HEAVY METALS – VANADIUM IN POULTRY

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RESUMO

METAIS PESADOS: VANÁDIO PARA AVES

Os minerais essenciais devem ser fornecidos na dieta em concentrações suficientes para promover uma resposta adequada, e a ingestão de outros minerais e deve ser baixa o suficiente para garantir a total segurança tanto para os animais quanto para a população humana que consumirá a carne e os ovos. Os níveis dietéticos máximos tolerados têm sido gerados, o que pode servir como orientação para garantir a segurança tanto para animais domésticos quanto para humanos. Embora existam várias fontes dietéticas de elementos tóxicos em potencial para aves, programas de controle de qualidade adequados na fábrica de ração, associados com boas práticas de produção e um nutricionista capacitado, devem diminuir consideravelmente a ocorrência de uma contaminação significativa. Lençóis freáticos contaminados por dejetos e outras formas de poluição industrial assim como sistemas uubanos de esgoto representam provavelmente as fontes mais comuns de contaminação nos EUA atualmente.

PALAVRAS-CHAVE: minerais, desempenho, toxidcez, nutrição

SUMMARY

All mineral elements can produce adverse effects in animals if they are consumed at sufficiently high levels. Thus, the essential minerals must be provided in the diet at concentrations sufficient to promote optimum response and the dietary intake of other minerals must be low enough to provide complete safety for both the animals and also the human population which will consume the meat and eggs. Dietary maximum tolerable levels have been generated which can serve as guidelines in ensuring safety for both domestic animals and humans. Although there are several potential dietary sources of toxic elements for poultry, adequate quality control programs at the feed mill coupled with good manufacturing practices and a knowledgeable nutritionist should greatly decrease the likelihood of significant contamination. Contaminated ground water from hazardous waste sites and other forms of industrial pollution as well as municipal waste systems probably represent the most likely sources of contamination in the United States at the present time.

KEY WORDS: minerals, performance, toxicity, nutrition

INTRODUCTION

Certain mineral elements are essential dietary nutrients for poultry and livestock. One classification of these minerals, generally referred to as "macro elements" includes calcium, phosphorus, sodium, chlorine, magnesium potassium, and sulfur. Another group, referred to as "micro" or "trace" elements, has dietary requirements which are considerably lower than those of the macro classification. Elements in the latter class include iron, manganese, copper, zinc, selenium, iodine, cobalt, molybdenum, and chromium. Additional trace minerals for which there is evidence of their essentiality include fluorine, vanadium, nickel, tin, silicon, lead, boron, and arsenic. Deficiencies of these elements have not been verified in domestic animals in dietary ingredients to cause concern from the standpoint of their toxicity. The designation "heavy metal" has been used historically although it is loose, nebulous term which is rapidly losing favor with scientists and the regulatory community. This term denotes a potentially toxic source and those elements

generally included in this classification by nutritionists are cadmium, lead, mercury, and arsenic. The dictionary definition of "heavy metal" is "an element with a specific gravity greater than 5". Other mineral elements of nutritional significance which also fit in this category include vanadium, cobalt, copper, iron, manganese, molybdenum, zinc, and chromium.

DIETARY MINERAL CONCENTRATIONS AN AVIAN PERFORMANCE

All mineral elements, whether considered to be essential or potentially toxic, can have an adverse effect upon the bird if included in the diet at excessively high concentrations. An animal's functional response (growth, egg production, etc.) to increasing dietary amounts of a mineral element is the classic bell-shaped curve. There is an initial portion increasing "linearly" which indicates "deficient" consumption of a mineral, followed by a plateau area which is considered the "normal" range of intake. When a "toxic" amount is reached, then production will decrease until eventual death occurs. The dietary mineral concentration which will just promote an optimum response can be considered as the minimum requirement and this amount will vary. The concentration at which a mineral exhibits a toxic effect is also quite variable. There is no single requirement for a mineral by an animal nor is there a single safe concentration at which any mineral can be tolerated whithout adverse effect. There is in actuality a series of minimum required levels and also of maximum tolerable levels that vary from animal to animal and from day to day within the same animal. Factors which can influence both the minimum requirement and also the maximum amount at which a mineral element can be tolerated by an animal include the following:

1. Solubility of the mineral source
2. Growth potential of the animal
3. Physiological function (growth, egg production, etc.)
4. Previous nutrition
5. Age
6. Environment
7. Balance of other nutrients
8. Diesease

ESTABLISHMENT OF MINERAL TOLERANCE LEVELS

Mineral tolerance levels for poultry have generally been established in some type of feeding study in which graded levels of the element were offered and specific criteria were examined. Ideally, production cycle feeding trials should be conducted initially and additional studies involving two or more generations continued with breeder flocks. Extensive studies of this description generally are not conducted and, depending upon the mineral, may not be necessary. In practice, studies of much shorter duration are done in which criteria including growth rate, biochemical lesions, mortality, and deposition of the element in meat or eggs will be measured. A highly soluble, reagent grade form of the mineral being tested is generally used to create a test situation in which adverse effects are most likely to occur. Form of the element, lenght of study, criteria examined, and species of bird must all be considered in applying experimental data under practical conditions.

Maximum tolerable levels of certain dietary minerals for poultry as published by NRC (1980), as well as their required concentrations (NRC, 1994) are presented in Table 1. The maximum tolerance level as used in the table is defined as "... that dietary level that when fed for a limited period, will not impair animal performance and should not produce unsafe residues in human food derived from the animal". These values are not the LD_{50} (lethal dose to 50% of population) calculated for acute exposure. It should be emphasized that toxicosis is a function of concentration and time. Good nutritional practices dictate that dietary concentrations of the mineral elements should be well below the maximum tolerable levels. With toxic metals such as lead, cadmium, and mercury, dietary concentrations should be maintained as low as possible to minimize the carry over into the human diet.

CONCERN FOR EXCESSIVE MINERAL INTAKES

There are several reasons for concern about the possibility of excessive mineral intakes by poultry. Natural water supplies can contain high concentrations of sulfur, fluorine, sodium, magnesium, and iron. In addition, numerous toxic elements, especially heavy metals, can be added to ground water from hazardous waste sites, industrial pollution, and municipal waste systems. The potential for contamination due to leaching of toxic elements from natural soils was demonstrated dramatically in wild fowl at the Kesterson Reservoir Natural Wildlife Refuge 90 miles southeast of San Francisco back in the early 1980s. Ground water purity should be checked by a qualified laboratory before building a poultry facility at any site and then monitored periodically for quality. The burden of safety falls largely on the individual producer for this potential source of contamination. Your local extension service, an agricultural university or local government consumer protection agency should be able to provide assistance to locate a suitable commercial laboratory to conduct these analyses. Early detection of contamination from water-borne sources could be well worth the monetary investment.

Some elements, such as manganese, selenium and molybdenum can occur in plants at such elevated concentrations, that adverse effects may result in animals consuming these forages. This is obviously a greater potential source of contamination for grazing ruminants, but could become an issue for free range birds or ratites. Plants can also become contaminated on the surface with zinc, lead, fluorine, and several other heavy metals from industrial processing plants, resulting in sufficient amounts to cause harm to animals. Another potential source of contamination is that resulting from human error during transport and mixing of ingredients and final delivery of a finished feed to the poultry house. This accidental administration can result in acute signs of toxicosis which may be quite different from the chronic effects displayed after minerals have been fed at greater than normal concentrations over an extended period of time. Good manufacturing practices and quality control should minimize problems of this nature.

Many diets formulated from natural feedstuffs are deficient in one or more of the essential minerals which must be provided in supplemental forms. Generally the elements are not supplied as purified reagent grade chemicals. Some may be ores which have had little processing or purification while others may be by-products of some industrial process. The amount of contaminating elements will depend on the native material from which the mineral supplement is obtained and the type of processing which it undergoes. Certain elements present may be beneficial while others may be potentially toxic to poultry. Compositional data for several supplemental trace mineral sources that have been used for poultry diets are presented in Table 2 (Ammerman et al., 1977). Complete analyses on individual samples are limited and those data presented the variability that occurs in the composition of various mineral sources. The suggested guidelines established by AAFCO (1999) for contaminant levels in mineral feed ingredients are shown in Table 3. Typical contaminant concentrations of mineral feed ingredients used in the United States are found in Table 4. Several values found in the individual samples presented in Table 2 exceed these typical concentrations.

The economic importance of feed grace phosphates and the presence of fluorine, vanadium, and other potentially harmful elements has resulted in several recent publications on the composition of various phosphate sources as illustrated in Tables 5 through 8 (Sullivan et al., 1994; Lima et al., 1995).

The use of animal wastes and certain by-product feedstuffs has resulted in higher mineral intakes by animals. As animals digest and utilize the nutrients in their diet, there is a proportionately greater disappearance of organic nutrients with a subsequent increase in the concentration of minerals in the excrement. Litter materials contain both living and dead microorganisms which have been shown to remove added lead through the processes of bioaccumulation and biosorption (Gupta and Keegan,

1998). This material may have potential for removing contaminating elements from accidental toxic spills, but could prove problematical when used as a feed ingredient for livestock.

The use of iron as a flocculating agent in the offal and meat meal industry has added excess concentrations of this element to poultry diets. If these ingredients are fed in combination with a phosphate supplement also high in iron or with drinking water containing high concentrations, toxicosis could occur. It is critical for the nutritionist to investigate fully the various properties of ingredients other than corn and soybean meal used in poultry diets to make necessary adjustments to the computer matrix limits used in formulation. Kan (1994) reviewed numerous harmful substances including heavy metals which could be encountered in poultry feeding and discussed methods to decrease their toxicity.

Unfortunately, analysis of several of the heavy metals responsible for animal poisoning requires specialized equipment and techniques which are not available at some university or commercial laboratories. In addition to the standard flame atomic absorption spectrophotometer used in most laboratories which could detect high concentrations of most cations, analysis of mercury requires a cold vapor system. Precision measurement of arsenic or selenium in feedstuffs requires a hydride generator coupled with a graphite furnace atomic absorption spectrophotometer (Fecher and Ruhnke, 1998). A graphite furnace is also needed to accurately determine the extremely low concentrations of vanadium and lead in complex biological samples as well as cadmium or lead in feedstuffs (Blake and Bourqui, 1998). An excellent review of the many pitfalls concerning laboratory methods for determination of fluorine in complex samples is available (Venkateswarlu, 1983). The ion specific electrode is currently used most frequently in fluorine analysis. Samples of feed ingredients and water should only be analyzed by qualified personnel at a laboratory at which the elements in question are determined on a routine basis with proper internal standards to check the accuracy and precision of the measurements.

EFFECTS OF HEAVY METALS IN POULTRY

Numerous experiments concerning toxicosis of mineral elements were conducted during the decades of the 1960s and 70s. Invention of the atomic absorption spectrophotometer by Walsh in the mid 1950s made the determination of many cations a rapid, fairly simples procedure so that research on all aspects of mineral metabolism fluorished. Additionally, during those decades the use of radioisotopes and disposal of contaminated materials and animals was not policed with anywhere near the degree of concern exercised at the present time. Many trials with animals were conducted to trace the kinetics of numerous elements including various toxic heavy metals.

Although the volume can no longer be considered recent in terms of modern laboratory methods making use of cells cultures and nucleic acid analyses to further refine our understanding of the mechanisms of mineral concerning the toxic effects of mineral elements in animals. Research conducted prior to that time was sufficient to establish guidelines for safe levels of mineral administration for life cycle feeding of animals. Due to the rise of the animal rights movement as well as improved cellular techniques, in the future university animal care committees will probably be reluctant to grant approval for simple studies on mineral toxicosis in which livestock and poultry are the test animals.

VANADIUM

The maximum tolerable level designated for vanadium in diets for poultry is 10 ppm. As opposed to fluorine which is less toxic to poultry, vanadium is better tolerated by ruminants which have a maximum dietary concentration of 50 ppm. Therefore if high-vanadium phosphates must be used during a crisis situation, the material would have less of a toxic effect in diets for cattle than poultry. High-vanadium phosphate sources generally originate in the Rocky Mountains in the western part of the U.S. and not in Florida deposits. Romoser et al. (1960) reported vanadium concentrations as high as 6,000 ppm in some rock

phosphate deposits. Berg (1963) found 1,400 ppm vanadium in a commercial tricalcium phosphate. Addition of the former source at 2% in the total diet for poultry would add 120 ppm vanadium, while addition of the latter would provide 28 ppm. As observed in Tables 5 through 8, vanadium concentrations in commercial phosphate sources ranged from 26 to 796 ppm for individual samples with most values falling within the range of 50 to 200 ppm.

Growing bone and kidney are the principal targets for vanadium administered orally to birds (Hathcock et al., 1964). Sell et al. (1986) also reported high concentrations in the liver and magnum as well as kidney and the skeleton following a radio-tracer dose of the element to laying hens. Vanadium appears to exert its toxic effect through inhibition of enzymes and cell damage from lysis (Waters et al., 1975). The vanadyl cation behaves like a simple divalent ion which can compete for ligand binding sites with Ca^{+2}, Mn^{+2}, Zn^{+2}, and Fe^{+2}. The vanadate form of the element competes with phosphate in many biochemical processes (Nielsen, 1997). Vanadium also exists in a peroxo form which can mimic the action of insulin (Fantus et al., 1989). Deterioration of internal egg quality has been speculated to be mediated by an inhibition of magnum motility during egg formation (Eyal and Moran, 1984). Toussant et al. (1995) studied oviduct morphology with a light microscope and computer image analyzer. They were able to show statistically significant differences in magnum fold height and surface epithelium height along with isthmus epithelium height in strains of Leghorn layers selected for production of eggs with normal and low Haugh units. In another study of morphological changes between control hens and birds producing low Haugh unit eggs in response to dietary vanadium, there were no clear changes evident.

The NRC publication (1980) and Nelson (1983) reviewed the literature concerning vanadium toxicosis in poultry, so an extensive review of dated material will not be repeated herein. Growth depression has been reported in birds consuming diets containing greater than 10 ppm vanadium for more than 21 to 28 days. The toxic threshold occurred between 8 and 50 ppm of the element from reagent grade sources in the nine publications reported by Nelson (1983). The vanadium in phosphates may be less soluble and therefore, somewhat less toxic than the reagent grade sources used in most of these animal feeding trials. Poorer albumin quality has been reported in eggs from laying hens consuming as little as 6 ppm vanadium in a dicalcium phosphate (Sell et al., 1982). Approximately 28 ppm were needed to decrease egg production in that same experiment. Hatchability was decreased by feeding 25 ppm vanadium as calcium orthovanadate fed for 20 weeks (Kubena et al., 1980), whereas 40 ppm were required to affect egg weight and egg specific gravity (Ousterhout and Berg, 1981). Kubena and Phillips (1982) reported no mortality in hens fed 50 ppm vanadium as calcium orthovanadate for five consecutive 28-day periods. However, in hens fed 100 ppm vanadium, there was 11% mortality in period 3, which increased to 39 and 56% in periods 4 and 5, respectively.

Since these earlier reports were reviewed, Sell et al. (1986) observed a decrease in interior quality of eggs from two strains of hens fed 3 or 6 ppm added vanadium from dicalcium phosphate from 25 to 80 weeks of age. The detrimental effect was generally overcome by addition of 5% cottonseed meal to the diet. Feed intake, egg production and egg weight were not affected by dietary vanadium in this experiment. Previously, Ousterhout and Berg (1981) had indicated that 20% cottonseed meal or 0.4 to 0.5% ascorbic acid in the diet protected hens from toxic effects of 40 ppm vanadium.

A significant decline in Haugh units was observed within 3 days following feeding of a diet containing 20 ppm vanadium as ammonium metavanadate (Toussant and Latshaw, 1994). This adverse effect was not ameliorated by addition of as much as 3,000 mg ascorbic acid/kg added to the diets. Davis et al. (1995) reported a similar decline in interior quality whithin 2 days after feeding 10, 20 or 30 ppm added vanadium. The detrimental effect on egg interior quality induced by feeding hens 10 ppm vanadium was partially improved by addition to the diet of the antioxidants

ascorbic acid (100 ppm), vitamin E (200 IU/kg), or $-carotene (500 ppm) (Miles et al., 1997). Hill (1988) reported that addition of 400 ppm copper or 100 ppm mercury overcame the growth depression observed in broiler chicks fed for 19 days. It would hardly be within the realm of good nutritional practice to add potentially harmful amounts of other elements to poultry diets to alleviate toxicosis resulting from vanadium.

Benabdeljelil and Jensen (1990) fed 0, 10, 30, or 100 ppm vanadium as ammonium metavanadate to laying hens (43 weeks of age) for 4 weeks and reported a decrease in feed intake and egg production in those fed 30 or 100 ppm vanadium. Egg weight, shell thickness and shell breaking strength were not affected by vanadium. Albumen height was decreased by feeding 10 ppm or greater of the element. In a second study, when these authors fed hens (47 weeks of age) either 0 or 10 ppm vanadium in combination with 0, 100, 1,000 or 5,000 ppm ascorbic acid, albumen height increased linearly with increasing antioxidant in the hens also given vanadium. However, addition of chromium as chromium chloride at 0, 10 or 50 ppm to diets containing 10 or 30 ppm vanadium for 4 weeks did not improve interior egg quality.

Duyck et al. (1990) demonstrated the importance of proper storage conditions on egg albumen quality in birds fed 10 ppm vanadium for 30 days. Haugh units decreased from 82 in eggs from hens fed the control diet and stored in the cooler under proper conditions (62°F, 60% relative humidity) for 1 day to 74 in eggs from those same hens stored for 7 days. Eggs from hens fed 10 ppm vanadium averaged 71. Haugh units after 1 day of storage and 64 after 7 days. By comparison, eggs from birds fed the control diet, but stored in the house were the daytime temperature averaged 81°F, ranged from 79 to 59 Haugh units at 1 and 7 days, respectively. Similar values in eggs from birds fed 10 ppm vanadium were 68 to 53 Haugh units, respectively. In this study, egg production and egg weight were not affected by dietary vanadium and the element did not influence the rate of albumen deterioration in either the refrigerated or non-refrigerated eggs. Unfortunately, in some locations overseas where high quality ingredients may be more difficult to obtain at times due to economic and political constraints, it may also be far more difficult to store eggs under refrigerated conditions.

The detrimental effect of vanadium interior egg quality is well documented. Eyal and Moran (1984) further characterized these changes to albumen in hens (33 weeks of age) fed 20 or 30 ppm added vanadium as ammonium metavanadate for 4 weeks. The percentage of outer thin albumen increased and the percentage of inner thin albumen decreased with increasing dietary vanadium. The percentage of thick albumen was not affected and neither was the distribution of component proteins in any albumen fraction. The authors concluded that the functional properties which could affect the consumer were not altered by dietary vanadium. Hence, the deterioration in interior egg quality is a matter of aesthetics rather than food safety at the consumer level. Unfortunately, as demonstrated by Berg et al. (1963) the detrimental effect of vanadium on egg quality can persist for more than a month after excess concentrations of the element are removed from the hens' diet. The liquid egg market may be the best option for sale of eggs with poor albumen quality.

Davis et al. (1995) fed comercial egg-type hens diets containing 0, 10, 20, or 30 ppm vanadium for 6 weeks. After 24 days, hens given 20 or 30 ppm vanadium showed a decline in egg weight and eggshell weight as well as the detrimental influence on albumen quality. Excess vanadium, however, did not affect cell mediated or humoral immune responses.

Bressman and Miles (personal communication) fed hens (32 weeks of age) diets containing 0, 20, 40, or 60 ppm added vanadium as ammonium metavanadate for 56 days. In addition to the detrimental effects on feed consumption and egg production observed at the two higher dietary concentrations, albumen quality was poorer for all hens supplemented with vanadium. Fecal moisture also increased linearly from 76% in hens fed the control diet to 80, 81 and 83% with increasing dietary vanadium concentrations, respectively. This almost 10% increase in excreta moisture could represent a problem for manure management in the layer industry, especially for fly control which is highly dependant on manure moisture. More recently, Miles

(unpublished data) fed broiler breeders a corn soybean meal based diet that had been supplemented with either 0, 50 or 100 ppm vanadium as amonium metavanadate and observed a de-pigmentation of the egg shell at both the 50 and 100 ppm dietary concentration of vanadium when compared to the un-supplementend control diet.

CADMIUM

With a maximum tolerable level of 0.5 ppm (NRC, 1980), cadmium can be considered the most toxic of the heavy metals. However, that value was chosen based more on the safety of carryover to the human food supply as opposed to signs of toxicosis observed in the animals consuming the element. Severe effects have been reported in animals consuming 5 ppm in the diet or 1 ppm in drinking water with adverse effects occurring at dietary levels as low as 1 ppm. The most likely source of contamination in the animal feed industry is in conjunction with the use of zinc sulfate or poorly processed zinc ores as sources of supplemental zinc. Other potential sources include mining and smelting operations, corrosion of metal-plated iron, discarded cadmium-chloride products, and the use of urban sewage sludges to fertilize pastures or croplands (NRC, 1980). Cadmium shares similarities in chemical reactivity with zinc and these two elements have common metabolic pathways in biological systems. The cadmium content of practical-type diets for poultry generally contain from about 0.05 to 0.35 ppm (NRC, 1980). Corn grain contains less cadmium than the plant leaves, but in soybeans and wheat the content of the element is similar between seeds and leaves. Some leafy vegetables can contain 100 ppm (dry basis) without evidence of toxicosis in the plant. Meat meal containing a high percentage of organ meat then muscle tisse could potentially contribute higher concentrations of the element. The 30 samples of dicalcium phosphate found in Tables 5 through 7 ranged from less than 0.1 ppm cadmium to 67 ppm with an average of 9.4 ± 14 ppm. Addition of the highest cadmium-containing phosphate to the diet at 2% would add approximately 1.5 ppm cadmium while the average concentration (9.4 ppm) would add less than 0.2 ppm of the element. The defluorinated phosphates are practically devoid of cadmium (Table 8), probably as a result of volatilization of element during thermal processing. As illustrated in Table 2, zinc sources contain higher concentrations of cadmium than other trace element supplements, but addition of 60 ppm zinc to the diet would contribute only 0.1 ppm cadmium from the zinc oxide containing 1,290 ppm of the element and about 0.008 ppm from the oxide containing 79 ppm. Sources of zinc sulfate generally contain greater amounts of cadmium than oxide forms and should be analyzed carefully prior to use.

Induction of synthesis of the protein metallothionein in the intestinal tract is the primary protective mechanism the animal has to prevent absorption of toxic amounts of cadmium. The element is sequestered by the protein and the epithelial tissue subsequently sloughed and eliminated in feces. When japanese quail were fed a diet containing 1 ppm radio-cadmium for 1 week, followed by 50 days on a basal diet, less than 4% of the initial dose remained (Jacobs et al., 1978). Approximately 25% was found in liver and another 25% in kidneys with 12% in the intestinal tract, probably in conjunction with metallothionein. Cadmium which is absorbed into tissues is retained by the body for a considerable period of time, thus exposure should be minimized, especially in breeders and layers as opposed to broilers or turkeys. Kidney is the primary site of damage from the element.

The anemia observed during cadmium toxicosis can be alleviated to a certain extent by addition of iron to diets (Hamilton and Valberg, 1974). Addition of dietary ascorbic acid and selenium had the greatest protective effects on broiler kidney damage from cadmium consumption (Rambeck and Kollmer, 1990); however, use of selenium in this capacity would be questionable.

LEAD

Earlier this century, the majority of poisoning cases in animals resulted from lead consumption (NRC, 1980). Although it is no longer used in the United States, the addition of lead to gasoline increased the concentration of this element in air, soil and water, as well as plants grown along roadways. Consumption of lead-containing paint chips also

resulted in numerous cases of toxicosis in humans and animals and, consequently, this source of the element has also been removed from the consumer market in the U. S. Spent lead shot was also a source of the element, especially in wild water fowl, but shotgun shells have made use of steel shot since the latter 1980s. The normal content of feed ingredients ranges from 0.02 to 3 ppm lead on a fresh basis (Reichlmayr-Lais and Kirchgessner, 1997). Lead is generally of little concern in feed grade phosphates and ranged from less than 1 to 77 of little concern in feed grade phosphates (Tables 5 to 7) and from less than 1 to 5 ppm in defluorinated phosphate (Table 8). Due, in part, to the discontinued use of lead-containing gasoline, the environmental lead burden in the United States during the last twenty years has decreased. The current most likely source of dietary contamination with the element would result from poor quality sources of zinc oxide in trace mineral premixes. As illustrated in Table 2, zinc oxides can contain excessive amounts of lead. However, addition of zinc to a diet from the zinc oxide containing 30,000 ppm lead would only add 3 ppm lead, which is considerably below the maximum tolerable level of 30 ppm listed in Table 1.

Lead accumulates in bones and appears to be relatively immobile. Birds absorb approximately 10% of dietary lead (NCR, 1980). Jeng et al. (1997) reported increased lead in kidney, liver, and egg yolks of Tsaiya ducks dosed with 10 mg/kg body weight for 3 months, but these authors did not analyze lead weight, but not by the lower dose. Feed intake during the experiment was not reported and, therefore, it was not possible to convert these dosages to a dietary concentration basis.

Clinical signs of lead poisoning usually include feed refusal, anemia, intestinal problems, and kidney dysfunction (NRC, 1980). A more recent finding is an increased concentration of the highly unsaturated fatty acid, arachidonic acid in tissues of poultry poisoned with lead. Knowles and Donaldson (1996) observed this change in birds which were fed 1,500 ppm lead in the diet or administered a similar dose by gastric intubation. This demonstrated that lipid peroxidation observed in feed resulting from lead was independent of this effect in birds. Embryonic exposure to the element via injection into the air sac at day 12 of incubation with from 50 to 400 µg lead/egg adversely affected imune function in the resulting pullets tested at 6 weeks of age (Chen et al., 1998). Again, the tolerance level of 30 ppm was selected based on food residues for human consumption. No adverse effects were noted in chickens given 100 ppm lead as lead oxide for 546 days (Hermayer et al., 1977) or as lead acetate for 28 days (Damron et al., 1969) whereas 1,000 ppm from either source caused poorer growth and egg production. Excess dietary calcium and phosphorus have been demonstrated to decrease the toxic effects of lead (Berg et al., 1980).

ARSENIC

In addition to environmental arsenic contamination in conjunction with lead and copper smelting, there are several potential sources of the element for poultry. Commercial fishmeal may contain 3 to 20 ppm on a dry matter basis (Anke et al., 1997), while shellfish and crustaceans have been reported to concentrate the element to the extent of 175 ppm (NRC, 1980). Soil and plant concentrations can become elevated from the use of arsenical sprays for control of insects and weeds. Cereal grains generally contain 0.05 to 0.4 ppm arsenic on a dry matter basis (Anke et al., 1997). The greatest potential source of excessive arsenic for poultry is accidental over supplementation with organic arsenicals such as Roxarsone, arsanillic acid, nitarsone and carbarsone (AAFCO, 1999). These compounds have been used successfuly as growth promoters and coccidiostats by the poultry industry for over 50 years. A 5-day withdrawal period is required before marketing birds to reduce tissue levels in food for human consumption. Good manufacturig pratices greatly reduce any likelihood of accidental poisoning in a commercial flock, but the possibility is there.

Unlike cadmium, arsenic is rapidly excreted from the animal body. The organic forms of the element are tolerated more readily than the inorganic forms as shown in Table 1. The maximum tolerable level for organic arsenic is 100 ppm, whereas that for inorganic forms is 50 ppm. Arsenic can substitute for phosphorus, disrupting cellular oxidative processes and result in capillary injury and

tissue hypoxia. Generalized signs of acute toxicosis involve gastrointestinal, cardiovascular, neurological, and hematological functions.

Vodela et al. (1997 a, b) studied the effect of multiple addition of heavy metals and volatile organic compounds to drinking water to mimic the concentrations often found in ground water contaminated by leaching from facilities. Concentrations of arsenic, cadmium, and lead were 0.8, 5.1, and 6.7 ppm, respectively, in the low-dosing group and ten times those concentrations in a high-dosing group compared with filtered control water. Reduction of water intake was accounted for with pair-watered controls. In one experiment, vitamin and mineral premixes were also fed at 100, 50 or 0% of recommended requirements. Addition of heavy metals, even at the low dosing rate, decreased feed intake and weight gain and suppressed humoral, and cell-mediated immune response in broilers. There was a significant interaction in which adverse effects of the heavy metals were further increased in the sub-optimal diets. In similar studies with broiler breeders, contaminated drinking water decreased egg production and egg weight and increased percentage of embryonic mortality.

Hassan et al. (1998) also conducted experiments with multiple heavy metals including cadmium, copper, lead and mercury added to water at high and low concentrations for 7 weeks. They reported subtle adverse effects on some immune parameters of chicks. The main sites of accumulation for lead and copper were bone and liver, respectively, while cadmium and mercury were found in kidney. The residue found in muscle was lower than that in other tissues.

MERCURY

Although the maximum tolerable level of mercury for poultry is only 2 ppm, there is relatively little problem with contamination of this element in diets unless as the result of direct contamination in the vicinity of smelting plants or from excessive fossil fuel combustion. Inorganic mercury occurs at very low concentrations on the order of less than 0.2 ppm in supplemental mineral sources (Tables 2 and 5 through 8). The greatest potential sources are that of fish protein concentrates or seed grains which have been treated with mercury as a fungicide. Fish can greatly concentrate methylmercury from contaminated food or direct uptake from water. Mercury can also be found in hair and feather meals used as protein supplements for animals.

Poultry can tolerate greater amounts of inorganic mercury than the organic methylated form. However, biomethylation of inorganic forms can occur in the environment or in the animal and increase the potential for toxicity. Hens given 8 ppm mercury as methylmercuric chloride for 70 days concentrated 55% of the dose in eggs, especially in albumen (Sell et al., 1974). Young chicks tolerated 25 ppm mercury as inorganic mercuric chloride in water for 42 days with no adverse effect (Thaxton et al., 1975), whereas 5 ppm in feed for 33 days as organic methylmercuric chloride resulted in 50% mortality (Soares et al., 1973). There are conflicting reports concerning the efficacy of selenium to reduce the toxicity of mercury (NRC, 1980). The dietary concentrations of selenium needed to have an effect (5 to 8 ppm) are above the maximum tolerable levels for this element and hence of little practical importance.

OTHERS

Addition of aluminium sulfate (alum) to poultry litter has been used to control phosphorus runoff and subsequent freshwater eutrophication when litter is applied to land as fertilizer. Huff et al. (1996) investigated toxic effects of dietary aluminium sulfate in broilers to predict adverse effects in birds produced where treated litter can be consumed. Dietary addition of 0.93% alum (0.08% aluminium) for 3 weeks decreased body weight. Decreased bone ash concentration, tibia breaking strength and intestinal strength were observed when 3.7% alum (0.32% aluminium) was fed. Bone aluminium concentration increased at the higher level, but muscle aluminium was not affected. Experiments lasting an entire 7-week growout cycle are needed to determine safety for longer periods.

Feed intake and gain of poults were suppressed

and liver copper concentration increased when water containing approximately 200 ppm copper was given in conjunction with diets containing 200 ppm of the element for 5 days (Ward et al., 1994). Proventriculitis was reported in broilers fed diets containing copper as copper sulfate in excess of 200 ppm of the element for 4 weeks (Wideman et al., 1996). The maximum tolerable level of copper listed in Table 1 is 300 ppm.

TABLE 1. Dietary requirements[a] and maximum tolerable levels[b] (ppm) of trace mineral elements for poultry

	Requirements			Maximum
Element	Broiler	Leghorn	Turkey	tolerance
Aluminium	NGc	NG	NG	200
Arsenic	NG	NG	NG	
inorganic				50
organic				100
Cadmium	NG	NG	NG	0.5
Chromium	NG	NG	NG	
chloride				1,000
oxide				3,000
Cobalt	NG	NG	GN	10
Copper	8	NG	6 -8	300
Fluorine	NG	NG	NG	150, 200[d]
Iodine	0.35	0.29 - 0.044	0.4	300
Iron	80	38 - 56	50 - 80	1,000
Lead	NG	NG	NG	30
Manganese	60	17 - 25	60	2,000
Mercury	NG	NG	NG	2
Molybdenum	NG	NG	NG	100
Nickel	NG	NG	NG	300
Selenium	0.15	0.05 - 0.08	0.2	2
Silicon	NG	NG	NG	---
Vanadium	NG	NG	NG	10
Zinc	40	29 - 44	40 - 70	1,000

[a] NRC (1994). Range for all age groups within a classification.
[b] NRC (1980).
[c] Not given.
[d] For turkeys and chickens, respectively. As sodium fluoride or source of similar solubility. Fluoride in certain phosphates may be less toxic.

TABLE 2. Potentially toxic elements in feed grade trace mineral sources[a]

Source	Primary element, %	Toxic element, ppm				
		Pb	As	Cd	Al	Hg
Manganese						
Oxide	56.3	660	213	-	-	-
Oxide	45.9	2,180	1,400	-	15,200	0.009
Oxide	55.3	1,280	119	-	-	-
Iron						
Carbonate	47.8	20	1	-	960	-
Sulfate	19.8	15	<1	-	18	-
Sulfate	20.2	16	<0.15	-	28	-
Oxide	63.0	70	30	-	6,050	-
Oxide	61.7	4,630	23	-	290	-
Zinc						
Oxide	70.2	30	2	1,290	-	-
Oxide	79.9	4,770	9	170	-	-
Oxide	60.1	30,000	149	79	-	-
Copper						
Oxide	84.1	130	7	-	-	-

[a] Ammerman et al. (1977). Each line represent one sample.

TABLE 3. Official guidelines suggested for contaminants in individual mineral ingredients[a]

Category	Typical analysis not suggested if below (ppm)	Typical analysis suggested if between (ppm)	Prohibited above (ppm)
Highly toxic	5	5 - 500	500
Cadmium			
Mercury			
Selenium			
Toxic	100	100 - 1000	1000
Cobalt			
Molybdenum			
Vanadium			
Barium			
Tungsten			
Copper			
Lead			
Moderately toxic	500	500 - 2000	2000
Arsenic			
Nickel			
Iodine			
Antimony			
Slightly toxic	2000	>2000	None
Boron			
Aluminum			
Bromine			
Zinc			
Bismuth			
Manganese			
Chromium			

[a]AAFCO (1999).

TABLE 4. Typical contaminant levels (ppm) of AAFCO-defined mineral feed ingredients[a]

Element	Arsenic	Cadmium	Lead	Mercury
Calcium	2 - 5	5 - 10	5 - 30	0.05
Phosphorus	2 - 5	5 - 10	5 - 30	0.05
Potassium	1	-	1	1
Salt	-	-	-	-
Sulfur	1	-	1	1
Cobalt	2 - 20	2 - 200	1 - 100	1 - 20
Copper	3 - 100	2 - 100	9 - 600	1
Iron	1 - 50	-	1 - 90	1
Iodine	2	1	3	2
Manganese	2 - 100	1 - 20	1 - 90	-
Magnesium	1 - 10	-	1 - 20	0.1 - 5
Selenium	-	1 - 5	-	10 - 1000
Zinc	10 - 800	80 - 500	100 - 2000	1

[a]AAFCO (1999). Adapted from NFIA Mineral Ingredients Handbook (1979).

TABLE 5. Potentially harmful elements in samples of different domestic and foreign commercial dicalcium phosphate sources[a]

	Phosphate source							
Element	1[b]	2	3	4	5	6	7	8
Iron	20	8,100	11,100	6,300	5,200	8,500	9,900	11,000
Albuminum	30	1,500	9,200	1,500	2,100	1,100	8,000	2,400
Arsenic	1	9	15	3	13	98	12	16
Cadmium	<0.1	7	5	<0.1	14	<0.1	6	17
Chromium	1	754	82	6	131	6	89	67
Fluorine	70	1,600	1,400	1,400	1,200	1,500	1,400	2,100
Lead	1	2	30	3	2	1	77	19
Mercury	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Nickel	11	28	21	12	44	17	23	54
Vanadium	2	160	195	1210	168	159	185	91

[a] Lima et al. (1995).
[b] Reagent grade dicalcium phosphate dihydrate.

TABLE 6. Potentially harmful elements in samples of different domestic and foreign mono-dicalcium phosphate sources[a]

	Phosphate source								
Element	1	2	3	4	5	6	7	8	9
Iron	14,100	8,760	7,710	8,880	5,610	8,860	8,120	8,680	8,520
Aluminum	7,400	3,700	9,600	7,200	4,800	8,900	7,400	3,000	8,200
Arsenic	6	2	5	6	2	5	4	4	5
Cadmium	5	24	4	4	<1	3	67	22	4
Chromium	82	194	74	85	538	65	493	200	96
Fluorine	18,000	1,500	1,600	1,500	1,900	1,400	1,900	1,400	1,400
Lead	4	<1	2	12	1	<1	<1	<1	30
Mercury	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	,0.1
Nickel	21	24	9	10	8	7	110	28	16
Vanadium	149	69	140	179	47	145	796	102	181

[a] Sullivan et al. (1994).

TABLE 7. Potentially harmful elements in samples of different domestic and foreign di-monocalcium phosphate sources[a]

	Phosphate source												
Element	1	2	3	4	5	6	7	8	9	10	11	12	13[b]
Iron	9,260	8,380	9,650	2,410	7,070	9,220	8,480	4,540	8,640	390	11,260	4,490	290
Aluminum	9,800	6,700	5,500	2,300	6,000	4,800	2,800	3,300	2,800	20	4,300	4,500	30
Arsenic	6	8	8	10	6	5	4	12	2	<1	5	8.3	<1
Cadmium	4	4	4	121	3	4	40	<1	22	<1	4	<1	<1
Chromium	84	77	80	116	63	77	271	12	188	<4	99	11	<4
Fluorine	1,500	1,200	1,400	2,500	1,300	1,400	7,300	1,300	1,400	700	1,600	600	200
Lead	7	14	<1	4	16	<1	4	<1	1	2	<1	<1	<1
Mercury	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<1	<1
Nickel	9	9	10	18	16	13	42	9	28	<1	12	7	1
Vanadium	164	151	84	159	161	138	83	44	71	<20	140	26	21

[a] Sullivan et al. (1994).
[b] reagent grade dicalcium phosphate dihydrate.

TABLE 8. Potentially harmful elements in samples of different domestic and foreign thermochemically produced defluorinated phosphate sources[a]

	Phosphate source												
Element	1	2	3	4	5	6	7	8	9	10	11	12	13
Iron	7,600	5,460	9,180	11,790	7,03	2,500	4,630	5,360	4,680	7,420	7,690	7,110	6,420
Aluminum	8,200	3,400	6,800	6,300	7,700	3,000	1,500	4,500	3,400	6,900	7,500	6,400	7,200
Arsenic	<1	<1	<1	<1	1	<1	<1	<1	<1	<1	1	2	<1
Cadmium	<1	<1	<1	<1	<1	<1	<1	<1	1	<1	<1	<1	<1
Chromium	88	114	48	30	87	<4	95	94	105	287	229	216	51
Fluorine	1,500	1,200	1,400	2,500	1,300	1,400	7,300	1,300	1,400	700	1,600	600	200
Lead	1	<1	5	2	<1	<1	<1	<1	<1	<1	<1	<1	<1
Mercury	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Nickel	11	12	13	14	13	1	13	26	20	131	106	99	18
Vanadium	97	53	117	84	97	127	53	185	45	105	110	101	104

[a] Sullivan et al. (1994).

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