

Salt and Trace Minerals for Livestock, Poultry and Other Animals
(printable version)

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THE ROLE OF SALT IN ANIMAL NUTRITION

Historical need for salt

For thousands of years it has been known that domestic and wild animals need salt just as man does. The virtues of salt for animals were extolled by the ancient Greeks. Early explorers in Africa, Asia and North America recorded observations of grazing animals traveling to salt springs or deposits to satisfy ravenous appetites for salt. Animals deprived of salt will risk grave danger or resort to unusual behavior to obtain it. Considerable evidence exists that early nomads and hunters took advantage of this fact to lure and capture animals by locating areas with salt and waiting for animals to come there periodically. That livestock and poultry need salt was recognized long before scientific knowledge of foods or nutrition became available. In the early 1800s the value of salt for experimental animals was demonstrated. Since then, many studies have been conducted and a summary of these results are reported herein.

The role of salt in animal nutrition

Common salt contains both sodium and chloride and is also called sodium chloride. Salt is unique in that animals have a much greater appetite for the sodium and chloride in salt than for other minerals. Because most plants provide insufficient sodium for animal feeding and may lack adequate chloride content, salt supplementation is a critical part of a nutritionally balanced diet for animals. In addition, because animals have a definite appetite for salt, it can be used as a delivery mechanism to ensure adequate intake of less palatable nutrients and as a feed intake limiter.

Even though the body only contains about 0.2% sodium, it is essential for life and is highly regulated. About half of the sodium in the body is in the soft tissues of the body; the other half in bones (129). Sodium makes up about 93% of the basic mineral elements in the blood serum and is the chief cation regulating blood pH. The ability of muscles to contract is dependent on proper sodium concentrations. Sodium plays major roles in nerve impulse transmission and the rhythmic maintenance of heart action (129). Efficient absorption of amino acids and monosaccharides from the small intestine requires adequate sodium (184).

The other nutrient in salt, chloride, is also essential for life. Chloride is the primary anion in blood, and represents about two thirds of its acidic ions. The chloride shift, movement of chloride in and out of the red blood cells, is essential in maintaining the acid-base balance of the blood. Chloride is also a necessary part of the hydrochloric acid produced by the stomach which is required to digest most foods.

Unfortunately, it is often assumed that if the sodium requirement is met, the chloride requirement will automatically be met also. However, recent evidence indicates this may not always be the case. For example, Belgian studies showed a close correlation between potassium and chloride in the urine of cows (72). They concluded that the necessity for the ruminant to eliminate high amounts of dietary potassium (as potassium chloride) can dramatically increase the chloride requirement. Therefore, since many ruminant feedstuffs are quite high in potassium, the potassium-to-chloride ratio in the diet is important.

In monogastrics, a chloride deficiency can also develop when low levels of salt are fed. Leach and Nesheim, (76) reported that a chloride deficiency in chicks results in extremely poor growth rate, high mortality, nervous symptoms, dehydration and reduced blood chloride.

THE NEED FOR SALT

Signs of a salt deficiency

When salt intake is below that required to meet the animal's need for sodium and chloride, the animal adjusts by conserving (77). Urine output of sodium and chloride nearly stops. A continuous low salt intake affects the health of animals through a loss of appetite and weight. Feed utilization decreases and it takes more feed per unit of gain or product produced (78, 83, 84, 128). Animals soon develop a craving for salt. They may consume considerable amounts of dirt, wood, rocks and other materials. They will also lick manure and urine in an attempt to obtain the needed salt. Lactating animals are most susceptible to a salt deficiency because milk contains a considerable amount of sodium and chloride. Because the composition of milk is highly regulated, a deficiency of sodium or chloride in the diet will ultimately decrease milk production.

Factors affecting salt needs

Many scientists have shown that the salt needs of animals vary. Some of the factors that influence salt needs are as follows:

1. Diet can have a great impact on the salt needs of animals. Diets containing different amounts of concentrates, pasture, hay, silage or byproduct feeds account for much of the variation in salt requirements due to the wide range of sodium and chloride concentrations.
2. The level of sodium, chloride and other minerals in the water is another important factor. Animals typically will consume 2-3 times as much water as dry food. Locality can have a major impact on the minerals present in the water and, thus, the need for salt.
3. Level of production can have a great influence on the need for supplemental sodium and chloride. For example, cow's milk contains approximately 630 ppm sodium and 1150 ppm chloride. As milk production increases so does the need for salt (130, 131). A Canadian study (123) showed that lactating gilts consumed twice the sodium chloride of open gilts of the same age. Increases in rate of growth, reproduction, egg production, etc. will all increase the need for these minerals.
4. The temperature and/or humidity can be an important factor. The University of Florida (130, 131) showed that heat stress increased the need for potassium in the diet of high-producing dairy cows. Increased milk production occurred due to 1.5% potassium in the diet. Texas studies verified the Florida finding on a need for up to 1.5% potassium for maximum milk production during hot weather (139). The Florida studies also showed that sodium needs were increased with the higher levels of potassium in the diet (130, 131).

During heat stress, certain animals can lose large amounts of sodium through sweating. For example, working horses have been shown to increase their salt consumption five-fold during heat stress (31). Providing free-choice salt is the best way to meet individual needs in this situation.

5. The sodium concentration of the same feedstuff grown in different areas can be highly variable. This results in different supplemental sodium needs even though the diets may be similar. A recent

survey (185) has shown that sodium concentrations for feedstuffs given in the third revision of the U.S.-Canadian Tables of Feed Composition are often 2-3 times greater than values being obtained by commercial laboratories. Consequently, the animal's requirement for supplemental sodium may well be greater because the concentration in the basal diet is overestimated.

6. Availability of sodium and chloride in feeds may be over-estimated. Recent work with forages suggest that mineral availability decreases with plant maturity because more and more of it is associated with the indigestible fiber fraction.

7. Potassium concentration in the diet can influence requirements for sodium and chloride. Sodium is required in the kidney for potassium conservation and to balance bicarbonate excretion electrically (186). An excess of potassium can aggravate a marginal sodium deficiency. This can even occur when high forage (pasture, hay or silage) diets are fed. For example, certain pastures may have up to 18 times more potassium than sodium. This helps explain why cattle choose to consume more salt on high forage diets than on high concentrate diets.

Adding supplemental potassium to the diet can have the same effect. Recent research from Florida showed that adding potassium to reduce heat stress markedly increased the sodium requirements of the lactating cow (131).

8. The concentration of chloride and/or sulfate in the diet can impact the sodium requirement. Cornell studies showed that excessive levels of sulfate or chloride ions depressed growth in the chick unless equimolar amounts of sodium and potassium were also supplied in the diet (59). Their studies provide a possible explanation for why animal performance may be enhanced with salt additions, even when sodium and chloride concentrations are above the NRC requirement.

9. Recent studies with poultry indicate that higher levels of sodium and chloride may be required for normal immunity and maximizing resistance to diseases (187) than is required for maximum growth. Most nutrient requirement studies are conducted under conditions to minimize stress from disease or the environment. It should not be surprising that requirements for sodium and/or chloride may be increased in less than optimal conditions.

10. Genetic differences in animals affect salt requirements. As we select animals for maximum performance while being fed diets with greater caloric density, sodium and chloride concentrations required to achieve maximum performance may be increased.

These factors help explain why salt needs vary among localities and with different feeding and management situation.

Salt and Coping with Stress

Modern production agriculture exposes animals to environments that they would not usually be exposed to in the wild. Although efforts are made to minimize the stress these animal experience, some animals do experience increased stress which is reflected in their endocrine profile. Recent research suggests that the changes in hormonal profile may cause an increased appetite for sodium. This increased appetite for sodium may encourage stereotypies behavior.

In this review, the term “stress” as applied to farm animals is a potential damaging stimulus that evokes a largely adaptive response (349). Stress is a normal part of animal life. Animals raised in the wild are exposed to a lack of food, heat, cold, antagonistic social interactions, predators, etc., all of which cause stress. The point is that animals will experience stress in both “natural” and “production” settings.

Stress and Behavior:

Stress encourages stereotypies behavior in laboratory and farm animals. Stereotypies is defined as behavior of an unvarying, repetitive nature with no direct purpose (353). Rats when they become sodium deficient exhibit stereotyped fixed action patterns that are ingestive in nature (348). Sodium deficient cattle frequently display excessive licking behavior (355). Cattle that are tethered in a restricted area or raised individually as calves in isolated stalls, exhibit similar licking behaviors.

In the past few years scientist have learned a great deal about how hormonal changes resulting from stress can affect brain chemistry and behavioral changes. Animals respond to stress by releasing adrenocortiotropic hormone (ACTH) from the anterior pituitary gland. The ACTH then causes the adrenal cortex to release aldosterone and corticosterone. Aldosterone is the main hormone that controls sodium balance by changing the kidney’s reabsorption of sodium and thus the amount excreted in the urine. Corticosterone increases blood glucose and carbohydrate metabolism to supply energy. These hormones also act directly on the brain through the activation of the neuropeptide angiotensin II. Angiotensin II is a powerful stimulus for thirst and sodium appetite (351). When it is injected directly into sensitive areas of the brain, it causes an immediate increase in water intake followed by a slower increase in sodium intake. However, the appetite for salt is more persistent and may be affected by previous experience. Some researchers believe that the angiotensin II may influence neuronal organization in the brain that can cause long-term changes in sodium appetite (351). Stress has been shown to increase the salt appetite in rats, mice, rabbits and sheep.

Cattle:

Phillips et al., (354) conducted an experiment to determine whether salt intake influenced the behavior of cattle in stressful environments. In this experiment, 36 Estonian Red dairy cows were allocated to three treatments, 0, 200, or 400 grams of salt added to a standard winter ration, daily. The basal diet was grass silage and ground barley. The final diets contained 1.0, 6.0 and 11.0 g sodium/ kg dry matter for the control, low and high sodium diets, respectively. The salt supplements were mixed with the barley and no feed refusals observed. Cows were individually housed and milked twice daily in their tied stalls. Each cow was observed for a total of 18 5-minute periods and the amount of time doing various behaviors recorded. Stereotypies behavior recorded included: mouthing the feed trough bars or tethering chain, rubbing against feed trough bars or tethering chains, pawing the ground or self-grooming. None of the individual stereotypies behaviors was significantly affected by sodium level, but collectively there was a reduction in total time spent in stereotypic behavior at the high sodium level. The fact that stress increases the sodium appetite of other herbivores suggests that the reduction of stereotypies measured in this experiment may be a consequence of the physiological relationship between stress and sodium status.

In a second experiment (354), 16 British Friesian female calves were selected at birth and allocated to pairs of similar weight. Within each weight, calves were assigned to no additional salt or 13.5 grams of salt/kg of concentrate fed. Adding the salt to the concentrate increased the sodium concentration from 4 to 9 g sodium/kg concentrate. Calves were housed in individual pens and weighed weekly for 6 weeks. Behavior was recorded for 12 hours after the calves received their concentrates on day 1 of each week. Adding sodium to the concentrates increased feed intakes, water intakes, and live weight. Calves with supplementary sodium spent less time grooming themselves, licking the pen, licking the buckets and ear sucking. The stereotypic behavior was more pronounced in calves than in the cows in the previous experiment. The sodium intake of the control treatments was greater than the requirement given by the British Ministry of Agriculture. In that sense they were not sodium deficient diets. However, the stress experienced may have increased the desire for sodium that resulted in behavior patterns associated with stress. Increasing the sodium level was helpful in controlling abnormal behaviors.

Pigs:

Abnormal behaviors may also be influenced by sodium levels in the diets of other farm animals. For example, tail biting in finishing pigs can be a real problem in high-density confinement buildings. Tail biting begins with the occasional chewing of another pig's tail. Once a wound has been established, the biting becomes more frequent and intense. Docking the tail at birth has become standard practice to try to avoid this problem later.

Diets containing less than 0.3% salt are associated with high levels of tail-biting (352). Most swine nutritionists recommend 0.5% salt in the diet. However, salt concentrations are often raised to 1% of the diet following an outbreak of tail biting. Other factors that may contribute to tail biting in pigs include protein deficiency, amino acid imbalance, thermal stress, high ammonia levels, overcrowding, large group sizes, and poor ventilation.

Because blood is relatively high in sodium, some researchers have proposed that tail biting was an effort to find more sodium. Canadian researchers (356) have tried to determine if this was the case by allowing pigs access to ropes (similar to pig tails) soaked in blood, salt water, and pure water. The pigs were given ACTH injections to simulate stress conditions. In this study the blood-soaked ropes were the most popular, but there was no difference in the number of pigs that preferred the salt water and pure water ropes. This suggests that salt taste may not be the only factor that makes blood attractive to stressed pigs (356).

Poultry:

A similar problem associated with a salt deficiency in poultry is cannibalism. Cannibalism occurs when birds peck at the feathers, toes, heads, and vents of other birds. If there is bleeding and further pecking, it may result in the death of the bird. Poultry nutritionists often recommend that the diet contain 0.15 to 0.20% sodium to minimize cannibalism. If cannibalism does become a problem, sometime it can be controlled by adding 5-10 grams of salt per gallon of drinking water. Other factors that can contribute to cannibalism include vitamin and amino acid deficiency, feed deprivation, over-crowding, over-heating, inadequate ventilation and bright lighting.

Humans:

The drive to consume adequate sodium can have a powerful influence on behavior. Dr. Derek Denton in his book *The Hunger for Salt* builds a strong argument that the incidence of cannibalism in primitive people was highly correlated with a lack of sodium in their diet. Cannibalism was most common in the tropical areas of the world that lacked access to salt. The equatorial jungles and mountains are noted for their very low sodium status. Requirements for sodium were also increased in these hot environments due to its loss in sweat.

Production environments that increase the stress in farm animals will also increase the appetite for sodium. The endocrine changes in the brain as a result of stress will stimulate the appetite for salt. Stereotypic behaviors that seem to have no meaningful purpose may be aimed at increasing the contact with and consumption of sodium sources. Salt requirements have been determined in production settings where efforts have been made to minimize stress. Sodium levels required to minimize undesirable animal behaviors in stressful environments may be greater than that required to meet nutrient needs in a low stress production settings. In that sense animals may have two sodium requirements, one to maximize animal production and another to modify behavior.

Overestimation of Sodium in Feeds

Previous researchers (1,2) have noted that the sodium concentrations reported in NRC feed composition tables were inflated for many common feedstuffs relative to values being reported. Minson (217) reported that the distribution of sodium concentrations in pasture samples was skewed to the low values. In this study 50% of the samples contained less than 1.5 grams of sodium per kg dry matter. Other researchers had shown that the sodium concentration decreased rapidly in some forages as they mature. Morris (218) found that California rangeland pastures in September only contained 0.1 to 0.2 grams of sodium per kg dry matter while the same pastures in the spring contained 0.5 grams of sodium per kg dry matter.

With this in mind, a survey was conducted to compare the mineral concentrations of common feedstuffs as determined in commercial laboratories with NRC values as reported in the third revision of the United States-Canadian Tables of Feed Composition. Data from laboratories in New York, Indiana, Idaho and Arizona were pooled and summarized. Feedstuffs from all 50 states were analyzed by these laboratories, but no attempt was made to summarize the data by region. Of all the minerals analyzed, sodium was found to be consistently below the NRC values. For example, the sodium concentration in corn was 23%, barley 47%, oats 12%, wheat 18%, corn silage 31%, alfalfa hay 10%, distiller's grains 57%, brewer's grains 16%, and whole cottonseed only 3% of the NRC value. With most feedstuffs, over 100 samples were analyzed and with corn silage and alfalfa over 3500 samples were involved. Soybean meal was the only common feedstuff compared where sodium was equal to or greater than NRC values (185).

More recent data suggests that sodium concentrations in grazed forages are also lower than many book values. Between 2001 and 2004, 1021 forage samples harvested by county agents across the state of Tennessee were analyzed for sodium (293). The average sodium concentration was 0.01%. Because most of these samples were not from pure stands of forage, it is difficult to compare with book values. However, 0.01% is below book values for most grasses.

The reason for this discrepancy is difficult to explain. Regional difference could be a contributing factor since most of the feeds were grown in the eastern half of the U.S. However, this logic does not hold for feedstuffs like whole cottonseed or wheat that were from the South and High Plains regions, respectively. One explanation that has been offered, but not confirmed, is that with some of the early feed analysis procedures, chlorides were measured and then multiplied by 0.649 to get the sodium concentration (188). This assumed that sodium was present in equal molar concentrations to chloride. The practical ramification of this information is that many diets formulated for specific sodium concentrations are actually below the desired amount because NRC values were used in the formulation.

Salt in water

Water quality is an issue receiving national attention. High quality water is essential for successful animal production systems. Rarely is salt the only mineral in high concentrations in saline water. Besides sodium and chloride, calcium, magnesium, bicarbonate, sulfate and nitrate ions are commonly present. Each ion may exert its own specific toxic effects separate from osmotic effects normally associated with the total dissolved solids. The nitrate ion is a good example.

The amount of sodium and chloride tolerated in drinking water is largely determined by the total soluble salt content of the water. Consequently, the NRC (189) guidelines for the use of saline waters by livestock and poultry are based on the total soluble salts (Table 1). The NRC committee (189) suggests the following points should be considered when making recommendation based on Table 1.

Table 1. A Guide to the Use of Saline Waters for Livestock and Poultry (189)

Total Soluble Salts Content of Waters, ppm	Comment
Less than 1,000	These waters have a relatively low level of salinity and should present no serious burden to any class of livestock or poultry.
1,000 – 2,999	These waters should be satisfactory for all classes of livestock and poultry. They may cause temporary and mild diarrhea in livestock not accustomed to them or watery droppings in poultry (especially at the higher levels) but should not affect their health or performance.
3,000 – 4,999	These waters should be satisfactory for livestock, although they might cause temporary diarrhea or be refused at first by animals not accustomed to them. They are poor waters for poultry, often causing watery feces and (and the higher levels of salinity) causing increased mortality and decreased growth, especially in turkeys.

5,000 – 6,999	These waters can be used with reasonable safety for dairy and beef cattle, sheep, swine, and horses. It may be well to avoid the use of these waters approaching the higher levels for pregnant or lactating animals. They are not acceptable waters for poultry, almost always causing some type of problem, especially near the upper limit, where reduced growth and production or increased mortality will probably occur.
7,000 – 10,000	These waters are unfit for poultry and probably for swine. Considerable risk may exist in using them for pregnant or lactating cows, horses, sheep, the young of these species, or for any animals subjected to heavy heat stress or water loss. In general, their use should be avoided, although older ruminants, horses and even poultry and swine may subsist on them for long periods of time under conditions of low stress.
More than 10,000	The risks with these highly saline waters are so great that they cannot be recommended for use under any conditions.

(1) Alkalinities and nitrates should be considered when using water containing more than 3,000 ppm total salts. Hydroxides are more detrimental than carbonates that are, in turn, more detrimental than bicarbonates. (2) Animals can consume high salinity water for a few days without risk if given access to good quality water thereafter. (3) If given a choice between highly saline and good quality water, animals will not drink the saline water. (4) Water consumption will usually increase as salinity increases up to the point where they refuse to drink. (5) Depressed water consumption will decrease feed intake and reduce performance. (6) Abrupt changes from low salinity to high salinity water will have greater detrimental effects on animal performance than gradual changes.

Information on the tolerance of livestock and poultry for sodium chloride in the water will be given in each species section. This information should be considered as one evaluates the tolerance of farm animals to sodium chloride in drinking water.

Salt in manure

Salt concentration in manure is a matter of concern only with the confinement rearing of livestock and poultry. Usually, the reference is to total mineral salts in the manure, not just sodium chloride (salt). The percent "salt" in manure is estimated by adding the percents potassium, calcium, sodium and magnesium and then multiplying by 2 (190). In most cases, the order of contribution to the salt concentration is potassium, calcium, magnesium, sodium. Often the contribution of sodium chloride to the total salts concentration in manure is overestimated. In fact, sodium accounts for only about 7% of the four major salts in manure from livestock and poultry (Table 2). Consequently, if the total salt concentrations contribute to a waste disposal problem, then diets should be evaluated as to the optimal concentration of each mineral before sodium chloride is removed.

Table 2. Estimated Quantities and Constituents of Livestock and Poultry Manures Produced Yearly¹ (190)

Animal		Quantity	Per	Animal-	Year		
Type	N²	P	K	Ca	Na	Mg	COD
Dairy	123	21.0	98	72.0	15.0	22.0	3,340
Beef	61	18.0	39	11.5	4.2	5.7	1,510
Swine	32	7.4	11	11.0	1.9	2.9	416
Layers ³	94	40.0	40	170.0	18.0	13.0	1,741
Broilers ³	78	22.0	25	91.0	9.2	9.2	1,183
Turkeys ³	304	84.0	99	355.0	36.0	36.0	4,599

¹ Manure production was derived from ASAE standards and Midwest Plan Service and Gilbertson et al. The values are commonly used for calculating storage volume and equipment requirements and do not indicate quantities available for land application. Based on average animal weight as follows: Dairy and Beef, 1,000 lb; swine, 200 lb; sheep, 100 lb; layers 41 lb; broilers, 2 lb; and turkeys, 10 lb. These values do not include bedding or materials such as spilled feed, soil or water from precipitation. Neither do they reflect the decomposition processes that start as soon as the manure is voided by the animal.

² Nitrogen (N), phosphorus (P), potassium (K), calcium (Ca), sodium (Na), magnesium (Mg) and chemical oxygen demand (COD)

³ Per 100 birds

Several research studies have been conducted to evaluate the effect of sodium chloride in manure on crop yields when manure is applied at various rates. Soil type, rainfall and crop grown are the major agronomic factors determining the optimal manure application rates. Under the conditions of west Texas, Texas Tech studies have shown that 10 to 15 tons per acre every 3 to 4 years will not affect crop yields or runoff problems (49, 51). In contrast, Michigan studies with sandy loam soil showed 10 tons per acre yearly to be the optimal application rate (50). This loam soil could handle 10 to 15 tons of manure annually, without nutrient accumulation, especially if the corn was harvested as silage.

A new concept that is being researched in areas where ground water contamination is not a problem, is for the livestock producer to rent the land for 1 to 3 years and apply very high levels of manure. The farmer can grow crops on the land during this period but recognizes that some reduction in yield may occur. Crops that can tolerate saline soils such as bermudagrass, tall wheatgrass, barley, cotton or sugar beets are best suited for this system (190). For example, Texas A&M has experimented with adding up to 900 tons of manure per acre in the El Paso and Pecos, Texas area. Crop growth was achieved at this application rate, but special management considerations are required for this approach to be used successfully.

In addition to the solid manure, feedlot runoff needs to be monitored. Often the runoff is collected in a settling basin and the water is used for irrigation purposes. Using runoff from the Pratt Feedlot, Kansas State researchers showed that approximately 20 inches of water could be applied before corn yields were depressed due to soil salinity (191). USDA scientists at Nebraska reported that application of up to 24 inches of feedlot runoff per irrigation season did not cause a build up of nitrate, nitrogen or salts in the soil after two years of application.

These and other studies show that dietary sodium chloride concentrations required for optimal performance does not create major manure disposal problems. If the total salt level in manure does restrict land application rates, the concentration of potassium, calcium and magnesium in the diet may be adjusted to bring it into an acceptable range.

Appetite for salt

Animals have a more well defined appetite for sodium chloride than any other compound in nature except water. Ruminants have such a strong appetite for sodium that the exact location of salt source is permanently imprinted into their memory which they can then return to when they become deficient. Bell (133) showed that when steers were trained to receive their sodium in response to pressing a panel, maximum effort to receive the sodium occurred at eight days and after, on a sodium deficient diet. Cattle also have a keen sense of smell for sodium (192). Sodium deficient steers were offered a cafeteria of 12 buckets of water with only one containing moderate levels of sodium salts. Steers would quickly choose the water containing sodium salts without having to taste the water sources.

Horses have been shown to have a specific appetite for salt if the diet is deficient in sodium (132). This is not true for the other nutrients. For example, horses do not develop a preference for calcium supplements when fed a calcium deficient diet (193). This natural appetite for salt is what makes salt such an excellent delivery mechanism for other nutrients that need to be consumed regularly, but where a natural appetite is lacking. The 1984 NRC Beef Cattle committee (157) recognized this fact in stating that minerals lacking in the diet can be provided by "self-feeding" common salt-mineral mixtures when the mixture is consumed in amounts to satisfy the animals' appetite for salt.

Cattle are also able to detect when they are chloride deficient. In a Cornell study, lactating cows fed a chloride deficient diet (.18% chloride) consumed 337 grams of salt a week while those fed the chloride adequate diet (0.40% chloride) ate 149 grams per week (134). Sodium levels were adequate so that cows were consuming the salt to satisfy their chloride requirement.

Chloride level needs attention

The study described above shows the risk of assuming that if the sodium requirement is met then the chloride level is also automatically met. Fettman et al. (194) reported that lactating dairy cows fed a diet containing 0.10% chloride developed clinical deficiency signs characterized by reduced feed intake, body weight, and milk production, lethargy, cardiovascular depression and mild dehydration. Many vegetable protein supplements are low in chloride (0.3-0.7 grams/kg dry matter). It is quite possible that mixtures of the protein supplements and cereal grains can be deficient in

chloride for pigs and poultry (219). More attention needs to be paid to diet chloride levels, especially where some of the sodium needs are being met with sources other than NaCl.

SALT FOR BEEF CATTLE

Required for Optimum Nutrition

Feed represents about 70% of the total cost of beef production. Numerous studies have shown that sodium chloride supplementation is an essential part of a balanced diet required for efficient beef production. If diets are deficient in salt, the producer risks significant economic loss long before outward deficiency symptoms appear. For example, in a Kansas State trial, calves were wintered on silage plus one pound of a soybean meal pellet daily. Those calves that were self-fed salt gained 1.26 pounds daily; while those not receiving any salt only gained half as much 0.65 pounds per day (9). Calves fed salt consumed 28.5 pounds and those without salt 26.1 pounds of feed daily, so that the amount of feed required per unit of gain was almost doubled when salt was absent.

In another Kansas study, steers fed salt gained 65 pounds more than those without supplemental salt over a 327-day period (9). In a 1971 trial, steers on full feed consumed about 5 cents (20 cents in 2000 dollars) worth of extra feed, but gained approximately 50 pounds more than steers not receiving salt (22).

A salt deficiency can also cause cattle to choose an imbalanced diet in order to get additional sodium or chloride. For example, Harbers et al. (23) reported that when feedlot heifers were fed whole shelled corn and supplement separately, cattle without any supplemental salt consumed 1.15 pounds more protein supplement per day compared to those receiving free-choice salt. The supplement contained 0.26% sodium compared to the 0.01% sodium in the corn. Since the supplement was the most costly ingredient in the diet, costs of gain increased significantly due to a lack of salt.

A 1973 Australian report (64) showed that salt supplementation significantly increased the gains of lactating beef cows and their calves while grazing native pastures.

In the examples discussed above, significant improvements in performance and reductions in the costs of production occurred with salt supplementation. It is important to note that these improvements were obtained prior to any deficiency symptoms.

Deficiency Symptoms

Usually the first sign of a salt deficiency in beef cattle is a craving or abnormal appetite for salt. Cattle may lick various objects such as rocks, wood, soil and the sweat of other animals. Sometimes, no other visible symptoms occur for months. Then appetite begins to decline, the animal subsequently develops an unthrifty appearance with a rough hair coat. This is followed by a rapid loss of body weight and a reduction in milk yield. Eventually, a sudden death will follow a prolonged salt deficiency.

Salt in Grazing Distribution

Because cattle have a very strong appetite for salt, it can be used as a management tool as well as a source of nutrient. For example, salt blocks or salt stations can be used to increase the carrying capacity on range by causing more even grazing. This procedure causes animals to go to lightly used forage areas that have less succulent or less palatable forage and to less accessible areas in rough or wooded terrain or farther from water or shade. This method was successfully used with salt-protein supplement mixtures by the USDA Woodward, Oklahoma Station, where salt-protein mixtures placed up to three miles from water helped distribute grazing over the range (25). Other scientists have also placed salt blocks in less frequently utilized forage areas to increase forage use (63). When salt was placed approximately 1.5 - 2.5 miles from water, range utilization was improved by increasing grazing 12% on the under-utilized area and reducing grazing 10% on the over-utilized area closest to the water. In many mountainous or inaccessible areas, salt blocks or complete mineral blocks that include salt are dropped by plane to get minerals to the animals and help distribute grazing (60). However, recent Oregon research suggests that sodium concentration in the forage may regulate the effectiveness of using salt to improve grazing distribution (320).

Salt as an Intake Regulator

The 1996 Beef NRC states, “high concentrations of salt have been use to regulate feed intake” (278). However, the use of salt to control the intake of protein and energy supplements on pasture is not a new concept. Recent research and changing economics in the beef industry have caused a resurgence of interest in this topic.

With the increased selling price of feeder cattle, the economics of supplementing protein and energy to grazing cattle has become much more favorable. With feed costs at approximately 50 cents per pound of gain, and selling price in excess of 75 cents per pound, the profit potential for feeding supplements on grass is obvious.

In addition, there are always those situations where due to drought or over stocking, supplemental feed on pasture is needed to extend the carrying capacity of the range. When feed supplies are short, it is impossible to achieve uniform intakes with hand-fed supplements because dominant animals often get much more than their portion while timid animals may get none. In all these situations, opportunities exist to use salt to control the intake of a self-fed supplement without the large investments in facilities and labor required to hand-feed a limited amount of supplement on grass.

Numerous research studies over the past 40 years have demonstrated the efficacy of using salt to control intake and support performance equal to hand-feeding. Meyer et al. (118) fed a diet containing approximately 50% forage and 50% concentrate with or without 9.3% added salt to finishing steers. At the end of the 84-day feeding period, average daily gain, feed efficiency and dressing percentage were nearly identical for both groups. Steers fed the high salt diet consumed 7.0 gallons more water per day and tended to have heavier kidneys per unit of carcass weight. These researchers did demonstrate that because of the increased water intake, digestive tract fill will be increased for several hours after a meal. However, if cattle are fed normal salt intakes, differences in gut fill are eliminated in a short time as indicated by the equal dressing percents for steers on both treatments.

Salt, as high as 30% of the supplement, has been used to restrict supplement intake to 1.9 pounds per day (21). This high level of salt was required because the Pangola grass pastures were of poor quality containing only 5.5% crude protein. Steers fed the salt-limited supplement gained 1.04, while steers hand-fed the same amount of supplement, minus the salt, gained 1.12 pounds per day. In this study there was no difference in blood sodium, hemoglobin or hematocrit due to high salt intakes. Feeding regimes used in this trial did not affect dressing percentage, rib-eye area or carcass grade when the cattle were slaughtered directly off grass.

More recently, Harvey et al. (117) compared the efficacy of using salt to limit supplement intake in hay and corn silage based diets in two different years. Soybean meal based supplements were used containing approximately 28% salt, with an intake of about 1.1 pound of soybean meal per day. This high level of salt did result in considerable variation in individual intakes of supplement and somewhat reduced gain on the corn silage based diet during year one of the study. However, performance was nearly identical for steers fed the two hay diets during the first year and on both the corn silage and hay diets during the second year.

In this trial, rumen parameters were monitored to determine how salt may affect rumen fermentation. One could theorize that increased consumption of water associated with salt intake may increase rate of passage of undigested residue from the rumen resulting in increased intake. In this study, rumen liquid dilution rates were increased from 6.3% to 10.4% per hour on the corn silage diet and from 7.8% to 9.1% per hour on the fescue hay diet due to salt intake. However, solid dilution rate was not affected which explains why there was no difference in forage intake. Previous studies (19) showed that cows fed alfalfa hay based diets containing 10.75% salt did not reduce cellulose digestibility compared to cows fed the same diet without salt. Similarly, Chicco et al. (21) showed that cellulose and protein digestibility was not affected by using salt to control intake.

Salt additions to the diet can change the rumen fermentation pattern. Rogers et al. (119) infused 500 or 1,000 grams of salt per day into the rumen of Holstein steers fed high roughage or high concentrate diets. These levels of salt infusion are equal to 5.2% and 9.5% of diet dry matter, respectively. With the high concentrate diet, molar percent acetate in rumen fluid increased and molar percent propionate decreased due to salt infusion. Acetate:propionate ratio was not changed on the high roughage diet. These data suggest that with steers fed high concentrate diets, some starch or soluble carbohydrates are flushed out of the rumen due to increased liquid dilution rate, resulting in fermentation higher in acetate. These changes in rumen fermentation could be beneficial to lactating dairy cows by helping to increase butterfat in milk, but would have little impact on performance of grazing cattle.

While the "science" of using salt to regulate intake has been adequately researched, the "art" of using this technology is still developing. The following is a list of pertinent considerations to keep in mind as one develops a system to use self-fed, salt-limiting mixtures for cattle and sheep.

First, the proportion of salt in the self-fed mixture may vary anywhere from 5% to 40%. To determine how much salt is needed, you first need to know the desired amount of supplement intake. If you want to restrict intake to 1 to 2 pounds per day, as high as 30% to 40% salt may be required with mature range cows. Yearling cattle grazing high quality forage may require only 5% salt initially to limit intake to a pound per day. However, as cattle grow and the grass gets more

mature and less plentiful, 20% to 30% salt may be required to maintain desired levels of intake (117). California researchers found that 8% to 10% salt was required to limit concentrate intake to 1% of body weight for yearling steers grazing lush, irrigated pastures.

From these data, it is obvious that factors other than desired intake also affect the concentration of salt required. These include such factors as age and weight of the animals, in that older animals require more salt to obtain the same level of restriction. As quality and quantity of the forage decreases more salt will be required. As animals become accustomed to the salt mixture, it may be necessary to increase the proportion of salt. The finer the grind of the salt being used, the less is required to obtain the same restriction. As salinity of drinking water increases less salt is required. As weather becomes more severe, more salt is required because animals are less prone to graze.

When used to control intake, salt mixes are most effective when fed in meal form. Pelleting feeds prevents the salt from inhibiting intake as rapidly and can result in overeating. Similarly, if animals are very hungry, it is recommended to hand-feed the salt mixture for a week before allowing free-choice access. During severe feed restriction, some animals may over consume if they are not adjusted to the salt mixture gradually.

A clean, plentiful water supply is a must when using salt to control intake. Water requirements can easily increase by 50% to 100% when using this system. During severe cold or blizzard, when stock tanks or ponds may freeze and animals tend to increase their intake of the salt mixture, adequate water is required to prevent the risk of salt toxicity. However, it is best not to place the salt mixture next to the water supply as it can restrict grazing distribution on the range.

Self feeders should protect salt mixtures from wind and rain and be portable. Usually about 20% of the animals should be able to eat from the feeder at one time. By knowing initial volume and weight of the salt mixture placed in the feeder, and marking the level of feed in the feeder every few days, one can calculate the amount being consumed per head per day. This will allow salt concentrations in future batches to be adjusted without having to let feeders become empty before intake per head can be determined. If it is important to keep intake at a constant level, self feeders should be filled every two to three weeks to allow for adjustments in salt concentrations. It is not uncommon to adjust salt concentrations 4 to 7 times to maintain desired intake over a normal grazing season.

Recent research (137) has shown that the number of adjustments in salt concentration required to maintain the desired intake can be cut in half when monensin is included in the salt mixture. Monensin, marketed as Rumensin, is cleared as a feed additive to increase daily gain of grazing cattle. Another advantage of including monensin is that it allows the use of a salt concentration that reduces the animal to animal variation in intake. In the studies reported by Muller, (137) self-feeding a salt-monensin-supplement gave the same improvement in daily gain (0.2 pounds per day) as hand feeding the monensin supplement without salt. These data show that salt, an already proven intake regulator, can be made even better when combined with monensin. Although less data are available with lasalocid, a Georgia study showed that lasalocid fed in a free-choice salt and mineral mix increased the gains of replacement heifers, cows and calves (151). Using salt as an intake regulator of self-fed supplements on grass is one management tool that can increase the profit of many cattle producers.

Salt as an Attractant

By combining a salt feeder with a cattle rub in the same apparatus two functions can be accomplished simultaneously (323). Cattle rubs are filled with an oil-based insecticide to control external parasites. By attaching it to a salt-mineral feeder at one end and pole at the other the cattle are encouraged to use the rub whenever they consume salt. This approach is very effective at controlling parasites while the animal's mineral needs are being met. Because it is portable it can also be moved to improve grazing distribution.

Effects of High Salt Intake

Cattle can tolerate high salt intakes if adequate water is supplied. For example, in an Arizona study (10) two pounds of salt were placed directly into the rumen through a fistula. When given free access to water no toxicity symptoms developed. When the salt was mixed with three gallons of water and no other water offered, salt toxicity symptoms began in 8 to 12 hours. Symptoms observed were anxiety and hypersensitivity, followed by loss of coordination and finally collapse. Rumen contractions increased in rate and intensity. When the excess salt was flushed from the rumen, the cow recovered rapidly.

In Texas studies, cows were fed as much as 2.99 pounds of salt per day for 22 days without harmful effects (11, 13). High salt intakes had no detrimental effect upon the reproductive performance of the cows. Total chloride excretion of the "high-salt" cows was 11.5 times that of the controls, with 98.3% of the chloride intake excreted in the urine. Histological studies showed no kidney damage. The cows fed the high salt levels consumed considerably more water and excreted a much greater volume of urine than cows receiving only a normal salt allowance.

An Arizona study showed that the maximum salt (NaCl) concentration in the urine was 2.3% (14). Seawater has 2.6% NaCl. Consequently, for each pound of salt absorbed, the animal must produce about five gallons of urine to eliminate it (14). This is why plenty of water is needed when cows are fed high levels of salt.

A 1976 Purdue study showed that cattle offered ground corn mixed with 10% salt consumed an average of 1.4 pounds of salt daily with no harmful effects (61).

Hereford steers were fed high grain diets containing 0.5%, 3%, 5% or 7% NaCl for 126 days. Growth and feed efficiency were not affected by the level of NaCl, although feed intakes, organic matter intakes and carcass weights were reduced at the 7% level as compared to the 0.5% salt level (136).

Although high salt intakes are safe for cattle, a question often asked is whether it affects the utilization of other minerals. To answer this question, USDA conducted a study involving 700-pound steers fed a pelleted diet containing 1%, 2%, 4% and 8% salt (62). The study was designed to determine what the effect of high levels of sodium chloride (salt) would have on the retention of calcium, phosphorus, magnesium, sodium, potassium, chloride and water in beef cattle. The results were as follows:

1. Calcium - Calcium retention was increased significantly by each increase in the dietary salt up to the 4% level. Decreased fecal loss of calcium was responsible for this effect. There was no difference between the calcium retention on the 4% or 8% salt levels.

2. Phosphorus - Phosphorus retention was higher when 2% or 4% salt was fed than for 1% or 8% salt. The decreased fecal phosphorus excretion appeared to be primarily responsible for the difference in retention at the 2% and 4% salt levels, but this was offset on the 8% level by the increased loss of phosphorus in the urine.

3. Magnesium - Magnesium retention was not affected significantly by the salt level. Similar to the excretion of calcium and phosphorus, the majority of the magnesium excreted was in the feces.

4. Potassium - There tended to be less potassium retention on the higher levels of salt. The urine accounted for the majority of the potassium excreted. There was considerable variation between treatments, which made it difficult to analyze the data and show significant differences at the four salt levels used.

5. Sodium - The amount of sodium retained was higher for the 4% and 8% salt levels, but the percentage of sodium consumed that was retained was not significantly different among the four levels of salt. Urine sodium tended to increase with increasing level of dietary salt. The majority of the sodium and chloride is excreted in the urine.

6. Chloride - The 4% salt level caused the highest retention of chloride, both in quantity and percentage of that ingested. More chloride was retained at the 8% salt level than at the 1% or 2% salt levels, but the percentage retained was no different at the 2% and 8% salt levels.

7. Water - The amount of water retained was not affected significantly by the dietary salt. The amount of water consumed and urine excreted increased with increasing levels of salt in the diet.

This study indicates that feeding levels of salt much higher than ordinarily used in cattle feeding did not have any harmful effects on the cattle. In fact, in most cases mineral retention increased. The 1984 NRC publication recommends that 10% NaCl in the diet is the maximum tolerable level to use (157). Salt intakes should be controlled for the first few days if the cattle have been deprived of salt. Rapid ingestion of 2-6 lbs of salt can cause salt toxicity even when water is available, if they have been deprived of salt previously (292).

Salt Tolerance Levels in Water

In a series of studies at the University of Nevada, it was found that growing heifers could consume, during the winter, water containing 1% (10,000 ppm) NaCl without harmful effect if water was continuously available (15, 16, 17, 18). Water containing 2% salt (20,000 ppm) was toxic, however, and caused loss of appetite, weight loss and anhydremia (a deficiency of the fluid portion of the blood). Two percent is a high level of salt when one realizes that seawater contains 2.6% salt.

Since cattle on the range often drink infrequently, water with salt was offered only once daily or every two days in some Nevada trials. Heifers refused to drink water with 1% salt when available

only once per day or every two days. Subsequent experiments, however, showed that water with a level of 0.5% salt (5,000 ppm) was consumed when offered only once a day. The cattle continued to consume water once daily when the salt level was raised to 0.65%. However, feed consumption decreased under these conditions.

These studies indicate that cattle tolerate more salt in drinking water if it is continually available. This information is of value to those living near the ocean where salt intrusion into water supplies is possible. Work at South Dakota showed 7,000 ppm salt (NaCl) in drinking water was acceptable for beef cattle (55). A level of 10,000 ppm decreased rate of weight gain but did not cause other effects.

Both the Nevada and South Dakota studies indicate that a level of 7,000 ppm salt (NaCl) might be the top level to allow in drinking water for beef cattle. Other mineral salts in the water must also be taken into consideration. For example, sulfate salts are more harmful than chloride salts. In evaluating water quality, therefore, total dissolved solids in the water must be considered, not just NaCl. In many cases, sodium chloride is blamed for harmful effects actually caused by other mineral salts.

Salt Effect on Rumen Fermentation

Arizona digestion studies with cattle and artificial rumen fermentation experiments showed that the increased salt concentration of the rumen caused by feeding high-salt diets does not decrease rumen microbial activity (19). Both types of experiments showed that digestible cellulose, as well as digestible gross energy, is not altered by the increased salt consumption. The Arizona results have been confirmed in other research, including an Oklahoma study and a 1971 Venezuelan trial, where digestibility of nutrients was not affected by high salt levels (20, 21).

In many trials, the addition of salt to diets lacking salt has increased the digestibility of certain nutrients. This was shown in a number of Kansas trials (9, 24). Thus, it appears that the use of salt at high levels will not hinder digestibility of any nutrients, and it may increase the digestibility of some.

Salt Requirements

The sodium requirement for beef cattle is the primary factor determining how much salt should be fed. Morris (195) reviewed several papers and concluded that the sodium requirement was approximately 0.08% for growing cattle and 0.10% for lactating cattle. If the sodium was supplied from salt, this would be equivalent to 0.2% and 0.25% salt in the diet, respectively. The sodium requirement is increased with lactation because cow's milk contains approximately 630 ppm sodium. The sodium requirement will also be increased when cattle are fed diets that increase fecal water excretion (196). Fecal water is the major route of sodium loss for cattle fed lush forages. Lush forages are also high in potassium which will tend to increase sodium excretion. This undoubtedly is the major reason that grazing cattle and or cattle fed silages have increase voluntary salt intakes compared to cattle fed high concentrate diets. Table 3 contains Montana data showing the increased salt intakes of cattle fed high roughage (limited-fed grain) diets compared to those fed a full feed of grain (58).

Table 3. Average Daily Salt Consumption in Pounds by Calves, Yearlings and 2-Year Olds on Full and Limited Feed (58)

Year	Full-fed Grain-lb Calves	Limited-fed Grain lb Calves	Full-fed Grain-lb Yearlings	Limited-fed Grain lb Yearlings	Full-fed Grain-lb 2-Year Olds	Limited-fed Grain lb 2-Year Olds
1935-36	0.060	0.200				
1936-37	0.060	0.170	0.080	0.240		
1937-38	0.060	0.180	0.080	0.240	0.150	0.510
1938-39			0.080	0.300	0.110	0.310
1939-40					0.140 ¹	0.510 ¹
Ave. (lb)	0.063	0.183	0.080	0.260	0.133	0.443
Ave. (oz)	1.000	2.900	1.300	4.200	2.100	7.100
Ratio full fed to limited	1:2.90		1:3.25		1:3.33	

¹ In 1939-40 steamed bone meal was fed with salt (2 parts salt and 1 part bone meal). These data show much greater salt intake where cattle consume predominately roughage rather than grain.

Free-choice feeding of salt for grazing cattle is the safe recommendation. Lactating cows grazing lush forage will have a higher sodium loss than other classes of cattle so that free-choice feeding allows them to adjust their intake. Feedlot cattle should receive enough salt to provide approximately 0.08% sodium. Although additional salt may not be needed routinely (317, 318), higher levels of salt may be beneficial in unique situations. For example, feedlot diets containing 0.5% to 1% salt have reduced the incidence of urinary calculi. The salt increases water intake resulting in a more dilute urine that is less conducive to stone formation (197).

Salt Affects Carcass Value

In today's beef production system, carcass quality grade is an important factor determining the value of feedlot cattle at slaughter. Research at the University of Minnesota has shown that salt level in the diet can affect marbling score, which is the most important factor determining quality grade (57). Steers fed a diet devoid of supplemental salt had significantly lower marbling scores than those receiving 12.5 grams of salt daily. In the same trial, steers fed 37.5 grams of salt per day had significantly larger rib eyes than those without salt.

SALT FOR SHEEP

Deficiency Symptoms

Sheep deficient in salt develop a craving for it. Like cattle, they may lick and chew wood, dirt, rocks and other materials. They may also consume toxic amounts of poisonous plants (155). These are manifestations of an unsatisfied craving for salt. In sheep, inadequate salt intake results in decreased feed consumption and decreased efficiency of nutrient utilization. Eventually, milk production, wool production and reproduction rate decline. A very severe deficiency may cause death.

Required for Optimum Nutrition

Feed costs are the greatest single expense in sheep production. A number of early experiments at Kansas, Iowa and elsewhere showed that adding salt to sheep diets reduces feed costs. A 1953 California study showed that adding salt to an alfalfa-barley diet increased rate of gain and feed efficiency (27). An Australian study showed that salt supplementation increased wool growth (68).

Salt and Grazing Distribution

The use of salt blocks or salt stations can be used to distribute sheep over a range area in the same manner as previously discussed in the beef cattle section.

Effects of High Salt Intake

In a California study, ewes were fed diets with sodium chloride contents of 0.5%, 4.8%, and 9.1% for 253 days during growth, finishing, breeding, gestation and early lactation (26). These levels of salt did not cause any differences in performance during growth, finishing, breeding or gestation. The findings are shown in Table 4. However, the 13.1% salt level did cause more weight loss during lactation and somewhat decreased the number of lambs raised. None of the salt levels affected the gain of the lambs, blood hematocrits, serum albumin and sodium, or milk protein, sodium or potassium. The blood and milk chlorides were increased, however, at the high salt levels.

Australian researchers have shown that salt intake prior to harvest may influence dehydration associated with shipment from the farm to the processing plant. Lambs consuming a high-salt diet based on saltbush had significantly higher muscle water content than lambs fed a low salt diet (321).

Table 4. **Growing and Finishing Period, 71 Days (26)**

Items Compared	Lot 1 0.5% NaCl	Lot 2 4.8% NaCl	Lot 3 9.1% NaCl	Lot 4 13.1% NaCl
Ewes per lot	10	10	10	10
Average initial weight, lb	70	68	74	70
Average final weight, lb	102	104	105	104
Average daily gain, lb	0.47	0.52	0.45	0.48
Average daily feed, basal ration	4.1	4.1	4.1	3.9
NaCl, lb intake daily	--	0.20	0.37	0.58
Basal ration per 100 lb of gain, lb	884	790	842	812
Average daily water intake, lb	14.7	25.4	32.6	45.8
Average water intake per gram of added NaCl, ml	--	54.9	52	56.5

Effect of Salt in Water

The maximum amount of salt sheep can bear depends on the dissolved solids in the water and mineral concentrations in the feed. For example, tolerance of sheep to drinking water containing 1.3% sodium chloride was remarkably good, according to Australian workers (29) when the diet had normal sodium concentrations. Productivity, in terms of body weight and wool production, was unaffected by prolonged drinking (15 months or more) of solutions with such a salt level. The use of water with 1.3% salt instead of fresh water resulted in greater voluntary intakes of water and, consequently, a greater flow of fluid through the rumen.

Other Australian studies show that 1.0% and 1.3% salt in the water may occasionally have detrimental effects (66, 67). These differences in response are due to differences in the sodium and chloride contents of the pasture. Wilson suggested that sheep in Australia entirely dependent on salt bush, should have no more than 0.6% sodium chloride (6,000 ppm) in the drinking water (69).

Effect of Salt on Nutrient Digestibility

A California study showed that sodium chloride levels of 0.66%, 4.8%, 9.4% and 12.8% in the diet (or daily intakes of 0.03, 0.16, 0.33 and 0.46 pounds of salt) fed to growing and finishing sheep had no detectable influence on nitrogen digestibility, nitrogen retention or total digestible nutrient

content of the diet (12). The high salt intakes were not detrimental to efficiency of feed utilization or average daily gain. In another California study, nitrogen digestibility and nitrogen retention were increased with high salt levels of 5.2%, 9.5% and 13.7% in the diet (27). Australian researchers found that increasing salt levels from 0 to 8% reduced diet digestibility from 59.1 to 57.3% in sheep (316). Thus, it appears that these high levels of salt have little or no harmful effect on diet digestibility with sheep, provided they have plenty of water to drink.

Salt as Feed Regulator

A great deal of work has shown that salt can be added to protein supplements to limit their intake when they are self-fed to sheep. The results obtained are similar to those discussed in the beef cattle section. Such mixtures usually contain from 10% to 50% salt (155).

Urinary Calculi Prevention

Formation of calculi (stones or crystals) within the urinary tract of sheep is a common disease that can be minimized by feeding extra salt. The most common calculi found in lambs is the struvite type, which contains calcium, magnesium and ammonium phosphates. The mineral composition of drinking water, in conjunction with mineral imbalances in the diet, probably contributes more to the initiation of calculi formation than does the lack of water itself. Wethers with calculi have abdominal discomfort as indicated by restless, kicking at their belly and make frequent attempts to urinate. Attempts to urinate are often accompanied by rapid twitching of the tail. Animals may also groan or bleat while attempting to urinate.

Prevention of this condition is much easier and more effective than attempts at treatment. The following are offered for consideration in the development of a urinary calculi prevention program. First, delay castration of young lambs as long as possible. Second, clean, cool water is a must. The diet should contain a 2:1 calcium to phosphorus ratio. In a flock where lambs have been diagnosed with urinary calculi, adding of 3-4% salt in the ration stimulates water intake, dilutes the mineral concentration in the urine, and helps to reduce the incidence in the rest of the flock. Finally, ammonium chloride should be added to the feed at the rate of 0.5-1.5% of the diet to prevent stone formation (291).

Salt Feeding Recommendations

A 1975 Purdue study showed that the dietary salt requirements for growing lambs ranged between 0.33% and 0.43% of the air-dry (90% dry matter) (65). The 1985 National Research Council sheep publications state, "In mixed feeds, it is customary to add 0.5% salt to the complete diet or 1.0% to the concentrate portion" (155). The NRC gave a requirement for salt as a range of 0.23% to 0.46% NaCl in the diet dry matter. It also indicates that 9% salt in the diet should be the maximum tolerable level used.

Salt and Stress

Because blood sodium, potassium and chloride are important in regulating water and electrolyte balance and the humoral immune response, pre-stress salt levels may be important in determining

post-stress recovery. Cole (273) reported that sodium chloride content of lamb diets prior to feed and water deprivation affected post-deprivation sodium and potassium status. These data suggest that adding 4 grams of salt per day to the diet of feeder lambs may be important in decreasing the adverse effects of marketing and transport stress and thereby improve subsequent animal health and performance.

SALT FOR GOATS

Deficiency Symptoms

Goats developed deficiency symptoms in 4 to 6 weeks after being fed a low-sodium diet (121). The deficiency symptoms included persistent licking, restlessness, dull shaggy hair, poor growth and intake of feed and marked emaciation during lactation. After 224 days, the deficient goats weighed 20% less than those supplemented with salt. They ate an average of 6% less feed per day and required 18.5% more feed per unit of gain.

Salt Helps Regulate Grazing

The 1981 Goat NRC committee stated "placing salt in less frequently grazed pastures may influence goats to move to these areas" (100). This principle is the same as that for cattle and sheep wherein the proper location of salt blocks or salt boxes can be used to help regulate grazing into less accessible range areas.

Salt Regulated Feed Intake

The 1981 Goat NRC committee (100) also states that "salt is often incorporated at high levels to regulate the free intake of nutritional supplements" This is similar to the practice followed with sheep and cattle, wherein, salt at levels of 10% to 50% is added to the feed as a means of limiting how much the animal consumes daily. However, plenty of water must be available to prevent harmful effects from the excess salt intake.

Salt Consumption

It is estimated that milking goats consume about 18 pounds of salt yearly. Meat goats and kids consume about 9 pounds and 4.5 pounds of salt, respectively. These are average figures and can vary due to many factors, as has been discussed for other animals. Heavy milk-producing goats would require higher levels of salt, since milk contains considerable sodium and chloride.

Salt Feed Recommendations

The 1981 Goat NRC committee states that, "..if goats are not provided salt free-choice, salt should be added to the feed. A recommended level would be 0.5% of the complete feed or proportionately higher levels in supplements" (100). It is recommended that 0.5% salt be added to the total diet or 1.0% salt to the concentrate portion of the diet. Goats on pasture should be self-fed salt in a mineral box.

SALT FOR DAIRY CATTLE

Deficiency Symptoms

The sodium and chloride nutrition of lactating dairy cows is critical to optimum production because of the obligatory loss of these nutrients in milk. In addition, dairy cattle are commonly fed high forage diets that are low in sodium and high in potassium. Consequently, a sodium chloride deficiency can develop rapidly in a high producing cow.

The following are the symptoms of salt deficiency reported in Cornell studies (28). The first symptom was a craving for salt that was noticed within two weeks and by four weeks was a consistent observation. After two months, the cows showed a depraved appetite. It was manifested by licking the hands and clothing of barn personnel, consuming quantities of soil soaked with urine or the run-off from the manure pile, licking the barn walls and drinking the urine from other cows during urination. This was followed by a loss of appetite and body weight. In some cows feed intake was reduced to near zero. Milk production decreased as appetite decreased. The cows then assumed an emaciated appearance, developed a dry, rough skin (particularly on the neck), the hair coat became unkempt and the cattle became listless. In terminal cases, there was shivering, a staggered gait (most noticeable in the hind legs), weakness, abnormal heart activity, low body temperature (as low as 96.3°F) and then death. In two cases, where cattle had arrived at this terminal condition, they were given 200 grams of salt and made complete recoveries.

In this study, cattle placed on the salt-deficient diet had averaged approximately 14,000 pounds of milk the previous year (Table 5). During the first year on the salt deficient diet, production dropped to 7,150 pounds, even though the water supplied 10-15 grams of salt daily. During the second year production dropped to approximately half of the first year's production when a low-salt water source was used. It is likely that the impact on milk production would have been even greater during the first year if the low-salt water would have been used. These data illustrate the importance of sodium chloride nutrition to optimum milk production.

Table 5. **Milk Production** (28)

Treatment Daily per Cow, g Salt	Previous Production lb	Production Experiment for 301 Days, lb
0	14,000	7,500
15	16,000	11,265
60	14,600	11,550
120	14,700	11,360

Recently Cornell researchers (198) have fed dairy cows diets containing 0.10%, 0.27% or 0.45% chloride to look at the effects of a chloride deficiency, independent of sodium. Dry matter intake was significantly reduced after 4 weeks, body weights dropped from an average of 575 kg initially to 476 kg by the eighth week of the trial and milk production decreased from 27.7 to 19.2 kg/day for cows fed 0.10% chloride. Other chloride deficiency signs included lethargy, hypophagia, emaciation, hypogalactia, constipation, cardiovascular depression and mild dehydration. The cows frequently licked urine of other cows, chewed on wooden stall dividers and licked metal pipes. These data show that chloride nutrition can be of critical importance in diets of lactating cows. The actual requirement is probably between 0.18% and 0.27% chloride (199).

Salt Feeding Recommendations

The 1989 National Research Council publication "Nutrient Requirements of Dairy Cattle" states that "A reasonable estimate of the dietary requirement for milk cows is 0.18% sodium, which is equivalent to 0.46% sodium chloride in the total diet dry matter" (156). This level of salt would provide 0.30% chloride which should be adequate. The 2001 Dairy NRC (286) reported that empirical modeling of data from 15 experiments with lactating cows (1,444 cow-period observations) conducted in either cool or warm seasons showed that dry matter intake and milk yield were improved by dietary concentrations of sodium well above those needed to meet sodium requirements (287, 288). 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.

Many dairyman feed total mixed rations, but some still feed the roughage and concentrate separate. In those cases, most dairy scientists recommend that 0.5% to 1.0% salt be added to the concentration mixture fed to dairy cattle (70, 71).

Heat stress increases the sodium requirement above that prescribed by the NRC for cows in a thermal-neutral environment (131). Not only does the sodium requirement, as a percent of the diet, increase due to sweating and decreased feed intake, but prolonged heat stress also reduces blood aldosterone concentrations resulting in increased loss of sodium in the urine (200). In non-lactating Holstein cows, aldosterone concentrations have decreased over 40% during heat stress.

In a 1986 review article, a University of Florida dairy scientist recommends that the total dietary sodium be about 0.5% of the ration dry matter (149). He states that a level of 1% salt supplementation will provide about 0.4% sodium, which, when added to the sodium level naturally found in feed ingredients of the ration, will typically yield about 0.5% sodium in the total ration dry matter. He further states that if dietary sodium is increased from 0.18% to 0.5% in the ration of a lactating dairy cow consuming 42 pounds of dry matter, this will amount to an additional 0.34 pounds of salt per cow per day. This extra salt will cost about 2 cents daily. Based on previous experiments, if two additional pounds of milk are produced daily, this will yield 20-30 cents, which is a sizable return on investment (149).

Israeli researchers (220) have also shown the importance of adequate chloride intake for early lactation cows. Their research showed that during heat stress milk yield was positively and significantly related to chloride and potassium status. These ions are high in sweat and large

negatively balances may occur during early lactation. Their data were interpreted to suggest that an increased supply of chloride and potassium above NRC requirements during early lactation may result in increased dry matter intakes and milk yields.

The degree of heat stress will vary with animal and the production potential of each cow. Cows with the highest production potential will usually consume the most feed and thus have the greatest heat load. This is a good reason to feed free-choice salt above that in the basal diet. This allows individual animals to adjust their intake.

Salt Fertilization can Improve Milk Production

Intensive rotational grazing is a dairy management practice that is becoming increasingly popular. Maximizing high-quality forage intake is essential to optimizing milk production with this approach. Recent British research (221, 222, 223, 224, 225, and 226) has shown the potential benefits of using salt as a fertilizer when the soil and forage grown on the soils is low in sodium. These researchers have repeatedly reported increases in milk production in cows grazing perennial ryegrass pastures when forage sodium levels were increased from 0.02% to 0.05% by salt fertilization. When given a choice, cows grazed the salt fertilized pasture compared to the unfertilized pastures, spent more time grazing, and had a greater bite rate. Milk yield and weight gain were increased by salt fertilization beyond that achieved by direct supplementation. Water intake and rumen pH were also increased due to salt fertilization which may have allowed the greater dry matter intakes. These data are interpreted to show that salt fertilization of low-sodium forages may increase the potential of using rotational grazing as a management strategy for dairy production.

SALT FOR SWINE

Deficiency Symptoms

Studies at Wisconsin and Purdue showed that a deficiency of salt decreased daily gain and feed efficiency in the pig (6, 78). They also showed that salt supplies sodium and chloride in about the proper proportions for growing pigs (6). The salt-deficient pigs licked their cages looking for salt.

Value of Salt

Four studies at Purdue showed that pigs receiving no salt in their diet required 174 pounds more feed per 100 pounds of gain; their rate of gain was only half as fast as that of pigs given adequate salt (3). In this trial, one pound of salt saved 287 pounds of feed. Furthermore, the pigs deficient in salt ate 12.5 times as much of the mineral mixture as the pigs fed salt. This reflected the craving for something they could not get except in common salt. Table 6 shows one year's data (1945).

Table 6. **Effect of a Lack of Salt in the Diet** (3)

Lot	How Salt Was Fed	Average Final Weight of Hogs, lb (85 day trial)	Average Daily Gain, lb	Feed per 100 lb of Gain, lb	Feed Cost Per 100 lb Gain
1	No salt fed	174	0.99	Corn 451.0 Supplement 100.7 Mineral 17.0 Total feed 568.7	\$12.53
2	Loose salt fed, but none in the mineral mixture	255	1.94	Corn 321.4 Supplement 73.1 Mineral 0.7 Total feed 395.2	\$8.68

In another trial at Purdue, the salt-fed pigs ate 23% more feed and gained at nearly double the rate on 33% less feed (3). One pound of salt saved 185 pounds of feed.

A study at Purdue showed that corn-soybean meal diets were improved by the addition of 0.2% salt (4). Pigs fed no salt showed a lower rate of gain and reduced feed utilization.

Salt Requirement Information

The 1998 National Research Council Swine Nutrient Requirements Committee recommends, "...that 0.20% to 0.25% added sodium chloride will meet dietary sodium and chloride requirements of growing-finishing pigs fed a corn-soybean meal diet" (277). Studies at Purdue (74, 75) serve as the basis for this recommendation. This is similar to the European ARC which recommends a level of 0.33% salt in the diet for the growing pig (124). However, recent research with the early weaned pig suggests that the sodium and chloride requirement maybe higher (272). Pigs weaned at 21 days of age required .40% salt for maximum performance through five weeks of age. During this time the gain:feed ratio was greater than 0.5:1, so that a nutrient dense diet is required for optimum performance.

The sodium and chloride requirements of breeding swine are not well established. Results of one study (123) showed that diets containing 0.3% salt were deficient for pregnant sows. A 1989 regional university study (Clemson, Florida, Georgia, Kentucky, Oklahoma, Texas and Virginia) evaluated adding 0.25% vs 0.5% salt to a corn-soy diet for sows through three parities (litters). The sows fed 0.5% supplemental salt resulted in a small increase in pigs farrowed, live pigs at birth, pigs weaned, birth weights and weaning weights. The authors concluded, "when continued for more than one reproductive cycle, feeding less than 0.5% salt appears to reduce litter size at birth and weaning" (150).

Effect of High Salt Intake

University of Alberta studies showed no harmful effect when salt was added to the drinking water of growing-finishing pigs at a level of 5,000 ppm (5). These and other studies indicate it is extremely difficult to salt-poison pigs when salt is added to the diet and the pigs are allowed free access to water.

In order to poison pigs with salt, the Wisconsin Station salt-starved the pigs for several months (7). They fed the pigs swill containing salt in a crowded trough space with no water available. Salt in the swill totaled 1.5% to 2.0% (wet basis) or 6% to 8% on a dry matter basis. It required more than one day of excessive salt feeding before symptoms of poisoning became evident. If water had been available, the pigs might have drunk enough to satisfactorily dilute the salt content of their digestive tract and body tissues.

In USDA studies, pigs were fed levels of salt of 0%, 1%, 2%, 4%, 8%, and 12% of the diet (8). The average daily gain for the pigs fed 2% salt exceeded those of the other pigs. Increasing levels of salt above 2% decreased gain and feed efficiency. The pigs on 12% salt ate enough feed to give them an intake of about one half pound of salt daily. One pig on the 8% salt level showed symptoms of possible salt poisoning. In another USDA trial, one pig weighing 218 pounds, after being fed 26 days on a high-salt diet, consistently consumed an average of 8.4 pounds of total diet and 1.1 pounds of salt daily. A daily water intake of 2.35 gallons was apparently sufficient to permit elimination of the salt without ill effects (8). The important fact is that swine are very resistant to salt poisoning as long as adequate water is available.

Salt in Manure

Manure disposal is an important consideration for many large swine confinement units. A Purdue study involved feeding pigs 0.2% versus 0.5% salt in the diet (73). Application of 40 tons of swine waste (10,000) gallons per acre per year resulted in maximum corn yields. Exchangeable soil sodium concentration increased from the swine waste application; however, the sodium accumulation (at both levels of salt in the diet) was not high enough to cause any problem with soil productivity. These data are interpreted to show that feeding the recommended salt concentrations should not limit the soil application of swine manure.

SALT FOR HORSES

Deficiency Symptoms

Horses deficient in salt will lick or chew on mangers, fences, dirt, rocks and other objects. They also develop depraved appetites, rough hair coat and reduced growth. Mares experience reduced milk production, according to reports from the Mississippi Station (30). Horses with severe salt deficiency, brought about by a low salt intake and considerable sweating, become fatigued and exhausted. Horses that sweat profusely can lose as much as 82.5 grams of sodium per day (201). Consequently, level of activity and degree of heat stress has a greater affect on salt requirement in horses than any other farm animal species.

Effect of High Salt Level

The 1989 National Research Council horse publication states that "horses are tolerant of high levels of salt in their diets if they have access to fresh drinking water (154). However, salt poisoning can occur if salt-starved animals are suddenly exposed to an unlimited supply of salt, or if liberal amounts of water are not available. In such cases, the horses may develop digestive disturbances. Severely poisoned animals die of salt cramps. Horsemen can easily prevent this occurrence by not giving salt-starved horses free access to salt until they start leaving some behind in the mineral box and by making sure plenty of water is always available to drink.

Salt Requirement Information

The need for salt varies considerably, depending on a horse's level of work, riding or heat stress. Because sweat contains about 0.7% salt, the more a horse exercises the more salt is lost via sweat and the more it is needed in the diet.

Michigan researchers measured daily salt consumption of 12 horses during the harvest season in the hot months of the year from May to August (31). Salt consumption averaged 0.64 ounces per horse in May and rose each month to a high of 3.18 ounces per horse in August. This variation is shown in Figure 1. Such variations in monthly salt intake and among individual horses show why it is risky to feed a specific amount per horse or a fixed percentage in the feed. The study also explains why salt is often self-fed to horses, even though it is also added to the diet.

Salt Feeding Recommendations

Typically 0.5% to 1.0% salt should be added to horse concentrate feeds. If the concentrate feed is used as a small part of the diet, it should contain 1% salt. If it is used as a major part of the diet, such as a complete feed, then 0.5% salt should be added. If extra salt is lost due to sweating or if the horses are being managed in a grazing situation, free-choice feeding of salt is recommended to ensure adequate intake.

SALT FOR POULTRY

Deficiency Symptoms

Salt deficiency results in lower feed consumption, loss of weight, lower egg production, occasionally a loss in egg size and slower growth in broilers (79, 80, 81, 203). Sodium chloride deficiency can also increase the bird's susceptibility to disease by suppressing the immune system. Recent work by Pimentel and Cook showed that Hubbard broiler chicks fed diets containing less than 0.14% sodium or 0.17% chloride had depressed immunity to sheep red blood cells as compared to chicks fed higher levels of sodium and chloride (203).

Diet ingredients used today in poultry feeding are deficient in sodium and chloride, so salt must be added for optimum performance. Mississippi data indicate that the body stores of sodium reserves in hens are negligible and last only about a week. Salt is not self-fed to poultry, so their needs must be met by an adequate level in the diet. Salt deficiencies will occur occasionally in commercial practice, as was found in Colorado (80). This could be due to situations where not enough salt is fed or salt requirements are higher than normal due to heat or other causes.

Rapid changes in the physiology of the small intestine occur in layers on a sodium deficient diet (227). After three weeks on a low-sodium diet the microvillus surface area more than doubled due to an increase in the number and size of epithelial cells. Greater density of open sodium channels also increased the net sodium transport. These observations demonstrate the physiological importance the laying hen puts on absorbing adequate sodium.

Recent research with broilers has shown that sodium and chloride concentrations recommended by the 1984 NRC publication for poultry was below the requirement for maximum growth using a corn-soy diet (202). These data suggests that the sodium requirement for broilers is 0.5% for the first week decreasing to 0.3% by three weeks of age. The higher sodium and chloride requirement is probably due to the faster growing strains of broilers being used today, the higher energy diets being fed, and the fact that sodium and chloride in drinking water was not taken into account in initial estimates of the requirement.

Effect of Type of Salt

Mississippi studies showed that two different types of salt (rock salt and evaporated salt) produced essentially equal growth responses and feed utilization in chicks when the particle size was equal (45, 46). Salt with a particle or crystal size range between U.S. Bureau of Standards Number 18 to Number 25 sieve sizes (0.0394 to 0.0278 inch openings) achieved maximum growth rate. Salt of a size range either larger or smaller than Number 16 through Number 30 sieve sizes (0.0469 to 0.0234 inch openings) supported a lower growth rate.

Effect of High Salt Levels

Excessive levels of salt are toxic to poultry, but there is considerable variation in the levels considered toxic by various investigators. Younger birds seem more susceptible than older birds to salt toxicity. The feeds in the diet as well as the level of certain nutrients can influence toxicity

levels. Chicks also exhibit individual differences in tolerance for excess salt. Poultry on high-salt diets increase their consumption of water almost in proportion to the excess salt (127).

Illinois work showed that chickens may be raised from 9 to 21 weeks of age on diets containing as high as 8% salt with no apparent detrimental effect on their condition (32). Maryland studies showed that baby chicks fed 8% or more salt experienced retarded growth (33). Canadian data from a more recent experiment showed that increased mortality occurred when 3% or more salt was added to the diets of chicks reared from hatching to 9 weeks of age (34). On the other hand, a Cornell study showed no effect of 4% salt in the diet on mortality of chicks to 8 weeks of age (40).

Table 7 shows data on the percent water in tissues of the chicks in the Canadian study (34). It is interesting that skin was the tissue most affected by an increase in water content as the salt level increased.

Table 7. Water Content of Some Tissues Affected by the Salt Content of the Diet (34)

Percent Salt		Percent	Water	
Added to Diet	Skin	Liver	Heart Muscle	Leg Muscle
½	50.0	72.2	75.7	76.1
3	63.6	72.2	75.1	77.5
5	59.0	73.4	78.5	78.0
7	76.9	72.4	78.6	73.5
9	82.9	73.7	82.5	77.8

Values represent the average of duplicate determination on each two birds.

Turkeys are more susceptible to salt toxicity than chickens. Connecticut workers reported that turkey poults readily tolerate levels of salt up to 2% if the diet is otherwise relatively free of salt (35). James reported that 2% salt in turkey poult diets increased mortality rate (36). However, mortality did not occur with a level of 1% salt added to the same diet.

A Canadian study showed that turkey poults fed up to 1.5% salt and 1.5% sodium did not exhibit signs of round heart disease (82). The study did not verify field recommendations to reduce salt intake, because round heart disease could be due to high salt intake.

An Illinois study showed that pheasants can tolerate 5% salt in the diet without any harmful effects (37).

Care should be taken with levels of salt added to poultry diets. The margin between a safe level and a harmful level is not great under some conditions. In addition, excess salt can increase fecal

moisture levels which can be a management problem in most housing units. Cornell research suggests that 2.5 g salt/liter of drinking water increased water intakes by 15%, but did not visually alter the texture of the excreta (290)

In summary, it seems that the addition of one percent salt, probably the highest level to add to any poultry diet, is safe and will cause no toxic effects even with very young birds.

Effect of Salt in Water

Controlling salt in water is more important than in feed. Levels of 0.4%, 0.9%, 1.2% and 2.0% salt in drinking water have shown harmful effects with poultry. Doll and co-workers reported no clinical evidence of toxicity with levels of 0.25% (2,500 ppm) sodium chloride in drinking water for chicks (38). A Canadian study showed that chicks can tolerate water containing as much as 0.5% salt even when the feeds contained 0.5% added sodium chloride (44). Clearly, water containing high salt levels should not be used for poultry. Based on current evidence, a level of 2,500 ppm salt in the water probably would not cause toxic effects (127), but may increase egg shell defects (290).

Salt Requirement Information

1984 NRC recommendations are for 0.15% sodium in all diets for chickens, ducks, pheasants, bobwhite quail and Japanese quail, while turkey poults start out at 0.17% and decrease to 0.12% sodium (141). The chloride requirement for these same species ranges from 0.12% to 0.15%. However, recent Georgia data show that optimal concentration of both sodium and chloride is 0.4% for broilers up to three weeks of age (202). Depending on the sodium and chloride in the basal ingredients, this would require approximately 0.9% salt. Canadian workers found the addition of 1% salt to a mash diet for chicks to be optimum for growth and superior to a group with 0.5% salt added (34). In a Wisconsin study with corn, wheat bran, standard wheat middling, dried skim milk and meat scraps, researchers concluded after a number of trials that the addition of 0.5% salt to the diet will meet the salt requirements of either growing chicks or laying hens (39).

Florida studies (204) have shown that feeding a corn-soy diet without added salt to Leghorn or broiler breeder hens causes an immediate drop in feed consumption and body weight with egg production dropping to near zero in 21 days.

A Cornell study with chicks up to eight weeks of age showed the minimum salt requirement to be 0.65% of the diet (40). An Illinois study with purified diets found the sodium requirement of the chick, up to 4 weeks of age, to be 0.11% on one diet and 0.20% on another (41). This is the equivalent of 0.28% to 0.50% sodium chloride in the diet.

Summers and co-workers in 1967 demonstrated that a chloride deficiency condition can exist on a practical corn-soybean meal diet in which the sodium chloride has been removed and sodium is provided from a source other than sodium chloride (42). The study was conducted with chicks up to three weeks of age. The study suggested that the requirement for chloride lies somewhere between 0.073% and 0.22% of the total diet.

A Nebraska study (43) showed that a level of 0.15% to 0.2% sodium in the diet was required for turkeys up to four weeks of age for maximum gain and feed efficiency. It also found that a level of 0.4% sodium chloride was required in a corn-soybean meal diet containing no ingredients of animal origin. The scientists also reported an interaction between sodium and potassium and suggested an optimum ration of about 2 to 2.5 parts potassium to one part sodium in young turkey diets.

An Illinois study showed that under practical conditions, the sodium requirement of both pheasants and quail was approximately 0.085%, and chloride requirement was between 0.048% and 0.11% of the diet (37). The addition of 0.15% salt to the diets satisfied both the pheasant and quail sodium and chloride requirements. The 1984 National Research Council poultry publication recommends a sodium level of 0.15% and a chloride level of 0.11% of the diet (equivalent to 0.21% salt) for pheasants and quail (141).

Mississippi studies have shown that the sodium requirements of cage hens are approximately twice that of hens kept on floor (47, 48). Floor hens recycle some sodium from their droppings, a fact to take into consideration in adding salt to diets of hens in cages.

Salt Feeding Recommendations

The level of salt used in chicken, turkey, duck, goose, pheasant and quail diets will vary from 0.25% to 0.5%. The kind of diet fed, the stage of the life cycle, the environmental conditions encountered, the level of productivity and other factors will influence the total level of NaCl to use in the diet.

SALT FOR DOGS, CATS, RABBITS, MINK, FOXES & OTHER SMALL ANIMALS

There is little information on salt requirements for dogs and cats, mink, foxes, rabbits and other small animals.

The 1985 NRC publication, Nutrient Requirements of Dogs, states the following: "One percent of sodium chloride (salt) in the total dry-type diet will supply normal needs and is not excessive for normal dogs." (142). AAFCO (289) recommends 0.3% sodium and 0.45% chloride in the diet of growing and reproducing dogs. Symptoms of a deficiency are fatigue, exhaustion, inability to maintain water balance, decreased water intake, retarded growth, dryness of skin and loss of hair (142). Dr.C. M. McCay of Cornell University reported that dogs fed diets containing 2% added salt consumed more water than usual but remained in good health (56).

The National Research Council publication on Nutrient Requirements of Mink and Foxes states as follows: "There are no data on the requirements of foxes for sodium and chloride. However, the requirements are met by fortifying the dry diet with 0.5% salt"(143).

There are no data on the minimum requirements of the growing mink for sodium and chloride. However, it is suggested that 0.5% salt in the wet feed or 1.3% salt in the dry diet be used for pregnant and nursing females to prevent "nursing sickness." Sodium and chloride requirements at other times may be lower (143).

The National Research Council publications on Nutrient Requirements of Rabbits state that in practice, salt is generally added to the diet at a level of 0.5% or provided ad libitum by means of a salt block for free-choice consumption.(89)

Table 8 gives recommended levels of salt to use with other laboratory animals and fish. The 1993 NRC (285) did not give dietary sodium or chloride requirements for fish. Very little research has been conducted on salt needs of these animals; hence, the recommended levels may change as more definitive information is obtained in the future.

Table 8. Recommended Salt Levels for Other Animals by National Research Council Publications

Name of NRC Publication on Nutrient Requirements of Small Animals	Year of Publication	Reference	Level of Salt Recommended in Total Diet, %	Signs of Deficiency of Salt in the Diet
Cats	1986	102	500 mg Na and 1900 mg Cl per kg dry diet	Weight loss, severe alopecia and skin dryness

Rats	1978	103	0.125.	Retarded growth, corneal lesions, soft bones, infertility, death
Mice	1978	103	0.5-0.7	No data reported
Gerbils	1978	103	0.39-1.0	No data reported
Guinea pigs	1978	103	0.5	No data reported
Hamsters	1978	103	0.38	No data reported
Warmwater fishes	1983	145	0.6	No data reported
Coldwater fishes	1981	144	0.5	No data reported
Non-human primates	1978	101	0.4-1.0	No data reported

TRACE MINERALS

There are seven trace minerals that have been shown to be needed in supplementing animal diets. They are iron, copper, zinc, manganese, cobalt, iodine and selenium. They are needed in very small amounts, or traces, in the diet, and hence their name, "trace minerals." Many times their requirement is expressed as parts per million (ppm) or as milligrams per kilogram (mg/kg) of diet dry matter. One ppm concentration is the same as one penny in \$10,000. With some minerals, such as cobalt the concentration in the diets may be expressed on a parts per billion basis (ppb). If there are approximately 300 million people in the United States, then each person is 3.33 ppb of the population. Even though the concentrations are very low, they are still significant nutrients.

Subclinical trace mineral deficiencies occur more frequently than recognized by most livestock producers. Currently this is a bigger problem than clinical mineral deficiencies, because the farmer does not see specific symptoms that are characteristic of a subclinical trace mineral deficiency. Instead, as shown in Figure 1, the immune system is depressed, the animal begins to grow more slowly, and fertility is impaired (306). The end result is inefficient production and lower profitability. Therefore, a profitable and efficient farm operation must provide the supplemental trace mineral elements. In highly competitive animal enterprises, it can be the difference between profit and loss.

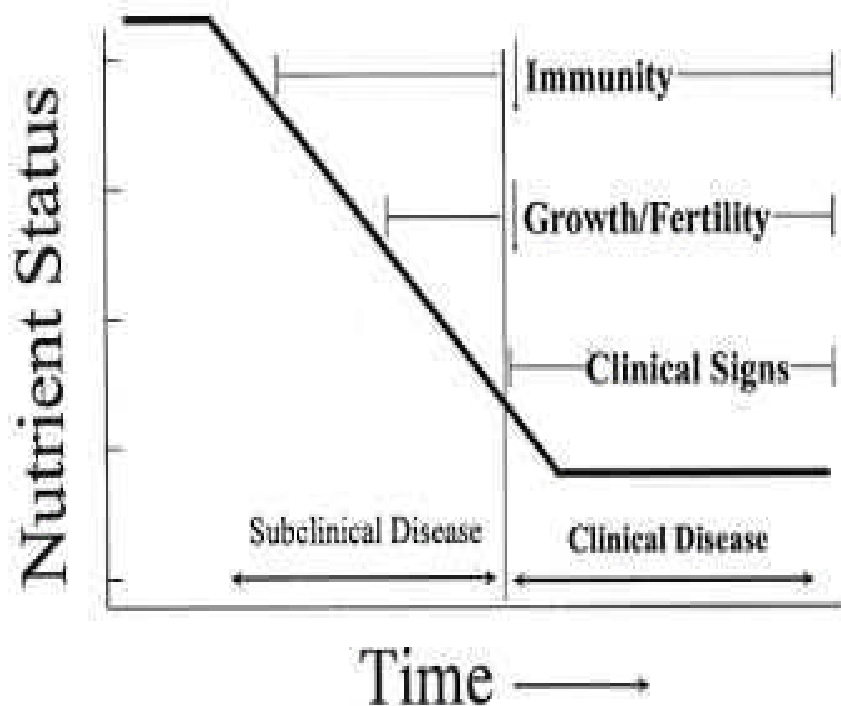


Figure 1. Schematic depiction of the relationship between nutrient status and presence of subclinical or clinical disease manifestations. (Redrawn from S. Wikse, 1992, Texas A&M University Beef Cattle Short Course).

Deficient Area Problem

There are several examples where an area of the country was not recognized to be trace mineral deficient in the past but now has been shown to require supplementation. For example, a selenium deficiency was not considered a problem in the United States until relatively recently. Now at least 44 states have been shown to contain low-selenium areas. In only a few states have the classical selenium deficiency symptoms been observed, but performance responses have demonstrated the need for selenium supplementation. When a cobalt deficiency was first found in Western Australia, the problem was believed to be confined to about 5,000 acres. Further studies showed that at least 25 million acres are cobalt-deficient in Western Australia.

Another factor to consider is the shipment of feeds from one region to others. This alone makes it almost impossible to isolate areas of specific trace mineral deficiencies. There is nothing to prevent feed grown in a trace mineral deficient area from being shipped to another area where the feed grown is supposedly adequate in that mineral. For example, corn and soybeans grown in Midwestern states with areas deficient in selenium, iodine and other trace minerals are shipped to and fed in many other areas of the United States and the world. For example, selenium deficiencies have been observed in pigs fed U.S.-produced corn and soybean meal in Taiwan in 1978. Locally grown feeds in Taiwan are usually adequate in selenium.

The only precaution is to make sure the trace mineral is not fed in areas where selenium may be in excess. This can be accomplished by working with the local feed manufacturer or consulting nutritionist to determine if there are any potentially toxic minerals in that area. For example, sheep producers should be aware of the copper levels of locally grown feeds. If the feeds are high in copper and low in molybdenum, then feeding a trace mineralized salt with little or no copper is recommended. Most feed manufacturers will be aware of situations where near toxic levels of one of the trace minerals are present in feeds and will provide trace minerals mixes formulated accordingly.

TRACE ELEMENT CONCENTRATIONS OF FORAGES

Forages, either harvested mechanically or by grazing, are the basal dietary ingredients for beef cattle, dairy cattle, sheep and horses. Based on the National Animal Health Monitoring System's Beef Study (1997) only 9% of the cow-calf producers analyzed the nutrient content of their feeds. This number is probably higher for dairy producers, but lower for sheep and horse producers. With this in mind, Mortimer et al. (264) summarized feed analysis data from 709 forage samples, collected from 678 producers in 23 states. The number of feed samples per state ranged from 98 for Texas to 4 for Florida and New Mexico. The states were primarily those with major beef cow populations. All sections of the U.S. were represented except for the Northeast. Wet chemistry was used for the analysis of aluminum, copper, iron, manganese, molybdenum, sulfur, selenium and zinc.

For purposes of analysis, data were combined into 11 different forage categories. The copper, manganese, zinc and selenium concentrations were classified as deficient, marginally deficient, or adequate (Table 9). The Maximum Tolerable Concentration (MTC) for each element is defined as

the dietary level, when fed for a limited period of time, will not impair animal performance and should not produce unsafe residues in human food derived from the animals.

Table 9. Classification of Trace Elements in Forage Relative to Their Abilities to Meet Either Dietary Requirements or Cause an Antagonistic Problem with Copper

<u>Trace Minerals</u>	<u>Deficient</u>	<u>Marginally Deficient</u>	<u>Adequate</u>	<u>MTC*</u>	
Aluminum (ppm)	--	--	--	1000	
Copper (ppm)	below 4	4-9.9	=10	100	
Manganese (ppm)	below 20	20-39.9	=40	1000	
Zinc (ppm)	below 20	20-29.9	=30	500	
Selenium (ppb)	below 100	100-199.9	200	2000	
Copper:Mo Ratio	below 4.0:1	4.0-4.5:1	>4.5-5:1	--	
*Maximum Tolerable Concentration					
<u>Copper Antagonist</u>	<u>Deficient</u>	<u>Ideal</u>	<u>Antagonistic Level**</u>	<u>Antagonistic Level**</u>	<u>MTC*</u>
			<u>Marginal</u>	<u>High</u>	<u>-</u>
Iron (ppm)	below 50	50-200	>200-400	>400	1000
Molybdenum (ppm)	--	below 1	1-3	above 3	5
Sulfur (% DM)	Below .10	.15-.20	>.20-.30	>.30	.40
*Maximum Tolerable Concentration **Levels above these can potentially adversely affect copper availability					

For trace minerals the concentration of one can have a great effect on the requirement of another. For example, copper requirements are greatly affected by the concentrations of antagonists such as molybdenum, sulfur, iron, and zinc. If molybdenum concentrations are below 1.0 ppm it will have no effect on copper requirement, but if molybdenum is above 3 ppm it can be very antagonistic to copper absorption. Thus the relationship between iron, molybdenum and sulfur relative to copper is described as ideal, marginally antagonistic, or highly antagonistic. These relationships apply to beef and dairy cattle, but may not be relevant to sheep.

Table 10. Alfalfa/Alfalfa Mix

Samples	Aluminum	Copper	Manganese	Zinc	Selenium	Copper to Molybdenum Ratio
N = 196	(ppm)	(ppm)	(ppm)	(ppm)	(ppb)	
Mean ± S.E.	151.54 ± 8.77	10.50 ± .32	56.97 ± 2.17	23.03 ± .50	376.64 ± 35.73	11.27 ± .97
Classification						
MTC*,%**	0	0	0	0	1.02	--
Adequate, %	--	41.84	74.49	13.78	54.59	66.84
Marginal, %	--	57.65	23.98	52.04	20.41	6.63
Deficient, %	--	0.51	1.53	34.18	23.98	26.53

Copper Antagonists					
Element	Mean ± S.E.	% Ideal	Antagonistic Level % Marginal	Antagonistic Level % High	%>MTC**
Iron	210.01 ±13.15 ppm	64.80	26.02	8.67	1.02
Molybdenum	1.96 ± .12 ppm	29.59	54.08	16.33	5.61
Sulfur	0.23 ± .01%	22.96	48.47	18.88	2.04

*In some cases, not all samples were available for determination of calcium content.

**Maximum Tolerable Concentration (MTC) also included in % High Antagonistic Level.

NOTE: Percentages may not add to 100% as samples deficient of the specified elements were not included.

The alfalfa and alfalfa mix was the largest forage category with 196 samples (Table 10). Each trace mineral has a mean and standard error (S.E.) value. For example the copper concentration averaged 10.5 ppm with a standard error of 0.32. The 1996 NRC for beef cattle gives the copper requirement at 10 ppm. On average it would appear that alfalfa based diets would meet this requirement, but when the individual samples are compared a different picture develops. Only 41.84% of the samples were adequate with 57.65% of the samples being marginal and 0.51% deficient. However, the copper:molybdenum ratios were such that 54.08% were marginal and 16.33% were highly antagonistic and could produce a copper deficiency. In addition, the iron and sulfur concentrations were such that 26.02 and 48.47% were marginally antagonistic and 8.67 and 18.88% of the samples were highly antagonistic, respectively. These data illustrate the potential problems that can occur when just looking at mean book values to decide whether trace mineral supplementation is required. While the mean value suggest that alfalfa is an adequate source of copper, in reality over 60% of the samples required copper supplementation. Similar data were reported by Adams (284) from alfalfa and alfalfa-mixed forages grown in Pennsylvania from 1969 to 1973. In that data set the standard deviation for copper concentration was over 5.7 ppm. With that sort of variation, book values are relatively meaningless.

Manganese was marginal in approximately 24% of the samples with 1.53% being deficient. In contrast, only 13.78% of the samples contained adequate zinc with approximately one-third being deficient (Table 19). With selenium, 54.59% was adequate, 20.41% marginal, and 23.98% deficient. The MTC was exceeded by 5.61% of the sample for molybdenum. Even though alfalfa is often considered an excellent source of trace minerals, these data show that copper, zinc and/or selenium are often deficient and require supplementation to optimize cattle performance.

Brome is a common forage fed to cattle, sheep and horses in the Midwest (Table 11). Copper was marginal in 75% and manganese was adequate in 85% of the samples tested. Only 5% of the samples were adequate in zinc and 80% were deficient. Selenium was marginally deficient in 25% and deficient in 45% of the brome samples. These brome samples did not contain highly antagonistic concentrations of iron, molybdenum or sulfur. With only 20 samples in this data set, these results are less conclusive than with some of the other forages.

Table 11. Brome

Samples N=20	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	115.22 ± 67.45	9.53 ± 1.12	69.31 ± 6.02	19.46 ± 1.54	189.95 ± 39.55	12.54 ± 3.25
Classification						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	25.00	85.00	5.00	30.00	80.00
Marginal, %	--	75.00	15.00	15.00	25.00	5.00
Deficient, %	--	0	0	80.00	45.00	15.00
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	155.78 ± 20.91 ppm	50.00	35.00	0	0	
Molybdenum	1.23 ± .17 ppm	50.00	45.00	5.00	0	
Sulfur	0.23 ± .01%	35.00	10.00	0	0	
*Maximum Tolerable Concentration						

There were 120 Bermuda grass samples analyzed and they came primarily from the Southern tier of states in the U.S (Table 12). Although there were no samples that were definitely deficient in copper, approximately two-thirds were marginal. Nearly half the samples were marginally deficient in zinc, and over half were deficient in selenium. Sulfur levels were reasonably high (0.27%) such that approximately 34 and 45% were highly or marginally antagonistic to copper. Over 8% of the samples exceed the MTC of sulfur. If animal diets were based on Bermuda grass, manganese is the only trace mineral not required as part of a routine supplementation package, according to this data set.

Table 12. Bermuda

Samples <u>N=112</u>	Aluminum <u>(ppm)</u>	Copper <u>(ppm)</u>	Manganese <u>(ppm)</u>	Zinc <u>(ppm)</u>	Selenium <u>(ppb)</u>	Copper to Molybdenum Ratio
Mean ± S.E.	182.30 ± 67.21	10.59 ± .72	126.51 ± 7.18	33.47 ± 1.87	142.26 ± 14.96	39.22 ± 5.03
Classification						
MTC*, %**	0.89	0	0	0	0	0
Adequate, %	--	35.71	92.86	43.75	19.64	94.64
Marginal, %	--	64.29	7.14	48.21	27.68	1.79
Deficient, %	--	0	0	8.04	52.68	3.57
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	165.22 ± 36.96 ppm	83.04	10.71	2.68	0.89	
Molybdenum	0.61 ± .09 ppm	82.14	15.18	2.68	1.79	
Sulfur	0.27 ± .01%	13.39	44.64	33.93	8.04	
*Maximum Tolerable Concentration						

Fescue is a common forage grown in the Southeastern one-third of the U.S. A total of 73 samples were analyzed in this data set (Table 13). Only 26% , 15%, and 4% of the Fescue samples were adequate in copper, zinc, and selenium, respectively. Selenium was likely to be the most limiting trace mineral with over 78% being deficient. In addition, over 40% of the samples had molybdenum and sulfur concentrations that were at least marginally antagonistic to copper. In a more recent study 1021 forage samples, primarily tall fescue, were analyzed by Tennessee researchers (293). Copper and zinc were at least marginally deficient in 92.4 and 89.3% of the samples, respectively. Sulfur was at least marginally antagonistic to copper in 89.3% of the samples. This is consistent the Virginia Tech research that showed that low copper availability is common in endophyte fungus infected Kentucky 31 tall Fescue (301). Fescue was a good source of manganese with over 97% of the samples being adequate. Selenium, zinc and copper should be added to Fescue-based diets routinely.

Table 13. Fescue

Samples N=73	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	112.82 ± 12.28	9.26 ± .51	151.84 ± 11.38	23.62 ± 1.08	79.29 ± 6.31	17.13 ± 1.91
Classification						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	26.03	97.26	15.07	4.11	80.82
Marginal, %	--	72.60	2.74	46.58	17.81	5.48
Deficient, %	--	1.37	0	38.36	78.08	13.70
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	153.83 ± 21.65 ppm	82.19	6.85	5.48	1.37	
Molybdenum	1.08 ± .10 ppm	49.32	46.58	4.11	0	
Sulfur	0.19 ± .01%	38.36	42.47	1.37	0	
*Maximum Tolerable Concentration						

This data set contained 34 samples classified as orchardgrass (Table 14). Selenium was the most commonly deficient element with only 5.88% of the samples being adequate, followed by 11.76% of the samples being adequate for both copper and zinc. Molybdenum was classified as either marginally or highly antagonistic to copper in 47.1% of the samples. Over 20% of the samples had at least marginal or higher antagonism to copper due to iron or sulfur concentrations. The only trace element that could be considered adequate in most Orchard grass samples is manganese.

Table 14. Orchardgrass/Orchardgrass Mix

Samples	Aluminum	Copper	Manganese	Zinc	Selenium	Copper to Molybdenum Ratio
<u>N=34</u>	<u>(ppm)</u>	<u>(ppm)</u>	<u>(ppm)</u>	<u>(ppm)</u>	<u>(ppb)</u>	
Mean ± S.E.	100.88 ± 9.65	7.44 ± .42	121.46 ± 12.26	24.13 ± 2.02	100.38 ± 15.47	15.71 ± 2.76
<u>Classification</u>						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	11.76	94.12	11.76	5.88	76.47
Marginal, %	--	82.35	5.88	44.12	26.47	8.82
Deficient, %	--	5.88	0	44.12	67.65	14.71
<u>Copper Antagonists</u>						
Element	Mean ± S.E	% Ideal	Antagonistic level	Antagonistic Level	%>MTC*	
			% Marginal	% High		
Iron	119.10 ± 15.40 ppm	73.53	20.59	0	0	
Molybdenum	1.50 ± .54 ppm	52.94	44.12	2.94	2.94	
Sulfur	0.18 ± .01%	44.12	17.65	8.82	0	
*Maximum Tolerable Concentration						

Sudan grass or sorghum-Sudan is a warm-season hybrid forage commonly grown in the High Plains and Southern part of the U.S. There were 61 samples analyzed (Table 15). Manganese was the only trace mineral that was classified as adequate in most (83.6%) of the samples. Approximately 23% of the samples were adequate in selenium, 26% were adequate in zinc and 49% were adequate in copper. However, over 50% of the samples had marginal or highly antagonistic levels of iron and molybdenum. Adams (284) reported that 91 sorghum-Sudan forages grown in Pennsylvania also had an average iron concentration of 320 ppm, but a standard deviation of 278 ppm. Thus copper, zinc and selenium supplementation is critical for Sudan based diets.

Table 15. Sudan

Samples N=61	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	279.26 ± 39.13	12.00 ± .91	67.54 ± 4.56	25.64 ± 1.49	165.89 ± 19.45	22.09 ± 3.86
Classification						
MTC*, %**	4.92	0	0	0	0	0
Adequate, %	--	49.18	83.61	26.23	22.95	91.80
Marginal, %	--	49.18	16.39	42.62	45.90	1.64
Deficient, %	--	1.64	0	31.15	31.15	6.56
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	320.63 ± 41.26 ppm	50.82	24.59	24.59	6.56	
Molybdenum	1.09 ± .12 ppm	49.18	44.26	6.56	0	
Sulfur	0.12 ± .01%	13.11	4.92	1.64	0	
*Maximum Tolerable Concentration						

There were 46 cereal-type forages analyzed in this study (Table 16). Although not described, the most common forages grown in the area from which samples were collected would be wheat, oats and barley. Only approximately 20% of the samples were adequate in copper, zinc or selenium. Zinc and selenium was definitely deficient in 45.6 and 52.2% of the samples, respectively. Nearly 85% of the forages were adequate in manganese. The copper to molybdenum ratio would have caused a copper deficiency in 8.7% of the samples.

Table 16. Cereal

Samples N=46	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	168.34 ± 31.57	8.27 ± .42	94.98 ± 14.46	23.00 ± 1.23	146.72 ± 30.83	23.42 ± 3.26
Classification						
MTC*, %**	2.17	0	0	0	0	0
Adequate, %	--	23.91	84.78	21.74	19.57	91.30
Marginal, %	--	76.09	13.04	32.61	28.26	0
Deficient, %	--	0	2.17	45.65	52.17	8.70
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	174.21 ± 32.09 ppm	71.74	13.04	6.52	2.17	
Molybdenum	1.01 ± .28 ppm	65.22	30.43	4.35	2.17	
Sulfur	0.17 ± .01%	39.13	21.74	2.17	0	
*Maximum Tolerable Concentration						

Native grasses were represented by 38 samples in this analysis (Table 17). Native grasses are mixture of grass species that exist naturally under range conditions and probably represent hand-clipped samples. Manganese was adequate in a high percentage (86.6%) of the samples. Zinc and copper concentrations are of concern because nearly 80% of the samples are marginal and or deficient. Also, a fairly high percentage of the samples had marginal or highly antagonistic levels of iron, molybdenum or sulfur. Thus copper supplementation needs careful evaluation.

Table 17. Native Grass

Samples	Aluminum	Copper	Manganese	Zinc	Selenium	Copper to Molybdenum Ratio
N=38	(ppm)	(ppm)	(ppm)	(ppm)	(ppb)	
Mean ± S.E.	139.93 ± 25.21	8.53 ± .58	117.25 ± 18.63	24.30 ± 1.56	163.39 ± 29.88	17.63 ± 4.43
Classification						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	18.42	86.84	23.68	23.68	76.32
Marginal, %	--	81.58	13.16	39.47	36.84	2.63
Deficient, %	--	0	0	36.84	39.47	21.05
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level	Antagonistic Level	%>MTC*	
			% Marginal	% High		
Iron	178.58 ± 33.94 ppm	71.05	18.42	5.26	2.63	
Molybdenum	1.24 ± .20 ppm	63.16	28.95	7.89	2.63	
Sulfur	0.17 ± .01%	21.05	23.68	7.89	2.63	
*Maximum Tolerable Concentration						

There were 70 samples classified as grass, which can be a mixture of native and cultivated forages. (Table 18). Nearly half the samples were deficient in selenium and over a third were deficient in zinc. A high percentage of the forages contained sufficient copper antagonists to make copper supplementation important even for the 30% of the forages which were considered adequate in copper. For example, 20% of the forages had copper to molybdenum ratios that could cause a copper deficiency. Adams (284) summarized data from 352 grasses grown in Pennsylvania. The standard deviations for zinc and copper concentrations were 10.7 and 8.4 ppm, respectively. Again, these data show that nutritionist must look at the variation among samples and the concentration of antagonistic elements to devise a supplementation package that will be adequate in a majority of the grazing situations.

Table 18. Grass

Samples N=70	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	115.96 ± 12.72	9.96 ± .65	114.32 ± 11.38	29.39 ± 2.23	165.77 ± 25.60	15.01 ± 2.70
Classification						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	30.00	88.57	34.29	22.86	71.43
Marginal, %	--	70.00	11.43	30.00	28.57	8.57
Deficient, %	--	0	0	35.71	48.57	20.00
Copper Antagonists						
Element	Mean ± S.E	% Ideal	Antagonistic level % Marginal	Antagonistic Level % High	%>MTC*	
Iron	152.94 ± 17.90 ppm	84.29	11.43	4.29	0	
Molybdenum	1.68 ± .27 ppm	38.57	51.43	10.00	2.86	
Sulfur	0.19 ± .01%	34.29	31.43	7.14	0	
*Maximum Tolerable Concentration						

Ensiled forages (n=31) was the last category of forages (Table 19). These ensiled forages were primarily corn, sorghum and small grains. Over 75% were marginal or deficient in copper and zinc and 55% were marginal or deficient in selenium. Three-fourths of the samples were adequate in manganese. The copper to molybdenum ratio was such at over 19% would be marginal or deficient in copper. Over 35% of the samples had marginal or highly antagonistic levels of iron and molybdenum.

Table 19. Silage/Silage Grass

Samples N=31	Aluminum (ppm)	Copper (ppm)	Manganese (ppm)	Zinc (ppm)	Selenium (ppb)	Copper to Molybdenum Ratio
Mean ± S.E.	135.08 ± 20.98	10.49 ± 1.79	61.13 ± 8.37	24.97 ± 1.84	207.68 ± 28.41	26.88 ± 6.76
Classification						
MTC*, %**	0	0	0	0	0	0
Adequate, %	--	22.58	74.19	22.58	45.16	80.65
Marginal, %	--	77.42	25.81	45.16	22.58	6.45
Deficient, %	--	0	0	32.26	32.26	12.90
Copper Antagonists						

Element	Mean ± S.E	% Ideal	Antagonistic level	Antagonistic Level	%>MTC*
			% Marginal	% High	
Iron	252.12 ± 36.72 ppm	61.29	19.35	19.35	0
Molybdenum	.91 ± .15 ppm	64.52	35.48	0	0
Sulfur	0.14 ± .01%	16.13	9.68	3.23	0

*Maximum Tolerable Concentration

Results of this study with 709 forage samples from 23 states shows that trace mineral supplementation of cattle, sheep and horses fed forage based diets is critical. Zinc, selenium and copper were either marginally deficient or deficient in 77.0, 69.5, and 66.7% of the forage samples analyzed, respectively. In other words, only 23.0, 30.2 and 33.3% of the samples were adequate in zinc, selenium and copper, respectively. The ability of these forages to provide copper is further compromised by the relatively high incidence of marginal or highly antagonistic concentrations of iron, molybdenum and sulfur.

A similar study was done by the government of Alberta. They determined the trace mineral concentrations in barley grain, grass-alfalfa hay, legume hay, brome hay, oat hay and cereal silages. Over 90% of the feeds tested were deficient in copper, zinc and iodine. Approximately 45% of the alfalfa and 65% of the grasses and cereal forages were deficient in selenium (294). In the Tennessee forage mineral survey involving 1021 forage samples, 92.4% were at least marginally deficient in copper and 83.1% were at least marginally deficient in zinc (293). Results of a study evaluating the variability of trace mineral concentrations within common feedstuffs are striking (319). For example, corn silage copper, manganese, zinc and selenium concentrations varied from 2 to 88, 4 to 304, 9 to 348 and 0.005 to 0.63 ppm, respectively. These studies show that proper trace mineral supplementation is critical for animals fed forage-based diets.

Suspected Mineral Problem

University of Tennessee researchers have developed a series of recommendations in trying to solve an apparent mineral problem (296). Although this approach was designed for cattle producers, the principles apply to all livestock producers. First, not all problems are due to minerals. Protein and or energy deficiencies, infectious diseases, heavy metal toxicities, etc. may have symptoms similar to a mineral deficiency. Secondly, it is important to be able estimate mineral intake. Knowing the mineral concentration in a supplement or totally mixed rations is important, but combining that information with an accurate estimate of intake is critical. Third, work with your mineral supplier to get the best mineral supplementation package for your animals. While certain mineral packages may be effective under a wide range of dietary conditions, a “one size fits all” approach may not be the best for your conditions. Next, remember that with minerals you can get too much of a good thing. Adding high levels of one mineral without looking at its concentration relative to the other minerals in the diet may solve one problem but create another. On the other hand, remember there may be situations when concentrations above the recommended requirement, may not be adequate. For example, variation due to genetics of the animal, disease or environmental stress, antagonist,

and bioavailability of the mineral source need to be considered. Finally, work with a trained nutrition professional to help sort out the complexities of your situation.

Salt as a Carrier of Trace Minerals

Salt is a natural carrier for trace minerals, since all farm animals have a natural appetite for salt. Moreover, when cattle, horses, sheep and other animals are on pasture with little, no or varying amounts of concentrate feeding, producers can supply trace mineralized salt free-choice in the form of a mineral block or as loose trace mineral salt in a box. Then, regardless of the amount of concentrates fed, and especially if none is fed, the animal can still consume salt and the trace minerals it contains.

Wisconsin studies with sheep showed that there was a large variability in salt-mineral mixture intake. They stated, however, that salt-mineral self-feeding is usually effective in preventing mineral deficiencies and is the most cost effective method of delivery (138).

The trace mineral levels in salt or salt-based mineral products are guaranteed on the package. Different levels of various minerals are added to salt for specific and different situations. Table 20 shows an example of the information salt companies provide on each of their products. Therefore, farmers and others can obtain various salt products to fit their specific needs. Arizona researchers demonstrated the advantage of being able to choose a trace mineral package best suited for each producer's unique situation. When a long-acting trace mineral bolus was given to beef cows over two years there was a breed by year by bolus interaction (315). These researchers concluded that there may be a need for a breed- and year-specific supplementation program.

Table 20. An Example of a Swine-Mineral Guaranteed Analysis

In Percentage	
Minimum Manganese (Mn)	1.00%
Minimum Zinc (Zn)	1.00%
Minimum Iron (Fe)	0.80%
Minimum Copper (Cu)	0.80%
Minimum Iodine (I)	0.02%
Minimum Cobalt (Co)	0.01%
Maximum Salt (NaCl)	95.5%
Minimum Salt (NaCl)	92.5%

Cost of Adding Trace Minerals to Salt

A U.S. Survey conducted in 1966 by the late Dr. G. Bohstedt, Emeritus Professor, University of Wisconsin, indicated that livestock and poultry consumed the annual levels of salt shown in Table 21. Dr. Bohstedt's figures on salt consumption are similar to those that Dr. Cunha obtained on a worldwide survey on salt consumption by animals (52).

Table 21. Average Salt Consumption per year in the U.S.

Animal	Pounds of Salt Consumed Yearly	Approximate Yearly Cost for Six Trace Minerals in Salt (cents)¹
Dairy Cattle	53.5	240
Horses	24.0	108
Beef Cattle	22.0	100
Heifers (1-2 yr old)	18.0	81
Calves	9.0	40
Swine	9.0	40
Sheep	9.0	40
Lambs	4.5	21
Milk Goats	18.0	81
Meat Goats	9.0	40
Kids	4.5	21
Water Buffalo Cows	29.25	180
Poultry (in feed)	0.417	2

¹ Assuming that trace mineralized salt (with copper, cobalt, iron, manganese, zinc and Iodine) costs \$90.00 more per ton, or 4.5 cent more per pound than plain salt. This does not include the cost of selenium, which will increase the costs depending on the selenium concentration.

By using the data in Table 21, the amount of salt consumed annually by various classes of animals can be determined. This can be used as a guide on how much salt to supply in the feed, as loose salt in mineral boxes or with salt blocks. The levels of salt consumption shown in Table 8 vary,

depending on the diet fed, the level of production, the area of the country and many other factors. They are good, average figures to use as a guide.

The cost of adding the six trace minerals to salt is very low, ranging from two cent for poultry to \$2.40 for dairy cattle for a whole year. Horses, beef cattle and dairy goats can be supplied trace minerals with salt for a year for approximately \$1.00; and calves, swine, sheep and meat goats for less than 50¢. This is certainly low-cost insurance compared to the benefits derived. If selenium is also added to salt, at a level of 20 to 30 ppm, the cost will be about one cent more per pound.

Value of Trace Minerals

A number of recent studies have shown that adding trace minerals to animal diets benefits animal productivity even when no deficiency symptoms are obvious. For example, a recent Texas study (205) demonstrated the effectiveness of feeding supplemental zinc, copper and manganese to improve reproductive efficiency of cows grazing native range. The four treatments studied were: (1) control (no supplementation); (2) 2.2 pounds of grain-urea mix; (3) grain mix plus 15 grams phosphorus and; (4) grain mix plus phosphorus plus the three trace minerals. The average length of time from the start of the breeding season to conception was 42, 35, 29 and 22 days respectively for the four nutritional treatments. The seven-day earlier conception rate would result in approximately 14 pounds increased weaning weight due to trace mineral supplementation alone. Applying recent economics, the return on investment is approximately 29:1 for trace mineral supplementation. The authors stated that, "Possibly, specific nutrient requirements, especially trace minerals, are at times of greater importance than body conditions for satisfactory reproductive performance in the beef cow."

Alberta researchers have also documented the value of trace mineral supplementation for their beef producers. In "Farming for the Future" demonstration projects trace mineral supplementation improved weaning weights by 14 to 26 lbs per calf and yearling weights on pasture were increased 20 to 36 lbs. These improvements occurred without clinical trace mineral deficiency symptoms and where producers did not perceive that growth rates were impaired (294). Paterson and Engle (2005) recently published an excellent review of the benefits of trace mineral supplementation of beef cattle with an emphasis on economically important traits (302).

In a review on trace minerals, Dr. A. A. Jimenez stated that some deficiencies of trace minerals in dairy cows have been implicated in reproductive problems such as retained placenta, anestrus, infertility and abortions (140). He also stated that trace mineral fortification of dairy cows producing 65 pounds of 3.5% fat-corrected milk amounted to 0.11% of the total feed costs.

Similar responses to trace mineral supplementation have occurred in growing pigs. Researchers at the University of Nebraska (206) conducted a growing trial with or without trace mineral supplementation of pigs on pasture. On average, trace minerals increased gains 0.09 lbs per day. The study showed that the cost of trace minerals amounted to 4.7 cents per pig, but returned \$1.64 per pig. Consequently for every penny invested in trace minerals, the producer would have received 35 cents in return. Another swine study (207) showed that omitting salt and six trace minerals from a corn-soy diet resulted in a 17% depression in growth rate. Although impossible to calculate return on investment in trace minerals because the diet was deficient in salt, this trial affirms the necessity

of fortifying corn-soybean meal based diets with trace mineralized salt. Similarly, a study at Ohio showed that deleting trace minerals from growing-finishing pig diets lowered feed intake and gain but did not affect feed efficiency (54). When trace minerals were added back to the diets, an immediate growth response was observed. A Purdue study with growing pigs showed that adding a trace mineral mixture improved gains 15.4% compared to controls fed no trace minerals (53). Research by Edmonds and Arentson (279) showed that deleting trace minerals from the diet of finishing pigs can reduce the nutrient concentration of the meat. Ham from pigs without trace mineral supplementation has significantly lower copper concentrations compared to those that were supplemented. Other researchers are concerned that removing trace minerals may compromise the welfare of the animals (308). These are only a few of the many examples that could be cited on the value of adding trace minerals to animal diets. More detail on specific trace mineral deficiency symptoms will be given later in this publication.

Responses to trace mineral supplementation can be subtle and not easily measured. For example, small changes in fertility, immunity, or longevity are economically important, but difficult to connect with a given trace mineral. Australian researchers reported that when sheep were supplemented with cobalt while grazing a pasture known to be cobalt deficient, a measurable response was only observed in 9 of 14 years (299).

Some consumers have questioned the wide spread practice of trace mineral supplementation when clinical trace mineral deficiencies are not observed. Their concern is that trace minerals may be stored in the body and that meat products may contain abnormally high levels. Irish researchers measured the copper, selenium, and iodine concentrations in tissues from 2,612 cattle harvested in the late 1990s. This followed marked improvements in blood copper and selenium concentrations due to increased supplementation over the past 20 years. Only 0.3, 2.3, and 4.0% of the cattle had high copper, selenium, or iodine concentrations, respectively. The term “high” means above of the normal range of concentrations, but does not imply anything approaching toxicity. The conclusion of this study was that current levels of trace mineral supplementation posed absolutely no threat to the human food chain (295).

ZINC FOR ANIMALS

Zinc is widely distributed throughout the body and plays an essential role in many body processes. Radioactive zinc given orally or intravenously reached peak concentrations in the liver within a few days, but concentrations in red blood cells, muscle, bone and hair do not peak for several weeks. Zinc is present in many enzyme systems that are concerned with the metabolism of feed constituents. For example, zinc is a constituent of carbonic anhydrase, carboxypeptidase A and B, several dehydrogenases, alkaline phosphatase, ribonuclease and DNA polymerase. Zinc is required for normal protein synthesis and metabolism, and it is also a component of insulin so that it functions in carbohydrate metabolism. Because zinc plays so many important roles in the body, it is required by all livestock and poultry (Table 22).

Table 22. **Zinc Requirements and Toxic Levels¹**

Class of Animal	Zinc Requirement in total Diet (ppm)	Toxic Level in Total Diet (ppm)
Beef Cattle	30	500 ³
Dairy Cattle	23-63 ²	300-1,000
Swine	50 (50-100) ²	2,000-4,000 ²
Horses	40	Over 700
Sheep	20-33	750 ³
Goats	45-75	750 ⁴
Chickens	40-50	800-3,000
Turkeys	40-75	4,000

¹ Levels recommended by NRC committees, unless otherwise indicated.

² Range given in latest NRC publication.

³ Maximum tolerable level in diet.

⁴ Same level as for sheep.

Absorption of zinc occurs throughout the small intestine and usually ranges from 5% to 40% of the intake. Transfer of zinc out of the intestinal mucosal cells to the plasma is regulated by metallothionine. Zinc absorption is reduced whenever diets are high in calcium or phytate (215). Common sources of supplemental zinc include zinc sulfate, zinc oxide, zinc chloride, zinc carbonate and zinc chelates.

Beef Cattle

A severe deficiency of zinc in young calves results in parakeratosis (a condition that resembles mange). The nose and mouth become inflamed with sub-mucosal hemorrhages. The animal also develops an unthrifty appearance, a roughened hair coat and joint stiffness. A mild zinc deficiency in finishing cattle results in lowered weight gains, but they show no clinical signs of a deficiency (85, 157). Excessive salivation is an early sign peculiar to ruminants. It may be caused by a reluctance to swallow the large amount of saliva that is normally produced (228). Hypogonadism is a common occurrence in zinc deficient bull calves.

Many recent studies have shown zinc to be essential to maximum immune function in stressed feedlot cattle. Texas researchers (208) reported that when steer calves were challenged with virulent infectious bovine rhinotracheitis (IBR) virus, serum zinc levels decreased significantly. The same author showed that during a natural outbreak of bovine respiratory disease, serum zinc levels were lowest at the time of peak morbidity. Substantial losses in immune capacity can occur due to inadequate zinc intakes before typical zinc deficiency symptoms appear (209).

A USDA study in Idaho by H. R. Mayland and co-workers showed that cows and their suckling calves grazing mature dry forage supplemented with zinc resulted in calves gaining 6% more weight (90). The forage being fed contained less than 20 ppm zinc. In cattle zinc plays a critical role in the proteolytic enzyme systems associated with muscle protein turnover. Muscle protein accretion decreased when supplemental zinc was removed for 21 days and then returned to normal after 14 days of supplementation (307). Some foreign scientists have reported signs of zinc deficiency in cattle grazing forages containing 20 to 30 ppm zinc. Missouri researchers drew blood on 529 feeder calves to evaluate zinc status. They found that 2.4% of the calves were zinc deficient and 24.4% were marginally deficient (324). In a recent Tennessee study, 83.1% of the 1021 forages samples analyzed were considered deficient or marginally deficient in zinc (293). Florida researchers have reported zinc deficiencies in four regions of the state (125). One must keep in mind that forages may differ in zinc level and availability and that the stage of maturity may also affect zinc availability. Data of Emanuele and Staples (210) showed that the maximum availability of zinc in bermudagrass and alfalfa was only 62.1% and 79.4%, respectively. The Idaho, Florida and other studies indicate a need to be concerned about the adequacy of zinc in cattle grazing forages, especially with mature, dry forages or hay made from them.

The zinc requirement for normal beef cattle appears to be between 20 and 40 ppm in the total diet. However, this requirement probably doubles during time of stress. Hutcheson (208) suggests that dietary zinc should be increased to approximately 80 ppm for stressed and sick feedlot cattle due to decreased feed intake and increased excretion of body zinc stores. Excess calcium in the diet may also increase the zinc requirement. The pig, for example, may need at least twice as much zinc if excess calcium is consumed in the diet. The influence of calcium level on zinc requirement is probably less in ruminants than non-ruminants (337). Zinc toxicity is seldom a problem. However, high levels of zinc caused harmful effects in beef cattle fed 900 ppm, which results in reduced gains and feed utilization (157). The 1984 NRC publication (157) gives 500 ppm as the maximum tolerable level.

Dairy Cattle

Lowered feed intake is one of the first changes observed in a zinc deficiency. The cattle grow slower due to a decreased feed intake and less efficient feed utilization. Other symptoms in a severe zinc deficiency are skin parakeratosis (usually most severe on the legs, neck and head), hair loss, unthrifty appearance, stiffness of joints, teeth gnashing, retarded testicular growth and excessive salivation. Reduced reproductive performance has been observed in both males and females fed zinc deficient diets (211). Another effect of a zinc deficiency is a failure of wounds to heal normally. In most cases, when a zinc deficient animal is given zinc, there is a dramatic and quick recovery. Improvements are observed within 24 hours after supplementation.

The estimated zinc requirement for dairy cattle is 40 ppm in the diet (156). There may be certain conditions or an interrelationship with other nutrients that might increase zinc needs. For example, a small percentage of Dutch-Friesian calves are born with an apparently inherited defect that causes a very severe zinc deficiency which can be temporarily corrected by very high amounts of zinc. Whether this means there are genetic differences affecting zinc needs is not well established. A review of the various experiments conducted on the zinc requirements of dairy cattle and the levels needed in each one indicate there is considerable variation in the requirements obtained. However, factors other than genetics are also involved (91).

Studies on excess zinc levels indicate that lactating dairy cows fed 1,279 ppm zinc in the diet did not experience reduced performance (212). Growing cattle fed 900 ppm zinc exhibited decreased weight gains and decreased feed efficiency. Based on these and other studies, the 1980 NRC publication, *Mineral Tolerance of Domestic Animals*, suggest that 1,000 ppm zinc in the diet is the point at which adverse physiological effects are observed (97).

Regardless of the level of zinc fed previously, cattle fed a severe zinc deficient diet may develop a deficiency within a few weeks. In other words, body stores of zinc do not last very long. The average zinc content of milk is about 4 ppm, but there is considerable difference among cows in the level of zinc in their milk. Milk zinc concentrations will decrease rapidly in response to a dietary deficiency (213).

Swine

The symptoms of zinc deficiency are reduced appetite and growth rate, skin lesions (parakeratosis) that look like mange, diarrhea, vomiting, and death in severe cases. Borderline deficiencies produce decreased appetite and growth in some animals, while others may show a fading or bleaching of the hair coat. A decrease in litter size and weight of pigs occurs with the sow, and retarded testicular development occurs with the growing boar. The zinc deficient pig also shows reduced tissue and blood zinc levels and reduced blood alkaline phosphatase activity (87).

Zinc is also critical to immune function in the pig. Miller (214) reported that zinc deficient pigs died following an intraperitoneal injection with *Salmonella pullorum* antigen, while there was no mortality in pigs receiving adequate dietary zinc and challenged similarly. One mechanism by which a zinc deficiency can impair the immune system is by causing atrophy of the thymus gland. This was demonstrated dramatically in the Miller study (214) where pigs fed zinc adequate diets

(100 ppm) had thymus glands weighing 51 grams while the thymus glands of pigs fed 12 ppm zinc weighed only 2 grams. The small intestine is an organ that plays a key role in immune function. Louisiana State researchers showed that adding an additional 100 ppm zinc from zinc sulfate to a control diet containing a 100 ppm zinc during gestation increased the increased the jejunal villus height of piglets at 28 days of age (359). The mechanism by which zinc level consumed by the mother influences gut development of her offspring a month after birth is being investigated.

A pig with parakeratosis responds very quickly and dramatically to zinc. Appetite increases immediately and an improvement in skin condition and weight gain is quite obvious within a week. The pigs soon recover from the skin lesions and other symptoms and may be completely recovered within one month.

The requirement for zinc by the pig is about 50 ppm in the diet. If the calcium level in the diet is excessive, the addition of 50 to 100 ppm zinc to the diet will not always completely prevent the growth depression and poor feed conversion associated with parakeratosis, although it will prevent the typical skin lesions. Therefore, under some conditions, a level of 100 to 150 ppm zinc is needed. However, in most cases, a level of 100 ppm zinc should be adequate. The level of calcium that causes parakeratosis will vary considerably. Sometimes a high calcium diet that supposedly should cause parakeratosis does not do so, and sometimes parakeratosis occurs with a low level of calcium (87, 92).

The zinc in soybean meal, cottonseed meal, sesame meal and other plant protein supplements has low availability to the pig (and also to the chick). The reason for this is these supplements are high in phytic acid, which combines with zinc to form zinc phytate, this complex is insoluble in the intestinal tract and cannot be absorbed. Therefore, the zinc in plant protein concentrates is less available than in animal protein supplements, such as meat meal and fish meal which contain no phytic acid. For example, the zinc requirement of the pig fed soybean meal is 50 ppm, whereas it is 18 ppm for pigs fed casein (animal protein) as the source of protein in the diet (87).

There is no danger in feeding pigs up to 150 ppm zinc in the diet (which occasionally is done) since over 1,000 ppm has been fed without any harmful effects. However, a level of 2,000 ppm zinc in the diet of the pig can cause toxic effects including growth depression, enteritis, arthritis, gastritis and hemorrhage in the axillary spaces (87). The form and bioavailability of the zinc source can greatly influence the toxic dose. For example, several experiments have shown a performance benefit from feeding high levels of zinc oxide to weanling pigs. Poulsen (229) reported that feeding 28-day old weanling pigs diets containing 2500 ppm zinc from zinc oxide improved gains and feed efficiency. A supplement of 2500 ppm zinc for two weeks post-weaning reduced the incidence of diarrhea by up to 50%. Smith (230) studied the effect of replacing corn starch with zinc oxide to provide 165, 1000, 2000, 3000, and 4000 ppm zinc in the diet on the performance of piglets weaned at 13 days of age. From 0 to 14 days postweaning, increasing the zinc oxide level linearly increased feed intake and feed efficiency. However, the 4000 ppm zinc addition began to depress performance as the pigs got older. The authors concluded that maximum performance was achieved if 4000 ppm zinc was fed from day 0 to 14, and 2000 ppm from day 14 to 28 postweaning. It is unlikely that these performance responses were due to the high dietary zinc improving the nutrient status of the piglets. This was confirmed recently when it was shown that supplemental zinc whether in the form of zinc oxide or in an organic form, was not as efficacious for improving gain

and feed intakes as 2000 ppm zinc from zinc oxide (358). Most nutritionists believe that the high concentrations of zinc from zinc oxide are inhibiting the growth of pathogenic bacteria that commonly affect the early-weaned pig.

The next question to consider is whether the responses to these high levels of zinc are additive to the wide spread practice of feeding 250 ppm of copper. A cooperative study involving 12 universities was reported to address this issue (275). Their summary showed that growth and feed efficiency was improved in nursery pigs when fed either 3,000 ppm of zinc or 250 ppm of copper from copper sulfate. However, no additive or synergistic benefit from feeding the combination was observed.

Monitoring the zinc status of pigs by measuring the zinc concentration in the blood serum or plasma has been a common practice in the past. However, recent research shows that blood zinc concentrations may not be a very sensitive diagnostic tool. Wedekind (231) reported that blood concentrations of zinc were higher in unfed animals than fed animals. The difference actually widened as zinc intakes decreased, to the point that unfed animals had plasma zinc levels twice that of fed animals on the same low-zinc diet. Assessing zinc status is difficult because there are no effective tests for marginal zinc deficiency. In general, if plasma zinc is below 0.4 mg/liter, pigs are considered deficient in zinc.

Horses

The symptoms of a zinc deficiency are similar to those obtained with cattle, swine and sheep. A zinc deficiency in the foal results in reduced appetite and growth rate, parakeratosis with considerable lesions in the feet, legs and head and loss of hair. The horses also show reduced tissue and blood zinc levels and reduced blood alkaline phosphatase (154).

The zinc requirement of the horse is approximately 40 ppm (154). However, some animal scientists recommend the use of 100 ppm zinc in the total diet because many horses will be fed higher levels of calcium than required. One hundred ppm will ensure an adequate level of zinc in the diet and provide a safety margin against the many factors that affect zinc needs. It will also protect the horse against loss of zinc, which may be tied up by the phytate phosphorus in soybean meal and other plant protein supplements. Moreover, most owners like to have their horses with beautiful looking skin and hair which makes it essential that zinc levels in the diet be adequate (98).

Research suggests that zinc along with copper and calcium play key roles in the prevention of Developmental Orthopedic Disease (DOD). Anderson (297) defined DOD of the young horse as any disturbance in the changing of the cartilaginous precursor of the skeleton to functional bone. For optimal bone mineralization in the young growing horse, 60-80 ppm zinc are recommended (298).

The danger of feeding excess zinc is low, since the feeding of 700 ppm in the diet was not detrimental to mares or their foals. Foals fed 20,000 ppm zinc in the diet, however, developed enlarged epiphyses followed by stiffness, lameness and increased tissue zinc levels (154).

Sheep

Zinc deficiency in lambs results in a lack of appetite, reduced growth, slipping of wool, swelling around the eyes and hooves, excess salivation, general listlessness, impaired growth of testes and cessation of spermatogenesis (155). Loss of appetite is the first sign of a zinc deficiency in growing lambs. Recent studies have shown that lambs switch from meal eaters to nibblers (232) as they become zinc deficient. Pair-feeding studies show that many of the signs of a severe zinc deficiency are secondary to a loss in appetite. In a USDA study in New York, ewes were fed a low-zinc diet during the last third of gestation and for the first six weeks of lactation (99). The zinc deficiency caused a few deaths, a continuous loss in body weight during lactation and development of skin lesions and frothy saliva. The rapid deterioration of the ewes after lambing suggests the zinc stores were depleted by the end of pregnancy and the marginal zinc levels may have contributed to the deaths that occurred. A recent study showed that 7 of 30 ewes fed a low-zinc diet aborted, reabsorbed or delivered mummified and deformed lambs, while the other 23 ewes delivered lambs that were 20% smaller than the controls (153). Feeding a diet containing only 3 ppm zinc during pregnancy reduced survival of the newborn lambs and caused pregnancy toxemia in the ewes as a result of anorexia (234). White (233) showed that zinc-deficiency-induced anorexia caused reduced secretion of gonadotrophin-releasing hormone from the hypothalamus of ram lambs. This will lead to impaired fertility in the ram.

The requirement of zinc for sheep is 20-33 ppm in the diet. The maximum tolerable level in the diet is 750 ppm (155). Excess zinc will cause a copper deficiency.

Goats

Zinc deficiency in goats includes reduced feed intake, weight loss, parakeratosis (mange-like condition), stiffness of joints, excessive salivation, swelling of the feet and horny overgrowth, small testicles and low libido (120).

A level of 45 to 75 ppm zinc should be used in the total diet of goats until their zinc requirements are met.

Poultry

Zinc deficiency causes growth retardation and abnormal feather development in poultry. Feather fraying occurs near the end of the feather. The severity of the fraying varies from almost no feathers on the wings and tail to only slight defects in the development of some of the barbules and barbicels. The hock joint may become enlarged. The long bones of the legs and wings also become shortened and thickened with a zinc deficiency. Other symptoms include scaling of the skin, especially on the feet, loss of appetite, reduced efficiency of feed utilization, and mortality in severe cases. Zinc deficiency in the breeder diet reduces egg production and hatchability. Embryos produced in zinc deficient eggs show a wide variety of skeletal abnormalities in the head, limbs and vertebrae. The hatched chicks also may not stand, eat or drink (95, 141). Proper zinc supplementation has proven to be important in reducing early mortality of turkey poults (306).

The 1984 NRC publication, Nutrient Requirements of Poultry, recommends a level of 40-75 ppm zinc in various poultry diets (141). This publication indicates that the toxic level of zinc varied from 800 to 4,000 ppm in the diet (141).

Other Animals

There is a lack of quantitative data on the zinc requirements of small animals. In some cases the latest National Research Council publications give suggested levels to use or levels of zinc that have been used successfully. These levels in ppm in the total diet are as follows: dogs, 60 to 90; cats, 30; fish, 15 to 78; rats, 12; mice, 30 to 58; guinea pigs, 20; hamsters, 9.2 and gerbils, 8.4. These levels can be used as guides until more definitive information is obtained. All animals need zinc in the diet but, in most cases, research has not been conducted to determine how much is needed. In a few instances where some deficiency symptoms were given, there was some similarity to those reported for domestic livestock.

Zinc Sources

Supplemental zinc is usually added to animal diets in the form of zinc oxide or zinc sulfate. Recent comparisons of bioavailability in chicks suggest that feed grade zinc oxide has only 44-78% the availability of zinc sulfate when added to purified (235) or practical (236) diets. A recent comparison of availabilities of zinc sources for pigs in corn-soy diets ranked zinc sulfate > zinc methionine > zinc oxide > zinc lysine (237).

IRON FOR ANIMALS

Iron has been recognized as an essential nutrient for over 100 years. Despite this fact, iron deficiency is still a major problem in several segments of the livestock industry.

Approximately two-thirds of body iron is present in hemoglobin in red blood cells and myoglobin in muscle, 20% is in labile forms in liver, spleen and other tissues with the remainder in unavailable forms in tissues such as myosin and actomyosin and in metalloenzymes. In hemoglobin, which contains 0.34% iron, an atom of ferrous iron in the center of a porphyrin ring connects heme, the prosthetic group, with globin, the protein. The iron in hemoglobin is essential for the proper function of every organ and tissue of the body. Iron also plays a role in other enzymes involved in oxygen transport and the oxidative process, including catalase, peroxidases, flavoprotein enzymes and cytochromes.

Iron in blood plasma is bound in the ferric state (Fe^{+++}) to a specific protein called transferrin. Transferrin is the carrier of iron in the blood and is saturated normally only to 30-60% of its iron-binding capacity (215).

The duodenum is the main site of iron absorption. However, only 5% to 10% of what is consumed is absorbed and then only if it is in the ferrous state (Fe^{++}). Once absorbed, the body tenaciously holds on to the iron for reuse. For example, most of the iron released from red blood cell breakdown is used to synthesize new hemoglobin. However, if the red blood cells are not replaced as rapidly as they are destroyed, anemia occurs. Anemia may occur at any stage of life, but it is especially likely to occur in certain species during the suckling period, since milk is very low in iron.

Iron is very low in the milk of cows, goats and sows. It varies from 0.5 to 1.0 ppm. Since pigs depend heavily on mother's milk during the first two to three weeks of life, they need iron supplementation because their body stores are unusually low.

This magnitude of growth rate imposes a greater demand on iron needs than occurs with young ruminants. There is little evidence of an iron deficiency occurring with calves, lambs and kids raised under grazing conditions, except when blood loss or disturbance in iron metabolism occurs because of parasitic infection or disease. This is because they start early to eat food other than mother's milk. Iron supplementation is needed, however, when young ruminants are fed an exclusive whole milk diet. Young nursing calves and lambs, receiving no supplemental source of iron, have responded to intramuscular injections of iron-dextran by improved hemoglobin levels and growth rate.

Table 23 gives information on the iron requirement of animals as well as the levels that may result in harmful effects.

Table 23. **Iron Requirement and Toxic Levels**¹

Class of Animal	Iron Requirement in Total Diet (ppm)	Toxic Level in Total Diet (ppm)
Swine		
Baby Pigs	140 –150	3,000
Growing-Finishing	80	3,000
Sows	80	3,000
Dairy Cattle		
Calves	100	1,000
Other Cattle	25	1,000
Beef Cattle	50	1,000 ²
Sheep	30-50	500 ²
Goats	30-50 ³	4
Horses	40-50	4
Poultry	50-80	4,500

¹ These are the nutrients given by the latest NRC publication on nutrient requirements of animals, unless indicated otherwise.

² Maximum tolerable level in diet.

³ Requirements not known. The same level as recommended for sheep is used.

⁴ NRC gives no recommendation.

Swine

The pig is born with about 47 mg of iron in body stores. The iron storage will last about a week, since it is estimated the pig will need 7 mg of iron absorbed daily for normal growth. In many cases, however, the iron stored is dangerously low by 3 or 4 days of age. Sow's milk is very low in iron and contains about 1 ppm, whereas the baby pig's iron requirement is 140 to 150 ppm in the total

diet. Plasma volume increase markedly during the first 12 hours after the newborn begins nursing. However, because there is little change in red blood cell volume the piglet develops physiological anemia rapidly. Therefore, unless the pig is given extra iron, it will become anemic as early as 5-10 days of age. During the first two to three weeks of life, the baby pig depends mostly on milk for its food supply. Baby pigs usually double their weight by one week of age and redouble it again in another week or ten days. Therefore, the pig rapidly outgrows its iron supply. So, iron supplementation is very important for the baby pig (87, 92).

Some of the symptoms of an iron deficiency are poor growth, listlessness, rough hair coat, anoxia, wrinkled skin, paleness of mucous membranes, hypochromic microcytic anemia, enlarged heart and spleen, enlarged fatty liver and ascites. A characteristic sign is labored breathing after minimal activity from which the term "thumps" arose. Blood hemoglobin is a reliable indicator of the iron status of the pig. Usually, a level of less than 9 grams of hemoglobin per 100 milliliters of blood results in borderline anemia (87, 92).

Attempts to significantly increase the iron content of sow's milk by feeding or injecting high levels of iron during late gestation and lactation have not been successful to date. Therefore, the practice of injecting the baby pig with 150 to 200 mg of iron during the first three days after birth is recommended. Moreover, iron supplementation of prestarter and starter diets is widely used by industry (104). Nursery pigs were fed diets supplemented with 0, 25, 50, 100, 150 ppm iron in the diet (as-fed basis) from ferrous sulfate. Whole body iron stores increased linearly due to increasing dietary iron concentrations (314). These results show that the basal diet was inadequate to maintain indices of iron status and that there was an increased risk for the pigs to develop anemia during the grower and finisher periods.

Proper iron nutrition is essential for optimum immune function. Nursing pigs made anemic by withholding supplemental iron were more susceptible to the lethal action of bacterial endotoxins than their littermates that had been given iron (158). Parsons et al. (159) reported that pigs receiving iron intramuscularly prior to four weeks of age were more likely to survive a transmissible gastroenteritis outbreak and recovered more rapidly than anemic pigs.

Dairy Cattle

Iron reserves of the newborn calf are usually sufficient to prevent a severe iron-deficiency anemia if dry feeds are fed within a few weeks after birth. Iron supplementation is needed, however, when calves are fed exclusively a whole milk diet. With older cattle, an iron deficiency seldom occurs unless there is a loss of blood caused by a parasitic infection or disease.

Iron deficiency results in anemia, reduced gain, listlessness, inability to withstand circulatory strain, labored breathing after mild exercise, reduced appetite, decreased resistance to infection, blanching of visible mucous membranes and a pale color of the muscle meat (91, 156).

Beef Cattle

The iron requirement of calves is about 100 ppm and 50 ppm appears adequate for older cattle (157). Calves on an exclusive milk diet develop an iron deficiency. Anemia and decreased growth rate occur (85, 157).

Sheep

Little is known about iron requirements for sheep. An iron deficiency anemia sometimes occurs with lambs raised on slotted, wooden floors. The anemia can be prevented by iron supplementation in the creep-feed or by intramuscular injections of iron-dextran. Two injections given three weeks apart, each of 150 mg iron, are effective (155).

Goats

Very little is known about iron requirements for goats. A deficiency may occur with young goat kids because of their low body stores at birth and the low iron content of milk. If iron deficiencies are observed and the kids are continued on a milk diet, injection of 150 milligrams iron-dextran at two-to-three-week intervals is recommended by the 1981 NRC publication, Nutrient Requirements of Goats (100).

Horses

Little is known about iron requirements for horses. An iron deficiency will result in anemia. Anemia may result with horses that are heavily parasitized. This is important, since horses are very susceptible to parasites and are treated for them more frequently than are other classes of livestock. Adequate iron is very important for racing and performance horses, which need a high hemoglobin level for proper oxygen transport and endurance (98, 154).

Poultry

Iron deficiency produces a microcytic, hypochromic anemia in chickens. This type of anemia also occurs with all other animals. Iron deficiency also produces a complete depigmentation of the normally red and black feathers of the Hampshire chicken (141). The iron requirement of chicks fed a casein, dextrose, and isolated soybean protein concentrate-based diet was studied by Aoyagi and Baker (238). Hemoglobin data were used to estimate the chick's requirement at 85 ppm in the diet. Heart hypertrophy was observed if less than 70 ppm iron were fed. Duck diets are usually formulated to provide 70-80 ppm of iron (300).

Other Animals

There is a lack of adequate information on iron needs and usage with most small animals. Iron requirement of the rabbit is not known. At birth, rabbits have a very large iron reserve and, therefore, are not as dependent on a supply of iron in the milk and are not as susceptible as some other animals to iron deficiency anemia during pre-weaning (89).

The dog needs 31.9 ppm iron in the diet. An iron deficient dog develops anemia and tissue anoxia (142). The cat requires 100 ppm iron in the diet. A deficiency of iron results in anemia (102).

Iron requirement of foxes is not known. In mink, a fur abnormality known as cotton fur, has been linked to an iron deficiency. A normal pelt was produced with 114 ppm iron in the diet, so this level might be used as a guide until a more adequate level is determined. Levels of 20 to 30 ppm iron are considered adequate by some, but others recommend levels of 60 to 88 ppm iron, and Scandinavian mink diets contain as high as 156 to 352 ppm iron (143). Iron deficiency symptoms in mink usually include growth retardation, anemia, severe emaciation, roughened fur and a lack of underfur pigmentation (achromotrichia) (143).

Iron supplementation increased trout growth and prevented anemia, but the iron requirement is not known. Trout may be able to absorb iron directly from water to meet requirements (144).

Iron requirement of non-human primates is 180 ppm in the diet. Iron deficiency results in anemia (101). Other laboratory animals need iron for anemia protection, but the requirement is not known in most species. The rat requires 35 ppm iron in the diet. The mouse requires 25 ppm for growth; a level of 120 ppm iron in the diet is the lowest level tested for reproduction and was satisfactory. The guinea pig needs 50 ppm in the diet for growth (103).

COPPER FOR ANIMALS

Copper is required for the activity of enzymes associated with iron metabolism, elastin and collagen formation, melanin production, and the integrity of the central nervous system. It is required for normal red blood cell formation by allowing iron absorption from the small intestine and release of iron in the tissue into the blood plasma. Ceruloplasmin is the copper-containing transport protein. Copper is required for bone formation by promoting structural integrity of bone collagen and for normal elastin formation in the cardiovascular system. Copper is required for normal myelination of brain cells and spinal cord as a component of the enzyme cytochrome oxidase which is essential for myelin formation. Maximum immune response is also dependent on copper as indicated by depressed titers in deficient animals.

The process of normal hair and wool pigmentation requires copper. It is believed that copper is a component of polyphenyl oxidase which catalyzes the conversion of tyrosine to melanin and for the incorporation of disulfide groups into keratin in wool and hair (215).

A minimum requirement for copper cannot be given with great accuracy, since copper absorption and utilization in the animal can be markedly affected by several mineral elements and other dietary factors. Zinc, iron, molybdenum, inorganic sulfate and other nutrients can reduce copper absorption. For example, it has been known that molybdenum can depress copper absorption in grazing ruminants. However, only recently have we understood the role of sulfur in intensifying the interaction of molybdenum and copper. Sulfur has its affect by forming thiomolybdates in the rumen (160). In 1991, Spears (161) described the following reactions involved in the formation of thiomolybdates which inhibit copper metabolism.

1. $\text{MoO}_4^{2-} + \text{H}^+ + \text{HS}^- \rightarrow \text{H}_2\text{O} + \text{MoO}_3\text{S}^-$ (monothiomolybdate)
2. $\text{MoO}_3\text{S}^- + \text{H}^+ + \text{HS}^- \rightarrow \text{H}_2\text{O} + \text{MoO}_2\text{S}_2^{2-}$ (dithiomolybdate)
3. $\text{MoO}_2\text{S}_2^{2-} + \text{H}^+ + \text{HS}^- \rightarrow \text{H}_2\text{O} + \text{MoOS}_3^{3-}$ (trithiomolybdate)
4. $\text{MoOS}_3^{3-} + \text{H}^+ + \text{HS}^- \rightarrow \text{H}_2\text{O} + \text{MoS}_4^{4-}$ (tetrathiomolybdate)

Thiomolybdates bind with copper in the rumen to form insoluble complexes which are poorly absorbed. However, some thiomolybdates are absorbed and affect copper metabolism in the body (160). It has been discovered that thiomolybdates cause copper to be bound to blood albumins which renders the copper unavailable for any biochemical reaction in the body. Price (162) reported that the tri- and tetrathiomolybdates were the sulfur-molybdenum complexes responsible for reducing copper absorption while the di- and trithiomolybdates had the greatest effect on copper metabolism in the body. These data show how important it is, when evaluating the copper intakes of ruminants, to consider the amount of molybdenum, and the amount of sulfur in the diet.

Likewise, copper availability changes with the forage specie and preservation technique (219). For example, fresh grass has the lowest availability and is further reduced by small increments of molybdenum and sulfur. In forages preserved as hay, the inhibitory effects of molybdenum are detectable, but less than that of sulfur. In silages, molybdenum has a small effect but sulfur reduces copper availability in a logarithmic manner. Estimated copper availability for grazed forage, silage and hay were 1.4, 4.9 and 7.3%, respectively, in Scottish Balckface ewes when molybdenum was

less than 2.0 ppm. These variations in copper availability with preservation method are probably due to changes in the release rates in the rumen of copper and its antagonists.

When evaluating mineral interactions and their effect on copper, it is essential to consider not only the feed, but also minerals in the water. For example, Smart et al., (163) found that reducing the sulfate content of drinking water from 500 to 42 mg per liter increased copper availability. This effect is independent of molybdenum, and probably results from the formation of insoluble copper sulfide. Ivan (164) proposed that rumen protozoa were important to this reaction. It appears that the protozoa degrade sulfur amino acids to sulfide which then reacts with the copper to form an insoluble complex.

The milk of certain species such as cattle, sheep, goats, pigs, dogs and rats is quite low in copper. In dairy cows, the level of copper is generally below 0.1 ppm, although it may go as high as 0.2 ppm after calving. But, if the dam's intake of copper is adequate, the newborn will have a substantial storage of copper in the liver. The liver is the main storage organ for copper. With most species, liver copper levels are higher in newborns than in adults. Copper in the milk cannot be increased beyond the normal range by adding extra copper to diets already adequate in copper.

Table 24 gives information on the copper requirements and levels that are apt to be toxic.

Table 24. **Copper Requirement and Toxic Levels¹**

Class of Animal	Copper Requirement in Total Diet (ppm)	Toxic Level in Total Diet (ppm)
Swine	5-6	>250 ³
Poultry	6-8	250-800
Horses	9	Not known
Dairy Cattle	12-16	40
Beef Cattle	10 (4-15)	100 ³
Sheep	7-11	25 ³
Goats	10 ⁴	8-25 ⁴
Other Animals	0.4-7.3	Not known

¹ Recommended by NRC publications, unless indicated otherwise.

² May produce a toxic effect if the diet is low in iron and zinc.

³ Maximum tolerable level in diet.

⁴ Since information is not available with goats, the levels recommended for sheep are suggested.

Ruminants are most sensitive to copper toxicity, while most non-ruminant animals have a relatively high tolerance for copper, especially if the diet is adequate in zinc and iron.

Swine

A deficiency of copper leads to poor iron mobilization, abnormal hematopoiesis, keratinization and decreased synthesis of elastin, myelin and collagen. Leg weakness, various types and degrees of leg crookedness, and the incoordination of muscular action also result. A subclinical deficiency causes reduced blood serum copper and ceroplasmin, a microcytic, hypochromic anemia, aortic rupture and cardiac hypertrophy (87, 92). Recent research suggests that copper and other trace mineral may be critical during early embryonic development. Copper concentrations in the developing pig embryo are much higher than in the uterine endometrium or ovary from day 12 to 30 of gestation (360). These data are interpreted to suggest that the developing conceptus has an increased demand for copper such that the maternal system shunts copper to the developing embryo.

Some producers are using copper at levels of 125 to 250 ppm in the diet as an antimicrobial compound. No toxic effects have been reported when levels of 125 to 150 ppm copper are used in the diet. However, occasional toxic effects have been reported when 250 ppm copper is used. The harmful effects are probably due to a lack of zinc and iron. The addition of 130 ppm zinc and 150 ppm iron has prevented harmful effects when using 250 ppm copper in the diet. With the baby pig, however, a higher level of iron, 170 ppm, is needed to counteract 250 ppm copper in the diet (92). Diets high in calcium may reduce zinc availability, which in turn could reduce the level at which copper is toxic. The use of high levels of copper in the diet will decrease by about 20% the rate of microbial decomposition of pig manure in the lagoon (105). If the manure is to be used as fertilizer on the land, this would be an advantage.

The use of 250 ppm copper in the diet increases the liver level of copper to a much greater extent than 125 ppm in the diet. The liver copper level can be decreased, however, by restricting the use of 250 ppm for pigs up to 100 to 125 pounds or by using a sulfide in the diet (106). Based on present knowledge, the safest procedure would be to use copper at a level no higher than 125 to 150 ppm in the diet, since it gives about the same effect as 250 ppm (92).

The source of copper is critical to achieving the anti-microbial effect in swine. Recent research comparing copper sulfate with copper oxide showed no effects by feeding up to 500 ppm copper from copper oxide while the 125 to 550 ppm copper from copper sulfate was efficacious (166).

Feeding high levels of copper may have non-nutritional advantages in that it improved the odor characteristics of swine waste (270). The mechanism of action is believed to result from the antibiotic-like functions of copper in the intestinal tract of pigs. Lower concentrations of cupric citrate seem to be as effective as 225 ppm of cupric sulfate. The replacement of all sulfate trace minerals with non-sulfate sources may further improve the odor characteristics.

Poultry

A copper deficiency causes a microcytic, hypochromic anemia. Bone weakness, deformities and depigmentation in New Hampshire chicks are also observed. Dissecting aneurysm of the aorta

occurs in copper deficient chicks. A marked cardiac hypertrophy occurs in turkey poults. A copper deficiency in the laying hen results in anemia and the production of eggs that are abnormal in size and shape, and some eggs have wrinkled and rough shells (95, 141).

The requirement for copper set by the National Research Council (Table 13) is 6-8 ppm (141). There are some poultry scientists, however, who feel that a higher level is needed and prefer a level of 10 ppm in the diet (95). Duck diets should contain 6-8 ppm of copper (300).

Horses

Very little information is available for copper needs of horses. A recent Cornell study indicates the copper requirement for the maintenance of mature ponies is about 3.5 ppm in the diet (107). A Minnesota study indicated that ponies were very resistant to chronic copper toxicity and could tolerate as high as 791 ppm in the diet (108). One horse study reported an apparent relationship between low blood serum copper levels and hemorrhaging in aged parturient mares. Another report indicated a copper deficiency caused bone abnormalities in the foal in Australia (154).

Copper is also required in the young horse to prevent Developmental Orthopedic Disease (DOD). Because this disease causes serious problems in the growing horse, most creep feeds are supplemented with 50 ppm copper and most weanling feed with 25 ppm (298).

Source of copper can have a huge impact on bioavailability in horses. Lawrence (2005) reviewed the literature describing apparent and true digestibility of copper sources in different horse diets. Apparent digestibility of copper varied from 9 to 48% and estimated true digestibility varied from 24 to 59%, respectfully (303). Copper concentrations in these diets varied from 7 to 52 ppm.

Dairy Cattle

A deficiency of copper can result in reduced growth or weight loss, unthriftiness, decreased milk production, diarrhea, changes in hair coat color and texture, bone weakness and fractures, "pacing gait" in older cattle, reduced reproduction, difficult calving, retained placenta, birth of calves with congenital rickets and "falling disease" which is sudden death due to acute heart failure and anemia. Sometimes the black hair around the eye loses pigment and develops a gray-spectacled appearance (91, 156).

Copper nutrition can have a great impact on fertility in dairy cows. Suboptimal ovarian activity, delayed or depressed estrus, reduced conception rate and calving difficulty have been associated with copper deficiencies in dairy cattle (165).

Copper absorption is low in ruminants, usually only 1-10% of that reported for non-ruminants (337). More copper is required by cattle on pasture than when they are fed dry forage or concentrates. Copper in silage appears to be of intermediate availability compared to other forage sources.

Breed of dairy cow can have a dramatic effect on copper metabolism. Du (239) reported that Jerseys accumulated copper in their livers faster than Holsteins when given the same copper-rich

diet ad libitum. Several reports from the field have suggested that Jersey and Guernsey cows are more susceptible to copper toxicity than Holsteins.

Beef Cattle

Copper deficiency symptoms include depraved appetite, stunted growth, suppressed immune system, rough hair coat, anemia, diarrhea, straight pasterns, depigmentation of the hair, and sudden death (falling disease). Reduced growth in cattle seems to be most dramatic when excess molybdenum causes the copper deficiency (240). In these cases liver and plasma copper concentrations may not reflect a copper deficiency. This has caused some to hypothesize that the excess molybdenum causes a “localized copper deficiency” within the body. Genglebach’s (241) research supports this concept in that neutrophils from heifers supplemented with molybdenum had lower copper zinc-superoxide dismutase activities than heifers that were made equally copper deficient using excess iron supplementation. Stress also affects how the cattle’s immune system responds to copper supplementation. In one study copper supplementation decreased antibody titers in unstressed calves, but increased the titers in stressed calves (265). Although copper is critical to a healthy immune system, there are other physiological barriers that can override copper status.

Copper requirement of beef cattle is 4 to 10 ppm in the total diet when the diet contains low levels of molybdenum and sulfate (157). In locations where diets contain high levels of molybdenum and sulfate, the level of copper required may increase threefold or more. Copper deficiency may occur when forages have a copper level below 5 ppm and the molybdenum level is above 3 to 5 ppm. In an Irish study 19% of 2612 cattle tested were deficient in copper despite wide spread copper supplementation (295).

Forages are more deficient in copper and higher in antagonist, such as sulfur, than commonly recognized (293). County extension agents collected 1021 forage samples from across the state of Tennessee. Copper was deficient or marginally deficient in 92.4% of the forage samples. Sulfur was considered at least marginally antagonistic to copper in 89.3% of the samples.

Breed also has a role in susceptibility to copper deficiency. Ward (242) reported that Simmental and Charolais cattle had a greater copper requirement than Angus. Differences in biliary secretion may explain the differences among breeds (243). Regardless of dietary treatment, biliary copper excretion was twice as high in Simmental compared to Angus cattle.

Several studies by Engle and Spears have shown the role of copper supplementation on carcass composition (266, 267, and 268, 269). Copper supplementation (10 mg/kg diet) has altered lipid metabolism and tended to increase plasma norepinephrine concentrations in feedlot cattle. This reduced back fat will improve the red meat yield per carcass and result in less waste. The increased polyunsaturated fatty acid concentration in muscle of copper-supplemented steers may result in a healthier product for human consumption.

Copper deficiency may occur in calves that are fed all milk diets for long periods of time or in cattle subsisting on forages produced in copper deficient soils or in soils that contain excess molybdenum (85, 157).

Curing or drying of forages may alter the chemical form of copper, making it more available than copper in fresh green plants (157). Plants that contain high levels of molybdenum, sulfur, phytate or lignin may reduce copper bioavailability (337).

Sheep

Copper deficiency in suckling lambs results in a lack of muscular coordination, partial paralysis of the hindquarters, a swayback condition, degeneration of the myelin sheath of nerve fibers, and weak lambs at birth that may die because of their inability to nurse. Researchers have determined that the swayback condition results from vacuolation, chromatolysis, and necrosis of the large motor neurons in the brainstem nuclei and ventral horn of the spinal cord (305). Anemia, bone disorders and a lack of fertility also occur with a copper deficiency. Sheep produce "steely" or "stringy" wool, which is lacking in crimp, tensile strength, affinity for dyes, and elasticity. Black sheep show depigmentation of the wool (155).

Excess molybdenum in sheep diets causes a copper deficiency. Sheep start to scour a few days after grazing in a pasture with 5 to 20 ppm molybdenum. When the dietary copper level falls below normal, or if the sulfate level is high, molybdenum intakes as low as 1 or 2 ppm may prove toxic. Molybdenum toxicity is usually controlled by increasing the copper level in the diet by 5 ppm. But it is difficult to give the exact copper level needed because of the complexity of the copper-molybdenum-sulfur interrelationship and all the factors affecting it. Merino sheep may need 1 or 2 ppm more copper in the diet than other sheep breeds, probably due to their high level of wool production (93, 94, 96). The dietary amounts of copper that are adequate for some breeds of sheep are deficient for others, and possibly toxic to some (155).

Sheep are very intolerant of excess copper and toxicities have occurred in sheep with concentrations as low as 10 ppm (215). Growing swine are often fed copper concentrations as high as 250 ppm in the diet to improve performance. Cattle can consume diets containing 100 ppm copper without problem.

Copper toxicity in sheep usually results from the accumulation of copper in the liver over a period of a few weeks to more than a year with no clinical signs followed by a sudden release of liver copper stores to cause toxicity. In these situations, chronic copper poisoning may result from excessive copper intake or from low intakes of molybdenum, sulfur, zinc, or calcium or following liver damage (167). Sheep accumulate copper in the liver more readily than other farm animals, and over a period of time, 1,000 to 3,000 ppm on a dry weight basis may be achieved. During the accumulation phase, blood copper levels are normally in the range of 0.10 to 0.20 mg per deciliter. Toxicity results when stress conditions cause the liver cells to die and release the stored copper into the blood. Plasma copper levels then increase 10 to 20 fold. These elevated blood copper levels (500 to 2,000 mg/dl) usually precede clinical signs by 24 to 48 hours (167). The most common symptoms are anorexia, excessive thirst, and depression. These are accompanied by severe hemoglobinemia, anemia, icterus and methemoglobinemia. Most sheep will die within 1 to 2 days of the onset of these signs (168).

The ratio of copper to molybdenum is the most important dietary factor affecting copper toxicity in sheep. Ratios of 10:1 or less will prevent toxicity in most cases. The exact mechanism by which

molybdenum prevents copper toxicity is poorly understood. However, it is known that an insoluble complex, CuMoO_4 , can be formed in the gastrointestinal tract, thus reducing copper absorption. This theory is substantiated by the fact that increasing dietary copper is an effective treatment for molybdenum toxicity.

Molybdenum concentrations in most feeds are in the range of 1 to 3 ppm. If molybdenum concentrations are less than 1 ppm, diets containing copper in the normal requirement range of 8 to 11 ppm have been known to produce toxicity (155). Sheep producers who live in or buy feed from molybdenum deficient areas should pay close attention to dietary copper levels. Such feeds as distillers dried grains and soybean meal, which are normally high in copper, should be limited in the diet. Importantly trace mineralized salt should be retained in the diet because it contains zinc which also reduces copper absorption. Diets containing high concentrations (100 ppm) of zinc have been shown to reduce liver copper stores. Eliminating trace mineral supplementation may actually worsen the situation by creating an even greater mineral imbalance.

Although prevention is much preferred, there are times when mass treatment is indicated. The most common treatment is to give a drench daily containing 50 to 100 mg of ammonium molybdate and 0.5 to 1.0g of sodium sulfate per animal for three weeks. To reduce labor, an aqueous solution of the two salts can be sprayed onto the feed. The Food and Drug Administration does not recognize molybdenum as safe for therapeutic purposes, so it can not be added to sheep diets as a preventative measure. Consequently, producers should consult a veterinarian with expertise in treating copper toxicity in these cases.

Besides nutrition, animal management factors can affect the incidence of copper toxicity in sheep. For example, although this disease can occur in both sexes of all breeds, mature ewes of British breeds seem to be the most susceptible. In the United States this disease is most common in the western states of the intermountain region. Although the disease can occur any time, peak incidence usually is in the fall and winter.

Environmental factors and stress can also affect the susceptibility of sheep to this disease. For example, grazing sheep in areas containing certain potentially toxic plants may predispose them to copper toxicity. Plants such as lupines, which contain toxic alkaloids, produce copper toxicity by impairing the liver's ability to metabolize ingested copper. Chronic toxicity is also common in sheep grazing subterranean clover and is associated with normal levels of copper, low levels of molybdenum, and no apparent liver damage. The stress associated with shipping ewes from mountain ranges to pastures some distance away appears to make ewes more susceptible. Caution should also be exercised when feeding by product feeds known to contain high copper concentrations. For example, broiler litter which can be high in copper should not be fed to sheep (146).

Sheep producers should become familiar with copper and molybdenum status of feeds grown in their area. If the area is deficient in molybdenum or high in copper, feed samples should be analyzed routinely to monitor the copper:molybdenum ratio in the diet. Supplemental feeds which are known to be low in copper should be used whenever possible.

Goats

Goats appear to be more similar to cattle than sheep in their copper metabolism. For example, in research cited by Haenlein (335), the average copper concentration in the livers of goats was 10 ppm, while in sheep it averaged 196 ppm (fresh weight). These data are derived from animals from many sources on different diets, but it does suggest that goats do not store copper in their livers. A report by the European Commission entitled, "Opinion of the Scientific Committee for Animal Nutrition on the use of Copper in Feedingstuffs," showed similar result. Sheep fed 7 ppm copper had 300 ppm (dry matter basis) copper concentrations in their livers, while goats fed similar copper levels had about one-third the copper at 100 ppm. Consequently, copper deficiency is much more common in goats than copper toxicity.

The potential signs of copper deficiency include diarrhea, poor weight gain, light hair coats, anemia and unthrifty appearance. California veterinary pathologist (336) reports the most common copper deficiency in goats they receive is weak kids, one to two months of age that are uncoordinated. The deficiency originates with the doe that is unable to transfer enough copper to the developing fetus. Copper deficiency in goats is usually determined by measuring the concentration in blood. Goats normally have 0.8 to 1.2 ppm copper in the serum. The exact copper requirement for goats has not been determined, but many producers with experience believe that it is 10-20 ppm. This assumes normal levels of molybdenum, iron, and sulfur, which can reduce copper absorption.

When sheep and goats are fed together, it is not uncommon to feed a low-copper mineral supplement designed for sheep. This increases the risk for developing copper deficiencies in kids born to does fed basal diets that are low in copper. Solaiman et al., (2005) fed Boer X Spanish goat kids a diet contain 14.5 ppm copper and then added 100 or 200 mg per head daily additional copper from copper sulfate (304). These data indicate that the 100 mg copper treatment improved gains, feed intake, and feed efficiency without adversely affecting health and well being of the goats. Practically, it is very unlikely that the same diet can be fed to sheep and goats without risking a copper toxicity in the sheep or a copper deficiency in the goats.

In summary, sheep and goats are dramatically different in their copper requirements. Based on these data it appears that copper requirements of goats must be re-evaluated and the NRC requirement adjusted accordingly (100).

Other Animals

With a few exceptions, studies on copper needs of other animals are quite limited. Based on the latest National Research Council publication on nutrient requirements of the other animals, the following are given as recommended levels or suggested requirements copper [(ppm)]: dog 2.9, cat 5, rat 5, mouse 4.5, rabbit 3, guinea pig 6, hamster 1.6, and gerbil 0.4 to 4.5 ppm in the total diet. When deficiency symptoms are given, they show some similarity to those of large animals. In all cases, copper is needed for the utilization of iron for the prevention of anemia. The range of copper needed is not much different from that for large animals (89, 102, 103, 142).

EXCESS MOLYBDENUM

Excess molybdenum causes a copper deficiency and results in extreme diarrhea, weight loss, decreased productivity, graying of hair in cattle, pigment formation, bone and joint disorders, problems with reproduction and heart function, loss of crimp in the wool, anemia, and many other problems with ruminants. Molybdenum is an essential nutrient because it is a constituent of the enzyme xanthine oxidase and other enzymes.

Molybdenum has been shown to be essential for lambs, chicks, and turkey poult fed highly purified diets. Molybdenum does not need to be added to practical diets yet, although that may be a possibility sometime in the future. In a few small, isolated areas that are very low in molybdenum, supplementary molybdenum in the diet may prevent copper toxicity in sheep. At this time, however, the Food and Drug Administration does not recognize molybdenum use as safe, and current regulations prohibit adding it to feed for sheep unless prescribed by a veterinarian for therapeutic purposes (155). Molybdenum is well known for its toxicity in areas of known excess molybdenum that occur in certain locations in the United States (California, Nevada, Oregon, Hawaii, Utah, Montana, Colorado and Florida) and in other countries of the world (England, New Zealand, Canada, Ireland, Argentina, Peru, Cuba and others) (128, 147).

There is considerable difference among animals in the tolerance of high levels of molybdenum. Cattle have the least tolerance, followed by sheep. Horses have failed to show any signs of toxicity in pastures that severely affect cattle. One study, however, showed that the presence of 5 to 25 ppm molybdenum in forages caused some disturbance in copper utilization in horses (98). Chick growth is inhibited at 200 ppm and turkey poult growth is depressed at 300 ppm molybdenum in the diet. Swine are the most tolerant to high levels of molybdenum. Pigs have been fed diets containing 1,000 ppm molybdenum for three months with no harmful effects (92).

Tolerance of non-ruminant animals to molybdenum is much higher than that of ruminant species. Because of this high tolerance, little research has been conducted to determine whether excess molybdenum might cause problems with non-ruminants under certain conditions. Tolerance to high levels of molybdenum varies with the age of the animal, quantity and form of ingested molybdenum, inorganic sulfate content in the diet and copper status. Molybdenum tolerance also varies with intake of methionine, cystine, and protein, which are capable of being oxidized to sulfate in the body. The level of sulfate in the diet can alter the absorption, retention, and excretion of absorbed molybdenum. The effect of excess molybdenum is a deficiency of copper, although there are many interrelationships involved that make it a complex matter. As discussed previously, the copper-molybdenum-sulfate interrelationship is very important, but other nutrients also can have a modifying influence on this interrelationship.

Under normal conditions, the liver contains 2 to 4 ppm molybdenum. This level can increase to 25 to 30 ppm with excess molybdenum intake. The level of molybdenum in the liver returns to normal when excess molybdenum intake ceases. If the excess molybdenum intake is prolonged, a depletion of tissue copper levels occurs and a copper deficiency results. High sulfate intake helps counteract the effect of high molybdenum levels in forage. The degree of molybdenum absorption and retention in the body tissues is decreased by the sulfates. This means that if the copper intake of the diet is about normal, sulfates can counteract a slight excess of molybdenum.

Molybdenum from dried forage may be less available than from green forage, because forages that interfere with copper metabolism when grazed do not interfere when fed as dry forage (157). The normal level of molybdenum in forages is 3 to 5 ppm. In areas of excess molybdenum, forages may contain from 20 to 100 ppm. Copper deficiency may occur when forages have copper levels below 5 ppm, and molybdenum levels above 3 to 5 ppm. Excess molybdenum levels in forages can usually be counteracted by increasing the level of copper in the total diet to two or three times normal. Sometimes even higher copper levels are needed, depending on the many interrelationships of copper-molybdenum-sulfates and other nutrients. To further complicate the matter, it is known that alkaline soils increase molybdenum availability but decrease copper availability to plant life. Therefore, alkaline soils increase the severity of the problem in excess molybdenum areas. Soil wetness in poorly drained soils may also increase the molybdenum level in the forages produced there (85, 91, 92, 93, 94, 96, 98).

In most excess molybdenum areas, a mineral mixture with two to three times the normal copper level is fed. In a few areas, a copper compound is injected. Livestock owners should follow the recommendations of a nutritionist or a veterinarian familiar with the excess molybdenum area in which they live. The recommendations made in each area may differ somewhat to meet the specific local excess molybdenum problem.

IODINE FOR ANIMALS

Around 1900, scientists first recognized that iodine was required for the proper functioning of the thyroid gland and that an iodine deficiency caused goiter. Shortly thereafter, iodized salt became widely accepted as a means of preventing goiter in man and animals.

In humans goiter and cretinism are no longer considered the main consequence of iodine deficiency, although these are the easiest signs of an iodine deficiency to detect. Recent research has shown that for every cretin living in an iodine deficient community, two pregnancies have ended in miscarriage, stillbirth or neonatal death (245). The survivors in an iodine-deficient population suffer from a higher incidence of developmental and functional abnormalities, including speech, hearing and neuro-muscular function. These symptoms are also expressed in a 10-15 point decrease in average I.Q. The term "Iodine Deficiency Disorders or IDD" has been coined to describe this condition.

The thyroid gland contains the highest concentration (0.2% to 5% on a dry weight basis) of iodine in the body; between 70% and 80% of the total body stores. Approximately 90% of the iodine which passes through the thyroid gland is captured by that organ (244). Iodine is then combined with tyrosine in the thyroid to form diiodotyrosine. Two molecules of this compound are then combined to form thyroxine. Approximately 80% of the thyroxine entering the circulation is broken down through de-iodinization by the liver, kidney and other tissues.

De-iodinization by these organs may appear counter productive at first glance, but recent research has elucidated its beneficial effect and the role selenium plays in the process. The first clue that selenium deficiency influenced iodine metabolism from selenium depleted rats where there was an increase in the thyroxine to triiodothyronine in the blood. Research by Arthur (246) and Donald (247) confirmed that the same changes occurred in cattle and sheep, respectively. A seleno-protein has been identified that is capable of transforming thyroxin to the more biologically active triiodothyronine. This deiodinase is located primarily in the liver and kidney with none in the thyroid gland itself (219). A second selenium containing deiodinase is located in the brain and brown adipose tissue. This is especially important in the newborn where thermogenesis is directly related to the activity of the brown adipose tissue. Consequently, iodine metabolism is greatly influenced by selenium nutrition, and thus indirectly influences basal metabolic rate and a host of physiological processes. Free iodine is conserved and recycled by the body with only 20% being excreted in the urine and feces (215).

When inorganic iodine is consumed, it is absorbed from the gastro-intestinal tract by two processes. One is common to other halides such as chlorine or bromine, and the other is specific for iodine. The stomach and duodenum not only absorb iodine, but also secrete it in gastric juice. In fact, gastric juice often has an iodine concentration 40 times higher than blood plasma. Iodine absorbed from the blood by the thyroid gland is stored as thyroglobulin. Free iodine is conserved and recycled by the body with only 20% being excreted in the urine and feces (215).

Several dietary factors are goitrogenic. In fact, the dietary concentration of goitrogens is probably a more important determinant of iodine status than dietary iodine levels, in many cases. Cruciferous plants contain potential goitrogens of the thiouracil type, while brassicas and white clover contain

cyanogenetic glycosides that are goitrogenic (219). Canola meal has resulted from the selection of rape-seed that is low in glucosinolate, a common goitrogen. There are also specific goitrogenic substances in other feeds (carrots, linseed, cassava, sweet potatoes, lima beans, millets, peanuts, cottonseed, soybeans and others) which slow down the hormone secreting activity of the thyroid gland. Goiter can occur, therefore, even though the iodine level might otherwise be adequate. Other substances can also reduce iodine absorption. For example, a high calcium level in drinking water may slow iodine absorption resulting in goiter, particularly if the iodine level is borderline in meeting body needs.

Crops grown inland, away from the ocean, are usually low in iodine, whereas crops grown near the ocean may be adequate in iodine. For example, it is estimated that 20-50 mg of iodine per acre fall annually in rain along the Atlantic coastal plain, but on 0.7 mg iodine per acre falls in rain in the Great Lakes region of the U.S. The Midwest and Great Lakes region have been the areas where iodine deficiency is most common. Supplemental iodine provided by iodized salt is recommended for use in all areas of the country, because of the uncertainty of where feeds originate.

Some iodine compounds are easily broken down by heat and moisture. Therefore, a stable source of iodine must be used to prevent losses from exposure to sunlight, moisture, or other conditions. Table 25 shows the level of iodine recommended by the latest National Academy of Sciences-National Research Council publications on the nutrient requirements of the species involved. The toxic levels in Table 25 are those indicated in the same NRC publications. Feeding excess iodine should be avoided because it may interfere with thyroid gland function.

Table 25. Iodine Requirement and Toxic Levels¹

Class of Animal	Iodine Requirement in Total Diet (ppm)	Toxic Level in Total Diet (ppm)
Swine	0.14	800
Beef Cattle	0.5 (0.2-2.0)	50 ²
Dairy Cattle	0.25-0.50	50
Horses	0.1	4.8
Sheep	0.1-0.8	50 ²
Goats	0.15-0.8 ³	8 ³
Poultry	0.3-0.4	625

¹ Levels recommended by the latest NRC publication on nutrient requirements, unless indicated otherwise.

² Maximum tolerable level in diet.

³ Same level as recommended for sheep.

Swine

Symptoms of iodine deficiency in piglets born to iodine deficient sows are thickened skin, puffy necks, hairlessness, and bloated appearance. Some of the piglets are born dead, others are alive but weak and usually die within a few hours. At necropsy, the thyroid is enlarged and hemorrhagic. Iodine deficiency can cause an approximate six-fold enlargement of the thyroid gland (87, 92).

Beef Cattle

Goiter in newborn calves is a sign of borderline or definite dietary iodine deficiency, even though the cows may appear normal. Thyroid glands four to five times normal size sometimes occur in calves, but the abnormality cannot be detected definitely until the calf is slaughtered. Iodine deficient calves may also be born blind, hairless, weak or dead, depending on how severe the deficiency is. Long-term deficiencies may result in decreased milk yields, and decreased fertility in females and males. Iodine deficiency may occur in cattle even at iodine intake levels recommended in Table 12. If as much as one fourth of the feed used is from strongly goitrogenic crops, iodine requirements increase to 0.5 ppm for growing and non-lactating cattle and 1.0 ppm for late gestation and lactating cows (85, 157). In a recent Irish study, iodine deficiency was the most the most prevalent deficiency observed. Plasma iodine concentrations were low in 69% of the 2,612 cattle tested (295).

Dairy Cattle

Lactating dairy cattle require more iodine than beef cattle because approximately 10% of the iodine intake is normally excreted in milk, and this percentage may increase as milk production increases (156). To ensure that the iodine requirements of the high-producing cow are met, 0.6 ppm iodine is recommended.

Horses

Pregnant mares may not show external signs of iodine deficiency but may produce a stillborn foal or one showing extreme weakness at birth, resulting in an inability to stand and suckle the mare. Foals born alive with a well developed goiter will usually die or will remain weaklings if they live. Deficient mares may exhibit abnormal estrus cycles in the absence of goiter. Limited data indicate that "navel ill" in foals may be lessened by feeding iodine to brood mares, but more verification is needed. A decline in libido (sex drive) and semen quality has been noted in Europe with stallions, but this also needs more verification (98, 154).

There is a wide margin between iodine requirement and the maximum tolerable level for all farm animal species except the horse (248). The maximum tolerable level for equines has been estimated as low as 5 ppm. With normal feedstuffs iodine toxicity is extremely rare. The only feed ingredient that could pose a toxicity risk is seaweed. Seaweed can contain 4,000-6,000 ppm of iodine.

Sheep

In newborn lambs, the more common symptom of iodine deficiency is goiter. If the condition is not advanced, the lambs may survive. Other symptoms are lambs born weak, dead, or without wool. Mature sheep seldom show a change in their appearance. But an iodine deficiency may result in reduced yield of wool and reduced conception rate (155).

Goats

Kids with goiter, as well as weak and dead kids, have been reported at birth. Very little experimental information is available with the goat. Therefore, extrapolated data from sheep studies must be used until additional experimental data become available from studies with goats (100).

Poultry

Goiter occurs in the thyroid gland, causing the thyroid gland to grow to many times its normal size. Histological examination of the thyroid shows hyperplasia and an absence of colloid. Iodine deficiency in breeding hens results in reduced egg iodine levels, reduced egg production, decreased hatchability, prolonged hatching time, and thyroid enlargement in the embryos (95, 141).

Other Animals

"Other animals" include dogs, cats, mink, foxes, rabbits, fish and laboratory animals. In all of these animals and fish, an iodine deficiency results in an enlarged thyroid. With many animals, a level of 0.1 to 0.3 ppm iodine in the diet will meet the iodine requirement. With others, a level as high as 1 ppm is required. Iodine in excessive levels should be avoided, because it may have harmful effects (89, 102, 103, 142, 143, 144, 145).

COBALT FOR ANIMALS

The only known animal requirement for cobalt is as a constituent of Vitamin B₁₂, which has 4% cobalt in its chemical structure. This means that a cobalt deficiency is really a vitamin B₁₂ deficiency.

Microorganisms in the rumen are able to synthesize vitamin B₁₂ needs of ruminants if the diet is adequate in cobalt. Normally, cobalt is not stored in the body in significant quantities. The small amount that is stored does not easily pass back into the rumen or intestinal tract where it can be used for vitamin B₁₂ syntheses. Therefore, ruminants must consume cobalt frequently in the diet for adequate B₁₂ synthesis. Injected cobalt is ineffective.

The fact that injected cobalt is ineffective agrees with recent research which suggests that cobalt deficiency in the rumen may be more important than a vitamin B₁₂ deficiency at the tissue level. Traditionally, a breakdown in propionate metabolism at the point in the metabolic pathway where methylmalonyl-CoA is converted to succinyl-CoA, was thought to be the reason for the depression in appetite. However, Kennedy (249) showed that there were massive increases in succinate concentrations in the rumen within two weeks of when sheep were fed a cobalt deficient diet. When sheep were fed a diet with only 0.02 ppm cobalt, succinate accumulation in the rumen began in two days (250). It has been well documented that changes in the rumen microbial population occurs in cobalt deficient ruminants. It now appears that a cobalt deficiency causes a vitamin B₁₂ deficiency which inhibits propionate producing bacteria such as *Selenomonas ruminantium*.

Cobalt was first shown to be of value to ruminants in 1935. Prior to that time, ruminants could not be successfully produced in many areas of the world because of severe cobalt deficiencies. In these locations, including Florida, cattle were limited to certain areas that were known as "healthy areas" (122). Animals in cobalt deficient areas, known as "sick areas" would respond when transferred to "healthy areas." In certain "sick areas" the lack of cobalt could be alleviated by periodic removal of the animals to "healthy areas" for varying periods. The discovery of cobalt as the cause of the problems in "sick areas" was of tremendous value in increasing ruminant productivity throughout the world. Even today, new cobalt deficient areas are being found as livestock production increases in newly developed areas, and as research efforts are increased to determine mineral needs of animals (93, 94, 96, 125, 126, 128, 135). University of Florida scientists reported in 1976 that 43% of the 140 forage samples taken throughout Latin America had cobalt levels of 0.1 ppm or less (86). Cobalt deficiency is most common in high rainfall areas where the soil is derived from acid igneous rocks such as granite and subject to leaching. Heavy liming of pastures has been associated with increased risk of cobalt deficiency (299).

With current knowledge, the most convincing evidence of a cobalt deficiency is determined by the response of the animal to cobalt feeding. The response is quick, with appetite increasing in about a week, and weight gains quickly follow. The remission of the anemia, however, occurs more slowly.

Table 26 provides information that indicates the cobalt status in ruminants as determined by vitamin B₁₂ levels in the liver.

Table 26. Liver B₁₂ as Cobalt Status Indicator¹

B₁₂ in Fresh Liver (ppm)	Cobalt Status of Animal
Less than 0.07	Severe cobalt deficiency
0.07-0.11	Moderate cobalt deficiency
0.11-0.19	Mild cobalt deficiency
0.19 or more	Sufficiency

¹ W.J. Miller, University of Georgia (91)

Table 27 gives data on the requirements for cobalt by the various classes of animals. It also gives the maximum tolerable level of cobalt as established by the National Research Council. Cobalt has a relatively low order of toxicity in all animals. Cobalt toxicity in ruminants is rare because toxic levels are about 300 times requirement levels (157). Excesses should be avoided, however.

Table 27. Cobalt Requirement and Toxic Levels¹

Class of Animal	Cobalt Requirement in Total Diet (ppm)	Toxic Level in Total Diet (ppm)
Dairy Cattle	0.1	10
Beef Cattle	0.1 (0.07-0.11)	10
Sheep	0.1-0.2	10 ²
Goats	0.1-0.2	10 ³

¹ Levels recommended based on the latest NRC publication on nutrient requirements of each animal.

² Maximum tolerable level in diet.

³ This level is not known so the level used for sheep is presented.

Cobalt protects sheep and to a lesser extent cattle, from "Phalaris Staggers," or "Ronpha Staggers," which sometimes occur with the perennial grass *Phalaris tuberosa* or one of several other related grass species. The toxic principal may be an alkaloid. The detoxification role of cobalt is not related to its role in vitamin B₁₂.

Dairy Cattle

Cobalt deficiency usually occurs after animals have been on a cobalt deficient diet for a considerable time and as vitamin B₁₂ stores in the liver and other tissues are depleted. Cobalt deficiency results in decreased appetite and feed consumption which leads to listlessness, retarded growth, weight loss, and decreased milk production. With an extreme cobalt deficiency, symptoms may include emaciation or wasting of the musculature, paleness of the skin and mucous membranes, muscular incoordination, a stumbling gait, rough hair coat, and high mortality rate among calves. Starvation associated with cobalt deficiency is caused, at least partially, by the animal's inability to metabolize propionate. This inability results from a lack of vitamin B₁₂, which is needed for the body to utilize propionate. Propionate is a volatile fatty acid, a product of rumen fermentation and an important source of energy for the animal (156).

Generally, 10 ppm cobalt in the diet is accepted as the maximum safe level. However, there are studies indicating that 20 to 30 ppm does not produce adverse effects (91, 156).

Beef Cattle

The appearance of a cobalt deficient animal is similar to that of a starved one. An early symptom is loss of appetite, which is alleviated quickly following cobalt supplementation if the deficiency symptoms have not advanced too far and caused body organ damage. The decrease in feed consumption results in emaciation and wasting of the musculature. Paleness of the skin and mucous membranes results from the anemia that develops progressively with the severity of the cobalt deficiency (85, 157). Although infertility can always occur as a secondary consequence of poor body condition in cattle, recent research (251) suggests that cobalt deficiency can cause infertility when cows are in good condition.

Sheep

Cobalt deficiency causes lack of appetite, lack of thrift, severe emaciation, weakness, anemia, decreased fertility, and decreased milk and wool production (155). Weeping eyes, leading to a matting of wool on the face, is another common symptom (299). Sheep are more susceptible to cobalt deficiency than cattle and the accumulation of fat in the liver of B₁₂ deficient sheep, but not cattle, may be related to a methyl-group deficiency affecting liver lipid metabolism (252). Another metabolic anomaly of cobalt deficient lambs is the accumulation of homocysteine in the plasma which leads to an accumulation of oxidation products, depletion of vitamin E, and damage to the mitochondria (249).

Goats

Cobalt deficiency symptoms include a loss of appetite, emaciation, weakness, anemia, and decreased production. The 1981 NRC committee on Nutrient Requirements of Goats assumes that a level of 0.1 ppm cobalt in the diet is adequate for goats since it is adequate for sheep (100).

Non-Ruminants

Cobalt deficiency has not been demonstrated in non-ruminant animals. In a few instances where cobalt has created some response, it is assumed that the diet lacked vitamin B₁₂. It should be indicated, however, that non-ruminants also synthesize a limited amount of vitamin B₁₂ in their digestive tract. How much is absorbed is not known, but vitamin B₁₂ would be available in the feces for animals that practice coprophagy. Horses have thrived on pastures so low in cobalt that cattle and sheep confined to them soon waste and die. The utilization of cobalt by the microbial flora in the rabbit is much more efficient than in ruminants. The absorption of vitamin B₁₂ by rabbits is very efficient (88). There is some evidence that cobalt may have a sparing action on zinc in zinc deficiency in pigs (92).

If the diet of non-ruminants is adequate in vitamin B₁₂, there is no evidence to indicate a need for cobalt. It is possible, however, that if dietary vitamin B₁₂ is limiting, a need for cobalt for intestinal synthesis of B₁₂ will be of some importance with non-ruminant animals. All-plant diets contain little or no vitamin B₁₂. Therefore, non-ruminant animals consuming all-plant diets would need some dietary cobalt in order to enable their microflora to synthesize vitamin B₁₂. This fact causes many producers of non-ruminants to supplement diets with 0.1 ppm cobalt just in case the diet might not supply all the vitamin B₁₂ required (87, 92, 93, 95, 141). Therefore, in practice, trace mineralized salt that provides cobalt, which is needed by ruminants, is also used for non-ruminant animals. This eliminates the need for manufacturing a separate trace mineralized salt for non-ruminants that does not contain cobalt. Moreover, the presence of cobalt provides some insurance in case the non-ruminant diet is lacking sufficient vitamin B₁₂.

MANGANESE FOR ANIMALS

Manganese was first recognized as a necessary nutrient for animals in the early 1930s. Because manganese is found in many different feeds, a deficiency is less likely than with most of the other trace minerals. However, manganese deficiency does occur in sufficient magnitude to justify consideration in this text.

Bone, kidney, liver, pancreas, and pituitary gland are the sites of highest manganese concentration. Relative concentration is quite low compared to the other trace minerals. For example, in humans, total body manganese is approximately 1% of the zinc and 20% of the copper. Although concentrations are low, it is a critical nutrient for several functions.

Manganese is essential for chondroitin sulfate synthesis, which is critical to the organic matrix of bone. Many enzymes required for the synthesis of polysaccharides and glycoproteins require manganese to be active. Manganese is a key component of the metalloenzyme, pyruvate carboxylase, a critical enzyme in carbohydrate metabolism. Lipid metabolism is also dependent on manganese to allow the liver to convert mevalonic acid to squalene (215).

High dietary intake of calcium, phosphorus, and iron reduce manganese absorption. The body has only a limited storage of mobilizable manganese reserves. The absorption of manganese in all domestic livestock is poor (93). Research with humans showed that manganese absorption from normal dietary ingredients ranged from 1.7 to 5.2%. Even manganese chloride was only absorbed at a rate of 8.9% (252).

Poultry

Manganese deficiency in the diet of growing chicks and poults results in perosis, or slipped tendon. Manganese-deficient chicks have less proteoglycan in the cartilage of the tibial growth plate than manganese-repleted chicks (253). This contributes to the perosis condition which involves a twisting and bending of the tibia, and slipping of the gastrocnemius tendon from its condyles. With increasing severity chicks are reluctant to move, squat on their hocks and soon die. In laying and breeding birds, a manganese deficiency results in lowered egg production and hatchability and reduced eggshell strength. In many cases, embryos that die as a result of manganese deficiency exhibit chondrodystrophy, a condition characterized by a parrot-like beak, wiry down and shortening of the long bones (95, 141).

Table 28 gives information on the manganese requirement of poultry and other animals. It varies considerably with the different classes of poultry. Ducks normally require 30-50 ppm (300). Manganese source also influences requirement. Manganese sulfate has the highest bioavailability, while manganese oxide and manganese carbonate are only about 30 and 55% as available as the sulfate (254).

Table 28. Manganese Requirement and Toxic Levels¹

Class of Animal	Manganese Requirement in Total Diet (ppm)	Toxic Level in Total Diet (ppm)
Poultry	25-90	4,800
Dairy Cattle	17-24	1,000
Swine	3-20	4,000
Beef Cattle	40 (20-50)	1,000 ²
Horses	40	--
Sheep	20-40	--
Goats	45-50	--
Other Animals	25-95	--

¹ All levels are those recommended by the latest NRC publications, except as indicated.

² Maximum tolerable level in diet.

Dairy Cattle

Only about 1% of the manganese in ruminant diets is absorbed (337). General symptoms of manganese deficiency include impaired growth, skeletal abnormalities, disturbed or impaired reproduction, and abnormalities of the newborn. Deformed calves at birth have weak legs and pasterns, enlarged joints, stiffness, twisted legs, general weakness, and reduced bone strength. Low-manganese diets cause heifers and cows to be slower in exhibiting estrus and more likely to have "silent heats," and a lower conception rate. Abscessed livers and practically no bile in the gall bladder have also been reported (91, 156).

In cattle the manganese requirement for growth is lower than that required for reproduction and birth of normal calves. Ohio researchers used regression techniques to estimate the intake required to meet the metabolic fecal manganese requirement (313). The corresponding dietary concentrations, assuming dry matter intakes of 21 and 12 kg/day for lactating and dry cows, respectively, were 28 and 49 ppm in the diet on a dry matter basis. These concentrations are approximately 1.6 and 2.7 times higher than those needed to meet the manganese requirements for lactating and dry cows, respectively, as calculated using the 2001 National Research Council dairy requirements model.

Swine

Signs of a manganese deficiency include abnormal skeletal growth with an altered ratio of fat-to-lean body tissue; absence of, or irregular, estrual cycles; poor mammary development and lactation; resorption of fetuses; and at birth, small, weak pigs whose sense of balance is poor. Decreased growth rate and feed efficiency also occur with a manganese deficiency (87, 92).

Data on the requirement for manganese have varied a great deal. This is probably due to the high ash, calcium, phosphorus, and other constituents in some diets which can increase manganese needs.

Beef Cattle

Deficiency symptoms in the cow are characterized by reproductive disorders. These include delayed estrus, reduced fertility, abortions, and deformed young. Calves born to cows deficient in manganese have exhibited poor growth, weak and shortened bones, and deformed legs with enlarged joints, stiffness, twisted legs, and "over-knuckling." In the male, manganese deficiency causes impaired spermatogenesis, testicular and epididymal degeneration, sex hormone inadequacy, and eventual sterility (85, 157).

Information on manganese requirements indicates that 20 ppm is probably adequate for growth, whereas about 40 ppm is needed for satisfactory reproductive performance. High intakes of calcium and phosphorus increase manganese needs (85, 93, 157).

Horses

Manganese requirement and the effect of a deficiency of manganese are not known. The National Research Council's Committee on Nutrient Requirements of the Horse recommends the 40 ppm manganese in the diet (154). Recent studies indicate that 45 ppm manganese are required for mature idle horses (298). This is a good recommendation because manganese is associated with proper bone development and formation, which is very important in all horses. Moreover, manganese can affect reproduction in both the male and female. Severe deficiency can cause resorption of the fetus in utero or death at birth (298). Because the foal crop is often low, it is important that manganese levels be adequate.

Sheep

Depressed or delayed estrus and poor conception rates occur in ewes with a manganese deficiency. More services per conception have also been reported (93). One report indicates that manganese may have a possible role in the functioning of the corpus luteum (109). Early weaned lambs on a purified diet containing less than 1 ppm manganese exhibited bone changes similar to those seen in other manganese deficient animals (155). This indicates that sheep probably need manganese for skeletal development, as various other species do. The levels of manganese in wool appear to be quite sensitive to changes in the manganese status of the lamb (155).

The exact requirements for manganese are not known, but the National Research Council committee on sheep recommends a level of 20 to 40 ppm in the diet (155). High intakes of calcium and iron may increase manganese needs.

Goats

Symptoms of manganese deficiency include reluctance to walk, deformity of the forelegs, and reduced reproductive efficiency (100). The growth of female goats fed 20 ppm manganese for the first year of life and 6 ppm the following year was not affected, but the low manganese level delayed the onset of estrus and required more inseminations per conception (155). In mature goats the manganese content of the hair is a better indicator of manganese status than manganese in any other part of the body (155). Goats also show tarsal joint excrescences, leg deformities, and ataxia due to a manganese deficiency (93). A decrease of 25% in the body fat content of kids born from goats on manganese deficient diets has been reported (93).

Information on manganese requirements of the goat is meager. A level of 45 to 50 ppm is suggested for sheep until more definitive data become available (100).

Other Animals

Unfortunately, there is very little information on manganese requirements of small animals. In some cases, the latest National research Council publications suggest levels of manganese that have been used successfully. They are, in ppm in the total diet: fish, 95; rat, 50; dog, 5; cat, 10; rabbit, 2.5 to 8.5; guinea pig, 40; non-human primate, 40; mouse, 45; gerbil, 3.3 to 45; and hamster, 3.65 ppm. These levels can be used as a guide until more definitive information is obtained. In the few instances where deficiency symptoms were given, there was some similarity to those reported for domestic livestock.

SELENIUM FOR ANIMALS

Selenium has become one of the most widely recognized nutrient deficiencies in the U.S. USDA scientists estimate that beef, dairy and sheep producers lose \$545 million yearly due to selenium deficiency (331). Selenium was recognized as a potentially toxic mineral many years before it was identified as an essential nutrient. It was not until 1957 that the role of selenium in preventing liver necrosis in the rat and exudative diathesis in chicks was recognized. Selenium is present in all cells of the body, but the concentration is normally less than 1 ppm. Toxic concentrations in liver and kidney are normally between 5 and 10 ppm (215).

Selenium is an important part of the enzyme glutathione peroxidase. This enzyme destroys peroxides before they can damage body tissues. Vitamin E is also effective as an antioxidant. Therefore, both selenium and vitamin E prevent peroxide damage to body cells. This aids the body's defense mechanisms against stress. Most feeds contain compounds that can form peroxides. Unsaturated fatty acids are a good example. Rancidity in feeds causes formation of peroxides that destroy nutrients. Vitamin E for example, is easily destroyed by rancidity. Selenium spares vitamin E by its antioxidant effect as a constituent of glutathione peroxidase.

Selenium and vitamin E are interrelated. Both are needed by animals and both have metabolic roles in the body in addition to an antioxidant effect. In some instances, vitamin E will substitute in varying degrees for selenium, or vice versa. However, there are deficiency symptoms that respond only to selenium or vitamin E. Although selenium cannot replace vitamin E in nutrition, it reduces the amount of vitamin E required and delays the onset of E deficiency symptoms.

Selenium plays a critical role in increasing the immune response in animals. For example, Dimitrov et al. (169) reported that pretreatment of polymorphonuclear neutrophils from selenium deficient pigs, with selenium, restored their oxidative metabolism, which is closely related to the ability of the neutrophils to kill microorganisms. Researchers (170) recently showed that adding 0.2 mg selenium and 20 mg vitamin E per kg diet increased antibody titers to parainfluenza3 virus. Titer levels increased more initially due to selenium supplementation, but responses to vitamin E were greater than for selenium to a secondary challenge. These authors concluded their paper by stating, "It may be necessary to reevaluate established recommended intakes for nutrients that may be classified as immuno-stimulatory."

Selenium is important in sulfur amino acid synthesis. Sulfur amino acids protect animals against several diseases associated with low intakes of selenium and vitamin E. This protection is believed to be due to the antioxidant activity of selenium and vitamin E. Therefore, the sulfur amino acids, methionine and cystine, can spare vitamin E and selenium through their antioxidant role.

Selenium can be added to diets of all food animals. Either sodium selenite or sodium selenate can be used. Selenium is added to feed as follows: Up to 0.1 ppm in complete feed for swine, beef cattle, dairy cattle, sheep, poultry, rabbits, dairy goats, etc. Turkeys may be fed 0.2 ppm and small pigs up to 50 pounds, may be fed up to 0.3 ppm. Horses and other non-food animals (game birds, zoo animals, laboratory animals, etc.) can be fed up to 0.1 ppm selenium in the total diet.

In August 25, 1997 Federal Register, FDA published a final rule which adopted, without change, the provisions of an interim rule published in the Federal Register on October 17, 1995 (60 FR 53702) regarding the approved use of selenium as a food additive in animal feeds. The current selenium food additive regulations (Title 21 Part 573.920 in the Code of Federal Regulations) provides for the use of sodium selenate or sodium selenite as sources of selenium for selenium supplementation of complete feeds for chickens, swine, turkeys, sheep, cattle, and ducks at a maximum level of 0.3 ppm; in feed supplements for limit feeding of sheep not to exceed an intake of 0.7 milligrams per head per day and in beef cattle not to exceed an intake of 3 mg per head per day; in salt-mineral mixtures for free-choice feeding for sheep at levels up to 90 ppm at a rate not to exceed an intake of 0.7 mg per head per day and for beef cattle at levels up to 120 ppm at a rate not to exceed an intake of 3 mg per head per day. Updates to this ruling should be available at (311). http://www.fda.gov/cvm/CVM_Updates/selenium.html

Excess selenium in animal diets must be avoided. However, selenium is no more toxic than some of the other trace minerals. Using the pig as an example, the toxic level of selenium is 5 to 10 ppm in the diet, which is 50 to 100 times the current level of 0.1 ppm selenium allowed by the Food and Drug Administration in most swine diets. This margin of safety for selenium is greater than for copper, zinc, iron and possibly manganese for the pig. Therefore, selenium can be used if proper precautions are taken in its addition to animal diets. These same precautions should be taken with the addition of other trace minerals. The National Research Council publications show that the following levels of selenium in the total diet are toxic: swine, 5 to 10 ppm; chickens, 5 to 20 ppm; dairy cattle, 3 to 5 ppm; beef cattle, 8.5 ppm; horses, 5 to 40 ppm; and sheep, 3 ppm. All of these animals require selenium at a level of 0.1 ppm in the total diet (except the turkey, which requires 0.2 ppm, and the baby pig, 0.3 ppm). Therefore, there is a significant safety factor between the level needed in the diet and what is toxic. Generally, higher levels of protein, sulfur and arsenic will partially protect against toxicity of excess selenium. Selenium should not be added to diets in areas where excess selenium occurs. Selenium accumulates in the body, but mild chronic signs can be overcome readily. Selenium is eliminated rapidly from the body of the affected animal when the animal is fed selenium-low forage (155). Small amounts of arsenic acids are effective in reducing the toxicity of selenium (155).

Selenium deficiencies have occurred throughout the United States and in all areas of the world. In the United States, selenium deficient areas have been found in 44 states. Feeds produced in selenium deficient states are shipped to other states and to many foreign countries. Therefore, selenium deficiencies are likely to occur almost anywhere. Countries that import U.S. produced grain and soybeans are likely to encounter selenium deficiencies, even though native soils may contain adequate levels of selenium. Taiwan is a typical example, because its swine industry imports grain and soybean meal from the United States.

Swine

Sudden death is a prominent feature of selenium deficiency. Gross necropsy lesions of a selenium deficiency are identical to those of a vitamin E deficiency (87). They include massive hepatic necrosis, and edema of the spiral colon, lungs, subcutaneous tissues, and submucosa of the stomach. Bilateral paleness and dystrophy of the skeletal muscles (white muscle disease) are often found. Occasionally, mottling and dystrophy of the myocardium (mulberry heart disease) are also

observed. Mulberry heart disease in pigs is most common when cereal-based diets contain less than 0.05 ppm selenium. Recent research by Mahan showed that adding .15 or .30 ppm selenium from sodium selenite from late gestation through day 14 of lactation increased milk selenium content and serum selenium concentration in nursing pigs compared to the unsupplemented controls (271). Organic selenium sources (selenium yeast) did increase milk selenium 2.5 to 3.0 times higher than the sodium selenite. Selenium supplementation of the sow may reduce the incidence of mulberry heart disease in the young pig. The incidence and degree of selenium deficiency may be increased by environmental stress. Dietary arsenicals help to alleviate selenium toxicity (87, 92).

Poultry

The main symptom of selenium deficiency is exudative diathesis. This disease characterized by edema of the breast, wing and neck regions. This edema is caused by an abnormally high permeability of the capillary walls that allows fluid to accumulate between the muscle and skin. In broilers fed low-selenium grains, chicks between 3 and 6 weeks of age, begin to show signs of weight loss, leg weakness and eventual death. With severe selenium deficiency, the growth rate is reduced and mortality increased even in the presence of adequate vitamin E. Pancreatic fibrosis and a reduction in pancreatic output of lipase, trypsinogen, and chymotrypsinogen are associated with selenium deficiency. Pancreatic lesions can occur as early as 6 days of age and usually return to normal within two weeks of selenium supplementation. In laying hens egg hatchability is the most sensitive criteria of selenium deficiency. Selenium is also required to prevent myopathies of the gizzard and heart in turkeys and ducks. Encephalomalacia, membrane lipid peroxidation, erythrocyte hemolysis, and muscular dystrophy are benefited by selenium (95, 110, 141).

Dairy Cattle

A major symptom of selenium deficiency is white muscle disease, which usually occurs in young calves. This causes chalky white striations, degeneration, and necrosis in cardiac and skeletal muscles. Heart failure, paralysis (usually of the hind legs), a dystrophic tongue and elevated SGOT (serum glutamic oxaloacetic transaminase) values may also be evident. Smith (255) reported that selenium injections reduced the duration, but not the incidence of mastitis when dietary selenium was deficient. Other symptoms include unthriftiness, growth depression, diarrhea, retained placenta, and lower reproductive efficiency (which includes increased services per conception) and birth of premature, weak, and dead calves (91, 156). Harrison and Hancock (333) recently reviewed the role of selenium and vitamin E in reproductive diseases in dairy cows.

Part of the reason ruminants are susceptible to selenium deficiency is that absorption is less efficient and more variable than in non-ruminants (256). Most of the selenium that is ingested leaves the rumen attached to the cell membranes of bacterial. Part of these cell membranes passes through the small intestine unabsorbed. The true absorption of selenium from grass hay has ranged from 10 to 16% (257). Organic selenium in selenized yeast increases blood and milk selenium more than an equal amount of selenium from selenite (337).

Beef Cattle

Symptoms of selenium deficiency are white muscle disease, heart failure, and paralysis. Paralysis can range in severity from slight lameness to inability to stand. Hollow or swayed back is typical. A dystrophic tongue is often seen in selenium deficient animals. Cows grazing forages lacking in selenium tend to produce calves with nutritional muscular dystrophy or white muscle disease (85, 157). White muscle disease is a degenerative rather than a dystrophic disease of the striated muscles. Lesions are probably the result of free-radical damage (258). Affected calves have muscle stiffness, arrhythmia, tachycardia and abdominal breathing. A selenium-vitamin E mixture injected into cows one month before calving prevented losses from birth of premature, weak, or dead calves in parts of California (112), and greatly reduced the incidence of retained placenta in cows in Scotland (113). French researchers (274) looked at the effect of selenium supplementation before and after calving on selenium status in deficient cows and their calves. This study demonstrated that adding 13.0 to 45.5 mg of dietary selenium daily to beef cows for 15 days in late pregnancy produced satisfactory selenium status in cows and their calves for up to three months after the end of supplementation. However, a more dependable approach is to prevent the selenium deficiency from developing in the first place by feeding supplemental selenium throughout gestation.

Missouri researchers determined the selenium status of 532 feeder calves representing 178 herds from all nine agricultural districts of the state (331). Deficient status was defined as less than 70 ppb whole blood selenium wet weight. On average, 16.5% of the calves were deficient in selenium. By district the deficiency rate varied from 4.7% to 40.0%. The districts with the highest proportion of deficient calves have higher annual rainfall than the rest of the state.

In most cases salt and inorganic selenium will be adequate, however, in some cases a more available source of selenium may be beneficial. Florida researchers (361) showed that adding selenium-yeast to free-choice salt increased calf plasma selenium concentrations more than giving cows selenium injections. Nursing calves eat little salt and so depend primarily on milk and forage to meet their needs. Arkansas researchers showed that calves born to cows fed selenium-yeast had higher whole blood selenium concentrations and greater glutathione peroxidase activity than calves from cows fed sodium selenite (363). Previous research had shown that selenium yeast increased milk selenium concentrations more than feeding selenite or selenate (362).

Sheep

Selenium deficiency has serious effects on lamb production. The manifestations are reduced growth and white muscle disease, which affects lambs at two to eight weeks of age (155). New Zealand studies have shown that lack of selenium causes high embryonic mortality, infertility, and high lamb mortality (111). Selenium supplementation increased the number of lambs marketed per 100 ewes lambing from 43 to 93% in the selenium treated group. Neither vitamin E nor an antioxidant was of much benefit in this study, which indicated that selenium per se was primarily responsible.

Goats

Research information is lacking on selenium for goats. There is a good possibility, however, that they would respond similarly to sheep in deficiency symptoms and in the need for selenium. Research scientists in France (114) and Nigeria (115) recommend the use of 0.1 ppm selenium in goat diets.

Horses

White muscle disease in foals, which can be prevented by selenium injection, has occurred in several countries. At necropsy the affected foals have alopecia, degenerative skeletal muscle, yellow-brown fat, and many small hemorrhages (154). Selenium deficiency results in reduced blood serum selenium and elevated serum glutamic oxalacetic transaminase levels. White muscle disease in foals has occurred in a number of foreign countries in areas where cattle and sheep respond to selenium. The foals show muscular stiffness, difficulty in walking, and may be unable to nurse. In severe cases, foals have a dejected appearance, become prostrate, and usually die in one to seven days. Some may excrete myoglobin, which gives the urine a peculiar brownish color. "Tying-up" in horses may respond to selenium and vitamin E injections. Sometimes a single dose of 25 mg of selenium and 250 mg of vitamin E cures horses of "tying-up." Some horses are cured immediately, whereas others respond only partially or take a longer time to respond (93, 98).

Selenium can be quite toxic to horses. In fact some have speculated that General Custer's horses were lame in the Battle of the Little Big Horn from grazing forages containing high levels of selenium. Some selenium accumulator plants can contain 50-10,000 ppm selenium (298). Generally these plants are not very palatable for horses.

Other Animals

Small animals also need selenium in the diet and a level of 0.1 ppm can be added to their diets. In some instances where selenium deficiencies have been studied, the symptoms obtained show some similarity to those of domestic farm animals..

Optimal Selenium Levels

Evidence is accumulating to indicate that higher levels of selenium than now approved by the Food and Drug Administration may be beneficial. The 1984 National Research Council report, Nutritional Requirements of Poultry, recommended selenium levels of 0.15 ppm for chicks 0-6 weeks old; 0.15 ppm for broilers; 0.2 ppm for turkeys; 0.14 ppm for ducks; and 0.2 ppm for Japanese quail (141). Except for turkeys, all the selenium levels are higher than FDA has approved. The 1985 NRC report, Nutritional Requirements of Sheep, recommended a level of 0.1 to 0.2 ppm selenium sheep diets which is higher than the current FDA approved level of 0.1 ppm (155). The 1983 NRC report, Selenium in Nutrition, stated that dietary requirements of most animals for selenium appear to fall within the range of 0.05 to 0.30 ppm (148).

The National Research Committee on Selenium Nutrition has proposed a maximum tolerable level of selenium of 2 ppm in the diet (148). This is a safe level that should not cause harmful effects. It

should not be confused with a level that causes toxic effects. The 2 ppm level would allow a reasonable safety factor between the nutritional requirement for selenium and the maximum tolerated level allowed in the diet.

INJECTABLE TRACE MINERALS

Injectable trace minerals are restricted to use by or on the order of a licensed veterinarian. Selenium is the most common trace mineral administered by injection. When diseases like white muscle disease have been diagnosed, injectable selenium is the most rapid approach to correcting the deficiency. Injectable selenium typically increases selenium levels in treated animal for about 45 days (334). However, injectable trace minerals should be viewed as a transitory, short-term solution to an acute deficiency problem. In intensively managed dairy cows a single dose of injectable trace minerals before breeding had no beneficial effects on first-service conception rates. Cows receiving a does of injectable trace minerals before calving and another dose before breeding had lower conceptions at first service (312).

Barium selenate is the most common injectable selenium source. In horses, New Zealand researchers found that selenium status was improved, but swelling and fibrosis occurred at all dosages evaluated (310). When cattle were injected with 1 mg selenium/kg body weight, between 76 and 99% of the selenium remained at the injection site 119 days post-treatment (309). Using this approach as a routine selenium delivery method is problematic. Most consumers will be apprehensive about consuming meat from animals injected with barium selenate knowing that there is a risk the meat may contain pharmacological doses of selenium. Injectable trace minerals are the most expensive delivery method per unit of nutrient provided.

BIOAVAILABILITY OF TRACE MINERAL SOURCES

Mineral bioavailability is defined as the proportion of the element consumed that is utilized for a biochemical or physiologic function (325). For a mineral to have high bioavailability it must be readily absorbed and easily assimilated by the body. Consequently, bioavailability is influenced by the chemical form of the mineral, the amount in the diet, the amount stored in the body, the concentration of other minerals in the diet, and the health age, and physiological state of the animal to which it is fed. To accurately assess bioavailability, more than one indicator of nutritional status should be monitored. Generally, the animals must be fed a deficient diet before or during the study to which graded levels of the mineral source in question are added (325). Although growth rate, milk production, egg production, etc. are economically important criteria to compare mineral sources, they may not reflect differences in bioavailability. Indicators such as metal-dependent enzyme activity, metallo-protein concentration, chemical balance and pathologic signs are more likely to reflect differences in bioavailability. Miles and Henry (338) have an excellent review of factors to consider when measuring trace mineral bioavailability.

Relative bioavailability will also vary between species. For example, phytate phosphorus is readily available to ruminants, but poorly utilized by monogastric animals. In contrast, copper is much more available to non-ruminants than to ruminants. Consequently, it is risky to predict relative bioavailability of different mineral sources without having measured it for the animal in question.

Knowing the approximate bioavailability of a mineral source is essential to being able to estimate the amount required in the diet. For example, Lawrence (303) used research data to estimate that a 500 kg horse needs 16 mg of bioavailable copper per day to replace endogenous loss. If the availability of the copper source was 70%, then 23 mg per day would meet the requirement. If the availability was 35%, then 46 mg of that copper source are needed to meet the requirement. The amount of supplemental copper required in the diet is doubled due to differences in bioavailability. In addition, twice as much copper will be excreted into the environment with the low compared to the high bioavailable sources. Lawrence (303) summarized nine research studies measuring apparent copper digestion. The values ranged from a low of 9% to a high 48%. The amount of copper required and the amount excreted could be vastly different for the same horse fed these different diets.

When manure is applied at high rates or composted and then applied at lower rates, all of the trace mineral excreted are incorporated into the soil. Agronomists have begun to study the availability of the trace minerals in the soil and its impact on the concentrations in plants. Initial research suggests that the amount of copper and zinc in the soil will influence the amount taken up by some crops but not others (326). Consequently, feeding trace mineral sources with high bioavailability is good for the animal and good for the environment. Bioavailability data for the common trace mineral sources has been summarized for swine (327, 357), cattle (328, 337, 357) and poultry (338, 357).

INORGANIC AND ORGANIC MINERALS

Recently there has been a dramatic increase in the potential trace mineral sources that can be fed to animals. For example in the official publication of the Association of American Feed Control Officials (AAFCO, 289) there were at least 16 different copper sources available to feed manufacturers. There are the traditional inorganic sources, copper sulfate, copper chloride, copper carbonate, copper oxide, etc. and then the organic sources defined by the organic molecule to which they were bound. They are listed as metal amino acid complex, metal amino acid chelate, metal polysaccharide complex, metal proteinate and metal propionate. Deciding which source is most appropriate is a complex issue and the one size fits all approach is not recommended. Miles and Henry (338) have an excellent review on the relative bioavailability of the different inorganic and organic trace mineral sources across all species.

First using a highly available inorganic source will regularly provide the lowest cost per unit of available nutrient. In the majority of situations this is the most cost effective means of optimizing animal health and production. Although there are numerous studies where organic trace minerals have higher bioavailability than traditional sources that does not prove they are more effective. Depending on the experiments chosen, one can find studies that show organic trace minerals to be more, equally, or less effective than their inorganic counterparts. In situations where a deficiency exists or high levels of antagonists bind the inorganic sources, the use of organic trace minerals may be indicated. In most of these cases a mixture of two-thirds inorganic and one-third organic provides the full benefit of the organic source, with less cost. Under the majority of production conditions, using a properly fortified trace mineral salt containing highly available inorganic sources will provide the most cost effective supplementation program.

TRACE MINERAL ANTAGONISTS

Mineral deficiencies may be characterized as primary or secondary depending on the cause of their development. Primary mineral deficiencies are caused by diets that are naturally deficient in one or more minerals. These deficiencies are corrected by the addition of one or more minerals to the diet. Secondary mineral deficiencies are caused by the consumption of one or more mineral antagonist that prevents the absorption or metabolism of another mineral. Simply measuring mineral concentration in the diet may not reveal the source of the problem because all minerals are at or above the requirement. In addition antagonistic mineral concentrations may only be present during certain growing seasons and not create deficiency problems until months later.

Copper is a perfect example of where deficiencies are often the result of antagonistic concentrations of other minerals. Sulfur, molybdenum and iron are three minerals that can readily inhibit copper absorption. High sulfur intakes can result from sulfur in both the feed and water. The increased use of ammonium sulfate fertilizers can increase forage sulfur concentrations to the point that copper deficiency becomes a problem. Florida researchers (329) reported liver copper concentrations of 204, 137, and 72 ppm for cows grazing pastures with no fertilizer, ammonium nitrate, and ammonium sulfate, respectively. Liver copper concentration less than 75 ppm reflect a copper deficiency. Over three years, the forage samples from the ammonium sulfate fertilized pastures average 0.50%. Due to the high sulfur, cows in this study did not respond to a salt-trace mineral mix containing 0.25% copper.

Molybdenum combined with sulfur interferes with copper metabolism through the formation of thiomolybdates in the rumen (147). Suttle (330) showed that increasing dietary sulfur from 0.10 to 0.40% in the presence of molybdenum, increased the copper requirement 50%.

Iron is the second most common trace metal on earth and can be present in high concentrations in both feed and water. The maximum tolerable concentration of iron in cattle diets is 1000 ppm, but dietary concentration as low as 250 ppm have been linked to copper deficiency (278). Exactly how iron prevents copper absorption is poorly understood. One theory involves the disassociation of ferrous sulfide complexes in the acid pH of the abomasum. The free sulfide then binds with copper to form an insoluble copper-sulfide complex.

Many other antagonistic relationships exist between different minerals. For examples, the antagonists for zinc include iron, copper, and calcium. Iron, potassium and magnesium are potentially antagonistic to manganese.

TRACE MINERALS AND CADMIUM TOXICITY

Traditionally cadmium toxicity was associated with the waste from mining and smelting of metals such as zinc and lead and with municipal sewage sludge. Recently cadmium has been detected at high levels in some phosphate and zinc sulfate fertilizers. Some researchers suspect that cadmium is being taken up by certain plants and thus plays a greater role in fescue toxicosis and grass tetany than previously believed. The concentrations of calcium, copper, zinc, iron, and selenium in the diet can have a dramatic effect on cadmium absorption and metabolism.

Cadmium toxicity and metabolism:

Cadmium has been shown to manifest its toxicity in animals by accumulating in most organs with the kidney being the main target organ. It causes the loss of proteins in the urine both from the blood and the kidney itself. Cadmium is also toxic to the central nervous system. It causes alterations of cellular functions in lungs. It affects both humoral and cell mediated immune response in animals.

Cadmium toxicity is directly related to cadmium absorption and retention because the half-life in some tissues, such as kidney, liver and gastrointestinal tract is measured in years. Trace mineral nutrition and calcium levels are the most important factors affecting cadmium absorption in most practical diets. When copper zinc, iron and selenium are adequate, less than 1% of the cadmium consumed ends up being absorbed in most ruminants (347). Adequately nourished cows only retained 0.75% of the oral dose after 14 days, but 0.13% was still present after 131 days (346).

Zinc and copper have similar electron configurations in the outer shell as cadmium. Thus these elements are often antagonistic in that exposure to cadmium will lower the copper status, and increasing zinc consumption will lower cadmium uptake and retention (341). Similarly, increasing selenium intake reduces the toxicity of cadmium by shifting the tissue distribution of cadmium from metallothionein to high-molecular-mass proteins that are more easily excreted.

High cadmium intakes (> 40 ppm in diet) cause symptoms similar to a zinc deficiency and were prevented by zinc supplementation (344). The symptoms include loss of appetite, poor growth, retarded testicular development, and parakeratosis in sheep. When lower concentrations of cadmium were given to sheep that were marginal in their copper status, the symptoms resembled those of a copper deficiency, such as anemia, impaired bone mineralization, loss of wool crimp, abortion and stillbirths (343). When pregnant ewes were fed 3.0-3.4 ppm cadmium in their diets, copper status was impaired to the point that the crimp in the wool was compromised. However, when excess zinc (750 ppm) was added to the diet, the effects on copper status were reduced (342).

In cattle, feeding 5 ppm cadmium during gestation reduced liver copper concentration in the newborn calves by 29%. Feeding 1 ppm cadmium to the gestating cows was sufficient to reduce liver copper stores by 40% (345). Because cattle are susceptible to copper deficiency, excess cadmium is more likely to be detected as a copper deficiency than in other species. Kidney and liver cadmium concentrations increased 5-8 fold when grazed pastures were fertilized with sewage sludge for eight years. However, no histological kidney damage was noted because maximal kidney concentrations were 32 ppm on a fresh weight basis, which is well under the 200 ppm in humans required to cause kidney damage (219).

Ammerman et al. (339) reported the interactions of cadmium and zinc in poultry. Turkey poult fed 2 ppm cadmium had hock and feather abnormalities on a low-zinc diet (10 ppm), but not on a high-zinc diet (60 ppm). However, when 20 ppm cadmium was fed, the 60 ppm zinc did not prevent deficiency symptoms. In poultry, diets containing 60-75 ppm cadmium will reduce egg production, delay testes development in males and cause kidney damage. When rats are fed low-zinc diets, as little as 1.5 ppm cadmium can reduce blood copper and inhibit bone mineralization. In rats, 6 ppm cadmium was found to have the same inhibitory affect on copper absorption as 1000 ppm zinc (342). Confirmation of cadmium toxicity in non-ruminants is best achieved by measuring kidney cadmium concentrations and the presences of histological kidney damage.

Dr. Swerczek a veterinary pathologist at the University of Kentucky has proposed the concept that cadmium may play a role in grass tetany and other induced mineral deficiencies (340). Some plants such as tobacco stalks appear to concentrate cadmium from phosphate fertilization and when these stalks are spread on pastures the cadmium leaches out and is taken up by the grass. Many of the signs associated with grass tetany are similar to cadmium toxicity. It is possible that cadmium, even in very low concentrations in the forages, may have a greater impact on animal health than previously recognized.

Traditionally cadmium toxicity was associated with mining of zinc and lead, and with the application of municipal sewage sludge to pasture. However, the detection of cadmium in some phosphate and zinc sulfate fertilizers means that livestock producers need to be aware of the possibility of cadmium toxicity in environments where it has not been a problem previously. Dietary copper, zinc, iron and selenium can have a major impact on cadmium absorption and retention. Feeding a well-fortified trace mineral salt is the first line of defense to minimize the risk of cadmium toxicity in livestock and poultry.

THE ROLE OF MAGNESIUM AND SODIUM IN GRASS TETANY

Ruminant animals are susceptible to a disease condition characterized by low blood magnesium known variously as grass tetany, hypomagnesemic tetany, grass staggers, lactation tetany, wheat pasture poisoning, and winter tetany. Animal losses vary from year to year but are economically important. For example, in the United Kingdom clinical cases occur in an estimated 1% of the cattle and a third of these cases result in death. In the United States the most susceptible animals are mature beef cows in early lactation grazing lush, rapidly growing grass. Although difficult to measure, dollar losses to cattle producers are estimated to be several million dollars every year.

Magnesium is a mineral that is required by all animals and functions as an enzyme cofactor. It is very important to the central nervous system because it competes with calcium in the excitation-secretion coupling process. This role is directly related to the most common symptom of grass tetany, tetanic contraction of the muscles. Initial symptoms are usually characterized by excessive alertness, wary appearance, and fine twitching of muscles of the face and ears. The affected animal will be uncoordinated and walk with a stiff gait. After a few minutes to 3 to 4 hours, the animal may suddenly drop with convulsive spasms, lying on its side with rapid paddling movements of the limbs. Death often occurs in convulsions, or the animal may become comatose before dying. It is not uncommon for all symptoms to occur in a span of 2 to 3 hours so that the producer does not notice any problems until the cow is already dead. Usually levels of blood magnesium will be approximately 0.5 mg/100 ml of blood compared to normal values of about 1.7 mg/100 ml.

In reviewing the scientific literature it is obvious that the cause of grass tetany is very complex. Although it is characterized by a deficiency of available dietary Mg, many other factors interact to determine whether an individual animal will exhibit the symptoms. In spite of its complexity, it is imperative to begin to put the pieces of the puzzle together if effective prevention programs are going to be implemented. This is even more important because treatment is often unsuccessful.

The most consistent trait of grass tetany is its occurrence in older, lactating animals consuming lush, cool-season grasses that have received some degree of fertilization. The problem is seasonal and often occurs 5 to 10 days after the onset of cold, wet weather. The forage is low in magnesium, sodium, and soluble carbohydrates, but high in nitrogen, potassium, and higher fatty acids. Incidence of grass tetany is higher in herds composed of British beef or dairy breeds than in Brahman breeds (175).

Feeding high levels of potassium generally has depressed blood serum magnesium in ruminants (172) as a result of reduced magnesium absorption (178). The main effect of potassium is on preintestinal magnesium absorption. Tomas and Potter (180) reported that magnesium infused into the omasum or abomasum was completely recovered at the duodenum, but 36% to 61% of magnesium infused into the rumen was not recovered at the duodenum. Correspondingly, it has been shown that ruminal infusion of potassium in sheep resulted in a large decrease in magnesium absorption, but infusing potassium into the abomasum or ileum had no effect.

Magnesium is transported across the ruminal mucosa by an active sodium-linked process (172). In fact, Marten and Rayssiguier (177) suggested that dietary or salivary sodium deficiency decreases

the sodium:potassium ratio in rumen fluid, which results in depressed magnesium absorption. They reported a fourfold increase in magnesium absorption when the sodium:potassium ratio in rumen fluid increased from 0.5 to 5.0. These studies utilized sheep where the rumens were emptied and then filled with buffer solutions containing different sodium:potassium ratios.

Marten et al. (176) reported that magnesium absorption in sheep increased from 22.3% to 34.5% when 2.3 grams sodium were added to a low-sodium, dried grass diet. The ruminal sodium:potassium ratio in this study increased from 0.9 to 5.5. These data suggest that inadequate salt supplementation may increase the susceptibility of animals to grass tetany. Swerczek (322) also observed that cattle with access to loose salt rarely have grass tetany. Certainly when there is a marginal magnesium deficiency, increasing the sodium:potassium ratio in the rumen fluid may be critical to maximizing the absorption of the magnesium that is available.

Another characteristic of diets causing grass tetany is that they are high in soluble protein and low in soluble carbohydrates. Marten and Rayssiguier (177) suggested that the imbalance between protein and carbohydrate in the rumen may lead to a deficiency of absorbable energy from the rumen. If volatile fatty acids and carbon dioxide production are depressed due to a lack of carbohydrate, blood flow to the rumen wall may be decreased resulting in low magnesium absorption. This may be a self-perpetuating phenomenon in that Ammerman et al. (171) showed that ruminal cellulose digestion and thus volatile fatty acid production was decreased by a magnesium deficiency.

Increased lipid or higher fatty acid concentrations in fertilized grasses may increase grass tetany. Magnesium and fatty acids may react to form magnesium soaps which are largely unavailable. Feeding peanut oil has been shown to depress plasma magnesium concentrations of grazing dairy cows (181). Although the level of lipids in most grasses is probably not high enough to cause grass tetany by itself, it is likely one of several nutritional factors in a cascade that, when summed, prompt the disease.

The ultimate goal of any prevention program should be to increase magnesium absorption so that blood levels of magnesium are maintained above 1.5 mg/100 ml. Because the quantity of magnesium that can be mobilized from body stores decreases with age, sufficient magnesium must be consumed on a regular basis. O'Kelley and Fontenot (179) showed that gestating beef cows required about 10 grams magnesium per day. Because magnesium is plentiful in milk, requirements more than doubled to approximately 22 grams during peak lactation.

Compounds used as supplemental magnesium sources include magnesium in the form of oxide, hydroxide, carbonate, sulfate, chloride, or as dolomite. Magnesium oxide contains the highest concentration of magnesium and has been used most commonly to prevent grass tetany.

Most magnesium compounds are unpalatable to ruminants and can not be fed successfully by themselves. Mixing the magnesium source with salt has some very important advantages. First, ruminants have an appetite for salt. Their desire for salt will increase intake of the magnesium source and insure that consumption will occur on a regular basis. Secondly, consumption of sodium and magnesium simultaneously may be critical to increasing magnesium absorption. As discussed previously, proper sodium:potassium ratio in the rumen may be the key to obtaining efficient

utilization of supplemental magnesium. In addition, salt-magnesium mixtures can be self fed without the high-cost labor of hand-feeding a magnesium fortified supplement on a daily basis. Finally, grazing lush forages and lactation usually increases a ruminant's appetite for salt which means that increased magnesium intake is likely to occur at the time of greatest need.

Adding a readily available carbohydrate source to the salt-magnesium supplement may also be beneficial. Frye et al. (1973) found that adding dry molasses, ground corn, cottonseed meal or alfalfa meal to a salt-magnesium oxide supplement increased intake. The salt:magnesium oxide:carbohydrate sources were mixed in a 1:1:1 ratio. Palatability of the mixture is critical if lactating cows are going to consume the 22 grams of magnesium required per day. In addition, the availability in the mixture of readily fermentable carbohydrate sources such as molasses or corn may improve absorption of magnesium from the rumen by increasing volatile fatty acid production resulting in more blood flow to the rumen.

Salt-magnesium supplements should be placed where animals have easy access. In large pastures several mineral feeders should be located throughout the pasture to ensure ready availability. To minimize competition, one mineral feeder should be available for every 15 to 20 cows. If cows are going to be grazed on pasture that are tetany prone, feeding a salt-magnesium mixture a few days in advance will help animals adjust to the mixture and assure a more uniform intake.

Potential economic losses from grass tetany make prevention the cheapest insurance available. The salt-magnesium-carbohydrate supplements will only cost two to four dollars per head for the spring grazing season. In a 100 cow herd, preventing the loss of only one cow every three years would more than pay for the additional costs of supplementation.

Magnesium is important to all farm animals. Many horse scientists now recommend the addition of 5% magnesium oxide to salt for horses to protect against a possible magnesium deficiency. Dairy scientists are also investigating the use of magnesium oxide as one means of raising milk butterfat levels in cows fed high concentrate diets. High concentrate diets depress the fat level in milk. Therefore, magnesium supplementation, which at first was used only for beef cattle, is now used with dairy cattle, horses, sheep and other animals. An adequate and continuous supply of dietary magnesium is needed to prevent magnesium tetany, since animals have limited capacities to store and mobilize magnesium. More studies are needed on magnesium availability in feeds and the factors that affect it.

CHROMIUM HELPS WITH STRESS

Recent research with humans and mice has shown that various stress factors such as high glucose intakes, low protein diets, infection, strenuous exercise and trauma increase the urinary excretion of chromium. Chromium supplementation in mouse diets has been effective in reducing stress-induced losses of zinc, iron, copper and manganese in urine. Chang and Mowat, (182) showed that adding 0.4 ppm chromium from high-chromium yeast improved weight gains and feed efficiency in stressed feeder calves. In addition, supplemental chromium also decreased serum cortisol and improved immune response in stressed calves. In one Canadian study, chelated chromium reduced morbidity to less than one-third of that in the control group. In more recent studies where chelated chromium was used alone or in combination with multiple vaccines, the significant improvements were only found with the combined treatment of chromium and multiple vaccines (259). An additional benefit may be that lower levels of antibiotics would be required for the treatment of sick feeder cattle. Chromium under some conditions has improved cattle performance without the stress of transport (276). Chromium could well be the next trace element routinely added to salt.

NEW TRACE ELEMENTS FOR ANIMALS

As soils decline in fertility and crop or animal yields increase, higher levels of trace elements may be required in animal diets. Molybdenum, nickel, fluorine, vanadium, tin and silicone are some of the trace mineral elements that may become deficient in animal diets in the future. One by one, they may be added to the list of seven trace mineral elements currently being supplemented in animal diets (iodine, copper, iron, cobalt, zinc, manganese and selenium).

When using highly purified or specialized diets, several elements have been shown to be essential. For example, molybdenum is an important part of the enzyme, xanthine oxidase. Fluoride prevents dental caries, may be helpful in osteoporosis, and is needed for growth, and anemia prevention. Nickel is needed for normal reproduction. Silicone is required for growth and proper bone development. Growth rate is decreased with a deficiency of tin or vanadium. Reduced wing and tail feather growth in chicks occurred with vanadium deficiency. Eventually, deficiencies of these trace elements may occur under specialized conditions with practical diets. Already it is known that supplementation of molybdenum, in a few areas where forages contain very low levels of molybdenum, would be helpful in counteracting copper toxicity in sheep. A NRC committee has listed levels of nutrients that might be needed in the diet. However, because requirements for these elements have not been established, levels shown in Table 29 should be used as guidelines only (141).

Table 29. Guideline Levels on Newer Trace Elements (138)

Mineral Element	PPM in the Total Diet
Silicon	250
Tin	3
Chromium	3
Molybdenum	1
Vanadium	0.2
Nickel	0.1

NUTRITION AND DISEASE INTERACTION

An increasing amount of information is accumulating to show that many nutrients are needed at higher levels to improve the ability of animals' immune system to cope with infection. Sodium, chloride, zinc, copper, iron, selenium, phosphorus and magnesium already have been shown to be helpful in this regard (152). It is apparent from the Wisconsin (203) and other studies that nutrient requirements for growth, feed efficiency, gestation and lactation do not necessarily mean that those levels will be adequate for normal immunity and high disease resistance. This indicates the need for more sophisticated studies that include the animal's nutrient needs for normal immunity against diseases. It also means that higher nutrient levels than those recommended by NRC may be needed for maximum productivity and health of the animals.

Salt with additives

Salt can be obtained with or without additives. Most producers add six of the trace minerals (iron, copper, zinc, manganese, cobalt and iodine) and some also add selenium to trace mineralized salt. Salt producers formulate products with additives other than trace minerals. These include other nutrients as well as non-nutritive feed additives. Salt is an ideal carrier of many feed additives because salt is needed by all animals on a daily basis.

Anthelmintics (de-worming agents) may be added to salt. Salt is a convenient means of supplying an anthelmintic for parasite control.

Ionophores such as monensin and lasalocid can be added to salt, primarily for cattle and sheep under pasture conditions. Similarly, antibiotics such as oxytetracycline have been mixed with salt and self-fed (183). This use is most common in a grazing situation. Salt-drug combinations should be fed only if the product is purchased through a feed company or prescribed by a veterinarian.

Vitamins A and D are frequently added to salt. In some cases other vitamins are added, particularly with salt or salt-mineral mixtures for horses, swine or poultry. When vitamins and trace mineralized salts are fed together the mixture should be kept dry to prevent some of the trace minerals from catalyzing the breakdown of the vitamins.

Magnesium in salt or salt-mineral mixtures for grass tetany is being used with increasing frequency.

Sulfur is added to certain salt products. Calcium and phosphorus are added when a complete mineral mixture is desired. Specific amounts of calcium and/or phosphorus can be used, depending on the animal or the feeding program. Salt can be used plain, with added trace minerals or with added macro minerals calcium and/or phosphorus.

USING SALT IN ANIMAL NUTRITION

Form of salt to use

Salt can be provided to animals in many ways. It can be used in the form of loose salt or salt blocks. Loose salt is used in diets mixed for animal feeding. In relatively small pastures, both loose salt and block salt are self-fed to livestock. With very large pastures or extensive range conditions, where mineral boxes are difficult to reach, salt blocks are a good solution. In some cases, they are dropped by airplane onto range areas. Salt blocks can be made for use in specific areas.

Mineral boxes for salt and other minerals

It is very important to provide mineral boxes for livestock when loose salt is self-fed. Some livestock producers will also place blocks of salt or minerals in a mineral box to protect them from the weather. The feeders should be constructed so that the mineral will remain fresh and dry. Every pasture should have at least one mineral box. Location should be selected so the mineral box can be inspected and filled easily throughout the year from a truck or other utility vehicle. Mineral feeders are used more frequently by livestock if they are located near water sources, back-rubbers and shaded loafing areas. The number of feeders should be adequate for the stocking capacity of the pasture. Feeders should be positioned near the ground so young animals can easily reach the contents of salt and minerals. Reference 323 shows examples of mineral boxes that are practical and aid in controlling external parasites by having back-rubbers attached. Other types are also available.

TRACE MINERAL NUTRITION OF FISH

During the past several years a fair amount of new information has been generated on the trace mineral requirements of a few cultured species of fish. Although we know much less than with the other food animal species, the data presented here can serve as a foundation for the future.

Several studies on the mineral requirements of channel catfish have been reported (260). The copper requirement is approximately 5 ppm in the diet. When 2 ppm copper was fed, enzymes such as copper-zinc superoxide dismutase in the liver and heart cytochrome C oxidase were depressed. Gatlin and Wilson (261) reported that iron deficiency signs in channel catfish included decreased weight gains, feed efficiency, hemoglobin, hematocrit, erythrocyte count and transferrin saturation values. Their data suggest that 20 ppm of iron in the diet is necessary to prevent deficiency signs. Gatlin and Phillips (262) reported that 200 ppm zinc in the diet was required to provide adequate zinc for catfish fed diets high in calcium and phytate.

Because of their adaptability, tilapia is becoming an increasing popular culture fish. McClain and Gatlin (263) conducted two experiments to determine the zinc requirements of fingerling blue tilapia. Based on scale and bone zinc concentrations, 20 ppm zinc in the diet was the requirement to prevent zinc deficiency. Additional research is needed to expand our understanding of the trace mineral nutrition of cultured fish.

BSE: POTENTIAL ROLE OF TRACE MINERALS

BSE is a short for Bovine Spongiform Encephalopathy, more commonly referred to as “Mad Cow Disease”. BSE was first observed in Great Britain in 1984 and was identified as a specific disease in 1986. The scope of the disease developed rapidly so that by June of 1990, there were over 14,000 confirmed cases out of a population of approximately 10 million cattle in Great Britain. The epidemic peaked in 1992-93 with approximately 1,000 confirmed cases per week. Since 1986 over 200,000 head of cattle have been destroyed after showing signs of BSE.

The exact cause of the disease is still under scientific investigation. Bacteria and viruses have been ruled out as the root cause. Most scientists believe the disease is caused by a self-replicating protein called a prion. But the question still remains as to why the disease exploded in the British cattle population. Recently a new theory that is getting a great deal of attention is that a trace mineral imbalance may be the root cause.

The term Bovine Spongiform Encephalopathy is the technical name for this disease because brain tissue sections of infected cattle appear spongy and infiltrated with a starch-like plaque when examined under a microscope. BSE causes a progressive degeneration of the central nervous system in cattle

The infective dose required for BSE transmission in cattle is quite small. An oral dose of 0.5 to 1.0 gram (0.02 to 0.04 oz.) of infected brain tissue is all that is required.

The contagious agent has only been found in brain, spinal cord, and retina of infected cattle. Consumption of meat or milk has not caused the disease. The mechanism by which the infectious agent is transferred from the digestive tract to the central nervous system has not been explained.

Prion protein is a glycoprotein produced by nerve tissues. This protein may protect the nerves in that it has superoxide dismutase activity (280). Deactivating harmful oxygen free radicals appears to be a key role. Most diseases involving prions such as CJD, scrapie in sheep, and chronic wasting disease in deer arise sporadically without a known cause. Other diseases are associated with point mutations in the prion protein. Prion diseases are characterized by the conversion of the normal cellular form of the prion protein to an altered isoform. It appears that when the isoform is introduced into the central nervous system, it can catalyze the conversion of the normal prion protein to the abnormal isoform. This isoform then is resistant to normal proteinase enzymes and accumulates within the brain causing the neurological degeneration and behavioral signs. This seeding theory helps to explain why the disease develops very slowly at first and then as more isoforms are developed they spout additional isoforms and the animals deteriorate rapidly.

Recently it has been shown that normal prion protein contains copper. This copper can be utilized at the nerve synapse or incorporated in copper/zinc superoxide dismutase. The prion will bind up to four atoms of copper and assumes a structure that is susceptible to proteinases. However, Brown et al., (281) showed that the prion was also capable of binding manganese and nickel. But more importantly, when manganese replaced copper the three-dimensional structure was changed such that the resulting prion was over 100 times more resistant to proteinase degradation. In addition, as the manganese bound prion aged it became increasingly resistant to the proteinase in vitro.

Proteinase resistance and the resulting fibril formation is the most distinguishing characteristic of the isoform.

When the exact mechanism of BSE is finally understood there will still be the sporadic prion diseases that occur in nature without any known infective agent. Therefore some environmental factor that predisposes certain animals or humans to the disease is a distinct possibility. Recent research into finding a factor common among localized areas associated with scrapie in sheep (Iceland), CJD in humans (Slovakia), and chronic wasting disease in deer (Colorado), showed that the soil in these areas is low in copper and high in manganese (282).

Of course these finds do not explain why BSE has become such a problem in Great Britain. However a farmer, Mark Purdey in Britain has studied this problem extensively and made some interesting observations (283). Mark Purdey contends that the incidence of BSE is not associated with the feeding of meat and bone meal as much as it is with the use of an organophosphate called phosmet to control Warble fly maggots. One of the traits of Phosmet according to Purdey is that it binds copper making it unavailable to biological systems. Could it be possible that the manganese containing prion was present in the meat and bone meal and that small amounts were absorbed and because Phosmet bound the available copper this acted as the seed prion to initiate the development of BSE? The relationship of copper and manganese in the development of prion diseases is gaining credibility. Researchers at Cambridge and in France have looked at the copper and manganese concentrations of CJD victims. They found that manganese was 10 times higher in the brains of CJD victims than was present in the brains of unaffected people

The final chapter in the BSE story has not been written. Theories as to why and how BSE exploded in Great Britain are still being tested in the laboratories. Because BSE has an incubation period as long as 10 years, it takes a long time to prove or disprove a theory. However, the role of copper and manganese in the development of prion diseases is gaining increasing credibility. Maintaining the proper copper:manganese ratio in the diet and environment maybe the cornerstone of BSE prevention in the future.

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