs over time with phosphorus deficiency (2 to 4 mg/dl in plasma) in mature animals (no active growth plates) with failure of mineralization of the remodeled osteoid matrix. During mineralization phosphate and calcium ions are incorporated into cartilage of physes or the osteoid matrix in a ratio of 6-to-10. If dietary phosphorus is deficient, low concentrations of phosphorus in blood may not allow this process to proceed normally. In the adult, phosphorus in bone released during remodeling is used to maintain concentrations of phosphorus in blood rather than being reincorporated into bone. In young animals, bone cartilage remains unmineralized resulting in bone that can be flexed without breaking.

Animals with a deficiency of phosphorus are not able to detect or sense phosphorus in feeds or supplements (Miller, 1983b). With severe deficiency of phosphorus they can exhibit pica. Clinical signs of phosphorus and copper deficiency are similar, but can be differentiated by concentrations of plasma phosphorus and blood hemoglobin.

PHOSPHORUS TOXICITY

Long-term feeding of excess phosphorus can cause problems of calcium metabolism, inducing excessive bone resorption and urinary calculi, secondary to the elevated concentrations of phosphorus in blood (National Research Council, 1980). Most often, phosphorus toxicity due to high dietary phosphorus is complicated with low dietary calcium, although ruminants can tolerate a wider ratio of calcium-to-phosphorus than nonruminants, as long as phosphorus and calcium are adequate. Supplemental phosphates given in large oral doses are not considered highly toxic, resulting in mild diarrhea and abdominal distress. Dairy cattle are quite adept at excreting excess absorbed phosphorus to maintain concentrations of phosphorus in blood within a normal range via salivary secretion and fecal excretion (Challa et al., 1989). Urinary excretion of phosphorus also may increase, although its quantitative importance is small relative to fecal excretion. Feeding 0.69 percent phosphorus (dry basis) in the diet of Holstein-Friesian cows for 14 weeks prepartum through 22 weeks of lactation caused no problems or signs of toxicity (De Boer et al., 1981). However, over-feeding can be a concern. High (0.64 percent, dry basis) dietary phosphorus reduced apparent absorption of magnesium compared with 0.22 percent phosphorus in pregnant dairy heifers (Schonewille et al., 1994). Additionally, high phosphorus (greater than 80 g/cow per day) in the diet of late pregnant, nonlactating cows increased phosphorus in blood which apparently inhibits production of the active form of vitamin D (Tanaka and Deluca, 1973), consequently increasing the incidences of milk fever and hypocalcemia at parturition (Barton et al., 1978; Reinhardt and Conrad, 1980). Assuming the presence of adequate calcium in the diet, the maximum tolerable concentration of phosphorus in diets for cattle is estimated to be 1.0 percent, dry basis (National Research Council, 1980).

PHOSPHORUS AND REPRODUCTION

Published research reports from 1923 through 1999 were reviewed to assess the effects of dietary phosphorus on reproductive performance of cattle. In some studies, but not all, severe deficiency of dietary phosphorus caused infertility or reduced reproductive performance of cattle (Alderman, 1963; Morrow, 1969; McClure, 1994). Typically, phosphorus concentration was <0.20 percent of dietary DM, the deficient diet was fed for an extended length of time (1 to 4 years), and where measured, feed intake was depressed, causing coincidental deficiencies of energy, protein, and other nutrients. Low BW generally is considered the main cause of reduced reproductive performance in phosphorus-deficient cows (Holmes, 1981). Palmer et al. (1941) showed that reproductive performance of dairy heifers was compromised much more when both dietary protein and phosphorus were deficient, than phosphorus singularly. Little (1975) demonstrated that deficiencies of phosphorus and protein were additive on failure to exhibit first postpartum estrus in grazing multiparous beef cows. However, there is no evidence to support feeding dietary phosphorus in excess of requirements to improve reproductive performance of dairy cattle.

VIRGIN HEIFERS

In growing virgin heifers, experimentally induced reproductive failure caused by a dietary phosphorus deficiency was very difficult to produce. Huffman et al. (1933) found no reproductive problems in dairy heifers fed a diet with 0.20 percent phosphorus. In two trials with growing dairy heifers, increasing dietary phosphorus from 0.22 to 0.32 percent resulted in no improvement in reproductive performance (Noller et al., 1977). With dairy heifers fed diets with 0.13 to 0.22 versus 0.40 percent phosphorus for 5.5 months no differences in estrus exhibition, services per conception, or pregnancy rates were detected (Hecht et al., 1977). Beginning at 7 months of age, Hereford heifers fed 0.16 or 0.40 percent dietary phosphorus for 2 years had similar pregnancy rates (96 versus 100 percent) and percentages of live calves (91 versus 93 percent) (Call et al., 1978). Hurley et al. (1982) examined intensity of estrus in 12- to 16-month old dairy heifers fed diets containing 73, 138, or 246 percent of National Research Council (1978) requirements for phosphorus. Estrous behavior, ovarian activity, and concentration of progesterone and luteinizing hormone in blood serum were not different among heifers fed different amounts of phosphorus. Because heifers are still growing and bone phosphorus is readily available, they apparently can compensate for a short-term (e.g., <2 years) dietary deficiency, thus reproductive performance is not affected.

LACTATING COWS

With lactating dairy cows, evidence from available research to support feeding phosphorus in excess of requirements to improve reproduction is virtually nonexistent. Results of seven studies can be summarized very succinctly (Stevens et al., 1971; Carstairs et al., 1980; Call et al., 1987; Brodison et al., 1989; Brintrop et al., 1993; Wu and Satter, 2000; Wu et al., 2000). All of the various measures of reproductive performance compared within each study were not different due to concentration of dietary phosphorus with one exception. In the study of Stevens et al. (1971), services per conception were greater in the second year for cows fed 0.40 versus 0.55 percent phosphorus, but not in the first year of study.

Among these seven studies, dietary phosphorus ranged from 0.24 to 0.62 percent of dietary DM, length of feeding different dietary phosphorus concentrations ranged from the first 12 weeks of lactation to as long as three consecutive lactations; and, average milk yields ranged from about 15 to almost 32 kg/cow per day. As long as dietary phosphorus was greater than or equal to 0.32 percent, reproductive performance was normal and not improved with greater concentrations of phosphorus.

Cows in some of the studies would not be considered high producing cows by modern standards. However, with Holstein cows yielding an average of 30.8 and 30.5 kg/cow per day of lactation over two lactations and fed 0.35 or 0.45 percent total dietary phosphorus, respectively, days postpartum to first insemination, days not pregnant, and services per conception were not affected by dietary phosphorus concentration (Wu and Satter, 2000). Pregnancy rates at 120 or 230 days of lactation were not different between cows fed 0.35 or 0.45 percent phosphorus for two lactations.

Overall, evidence from the research literature does not support feeding dietary phosphorus at concentrations in excess of those needed to meet dietary requirements to improve reproductive performance. Additional studies with more, higher yielding cows would be useful.

PHOSPHORUS HOMEOSTASIS OF THE PERIPARTURIENT COW

The periparturient dairy cow represents a unique situation with respect to phosphorus homeostasis. Modulation of calcium homeostasis through endocrine regulation is paramount. During mobilization of 10 ions of calcium from bone, six phosphate ions also are released into blood circulation. Indirectly, this serves to increase the pool of phosphorus in blood. In this physiologic circumstance, increasing the supply of potentially absorbable phosphorus via the diet may be of little benefit. Braithwaite (1983) showed in ewes during late pregnant and early lactation that increasing dietary phosphorus (and calcium) did not increase net

retention or utilization of phosphorus. Instead, the increased amount of dietary phosphorus supplied was absorbed with lower efficiency and that which was absorbed appeared as a net increase in salivary phosphorus and endogenous fecal phosphorus, in excess of the animal's requirement. Resorption of phosphorus from bone appeared to occur merely as a consequence of greater demand for calcium in the periparturient period. Because of the high physiologic priority and regulatory mechanisms for calcium homeostasis in the periparturient period, it is doubtful that increasing the supply of dietary phosphorus above that to meet requirements will have any positive benefit. Mineral stores in bone were mobilized during late pregnancy and early lactation, irrespective of rate of phosphorus absorption. These stores were replaced later in lactation as long as intake of phosphorus was sufficient to meet requirements. Similar studies were not found for periparturient dairy cows, but physiologic events are presumed similar.

In the past, the calcium-to-phosphorus ratio was held as an important nutritional consideration in diet formulation and for proper utilization of both elements. This is important only if dietary phosphorus or calcium is deficient. With sufficient dietary phosphorus, wide ranges of the ratio can be tolerated (Agricultural Research Council, 1980; National Research Council, 1980; National Research Council, 1989). This, taken with the fact that the efficiencies of absorption of phosphorus and calcium vary greatly depending upon sources of the elements, provides no support for recommending a specific dietary calcium-to-phosphorus ratio. No differences in milk yield, persistency of milk production, milk composition, or reproductive performance were found in cows during early lactation fed diets with calcium-to-phosphorus ratios of 1-to-1, 4-to-1, or 8-to-1 (Smith et al., 1966), or 3-to-1 or 1.5-to-1 (Stevens et al., 1971). Nonetheless, it is important to insure that dietary requirements of each element are met.

Sodium

Cattle evolved without abundant dietary sodium to meet nutritional needs. Therefore, the body developed a tenacious ability to conserve sodium, via the kidney and efficient absorption from the lower small intestine and large intestine. Dairy cattle utilize dietary sodium very efficiently, but only very small amounts are stored in a form that is readily available for metabolism. Feeding sodium in excess of needs directly results in increased excretion, which may contribute to excess in the environment increasing salinity of water and soil, and toxicity to plants. However, when dietary concentrations of other macromineral electrolyte elements (e.g., chlorine) are fed in excess of requirements, additional dietary sodium improves animal performance.

PHYSIOLOGIC ROLES

Sodium, is the primary extracellular cation (Aitken, 1976). Additionally, 30 to 50 percent of total body sodium is in a non-exchangeable fraction in the crystalline structure of bone (Eldman et al., 1954). It, along with chlorine and potassium in proper concentrations and balance, are indispensable for a number of important physiologic functions. The exchangeable fraction of sodium modulates extracellular fluid volume and acid-base equilibrium (McKeown, 1986). Additionally, heart function and nerve impulse conduction and transmission are dependent on the proper balance of sodium and potassium. Sodium also plays an indispensable role in sodium-potassium adenosine triphosphate enzyme (Na-K ATPase) responsible for creating electric gradients for nutrient transport. The Na-K pump is essential for all eukaryotic cells, enabling transport of glucose, amino acids, and phosphate into cells, and hydrogen, calcium, bicarbonate, potassium, and chloride ions out of cells (Lechene, 1988). Sodium also is a major component of salts in saliva to buffer acid from ruminal fermentation (Blair-West et al., 1970).

By regression, the sodium content of cattle was estimated at 2.01 to 1.68 g/kg over the range of 75 to 500 kg empty body weight; about 0.4 percent of bone tissue is sodium (Agricultural Research Council, 1980). Typical concentrations of sodium in blood plasma are 150 meq/L and 160 to 180 meq/L in saliva. Concentration in milk is between 25 and 30 meq/L; it is increased during mastitis when serum leaks into milk, but not affected appreciably by dietary sodium content (Kemp, 1964; Schellner et al., 1971).

SODIUM UTILIZATION AND HOMEOSTASIS

Absorption occurs throughout the digestive tract, and dietary sodium generally is assumed to be almost completely available. Absorption occurs by an active transport process in the reticulorumen, abomasum, omasum, and duodenum. Passive absorption also occurs through the intestinal wall so there is a tendency towards equal concentrations in intestinal and fecal fluids. However, substantial active absorption against a sizable concentration gradient also occurs in the lower small intestine and large intestine (Renkema et al., 1962). Consequently, relatively little sodium is excreted in feces, especially in sodium-deficit animals. This mechanism helps ensure that ruminants can subsist on feeds relatively low in sodium content over long periods of time.

Sodium concentrations in blood and tissues are maintained principally via reabsorption and excretion by the kidneys. There is close synchrony between the excretion of sodium, and potassium and chloride. Sodium is the central effector of ion excretion and changes in renal reab-

sorption are chief determinants of sodium excretion. Endocrine control via tissue receptors and the renin-angiotensin system, aldosterone, and atrial natriuretic factor monitor and modulate sodium concentrations in various tissues, which consequently control fluid volume, blood pressure, potassium concentrations, and renal processing of other ions. Kidneys are tremendously efficient in reabsorbing sodium when dietary sodium is deficient. When cattle are depleted of sodium, salivary glands decrease secretion of sodium in saliva. The decrease in sodium content is replaced reciprocally by nearly the same concentration of potassium (Van Leeuwen, 1970; Morris and Gartner, 1971).

REQUIREMENT FOR ABSORBED SODIUM

Maintenance As with calcium and phosphorus, the factorial method was used to derive the absorbed sodium requirement. By definition, the maintenance requirement for absorbed sodium is equal to the inevitable losses in feces and urine of animals fed very near their true requirement. In the model, the daily maintenance requirement for absorbed sodium for growing cattle and non-lactating pregnant cows was set at 1.5 grams/100 kg body weight based on the estimates of total inevitable losses of 0.015 grams/kg body weight per day as reported by Todd et al. (1984) and Gueguen et al. (1989). During initial model development this value for maintenance also was tested for lactating cows. However, this resulted in diets containing amounts and concentrations of total dietary sodium which are known to result in signs of sodium deficiency (Babcock, 1905; Mallonee et al., 1982a), subnormal lactational performance (Aines and Smith, 1957; Kemp, 1964; Kemp and Geurink, 1966; Mallonee et al., 1982a), and negative sodium balance (Lomba et al., 1969). Therefore, the maintenance requirement for absorbed sodium for lactating cows was adjusted based on consideration of results from feeding experiments (see below). The maintenance requirement of absorbed sodium for lactating cows was set empirically at 0.038 g/kg of body weight per day. A higher maintenance requirement for lactating animals compared with growing and nonlactating animals seems logical due to the greater rate and extent of dynamics of some basic physiologic functions (e.g., ruminal buffering or systemic acid-base balance) during lactation that are not accounted for directly by the simple factorial calculation. Doubtless, more research estimates of inevitable losses of sodium in feces and urine by all classes of dairy animals could prove very useful in setting the maintenance requirements for absorbed sodium. Sweating, for aid in heat balance, includes secretion of sodium (Jenkinson and Mabon, 1973). At environmental temperatures between 25 and 30°C, an additional 0.10 g of sodium per 100 kg body weight was considered part of maintenance. With environmental temperatures >30°C, maintenance requirement was increased an additional 0.40 g of sodium per 100 kg BW for a total of 0.50 g per 100 kg BW (Agricultural Research Council, 1980).

Growth In the model, the requirement of absorbed sodium for growth was set at 1.40 g/kg of average daily gain for animals weighing between 150 and 600 kg live body weight (Gueguen et al., 1989).

Pregnancy Recent slaughter data are available from 18 multiparous pregnant Holstein cows to quantify the requirement for absorbed sodium of the conceptus during the last trimester (House and Bell, 1993). Quantitative requirements for all mineral elements are negligible to about 190 days of gestation. The sodium requirement of the conceptus is 1.39 g/d from 190 to 270 days of gestation, but should not be used to compute the sodium requirement for days of gestation <190 (House and Bell, 1993).

Lactation The average sodium concentration in milk from several studies was 0.63 g/kg (Agricultural Research Council, 1965), and this was taken as the requirement for absorbed sodium for milk yield. This constitutes a large portion of the total absorbed sodium requirement of sodium for lactating cows.

DIETARY REQUIREMENT AND EFFICIENCY OF ABSORPTION

As with calcium, phosphorus, and magnesium, the dietary requirement for sodium was established by dividing the absorbed sodium requirement by an absorption coefficient. Sodium from typical feeds is solubilized and released in the liquid matrix of digesta and is readily available for absorption. Feedstuffs commonly used in diets for dairy cattle do not contain enough sodium to meet requirements and supplemental sources are typically included. Because fecal excretion of sodium is low, especially when the element is fed below or very near the true requirement, apparent digestibility data are useful to determine the efficiency of absorption. Apparent absorption of sodium by dairy cows fed fresh forages ranged from 77 to 95 percent, with an average of 85 percent (Kemp, 1964). Semipurified diets containing corn silage, solka floc, or corn cobs as the primary fiber sources fed to dairy heifers had apparent absorption coefficients for sodium of 74, 86, and 91 percent, respectively (Martz et al., 1988). Common salts of sodium used in diets for dairy cattle are readily soluble within the digestive tract (Peeler, 1972). Sodium chloride is most often used and the sodium is essentially 100 percent available; efficiency of absorption of sodium from other salts (e.g., sodium bicarbonate) also is considered very high. Agricultural Research Council (1980) estimated that 91 percent of sodium consumed by cattle was absorbed.

Because common feedstuffs contain relatively little sodium, and supplemental inorganic sources with high sodium availability are used, an efficiency of absorption coefficient of 90 percent was used in the model to compute the dietary requirement for sodium with common feedstuffs and mineral sources. However, the sodium in some animal by-product feedstuffs containing bone may be less available as it is tightly bound in the crystalline structure.

LACTATIONAL RESPONSES TO VARYING DIETARY SODIUM CONCENTRATIONS

Aines and Smith (1957) established that sodium was the limiting nutrient in the diet of dairy cows that contained no supplemental sodium chloride. Furthermore, for dairy cows to produce 20 kg of milk, 30 g of sodium chloride per cow was needed, and 15 g was inadequate. Total sodium requirement at that rate of milk production was estimated to be 21.3 g/d per cow. Kemp (1964) calculated a comparable sodium requirement from balance trials of 23.3 g/d. Kemp and Geurink (1966) reported that 0.14 percent sodium in grazed forage was sufficient to support more than 30 kg of milk production per day. However, feeding lactating dairy cows a diet with no supplemental sodium chloride (0.16 percent sodium, dry basis) resulted in marked depressions in DMI and milk yield after just 1 to 2 weeks of feeding (Mallonee et al., 1982a). Sodium balance always was negative in experiments involving lactating dairy cows fed many different diets with <0.2 percent sodium, or when the diets of adult nonpregnant cows contained <0.1 percent sodium, dry basis (Lomba et al., 1969).

Empirical modeling of data from 15 experiments with lactating cows (1,444 cow-period observations) conducted in either cool or warm seasons showed that DMI and milk yield were improved by dietary concentrations of sodium well above those needed to meet requirements (Sanchez et al., 1994a,b). Dry matter intake and milk yield responses over a range of dietary sodium concentrations (0.11 to 1.20 percent, dry basis) were curvilinear, with maximum performance at 0.70 to 0.80 percent sodium, dry basis. However across all experiments, dietary concentrations for sodium, potassium, chloride, calcium, and phosphorus, ranged from below those needed to meet requirements to concentrations considerably higher (e.g., 0.11 to 1.20 percent sodium; 0.66 to 1.96 percent potassium; 0.15 to 1.62 percent chloride; and, 0.33 to 0.65 percent phosphorus, dry basis). Maximum feed intake and milk yield responses at higher concentrations of sodium than needed to meet requirements likely is due to the higher concentrations of other macromineral elements in the diets. There were interactions of sodium by potassium, sodium by chloride, and sodium by phosphorus on DMI indicating that responses to sodium differed, over the range of dietary concentrations of potassium, chloride, and phosphorus. Additionally, interactions of dietary sodium with potassium, chloride, and phosphorus on DMI differed in experiments conducted in the cool versus warm season. It is unknown if maximum performance responses occurring at 0.7 to 0.8 percent dietary sodium would have occurred if the dietary concentrations of other macromineral elements would have been closer to those needed to provide their requirements, or if the high concentrations of sodium (and other elements) may have affected feed intake and (or) digestive physiology, independent of requirements. In hot weather, milk yield and DMI increased when sodium was supplemented to a basal diet (0.18 percent sodium, dry basis) to 0.55 percent total dietary sodium (dry basis) with either sodium chloride or sodium bicarbonate; chlorine was equalized among diets (Schneider et al., 1986).

Few reports of feeding studies with dairy cattle fed graded concentrations of sodium to determine growth responses were found in the literature. Morris and Gartner (1971) considered 3.1 grams of sodium/day adequate for 300 kg bulls gaining 0.9 kg/day. This is estimated to be about 0.05 percent of dietary dry matter intake.

FREE-CHOICE FEEDING OF SODIUM CHLORIDE

Cattle consume salt liberally if given the choice. Smith et al. (1953) found that lactating cows consumed more salt when provided free-choice in granular versus block form, but consumption of block was sufficient to meet needs for lactation.

SODIUM DEFICIENCY

Babcock (1905) fed a diet very low in sodium to dairy cows and described intense craving for salt, licking and chewing various objects, and general pica. Deficiency signs were manifested within 2 to 3 weeks. Sodium deficiency signs may not develop for several weeks to months, depending upon rate of milk production. However, Mallonee et al. (1982a) found when feeding a diet with no supplemental sodium chloride (0.16 percent sodium) that feed intake and milk yield began to decline within 1 to 2 weeks, and pica and drinking of urine of other cows was observed. Although dietary chloride concentration was not measured in the study, potassium chloride was supplemented (1.0 percent total dietary potassium), so chloride deficiency was not the cause of the condition. The condition was reversed as quickly as it was caused by inclusion of sodium chloride in the diet. Other deficiency signs include loss of appetite, rapid loss of body weight; an unthrifty, haggard appearance, lusterless eyes, and rough hair coat (Underwood, 1981). More extreme signs of deficiency include incoordination, shivering, weakness, dehydration, and cardiac arrhythmia leading to death.

SODIUM (SODIUM CHLORIDE) TOXICITY

Demott et al. (1968) fed lactating cows 4 percent sodium chloride in a grain mix at 1 kg of grain for each 2 kg of 4 percent fat-corrected milk yield for 2 weeks without ill effects on milk yield, body weight, or general health. Although total feed intake was not measured, the sodium concentration of the total diet would have been about 0.8 to 1.0 percent, dry basis. High intake of sodium chloride can increase the incidence and severity of udder edema (Randall et al., 1974). Feeding diets with 0.88 percent sodium from sodium chloride or sodium bicarbonate to mid-lactation Holstein cows did not cause toxicity or reduce feed intake and milk yield compared with 0.55 percent sodium (Schneider et al., 1986).

Drinking water that contained 12,000 to 25,000 mg/L sodium chloride by growing cattle produced toxicosis (Weeth et al., 1960; Weeth and Haverland, 1961). Toxicity signs included severe anorexia, reduced water intake, anhydremia, weight loss, and ultimately physical collapse. However, growing cattle tolerated up to 10,000 mg/L sodium chloride in drinking water without ill effects. Jaster (1978) provided drinking water with 0 or 2,500 mg/L sodium chloride for a 28-day period to lactating cows. No changes were noted in feed intake or digestibility, or in macromineral element concentrations in blood or milk. However, milk yield declined and water consumption increased.

A major factor influencing the degree of exhibition of sodium chloride toxicosis is the availability and quality of drinking water. With an adequate supply of good quality drinking water cattle can tolerate large quantities of dietary sodium chloride. The National Research Council (1980) states that the maximum tolerable dietary concentration of sodium chloride is 4.0 percent for lactating cattle (about 1.6 percent sodium, dry basis); this was the highest level tested (Dermott et al., 1968). For other cattle, the maximum tolerable concentration was set at 9.0 percent of dietary DM based on work of Meyer et al. (1955).

Chlorine

The dietary requirements of chlorine for various classes of dairy cattle are the least studied of any of the macromineral electrolyte elements. Nonetheless, its physiologic roles and interrelationships with sodium and potassium are extremely important. Typically, chlorine is provided in the diet in a salt form which is solubilized, releasing the negatively charged chloride ion for absorption. Chloride is functionally important because of its propensity to accept electrons during metabolism.

PHYSIOLOGIC ROLES

Chloride is the major anion in the body involved in regulation of osmotic pressure, making up more than 60 percent of the total anion equivalents in the extracellular fluid. As a strong anion, it always is dissociated in solution. There is a close relationship between chloride, sodium, and potassium to maintain a strong ion difference (Stewart, 1981), and it is essential for transport of carbon dioxide and oxygen. It also is the chief anion in gastric secretions for protein digestion and is accompanied by hydrogen ions in nearly equivalent amounts. It is needed for activation of pancreatic amylase. Typical concentrations of chloride in blood plasma are between 90 and 110 meq/L, 10 to 30 meq/L in ruminal fluid, and 25 to 30 meq/L in milk. The concentration of chloride in cattle was estimated by regression to be between about 1.2 to 1.4 g/kg over the range of 100 to 500 kg empty-body (Agricultural Research Council, 1980).

UTILIZATION AND HOMEOSTASIS

About 80 percent of the chloride entering the digestive tract arises from digestive secretions in saliva, gastric fluid, bile, and pancreatic juice. Chloride is absorbed throughout the digestive tract. It, like sodium, is absorbed mainly from the upper small intestine by passive diffusion following sodium along an electric gradient. Chloride is transported across the ruminal wall to blood against a wide concentration gradient (Sperber and Hyden, 1952). Martens and Blume (1987) showed that chloride was co-transported actively with sodium across the rumen wall, although the exact mechanism is unclear. Appreciable absorption of chloride from gastric secretions (hydrochloric acid) occurs in the distal ileum and large intestine by exchange with secreted bicarbonate. Therefore, in the short term relatively large day-to-day differences in dietary intake of chloride have little effect on the total chloride entering the digestive tract. Excess chloride is excreted mainly in urine and feces, with smaller amounts in sweat mainly as sodium chloride or potassium chloride.

Regulation of the concentration of chloride in extracellular fluid and its homeostasis is coupled intimately to that of sodium, and is controlled precisely. Typically, it is thought that chloride's role in maintaining ionic and fluid balance is passive to that of sodium and potassium. However, Fettman et al. (1984b) showed that during chloride deficiency the ion functioned independently to mediate chloride conservation. Chloride was conserved by reducing excretion at the kidney, and in feces and milk. Chloride intake in excess of needs was excreted mainly in urine of steers and sheep (Nelson et al., 1955). In lactating cows, a significant amount of chloride was excreted in feces (Coppock, 1986). Normally, anion concentration in extracellular fluid is regulated secondarily to cation concentrations, and when the amount exceeds reabsorption capability of the kidney, excess chloride is excreted in urine (Hilwig, 1976). There is a reciprocal relationship between chloride and

bicarbonate ions in the kidney and excretion of each chloride ion is associated with reabsorption of a bicarbonate ion or visa versa, depending upon systemic pH (Fischer et al., 1983). Also, renal excretion of excess sodium is accompanied by excretion of chloride. Chloride excretion also is influenced by bicarbonate ion. If blood bicarbonate rises, a comparable amount of chloride is excreted by the kidneys to maintain systemic acid-base balance. If base or electrolyte cations need to be conserved, chloride excretion is accompanied by ammonium ions.

REQUIREMENT FOR ABSORBED CHLORIDE

Maintenance The inevitable endogenous losses of chloride in feces and urine are about 50 percent higher than that of sodium (Gueguen et al., 1989). Therefore, 2.25 g/100 kg of body weight was used to set the maintenance requirement for absorbed chlorine in the model. More experimental data from dairy animals in different stages of the production cycle and at different levels of performance could be very useful to establish maintenance requirements.

Growth For cattle with live body weights between 150 and 600 kg, the requirement for absorbed chloride for growth was set at 1.0 g/kg of average daily gain (Gueguen et al., 1989).

Pregnancy No research was available to directly establish the requirement for absorbed chloride for pregnancy. However, based on consideration of the daily sodium accretion rate of the conceptus and the fetus separately (House and Bell, 1993), and assuming that the relative proportions of chloride and sodium in the fetus and in a new born calf (41.5 percent chloride and 58.5 percent sodium; Agricultural Research Council, 1980) are similar, the requirement was set at 1.0 g/d from 190 days of gestation to parturition.

Lactation Average concentration of chloride in milk for several studies was 1.15 g/L (Agricultural Research Council, 1965). Chloride exists in milk almost entirely as the free ion (Holt, 1985). Chloride is highest in colostrum, declines rapidly to average concentrations soon after lactation commences, and increases towards the end of lactation (Flynn and Power, 1985). The concentration in milk is independent of dietary intake, except in severe deficiency, and high environmental temperatures increase the chloride content, whereas cold temperatures have the opposite effect. The requirement for chloride for lactation was calculated as 1.15 g/kg of milk produced.

DIETARY REQUIREMENT AND EFFICIENCY OF ABSORPTION

Relatively little research has been done in ruminants to measure the true absorption coefficient for chloride principally due to its widespread commercial availability, good palatability of inorganic sources (e.g., sodium chloride), and relatively low cost. Chloride, like sodium and potassium, from inorganic sources and common feedstuffs is freely released into the liquid phase of the digesta and readily absorbed (Underwood, 1981). Because the main excretory route of excess chloride is in urine, using apparent digestibility data collected from animals fed at or just below their true requirement yields values similar to those representing true absorption. Apparent absorption of chloride in lactating cows fed fresh forages ranged from 71 to 95 percent and averaged 88 percent (Kemp, 1966). This is comparable to other estimates of absorption efficiency of 85 to 91 percent in cattle and sheep fed mixed diets (Agricultural Research Council, 1980). Paquay et al. (1969b) found that apparent digestibility of chloride was not influenced by intake of chloride, but was correlated negatively with intakes of dry matter, energy, and pentosan; and positively correlated with intakes of potassium and nitrogen. Although factors such as lactation, pregnancy, and growth affect the requirement for chloride, these factors do not appear to alter the efficiency of absorption of chloride. The efficiency of absorption of chloride from sodium chloride is considered virtually 100 percent. The relative absorption of chloride from potassium chloride is 95 percent of that from sodium chloride. Overall, the absorption coefficient for chloride in feedstuffs and mineral sources commonly fed to dairy cattle is near or >90 percent (Henry, 1995c). Therefore, in the model an absorption coefficient for chloride of 90 percent was assigned for all types of feedstuffs and common supplemental mineral sources.

LACTATIONAL AND GROWTH RESPONSES TO VARYING CONCENTRATIONS OF DIETARY CHLORIDE

Coppock (1986) provided a thorough review of the estimated requirements of dietary chloride for lactating dairy cows. Coppock et al. (1979) compared 0.18 percent with 0.40 percent total dietary chloride fed to primiparous Holstein cows for the first 11 weeks of lactation. Cows fed the diet with 0.18 percent chloride conserved chloride by dramatically reducing excretion of chloride in urine and feces. Chloride in milk also tended to decline. However, intakes of feed and water, and milk yield and composition, did not differ due to dietary concentrations of chloride. One-half of the cows in each treatment group had free access to a trace-mineral salt block and cows fed the diet low in chloride consumed more of the salt block. Fettman et al. (1984b) fed diets containing 0.10, 0.27, and 0.45 percent chloride for the first 8 to 11 weeks of lactation. Cows fed 0.10 percent chloride rapidly exhibited clinical signs of deficiency and poor performance compared with those fed medium and high concentrations of dietary chlo-

ride. However, health, feed intake, and yield and composition of milk by cows fed the medium and high concentrations of dietary chloride were similar. Coppock (1986) suggested that 0.2 percent chloride in the diet would approximately meet the requirement of a lactating cow. Meeting the true requirement is complicated by the cow's apparent ability to reduce chloride in milk when a low chloride diet is fed. He also suggested a dietary recommendation of 0.25 percent for a cow near zero energy balance, but emphasized that this was likely too low for a cow in peak milk yield and negative energy balance.

Empirical models with a large data set (1,444 cow-period means) showed that increasing dietary chloride over a range of 0.15 to 1.62 percent decreased DMI and milk yield of midlactation cows (Sanchez et al., 1994a). The negative effects of increasing dietary chloride were much more dramatic in hot summer weather than in winter. This is consistent with the results of Escobosa et al. (1984) showing profound exacerbating effects of high dietary chloride on acid-base balance (metabolic acidosis) and lactational performance during heat stress. Loss of chloride from sweating in response to high ambient temperatures is small, compared with losses of potassium and sodium (Jenkinson and Mabon, 1973), and not of quantitative importance in determining the chloride requirement.

Feeding diets with 0.038 percent chloride for 7 weeks to male Holstein calves did not produce clinical deficiency nor depress feed intake, growth rate, or digestibility of feed compared with calves fed 0.50 percent chloride (Burkhalter et al., 1979). Calves fed the low chloride diet adapted by reducing urinary excretion of chloride, and their water intake and urine output were greater than that of calves fed more chloride. In another study, acid-base status of calves fed a low chloride diet (0.038 percent chloride) was characterized (Burkhalter et al., 1980). Sodium bicarbonate was used to equalize the sodium in the low chloride diet. The low chloride diet resulted in lower chloride and potassium in plasma, and increased blood pH, partial pressure of carbon dioxide and bicarbonate, but had no effect on sodium in plasma. However, the mild alkalosis was not severe enough to affect growth and calves adapted to the low intake of chloride. Results of growth performance to varying intermediate dietary concentrations (e.g., between 0.038 and 0.50 percent) of chloride are not available, and therefore, it is uncertain if a dietary concentration of 0.038 percent is adequate if fed for >7 weeks.

If sodium chloride (common salt) is used to meet the sodium requirement the chloride requirement is met or exceeded. However, if sodium bicarbonate or some other sodium-containing salt is used to supply sodium, it may be necessary to meet the chloride requirement with another supplemental source (e.g., potassium chloride). More research to establish the requirements and appropriate dietary concentrations of chloride (and sodium) considering its interrelationships with other nutrients (Sanchez et al., 1994a,b), could greatly reduce the amount supplemented (e.g., sodium chloride) and excreted. Application of manure from cows fed chloride and sodium in excess of animals' needs can increase soil salinity (Coppock, 1986).

Chloride in drinking water also may make a major contribution to intake of chloride and should be considered.

CHLORIDE DEFICENCY

Chloride deficiency was created in young calves (100 kg BW) by feeding 0.063 percent chloride in the diet and removing daily about 600 g of abomasal contents (Neathery et al., 1981). General clinical signs were anorexia, weight loss, lethargy, mild polydipsia, and mild polyuria. In latter stages, severe eye defects and reduced respiration rates occurred, and blood and mucus appeared in feces. Metabolically, chloride deficiency resulted in severe alkalosis and hypochloremia, which manifested secondary hypokalemia, hyponatremia, and uremia. Control calves also had abomasal contents removed daily, but were fed a diet with 0.48 percent chloride, and grew normally and showed no signs of deficiency. During the first 8 to11 weeks of lactation, dairy cows fed low (0.1 percent, dry basis) dietary chloride exhibited dramatic and progressive declines in intakes of feed and water, body weight, milk yield, and electrolyte concentrations in blood serum, saliva, urine, milk, and feces (Fettman et al., 1984b). A significant decline of chloride in blood serum was found within 3 days after switching cows from a diet containing 0.42 percent to a diet with 0.10 percent chloride. Clinical signs of deficiency were manifested as depraved appetite, lethargy, hypophagia, emaciation, hypogalactiae, constipation, and cardiovascular depression. Metabolic alterations were severe primary hypochloremia, secondary hypokalemia, and metabolic alkalosis. Similar deficiency signs were induced by low dietary chloride in other studies (Fettman et al., 1984b, c, d). Chloride deficiency, resulting from an inadequate dietary supply or loss of gastric juices, can lead to alkalosis due to an excess of bicarbonate, because inadequate chloride is partially compensated for by bicarbonate.

CHLORIDE TOXICITY

The amount or concentration of chloride in the diet *per se* to cause toxicity has not been determined. However, the previous discussion of sodium chloride toxicosis (sodium section) is of interest. High systemic concentrations of chloride, in the absence of a neutralizing cation, can cause disturbance of normal acid-base equilibrium (Stewart, 1981; Escobosa et al., 1984). The maximum tolerable concentration of chloride per se in diets for dairy cattle has not been established. However, the maximum tolerable concentration of dietary sodium chloride was set at 4.0

percent (dry basis) for lactating dairy cows (National Research Council, 1980), based on the work of Demott et al. (1968) although only about one-half of the total daily intake was as the grain mix containing 4.0 percent sodium chloride. For nonlactating dairy animals a maximum tolerable concentration of sodium chloride of 9.0 percent was suggested (National Research Council, 1980). Less tolerance is evident when calcium is the cation accompanying chloride (Escobosa et al., 1984).

Potassium

Potassium is the third most abundant mineral element in the body. It must be supplied daily in the diet because there is little storage in the body and the animal's requirement for potassium is highest of all the mineral element cations. Absorbed potassium in excess of requirements is excreted mainly in urine. Application of manures or fertilizers rich in potassium to crop land can result in excess potassium in the environment and very high potassium content of forages. This can cause problems with calcium and magnesium metabolism particularly for periparturient dairy cows, and may cause udder edema.

PHYSIOLOGIC ROLES

Potassium is involved in osmotic pressure and acid-base regulation, water balance, nerve impulse transmission, muscle contraction, oxygen and carbon dioxide transport; in phosphorylation of creatine, pyruvate kinase activity, as an activator or co-factor in many enzymatic reactions, in cellular uptake of amino acids and synthesis of protein, carbohydrate metabolism, and in maintenance of normal cardiac and renal tissue (National Research Council, 1980; Stewart, 1981; Hemken, 1983). It is the major intracellular electrolyte with concentrations in the range of 150 to 155 meq/L. In contrast to sodium and chloride, extracellular concentrations of potassium are very low (about 5 meq/L). Saliva typically contains <10 meq/L, whereas concentrations in ruminal fluid range from 40 to 100 meq/L. Blood plasma contains 5 to 10 meq of potassium per liter. However, the vast majority of potassium in blood is located within red blood cells (Aitken, 1976; Hemken, 1983). Potassium is abundant in soft tissues of the body. The potassium content of cattle from 75 to 500 kg empty body weight estimated by regression ranged between 2.37 and 2.01 g/kg, respectively (Agricultural Research Council, 1980). The concentration of potassium in milk is higher than any other mineral element (about 38 meq/L).

POTASSIUM UTILIZATION AND HOMEOSTASIS

Ruminants evolved on natural diets relatively rich in potassium, but deficient in sodium. Potassium is absorbed primarily in the duodenum by simple diffusion, and some absorption occurs in the jejunum, ileum, and large intestine. The main excretory route of excess absorbed potassium is via the kidneys. This route is primarily under regulation by aldosterone, which increases sodium reabsorption in the kidney with the concomitant excretion of potassium. Blood acid-base status also affects urinary excretion of potassium (McGuirk and Butler, 1980). With the onset of an alkalotic condition, intracellular hydrogen protons are exchanged with potassium in blood plasma as part of the regulatory mechanisms to maintain acid-base equilibrium and blood pH, reducing potassium in blood. A large gradient exists between intracellular renal tubule concentrations of potassium and that of luminal fluid (urine). This gradient affects the passage of potassium from the tubular cells into urine. Some endogenous as well as unabsorbed potassium also may be excreted in feces. Endogenous fecal losses of potassium increase with increasing DMI (Agricultural Research Council, 1980). However, Paquay et al. (1969a) using data from nonlactating and lactating dairy cows fed many different types of diets estimated that on average about 2.2 g of potassium/kg of dietary DM was excreted in feces.

REQUIREMENT FOR ABSORBED POTASSIUM

Maintenance By definition, the maintenance requirement for absorbed potassium is the sum of the endogenous losses in urine and feces when animals are fed very near the true requirement. Estimates of endogenous potassium loss in urine were 0.038 g/kg of body weight and 2.6 g/kg of dietary dry matter in feces in the study of (Gueguen et al., 1989). Therefore, in the model for growing animals and non-lactating pregnant cows the daily maintenance requirement of absorbed potassium was set at 0.038 g/kg of body weight plus 2.6 g/kg of dietary dry matter intake. During model development a value of 2.6 g potassium/kg of dietary dry matter intake was tested as the endogenous fecal loss for lactating cows. However, the computed amount of absorbed potassium for maintenance and the resulting total dietary concentrations and amounts of potassium were too low for optimum feed intake and milk yield based on results of feeding experiments (Dennis et al., 1976; Dennis and Hemken, 1978; Erdman et al., 1980a; Sanchez et al., 1994a,b). Therefore, in the model the maintenance requirement for absorbed potassium of lactating cows was set empirically as 0.038 g/kg body weight (endogenous urinary loss) plus 6.1 g/kg of dietary dry matter (endogenous fecal loss). A higher maintenance requirement for absorbed potassium for lactating cows compared with non-lactating animals is justified based on potassium's role in dynamic processes associated with ruminal function at higher levels of feed intake and maintenance of systemic acid-base balance. Doubtless, more definitive research data

certainly would be useful to estimate the maintenance requirement for absorbed potassium of all classes of dairy animals. In addition, thermoregulation by sweating at higher environmental temperatures is part of the true maintenance requirement. At environmental temperatures between 25°C and 30°C, an additional 0.04 g of potassium/100 kg body weight was considered part of maintenance. At environmental temperatures >30°C, an additional 0.36 g of potassium/100 kg body weight for a total of 0.40 g per 100 kg BW was used in the model.

Growth In the model, the requirement of absorbed potassium for growth was set at 1.6 g/kg average daily gain based on the estimate of Gueguen et al. (1989) for cattle between 150 and 500 kg live weight.

Pregnancy Recent slaughter data are available from 18 multiparous pregnant Holstein cows to quantify the requirement for absorbed potassium for conceptus accretion during the last trimester of pregnancy (House and Bell, 1993). Requirements for all nutrients are negligible up until about 190 days of gestation. The requirement of the conceptus for absorbed potassium is 1.027 g/d from 190 to 270 days of gestation, but should not be used to compute the potassium requirement for days of gestation <190 (House and Bell, 1993).

Lactation The concentration of potassium in milk is quite constant even under conditions of widely varying potassium intakes (Sasser et al., 1966). The average concentration in milk is 0.15 percent, which is greater than any other mineral element. Therefore, the requirement for absorbed potassium was computed as 1.5 g/kg of milk produced.

DIETARY REQUIREMENT AND EFFICIENCY OF ABSORPTION

Because the body does not store potassium, it must be consumed daily. The dietary requirement of potassium was established by dividing the requirement for absorbed potassium by an absorption coefficient. Potassium in feeds exists as simple ions, which typically are released in to the liquid matrix in the lumen of the digestive tract and are readily available for absorption (Emanuele and Staples, 1990, 1991; Ledoux and Martz, 1990). Hemken (1983) indicated that potassium is almost completed absorbed with a true digestibility of 95 percent or greater for most feedstuffs. Because potassium is excreted mainly in urine, urinary excretion and apparent digestibility (apparent absorption) are reliable criteria for estimation of efficiency of absorption. Paquay et al. (1969a) found that the apparent absorption of potassium by dairy cows fed alfalfa silage, clover silage, and cabbage silage ranged from 87 to 94 percent. Apparent absorption was slightly lower in four tropical forages fed to sheep, but efficiency of absorption was not affected by maturity of the forage (Perdomo et al., 1977). Average apparent absorption of potassium in eight forages fed to cattle and sheep was 85 percent (Miller, 1995). Supplemental potassium from inorganic sources such as potassium chloride, potassium carbonate, potassium sulfate, potassium acetate, potassium bicarbonate, dibasic potassium phosphate, and potassium citrate monohydrate are readily available for absorption (Peeler, 1972; Miller, 1995). In the model, an absorption coefficient value of 90 percent for potassium was used for all types of feedstuffs and mineral sources.

GROWTH AND LACTATIONAL RESPONSES TO VARYING CONCENTRATIONS OF DIETARY POTASSIUM

Growth Research delineating the optimum dietary concentration of potassium and requirement of potassium for growing dairy calves is sparse. Growth of dairy calves was maximized with 0.58 percent potassium in the diet and no benefits were noted with higher dietary concentrations (Bigelow et al., 1984). Weil et al. (1988) found no differences in BW gain (average 0.73 kg/calf per day for all dietary potassium concentrations), feed intake, or plasma macromineral concentrations when feeding diets with 0.55, 0.84, 1.02, or 1.32 percent potassium (dry basis) to Holstein and Jersey calves of both sexes starting at 4 weeks of age. In a second study, 16 Holstein calves, blocked by sex, were fed either 0.34 or 0.58 percent dietary potassium from 6 to 14 weeks of age. Average daily gain and feed intake were greater for calves fed 0.58 percent potassium. Weil et al. (1988) concluded that a dietary concentration of potassium between 0.34 and 0.58 percent (dry basis) optimized growth of young dairy calves, but suggested that 0.55 percent be recommended until the potassium requirement is more clearly delineated. Tucker et al. (1991) fed diets with 0.4 or 0.6 percent dietary potassium (supplemented as potassium chloride) and 0 or 2.0 percent sodium bicarbonate (2 by 2 factorial) to growing calves (56 to 70 days of age and 76 kg live BW at initiation of study) and found no effects on feed intake. However, average daily gain increased with higher dietary potassium, and tended to be reduced by addition of sodium bicarbonate. Concentrations of potassium in plasma were increased by higher dietary potassium. Authors suggested that dietary concentrations between 0.4 and 0.55 percent (dry basis) may be optimum for growth of young dairy calves. Feedlot cattle require approximately 0.55 to 0.60 percent potassium (National Research Council, 1996). For cattle under range conditions with slower growth rates than feedlot cattle, 0.3 to 0.4 percent potassium appears adequate.

Lactation The secretion of potassium in milk (1.5 g potassium/L) necessitates higher dietary concentrations for lactating cows compared with growing cattle. Early

research indicated that 0.75 and 0.70 percent dietary potassium (dry basis) were sufficient to meet requirements of early and mid- to late lactation cows, respectively (Dennis et al., 1976; Dennis and Hemken, 1978; Erdman et al., 1980a). Feed intake increased when cows previously fed 0.45 or 0.55 percent potassium were fed 0.66 percent, dry basis (Dennis et al., 1976). In another trial with midlactation cows (average milk yield = 23.0 kg/cow per day), 0.42 percent dietary potassium reduced DMI and milk yield; however, no differences in DMI or milk yield were noted for cows consuming diets with 0.69 or 0.97 percent potassium, dry basis (Dennis and Hemken, 1978). Similarly, during the first 10 weeks of lactation (average milk yield = 29.2 kg/cow per day), feed intake and milk production were similar with potassium concentrations of 0.75 and 0.99 percent, but feed intake declined with 0.51 percent dietary potassium (dry basis) compared with that of cows fed higher concentrations. The major animal response to marginally low concentrations of dietary potassium in these studies was reduced feed intake.

Empirical modeling of data from 15 experiments with midlactation dairy cows (1,444 cow-period observations) conducted in either cool or warm seasons showed that DMI and milk yield were improved with concentrations of dietary potassium well above those needed to meet requirements (Sanchez et al., 1994a,b). Dry matter intake and milk yield responses over a range of dietary potassium concentrations (0.66 to 1.96 percent, dry basis) were curvilinear, with maximum performance when diets contained 1.50 percent potassium, dry basis, in the cool season. In the warm season, DMI and milk yield increased over the range of dietary potassium concentrations in the data set. However, among all experiments in the data set, dietary concentrations for potassium, sodium, chloride, calcium, and phosphorus, ranged from below those needed to meet requirements to concentrations considerably higher (e.g., 0.66 to 1.96 percent potassium; 0.11 to 1.20 percent sodium; 0.15 to 1.62 percent chloride; 0.50 to 1.34 percent calcium; and, 0.33 to 0.65 percent phosphorus, dry basis). Maximum feed intake and milk yield responses at higher concentrations of potassium than needed to meet requirements likely are associated with the higher concentrations of other macromineral elements in the diets. Interactions of potassium by sodium, potassium by chloride, and potassium by calcium on dry matter intake and milk yield indicated that responses to potassium differed, over the range of dietary concentrations of sodium and chloride. Additionally, interactions of dietary potassium with sodium and chloride on dry matter intake differed in cool versus warm season experiments. It is unknown if maximum performance responses occurring at 1.5 percent potassium or greater would have occurred if the dietary concentrations of other macromineral elements would have been nearer those needed to meet dietary requirements. Or, if the high concentrations of potassium (and other elements) may have affected feed intake and (or) other physiologic functions, independent of true requirements. In a winter study in Florida, Mallonee (1984) found no benefit of increasing dietary potassium from 1.07 to 1.58 percent (dry basis) on feed intake or lactational performance of midlactation Holstein cows when diets contained 0.99 percent calcium, 0.43 percent phosphorus, and 0.28 percent magnesium. However, there were interactions on DMI and milk yield of dietary potassium with varying dietary sodium (0.16, 0.42, and 0.70 percent, dry basis).

Diets containing 1.6, 3.1, or 4.6 percent potassium (via supplemental potassium carbonate) were fed to cows during early lactation (Fisher et al., 1994). Feed intake and milk yield were reduced with the 4.6 percent potassium, and water intake, urinary excretion, and total potassium excretion were increased with increasing concentrations of potassium in the diet.

During heat stress, reduced DMI coupled with the requirement for lactation, increased the requirement for potassium for sweating (Johnson, 1967; Jenkinson and Mabon, 1973; Beede et al., 1983) and acid-base maintenance (Beede and Collier, 1986). Feeding higher concentrations of dietary potassium than needed to meet National Research Council (1989) recommendations of lactating cows in thermoneutral environments (0.8 to 1.0 percent potassium, dry basis), increased feed intake and milk yield compared with cows fed lower dietary concentrations (Beede et al., 1983; Schneider et al., 1984; Mallonee et al., 1985; Schneider et al., 1986; West et al., 1987; Sanchez, 1994a). A dietary potassium concentration of 1.5 percent (dry basis) during heat stress maximized lactational performance (Beede and Shearer, 1991). Many, but not all, diets fed to lactating dairy cattle inherently contain basal concentrations of potassium of 1.5 percent or greater and no additional supplementation is necessary.

POTASSIUM DEFICENCY

Signs of severe potassium deficiency were manifested in lactating dairy cattle fed diets with 0.06 to 0.15 percent potassium (Pradhan and Hemken, 1968; Mallonee et al., 1982b). Marked decline in feed and water intake, reduced body weight and milk yield, pica, loss of hair glossiness, decreased pliability of the hide, lower concentrations of potassium in plasma and milk, and higher blood hematocrit readings occurred within a few days to a few weeks after cows were offered the potassium-deficient diets. Rate of occurrence and severity of deficiency signs appear to be related to rate of milk production, with higher yielding cows (secreting more total potassium in milk) affected more quickly and severely than lower yielding cows. With severe potassium deficiency, cows will be profoundly weak or recumbent with overall muscular weakness and poor

intestinal tone (Sielman et al., 1997). In this case, hypokalemia syndrome was associated with treatment of ketosis.

When diets contained 0.5 to 0.7 percent potassium the only apparent sign of deficiency in lactating cows was reduced feed intake with corresponding lower milk yield compared with cows fed adequate potassium. Severe potassium deficiency under most natural conditions is rare. However, marginal deficiency can occur if diets contain predominantly low-potassium feedstuffs and are not supplemented.

POTASSIUM TOXICITY

The dietary concentration of potassium that leads to toxicity is not well defined (Ward, 1966b). Toxicosis is unlikely to occur under natural conditions, but could occur as a result of excess supplementation. Ward (1966a) described death from 501 g of potassium as potassium chloride given by stomach tube to a cow (475 kg body weight); death was apparently from cardiac arrest. It was pointed out that this amount represented approximately the daily amount consumed by similar cows fed 15 kg of alfalfa, apparently without ill effects. Dennis and Harbaugh (1948) administered 182 and 240 g of potassium as potassium chloride without detectable clinical signs of toxicity, but 393 g by stomach tube to cattle weighing about 300 kg resulted in one death, two that required treatment, and two exhibiting no signs of toxicity. The maximum tolerable concentration in the total diet for ruminants was set at 3.0 percent, dry basis (National Research Council, 1980). However, when 4.6 percent dietary potassium (via supplemental potassium carbonate) was fed to cows during early lactation, feed intake and milk yield were reduced, and water intake, urinary excretion, and total potassium excretion were increased (Fisher et al., 1994). High concentrations of dietary potassium, in excess of those needed to meet requirements, also depressed magnesium absorption. Feeding potassium in excess of that needed to meet requirements, can present metabolic and physiologic challenges to cattle, and can increase excretion of potassium into the environment.

DIETARY CATION-ANION DIFFERENCE

Interrelationships of the monovalent macromineral elements—sodium, potassium and chlorine—on dairy cattle performance have been of major interest in recent years. Mongin (1980) reviewed these interrelationships for nonruminants and suggested that the net acid intake could be extrapolated from the difference between macromineral cations (sodium and potassium) and the anion (chloride) in the diet. Considerations are inherently analogous to the strong-ion difference concept of basic acid-base chemistry (Stewart, 1981). In recent years, the dietary cation-anion difference concept has been studied in dairy cattle. More work has addressed diets of late prepartum dairy cows (see Transition Cow section, Chapter 9).

LACTATIONAL RESPONSES

A limited evaluation of cation-anion difference in diets of lactating dairy cattle and calves has been done. In studies with lactating dairy cows the dietary cation-anion difference generally has been expressed as: milliequivalents (sodium + potassium − chloride)/kg of dietary DM. Tucker et al. (1988) evaluated this concept in lactating dairy cows fed diets with −100, 0, +100, and +200 meq/kg of dietary DM. Dry matter intake and milk yield increased in cows fed +200 compared with those fed −100 meq/kg of dietary DM, independent of the effects of the individual elements (sodium, potassium, and chloride) used to alter the dietary cation-anion difference.

Because of the abundance of sodium and potassium relative to chloride in typical diets for lactating cows, the dietary cation-anion difference is rarely < +100 meq/kg of dietary DM. Sanchez et al. (1994b) used data from 10 separate experiments with midlactation cows to develop an empirical regression model characterizing effects of varying dietary cation-anion difference on feed intake and milk yield. The dietary cation-anion difference in experiments in the data set ranged from +58 to +612 meq/kg of dietary DM, averaging 324 meq. Dry matter intake and milk yield both responded in a curvilinear fashion over the range of dietary cation-anion difference. Both response variables were maximized with +380 meq (sodium + potassium − chloride)/kg of dietary DM. However, magnitude in difference of responses was not large (about 0.25 kg/cow per day) between +200 and +500 meq/kg of dietary DM. Many practical diets have cation-anion differences within this range. However, in the regression model from +380 to +612 meq, DMI declined (about 0.5 kg/cow per day), as did milk yield (about 1.0 kg/cow per day). Milk composition was not affected by varying dietary cation-anion difference. The empirical model was tested with independent data (Tucker et al., 1988; West et al., 1991, 1992) and reasonable agreement was found. Similar evaluations of effects of dietary cation-anion difference on performance during early lactation and with higher yielding cows may prove useful.

Escobosa et al. (1984) found that increasing the dietary cation-anion difference from −144 to +350 meq/kg of dietary DM during heat stress increased DMI and milk yield. Similar results were reported subsequently (West et al., 1991), even when different amounts of sodium or potassium were used to achieve the same dietary cation-anion difference (West et al., 1992).

GROWTH RESPONSES

The influence of dietary cation-anion difference on growth of calves has been examined. Xin et al. (1991) studied the effects of −100 and 200 meq [(sodium + potassium) − (chloride + sulfur)]/kg of dietary DM on feed intake, growth, acid-base status, and potential interactions with dietary copper source. Calves were fed dietary treatments beginning 4 to 11 days through 12 weeks of age. Growth rate increased with 200 compared with −100 meq/kg dietary DM, and blood pH was higher with 200 meq at 8 and 12 weeks of age. Copper source did not affect growth rate. In another study, growing dairy calves [(56 to 70 days of age and 79 kg (28 Holsteins) or 54 kg (4 Jerseys) live body weight at initiation of study)] were fed diets varying in cation-anion difference (sodium + potassium − chloride) of 0, 210, 370, and 520 meq/kg dietary DM (Jackson et al., 1992). Feed intake and average daily gain responded quadratically being greatest at 370 meq and lowest with 0 meq. Authors suggested that an optimal dietary cation-anion difference may exist for young growing ruminants. In a follow-up study, feed intake, growth rate, and calcium metabolism were compared for male and female Holstein calves (56 to 70 days and 72 kg average live body weight at the beginning of the study) fed diets with −180 or 130 meq [(sodium + potassium) − (chloride + sulfur)]/kg of dietary DM factorially with 0.42 and 0.52 percent dietary calcium (Jackson and Hemken, 1994). Feed intake did not differ due to dietary treatments. Calves fed the dietary treatment with 130 meq had greater growth rates than those fed diets with −180 meq; dietary calcium had no effect. Urinary calcium excretion was greater for calves fed diets with −180 meq compared with diets with 130 meq. Breaking strength of the 9th rib was greater for calves fed the 130 meq treatment compared with the −180 meq treatment; breaking strength of the 7th rib was greater with both diets that contained higher dietary cation-anion difference and higher dietary calcium. These studies indicate that there likely is an optimum dietary cation-anion difference for growing dairy calves and that a low or negative cation-anion difference may be deleterious to bone strength. Additional studies will be useful to elucidate the effects of dietary cation-anion difference on growth, bone metabolism, and acid-base physiology of growing calves.

Magnesium

Magnesium is a major intracellular cation that is a necessary cofactor for enzymatic reactions vital to every major metabolic pathway. Extracellular magnesium is vital to normal nerve conduction, muscle function, and bone mineral formation. The concentration of magnesium in plasma of cows is normally between 0.75 and 1.0 mmol/L or 1.8 and 2.4 mg/dl. In a 500-kg cow, there is about 0.7 g of magnesium in the blood, 2.5 g of magnesium in all extracellular fluids, 70 g of magnesium inside cells, and 170 g of magnesium within bone mineral (Mayland, 1988). Bone is not a significant source of magnesium that can be utilized in times of deficit, as bone resorption occurs in response to calcium homeostasis, not magnesium status. Maintenance of normal concentration of magnesium in plasma is nearly totally dependent on absorption of dietary magnesium.

ABSORPTION

Magnesium is absorbed primarily from the small intestine of young calves. As the rumen and reticulum develop they become the main, and perhaps the only, site for magnesium absorption (Martens and Gabel, 1986; Martens and Rayssiguier, 1980; Pfeffer et al., 1970). In adult ruminants, the small intestine is a site of net secretion of magnesium (Greene et al., 1983). Magnesium absorption from the rumen is dependent on the concentration of magnesium in solution in the rumen fluid (important to both the active and passive transport of magnesium across the ruminal wall) and the integrity of the magnesium transport mechanism. The magnesium transport system is a sodium-linked active transport process (Martens and Gabel, 1986), which is critical if the dietary concentration of magnesium is low. In preparing the equations to describe the requirement for magnesium in this model it is assumed that the mechanism for the active transport of magnesium across the rumen wall is intact and functioning. It should be kept in mind when formulating rations that this is often not the case. The following sections will help to clarify when interference with rumen magnesium transport can be expected.

FACTORS AFFECTING SOLUBLE CONCENTRATION OF MAGNESIUM IN RUMINAL FLUID

Dietary Magnesium Content Feeding forages with a low magnesium content and inadequate magnesium supplementation of diets will keep the concentration of soluble magnesium low in the rumen. Cool weather, common in spring and fall when pastures are growing rapidly, reduces uptake of magnesium by plant tissue as does potassium fertilization of pastures (Mayland, 1988). Legumes generally contain more magnesium than grasses. Low DMI associated with high moisture diets can also lead to inadequate magnesium in ruminal fluid.

pH of the Rumen Fluid and Magnesium Solubility Magnesium solubility declines sharply as ruminal pH rises above 6.5. Grazing animals tend to have higher ruminal pH because of the high content of potassium in pasture and the stimulation of salivary secretions associated with

grazing. Heavily fertilized, lush pastures are often high in nonprotein nitrogen and relatively low in readily fermentable carbohydrates. The ability of the ruminal microbes to incorporate the nonprotein nitrogen into microbial protein is exceeded and ammonia and ammonium ion build up in the rumen increasing ruminal pH. When high grain rations are fed ruminal fluid pH is often below 6.5 and magnesium solubility is generally adequate. This may explain why magnesium in concentrates is generally more available than the magnesium in forages (Miller et al., 1972).

Forage Forage can often contain 100 to 200 mmol/kg of unsaturated palmitic, linoleic, and linolenic acids, which can form insoluble magnesium salts. Plants also can contain trans-aconitic acid or citric acid. A metabolite of trans-aconitic acid, tricarballylate can complex magnesium and is resistant to ruminal degradation—but its role in hypomagnesemic tetany is unclear (Schwartz et al., 1988).

FACTORS AFFECTING MAGNESIUM TRANSPORT ACROSS THE RUMINAL EPITHELIUM

Dietary Sodium:Potassium Ratio Forages and pastures are generally low in sodium. Adding sodium to the diet can improve transport of magnesium across the ruminal wall when dietary sodium is low—though in high amounts it increases urinary excretion of magnesium so that the benefit to the animal may be negated. Dietary potassium in high concentrations can reduce the absorption of magnesium. Newton et al. (1972) fed lambs either a low potassium diet (0.6 percent potassium) or a high potassium diet (4.9 percent potassium) and found about a 50 percent reduction in apparent magnesium absorption. The exact mechanism for this interference by potassium is unknown, though it is thought to interfere with the sodium-linked transport of magnesium across the ruminal wall by depolarizing the apical membrane of the ruminal epithelium reducing the electric force driving magnesium across the rumen epithelial cells (Martens and Kasebieter, 1983; Leonard-Marek and Martens, 1996). The negative effects of a high potassium diet cannot be overcome by adding extra sodium to the diet (Martens, 1988). Increasing magnesium in the diet cannot overcome the effect of the high potassium diet on the sodium-linked active transport of magnesium. However, increasing magnesium in the diet will allow enough transport of magnesium across the ruminal wall by passive absorption to meet the animal's requirement for absorbed magnesium (Ram et al., 1998; Leonard-Marck and Martens, 1996). Feeding ionophores (monensin, lasalocid) can improve activity of the sodium-linked transport system for magnesium in the rumen, increasing magnesium absorption efficiency about 10 percent (Greene et al., 1986). However, ionophores are not approved for use in many of the animals they could benefit.

Lush High-Moisture Pastures Lush high-moisture pastures increase the rate of passage of material from the rumen. There is some evidence that this prevents the concentration of magnesium within the ruminal fluid from reaching high enough levels (about 11mmol/L in cows) to fully saturate transport sites for magnesium in the rumen (Martens, 1983).

Ingestion of High Amounts of Aluminum Some studies have noted that tetany is more likely when animals ingest large amounts of aluminum as a result of soil contamination of forages. Most studies have found no impairment of magnesium absorption as a result of aluminum ingestion. Aluminum absorbed into the blood can depress parathyroid hormone secretion, which might result in a somewhat reduced concentration of magnesium in plasma. However, aluminum is generally very poorly absorbed from the diet (Fontenot et al., 1989).

Energy Availability Several reports have demonstrated increased utilization of orally administered magnesium when administered with oral glucose suggesting that glucose supplied the ruminal epithelium with a source of energy to power active transport of magnesium. It is also possible that the rapid fermentation of oral glucose lowered rumen pH enough to solubilize more of the magnesium. Similarly adding glucose may have increased ammonia incorporation into microbial protein, reducing the inhibitory effect of ammonia on magnesium transport (Mayland, 1988).

MAGNESIUM REQUIREMENT

A factorial approach was taken to describe the magnesium requirements of dairy cattle. Fecal loss of endogenous magnesium will be considered to be 3 mg/kg body weight for adult cattle and heifers >100 kg BW, based primarily on the data of Allsop and Rook (1972), and in agreement with the figure adopted by the National Research Council's *Nutrient Requirements of Beef Cattle* (1996) and Agricultural Research Council (1980) publications. Obligate urinary loss of magnesium is negligible. In growing heifers the magnesium content of tissue is 0.45 g/kg body weight gain (Agricultural Research Council, 1980). In pregnant animals, the fetal-placental requirement for magnesium is about 0.181 g/day in Holsteins from day 190 until the end of pregnancy (House and Bell, 1993). Grace (1983), however, estimated the requirement of the fetoplacental unit in late gestation was 0.33 g/day. Considering all the problems associated with hypomagnesemia at parturition it was decided to use the higher figure of 0.33 g/day to describe the fetal requirement for magnesium. Colostrum contains about 0.4 g magnesium/kg (Lyford and Huber, 1988) and milk contains about 0.12 to 0.15 g magnesium/kg.

The efficiency of absorption of magnesium as determined by apparent absorption from natural feedstuffs varies from 11 percent to 37 percent with the majority of values falling between 20 and 30 percent (Agricultural Research Council, 1980; Rook et al., 1958; Forbes et al., 1916; Rook and Campling, 1962; Henry and Benz, 1995). Apparent absorption is considered as an estimate of true absorption because few studies have been conducted to determine true absorption of magnesium. In reviewing literature published before 1980, the Agricultural Research Council (page 204) (1980) determined that the average coefficient for absorption of magnesium from a wide variety of natural feedstuffs fed to ruminants averaged 29.4 percent with a standard deviation of 13.5 percent. Because overestimating the efficiency of absorption of magnesium is potentially detrimental, the coefficient of absorption for magnesium from natural feedstuffs was assigned a value of 16 percent, one standard deviation below the mean, as adopted by the Agricultural Research Council (1980); this approach should provide some margin of safety from magnesium deficiency.

Magnesium oxide is the most widely used inorganic source of magnesium in ruminant diets. Studies with cattle have determined that the coefficient for absorption of magnesium from magnesium oxide is between 28 percent and 49 percent (Moore et al., 1971; Storry and Rook, 1963), though in one study with animals that were on pasture that was associated with hypomagnesemia the coefficient of absorption of magnesium from magnesium oxide was between 5 and 10 percent. Ammerman et al., (1972) working with sheep determined that apparent absorption of magnesium from magnesium oxide was 52 percent and true biologic efficiency of absorption was 51 percent. They also determined that the true biologic efficiency of absorption of magnesium sulfate was 57.6 percent and the biologic efficiency of absorption of commercial magnesite (magnesium carbonate) was essentially zero. Reagent grade magnesium carbonate was 43.7 percent biologically absorbable. Most studies express efficiency of absorption of magnesium from other inorganic sources relative to the absorption from magnesium oxide. Unfortunately, the particle size of the magnesium oxide used can have a large effect on absorption of magnesium (Jesse et al., 1981; Schonewille et al., 1992) with solubility and hence absorption being increased greatly with finely ground magnesium oxide. The coefficient of absorption for magnesium from inorganic sources will be set at 50 percent based on magnesium oxide with a particle size where 99 percent of the material is <250 μm in diameter. Magnesium in magnesite and dolomitic limestone should be considered unavailable when formulating dairy rations. Magnesium sulfate and magnesium chloride are much more soluble and available for absorption.

At least some of the variation in estimates of efficiency of absorption of magnesium from both forages and inorganic sources is a result of the experimental diet fed during the trials. Diets high in potassium and nitrogen would result in lower biologic efficiency of absorption of the magnesium. High moisture diets may also reduce absorption of dietary magnesium by the animal. All three are often present in immature forages, especially when animals are at pasture. Because potassium can have such a large effect on magnesium absorption, the coefficient for absorption of magnesium should be decreased when a high concentration of potassium is present in the diet. Greene et al. (1983) determined that in steers fed a diet that contained 0.1 percent magnesium, and 0.6 percent potassium apparent absorption of magnesium was 28.7 percent. Raising dietary potassium to 2.4 percent and 4.8 percent reduced apparent magnesium absorption to 20.9 percent and 7.9 percent, respectively, or about 5 percent for every 1 percent increase in dietary potassium above the potassium requirement. To overcome this 5 percent reduction in the apparent absorption of magnesium caused by potassium theoretically one would need to increase the final concentration of dietary magnesium by 17 percent (5 percent divided by 28.7 percent times 100) to ensure adequate magnesium absorption. Adjusting the absorption coefficient for dietary magnesium for the effect of dietary potassium has not been incorporated into the diet evaluation section of this model as the data fail to allow an adequate equation to be developed. However the problem appears to be pervasive. Blood magnesium concentration should provide a ready index of the adequacy of diet magnesium supply and absorption (Goff, 1998).

Cattle can excrete large amounts of magnesium in the urine so magnesium toxicity is not a practical problem in dairy cattle, although a maximum tolerable level of 0.4 percent was used in previous National Research Council publications. The negative effects of diets high in magnesium are generally restricted to causing a reduction in feed intake (most magnesium salts are not very palatable, especially magnesium sulfate and magnesium chloride) and/or inducing an osmotic diarrhea. Earlier National Research Council publications listed 0.4 percent magnesium in diets as the maximum tolerable level of magnesium based on earlier work where cows fed 0.39 percent magnesium showed no adverse effects (O'Kelly and Fontenot, 1969). However, diets supplemented with magnesium oxide to bring dietary magnesium to 0.61 percent have been used in high concentrate diets to correct milk fat depression without apparent harm except for occasional diarrhea (Erdman et al., 1980b). Diets for dry cows are commonly supplemented with magnesium sulfate and magnesium chloride to raise dietary anion content and magnesium in an effort to control milk fever. Many of these diets will exceed 0.4 percent magnesium with no

adverse effect other than a possible reduction in feed intake (van Mosel et al., 1990).

Young calves fed 1.3 percent magnesium had lower feed intake and weight gain and diarrhea with mucus in feces (Gentry et al., 1978). Steers fed 2.5 or 4.7 percent magnesium exhibited severe diarrhea and a lethargic appearance; 1.4 percent magnesium reduced DM digestibility (Chester-Jones et al., 1989).

Sulfur

FUNCTION

About 0.15 percent of the body weight is sulfur. Sulfur is found in the amino acids methionine, cysteine (cystine), homocysteine and taurine; in chondroitin sulfate of cartilage; and in the B-vitamins thiamin and biotin. The disulfide bonds of the sulfur containing amino acids are largely responsible for determining the tertiary structure of proteins. Oxidation of methionine and cysteine causes sulfur to also exist in tissues as the sulfate anion, which influences the acid-base balance status of the animal.

Methionine, thiamin, and biotin cannot be synthesized by cattle tissues. These nutrients must either be supplied in the diet or synthesized by ruminal microbes.

The dietary requirement of sulfur for the cow is primarily to provide adequate substrate to ensure maximal microbial protein synthesis. In general, the sulfur content of feedstuffs is directly related to protein concentration. Corn silage is often low in sulfur (0.05 to 0.10 percent) (Hill, 1985) and protein and is the type of diet in which nonprotein nitrogen and sulfur can be successfully added to enhance microbial protein synthesis. Nonprotein nitrogen, such as urea added to these diets will not be incorporated into microbial protein unless adequate sulfur is present to allow formation of methionine.

Emery (1957a) and Emery et al. (1957b) reported that ruminal microbes produce twice as much cysteine as methionine from inorganic sulfate. Not all bacteria in the rumen utilize all forms of sulfur (Emery et al., 1957a). Bryant (1973) found that the predominant ruminal cellulolytic bacteria, *Fibrobacter succinogenes*, could utilize sulfide or cysteine but not sulfate. Many strains of *Ruminococcus* grew in media containing only sulfide or sulfate sulfur. Elemental sulfur is not well utilized by many ruminal bacteria (Ishimoto et al., 1954).

Sulfur incorporated into microbial protein is absorbed from the small intestine as cysteine and methionine. Some dietary sulfur is absorbed as the sulfate or sulfide anion. Bray and Hemsley, 1969 observed that ^{35}S-sulfide was absorbed more rapidly and efficiently from the rumen of sheep than was sulfate. Sulfate sulfur is absorbed more efficiently in the small intestine (Bird and Moir, 1971).

REQUIREMENT

Growth and production have been used to assess the requirement for dietary sulfur especially in low protein diets where nonprotein nitrogen is utilized. Bouchard and Conrad (1973 a,b) determined that 0.20 percent dietary sulfur, when supplied as inorganic sodium, calcium, potassium, or magnesium sulfate was adequate to sustain maximal sulfur retention (microbial protein synthesis of cystine and methionine) even in midlactation dairy cows producing 30 to 37 kg milk/ day. For efficient utilization of nonprotein nitrogen, the dietary nitrogen:sulfur ratio should be between 10 and 12:1 (Bouchard and Conrad, 1973b). The sulfur requirement is set at 0.20 percent of dietary DM.

FACTORS AFFECTING SULFUR REQUIREMENT

The sulfur-containing amino acids provide a major dietary source of sulfur for the cow and the ruminal microbes. Protection of proteins and amino acids from ruminal degradation could result in less sulfur being available for microbial protein synthesis in the rumen. Protection of proteins or amino acids from degradation in the rumen may help the cow obtain amino acids required for her tissues. However, failure of the rumen microbes to obtain adequate protein from rumen degradable sources may reduce the supply of sulfur from feed protein sources reducing ruminal cellulose digestion (Hunt et al., 1954; Spears et al., 1976) and reduce animal performance.

Methionine, methionine analogs, and sulfate salts are utilized equally well in meeting the dietary sulfur requirements of the cow and ruminal microbes (Bouchard and Conrad, 1973a,b; Bull and Vandersall, 1973; Thomas et al., 1951). Elemental sulfur is much less available, probably because it is not very soluble (Fron et al., 1990). Lignin-sulfonate is also a poorly utilized source of sulfur (Bouchard and Conrad, 1973a).

TOXICITY

Excessive dietary sulfur can interfere with absorption of other elements, particularly copper and selenium (see copper and selenium sections). Acute sulfur toxicity causes neurologic changes, including blindness, coma, muscle twitches, and recumbency (Coghlin, 1944). Post-mortem examination reveals severe enteritis, peritoneal effusion, and petechial hemorrhages in many organs, especially kidneys (Bird, 1972). Often the breath will smell of hydrogen sulfide—which is likely the toxic principal in sulfur toxicosis. Sulfates are less toxic, though they can cause an osmotic diarrhea as the sulfate is only poorly absorbed. Excess sulfate added to rations can reduce feed intake and performance (Kandylis, 1984). Water containing 5,000 mg sodium sulfate/kg (1,100 mg S/kg or 0.11%) reduced feed

and water intake resulting in reduced growth of cattle (Weeth and Hunter, 1971). Recent observations in beef cattle have determined that a polioencephalomalacia-like syndrome can be induced with diets containing 0.5 percent sulfur using sulfate salts as supplemental sulfur sources or from drinking water high in sulfates (Beke and Hironaka, 1991; McAllister et al., 1997). The strong reducing environment within the rumen can reduce dietary sulfate, sulfite, and thiosulfate to sulfide within the rumen (Lewis, 1954).

Sulfate anions have been added to rations of dry cows just before calving to decrease the dietary cation-anion difference of the ration to help prevent milk fever (Chapter 9), often to levels above 0.5 percent sulfur. No toxicities have been reported in dairy cows. However, the deleterious effects sulfur can have on copper and selenium absorption, and the recent reports of polioencephalomalacia in beef cattle fed 0.5 percent sulfur rations (Beke and Hironaka, 1991; McAllister et al., 1997) suggest the maximal tolerable level of sulfur should remain at 0.4 percent of dietary DM, as estimated earlier (National Research Council, 1980).

TRACE MINERALS

Cobalt

FUNCTION

Cobalt is a component of vitamin B_{12} (cobalamin). Ruminal microbes can produce all of the vitamin B_{12} required by the cow provided adequate cobalt is available in the diet. As much as 13 percent of the dietary cobalt will be incorporated into vitamin B_{12} when a cobalt insufficient diet is fed, though in general only 3 percent of dietary cobalt is incorporated into vitamin B_{12} (Smith and Marston, 1970). As dietary cobalt increases the ruminal microbes also produce a number of analogs of vitamin B_{12} which are not physiologically active. The presence of these vitamin B_{12} analogs in liver and blood reduces the utility of vitamin B_{12} determination to assess the status of cobalt (Halpin et al., 1984). However, a vitamin B_{12} content of liver below 0.1 μg/g wet weight is considered indicative of cobalt deficiency (Smith, 1987). A portion of dietary cobalt can be absorbed in the cation form (Smith, 1987); however it has no known function and once absorbed does not appear capable of re-entering the rumen so microbes could utilize it. Most is excreted in the urine and a smaller amount exits with the bile (Underwood, 1981).

Cobalt chloride and nitrate, and cobaltous carbonate and sulfate all appear to be suitable sources of cobalt for ruminants. Cobaltous oxide, which is less soluble is somewhat less available (Henry, 1995). Cobaltous oxide pellets and controlled release glass pellets containing cobalt that remain in the rumen-reticulum have been used successfully to supply cobalt over extended periods of time to ruminants on pasture, though regurgitation can cause loss of some types of pellets (Poole and Connolly, 1967).

DEFICIENCY

Ruminants appear to be more sensitive to vitamin B_{12} deficiency than nonruminants, largely because they are so dependent on gluconeogenesis for meeting needs of tissues for glucose. A breakdown in propionate metabolism at the point where methylmalonyl-CoA is converted to succinyl-CoA may be a primary defect arising from a deficiency of vitamin B_{12}. The appearance of methylmalonic acid in urine may be used as an indicator of a deficiency of vitamin B_{12} (Gawthorne et al., 1971). A deficiency of vitamin B_{12} may limit methionine production and limit nitrogen retention (Gawthorne and Smith, 1974). The advantages and disadvantages of methylmalonic acid and vitamin B_{12} determinations to assess vitamin B_{12} and (or) cobalt status have been reviewed (Mills, 1987). Without cobalt in the diet, production of vitamin B_{12} in the rumen rapidly (within days) declines (Underwood, 1981). Stores of vitamin B_{12} in the liver of adult ruminants are usually sufficient to last several months when they are placed on a cobalt-deficient diet. Young animals are more sensitive to dietary insufficiency of cobalt because they have lower reserves of vitamin B_{12} in the liver. Early signs of a deficiency of cobalt include failure to grow, unthriftiness, and loss of weight (Smith, 1997). More severe signs include fatty degeneration of the liver, anemia with pale mucous membranes (Underwood, 1981), and reduced resistance to infection as a result of impaired neutrophil function (MacPherson et al., 1987; Paterson and MacPherson, 1990).

Although the cow may have adequate stores of vitamin B_{12} to last several months, the ruminal microbes apparently do not. Within a few days of a switch to a diet deficient in cobalt ruminal concentrations of succinate rise. This may be the result of a blockade of microbial conversion of succinate to propionate, or a shift in ruminal bacterial populations toward succinate production rather than propionate production (Kennedy et al., 1996).

REQUIREMENT

The dietary requirement for cobalt was estimated to be 0.11 mg/kg of dietary DM (Smith and Loosli, 1957; Ammerman, 1970), and is based on the amount of cobalt that must be supplied to keep tissue concentrations of vitamin B_{12} above 0.3 μg/L (Marston, 1970). The critical concentration of cobalt in the ruminal fluid to allow production of adequate vitamin B_{12} is about 20 ng cobalt/ml. Ruminal fluid normally contains about 40 ng cobalt/ml (Miller et al., 1988).

Plant and animal derived feedstuffs will generally contain between 0.1 and 0.5 mg cobalt/kg DM. Soils along

the southeastern Atlantic coast are deficient in cobalt and forages grown on these soils may not meet the animal requirement for cobalt (Ammerman, 1970). Alkaline soils or liming of soils can prevent adequate uptake of cobalt by plants (Mills, 1981).

SPECIAL PROPERTIES OF COBALT

Dietary cobalt may also have some effects independent of its necessity for production of vitamin B_{12}. Cobalt fed at 0.25 to 0.35 mg/kg of dietary DM, well above those required for sufficient vitamin B_{12} synthesis, seems to enhance ruminal digestion of feedstuffs, especially lower quality forages (Lopez-Guisa and Satter, 1992; Saxena and Ranjhan, 1978). This effect may be due to selection of certain microbial populations with a higher cobalt requirement or may be a result of the divalent cobalt cation forming crosslinks between negatively charged bacteria and negatively charged forage particles, which allows bacteria to cling to forage particles more efficiently (Lopez-Guisa and Satter, 1992). Copper, calcium, and magnesium are divalent cations that may have some of the same ability to "cross-bridge" bacteria and forage particles (Somers, 1983; Storry, 1961). Addition of cobalt has been reported to increase total anaerobic bacteria in the rumen by 50 percent and increase lactic acid production in the rumen by 86 percent (Young, 1979). These results suggest that ruminal microbes may require more cobalt than previous research that focused on the ruminant's vitamin B_{12} status would suggest. However, these data do not yet justify increasing dietary cobalt above 0.11 mg/kg of dietary DM.

Phalaris staggers, a neurologic syndrome induced by alkaloids in the grass, *Phalarus tuberosa* (Ulvund, 1985), can be prevented by supplemental cobalt. Cobalt inactivates or interferes with the absorption of this neurotoxin (Lee and Kuchel, 1953).

TOXICITY

Cobalt toxicity causes reduced feed intake, loss of body weight, hyperchromemia, and eventually anemia—signs similar to those seen in cobalt deficiency (National Research Council, 1980; Ely et al., 1948; Keener et al., 1949). Though toxicity in these reports occurred when there was about 30 mg cobalt/kg of dietary DM, the maximal tolerable dietary cobalt concentration has previously been set at 10 mg/kg of dietary DM (National Research Council, 1980).

Copper

FUNCTION

Copper is a component of enzymes such as cytochrome oxidase, necessary for electron transport during aerobic respiration; lysyl oxidase, which catalyzes formation of desmosine cross links in collagen and elastin necessary for strong bone and connective tissues; ceruloplasmin, which is essential for absorption and transport of iron necessary for hemoglobin synthesis; tyrosinase, necessary for production of melanin pigment from tyrosine; and superoxide dismutase, which protects cells from the toxic effects of oxygen metabolites, which is particularly important to phagocytic cell function.

COPPER REQUIREMENTS

Endogenous losses of copper are approximately 7.1 μg/kg body weight (Agricultural Research Council, 1980). Copper content of growing tissues, when the liver is included as part of the carcass, is about 1.15 mg/kg based primarily on studies of sheep and cattle (Grace, 1983; Simpson et al., 1981). In liver, where excess absorbed copper is stored, copper concentrations can be much higher depending on diet. Copper content of colostrum is about 0.6 mg/kg (Lyford and Huber, 1988). Copper content of milk is about 0.15 mg/kg though it can be about 0.2 mg/kg when animals are fed a high copper diet (Schwarz and Kirchgessner, 1978). The requirement for absorbed copper during lactation that was used in the model is 0.15 mg copper/kg milk produced. This is a 50 percent increase over the value of 0.10 mg copper/kg milk produced used by the Agricultural Research Council (1980). In early gestation (<100 days) about 0.5 mg copper is incorporated into fetal, placental, and uterine tissue each day, increasing to between 1.5 and 2 mg/day during the last month of gestation (Agricultural Research Council, 1980; House and Bell, 1993). In the model, the requirement for absorbed copper is set at 0.5 mg/day for cows <100 days in gestation, 1.5 mg/day if gestation is between 100 and 225 days, and 2.0 mg/day if gestation is >225 days (Table 6-1).

EFFICIENCY OF COPPER ABSORPTION FROM DIETS

The amount of dietary copper required to supply the copper needed for maintenance, growth, and lactation will vary with the age of the animal, the chemical form of the dietary copper, and the presence of substances in the diet that interfere with absorption of dietary copper. In newborn calves, up to 70 percent of dietary copper is absorbed, similar to nonruminants. Bremner and Dalgarno (1973 a,b) found that 50 to 60 percent of dietary copper (supplied as copper sulfate) was retained in the liver of calves between 3 and 14 weeks of age. During the first 4 weeks of life (before weaning) the coefficient for absorption assigned to copper was 60 percent. With the development of the rumen there is a tremendous decrease in absorption of copper. Only between 1 and 5 percent of dietary copper will be absorbed by adult cattle. The absorption of dietary

TABLE 6-1 Comparison of Estimated Dietary Copper Requirements (mg/d) and Dietary Copper Concentrations (mg/kg of DM) for Cattle in Various Physiologic States

Cow Description[a]	Feed Intake (kg DM)	1989 NRC mg/day	1989 NRC mg/kg diet	1980 ARC mg/day	1980 ARC mg/kg diet	2000 NRC mg/day	2000 NRC mg/kg diet
300-kg heifer, ADG = 0.7 kg	6	60	10	71	11.8	72.6	12
500-kg heifer, ADG = 0.5 kg, day 250 of gestation	10	100	10	154	15.4	152	15.2
650-kg cow, 40 kg of milk per day	20	200	10	214	10.7	313	15.7
650-kg cow, day 270 of gestation	12	120	10	167	13.9	163.5	13.7

[a]ADG = average daily gain.

copper is reduced by the presence of sulfur and molybdenum in the diet. Sulfur sources can be converted to sulphide within the rumen leading to formation of copper sulphide precipitates rendering the copper unavailable for absorption (Bird, 1970). Allen and Gawthorne (1987) suggest that sulfur and molybdenum form tetrathiomolybdate in the solid phase of the ruminal digesta. Tetrathiomolybdate binds copper to form a highly insoluble complex that renders the copper unavailable for absorption. Suttle and McLauchlan (1976) developed a nomogram which models the effect of dietary sulfur and molybdenum on the efficiency of absorption of dietary copper (CopperAbsorbable) from a diet based on dietary sulfur (g/kg) and molybdenum (mg/kg) concentrations.

$$\log(\text{CopperAbsorbable}) = -1.153 - 0.076(\text{sulfur}) - 0.013(\text{sulfur} \times \text{molybdenum})$$

In Table 6-2, the calculated absorption coefficient for copper decreases from 4.6 percent when dietary sulfur is at the required concentration (0.20 percent), to 3.1 percent when dietary sulfur is at the maximum concentration (0.40 percent) in a diet that contains 1 mg molybdenum/kg.

TABLE 6-2 Calculated Copper Absorption Coefficients Across Various Dietary Sulfur and Molybdenum Concentrations

Dietary Sulfur (g/kg)	Dietary Molybdenum (mg/kg)	Cu absorption coefficient
2.0	1	0.046
4.0	1	0.031
6.0	1	0.021
2.5	0.5	0.043
2.5	1	0.042
2.5	2	0.039
2.5	5	0.0314
2.5	10	0.0217
2.5	20	0.010
2.5	100	0.003

Dietary molybdenum concentrations >10 mg/kg present a major obstacle to absorption of copper.

In the model, the assumption was made that molybdenum content of the diet is 1 mg/kg and dietary sulfur is 2.5 g/kg (0.25 percent sulfur). The coefficient of copper absorption utilized in the model was 4 percent. If the dietary molybdenum or sulfur content differs from these values, the user can adjust the amount of dietary copper required by calculating the proper copper coefficient or referring to Table 6-2, based on the work of Suttle and McLauchlan (1976). The required dietary copper would then be 0.04/"copper absorbable" times the requirement determined by the model, where "copper absorbable" is the calculated absorption coefficient of copper. This adjustment to the copper requirement is the adjustment suggested by the 1980 ARC publication.

However as summarized and discussed by Underwood and Suttle (1999), the relationship between copper availability and dietary copper and molybdenum is probably more difficult to predict than the equation of Suttle and McLauchlan (1976) would suggest. The effect of sulfur and molybdenum varies depending on the feedstuff serving as a source of copper. Underwood and Suttle (1999) conclude that the absorbable amount of copper in ensiled grass was not greatly impaired by an increase in dietary molybdenum but was greatly depressed by the addition of sulfur to the ration. When the diet was 0.2 percent sulfur about 5.5 percent of the copper was available, but when the diet was 0.4 percent sulfur the absorbable copper was reduced to about 1.5 percent. In hays, the inhibitory effect of molybdenum is present but relatively small. As sulfur increases from 0.2 to 0.4 percent in hays the percent absorbable copper decreases by 20–30 percent. The percent absorbable copper in fresh grasses is lower than that of hays or ensiled grasses at any given sulfur or molybdenum concentration and the addition of sulfur or molybdenum drastically decreases copper absorbability. The inhibitory effect of increasing diet molybdenum on copper absorption

is greatest when dietary molybdenum is low and seems to reach a plateau once diet molybdenum is about 4–5 mg/kg DM. Beyond this point higher dietary molybdenum concentrations do not impair copper absorbability significantly further (Underwood and Suttle, 1999; Gengelbach, 1994).

OTHER FACTORS KNOWN TO INFLUENCE THE ABSORPTION OF COPPER

Pasture Grazing Up to 10 percent of the DMI of pastured animals can be from soil ingested as they graze—especially if pastures are short. Suttle et al. (1975) found that inclusion of soil at 10 percent of DM reduced copper absorption by 50 percent, across several different soil types. The coefficient for absorption of copper for animals at pasture should be decreased by one-half to ensure adequate copper supplementation. This is not included in the model, and the user may decide to adjust requirements for copper accordingly, essentially doubling the copper required, as suggested by the model, when animals are at pasture.

High Dietary Zinc Zinc induces increased metallothionein in the intestine. Metallothionein coats the surface of the lumenal side of the intestinal cells and binds and sequesters copper at the lumenal surface. The bound copper is eventually lost to the feces upon desquamation of the intestinal epithelial cells. A fairly strong negative linear relationship was found between dietary zinc and copper retained in the liver of lambs by Bremner et al. (1976). Lambs fed diets with 40 (30 was considered the requirement), 220, or 420 mg zinc/kg retained 4, 2.8, or 1.5 percent of the dietary copper in their liver respectively, suggesting that the coefficient for absorption should be decreased by 16 percent for every 100 mg of zinc per kg diet above 40 mg/kg. However, this adjustment factor was not included in this model. In adult lactating cows, supplementation with 2000 mg of zinc/kg of diet reduced copper in plasma, but 1000 mg of zinc/kg of diet did not (Miller et al., 1989). These data suggest that under practical conditions zinc is not a major factor affecting absorption of copper unless the diet contains at least twenty-fold more zinc that is recommended. In some areas, zinc oxide is fed at $>$ 1000 mg zinc/kg DM in an effort to control facial eczema. However because of the poor solubility of zinc oxide it appears that in most cases this does not induce copper insufficiency (Lee et al., 1991).

High Dietary Iron Copper reserves in liver of calves were found to be depleted when calves were fed diets containing 1400 mg of iron/kg of diet, greater than ten-fold the concentration of iron recommended (Agricultural Research Council, 1980). Increasing diet iron from 500 to 800 mg/kg DM dramaticly reduced the liver copper content from 134 to 16 mg copper/kg DM in 8 weeks (Phillippo et al., 1987). There seems to be an interaction between high dietary iron and sulfur as when both are present the inhibition of diet copper absorbability is more pronounced (Underwood and Suttle, 1999). Water containing large amounts of iron also has been implicated as a cause of copper deficiency but no specific recommendation on how iron affects the coefficient of absorption of copper can be made and no adjustment is included in the model.

High Dietary Calcium Some studies have suggested a decrease in absorption of copper when calcium was added to the diet (Kirchgessner and Weser, 1965; Dick, 1954). In the study of Kirchgessner (1965) increasing calcium from 0.70 percent to 0.95 percent of the diet reduced copper absorption. However, other studies failed to show an effect of calcium on copper status (Huber and Price, 1971) despite larger changes in dietary calcium content. No adjustment of the absorption coefficient for copper was recommended based on dietary calcium.

Differences in Breeds Breed differences exist among cattle that may make some breeds more susceptible to copper toxicity. Jersey cattle fed the same diet as Holstein cattle accumulated more copper in their livers (Du et al., 1996). It is not clear whether this reflects differences in feed intake, efficiency of copper absorption, or biliary excretion of copper. Although these data caution that Jersey cows may be more prone to copper toxicity than Holstein cows, an adjustment of the copper requirement based on breed does not seem warranted at this time.

SUPPLEMENTAL COPPER ABSORPTION

Copper is usually supplemented in the sulfate, carbonate, or oxide forms. Recent studies indicate that copper oxide is not very available relative to copper sulfate (Langlands et al., 1986; Kegley and Spears, 1993). In early studies, copper carbonate was at least equal to copper (cupric) sulfate (Chapman and Bell, 1963). Various organic forms of copper also are available. In calves fed diets high in molybdenum, copper proteinate was more available than copper sulfate (Kincaid et al., 1986). However, Wittenberg et al. (1990) found similar efficiency of absorption of copper from copper proteinate and copper sulfate in steers fed high-molybdenum diets. Studies that compared copper lysine to copper sulfate have yielded inconsistent results. Ward and Spears (1993) reported that copper lysine and copper sulfate had similar efficiencies of absorption when fed to cattle; however, Rabiansky et al. 1999 and Nockels et al. (1993) found that copper lysine was more available than copper sulfate.

The studies cited above to determine the coefficient of absorption for copper are primarily based on the use of

cupric sulfate as the source of copper. The sulfates have the highest biologic efficiency of absorption of the common inorganic sources; the carbonate and oxide forms are of intermediate and low efficiency of absorption, respectively (Ammerman and Miller, 1972). Cupric oxide is nearly insoluble and does not serve as an adequate source of supplemental copper (Xin et al., 1991, Kegley and Spears, 1993). However, cupric oxide needles (oxidized copper wire particles - 24 g/cow) which degrade slowly in the rumen-reticulum have been successfully used to provide long term supplementation (6 to 8 months) to pastured animals (Judson, 1984; Richards, 1985). In general, supplemental forms of copper that are as soluble or exceed the solubility of cupric sulfate are good sources of copper for cattle. In some studies (Kincaid et al., 1986) the efficiency of absorption of copper proteinates has exceeded that of cupric sulfate. However, in most studies (Du et al., 1996; Wittenberg, 1990) the copper proteinates have nearly the same efficiency of absorption as cupric sulfate. Cupric chloride, which is highly soluble, may be up to 20 percent more available than cupric sulfate (Ivan et al., 1990). A chelated copper source demonstrating a specific mechanism for absorption that bypasses normal impediments to copper absorption could improve copper absorbability. However the data supporting this concept have not been reported in a refereed journal.

SIGNS OF COPPER DEFICIENCY

An early classic sign of copper deficiency in cattle is loss of hair pigmentation, particularly around the eyes. Scours is a clinical sign of copper deficiency that seems to be unique to ruminants, though the pathogenesis of this lesion is not understood. Anemia (hypochromic macrocytic), fragile bones and osteoporosis, cardiac failure, poor growth, and reproductive inefficiency characterized by depressed estrus also are observed in copper deficiency (Underwood, 1981). An effect of copper deficiency that is not easily observed is reduced immune function. Neutrophils have a reduced ability to kill invading microbes (Boyne, 1981) leading to increased susceptibility to infections. The dietary copper required for optimal immune function may exceed the amount required to prevent the more classic signs of copper deficiency (Xin et al., 1993; Xin et al., 1991; Stabel et al., 1993).

ASSESSING COPPER ADEQUACY

Forage concentrations of copper are of limited value in assessing adequacy of copper unless concentrations of copper antagonists in forage such as molybdenum, sulfur, and iron are also considered. Forages vary greatly in content of copper depending on plant species and available copper in the soil (Minson, 1990).

Concentrations of copper in liver <20 mg/kg on a DM basis (5 mg/kg wet weight) or plasma concentrations <0.50 mg/L are definitive of a copper deficiency. Copper concentration in normal liver is between 200 and 300 mg/kg of DM (Underwood, 1981; Ammerman, 1970; Puls, 1994). However, in the presence of high dietary molybdenum and sulfur which promote formation and absorption of thiomolybdates into the blood, copper in liver and plasma may not accurately reflect copper status because the copper can exist in tightly bound forms with the absorbed thiomolybdates, rendering it unavailable for biochemical functions (Suttle, 1991).

COPPER TOXICITY

Of all the minerals, copper is the most likely to become toxic if over-supplemented. Copper toxicosis can occur in cattle that consume excessive amounts of supplemental copper or feeds that have been contaminated with copper compounds used for other agricultural or industrial purposes (Underwood, 1977). When cattle consume excessive copper, they may accumulate extremely large amounts of the mineral in the liver before toxicosis becomes evident. Stress or other factors may result in the sudden liberation of large amounts of copper from the liver to the blood, causing a hemolytic crisis. Such crises are characterized by considerable hemolysis, jaundice, methemoglobinemia, hemoglobinuria, generalized icterus, widespread necrosis, and often death (Underwood, 1977; National Research Council, 1980).

Cattle are more tolerant of higher levels of dietary copper than are sheep, perhaps because of their greater capacity to eliminate copper from the body by way of bile. However, chronic copper poisoning is possible in cattle with supplementation of diets at just 4 to 5-fold the "required" amount of copper (Auza, 1999; Bradley, 1993). In most cases of copper intoxication, the concentration of copper in liver will exceed 900 mg/kg on a dry weight basis (Radostits et al., 1994); copper toxicity may occur at concentrations of copper in liver as low as 331 mg/kg liver on a dry weight basis (Auza, 1999). These data suggest the maximal tolerable dietary copper should be reduced to 40 mg/kg, unless dietary molybdenum is elevated greatly.

Iodine

FUNCTION

Iodine is necessary for the synthesis of the thyroid hormones thyroxine and triiodothyronine that regulate energy metabolism. The amount of iodine incorporated into thyroid hormones is about 0.4 mg/day in calves weighing 40 kg and increases to 1.3 mg iodine/day in nonpregnant heifers weighing 400 kg. Late gestation cows incorporate about

1.5 mg iodine/day into thyroid hormone. During lactation thyroid hormone production is increased, especially in high producing cows and iodine incorporation into thyroid hormones may reach 4 to 4.5 mg iodine/day (Sorensen, 1962). Thyroid hormone production also is increased during cold weather to stimulate an increase in basal metabolic rate as the animal attempts to remain warm (Goodman and Middlesworth, 1980).

About 80 to 90 percent of dietary iodine is absorbed and most of the iodine not taken up by the thyroid gland is excreted in urine and milk (Miller et al., 1988). Milk normally contains from 30 to 300 μg iodine/L and the iodine content of milk generally increases as dietary iodine increases making the iodine content of milk a reasonable indicator of iodine status (Berg et al., 1988). The availability of thyroid hormone assays provides more accurate assessment of actual thyroid function and the causes of thyroid dysfunction.

When the iodine content of the diet is more than adequate, <20 percent of the dietary iodine will be incorporated into the thyroid gland (Sorensen, 1962). Under conditions where intake of dietary iodine is marginal the thyroid gland will incorporate about 30 percent of the dietary iodine into thyroid hormones (Miller et al., 1975). When severely iodine deficient the hyperplastic thyroid can bind up to 65 percent of the iodine consumed by the cow (Lengemann and Swanson, 1957).

REQUIREMENT

Maintenance The daily thyroxine production of growing and mature nonlactating cattle is 0.2 to 0.3 mg thyroxine/ 100 kg of body weight, which contains 0.13 to 0.2 mg iodine (Miller et al., 1988). Miller et al. (1988) suggested that about 30 percent of dietary iodine actually was utilized by the thyroid gland to synthesize thyroxine and that 15 percent of the iodine that is used to synthesize thyroxine each day comes from recycling of iodine from the degradation of previously secreted thyroxine. Therefore, about 0.6 mg dietary iodine/ 100 kg of body weight is required to meet the requirement of the body for thyroxine synthesis. Pregnancy does not increase the requirement for iodine for thyroxine production to any significant degree (Miller et al., 1988). Assuming DMI of the 600-kg nonlactating pregnant cow is 1.8 percent of body weight, the diet must contain 3.6 mg of iodine/10.8 kg of dietary DM or 0.33 mg iodine/kg of dietary DM.

Lactation The rate of thyroxine production increases about 2.5-fold in heavily lactating cows (Sorenson, 1962). This increases the iodine requirement of lactating cows to 1.5 mg/100 kg body weight. Assuming that DMI of lactating cows is about 3.3 percent of body weight, the diet of a lactating cow should contain 0.45 mg iodine/kg DM.

FACTORS AFFECTING IODINE REQUIREMENT

Goitrogens are compounds that interfere with the synthesis or secretion of thyroid hormones and cause hypothyroidism. Goitrogens fall into two main categories. Cyanogenic goitrogens impair iodide uptake by the thyroid gland. Cyanogenic glucosides can be found in many feeds, including raw soybeans, beet pulp, corn, sweet potato, white clover, and millet and once ingested are metabolized to thiocyanate and isothiocyanate. These compounds alter iodide transport across the thyroid follicular cell membrane, reducing iodide retention. This effect is easily overcome by increasing supplemental iodine in the diet. The National Research Council (1989) included a safety factor (set dietary iodine requirement at 0.6 mg/kg dietary DM) to account for possible interference with iodine utilization by protein sources common in diets for lactating cows.

Progoitrins and goitrins found in cruciferous plants (rape, kale, cabbage, turnips, mustard) and aliphatic disulfides found in onions inhibit thyroperoxidase preventing formation of mono- and diiodotyrosine (Ermans and Bourdoux, 1989). With goitrins, especially those of the thiouracil type, hormone synthesis may not be readily restored to normal by dietary iodine supplementation and the offending feedstuff needs to be reduced or removed from the diet.

SOURCES OF IODINE

Most sources of iodine are readily available and the iodides of sodium, potassium, and calcium are commonly used. Potassium iodide tends to be easily oxidized and volatilizes before the animal can ingest it. Pentacalcium orthoperiodate and ethylenediamine dihydroiodine (EDDI) are more stable, less soluble, and are commonly used in mineral blocks and salt licks exposed to the weather.

Concentrations of iodine in forage are extremely variable and the concentration depends on the iodine content of the soil. Soil near the oceans tends to provide adequate iodine in plants. However, in the Great Lakes regions and Northwest United States, iodine concentrations in forages are generally low enough to result in a deficiency of iodine unless iodine is supplemented to the diet.

Supplemental iodine should not exceed 0.5 mg/kg diet due to concerns for human health (see toxicity section).

DEFICIENCY SYMPTOMS

Iodine deficiency reduces production of thyroid hormones slowing the rate of oxidation of all cells. Often the first indication of iodine deficiency is enlargement of the thyroid (goiter) of newborn calves (Miller et al., 1968). Calves also may be born hairless, weak, or dead. Fetal death can occur at any stage of gestation. Often the cows will appear normal (Hemken, 1970). In adult cattle, iodine

deficiency can cause enlarged thyroid glands, reduced fertility (males and females), and increased morbidity. Under conditions of marginal or deficient dietary iodine the maternal thyroid gland becomes extremely efficient in the removal of iodine from the plasma and in the recovery of iodine during the degradation of spent thyroid hormone and thyroglobulin. Unfortunately, this leaves little iodine for the fetal thyroid gland and the fetus becomes hypothyroid. The goiter condition is the hyperplastic response of the thyroid gland to increased stimulation of thyroid growth by thyroid-stimulating hormone produced in the pituitary gland. Under mild iodine deficiency, the hyperplastic thyroid gland can compensate for the reduced absorption of iodine (Hetzel and Wellby, 1997).

TOXICITY

Iodine toxicity has been reported in adult dairy cows with dietary intakes of just 50 mg/day (about 5 mg/kg dietary DM). Symptoms included excessive nasal and ocular discharge, salivation, decreased milk production, coughing and dry, scaly coats (Olson et al., 1984). High concentrations of dietary iodine in the diet also increase iodine concentrations in milk, and because humans are much more sensitive to iodine thyrotoxicosis than cows the danger of excess dietary iodine fed to cattle also is a public health issue (Hetzel and Welby, 1997).

Current US Food and Drug Administration Regulations set the maximum limit of iodine supplementation from EDDI at 10 mg/day.

Iron

Iron primarily functions as a component of heme found in hemoglobin and myoglobin. Enzymes of the electron transport chain, cytochrome oxidase, ferredoxin, myeloperoxidase, catalase, and the cytochrome P-450 enzymes also require iron as cofactors.

Iron deficiency results in hypochromic microcytic anemia due to failure to produce hemoglobin. Light colored veal is due to low muscle myoglobin as a result of restricted dietary iron. Anemic calves are listless and have poor feed intake and weight gain (Blaxter et al., 1957; Bremner and Dalgarno, 1973b). Another important aspect of iron deficiency is greater morbidity and mortality associated with depressed immune responses (Mollerberg and Moreno-Lopez, 1975). Increased morbidity was observed before there was an effect of iron deficiency on packed red blood cell volume.

Iron deficiency in adult cattle is very rare—in part because their requirement is reduced, but also because iron is ubiquitous in the environment, and soil contamination of forages (and soil ingested by animals on pasture) generally ensures that iron needs of the adult will be met or exceeded (Underwood, 1981).

ABSORPTION

Iron in the ferric form (Fe^{+3}) is poorly absorbed from the intestinal tract. Much of the dietary iron exists within feedstuffs in the ferric form. Some of the ferric iron can be reduced to the ferrous (Fe^{+2}) form upon reaction with the acid of the abomasum (Wollenberg and Rummel, 1987). During digestion, dietary non-heme iron in the ferrous state usually becomes bound to some chelator such as histidine, mucin, or fructose. These chelators enhance iron absorption by solubilizing iron and protecting it in the ferrous state. Other chelators (e.g., oxalate and phosphate) can inhibit iron absorption. During absorption, the iron binds to specific non-heme iron-binding receptors within the brush border of the enterocyte and is transported into the cell. Once inside the cell the iron can be transported to the basolateral membrane and becomes bound to transferrin for transport within the blood. If iron status of the body is adequate the iron entering the enterocyte is not transported to the basolateral membrane but is instead bound by ferritin, a protein produced by the enterocytes when iron is not needed by the body. Once bound to ferritin, the iron is excreted with the feces when the enterocyte dies and is sloughed (Beard and Dawson, 1997). The amount of dietary iron absorbed can be controlled by up-regulation or down-regulation of ferritin content of enterocytes. How concentrations of ferritin in enterocytes are regulated by iron status of somatic cells is unknown.

FACTORIAL REQUIREMENT DETERMINATION

The majority of iron incorporated into tissues is very effectively recovered and recycled so that maintenance requirements for iron are negligible. Milk contains about 1 mg iron/kg (National Research Council, 1989). The iron requirement of the conceptus of the pregnant cow between day 190 of gestation and the day of calving has been estimated to be 18 mg/day (House and Bell, 1993). Estimates of iron content in the body range from 18 to 34 mg/kg body weight of calves (Bremner and Dalgarno, 1973a). The absorbed iron requirement for growth of cattle has been set at 34 mg iron/kg average daily gain.

Matrone et al. (1957) estimated that 60 percent of iron supplemented as ferric chloride was absorbed in calves. In calves that are iron deficient, as assessed by anemia, about 55 percent of the iron in the diet can be retained in the calf's tissues (Miltenberg et al., 1993). Bremner and Dalgarno (1973a,b) using hemoglobin synthesis as their criterion, estimated the iron absorption coefficient in 9 to 12-week-old calves was 72 percent in an iron deficient

liquid diet providing just 10 mg iron/kg diet. When ferrous sulfate or ferric citrate were added to the diets to raise total dietary iron to 40 mg/kg diet, the iron absorption coefficient declined to 43 percent; yet these animals were still considered iron deficient as assessed by blood hemoglobin content and muscle pigmentation. At least in calves, the true efficiency of absorption of iron appears to decrease as the concentration of dietary iron increases—even before the absorbed iron requirement for growth has been achieved. An analysis of balance studies done in growing calves presented in the 1980 ARC publication (p. 238) suggests that the availability of soluble iron from liquid diets declines from 0.40 to 0.15 as the diet iron content increases from 40 to 100 mg/kg. Feeding calves <15 weeks of age as little as 39 mg of iron/kg DM will allow calves to grow at a normal rate but the muscles remain pale and the animals remain slightly anemic (Bernier et al., 1984). A recent study by Lindt and Blum (1994) suggests that 50 mg iron/kg diet (which would likely correspond to an absorption coefficient for iron of approximately 0.35) is adequate to support growth but the carcass remains pale. To maximize myoglobin synthesis and strength of the calf the diet must contain more iron.

Once the animal is ruminating the efficiency of iron absorption is considerably lower. In part this is because most forages will supply more than adequate amounts of iron to cattle as a result of soil contamination of the forage. Therefore iron absorption mechanisms tend to be down-regulated to protect the animal from iron toxicity as opposed to maximizing iron absorption. However a second factor is that the form of the iron in the forages is often ferric oxide which is less soluble and poorly absorbed. Studies utilizing radioactive iron determined that iron absorption efficiency was less than 2 percent in adult cattle fed a diet that supplied much more iron to the cows than was needed (Van Bruwaene et al., 1984). Though no data exists for cattle, when pregnant ewes were fed diets that provided adequate but not excessive dietary iron content (20 mg iron/kg diet) the animals were absorbing 21 percent of the dietary iron (Hoskins and Hansard, 1964). Assuming iron absorption in cattle is similar, it may not be desirable to base requirements on maximal iron absorption efficiency. Therefore, the absorption coefficient for dietary iron utilized to determine the requirement for iron in adult animals is set at 0.10 for iron in feedstuffs.

Using this approach a 6-week-old calf gaining 0.8 kg of body weight/day and consuming 0.9 kg dietary DM/day requires 150 mg iron/kg dietary DM. A 12-week-old calf gaining 1.8 kg body weight/day consuming 2.6 kg dietary DM/day requires 118 mg iron/kg dietary DM. A cow producing 25 kg of milk/day at 205 days of gestation and consuming 20 kg/day of DM requires just 24 mg iron/kg DM. Most feedstuffs will contain adequate iron to meet the iron requirements of adult cattle. Milk-fed calves are the only group of cattle that ordinarily require iron supplementation. Ferrous sulfate and ferric chloride are good iron supplements. Ferric oxide and ferrous carbonate are poorer iron sources (Henry and Miller, 1995).

TOXICITY

Concerns about Excessive Dietary Iron Iron can interfere with the absorption of other minerals, primarily copper and zinc. As little as 250 to 500 mg of iron/kg dietary DM has been implicated as a cause of copper depletion in cattle (Bremner et al., 1987; Phillippo et al., 1987).

If absorption of dietary iron exceeds the binding capacity of transferrin and lactoferrin in blood and tissues, free iron may increase in tissues. Free iron is very reactive and can cause generation of reactive oxygen species, lipid peroxidation, and free radical production leading to "oxidative stress," increasing anti-oxidant requirements of the animal (Halliwell, 1987). Free iron also is required by bacteria for their growth and excessive dietary iron can contribute to bacterial infection (Baynes et al., 1986). Iron toxicity is associated with diarrhea, reduced feed intake, and weight gain. The National Research Council (1980) recommended that dietary iron not exceed 1000 mg/kg DM.

Water containing more than 0.3 mg iron/L is considered unacceptable for human consumption by the US Environmental Protection Agency. Livestock can tolerate higher levels but iron in water will be much more available and therefore more toxic than iron in feedstuffs.

Manganese

Manganese deficiency can cause impaired growth, skeletal abnormalities (shortened and deformed), disturbed or depressed reproduction, and abnormalities of the newborn (including ataxia—due to failure of the inner ear to develop) (Underwood, 1977). The skeletal changes are related to loss of galactotransferase and glycosyltransferase enzymes that are vital to production of cartilage and bone ground substance mucopolysaccharides and glycoproteins. Manganese super oxide dismutase works in concert with other anti-oxidants to minimize accumulation of reactive forms of oxygen, which could damage cells. Manganese is found in highest concentrations within the mitochondria of cells. It also accumulates in the inorganic matrix of bone.

MANGANESE HOMEOSTASIS

The majority of the dietary manganese that is absorbed is removed from the portal circulation by the liver and is excreted into the bile. A small portion is bound to transferrin within the liver and released into the circulation for

transport to the tissues. Some of the absorbed manganese is bound to 2-macroglobulin and albumen and remains in the circulation. The proportion of manganese absorbed from the diet is <4 percent and generally is closer to 1 percent. A mechanism to enhance the efficiency of absorption of manganese during a deficiency of manganese does not appear to exist (Gibbons et al., 1976). The major homeostatic control for manganese appears to be regulation of biliary excretion of manganese absorbed in excess of tissue needs. Enterohepatic circulation of manganese also may be a factor in manganese homeostasis (Miller, 1978). Almost no manganese is excreted in urine.

Most of the manganese in the body is found in the skeleton, liver, and hair. Manganese accumulates in the liver in direct proportion to dietary manganese providing a more precise index of manganese status (Black, 1985), with an adequate concentration of manganese in liver being 10 to 24 mg/kg on a DM basis (Puls, 1994). The liver and perhaps other tissues have a limited ability to store manganese that can be mobilized, which may satisfy needs for several weeks during times of manganese deficiency (Lassiter and Morton, 1968).

MANGANESE REQUIREMENTS

There are no precise data on maintenance requirements for manganese in dairy cattle. However, manganese deficiency has been reported when diets contained 16 to 17 mg of dietary manganese/kg of DM. Assuming maintenance intakes of these diets did not exceed 2 percent of body weight and that <1 percent of ingested manganese was absorbed, we can estimate that the maintenance requirement for absorbed manganese of cattle is <0.002 mg/kg body weight or about 1 mg in a 500 kg cow. House and Bell (1993) determined that the fetus and placenta in cows at 190 days in gestation take up nearly 0.3 mg manganese/day. Colostrum contains 0.16 mg/kg and milk contains 0.03 mg/kg (Lyford and Huber, 1988). The concentration of manganese in carcasses of calves averages about 2.5 mg/kg of total carcass on a DM basis (Suttle, 1979). Assuming the carcasses used in these experiments were 27 percent DM, the manganese requirement for growth can be estimated to be 0.7 mg manganese/kg of body weight gain.

The coefficient for absorption of manganese from most diets is about 1 percent (Sansom, 1978; Gibbons, 1976) though Vagg (1976) found that just 0.5 percent of 54Mn was absorbed from the diet. For this model, the coefficient for absorption of manganese was set at 0.75 percent.

High concentrations of dietary calcium, potassium, or phosphorus increase excretion of manganese in the feces, presumably by reducing absorption of manganese (Lassiter, 1972; Hartmans, 1974). Excessive dietary iron depresses retention of manganese in calves (Ho, 1984).

In one experiment, all calves born from cows fed 16–17 mg/kg of dietary DM for 12 months had neonatal deformities (Dyer et al., 1965). The deformities included weak legs and pasterns, enlarged joints, stiffness, twisted legs, general weakness, and reduced bone strength. Heifers and cows that are fed low-manganese diets are slower to exhibit estrus, are more likely to have "silent heats," and have a lower conception rate than cows with sufficient manganese in their diet. Cows that were fed 7 to 10 mg of dietary manganese/kg of dietary DM for extended periods exhibited abscessed livers and had practically no bile in their gall bladders (Bentley and Phillips, 1951).

The National Research Council's *Nutrient Requirements of Beef Cattle* (1996) lists the requirement for manganese as 20 mg of manganese/kg dietary DM for growing cattle and 40 mg/kg dietary DM for breeding cattle. The Agricultural Research Council (1980) recommends 10 mg of manganese/kg dietary DM for growth of cattle increasing to 20 to 25 mg manganese/kg dietary DM to maintain normal reproduction. The National Research Council's *Nutrient Requirements of Dairy Cattle* (1989b) suggests that 40 mg of manganese/kg dietary DM should be adequate for all classes of cattle, though a requirement was not actually determined.

Using the factorial approach, a more precise determination of the manganese requirement can be made (Table 6-3). For example, a 500 kg heifer in late gestation gaining 0.5 kg body weight/d would require 1.65 mg absorbed manganese to meet her requirements. Assuming the coefficient of manganese absorption is 0.75 percent, then the diet must provide 220 mg manganese. If the heifer is consuming 10 kg DM/d, then the concentration of manganese would need to be 22 mg/kg.

The manganese content of feedstuffs is quite variable and is influenced by soil types, soil pH, fertilization, and plant species. Manganese sulfate is commonly used to supplement diets as it is a fairly soluble source of manganese. Other mineral sources of manganese and their efficiency of absorption relative to manganese sulfate are manganese carbonate (30 percent), manganese dioxide (35 percent), manganese monoxide (60 percent), and manganese methionine (125 percent) (Henry, 1995).

Manganese toxicity in ruminants is unlikely to occur, and there are few documented incidences with adverse effects limited to reduced feed intake and growth (Jenkins and Hidiroglou, 1991). These negative effects began to appear when dietary manganese exceeded 1000 mg/kg. The maximum tolerable amount of manganese, as given by the National Research Council (1980), is 1,000 mg/kg.

Molybdenum

FUNCTION AND REQUIREMENT

Molybdenum is a component of xanthine oxidase, sulfide oxidase, and aldehyde oxidase, enzymes found in milk and

TABLE 6-3 Comparison of Estimated Dietary Manganese Requirements (mg/d) and Dietary Manganese Concentrations (mg/kg of DM) for Cattle in Various Physiologic States

Cow Description[a]	Feed intake (kg of DM)	1989 NRC mg/day	1989 NRC mg/kg of diet	1980 ARC mg/day	1980 ARC mg/kg of diet	2000 NRC mg/day	2000 NRC mg/kg of diet
300-kg heifer, ADG = 0.7 kg	6	240	40	60	10	145	24.2
500-kg heifer, ADG = 0.5 kg, day 250 of gestation	10	400	40	200–250	20–25	220	22.0
650-kg cow, 40 kg milk per day	20	800	40	400–500	20–25	333	16.7
650-kg cow, day 270 gestation	12	480	40	240–300	20–25	213	17.8

[a]ADG = average daily gain.

many tissues (Mills and Davis, 1987). Concentrations of molybdenum in milk and plasma increase as dietary molybdenum increases (Underwood, 1981). Lesperance et al. (1985) determined that molybdenum in plasma of cows fed 3 mg molybdenum/kg diet were <0.1 g/ml. In animals fed 100 mg molybdenum/kg diet for 11 months, the plasma concentration of molybdenum had increased to 2.5 g/ml. A molybdenum deficiency in cattle has been difficult to reproduce. Shariff et al. (1990) observed that addition of 10 mg molybdenum/kg of a high forage diet containing 1.7 mg molybdenum/kg of diet increased the rate of in situ DM disappearance from the rumen of cattle, but adding molybdenum to a ground barley-based diet containing just 1.0 mg molybdenum/kg had no effect. Studies with lambs also were unable to define the dietary requirement for molybdenum (Ellis and Pfander, 1970; Ellis et al., 1958). It is not suggested to supplement molybdenum as it would seem very unlikely that dairy cattle would develop a deficiency of molybdenum when fed practical diets.

TOXICITY

Dietary molybdenum becomes a practical concern because it antagonizes the absorption of copper (and to a lesser extent phosphorus). Molybdenum toxicosis signs are essentially those associated with copper deficiency. Molybdenum and sulfate interact within the digestive tract to form a thiomolybbdate complex that has a high affinity for copper. Copper bound to this molybbdate is unavailable for absorption (see section on copper). The toxicity of molybdenum can be overcome by increased copper supplementation and copper toxicity can be reduced by molybdenum supplementation.

The maximal tolerable dietary concentration of molybdenum is suggested to be 10 mg/kg diet (National Research Council, 1980). However, as little as 5 mg Mo/kg diet has been demonstrated to cause copper depletion in heifers (Bremner et al., 1987; Phillippo et al., 1987).

Selenium

FUNCTIONS AND ANIMAL RESPONSE

The best understood function of selenium is as a component of the enzyme, glutathione peroxidase (GSH-px) (Rotruck et al., 1973). This enzyme converts hydrogen peroxide to water and is an important component of the cellular antioxidant system. More recently, selenium was identified as a component of Type I iodothyronine-5′-deiodinase (Berry et al., 1991); the enzyme that converts T_4 to T_3. Selenium is also found in other proteins but the functions of those proteins remain unclear (Deagen et al., 1991; Yeh et al., 1997).

White muscle disease or nutritional muscular dystrophy is caused by selenium deficiency. Clinical signs of this disease include leg weakness and stiffness, flexion of the hock joints, and muscle tremors (National Research Council, 1983). Cardiac and skeletal muscles have chalky striations and necrosis. Animals often die from cardiac failure. Marginal or short term deficiencies of selenium have been related to poor growth, general unthriftiness, and diarrhea (Andrews et al., 1968). In several studies, the prevalence of retained fetal membranes was reduced when supplemental selenium was fed or injected into dairy cows during late gestation (Harrison et al., 1984; Miller et al., 1993). The prevalence of retained fetal membranes was not reduced when injections of selenium were given to animals reared in areas of the country with adequate concentrations of selenium in feeds (Schingoethe et al., 1982). Other problems that have responded to supplementation of selenium include metritis, cystic ovaries (Harrison et al., 1984), and udder edema (Miller et al., 1993).

Selenium is involved with metabolism of arachadonic acid via GSHpx (Maddox et al., 1991). This relationship probably is one reason supplementation of selenium improves the killing ability of neutrophils (Hogan et al., 1993) and reduces the prevalence and severity of mastitis in dairy cattle (Smith et al., 1984; Erskine et al., 1989;

Maddox et al., 1991). Those experiments were conducted in areas where basal ingredients had low concentrations of selenium (<0.08 mg/kg) and supplementation consisted of either an injection of selenium (50 mg/cow) or supplementation of 0.2 to 0.3 mg of dietary selenium/kg of diet.

SOURCES

The concentrations of selenium in plant material are highly correlated with those in the soil. Fertilization of soil with selenium increases selenium concentrations in plants. Because of the strong relationship between concentrations of selenium in soils and plants, accurate maps have been developed that depict expected concentrations of selenium in feeds (National Research Council, 1983). In general, feeds grown in the central plains of the U.S. and Canada contain more than 0.1 mg selenium/kg of DM, and feeds grown east of the Mississippi river and west of the Rocky mountains typically contain <0.1 mg selenium/kg of DM. The leaves of forage plants contain 1.5 to 2 times more selenium than do stems (Harada et al., 1989; Gupta, 1991). When soils contain low concentrations of selenium, seeds and vegetative matter generally have similar concentrations of selenium, but as selenium in soil increases, concentrations of selenium in seeds increase more than do those in the vegetative matter (Harada et al., 1989; Stephen et al., 1989). Plant genetics also may influence concentrations of selenium in forages (McQuinn et al., 1991).

Most animal byproducts with the exception of milk products tend to have high concentrations of selenium. Fish meal often contains more than 1 mg selenium/kg of DM. However, the efficiency of absorption of selenium by nonruminants from these products, especially from fish meal, is low (Meltzer et al., 1993); comparative data for ruminants are not available.

Based on current regulations of the U.S. FDA (1997), the only two forms of inorganic selenium that can be added legally to diets in the United States are sodium selenite and sodium selenate at levels not to exceed 0.3 mg of supplemental selenium/kg of DM. Slow release ruminal boluses that contain selenium (from sodium selenite) also are available. Other sources of supplemental selenium include calcium selenite, selenium dioxide, and selenium enriched yeast.

EFFICIENCY OF ABSORPTION

The literature on the efficiency of absorption of selenium is not extensive for ruminants. Extrapolation of data collected from nonruminants should be done with caution because of extensive ruminal metabolism of selenium. Apparent digestibility of selenium in forages and concentrates is between 30 and 60 percent for sheep, goats, and nonlactating dairy cows (Harrison and Conrad, 1984a; Harrison and Conrad, 1984b; Aspila, 1988; Koenig et al., 1997). Limited data suggest that the true digestibility of selenium from feeds is between 40 and 65 percent for ruminants (Harrison and Conrad, 1984b; Aspila, 1988). The selenium from sodium selenate, sodium selenite, and Se-enriched yeast had apparent digestibilities by ruminants in the range of 40 to 50 percent (Harrison and Conrad, 1984b; Aspila, 1988; Koenig et al., 1997), but one study (Koenig et al., 1991) found that the apparent digestibility of selenium from sodium selenite fed to dairy cows was <4 percent.

Comparative absorption of different sources of selenium can be assessed by concentrations of selenium in tissue and blood and by activity of GSHpx. Overall, few differences have been found among the different inorganic sources of selenium or between dietary supplementation and ruminal boluses based on concentrations in tissue or blood of dairy cattle (Podoll et al., 1992; Gibson et al., 1993; Grace et al., 1995). Feeding organic sources of selenium (including Se-enriched yeast and feedstuffs with high concentrations of selenium) to ruminants usually increases concentrations of selenium in blood and tissues and the activity of GSHpx more than feeding inorganic sources of selenium (Conrad and Moxon, 1979; Johansson et al., 1990; Nicholson et al., 1991a, b).

REQUIREMENTS AND FACTORS AFFECTING REQUIREMENTS

The 6th revised edition (National Research Council, 1989b) defined the selenium requirement as 0.3 mg/kg of dietary DM for all classes of dairy cattle. No new data are available to dispute this requirement. However, the majority of data supporting this requirement were generated from experiments in which 0.3 mg of supplemental selenium/kg of dietary DM (DM basis) was fed so that total dietary selenium ranged from 0.35 to 0.40 mg/kg. Proper selenium nutrition of dairy animals in late gestation is important for preventing some periparturient disorders and also for ensuring that the calf is born with adequate selenium. Selenium efficiently passes through the placenta and calves born from dams receiving adequate selenium are in better selenium status than calves from dams not fed adequate selenium (Van Saun et al., 1989). The concentration of selenium in milk is increased when cows are fed additional selenium (Grace et al., 1997). Increased concentrations of selenium in milk may have positive effects on calf and human health.

Establishing requirements for selenium using the factorial approach is difficult because the deposition of selenium in body tissues, conceptus, and milk is dependent on selenium intake. As cows consume more selenium, the concentrations of selenium in milk and in the conceptus increase. Assuming a cow is fed a diet with approximately 0.3 mg of selenium/kg of dietary DM, the conceptus will accumulate

approximately 0.055 mg of selenium/day during the last trimester of gestation (House and Bell, 1994). Selenium concentration of milk ranges from 0.01 to 0.025 mg/kg (Conrad and Moxon, 1979; Lean et al., 1990; Van Dael et al., 1991). Endogenous fecal losses of selenium in dairy cattle range from 0.011 to 0.019 mg/kg of dry matter intake (Harrison and Conrad, 1984b; Koenig et al., 1991 a, b; Ivancic, 1999). Urinary excretion is dependent on Se intake (Ivancic, 1999). For lactating cows consuming approximately 2.5 mg of Se/day, urinary Se losses averaged 0.5 mg/d (Ivancic, 1999). Similar values were reported for dry cows fed similar amounts of Se (Harrison and Conrad, 1984b). Therefore, for a nonlactating cow in the last trimester of gestation that consumed 10 kg of DM/day, the requirement for absorbed selenium is approximately 0.7 mg/day. Assuming an absorption coefficient of 40 percent, the dietary requirement would be 1.75 mg/d. For a lactating cow producing 30 kg/d of milk, the requirement for absorbed selenium would be approximately 1.7 mg/day and the dietary requirement would be 4 mg/day. In agreement with those calculations, dry cows fed approximately 1.4 mg of Se/d (Harrison and Conrad, 1984b), and lactating cows fed approximately 4.2 mg/d (Ivancic, 1999) were in slightly positive Se balance. However, based on blood concentrations and prevalence of mastitis and retained fetal membranes, the calculated requirements are not adequate. Maus et al. (1980) reported that plasma concentrations of selenium in lactating dairy cows reached a plateau when selenium intake was 6 mg/day.

Current FDA regulations limit selenium supplementation to 0.3 mg/kg of diet (FDA, 1997) and in most situations, that amount of supplemental selenium will maintain dairy cattle in good selenium status. Based on the effect of selenium on mastitis, concentrations of selenium in whole blood should be greater than about 0.18 μg/ml or approximately 0.08 μg/ml for plasma (Jukola et al., 1996). Intake of approximately 6 mg/day of selenium should maintain those blood concentrations (Maus et al., 1980). Based on available data, the selenium requirement was maintained at 0.3 mg/kg of dietary DM.

Certain nutrients affect the absorption and metabolism of selenium and can alter the requirement for selenium. The requirements for vitamin E and selenium are clearly interdependent, but the relationship has not been quantified. Dairy cattle that are marginal in either selenium or vitamin E will require additional amounts of the other nutrient. The apparent digestibility of selenium is reduced when cows are fed diets with high (ca. 1.3 percent) or low (ca. 0.5 percent) concentrations of calcium (Harrison and Conrad, 1984a). Increased consumption of sulfur may increase the requirement for selenium. In sheep, increasing dietary sulfur (from sulfate) from 0.05 percent to 0.24 percent decreased selenium absorption (Pope et al., 1979) but had no effect on metabolism of selenium when dietary sulfur was increased from 0.28 to 0.78 percent (Paulson et al., 1966). In contrast, true digestibility of selenium by lactating dairy cows was reduced from about 56 percent to 46 percent when dietary sulfur was increased from 0.2 percent to 0.4 or 0.7 percent (from calcium and magnesium sulfate) (Ivancic, 1999). Supplementation of sulfur from anionic salts for the last three weeks of gestation did not influence selenium status of nonlactating cows (Gant et al., 1998). With beef cattle, long term feeding of diets that contained approximately 0.2 or 0.5 percent total sulfur did not affect concentrations of selenium in whole blood or the activity of GSHpx in red blood cells (Khan et al., 1987). A relationship between dietary copper and selenium also may occur. In sheep fed a diet with >0.3 mg/kg of selenium, increasing dietary copper from about 7 mg/kg of DM to 21 mg/kg of DM increased the selenium concentrations in liver but decreased the concentration of selenium in muscle (Hartmann and van Ryssen, 1997). An isotope study found similar results when sheep were fed low selenium diets (<0.1 mg/kg) (White et al., 1989). Koenig et al. (1991b) reported no effect of feeding diets with 0 or 16 mg of supplemental copper/kg on selenium balance in dairy cows (diets contained 0.2 mg of selenium/kg of DM). Increasing the amount of zinc in diets fed to rats from 5 to 20 mg/kg reduced absorption of selenium by 25 percent (House and Welch, 1989). Data concerning the effects of zinc with ruminants are lacking.

TOXICITY

Historic concerns regarding selenium were not about providing adequate selenium to cows, but rather about toxicity. Alkali disease and blind staggers result from selenium toxicity. Clinical signs include sloughing of hooves, lameness, loss of hair, and emaciation (National Research Council, 1983). Most cases of selenium toxicity have been related to consumption of selenium accumulating plants (e.g., *Astragalus* sp.). Chronic toxicity can occur when cattle are fed diets with 5 to 40 mg of selenium/kg for a period of several weeks or months (National Research Council, 1983). Acute toxicity can occur when cows are fed 10 to 20 mg of Se/kg of body weight. An injection of about 0.5 mg of Se/kg of body weight to young cattle (ca. 200 kg) resulted in a 67 percent mortality rate (National Research Council, 1983). The recommendation for dietary selenium is approximately 16 times less than the lowest dietary level that has been related to chronic toxicity.

Zinc

FUNCTION

Zinc is a component of many metalloenzymes such as copper-zinc superoxide dismutase, carbonic anhydrase,

alcohol dehydrogenase, carboxypeptidase, alkaline phosphatase, and RNA polymerase, which affects metabolism of carbohydrates, proteins, lipids, and nucleic acids. Zinc regulates calmodulin, protein kinase C, thyroid hormone binding, and inositol phosphate synthesis. Zinc deficiency alters prostaglandin synthesis, which may affect luteal function (Graham, 1991). Zinc is a component of thymosin, a hormone produced by thymic cells that regulates cell-mediated immunity.

ABSORPTION

Intestinal absorption of zinc occurs primarily in the small intestine (Flagstad, 1976). In animals that are zinc deficient, zinc readily enters the enterocytes and is transported across the cell by an intestinal protein (CRIP) that is rich in cysteine and is released into the portal circulation to be carried primarily by transferrin (Evans and Winter, 1975) and albumin. In animals that are zinc replete, metallothionein, a second protein rich in cysteine, is found in the mucosal cells and this metallothionein competes with the cysteine rich protein for zinc transported across the brush border membrane. Zinc bound to metalothionein will remain in the enterocyte and be excreted with the feces when the enterocyte dies and is sloughed (Chesters, 1997). By up-regulating or down-regulating metallothionein in mucosal enterocytes the amount of dietary zinc that is absorbed can be regulated. How the status of zinc regulates the concentration of intestinal metallothionein is unknown, but it requires days to weeks for the concentration of metallothionein in the intestine to adjust to a low zinc diet (Taylor et al., 1991).

About 50 percent of the zinc in milk is absorbed by calves (Miller and Cragle, 1965). Adding soybean protein to a milk diet adds phytic acid and reduces the efficiency of zinc absorption by more than one-half (Miller et al., 1967). In ruminating calves weighing 70 to 150 kg, efficiency of zinc absorption ranged from 16 percent to 51 percent, depending on the level of supplementation in one study (Miller et al., 1968) and was about 20 percent in another (Miller and Cragle, 1965). In adult cattle, only 12 to 14 percent of dietary zinc was absorbed in the study of Miller and Cragle (1965). Total zinc in the diet was not reported. In a study by Hansard et al. (1968), about 22 percent of dietary zinc was absorbed in adult cows fed a diet containing 28 mg zinc/kg diet, which was probably not greatly in excess of the needs of the animals. Animals that were adapted to a low zinc diet with no inhibitors of zinc absorption can absorb nearly 50 percent of dietary zinc (Kirchgessner and Schwarz, 1976). However, optimal zinc absorption is neither likely, nor desirable and a coefficient of zinc absorption of 15 percent is used for all ruminating animals, based on the results of Miller and Cragle (1965) with adult cattle. This approach might be too conservative. The Agricultural Research Council (1980) reviewed the literature on efficiency of zinc absorption and concluded that pre-ruminant calves absorb about 50 percent of dietary zinc, growing ruminants absorb 30 percent of dietary zinc, and adult ruminants absorb 20 percent of dietary zinc. Since intrinsic zinc from feedstuffs is apparently absorbed to about the same extent as added inorganic zinc (Stuart et al., 1986), no correction was made for zinc from inorganic or organic sources. The poor solubility of zinc oxide suggests that it may not be as well absorbed as zinc sulfate. However, when zinc oxide was added to diets to raise dietary zinc, it was comparable to other forms of zinc in preventing signs of zinc deficiency in most studies (Miller et al., 1967; Miller et al., 1970; Kincaid, 1979; Kincaid et al., 1997). Unfortunately, these studies used large amounts of zinc (200 to 600 mg/kg diet) in basal diets. A comparison of efficiency of absorption of zinc sources at dietary concentrations closer to or slightly below the metabolic needs of the animals remains to be done in ruminants.

DIETARY ZINC REQUIREMENT

A factorial approach was taken to determine the dietary requirement for zinc. The daily endogenous fecal loss of zinc is approximately 0.033 mg zinc/kg body weight and the obligate urinary loss of zinc has been estimated as 0.012 mg zinc/kg body weight for a total maintenance requirement for zinc of 0.045 mg zinc/kg body weight (Hansard et al., 1968). During gestation, the fetus and uterus retain about 12 mg zinc/day (House and Bell, 1993) between day 190 of gestation and the end of gestation and nearly double that estimated by Agricultural Research Council (1980). The zinc content of milk is about 4 mg/kg (range 3.4 to 5.8 mg) (Osis et al., 1972; Schwarz and Kirchgessner, 1975). The lactational requirement for zinc can be very large. The amount of zinc retained during growth of body tissues is estimated to be 24 mg zinc/kg average daily gain (range 16 to 31 mg) (Kirchgessner and Neesse, 1976; Miller, 1970). Using these values, the requirement for the amount of zinc that must be absorbed from the diet can be estimated. This requirement divided by the efficiency of absorption of dietary zinc, which is estimated to be 15 percent, is the total amount of zinc the diet must supply each day.

A comparison of the requirement for dietary zinc of cattle in various physiologic states estimated by the National Research Council (1989b), the Agricultural Research Council (1980), and the current model are presented in Table 6-4. The National Research Council (1989b) may have underestimated the dietary zinc required to support lactation. Kirchgessner and Weigand (1982) utilized semi-purified diets that contained between 6 and 436 mg of zinc/kg to derive a dose response relationship

TABLE 6-4 Comparison of Estimated Dietary Zinc Requirements (mg/d) and Dietary Zinc Concentrations (mg/kg of DM) for Cattle in Various Physiologic States

Cow Description	Feed intake (kg of DM)	1989 NRC mg/day	1989 NRC mg/kg of diet	1980 ARC mg/day	1980 ARC mg/kg of diet	2000 NRC mg/day	2000 NRC mg/kg of diet
300-kg heifer, ADG = 0.7 kg	6	240	40	151	25	202	33
500-kg heifer, ADG = 0.5 kg day 250 gestation	10	400	40	204	20.4	310	31
650-kg cow, 40 kg milk/d	20	800	40	946	47.3	1261	63
650-kg cow, day 270 gestation	12	480	40	178	14.8	274	22.8

between dietary zinc and the concentration of zinc in serum or milk or serum alkaline phosphatase. They reported that diets containing 35 mg zinc/kg were sufficient to provide 90 percent of the maximal response for zinc in serum, milk, and serum alkaline phosphatase activity during lactation. Semi-purified diets may not be an adequate indication of expected results in practical feeding situations where chelation may reduce the availability of dietary zinc.

FACTORS AFFECTING ZINC REQUIREMENTS

Two major dietary factors that can modify the efficiency of absorption of dietary zinc are interactions of zinc with other metal ions and the presence of organic chelating agents in the diet.

Zinc and copper are antagonistic to one another. In most cases, zinc interferes with absorption of copper to cause a deficiency of copper, if copper availability is marginal, but when dietary copper-to-zinc ratios are very high (50:1), copper could interfere with absorption of zinc, although this is unlikely in cattle (Van Campen, 1969). Excessive dietary iron can interfere with absorption of zinc in man and other species. In rats, a deficiency of iron enhanced both absorption of iron and zinc suggesting iron and zinc share a common absorption mechanism (Flanagan et al., 1980). In man, the effect of excess iron is evident when the iron-to-zinc ratio is >2:1 (Solomons, 1986). No data exist for the interaction between dietary iron and absorption of zinc in ruminants. Under practical conditions dietary iron is often well in excess of requirements for iron by the cow.

Cadmium is antagonistic to the absorption of both zinc and copper and also interferes with tissue metabolism of zinc and copper in the liver and kidneys (see cadmium section). Lead competitively inhibits absorption of zinc and also interferes with the function of zinc during heme synthesis (Finelli et al., 1975). Tin also may interfere with absorption of zinc (Johnson and Greger, 1984).

High dietary concentrations of calcium interfere with absorption of zinc in nonruminants, perhaps because the effect of phytate is exaggerated by dietary calcium (O'Dell et al., 1958). In cattle supplementation of calcium was associated with a reduction of zinc in serum of yearling steers and calves (Mills et al., 1967; Perry et al., 1968). Studies with sheep did not demonstrate a deleterious effect of increased dietary calcium on metabolism of zinc or growth (Pond and Oltjen, 1988; Pond and Wallace, 1986).

Organic chelators of zinc can increase or decrease efficiency of absorption of zinc. Those that interfere with absorption tend to form insoluble complexes with zinc. Phytate commonly binds zinc in plants and greatly diminishes absorption of zinc in calves and nonruminant animals. However, ruminal microbes metabolize most of the dietary phytate so it is not a factor affecting absorption of zinc in ruminating animals.

Deficiency symptoms for zinc were not observed in ruminating calves fed semi-synthetic rations containing as little as 8 mg zinc/kg DM. Most of these rations utilized urea to supply some of the nitrogen required by the animals (Agricultural Research Council, 1980, Table 6.13, p. 257) (Mills et al., 1967). However, a deficiency of zinc has been reported numerous times in calves of similar age fed natural diets supplying at least 8 and as much as 30 mg zinc/kg diet (Demertzis, 1973; Agricultural Research Council, 1980—Table 6.16, p. 261). These data raise suspicions that unknown organic factors found in common feed ingredients interfere with absorption of zinc by ruminants and other species.

Some naturally occurring chelators of zinc improve absorption of zinc. Scott and Ziegler (1963) demonstrated with chicks that adding distillers dried solubles and liver extract to soybean protein based diets improved the efficiency of absorption of the dietary zinc, though the factor remained unknown. Peptides and amino acids can form complexes with zinc and both cysteine and histidine bind zinc strongly and improve efficiency of absorption of zinc in chicks (Nielsen et al., 1966; Hortin et al., 1991). At the alkaline pH found in the intestine, it is likely that little free zinc cation exists in solution. One action of beneficial chelates is to form soluble zinc complexes within the small intestine permitting soluble zinc to reach the brush border membrane for absorption.

DEFICIENCY

Cattle that are deficient for zinc quickly exhibit reduced feed intake and growth rate. With a more prolonged deficiency the animals exhibit reduced growth of testes, weak hoof horn, and perakaratosis of the skin on the legs, head (especially nostrils), and neck. On necropsy thymic atrophy and lymphoid depletion of the spleen and lymph nodes are evident (Brummerstedt et al., 1974; Mayland et al., 1980; Miller and Miller, 1962; Mills et al., 1967). A genetic defect that greatly reduces absorption of zinc has been identified in Dutch-Friesan cattle. These animals become severely deficient in zinc unless fed extremely large amounts of dietary zinc (Flagstad, 1976).

Concentrations of zinc in serum are normally between 0.7 and 1.3 μg/ml. Concentrations of zinc in serum below 0.4 μg/ml are often considered deficient. However, stress or disease can cause a rapid redistribution of zinc out of extracellular fluids causing concentrations of zinc in serum to fall into the "deficient" range even when dietary zinc is adequate (Hambridge et al., 1986; Goff and Stabel, 1990). Concentrations of zinc in the liver are more reliable and should be 100 to 400 mg/kg on a dry weight basis (Puls, 1994). However, responsive conditions to zinc have been seen in cattle with concentrations of zinc in liver above 100 mg/kg because zinc in liver does not serve as a readily mobilizable source of zinc during times of dietary insufficiency of zinc (Miller, 1978). Thus, it is difficult to certify adequacy of zinc by concentrations of zinc in liver but it is possible to diagnose zinc deficiency when zinc in liver is below 100 mg/kg dry weight. Carbonic anhydrase and alkaline phosphatase activities in blood have been used to assess the status of zinc but these determinations are difficult to interpret because concurrent disease can affect these enzymes as much as a deficiency of zinc. A promising indicator of the status of zinc is metallothionein in plasma or urine. This protein is induced by zinc in tissues and released into blood (Garvey, 1984) and may be a better indication of the status of zinc in tissues.

TOXICITY

A large amount of dietary zinc is fairly well tolerated by cattle, however zinc toxicity was observed in cattle fed 900 mg zinc/kg diet (Ott et al., 1966). Very high levels of zinc have a negative effect on absorption and metabolism of copper (Miller et al., 1989), and it is for this reason primarily that dietary zinc should be limited. The maximal tolerable level of dietary zinc is suggested to be 300 to 1000 mg/kg diet. However very high levels of zinc oxide, providing more than 1000 mg Zn/kg DM are routinely fed to cattle in some areas to combat facial eczema with no sign of zinc intoxication.

Chromium

Chromium is primarily found in tissues as an organometallic molecule composed of Cr^{+3}, nicotinic acid, glutamic acid, glycine, and cysteine known as glucose tolerance factor (Toepfer et al., 1977). Without Cr^{+3} the glucose tolerance factor is inactive. Glucose tolerance factor can potentiate the effect of insulin on tissues, either by stabilizing the insulin molecule (Govindaraju et al., 1989), or by facilitating the interaction of insulin with its receptor in tissues (Mertz, 1993). Studies in rats have determined that chromium is absorbed primarily from the small intestine (Chen et al., 1973). Inorganic forms of Cr^{+3} ($CrCl_3$, Cr_2O_3) are very poorly absorbed (hence the utility of Cr_2O_3 as a marker for digestion studies). Complexing Cr^{+3} with organic compounds greatly increases the absorption of chromium. Chromium nicotinate and chromium picolinate are usually considered the most available sources of supplemental chromium. Chromium from naturally occurring sources such as brewer's yeast also is efficiently absorbed with as much as 10 to 25 percent absorbed by rats (Underwood, 1977a).

The essentiality of chromium as a required element necessary for normal glucose metabolism in the diet of humans is well accepted and it is recommended that the diet of adult humans supply from 50 to 200 μg chromium/day (National Research Council, 1989a). The role of chromium in animal nutrition recently was reviewed by the National Research Council (1997). Several studies in cattle have demonstrated a favorable response to chromium supplementation, especially if the animals are under some physiologic stress. These positive studies examined the effects of supplementation of chromium during the periparturient period in dairy cattle. The positive findings included improved humoral and possibly cell mediated immune response (Burton et al., 1993), improved energy status (reduced liver triglyceride accumulation), DMI, and milk production (Besong et al., 1996), and increased milk production in primiparous cows but not multiparous cows (Yang et al., 1996). In these studies the basal rations generally contained <1.6 mg chromium/kg diet and diets were supplemented with 0.5 to 10 mg chromium/kg diet. Together with reports of reduced morbidity of stressed feedlot calves supplemented with chromium (Moonsie-Shageer and Mowat, 1993), these studies suggest that cattle require chromium in their diets. Unfortunately, the amount of chromium required in the diet for optimal performance is unclear and the literature does not support a general recommendation for supplementation of chromium of typical cattle diets. Additional research on the bioavailable chromium contained in common feedstuffs and the effects of diets deficient in chromium on performance of dairy cows will be required, including appropriate titration studies, before a dietary requirement for chromium can be established (National Research Council, 1997).

It is generally accepted that trivalent chromium added to diets is safe and non-toxic. Chromium toxicity is primarily associated with exposure to hexavalent Cr^{+6} (chromium trioxide, chromates, bichromates). Hexavalent chromium passes into the interior of cells much more readily than trivalent chromium (Jennette, 1979) and is able to depress mitochondrial oxygen consumption by inhibiting alpha-ketoglutarate dehydrogenase (Ryberg and Alexander, 1990). If significant amounts reach the cell nucleus there can be a variety of pathologic changes in the DNA (Alexander, 1993). For livestock the maximum tolerable concentration of chromium in the diet is set at 3000 mg/kg for the oxide form and 1000 mg/kg for the chloride form of the trivalent forms of chromium. Hexavalent forms of chromium are at least five times more toxic (National Research Council, 1997).

Aluminum, Arsenic, Nickel, Silica, Tin, and Vanadium

These elements can be found in minute amounts in the tissues of animals. When removed from the diet of laboratory rodents, some of these elements have been demonstrated to be essential for these species. Data on essentiality in dairy cattle are non-existent and practical diets would not be expected to result in deficiency of any of these elements. Most of these elements can be toxic when provided in large amounts and this is occasionally a problem in dairy cattle.

Our current understanding of metabolism does not include any specific role for aluminum, arsenic, silica, tin, or vanadium. Nickel is required for activity of urease.

Aluminum is the third most common element in the earth's crust. It is found in only trace amounts in plants and animals. Animals on pasture can consume up to 10 percent of their total DMI as soil. Most of the aluminum ingested is not absorbed, and the majority of the small amount that is absorbed is rapidly excreted in urine. Bone and other tissues will accumulate aluminum, especially if renal function is compromised (Thurston et al., 1972).

Aluminum binds phosphate, which reduces absorption of phosphorus. In animals, aluminum toxicosis is primarily associated with malabsorption of phosphorus and other minerals (Allen, 1984). The maximum tolerable dietary aluminum is 1000 mg/kg of diet (National Research Council, 1980; Bailey, 1977; Valdivia et al., 1978).

A deficiency of arsenic has been reported in goats and mini-pigs fed diets containing <50 mg arsenic/kg of diet (Anke et al., 1976). Deficiency signs consisted of impaired reproductive performance and lower weight gains in second generation animals.

Organic arsenicals have been used in swine and poultry for their antibiotic and anti-coccidial properties (Frost et al., 1955). They have not been used in cattle. However, organic arsenicals, as well as inorganic forms of arsenic are well absorbed and can cause toxicosis when feedstuffs are accidentally contaminated with arsenic. Arsenic released from copper and lead smelters can contaminate soil and forages (Lillie, 1970). Unfortunately, sheep and cattle do not find arsenic unpalatable and may even develop a taste for it (Clarke and Clarke, 1975). Insecticides, wood preservatives, and herbicides containing arsenic also have been sources of contamination.

Arsenic binds to sulfhydryl groups of proteins interfering with their function. Inorganic arsenicals are more toxic than organic arsenicals. Cattle fed poultry manure containing 18 mg/kg organic arsenic/kg of diet exhibited no signs of toxicity (Calvert and Smith, 1972). The maximal tolerable dietary arsenic was set at 50 mg/kg and 100 mg/kg for inorganic and organic forms, respectively, per kg of diet (National Research Council, 1980).

Deficiencies of nickel have been produced in chicks, pigs, goats, and rats (Nielsen and Ollerich, 1974; Nielsen and Sandstead, 1974) and in lambs (Spears and Hatfield, 1978). Lambs fed a diet low in nickel had lower ruminal urease activity than lambs fed 5 mg of nickel/kg of DM (Spears, 1984). Nickel deficiency is associated with reduced growth, which is perhaps the result of pathologic changes in the liver (Nielsen et al., 1975). Nickel is relatively non-toxic with maximal tolerable dietary concentrations of 50 mg/kg for cattle (National Research Council, 1980). The main adverse affect of nickel is reduced feed intake.

Silicon is the second most abundant element on earth but is found in only very trace amounts in animal tissues. In chicks and rats, a deficiency in silicon has been demonstrated using carefully purified diets. A deficiency in silicon is associated with disturbances of bone formation and depression in growth rate (Carlisle, 1974).

Silicon is primarily found combined with oxygen to form silica. Silica is only poorly absorbed. Contamination of forages with soil makes a deficiency of silicon very unlikely in ruminants. Silicon in forages is more likely to present a problem as it can depress DM digestibility in ruminants (Van Soest and Jones, 1968).

While primarily a clinical problem in males, urinary calculi of ruminants often contain silica. Although urinary calculi in grazing steers has been associated with the silica content of forages (Parker, 1957), there is evidence that low dietary intake of magnesium is the main dietary factor associated with increased formation of silica containing uroliths (Schneider et al., 1952; Parker, 1957). The maximal tolerable silicon concentration was set at 0.2 percent of the diet, based primarily on the negative affects of silicon on dietary organic matter digestibility (National Research Council, 1980).

Tin has been demonstrated to be essential for growth in rats (Schwartz et al., 1970). However, because tin is so commonly used for production of "tin cans" and alloys, the greater concern is that tin will contaminate feedstuffs. Inorganic forms of tin are poorly absorbed and dietary

levels as high as 150 mg/kg were considered safe (deGroot, 1973). Organic forms of tin may be more toxic (National Research Council, 1980).

Vanadium at 0.1 mg/kg of diet optimizes growth in rats (Schwartz and Milne, 1971) and calves (Drebickas, 1966). How it acts is unknown. Less than 1 percent of dietary vanadium is absorbed in sheep (Hansard, 1975).

Vanadium toxicity is associated with inhibition of enzyme activity, particularly the Na-K ATPases (Cantley et al., 1977). The maximum tolerable vanadium was suggested to be 50 mg/kg of diet (National Research Council, 1980). However, ruminal function (DM digestibility) in lambs was disrupted with just 7 mg vanadium/kg of diet (Williams, 1973). Rock phosphates can contain up to 6,000 mg of vanadium/kg, which could be a potentially toxic source of vanadium (Romoser et al., 1960).

TOXIC MINERALS

Cadmium

Cadmium is a heavy metal that accumulates within the body, particularly the kidney, to cause renal damage. It is cleared from the body very poorly and very slowly. It is of particular concern to humans because of our long life span and because cadmium has become so commonly distributed in the environment. The Joint FAO/WHO Expert Committee on Food Additives (1972) established a provisional maximum tolerable intake of cadmium at 57 to 71 μg/day. Ryan et al. (1982) estimated that men and women consume 33 and 26 μg cadmium/day respectively. The maximal tolerable cadmium in the diet of cattle was set at 0.5 mg/kg in an effort to avoid adding cadmium to the diet of humans consuming products primarily derived from dairy cattle (National Research Council, 1980).

Cadmium is antagonistic to zinc and copper, and to a lesser degree iron. Diets containing from 5 to 30 mg cadmium/kg of diet generally decrease animal performance by interfering with copper and zinc absorption, resulting in symptoms usually associated with copper and zinc deficiency. Diets containing more than 5 mg of cadmium/kg can cause copper concentrations in liver to decline (Smith, 1986). Cadmium binds to metallothionein very tightly, which competitively decreases absorption of copper and to a lesser degree absorption of zinc. Liver and kidney contain metallothionein proteins (Kagi et al., 1974) that accumulate cadmium throughout the life of animals. Ruminant diets containing more than 30 mg of cadmium/kg have produced anorexia, reduced growth, decreased milk production, and abortion (Doyle et al., 1974; Wright et al., 1977).

Cadmium is a contaminant of the zinc sulfides used to galvanize iron to prevent corrosion. It is a component of nickel-cadmium batteries and is used as a stabilizer in polyvinylchloride plastics. Waste from zinc smelting operations and zinc plating operations have been sources of contamination that have resulted in cadmium intoxication. Urban sewage sludge contains significant amounts of cadmium and should not be used as fertilizer on farmland growing crops intended for consumption by humans or food animals. Some phosphate fertilizers can also contain significant amounts of cadmium (National Research Council, 1980).

High concentrations of cadmium (up to 10 mg/kg) have been found in forages grown in fields near industrial zinc-plating sites and where urban sludge has been used as a fertilizer (Smith, 1986). In most areas of the country, cadmium toxicosis is not a concern because most feeds and forages contain <0.50 mg/kg (National Research Council, 1980).

Less than 1 percent of dietary cadmium is absorbed by ruminants (Neathery et al., 1974). Intestinal metallothionein binds cadmium tightly and limits the absorption of cadmium. Cadmium can be detected in small amounts in milk but the mammary gland limits cadmium transport and the concentration of cadmium in milk is not increased by high dietary concentrations of cadmium (Sharma et al., 1979; Smith, 1986; Van Bruwaene et al., 1982). Concentrations of cadmium in muscle are much lower than in kidney (Sharma et al., 1979). However, significant accumulation in muscle has been reported after prolonged feeding of diets high in cadium to cattle (Smith, 1986).

Fluorine

Although fluorine in very small amounts can increase the strength of bones and teeth, it is generally not regarded as an essential dietary component (National Research Council, 1980). Fluorine is generally regarded as a toxic element with regards to domestic livestock because in large amounts fluorine will accumulate in bone to an extent that actually weakens bone, increasing lameness and increasing wear of teeth (Shupe, 1980; Crissman et al., 1980). The teeth of cattle intoxicated with fluorine become mottled and stained, and are eroded or pitted.

Soluble forms of fluoride, such as sodium fluoride, are rapidly and nearly completely absorbed by cattle (Perkinson Jr. et al., 1955). About 50 percent of fluorine in undefluorinated phosphates of bone meal is absorbed. Dietary calcium, aluminum, sodium chloride, and fat can reduce fluorine absorption (National Research Council, 1980). Fluorine does not pass readily into milk and fluorine in milk does not increase markedly with increased dietary fluorine (Greenwood et al., 1964).

Minor morphologic lesions can occur in young cattle receiving as little as 20 mg of fluorine/kg of diet when teeth are developing rapidly, but the relationship between these minor lesions and animal performance is unknown

(National Research Council, 1980). The maximal tolerable dietary content of fluorine was set at 40 mg/kg of diet (National Research Council, 1980).

Rock phosphates from Florida [fluorapatite, $Ca_{10}F_2(PO_4)_6$] can contaminate cattle when used in feed or when applied as a fertilizer without first being defluorinated. To qualify as defluorinated, feed grade phosphates can contain no more than 1 part of fluorine to 100 parts of phosphorus (AAFCO, 1977). Other potential sources of fluorine include bone meal, deep well water, and soil near volcanoes and fumaroles. Fluorine in the form of hydrofluoric acid, silicon tetrafluoride, or fluoride containing particulates can be released from industrial sites associated with aluminum or phosphate processing. These emissions can contaminate water, soil, and plants near these sites, resulting in fluorine intoxicosis in animals grazing in the areas (Bunce, 1985).

Lead

Lead is the most common cause of toxicoses in domestic livestock (Neathery and Miller, 1975). Lead halides and lead bromochloride, which were once added to gasoline as engine valve lubricants were emitted from automobile exhaust during combustion and continue to contaminate much of the American landscape. Lead based pigments were common until restrictions were imposed and paint chips from older structures remain a significant source of lead contamination in cattle. Lead intoxication also has occurred in cattle consuming lead from batteries, putty from window glazing, linoleum, asphalt roofing, and used engine or crankcase oil. From 3 to 10 percent of ingested lead is absorbed by adult ruminants (Fick et al., 1976). Elevated dietary calcium, phosphorus, iron, zinc, fat, and protein decrease the absorption and retention of lead (Mahaffey, 1983; White et al., 1985). Lead accumulates in bone (Schroeder and Tipton, 1968). Lead readily passes into milk so that increasing dietary concentrations of lead results in increased lead concentration in milk (Murthy et al., 1967; Lopez et al., 1985; Underwood, 1977).

Clinical intoxicosis interferes with normal metal-dependent enzyme functions. Lead causes derangements in porphyrin and heme synthesis, interferes with protein synthesis, basophilic stippling of erythrocytes, and causes microcytic hypochromic anemia (National Research Council, 1980).

Chronic exposure to low levels of lead is not associated with clinical symptoms in cattle because bones sequester lead and release it gradually to the blood for excretion. In humans, low exposure to lead is associated with a loss of cognitive powers—this is not generally detected in cattle. Acute intoxication with lead causes impaired neurologic function resulting in blindness and irritability (Radostits et al., 1994). Lead toxicity also causes intestinal pain and colic, and abortion. Lead accumulates in the kidney cortex and renal tubular inclusion bodies suggest impaired renal function. In cattle that have died from lead poisoning, lead in the kidney cortex is often >50 mg/kg and in the liver is often >20 mg/kg (fresh tissue) (Allcroft and Blaxter, 1950).

Whereas cattle can tolerate up to 100 mg of lead/kg in their diet without noticeable effects the maximum tolerable dietary lead was set at 30 mg/kg (National Research Council, 1980). A single dose of 200 mg lead/kg body weight is lethal to cattle (Allcroft and Blaxter, 1950). Young animals tend to be more susceptible to lead intoxication than adults because they have a higher rate of absorption of lead (90 percent versus 10 percent) and are more likely to exhibit pica (eating non-food substances).

Mercury

Mercury toxicity is uncommon. Most cases have been associated with ingestion of seed grain coated with an organic mercury fungicide (Radostits et al., 1994). Fish meal protein concentrates also have accidentally caused mercury poisoning. Fish concentrate methyl mercury that might be in the water (Annett et al., 1975). The organic mercury compounds, especially methyl mercury, are more toxic than the inorganic forms of mercury (Potter et al., 1972; Ansari et al., 1973; Sell and Dawson, 1973). Organic mercury compounds are more efficiently absorbed and are retained longer (Ansari et al., 1973). Because they are more lipophilic, they tend to cross the blood-brain barrier easier resulting in greater neurologic problems. Little organic or inorganic mercury is secreted into milk (Sell and Dawson, 1973; Potter et al., 1972; Neathery et al., 1974).

Inorganic mercury compounds are very caustic and cause acute gastroenteritis when ingested. Low doses of inorganic mercury ingested over time cause depression, anorexia, and a stiff-legged gait followed by paresis. Alopecia, pruritus, scabby lesions around the anus and vulva, shedding of teeth, and diarrhea are typical of later stages of inorganic mercury poisoning (Radostits et al., 1994). The primary cause of death is acute renal failure (Bulger and Siegel, 1975).

The organic mercury compounds (alkyl mercuries) primarily affect the nervous system and clinical signs are similar to those seen in calves with polioencephalomalacia: listlessness, incoordination, progressive blindness, and convulsions. However, animals poisoned by organic mercury compounds do not respond to thiamin (Oliver and Platonow, 1960; Herigstad et al., 1972; Davis et al., 1965).

A single 8-g dose of mercuric chloride was toxic to cattle (Radostits et al., 1994). Calves fed a diet of milk containing 10 mg of mercury from methyl mercury/kg of milk died in 36 to 81 days, whereas calves receiving 2 to 4 mg of mercury as methyl mercury /kg of milk remained clinically normal (Herigstad et al., 1972). The suggested maximum tolerable concentration of dietary mercury in organic or inorganic form is 2 mg/kg (National Research Council, 1980).

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