Element Concentrations Toxic to Plants, Animals, and Man

GEOLOGICAL SURVEY BULLETIN 1466

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By LARRY P. GOUGH, HANSFORD T. SHACKLETTE, *and* ARTHUR A. CASE

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An appraisal of the toxicity hazard to plants, animals, and man from natural and manmade element concentrations of environmental concern

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By LARRY P. GOUGH, HANSFORD T. SHACKLETTE, and ARTHUR A. CASE

INTRODUCTION

Investigators have long recognized the importance of certain elements, commonly called "minerals," in the diet of humans and animals and in the soil that supports plants, in that these elements are essential for the life or optimum health of the organisms. Deficiencies of 20 to 24 elements in animals and man (Frieden, 1972) and of 13 to 18 elements in plants (Epstein, 1965) have been recognized. At the same time, an understanding of the responses of these organisms to the insult of toxic concentrations of these and other elements also has been of interest. More recently, concern has arisen regarding the effects of an organism's exposure to the more subtle chronic and subchronic concentrations of certain elements that industrial and other human activities are releasing into the environment.

Many studies of element toxicities have been made, and reports of these studies are scattered through the biological, medical, geological, and other literature. The present requirement for preparing environmental impact statements before certain types of industrial and other operations are undertaken necessitates that judgments be made regarding the potential health effects, if any, of elements that may be released into the environment by these operations. Although these judgments may be outside the fields of the physical and biological scientists who must predict environmental impacts that are likely to occur, certain valid estimations can be made if adequate information is available. The purpose of this outline is to provide some specific data on the known toxicity levels of certain elements that may reach potentially dangerous concentrations in the environment. We have limited our emphasis to toxic levels that are acquired by organisms through absorption, ingestion, and inhalation; intravenous, subcutaneous, intramuscular, or intraperitoneal injections of toxic elements can hardly be considered of environmental concern. We have given but little emphasis to the effects of occupational exposure to hazardous chemical compounds.

Much has been written on the toxicities of some elements such as lead, mercury, and selenium, and our chief task with these elements was that of selecting the most useful reports to include. For some other elements, reports of toxicities are few and often in obscure publications. The elements selected for this discussion are those that may be environmentally important and especially those for which we have quantitative data. Some of the information in this outline is from unpublished data in our files; most statements, however, are identified by reference citations.

The elements are presented alphabetically by their common English names. We have divided the notes on each element into three parts—plants, animals, and man—and classified associated toxicities under natural and man-induced conditions, where appropriate. References to the toxicities of a few elements of possible environmental concern are inadequate for an evaluation to be made of their hazard. These elements are grouped near the end of this report, and a general discussion is given of the available information on each element.

Concentrations of elements generally are presented as parts per million (ppm), although other methods of expressing concentrations may be quoted. In some instances units such as mg/kg of body weight, μmoles/L, $\mu g/m^3$, parts per billion (ppb), or percent provide a clearer concept for a given example, and converting them to ppm was considered to be unnecessary. A part per million is 10^{-4} percent and a part per billion is 10^{-7} percent.

It will be noted in this report that many of the elements are essential for plants or animals, yet are toxic under certain conditions (Wallace, Romney, Alexander, and Kinnear, 1977). The toxicity of an element depends, first of all, on its chemical form. For example, chlorine in the elemental form (a gas) is highly toxic, whereas chlorine compounds may be relatively harmless, as is sodium chloride (common salt). Conversely, elemental arsenic is not toxic, although some arsenical compounds are highly toxic. Many reports do not specify the chemical form of an element when discussing its toxicity. It can be assumed for most of these reports that, under the specified natural or experimental conditions, the element existed in an undetermined compound and that only the total concentration of the element was known. For most reports in which the basis for expressing concentrations of an element in plant or animal tissues is not specified, a dry-weight basis can be assumed.

The toxicity of a substance is also determined by the dosage—that is, the amount (in relation to body weight or to the growth medium) and the frequency and duration of administering the substance to the organism. The duration of specified dosages of toxic elements was not given by some investigators, including Bowen in table 7.4 of his 1966

paper. Some reports define the minimum lethal dose (MLD); that is, the amount of a substance that kills the organism in one dose. In reports that relate toxicity to body weight of the experimental animals, "live" or "wet" weight of the animal is commonly used. Amounts of an element in relation to body weight are expressed as ingested amounts, unless otherwise noted. The term "LD_{50}" means that the specified dose is lethal to 50 percent of the experimental organisms. Quantities of total diets given are based on a one-day consumption, unless otherwise stated.

To be toxic, an element must be "available" to the plants or animals—that is, it must exist in a form that can enter tissues of the organism either in solution (generally aqueous), as a gas, or (uncommonly) as a solid; or it must emit ionizing radiation. Total amounts of a toxic element in the environment are not relevant to an adequate estimation of the toxicity hazard that may be present unless it can be shown that the element exists in, or is likely to assume, an available form under the environmental conditions in which it occurs.

The toxicities of elements resulting from ionizing radiation are not discussed in this report. Although these elements may be of primary concern in some environmental studies, an adequate appraisal of their impact on plants, animals, and man is beyond the scope of this report. Exposures of organisms to natural occurrences of radiation levels significantly higher than those found in the normal environment are not common, and the effects of these exposures have been but rarely noted. Because the areas where these radiation levels are found generally are very limited in extent, the mobility of the larger animals reduces their period of exposure to this radiation. We have reports of the effects of naturally occurring radiation only for plants. The studies by Shacklette of bog bilberry (1962a) and of fireweed (1964) that grew over a near-surface uranium (pitchblende) deposit demonstrated conspicuous morphological changes that were attributed to genetic modification caused by radiation. In later reports by Steere (1974; 1977, p. 426), the evolution of a new species of moss (*Coscinodon arctolimnia*) at the same pitchblende deposit noted above was suggested to be ". . . the end result of a progressive series of multiple mutations induced by natural radioactivity over a period of thousands of years." These are the only reports that we have of the influence of naturally occurring radiation on morphological, and perhaps genetic, changes in plants.

Experimental studies by Cannon (1957) demonstrated that morphological changes in native plant species (prince's plume, among others) could be induced by "salting" natural soils with radioactive agents. The extensive experimental studies of the effects of radiation from radioactive isotopes on many kinds of organisms, and the increased body burdens of radioactivity in native plants, animals, and

man caused by radioactive fallout from nuclear explosions, are not reviewed in this report.

Some types of landscape disturbance may result in a deficiency of certain elements that are essential to organisms in low, but toxic in high, concentrations. Amounts of these elements that are nutritionally required for the major classes of organisms cannot be generalized because they are highly dependent on characteristics of different species. Therefore, we have only noted the essentiality of an element, or lack thereof, for the larger classes of organisms, following the generally accepted concepts of essentiality for the element. The list of essential elements is subject to change because the metabolic role of the trace elements is continually being reappraised by experimental studies. Moreover, the definition of essentiality depends on the views of the investigators, which range from a strict concept that only growth and reproduction are required to broader concepts that include optimum health and growth of the organism. Generally accepted criteria for determining essentiality of an element for animals were given by Underwood (1958, p. 34) as follows: "(1) Repeated demonstration of a significant growth response to dietary supplements of the element and this element alone; (2) development of the deficiency state on diets otherwise adequate and satisfactory, i.e., containing all other known dietary essentials in adequate amounts and proportions and free from toxic properties; and (3) correlation of the deficiency state with the occurrence of subnormal levels of the element in the blood or tissues of animals exhibiting the response." The same principles can be applied to determining essentiality of an element to plants and other organisms. A method of determining whether an element is essential or nonessential in human tissues was proposed (Liebscher and Smith, 1968) based on essential trace elements having a "normal" distribution of concentrations and nonessential trace elements having a "log-normal" distribution, but the validity of this method has not been fully tested.

Gough and Shacklette (botanists) of the U.S. Geological Survey prepared this report with the assistance of Case (clinical veterinarian, toxicologist) of the School of Veterinary Medicine, University of Missouri—Columbia.

ALUMINUM

[Nonessential for plants and animals]

Plants

Under natural conditions—Because aluminum is abundant in most soils, toxicity is largely dependent on the exchangeable amount present due to low soil pH. Only small quantities of soluble aluminum

were found by Magistad (1925) in soils that had pH values between 4.7 and 7.8. Pierre (1931, p. 193-194) stated, in discussing his experiments, "It will be noted that the concentrations of aluminum in the soil solutions are in most cases very small, even at low pH values. . . . As was to be expected it was found that the greater the hydrogen-ion concentration of a particular soil, the greater is the concentration of aluminum in its displaced soil solution." For instance, in Cory silt loam he found 5.7 ppm aluminum in the soil solution at pH 4.43, but only a trace of aluminum at pH 6.00. Agronomists have demonstrated that varieties of cultivated species vary greatly in their aluminum tolerance. Foy and others (1967) reported that aluminum-sensitive wheat and barley cultivars apparently mobilize soil aluminum, and thereby increase its availability, by lowering the pH of the rhizosphere. Because of aluminum toxicity, certain crops cannot be grown on some naturally acid soils without applications of lime. In contrast, some plants are known as "aluminum accumulators" and may contain as much as 37 percent aluminum in ash.

Under man-induced conditions—Susceptibility to aluminum poisoning differs widely among plant species. McLean and Gilbert (1927) classified 12 crop plants according to their sensitivity to aluminum in culture solutions as follows: sensitive (depressed by 2 ppm aluminum)—barley, beet, lettuce, and timothy; intermediate (depressed by 7 ppm)—cabbage, oats, radish, rye, and sorghum; and tolerant (depressed by 14 ppm)—corn, redtop, and turnip. Wallace and Romney (1977) reported, "The threshold concentration of Al for toxicity was about 20 μg/g in rice shoots and about 30 μg/g in soybean leaves. The solution level necessary for these concentrations was 8 μg Al/ml. Plant concentrations which caused severe toxicity were 70 μg Al/g plant with 81 μg Al/ml solution. Most Al remained in roots, but leaves contained more than did stems of soybeans." Acidic mine spoil and drainage waters may be suspected of causing aluminum poisoning in some native and cultivated plants.

Animals

Under natural dietary conditions—We have no reports of animal toxicity attributed to naturally occurring aluminum in the environment. Bentonite, kaolin, $Al_2(SO_4)_3$, Al_2O_3, and $Al(OH)_3$ have been used as growth stimulants for rabbits, chickens, sheep, cattle, and hogs without adverse effects as long as they composed no more than 1–2 percent of the diet (Sorenson and others, 1974). It seems very unlikely that grazing animals, even those that consume a relatively large proportion of their diet as soil (such as sheep and horses), would ever achieve these concentrations naturally. Several investigators have reported that aluminum is a specific root poison for many species and that the poisoned roots are unable to translocate the element to other

parts of the plant (Pratt, 1966a); therefore there seems to be little likelihood that grazing animals are affected by these plants. On the other hand, sweetleaf, an aluminum accumulator (Robinson and Edgington, 1945), is a preferred browse shrub for horses and cattle (Small, 1933); yet no toxicity symptoms in these animals have been reported as due to aluminum. However, Hutchinson (1943) concluded that this plant, reported to contain 2.4–3.5 percent aluminum in dry matter, could not constitute the sole diet of these animals because of the inhibitory effect of aluminum on phosphorus metabolism.

Under man induced dietary conditions—Acute toxicity studies involving laboratory animals were reviewed by Sorenson and others (1974). These authors gave the LD_{50} for $Al_2(SO_4)_3$ administered orally to mice as 6.2 g/kg and for $AlCl_3$ as 3.85 g/kg. Further, the LD_{50} for $Al(NO_3)_3 \cdot 9H_2O$ to rats was given as 0.26 g/kg. Chronic toxicity results in several phosphorus-metabolism imbalances including excretion of phosphorus, decreased incorporation of phosphorus into phospholipids, and a drop in ATP and a rise in ADP levels in blood. Sorenson and others (1974) reported that 150 mg of $AlCl_3$/kg/day fed to rats will result in the expression of these imbalances. They also reported that concentrations of aluminum in excess of 1.5 μg/ml in the environment of rainbow trout fingerlings were lethal to some. Bowen (1966) listed 200 mg Al^{3+} in a 10-g/day dry-weight diet (20,000 ppm) as being toxic to rats, whereas 220 mg (22,000 ppm) was lethal.

Man

Aluminum poisoning in humans appears to be rare. Smith (1928) reported that ingestion of 150 mg of aluminum per day is without obvious effects on normal humans; 200 mg may, however, give rise to mild catharsis, which increases with the dose. Sorenson and others (1974) reported that 5.5 mg of aluminum per kilogram of body weight in food and drink does not cause adverse effects in humans, probably because the aluminum is usually in the less-toxic colloidal form. Regarding absorption of inhaled airborne aluminum, Hutchinson (1943, p. 252) wrote, "The fact that all investigators have found aluminum in the bile indicates that some of the aluminum present in the faeces, as well as in the urine, has been involved in a metabolic cycle. It is conceivable that this aluminum has in part entered the blood stream through the pulmonary epithelium, but if such a mode of entry were generally important significant negative balances would have been reported wherever balance sheets have been made." A number of observations indicate that high concentrations of aluminum may be toxic to the nervous system (Crapper and others, 1973). These authors analyzed the brains of persons who died with Alzheimer's disease (a disease occurring after the age of 40 and producing progressive dementia) and found concentrations of aluminum similar to those in the

brains of experimental animals that had been injected with 150–225 μg aluminum, as aluminum chloride, and that as a result had developed neurological symptoms similar to those of Alzheimer's disease in humans. Pulmonary fibrotic reactions to inhaled silica and certain aluminum-containing compounds can result in silicosis, aluminosis, aluminum lung, and bauxite pneumoconiosis (Sorenson and others, 1974).

ANTIMONY

[Considered nonessential for plants and animals]

Plants

Under natural conditions—Brooks (1972) listed toxicity to plants as moderate; we have found, however, no reported cases of natural poisoning. Shacklette (1965b) reported that a birch tree growing over, and having root contact with, mercury, antimony, and arsenic minerals in Alaska contained 50 ppm antimony in the ash of stems, yet the tree exhibited no symptoms of toxicity. Samples of 27 trees and shrubs that grew over this deposit, but which were not proven to have root contact with these minerals, contained 7–50 ppm antimony in stem ash without evidence of toxicity to the plants, even though the soil contained as much as 95 ppm antimony (Shacklette and others, 1978). Brooks (1972) reported 1 ppm as the concentration in plant ash. Soils were reported by Brooks (1972) to contain 0.5 ppm—only half the value he reported in plants—which suggests that plants concentrate this element above the levels in soils. The studies in Alaska cited above do not support this view, nor did Lisk (1972), who gave the plants/soil antimony ratio as 0.01. In analyses of 863 soil samples from throughout the conterminous United States, one sample was found to contain 500 ppm antimony; all other samples contained less than 150 ppm (the lower detection limit of the analytical method that was used) (Shacklette and others, 1971). The single high value was attributed to contamination of the sampling site by fluorspar mine tailings.

Under man-induced conditions—We have no reports of experimental studies of the toxicity to plants caused by this element.

Animals

Under natural dietary conditions—Because of the low concentrations of antimony believed generally to occur in plants, toxicity to animals from plant sources is unlikely. Sittig (1976) reported, however, that marine animals concentrate antimony in their muscle tissue, and that studies had indicated certain levels in this tissue that were toxic to some fish.

Under man-induced dietary conditions—Sea birds were killed in the Irish Sea in 1969 in a massive disaster, the cause of which was unknown but suspected to be ocean dumping of industrial wastes.

Holdgate (1971) reported that livers of the dead birds contained concentrations of antimony ranging from 40 to 400 ppm, and Tucker (1972) stated that the 400 ppm that was found could be considered the approximate lethal threshold. Extremely low concentrations of antimony in feed were shown by experiments to shorten the life span of small mammals (Schroeder, 1974a).

Man

Under normal environmental conditions—There seems to be little danger to man from concentrations of antimony that are commonly encountered in natural materials. The levels in foodstuffs, however, are not well established.

Under the effects of contamination by industrial operations—Exposure to antimony dust can cause irritation of nasal passages, eyes, and lungs. Ingestion of 100 mg is fatal (Schroeder, 1974a). Occupational exposure to antimony ore, elemental antimony, and antimony oxides has been implicated in a lung disease (pneumoconiosis), whereas exposures to antimony trisulfide have been associated with cardiovascular changes (Sittig, 1976). This writer reported further (p. 7), "There is no evidence that antimony is carcinogenic, teratogenic, or a mutagen."

ARSENIC

[Considered nonessential to plants and animals]

Plants

Under natural conditions—Although the free element is not considered poisonous, some of its compounds are extremely so (Liebig, 1966). Brooks (1972) stated that the toxicity of this element to plants was severe. It was reported (Embleton and others, 1976) that >5 ppm (dry matter) arsenic in the leaf tissue of Navel and Valencia oranges is probably an excessive amount. The arsenic in normal soils ranges from <1 to 40 ppm (Underwood, 1971); within this range, damage to plants is not expected, and the arsenic content of most species is low (<10 ppm; arsenic in the leaf tissue of Navel and Valencia oranges is probably based on dry weight). Soils in the vicinity of some heavy-metal deposits may contain much more arsenic, and Douglas-fir growing in such soils may contain as much as 8,200 ppm in ash (Warren and others, 1968).

Under man-induced conditions—The toxicity to plants of certain arsenic compounds, principally sodium arsenite and arsenic trioxide, is so great that these compounds were used as herbicides for many years. Arsenic compounds such as calcium arsenate, lead arsenate, and cupric arsenite (Paris green) formerly were widely used as insecticides. The long-term use of these materials in agricultural practice accounts for most recorded cases of plant toxicities. The National Research Council (1977a) reported from the literature that leaves of

fruit trees displaying toxicity symptoms (shot-holing and defoliation) had arsenic concentrations of 2.1–8.2 ppm; normal leaves, 0.9–1.7 ppm. The toxicity depends on the concentration of soluble, not total, arsenic in soils. Damage to alfalfa and barley was proportional to the concentration of soluble arsenic; if above 2 ppm, marked damage to the plants occurred (Vandecaveye and others, 1936). Soils "salted" with 500 ppm sodium arsenate and allowed to come to equilibrium for 1 month were toxic to all vegetable crops tested, whereas, at 10, 50, and 100 ppm, crops survived (National Research Council, 1977a). Studies of unproductive, old orchard soils in the Yakima Valley, from which apple trees had been removed, revealed 3.4–9.5 ppm of readily soluble arsenic in the top 15 cm of soil. In Missouri, orchard soils with total arsenic levels above 100 ppm were considered to be highly contaminated (Hess and Blanchar, 1977). These authors also found that most of the arsenic was near the soil surface (upper 20 cm). Arsenic is introduced into the environment as an air pollutant from burning coal, and from smelters and refineries (Underwood, 1971), but we have no reports of damage to plants from these sources of arsenic.

Animals

Under natural dietary conditions—Examples of toxicity of arsenic to animals under natural conditions are believed to be uncommon. Grimmett (1939) investigated livestock poisoning in a valley of New Zealand and found that soils and muds contained as much as 1.9 percent arsenic; water from springs, streams, and surface depressions varied from a trace to 13 ppm of arsenic; and pasture grasses contained 2.8–6.9 ppm (dry weight) arsenic. He concluded that the danger to stock was not from the arsenic in the forage, but from that in the mud or water which might be ingested.

Under man-induced dietary conditions—There is probably little danger of arsenic poisoning from feeding plants and plant products to animals. In fact, four organic arsenic compounds (arsenilic acid, 4-nitrophenylarsonic acid, 3-nitro-4-hydroxyphenylarsenic acid, and arsenobenzene) are used as growth stimulants for pigs and poultry (Underwood, 1971). Selby and others (1974), however, reported the death of several cattle that grazed in pastures contaminated by high arsenic in the soil (probably the result of drainage from a refuse heap suspected of containing arsenic residues). Further, Case (1974) stressed that numerous reports in the literature emphasize the danger of allowing sheep or cattle to graze in orchards sprayed with arsenicals. Trivalent compounds (arsenites) are far more toxic than the pentavalent forms (arsenates). Case (1974) reported the following lethal oral dosages of sodium arsenite for sheep: for 1 of 48, 2 g; for 4 of 30, 3 g; for 14 of 15, 4 g. In addition, he stated that all sheep given 1 g of lead arsenate per day for 6 days died within 6 to 94 days. The National

Research Council (1977a) stated that, in general for most animals, the lethal oral dose is 1–25 mg/kg of body weight as sodium arsenite. The 96-hour LD_{50} for mice and rats is 11.2 ppm arsenite in the diet, whereas it is 112 ppm for arsenate. Schroeder and Balassa (1966) reported that arsenic deactivates enzymes with thiol groups and acts as an antagonist of iodine and selenium.

Man

Under natural dietary conditions—Water is the most common natural material causing arsenic poisoning. Amounts in excess of 0.05 mg/L in drinking water constitute grounds for rejection (U.S. Public Health Service, 1962). Wyllie (1937) reported illness and one fatality in a farm family that used water from a deep well having 0.4–10 ppm arsenic (as As_2O_3). Large beds of FeAsS occurred about 16 km from this well. Water from shallow wells near this farm were free of arsenic. Borgono and Greiber (1971) reported that prolonged consumption (12 years) of water high in arsenic (0.8 ppm) produced cutaneous lesions in over 30 percent of the inhabitants of a city in Chile. Foods of plant origin contain so little arsenic that toxicity to humans is unlikely. Seafood contains much more arsenic—maximum values reported (fresh weight) follow: oysters, 10 ppm; mussels, 120 ppm; prawns, 174 ppm; and shrimp, 42 ppm (Underwood, 1971). This author stated further that the amounts of arsenic ingested daily in the food are greatly influenced by the amounts and proportions of seafoods included in the diet.

General notes on toxicity—Inhalation of high levels of arsenic released from powerplants and smelters may pose health problems. Friberg (1975) reported that coal from Czechoslovakia containing 1,000–1,500 g of arsenic/ton of coal was responsible for the release of 0.5–1.0 ton of arsenic per day into the atmosphere from a powerplant. Elemental arsenic is not toxic (Schroeder and Balassa, 1966), whereas most compounds of this element are highly toxic (Stecher, 1968). Schroeder and Balassa (1966) reported that mountaineers in Syria and the Alps have been known to consume large quantities of native arsenic ("arsen"), believing that it promotes endurance at high altitudes. The minimum fatal dose of arsenous oxide for man was given as 0.06–0.18 g by Monier-Williams (1949). This author also stated that the British Royal Commission set maximum limits of 1.4 ppm arsenic for solid foods and 0.14 for liquid foods including beverages. This limit for foods seems unrealistic, in view of the arsenic content of seafoods listed above. Thomas (1973) gave 5–50 mg/kg body weight as the probable lethal dose for an adult; Bowen (1966) listed as toxic 5–50 mg $As^{(III)}$ or $As^{(V)}$ in a 750-mg/day dry-weight diet (7–67 ppm) and as lethal, on the same basis, 100–300 mg (133–400 ppm), duration not given. The National Research Council (1977) gave the average esti-

mated fatal dose of arsenic trioxide as 125 mg. (For a 70-kg man, this is equivalent to about 1.4 mg/kg of body weight).

BERYLLIUM

[Considered nonessential for organisms]

Plants

Under natural conditions—The toxicity of beryllium to plants is given as severe by Brooks (1972). The geometric mean for beryllium in 847 samples of U.S. soils was reported to be 0.6 ppm with a range of <1 to 7 ppm (Shacklette and others, 1971). Only a small proportion of this total amount, however, is actually available for absorption by plants. Beryllium often substitutes for aluminum in secondary clay minerals. Further, it will displace divalent cations in the exchange complex and become strongly fixed in the soil. Beryl (beryllium aluminum silicate, the important beryllium-bearing primary mineral) contains about 5 percent beryllium, but beryl itself is biologically inert. Such compounds as beryllium chloride, beryllium fluoride, and beryllium sulfate are toxic to plants. Bidwell (1974) stated that beryllium is able to replace magnesium as an essential element in some fungi and partly in tomatoes. Further, it apparently stimulates the growth of some plants (ryegrass and kale) while inhibiting others (bean) (Bidwell, 1974). We have no reports of beryllium toxicity to plants under field conditions.

Under man-induced conditions—Because of new demands for the general use of beryllium in industry (particularly space and atomic energy research) there is concern that the concentration of beryllium in agricultural soils may be increasing. Very little is known, apparently, about beryllium cycling or its ability to enter the human food chain by being assimilated by crops. Romney and Childress (1965) found that beryllium (as beryllium chloride) at levels greater than 2 ppm in nutrient solutions reduced the growth of alfalfa, lettuce, peas, and soybeans. Also, they reported that the yield of beans and wheat was reduced when beryllium occupied the equivalent of 4 percent of the cation-exchange capacity of soils. (Their experiments included a cation-exchange capacity for beryllium as high as 16 percent.) In a nutrient solution containing 16 ppm beryllium, plants showing toxicity symptoms of brown-root and stunted foliage had concentrations of beryllium in plant tops ranging from 27 ppm (dry weight) in alfalfa to 75 ppm in peas. These authors concluded that, for the crops they studied, beryllium was not readily translocated to other plant parts from the roots. One report (Griffitts and others, 1977, p. 9) stated, "There is no evidence at present that beryllium is moving from soils

into food plants in the United States in amounts that are detrimental to plants, animals, or people."

Animals

Under natural conditions—We have no reports of beryllium toxicity to animals grazing on native vegetation.

Under man-induced dietary conditions—The toxicity of beryllium when ingested at chronic levels appears to be indirect. Monier-Williams (1949, p. 493) wrote, "Beryllium salts, when fed to rats, poultry, etc., produce the so-called beryllium rickets, due to immobilization of phosphorus by formation of insoluble beryllium phosphate." Beryllium, like cadmium and lead, has a long biological half-life and therefore tends to accumulate in animal tissues, particularly bone. When inhaled it generally causes chemical pneumonitis and once absorbed by the animal, competes with magnesium for enzymes (Dulka and Risby, 1976). These authors reported the LD_{50} for beryllium to mice as 0.5 mg/kg of body weight. The Subcommittee on Nutrient and Toxic Elements in Water (1974) reported from the literature that 28.5 mg/L beryllium was not very toxic to goldfish, minnows, and snails. Apparently, beryllium given to laboratory animals parenterally or by aerosols is strongly carcinogenic (Schroeder, 1974b).

Man

Although considered by Schroeder (1974b) to be, perhaps, the most toxic of all "environmental" metals, relatively few cases of beryllium poisoning in man have occurred. Ill effects on workers associated with beryllium was first noted in this country in 1940 (Schubert, 1958) among workers processing beryllium ores. Subsequently, many beryllium poisonings were reported in association with the fluorescent tube industry when exposure to phosphor dust was involved. Schubert (1958, p. 29) wrote, "By 1949 the list of cases had grown to such alarming proportions that the major manufacturers of fluorescent tubes decided in consultation with officials of the U.S. Public Health Service to discontinue the use of beryllium phosphors." Beryllosis is mainly a granulomatous disease of the lungs; however, ulcers and granulomatous skin lesions can result from beryllium absorbed directly into cuts and abrasions. Schroeder (1974b) reported that as little as 45 μg inspired beryllium has caused chronic beryllosis and that the disease has occurred when air containing 0.1 $\mu g/m^3$ was inhaled (no time period given). Schubert (1958, p. 33) stated that ". . . the acute disease could be attributed to air concentrations of soluble salts in excess of 100 micrograms per cubic meter, and that when the air level exceeded 1,000 micrograms of beryllium, nearly everyone developed acute beryllium poisoning." Schroeder (1974b) reported that the combustion of coal (which can contain from 1–3,000 ppm beryllium) con-

tributes to the total amount already in the air from other industrial sources.

BORON

[Essential only for higher plants]

Plants

Under natural conditions—The difference between required amounts of boron and toxic amounts is very small—commonly only a few parts per million in soil. Analysis of total-soil boron is not very useful in predicting toxicity; the boron may be held in relatively insoluble compounds such as tourmaline, or in organic residues or marine sediments where it is more readily available. The amount of water-extractable boron in soil is a good index of essential or toxic levels (Bradford, 1966a). For example, early work by Kelley and Brown (1928) in California showed that >2.3 ppm water-soluble boron in the soil of citrus and walnut orchards was probably the toxic threshold. Sauchelli (1969) reported that extractable boron in the range of 0.5-1.0 ppm promotes normal growth of orchard trees, whereas values above 1.0 ppm are high and values above 2.0 ppm indicate possible toxicity. Boron tolerance varies widely among plant species; examples of this range of tolerance follow: sensitive—lemon and other citrus trees, peach, navy bean, and pecan; semitolerant—lima bean, tomato, corn, wheat, and cotton; tolerant—carrot, lettuce, onion, alfalfa, beet, and asparagus. Branson (1976) reported that, in general, the limits of boron tolerance, as measured in saturation extracts of soil, are 5 ppm for semitolerant and 10 ppm for tolerant crops. The following concentrations (ppm, dry matter) of boron in leaf tissue are considered excessive: >260, Navel and Valencia oranges (Embleton and others, 1976); >80, pears and plums; >85, almonds; >90, apricots; >100, apples and peaches; >250, avocados (Jones and Embleton, 1976); >300, grapes (Cook and Wheeler, 1976); and >250, rutabaga (Gupta, 1977).

Under man-induced conditions—Because the difference between essential and toxic amounts is so small, most cases of toxicity are caused by excessive amounts applied in fertilizers. John, Chuah, and Van Laerhoven (1977) found that hot-water soluble boron concentrations ranging from 16.4 to 50.1 ppm (resulting from the application of 50–100 ppm boron) in three different soil types from British Columbia were toxic to spinach and corn. Tissue concentrations of boron in these experiments ranged from 348 to 409 ppm in spinach (dry matter) and from 1,077 to 1,758 ppm in corn. Toxic amounts may also be introduced in irrigation water (Kelley and Brown, 1928). Where the boron content of water is 0.15–0.30 mg/L or more and 45 cm/ha (or more) of water is used, boron deficiency is unlikely. But if the boron content of the water

is greater than 1.1 mg/L, some sensitive crops will begin to show toxicity symptoms (Bradford, 1966a).

Animals

Under natural dietary conditions—Underwood (1971, p. 434) stated, "Boron intakes by grazing animals must be very variable, depending upon the soil type and on the plant species consumed, because the boron concentrations are influenced by the species and the boron status of the soil. These intakes would invariably be much higher, per unit of body weight, than those of humans consuming mixed diets containing a substantial proportion of foods of animal origin." The pastures of solonetz and solonchak soils of the Kulundinsk steppe in Russia were reported by Plotnikov (1960) to be so high in boron that gastrointestinal and pulmonary disorders occured in lambs. Moreover, the water supplies were unusually high in boron (0.2–2.2 mg/L), which probably contributed to the boron toxicity.

Man

Underwood (1971, p. 434) stated, "The boron in food, and boron added as sodium borate or boric acid, is rapidly and almost completely absorbed and excreted, largely in the urine. Where high intakes occur either accidentally or from the treatment of large burns with boric acid, similar high absorption and urinary excretion take place, but sufficient boron may be temporarily retained in the tissues, especially in the brain, to produce serious toxic effects." Bowen (1966) listed 4,000 mg boron (borate) as toxic in a 750-g/day dry-weight diet, duration not given.

BROMINE

[Considered nonessential for plants and animals]

Plants

Under natural conditions—The bromine content (principally as bromides) in normal soil is so low, ranging from 10 to 40 ppm, that bromine toxicity has not been a problem in crop production in the past (Martin, 1966a).

Under man-induced conditions—Most cases of bromine toxicity to plants are related to residual bromine in the soil following fumigation with bromine compounds (principally ethylene dibromide and methyl bromide). However, the possibility exists of bromine contamination of soils by motor vehicle and industrial emissions. In land plants bromine normally ranges from trace concentrations to about 260 ppm in the dry tissue. It is especially high in melons, carrot tops, celery tops, and mushrooms. In contrast to land plants, marine plants contain as much as about 2,000 ppm (Martin, 1966a). Plant species differ greatly in their tolerance to soil bromine; onion is so sensitive that it has been

used as a test plant for detecting soil bromide (O'Bannon, 1958). Other sensitive plants include bean, beet, cabbage, celery, citrus, pea, peanut, pepper, potato, spinach, sugar beet, sweet potato, and turnip. The bromine content of dry leaf tissue has been used as an indication of toxicity; for example, leaf bromine concentrations in citrus seedlings of 0.17, 0.33, 0.4, 1.3, and 1.8 percent were associated with 12, 22, 31, 57, and 90 percent reduction in growth, respectively (Martin and others, 1956). Concentrations of water-soluble soil bromine were reported to have the following effects: 38–83 ppm reduced growth of bean and cabbage; 26–53 ppm injured carnation plants; 15 ppm reduced growth of citrus seedlings, and 600 ppm severely injured the plants; and growth of beans was greatly retarded by 300 ppm (summaries given by Martin, 1966a).

Animals

Under natural dietary conditions—Although the bromine content of animal tissues and milk is greatly influenced by the bromine in the feeds, we have no reports of bromine toxicity from feed sources.

Under man-induced dietary conditions—Bowen (1966) listed as lethal to rats 800 mg Br^+ in a 10-g/day dry-weight (80,000 ppm), duration not given.

Man

Dietary bromine is retained for only short periods in the tissues and is excreted mostly in the urine. Underwood (1971, p. 435) stated, "Human dietary bromine intakes have probably increased in recent years in areas where organic bromides are used as fumigants for soils and stored grains, and in motor fuels." However, there appear to be no toxicity problems associated with this intake. Thomas (1973) gave 500–5,000 mg/kg body weight of bromides (sodium, potassium, ammonium, and others) and 50–500 mg/kg body weight of bromate salts as the probable lethal oral dose for an adult.

CADMIUM

[Considered nonessential for plants and animals]

Plants

Under natural conditions—No toxicity has been demonstrated. Cadmium is a relatively rare element that is rather uniformly distributed in the most abundant rocks of the Earth's crust, which has an average content of 0.15–0.2 ppm (Fleischer and others, 1974). The only natural concentrations of commercial importance are those in sulfide deposits, especially those containing zinc, lead, and copper; and the toxicities of these elements in the deposits are most likely more severe than the toxicity of the low concentrations of the cadmium.

Under man-induced conditions—Brooks (1972) stated that the general toxicity of this element to plants was moderate. It has been noted (Allaway, 1968) that, in general, 3 ppm cadmium in the tissue of plants depressed growth. For example, Miller, Hassett, and Koeppe (1976) reported that growth depression occurred in soybeans when shoots had 3–5 ppm cadmium. Cadmium was reported to reduce plant growth at the following nutrient solution concentrations: 0.2 ppm, beets, beans, and turnips; 1 ppm, corn and lettuce; 5 ppm, tomato and barley; and 9 ppm, cabbage (Page and others, 1972). Traynor and Knezek (1973) reported that corn grown on cadmium-enriched soils readily absorbed and translocated the element. They also found growth reduction in corn at its maximum when 281 ppm cadmium was added to the soil resulting in a plant concentration of 131 ppm (ash weight basis). The concentrations of cadmium in soils that were required to damage plants were higher than even those in soils of contaminated areas (Fleischer and others, 1974).

Animals

Under natural dietary conditions—The cadmium content of plants under natural conditions is low (Shacklette, 1972), and various animal studies have suggested that the absorption of cadmium from the gastrointestinal tract is poor (Fleischer and others, 1974), therefore poisoning of animals is unlikely.

Under man-induced dietary conditions—Mammals have no effective mechanism for the elimination of ingested cadmium; with time it tends to accumulate in the liver and kidney. Its relative toxicity to mammals has been rated from moderate to high (Allaway, 1968). Cadmium from industrial or vehicular pollution can be absorbed by plants, or deposited on plant surfaces, to the extent that the plants are toxic to animals. For example, Goodman and Roberts (1971) reported that grass contaminated by industrial pollution contained 9.9 ppm cadmium, whereas control samples of the grass contained only 1 ppm; this contaminated grass caused the death of a horse (but the immediate cause of death was attributed to plumbism). They found that contaminated grass contained as much as 40 ppm cadmium in ash. Cadmium was suspected of killing a horse that contained 80 ppm cadmium in its liver and 410 ppm in its kidneys (Lewis, 1972). Thirty to sixty ppm cadmium in the diet of sheep for 191 days was shown to reduce growth and feed intake (Doyle and others, 1972). Forty-five ppm cadmium in the diet of rats for 6 months caused slight toxic symptoms (Underwood, 1971). Bowen (1966) stated that 0.5 mg/day Cd^{2+} in a total dry weight diet of 10 g (500 ppm) is toxic to rats, whereas 16 mg/day (1,600 ppm) is lethal, duration not given. A subtoxic level of 5 ppm cadmium in the drinking water of rats, given for 180-240 days, produced systolic hypertension. It has been used as an anthelminthic drug in swine and poultry at

concentrations ranging from 30 to 1,000 ppm in the diet (Stecher, 1968). Cadmium appears to be highly toxic to aquatic organisms at low concentrations, as follows: lethal to the flatworm *Polycelis nigra* at 2.7 ppm, to the stickleback fish at 0.2 ppm, to goldfish at 0.02 ppm, and to *Daphnia magna* at 0.003 ppm (Schroeder and Balassa, 1961).

Man

Under normal environmental conditions—The normal cadmium content of food plants is low, ranging from 0.1 to 1 ppm in dry material (Shacklette, 1972); therefore poisoning from this source is unlikely.

Under the effects of environmental pollution by cadmium—The first likely instance of cadmium poisoning in man due to general environmental contamination is that of itai-itai (ouch-ouch) disease in the elderly women who lived in the vicinity of a mine in Japan. It was demonstrated that the water, rice, and fish in the endemic area contained high concentrations of cadmium and other metals, probably due to contamination of the local river by the effluent from a zinc-lead-cadmium smelter (Fleischer and others, 1974). Friberg (1975) reported that, in 1972, Swedish school children showed severe gastrointestinal symptoms after the consumption of soft drinks made with water containing 16,000 μg/L cadmium. Bowen (1966) stated that the normal dietary intake of Cd^{2+}, based on a total dry weight diet of 750 g/day, is 0.6 (0.8 ppm), whereas 3 mg/day (4 ppm) is toxic, duration not given. Inhaled cadmium oxide fumes at a concentration of 1 mg/m^3 for 8 hours causes pulmonary edema, and 5 mg/m^3 for 8 hours is considered lethal (Bergqvist and others, 1977). A recent study of the environmental impact of cadmium (Fleischer and others, 1974, p. 299) concluded, "Based on present knowledge, and there are many gaps, there is no solid evidence of a hazard to the general population from cadmium in food, air, or water. Contamination of food and water rather than air would seem to present more of a potential problem. This might not be the case with smoking or excessive occupational exposures."

CHLORINE

[Essential for plants and animals]

Plants

Under natural conditions—The chloride ion is itself toxic if too greatly concentrated; also, if too concentrated, the common natural chlorine compound, sodium chloride, is toxic through its effect on osmotic pressure within the plant. Symptoms of excess chloride include burning and firing of leaf tips or margins, bronzing, premature yellowing and abscission of leaves and, less frequently, chlorosis (Eaton, 1966). Within the concentration ranges of sodium chloride tolerated by a species of plant, each increase in the osmotic pressure of

the substrate solution causes an increase in the osmotic pressure of the plant sap; beyond this range, lethal wilting occurs. Under natural conditions, areas inundated by seawater or strongly influenced by sea spray may accumulate toxic amounts of sodium chloride in the soil. Evaporative salt in closed geographic basins may also reach toxic concentrations. On natural undrained soil in Australia, the chloride in leaf petioles of grape (percent, dry matter) related to leaf toxicity symptoms were as follows: 0.81, no toxicity; 1.35, slight burning; 1.55, burning; and 2.50, severe burning (Woodham, 1956). The following concentrations (percent, dry matter) of chlorine in leaf tissue are considered excessive: >0.7, Navel and Valencia oranges (Embleton and others, 1976); >0.5, avocado (Jones and Embleton, 1976); >0.2, apricots; >0.3, almonds, apples, peaches, pears, plums, prunes, and walnuts; >0.5, olives (Beutel and others, 1976); and >0.5, grapes (Cook and Wheeler, 1976).

Under man-induced conditions—Chloride toxicity may be produced in plants by over fertilizing, such as with potassium chloride. Chloride toxicity may also result from irrigation using slightly salty water with insufficient flushing of the soil. Plants range widely in their resistance to toxicity, from the halophytes such as asparagus to highly sensitive species. Experimental studies in which grapefruit trees were watered with different levels of salt concentration in the water gave the following results (Pearson and others, 1957): 300 ppm salt, relative weight of tops was 100, 0.07 percent (in dry matter) in leaves; 1,300 ppm salt, relative weight of tops was 46, 1.28 percent in leaves; and 2,300 ppm salt, relative weight of tops was 38, 1.35 percent in leaves. The two higher concentrations produced defoliation, dieback, chlorosis, bronzing, and burning.

Animals

Under natural dietary conditions—Forage plants in general are so low in sodium chloride that salt must be provided for herbivorous animals; therefore, natural chlorine toxicity in these animals is very unlikely.

Under man-induced dietary conditions—Our only report is one given by Bowen (1966) who listed as lethal to rats >900 mg Cl^- in a 10-g/day dry-weight diet (90,000 ppm), duration not given.

Man

We have no reports of human toxicity attributed to excessive chlorine in the natural environment, although chlorine gas is highly toxic to plants, animals, and man. Sax (1975) gave the following toxicity ranges for chlorine gas: 3.5 ppm—detectable odor; 15 ppm—immediate irritation of the throat; 50 ppm—dangerous; 1,000 ppm—fatal even with short exposure. Thomas (1973) gave 50–500 mg/kg body weight as the probable lethal dose of chlorate salts for an adult.

CHROMIUM

[Considered nonessential for plants; essential trace element for animals]

Plants

Under natural conditions—The growth of only certain species on "serpentine soils" has commonly been attributed to the high chromium levels in such soils (Brooks, 1972); but excessive nickel, unfavorable Mg:Ca ratios, and deficiency of molybdenum may also contribute to this condition (Vanselow, 1966a). Soil from "poison spots" in Oregon contains as much as 2–3 percent chromic oxide (McMurtrey and Robinson, 1938), whereas in Maryland, infertility of the soil was associated with 1,000–3,900 ppm chromic oxide (Vokal and others, 1975). The concentrations (dry weight) of chromium in plants growing in serpentine soils and showing toxicity symptoms follow: 18–24 ppm in leaves and 375–410 ppm in roots of tobacco; 4–8 ppm in corn leaves; and 252 ppm in oat leaves (National Research Council, 1974a). Vokal and others (1975) wrote, "The concentration of chromium usually present in the air (traces to 0.02 $\mu g/m^3$ as Cr) are too low to have any significant effect on the growth or yield of vegetation."

Under man-induced conditions—Brooks (1972) reported chromium toxicity to plants as severe, whereas Allaway (1968) stated it to be moderate. Chromium in the form of chromates is particularly toxic (McMurtrey and Robinson, 1938). In soil, 1,370–2,740 ppm chromium caused chlorosis in citrus; 10-15 ppm chromium (as $K_2Cr_2O_7$) in the nutrient solution was toxic to barley (Mertz, 1974a); 150 ppm in soil was toxic to orange seedlings; 16 ppm (chromate) reduced growth in tomatoes, oats, kale, and potatoes whereas 10 ppm was toxic to corn and 5 ppm was toxic to tobacco (National Research Council, 1974a). In corn leaves, 4–8 ppm (dry weight) chromium was toxic (Soane and Saunder, 1959). Pratt (1966c, p. 136) stated that because of their toxic effects, the use of chromium salts should be avoided in all plant-production media. Wallace, Alexander, and Chaudhry (1977) reported: "Plants grown with $10^{-5}N$ Cr_2O_7 were decreased in yield by about 25% . . . while the same level of $Cr_2(SO_4)_2$ was essentially without effect. For the two salts, the leaf, stem, root concentration for Cr, respectively were 2.2 and 1.3, 0.7 and 0.7, and 140 and 104 $\mu g/g$." In aquatic plants, hexavalent chromium at 0.03–65 ppm has been shown to inhibit the growth of freshwater algae; in seawater, 1–5 ppm reduces photosynthesis in kelp (National Research Council, 1974a).

Animals

Under natural conditions—Chromium as a metal is considered biologically inert. Underwood (1971, p. 263-264) stated, "Chromium, particularly trivalent chromium, has a low order of toxicity. A wide margin of safety exists between the amounts ordinarily ingested and

those likely to induce deleterious effects." Allaway (1975) notes that even though $Cr^{(VI)}$ is more toxic to plants and animals and is more likely to be soluble and mobile in soil and waters than is $Cr^{(III)}$, no naturally occurring minerals contain $Cr^{(VI)}$. Further, he stated (p. 37) that ". . .minerals contain $Cr^{(III)}$, especially mixed oxides of $Cr^{(III)}$ and Fe, are generally inert and resistant to weathering."

Under man-induced dietary conditions—The hexavalent form of chromium may be 100 times more toxic than the trivalent form; however, both are relatively nontoxic to mammals and other vertebrates (Schroeder and others, 1962a). Underwood (1971) stated that 50 ppm chromium in the diet resulted in growth depression of experimental animals. Thirty to forty mg/kg of body weight as zinc chromate is lethal to calves within 1 month, and about 20 times that amount is lethal to cows. Thirty ppm chromium in the liver and 4 μg/ml in whole blood is diagnostic of chromium toxicity (Harrison and Staples, 1955). Five mg chromate in a 10-g/day dry-weight diet (500 ppm) is toxic to rats, duration not given (Bowen, 1966). Chromium is potentially carcinogenic (National Research Council, 1974a).

Man

"Chromium as a metal is biologically inert and does not produce toxic or other harmful effects in man or laboratory animals" (National Research Council, 1974a, p. 42). People who work with hexavalent chromium have, however, developed cutaneous and nasal mucous-membrane ulcers and contact dermatitis. The lung is the only organ that appears to accumulate chromium, and this may be related to airborne exposure to chromium-containing dust (Mertz, 1967). Bowen (1966) reported that in a 750-g dry-weight diet, normal consumption of chromate is about 0.05 mg (0.07 ppm), whereas the toxic level is 200 mg (270 ppm) and the lethal level is 3,000 mg (4,000 ppm). Allaway (1975) concluded that since there have been no reports of toxicity to people or animals from the consumption of $Cr^{(III)}$, chromium in plants represents an unlikely potential hazard. Schroeder, Balassa, and Tipton (1962a) reported that consumption of water containing 1.0–25 ppm chromium by a family over several years produced no apparent ill effects. They further stated that chromium is one of the least toxic of trace metals.

COBALT

[Essential trace element for blue-green algae and nodule bacteria; considered nonessential for higher plants, although growth and yield increases have been reported; essential trace element for animals]

Plants

Under natural conditions—Vanselow (1966b) reported that cobalt produces toxicity to plants when the amounts available to the plant exceed certain low levels, but that a naturally occurring excess of

cobalt in soils is improbable and that no instances so far had been noted. Plant species range widely in their content of cobalt, yet show no toxicity symptoms. Some trees have very high concentrations in their leaves; black gum leaves, for example, were reported to contain as much as 10,000 ppm in ash and persimmon leaves, 2,000 ppm; most other species contain much less (Connor and Shacklette, 1975).

Under experimental conditions—A number of authors (for example, Vanselow, 1966b) have reported that small amounts of cobalt in solution cultures, sometimes as small as 0.1 ppm, produced adverse or toxic effects on many crop plants. The symptoms of cobalt excess include depressed growth, chlorosis, necrosis, and even death of the plant. The chlorosis is frequently described as resembling that of iron deficiency (Vanselow, 1966b). From the literature, Vergnano and Hunter (1952) reported the following examples of cobalt toxicity to plants growing in nutrient solutions: maize and beans, 2–8 ppm; oats, 3–300 ppm; wheat, 6–590 ppm; barley and beans, 1–15 ppm; flax, 0.5–5 ppm; and oats and sugar beets, 15–30 ppm. Their own studies of cobalt toxicity to oats revealed symptoms ranging from slight chlorosis and occasional necrosis (5 ppm in nutrient solution) to stunted and severe necrosis (50 ppm). Sudan grass containing 19–32 ppm (dry weight) cobalt showed toxicity symptoms when grown in soils to which 908 kg of cobalt per 0.4 ha had been added (Fujimoto and Sherman, 1950).

Animals

Under natural dietary conditions—We have no reports of cobalt toxicity attributed to consumption of natural feedstuffs; pasturage deficiency in cobalt is of far greater concern than are potentially toxic concentrations in plants. Kubota (1967, p. 128) stated, ". . . there are no known areas of cobalt toxicity in the United States as there are areas of selenium toxicity."

Under man-induced dietary conditions—Cobalt is relatively nontoxic. Sheep can tolerate large doses of cobalt; 0.35 g/100 kg/day produced no ill effects (Becker and Smith, 1951). Smith (1962) listed the following in mg/kg body weight/day: rats, 2.5, nontoxic; dogs, 10, nontoxic; sheep, 3, nontoxic; and cattle, about 1, toxic. Bowen (1966) listed 0.7 mg Co^{2+} as toxic to rats in a 10-g/day dry-weight diet (70 ppm), duration not given. A concentration of 0.1 mg/m^3 of metallic cobalt powder inhaled by miniature pigs for 6 hours daily, 5 days/week is considered toxic (Hellsten and others, 1976).

Man

The edible parts of food plants contain only <1–10 ppm (ash base) cobalt, therefore toxicity attributed to this source is very unlikely (Connor and Shacklette, 1975). As with animals, the toxicity to man of nonradioactive isotopes of cobalt is of little importance. Hazards associated with the therapeutic use of this element were given (Hellsten

and others, 1976, p. 20) as follows: "Poisoning by ingestion has mainly occurred in connection with the medical use of cobalt in the treatment of certain anemias. In these therapies the daily intake varied between 25–30 mg cobalt, which is about 100 times the normal dietary intake." At a time when cobalt was used in beer in order to preserve the foam, it was suspected of causing the "beer drinkers cardiomyopathy" syndrome. Some evidence (Burch and others, 1973), however, implicates selenium in conjunction with cobalt as being responsible. Bowen (1966) listed 500 mg Co^{2+} as toxic in a 750-g/day dry-weight diet (670 ppm), duration not given.

COPPER

[Essential trace element for plants and animals]

Plants

Under natural conditions—Soil influenced by a high degree of copper mineralization may be toxic to native or cultivated plants. Fraser (1961) reported that large trees were absent and tree seedlings were killed, in a syngenetic copper deposit in a swamp in New Brunswick; he further reported that muck from this swamp killed cucumber plants to which it was applied. There are many reports in the literature of copper toxicity to plants on or near copper deposits, and the degree of resistance to copper toxicity by different species has been used as a geobotanical method of mineral exploration. One report by Malaisse and others (1978) gave the unusually high value of 1.3 percent copper (dry weight base) in the tissue of a copper-mineralization indicator plant *Aeolanthus biformifolius* (Mint Family) from Zaïre. Areas in Norway where vegetation had been poisoned (containing as much as 895 ppm copper in dry material) by naturally-occurring high copper levels in soils (as much as 7,400 ppm) could be detected by examining satellite photographs (Bølviken and others, 1977). The following concentrations (ppm, dry matter) of copper in leaf tissue are considered excessive: probably >22—Navel and Valencia oranges (Embleton and others, 1976); and >0.25—avocados (Jones and Embleton, 1976).

Under man-induced conditions—Copper sulfate (blue vitriol) has been used for more than seventy years as an algicide at an achieved concentration of 1 mg/L (1 ppm) for the upper 0.5 m of water (Mackenthun and Ingram, 1967). Concerning the toxicity of copper to vascular plants, Bennett (1971, p. 675) wrote, "Copper is highly toxic to roots, and toxic concentrations are translocated only negligibly via the stems. Thus Cu is a widely used toxicant for destroying roots that have encroached into sewer lines and septic tank fields." Most copper toxicites in crop plants have been caused by the use of Bordeaux mixture

(copper sulfate and lime) as a fungicide. Toxicity to copper in plants is usually manifested by chlorosis (yellowing) of the foliage, caused by the interference of excessive copper with iron metabolism in the plant. Reuther and Smith (1953) found appreciable chlorosis of citrus seedlings occurring when the total copper of a very sandy soil of pH 5.0 or below exceeded 150 ppm. Westgate (1952) reported copper toxicity in several crops on old vegetable fields having over 400 ppm total copper in the topsoil as a result of many years of Bordeaux mixture spraying of celery. Copper is added to the environment by industrial pollution and vehicular emissions (Shacklette and Connor, 1973), but we have no reports of copper toxicity attributed to these sources. The National Research Council (1977b) reported that toxicity to plants has been noted in areas receiving manure from swine and poultry that were given diets rich in copper (250 ppm). Copper deficiencies in agricultural soils are much more common than copper excesses; and fertilization with copper, especially on highly organic or very sandy soils, is a common practice.

Animals

Under natural dietary conditions—Dick (1956) reported that when sheep graze on pastures having normal copper but low molybdenum content (less than 0.1 ppm), copper accumulates in their livers. This accumulation sometimes results in chronic copper poisoning, followed by death. On the other hand, when pasture or forage contains a normal copper content but high molybdenum (5 ppm or more), a poisoning having many of the clinical symptoms of copper deficiency may occur. The average (geometric mean) copper content of U.S. soils was reported as 18 ppm, with a range of <1 to 300 ppm, and most soil samples contained <3 ppm molybdenum, with high values ranging from 3 to 7 ppm (Shacklette and others, 1971).

Under man-induced dietary conditions—Hemkes and Hartmans (1973) found that sheep feeding on either side, and within 20 m of, high-tension copper power lines became ill and died after consuming dry forage and soil containing copper in excess of 15 ppm. Sheep appear to be particularly sensitive to copper intoxication. Case (1974, p. 278) stated "Ingestion of as little as 1.5 g [copper]/sheep/day for 30 days was fatal to most sheep of the British breeds, an amount easily obtained from hog rations supplemented with copper sulfate. . ." Chronic copper poisoning of animals may occur as a consequence of excessive consumption of copper-containing salt licks or mixtures, or the unwise use of copper-containing drenches. Contamination of feeds with copper compounds from horticultural or industrial sources is a further cause. In all animals the continued ingestion of copper in excess of requirements leads to some accumulation in the tissues, especially in the liver (Underwood, 1971). These liver accumulations were reported by Case

(1974) to range from 247 ppm to >6,000 ppm in sheep dying of copper intoxication, with >500 ppm usually indicative of poisoning. Copper toxicity in mammals, compared to other animals, is of little significance because they possess barriers to copper absorption. Avian and mammalian resistance to copper is 100 to 1,000 times greater than is that of more primitive animals (Schroeder, Nason, Tipton, and Balassa, 1966). For aquatic organisms copper is the most toxic common heavy metal; this toxicity, however, is inversely related to the water hardness (principally calcium content). Various copper compounds (copper sulfate, copper pentachlorophenate, copper tartrate, and others) have been used since the 1920's as molluscicides in the control of schistosomiasis (National Research Council, 1977b). Bowen (1966) reported that a 20 mg Cu^{2+} (2,000 ppm) ration in a dry-weight diet of 10 g/day was lethal to rats, duration not given.

Man

Under normal environmental conditions—Underwood (1971, p. 106) stated, "Continuous ingestion of copper from food or water supply at intakes sufficient to induce chronic copper poisoning in man is extremely unlikely, judging by the amounts required for this purpose in other monogastric species." From a normal intake of 2–5 mg/day (Bowen, 1966), a net balance of zero copper is normally maintained. The National Research Council (1977b) reported that 800 μg/L in the drinking water of a 15-month-old infant may have caused acrodynia (pink disease).

Under excessive copper intake—Excessive buildup of copper in the liver and central nervous system caused by a metabolic inability to excrete copper is the fundamental cause of Wilson's disease (Scheinberg, 1969). Copper toxicosis in humans (and most higher animals) is not, however, a major problem. As stated by Hill (1977, p. 126) ". . . the ingestion of excessive copper salts leads immediately to vomiting and the expulsion of the copper. The highly reactive ions of copper result in complex formations in nature so that the free ion is seldomly encountered." Bowen (1966) reported that 250–500 mg Cu^{2+} (330–670 ppm) in a dry-weight diet of 750 g/day is toxic to normal individuals, duration not given. Soluble copper salts, notably copper sulfate, are strong irritants to the skin and mucous membranes (Stecher, 1968).

FLUORINE

[Considered nonessential for plants; essential trace element for some higher animals, improves teeth and bones]

Plants

Under natural conditions—Toxicity is unusual, but plants growing in acid soils have accumulated toxic levels of fluorine (Allaway, 1968). The amount of fluoride taken up from the soil by plants is usually

unrelated to total soil fluorine, but is controlled by soil type, calcium and phosphorus content of the soil, and soil pH (Brewer, 1966a). Brewer (1966a, p. 182) reported, "With few exceptions, the usual fluoride content of most plant foliage growing in areas removed from possible sources of fluoride air pollution is in the range of 2 to 20 ppm on a dry-weight basis. Notable exceptions include tea leaves, which have been found . . . to contain, on the average, 50 to 90 ppm of fluorine. The highest fluoride concentrations reported for vegetation, presumably attributable to fluorine absorption from the substrate, include 1,900 ppm in camellia leaves . . . and in excess of 8,000 ppm in tea leaves . . . ." Mitchell and Edman (1945, p. 82) reported, "Very few foods contain less than 0.1 ppm of fluorine on the fresh basis, and few foods uncontaminated with the residues of insecticidal sprays and fluoriniferous soils and dusts contain more than 2 ppm." They also stated that the maximum *chlorine* [apparently a misprint of fluorine] permitted in vegetables is 2.8 ppm and in apples and pears, 7 ppm. Embleton, Jones, and Platt (1976) reported that >100 ppm (dry matter) fluorine in the fruiting shoots of Navel and Valencia oranges is probably an excessive amount.

Under man-induced conditions—Vegetation in the vicinity of superphosphate and elemental phosphorus plants is often injured by fluorine emissions (for example, Georgetown Canyon, Idaho, in U.S. Department of the Interior and U.S. Department of Agriculture, 1977), and plants growing on soil that has been heavily fertilized with phosphate fertilizers may also receive toxic amounts of fluorine from the soil. However, most toxicity problems have been associated with industrially polluted air as the major source of fluorine, and this source is indicated by abnormally high fluorine content in the tops of plants and with a corresponding low content in the roots. Depending on species and growth conditions, 30–300 ppm fluorine (dry weight) generally reduces growth (National Research Council, 1971). Injury symptoms are leaf-margin necrosis, interveinal chlorosis, or both. As a result of fluoride emissions from a factory in Czechoslovakia, Marier (1968) reported that tree leaves within a 5-km distance were necrosed, had a decreased chlorophyll content, and contained 7–72 times more fluorine than that normally found. Vegetables and fruit were disfigured in shape and color, and contained 5–21 times the fluoride of control samples.

Animals

Under natural dietary conditions—Animals obtain their fluorine from the plants that they eat and the water that they drink (Brewer, 1966a). The fluorine levels of common uncontaminated feed materials are too low to constitute a fluorine hazard to livestock, but endemic fluorosis has been reported from areas where the drinking water con-

tains unusually high fluorine content (Underwood, 1971). The following classification of fluorine concentrations in water relative to the health of lambs was given (Case, 1974): 2 ppm, slightly toxic; 5 ppm, definitely toxic; and 10 ppm, likely to cause serious damage. Further, the recommendation was made that rations for lambs should not contain more than 100 ppm fluorine (dry matter). Lethal fluorosis of sheep and other livestock, caused by consuming contaminated vegetation and water, occurred following a volcanic eruption in Iceland (Thorarinsson, 1970). Some plants (particularly members of the Leguminosae) have the capability of synthesizing organo-fluoride compounds (specifically fluoroacetate and fluorocitrate) from inorganic sources. Although not particularly toxic to plants, these compounds are very toxic to grazing animals and have caused numerous ungulate deaths in Africa and Australia (Lovelace and others, 1968).

Under man-induced dietary conditions—Herbage and water supplies of animals that have been contaminated by dust from the mining of rock phosphate deposits have caused chronic fluorosis. Whereas surface waters usually contain less then 1 ppm fluorine, water from deep wells in endemic fluorosis areas contains 3–5 ppm fluorine and often 10–15 ppm (Underwood, 1971). Bowen (1966) reported that 0.1 mg F^- in a 10 g/day dry weight diet (10 ppm) is toxic to rats, whereas 30 mg (3,000 ppm) is lethal, duration not given. The concentrations of fluorine in dietary dry matter, above which normal performance may be affected, follow: beef or dairy heifers, 40 ppm; horses, 60 ppm; finishing cattle, 100 ppm; broiler chickens, 300 ppm; breeding hens, 400 ppm; and turkeys, 400 ppm (National Research Council, Committee on Animal Nutrition, Subcommittee on Fluorosis, 1974). The following conclusion regarding fluorine toxicosis in bovines is given by Church and others (1971, p. 511): ". . . long-term ingestion of fluorine at a rate much above 30 ppm will result in symptoms of fluorosis after a period of years. Ingestion of appreciably larger amounts (>100 ppm) can be tolerated for time periods of several months without undue hazard." These authors further stated that fluorine concentrations above 5,500–7,000 ppm (ash base) in bone are considered an indication of poisoning. Case (1974) reported that sheep fed 10 mg sodium fluoride/kg of body weight developed acute fluorine toxicosis; the likely lethal dosage was given as 15 mg/kg.

Man

Under natural environmental conditions—Food is the major source of fluorine to individuals not exposed to industrial contamination or to naturally or artificially fluoridated drinking water. Very few foods contain more than 1–2 ppm fluorine, and most of them contain less than 0.5 ppm (dry matter). The consumption of tea can be an important determinant of total dietary fluorine intake, as fluorine concentration

of 100 ppm in tea leaves are common, two-thirds of which passes into the infusion, so that one cup of tea can add 0.1–0.2 mg fluorine to the diet. Endemic fluorosis from natural waters from deep wells having 20–50 ppm fluorine has been noted as resulting in mottled teeth and bone deformities (Underwood, 1971). Mitchell and Edman (1945, p. 87) reported that, in humans, chronic fluorosis is generally caused by ingestion or inhalation of fluorine-containing gases and dust. Bowen (1966) listed 0.5 mg F^- in a 750-g/day dry-weight diet (0.7 ppm) as the normal dietary intake. Consumption of 20 mg (27 ppm) was considered toxic and 2,000 mg (2,700) was lethal, duration not given.

Fluoridated drinking water—Community water supplies commonly are treated with sodium fluoride or fluorosilicate to maintain fluoride levels ranging from 0.8 to 1.2 ppm, in order to reduce the incidence of dental caries; at concentrations much above these levels, some tooth mottling may occur (Underwood, 1971).

IODINE

[Considered nonessential for plants; essential trace element for animals]

Plants

Under natural conditions—Although iodine is highly toxic to plants, it usually is present in very small amounts in an available form in soils and, therefore, seldom presents a toxicity problem (Martin, 1966b). The iodine content of land plants varies with the species and the amount in the soil. Shacklette and Cuthbert (1967) reported the following average iodine concentrations (ppm, dry matter) for groups of land plants: fleshy fungi, 6.2; ferns, 5.7; coniferous trees, 3.9; monocotyledons, 5.9; dicotyledonous trees, 2.7; dicotyledonous vegetables (edible parts), 6.9. Iodine in marine brown algae ranged from 55 to 8,800 ppm.

Under man-induced conditions—Large applications of kelp (marine brown algae) to soil as fertilizer may cause iodine toxicity to sensitive plants (Martin, 1966b). Adding 2.5 ppm potassium iodide to soil has injured crop plants. In nutrient solutions and sand cultures, iodine is toxic to plants at concentrations greater than about 0.5–1 ppm (Martin, 1966b).

Animals

Under natural dietary conditions—We have no reports of iodine toxicity in animals fed normal rations. The iodine levels in vegetation in some areas are inadequate for animal requirements, and iodine supplements must be used in the feed.

Under man-induced dietary conditions—In experimental studies, significant species differences in tolerance to high intakes of iodide were found; however, in all species studied, the tolerance was high in comparison with normal dietary iodine intakes, indicating an ex-

tremely wide margin of safety for this element. Hens fed 312–5,000 ppm potassium iodide in their ration ceased egg production. High levels also increased embryonic mortality in rats and rabbits (Underwood, 1971).

Man

Under natural environmental conditions—Prolonged intake of large amounts of iodine by normal individuals markedly reduces thyroidal iodine uptake—the antithyroidal or goitrogenic effect. In an area in Japan, the consumption of large quantities of iodine in the diet was reported as the cause of endemic goiter (Underwood, 1971). However, areas that are deficient in iodine are far more common than areas that have excesses, and iodine supplements to foods are widely used.

Iodine tolerances—Bowen (1966) listed 0.2 mg I^- in a 750-g/day dry-weight diet (0.3 ppm) as normal, whereas 10,000 mg (13,300 ppm) was toxic, duration not given. Thomas (1973) gave 5–50 mg/kg body weight as the probable lethal dose of iodine for an adult.

LEAD

[Nonessential for plants and animals]

Plants

Under natural conditions—An area in Montana was reported (Mudge and others, 1968) where weathering of Devonian carbonate has produced a thin soil enriched in lead, containing >5,000 ppm lead in the A-, B-, and C-horizons. The pine trees of the area of known lead mineralization were dwarfed and misshaped, and their needles were yellow-green. Analyses of these trees were not given. The occurrence of naturally lead-poisoned soil in Norway was described (Låg and others, 1969) in which the vegetation was exceptionally sparse in an area about 100 m^2 that was believed to have been influenced by solutions from the weathering of galena-bearing quartzite. The richest soil samples contained 11 percent lead, and the maximum concentration in plants was 0.9 percent in dry material. Apparently normal vegetation in this area contained around 0.1 percent, and sickly vegetation contained about 0.3 percent in dry matter. The differences in tolerance of lead among the species at or near the site were indicated by the absence of common species in the lead-rich area. Lead, zinc, and other heavy metals often occur together in mineralized outcrops; therefore, it is difficult to associate observed toxicity symptoms in plants at these sites with lead alone. Plants at these sites may absorb large amounts of lead without exhibiting toxicity symptoms; concentrations in stems of certain shrubs may be as high as 350 ppm lead in ash of the samples (Shacklette, 1960) without producing visible toxicity.

Under man-induced conditions—Elevated levels of lead in plants affected by automotive and industrial contamination have been extensively reported. We have no reports that conclusively demonstrate lead toxicity to the plant from such contamination. Frank toxicity of lead to plants has been shown, however, by laboratory and field experiments. Prát (1927) studied uptake of lead from lead chloride solutions; he found that broadbean plants absorbed all the lead in less than 3 days, but grew very little as a result. Hooper (1937) found that French beans growing in solution cultures were damaged by 30 ppm of lead as the sulfate alone or in combination with nutrient salts. Plants sprayed with lead sulfate solutions were not injured unless sufficient lead residue was left to block the stomates. Wilkins (1957), using lead nitrate as the nitrogen source, grew sheep fescue in solution cultures and reported that root growth was measurably retarded with 10 ppm of lead, markedly reduced by 30 ppm, and stopped at 100 ppm. Miller and Koeppe (1970) showed that 60μmoles of lead nitrate added to a nutrient solution retarded growth of young corn plants when phosphate was deficient. (In young leaves the concentration of lead was 115 ppm in dry weight.) At 600μmoles and greater, marked reduction in growth of corn was noted with or without sufficient phosphate. (Concentration of lead in young leaves was 306 ppm, dry weight.) The use of lead arsenate as an insecticide in orchards for long periods has caused high lead concentrations in these soils. Regarding the toxicity of these soils, most of the observed damage to subsequent plant growth can be attributed to the resulting arsenic concentrations. Connor, Shacklette, and Erdman (1971) found 2 percent lead in ash of a red cedar tree that had been contaminated by dust from passing ore-carrying trucks; washing the samples with different solvents did not appreciably reduce the lead content. This tree showed no toxicity symptoms. Most of the lead in soils is sparingly soluble and largely unavailable to plants. Brewer (1966b) reviewed the reports of soluble lead in many different soils and found most soils to contain less than 1 ppm. Lead levels in epiphytic plants may be very high, although these plants have no root contact with the soil. Spanish moss believed to have been contaminated by industrial or vehicular pollution was reported to contain as much as 5 percent lead in ash (Shacklette and Connor, 1973). We have found some tree-growing pendant lichens (*Usnea* and *Alectoria* spp.) that contain 1.5 percent lead in ash, even though collected from areas having no obvious source of airborne pollution. None of these epiphytes exhibited evidence of toxicity.

Animals

Under natural dietary conditions—McMurtrey and Robinson (1938, p. 821) wrote, "Normally the quantities [of lead] in edible plants or parts thereof are so small as to have no effect on the health of the

animal eating the plant." Shacklette (1962b) estimated that a large browsing animal eating native tree and shrub foliage in Alaska might ingest from 1 to 4 g of lead annually, depending on the region of the State. The report on natural lead accumulation in Norwegian soils and plants (Låg and others, 1969, p. 151) stated, "Since lead is toxic to many animal organisms, even in small amounts, such concentrations as found in this case may have an injurious effect on fauna." Examples of this possible toxicity, however, were not given.

Under man-induced dietary conditions—Lead from industrial operations and vehicular traffic may be absorbed by, or accumulated on, forage plants to the extent that the plants are toxic to grazing animals. Lead poisoning in cattle and horses that grazed near smelters has been reported in Germany by Hupka (1955), in the United States by Dorn and others (1972), in Ireland by Egan and O'Cuill (1970), and in Canada by Schmitt and others (1971). The latter authors reported that excessive amounts of lead in ingested forage was the primary cause of a chronic debilitating disease of six young horses and less acute, but similar, disorders in 25 older horses. Cattle were less seriously affected. The lead values in the pasture grasses were as follows (ppm, dry weight basis): spring, 1–46; midsummer, 4–310; fall, 4–435; and overwintered grass, 25–2,800. Animals in the Staten Island Zoo, ranging from reptiles to primates, suffered acute lead poisoning attributed largely to intake of lead from atmospheric fallout (Bazell, 1971). Schroeder, Balassa, Gibson, and Valanju (1961, p. 417) stated, "It[lead] is not as toxic orally for mammals as cadmium and mercury, but is more so than copper, manganese, silver, vanadium, zinc, chromium, molybdenum, cobalt, nickel and probably arsenic." They also reported that 0.01–1.0 ppm lead (chloride) immobilized *Daphnia magna* and that 0.1–50 ppm was lethal to various fishes. For comparison, they noted that this was the range of toxicity to these organisms for zinc and nickel. Migratory waterfowl have been reported to have been poisoned by ingesting lead shot that were lodged in bottom sediments (Lisk, 1972). Many other reports could be cited of poisoning of animals by lead that was introduced into the environment by human activity.

Man

Under natural environmental conditions—Lead poisoning from natural sources is very unlikely. Brewer (1966b, p. 214) commented, "The extremely low lead content of edible parts of the majority of crops is reassuring, in light of the potential toxicity of lead to animals, including man." Poisoning of man has occurred when drinking water containing 0.18 to more than 1.0 ppm Pb^{2+} (0.4–2.5 mg daily, in addition to lead in food) has been consumed over an unspecified period of time (Schroeder and others, 1961). Water should not exceed 0.05 ppm lead (World Health Organization, 1963). "Normal" rural air contains about

0.05 μg lead per cubic meter (Patterson, 1965). However, it should be pointed out that very few humans today live in a natural, unpolluted environment.

Lead intakes and toxicities—The average daily intake of lead from food and beverages in the United States is now estimated at about 0.30 mg, with only about 10 percent of this being absorbed from the intestinal tract. In addition, some lead is inhaled and absorbed through the respiratory tract; the relative importance of this respiratory exposure is a matter about which there is some dispute (Goldsmith and Hexter, 1967). These authors concluded that the absorption of lead from the two sources may be of similar magnitude. In another study (Rabinowitz and others, 1973), in which a healthy man was fed a diet normal in lead content and labeled with ^{204}Pb it was concluded that about two-thirds of his assimilated lead was dietary in origin and the remainder was inhaled. Studies have generally shown that less than 10 percent of ingested lead is absorbed, whereas 25–50 percent of inhaled lead is absorbed (Goldsmith and Hexter, 1967). The possible physiological significance of environmental lead was discussed at length by Warren (1974), who listed the sources of lead insult as air, dust, leaded paint, cigarette tobacco, beer and wine, drinking water, food, fertilizers (particularly, sewage sludge), leaded gasoline, and miscellaneous, which included smelters, vegetables grown over refuse dumps, glazed pottery, plastics, solders, and fillers in alkaline piping. Bowen (1966) reported that the normal consumption of Pb^{2+} in a 750-g/day dry-weight diet was 0.3–0.4 mg (0.4–0.5 ppm) and that a consumption of 10,000 mg (13,300 ppm) on the same basis may be considered lethal, duration not given. Schroeder, Balassa, Gibson, and Valanju (1961b) speculated that poisoning in man would be eventually achieved if 1 or 2 percent of the total body content of lead (80 mg) were ingested daily (0.8–1.6 mg). Patterson (1965) noted that a concentration of Pb^{2+} in 0.5–0.8 ppm in the blood is the threshold for acute lead poisoning. Friberg (1975) reported that 53 percent of 1 to 9-year old children living near a lead smelter in El Paso, Tex., were found to have blood lead values above 40 μg/100 ml (0.4 ppm).

LITHIUM

[Considered nonessential for higher plants and animals, but an essential trace element for some micro-organisms]

Plants

Under natural conditions—Naturally occurring instances of lithium toxicity to plants are not known, except in the case of citrus (Bradford, 1966b), which are very sensitive to small amounts of lithium. (Embleton and others, 1976, reported that >35 ppm, dry weight, lithium in leaf tissue of Navel and Valencia oranges is probably an excessive

amount.) However, a report on lithium in the environment (Mertz, 1974b, p. 40) stated, "Crops grown in closed basins in California, Nevada, and Arizona are exposed to toxic levels of lithium." Lithium excesses occur most commonly in soils derived directly from igneous rocks rich in ferromagnesian minerals, and in soils derived from sedimentary deposits rich in clays or micas (Bradford, 1966b). The lithium concentration in 912 samples of soils from throughout the conterminous United States was reported (Shacklette and others, 1971) as follows: range, 5–136 ppm; arithmetic mean, 24.7. Bradford (1966b, p. 221) stated, "There is no evidence available to indicate that total lithium in soils is in any way related to plant availability. . . . Plant content of this element is at present the best guide to the lithium status of the soil."

Under man-induced conditions—Many crops are susceptible to injury when lithium is applied to the soil in the form of soluble salts (McMurtrey and Robinson, 1938), and many plants are tolerant of high lithium levels (Mertz, 1974b). Lithium was toxic to citrus when concentrated (as Li_2SO_4) in soil to 2–5 ppm, and when concentrated in leaves to 140–220 ppm in dry weight (Aldrich and others, 1951). Lithium toxicity has also been observed in avocado, celery, corn, olive, and wheat. In contrast, cotton seems to be very resistant to lithium toxicity, even to as much as 2,270 kg of lithium nitrate per hectare. Lithium content in citrus leaves ranging from 4–40 ppm in dry weight has caused moderate to severe toxic effects (Sauchelli, 1969). Beans grown in culture solutions were sensitive to 0.5×10^{-3} *M* lithium, which resulted in 10 ppm lithium in leaves, 48 ppm in stems, and 24 ppm in roots. Higher concentrations of lithium produced marked reductions in plant yield accompanied by increased lithium concentrations in leaf, stem, and root tissues (Wallace, Romney, Cha, and Chaudhry, 1977). Lithium ions are toxic to various lactic acid bacteria (Voors, 1969).

Animals

We have no reports of lithium toxicity in animals.

Man

Voors (1969, p. 1339) stated, "From the available data on lithium levels of commonly used foods and drinks it can be concluded that, in general, the daily lithium intake through drinking water exceeds the intake through foods. It has to be conceded, however, that there is incomplete knowledge concerning the presence of accumulator plants in human food." Our own data on lithium in fruits and vegetables from areas of commercial production in the United States show striking regional differences in concentrations of this element. Among field-collected samples of these food plants, the lithium content of those from Michigan, New York, New Jersey, and Florida was below the detection limit (<4 ppm in ash), whereas samples from Texas and States west-

ward commonly contained measurable, often high (as much as 28 ppm in California oranges), concentrations. For example, the concentration in snap bean samples from the Eastern States was <4 ppm, while in those from California it ranged from 5 to 27 ppm in ash. Toxic symptoms that have been reported at the pharmacological-dose level in humans include development of goiter and congenital malformations (Mertz, 1974b). Massive doses of lithium were reported to reduce aggressive behavior in humans and other vertebrates (Levy, 1968). Bowen (1966) gave 200 mg/day Li^+ in a 750-g dry-weight diet (270 ppm), duration not given, as the toxic dose in humans.

MANGANESE

[Essential trace element for plants and animals]

Plants

Under natural conditions—Berger and Gerloff (1947) reported that acid soils (pH 5.0) mobilized manganese and that soil solutions containing >2.0 ppm soluble manganese were toxic to potatoes in Wisconsin. An acid soil (pH 4.8) with an ammonium acetate-extractable manganese concentration of 7.8 ppm was toxic to cotton in Virginia (Foy and others, 1969). Morris (1949) found that the exchangeable manganese content of 25 naturally acid soils varied from 1.2–638 ppm. Lespedeza and sweet clover grown on these acid soils made poor growth, contained high concentrations of soluble manganese, and showed typical symptoms of manganese toxicity. In general, plants having more than 400–500 ppm manganese (dry weight basis) in their tissues showed toxicity symptoms. The following concentrations (ppm, dry matter) of manganese in leaf tissue are considered excessive: >1,000, Navel and Valencia oranges (Embleton and others, 1976); >1,000, avocados (Jones and Embleton, 1976); 200, soybean, >500, flax (Moraghan, 1977); and 160, soybean (Ohki, 1977).

Under man-induced conditions—Reuther, Smith, and Specht (1949) found that 600–800 pounds of manganese had accumulated in the top foot (approximately equivalent to 300–400 ppm) of acid sandy soils in Florida after 15 years of continuous fertilization of orange trees with manganese compounds. These amounts were toxic to some plants. Amounts found in comparable virgin soils were 34–46 kg total manganese per hectare. Plant species range greatly in their tolerance of excess manganese; Fergus (1954) found that kidney beans, a good indicator plant for excess manganese, showed toxicity symptoms when grown on a soil having a pH of less than 5.0 because of the easily reducible manganese in the soil. Vlamis and Williams (1967) reported that the following concentrations (ppm, dry weight) showed symptoms

of manganese toxicity in old leaves of plants grown in solution culture: 1,200, barley; 7,000, rice; 1,400, rye; and 800, ryegrass. Schroeder, Balassa, and Tipton (1966) reported the following toxic concentrations (ppm) of the divalent manganous ion in water supplied to cultures: yeast, 550; legumes, 1–10; orange and mandarin seedlings, 5; tomatoes, 5–10; soybeans, 10–25; flax, 25–100; and oats, 150–500.

Animals

Under natural dietary conditions—Cotzias (1962) reported from the literature that spontaneous lactation tetany has been recorded in cows grazing on pastures high in manganese. Further, consumption by animals of large amounts of certain tree foliage, which contains more manganese than do most herbaceous plants (Shacklette and Severson, 1975), potentially could be toxic. Herbage of mixed-species composition in pastures in Spain was reported to reach the toxicity limit (15 ppm, dry weight basis) for cattle in only 1 of 19 pastures studied (Gonzales and others, 1959).

Under man-induced dietary conditions—The Mn^{+2} form has a low order of toxicity to living organisms, especially to vertebrate animals (Schroeder, Balassa, and Tipton, 1966). The hexavalent form is highly toxic but does not occur in nature. The above report listed the following as toxic concentrations (ppm) of Mn^{2+} in diets: *Daphnia magna*, 50; flatworms, 700; freshwater fish, 2,420–3,450; birds, 4,800; chicks, 4,800; rats, >2,000; rabbits, 1,250–6,000; pigs, 500–2,000; and lambs, 5,000.

Man

Underwood (1971, p. 202) reported, "Chronic manganese poisoning occurs in miners working with manganese ores. The manganese enters the lungs as oxide dust from the air and also enters the body via the gastrointestinal tract. The disease is characterized by a severe psychiatric disorder (locura manganica) resembling schizophrenia, followed by a permanently crippling neurological (extrapyramidal) disorder clinically similar to Parkinson's disease." Cotzias (1962) reported that a particle size of manganic oxide $<5\mu$ was responsible for the disorder. The National Research Council (1973) reported that manganism in miners, voltaic-cell factory workers, iron and steel industry workers, and others has been reported from Spain, Sinai, Germany, Chile, Morocco, Cuba, Italy, and Japan, to name a few. From the literature, these authors also reported a case of acute manganese intoxication of a family that ingested well water contaminated by old buried dry cells. Bowen (1966) reported that the normal consumption of Mn^{2+} in a 750-g/day dry-weight diet was 3–10 mg (4–12 ppm), duration not given.

MERCURY

[Considered nonessential for plants and animals]

Plants

Under natural conditions—Mercury in living tissues is believed to be largely organic and primarily methyl mercury. The conversion of inorganic mercury to methyl mercury or dimethyl mercury is accomplished by anaerobic bacteria in the bottom muds of streams. Dimethyl mercury, stable in alkaline solutions, dissociates to ionic methyl mercury at low pH values such as may exist in the anaerobic bottom muds of streams and lakes. Methyl mercury is soluble in water and is available for incorporation into the tissues of aquatic organisms; thus, it can enter the food chain of higher animals and man. Or the methyl mercury may enter the higher organisms by direct assimilation from the surrounding medium (Greeson, 1970). Mercury compounds inhibit the growth of bacteria and have long been used as antiseptics and disinfectants. In contrast, most higher (vascular) plants are remarkably resistant to mercury poisoning, although they may accumulate high concentrations of mercury in their tissues. Shacklette (1970) reported as much as 3,500 parts per billion (ppb) in dry material of labrador tea that grew where its roots were above cinnabar mineral deposits, but the plant showed no toxicity symptoms. The geometric mean mercury concentration in 912 samples of soil from throughout the conterminous United States was reported as 71 ppb (Shacklette and others, 1971). Shacklette (1970) stated that the few available reports of mercury analysis of plants suggest that this metal is not concentrated to a great extent, if at all, in the tissues of most plants that grow in normal soils. Mosses are tolerant of high mercury levels in their substrate (Shacklette, 1965b).

Under man-induced conditions—Fungicides containing mercury compounds have been widely used on agricultural and horticultural crops. These compounds, if sprayed on plants, may be absorbed by the plants and translocated from one part of the plant to another. Novick (1969) stated that mercury compounds from sprays applied to apple leaves may be translocated to the fruits, and sprays applied to potato leaves may be moved to the tubers. In addition, the sprays may produce surficial deposits on the leaves that are not easily removed. The mercury compounds, as applied, have no toxic effects on the plants themselves. Shacklette and Connor (1973) reported 0.5 ppm mercury (dry weight basis) in Spanish moss plants that presumably resulted from airborne industrial pollution, but toxic effects on the plants were not noted. Some greenhouse flowers, notably roses, are sensitive to volatilized elemental mercury, in that very low concentrations in the air cause toxic symptoms (Shacklette, 1970).

Animals

Under natural dietary conditions—We have no reports of toxicity resulting from animals grazing on normal vegetation; it is likely that the small amounts present in the plants are well within the elimination rates of animals. Wershaw (1970, p. 31) stated, "Natural surface waters contain tolerably small concentrations of mercury except in areas draining mercury deposits."

Under man-induced dietary conditions—The concentration of mercury, that was derived from the soil and that is present in crop plants probably is never toxic. However, the mercury compounds applied as fungicides to plant leaves, seeds, and grains may reach levels that are toxic to animals. Bowen (1966) reported that the consumption of 8 mg/day Hg^{2+} in a total-dry-weight diet of 10 g (800 ppm) was lethal to rats, duration not given.

Man

Greeson (1970, p. 33) stated, "Aquatic organisms, as well as man, will concentrate mercury within their bodies. . . . The result . . . is a buildup with time to the extent that the accumulated mercury can become toxic and, eventually, lethal." In Japan about 50 persons, out of more than 100 affected, died of the "Minamata disease," caused by the consumption of fish and shellfish obtained from a bay that had received large amounts of methyl mercury compounds in the waste effluents from a plastics factory (Kurland and others, 1960). Mercury poisoning was reported among citizens of Iraq who consumed bread made from seed wheat that had been treated with methyl mercury (Bakir and others, 1973). They stated that the wheat contained 8 ppm mercury, and acute concentrations (resulting in many patient deaths) were measured at 0.5–0.8 mg of mercury per kilogram of body weight. The biological half-life of methyl mercury in man is about 70–80 days. It is readily absorbed by the gastrointestinal tract. Löfroth (1972, p. 66) stated, ". . . clinical methylmercury poisoning in adult people can occur at a mercury level of about 400 ng/g in the red blood cells . . . corresponding to a regular daily intake of 0.3 mg mercury as methylmercury, i.e. 0.004 mg/kg body weight." Bowen (1966) reported that the normal daily consumption of Hg^{2+} was from 0.005–0.02 mg when based on a 750-g dry weight diet (0.007–0.03 ppm). Consumption of 150–300 mg on the same basis (200–400 ppm) was considered lethal, duration not given. The U.S. Public Health Service (1962) proposed a tentative upper limit of 5.0 ppb mercury in drinking water.

MOLYBDENUM

[Essential trace element for plants and animals]

Plants

Under natural conditions—There are no recorded instances of a field

occurrence of molybdenum toxicity to plants (Johnson, 1966), although high levels may be accumulated in the plants. Dye and O'Harra (1959) reported that, among forage plants, legumes usually collect more molybdenum than do grasses; they cited a high value of 372 ppm molybdenum (dry weight basis) in a sample of black medic from a Carson Valley, Nev. ranch. Warren and Delavault (1965) stated that many trees and lesser plants growing over commercial, or potentially commercial, molybdenum deposits may be expected to carry upwards of 500 ppm molybdenum in their ashes and that relatively few should be found with less than 250 ppm. Deficiency levels for plants are usually indicated by less than 0.10 ppm in their tissues (Johnson, 1966), whereas Embleton, Jones, and Platt (1976) reported that >100 ppm (dry matter) molybdenum in the fruiting shoots of Navel and Valencia oranges is probably an excessive amount.

Under man-induced conditions—Toxicity in the plant is observed only under extreme experimental conditions; in solution-culture experiments using 1,000–2,000 ppm molybdenum, tomato plants developed an intense golden yellow color in their leaves, and seedlings of cauliflower turned an intense purple (Johnson, 1966). Wallace, Romney, Alexander, and Kinnear (1977) wrote the following: "A level of 10^{-3} *M* H_2MoO_4 was toxic to bush beans grown in solution culture. Leaves, stems, and roots, respectively, contained 710, 1054, and 5920 μg Mo/g dry weight."

Animals

Under natural dietary conditions—"Molybdenum toxicity, variously referred to as molybdenosis, teart disease, and peat scours, has been reported from many parts of the world . . . . In most instances toxic amounts of molybdenum in forage consumed by ruminants have resulted from naturally occurring excess molybdenum in the soil or irrigation water" (Johnson, 1966, p. 287). This toxicity in ruminants is a complex matter, involving not only excess molybdenum but also low copper levels and high sulfate-sulfur concentrations in the forage. Molybdenosis occurs when the copper in forage is normal (8–11 ppm in dry matter) and the molybdenum is above normal (more than 5-6 ppm for cattle or 10–12 ppm for sheep) (Dye and O'Harra, 1959). Molybdenosis in grazing animals has been observed in the San Joaquin Valley of California (Barshad, 1948) and in Nevada and Oregon (Kubota and others, 1961, 1967). The amount of molybdenum absorbed by plants is not strongly correlated with total molybdenum in the soil, because soil pH and other soil properties greatly affect availability to plants, molybdenum being more available at high pH values. The area in Kern County, Calif., reported by Barshad (1948) to produce molybdenosis in cattle, had alkaline soils with total molybdenum concentrations of 1.5–5.0 ppm; these soils supported pasture plants, a large proportion having 20 or more ppm molybdenum (dry weight).

Under man-induced dietary conditions—Applying molybdenum fertilizers, or liming to release previously unavailable molybdenum, may result in the forage containing amounts of the element that are toxic to animals. Mining activities may expose deposits having concentrations of molybdenum and other elements that can be considered anomalous in the natural geochemical environment of plants and animals. An area in Missouri was studied (Ebens and others, 1973) where clay mining operations had contaminated adjacent areas to the extent that beef cattle grazing these areas exhibited symptoms of metabolic imbalance thought to be chronic molybdenosis due principally to copper-molybdenum imbalance. G. A. Christianson and G. A. Jacobson (written commun., 1974) reported molybdenosis in beef cattle in North Dakota in the vicinity of a lignite ashing plant. Apparently emissions from the plant raised the molybdenum level in the forage, creating a molybdenum-induced copper deficiency in the cattle. Molybdenum concentrations in fecal material from contaminated fields ranged from 16 to 26 ppm (dry weight). Bowen (1966) reported that 5 mg molybdate in a 10-g/day dry-weight diet (500 ppm) was toxic and 50 mg (5,000 ppm) was lethal to rats, duration not given. Owing to poor absorption, horses and pigs have low susceptibility to molybdenosis (Underwood, 1971).

Man

At normal environmental concentrations of molybdenum, toxicity is of little concern. Underwood (1971, p. 135) stated, "Molybdenum is apparently of so little practical significance in human nutrition, either in health or disease, that few people have been stimulated to undertake studies with this element." The only incidence of molybdenosis in humans of which we are aware was reported by Agarwal (1975). He found that peasants in India who consumed sorghum that was grown in alkaline soils high in molybdenum and fluorine as a staple, developed a crippling syndrome of knock-knees (genu valgum). No instances of the disease were reported prior to the construction of a large dam that raised the water table in the surrounding district and caused an increase in alkalinity and concurrently the uptake of molybdenum by the sorghum. No specific concentrations of molybdenum in sorghum, soils, or humans were given.

NICKEL

[Suspected as being essential for some plants and animals]

Plants

Under natural conditions—Mitchell (1945) stated that the normal nickel content of plant material, dry weight basis, ranges from 0.10–5 ppm, depending on the species, the part of the plant, maturity, time of sampling, and other factors. Extremely high nickel concentrations of

1–2 percent (dry weight) in the leaves and 25.74 percent in the latex of *Sebertia acuminata* from New Caledonia have been reported by Jaffré and others (1976). The plant appears to be restricted in its growth to areas of ultrabasic substrates in which nickel concentrations are relatively high (0.85 percent in soil). No toxicity symptoms were expressed in response to these extreme tissue concentrations. Excessive nickel in serpentine soils may be a factor contributing to the restricted growth of plants on these soils, as reported by many investigators; but excessive chromium, unfavorable magnesium-calcium ratios, and deficiency of molybdenum may also limit plant growth (Vanselow, 1966a). The amount of nickel absorbed by plants on serpentine soils ranges widely among species; Shacklette (1966) reported the following concentrations (ppm in ash) from a site in Alaska: aspen, 10; white birch, 50; and low juniper, 150. The soil at this site contained 50–75 ppm nickel, and the basaltic greenstone on which the soil developed contained 100 ppm nickel. Malyuga (1964) described apetalous forms of anemone growing over nickel deposits, attributed to toxicity of the nickel.

Under man-induced conditions—Toxicity of nickel to many plants, even at relatively low concentrations, has been noted; 40 ppm (dry weight) in tomato plants was toxic, and 150 ppm stopped growth (Sauchelli, 1969). Soane and Saunder (1959) reported 12–246 ppm (dry weight) in leaves of corn and 14–34 ppm in leaves of tobacco—both kinds of plants developed toxicity symptoms. The following concentration ranges for nickel in nutrient solutions are reported from the literature by Vergnano and Hunter (1952) as being toxic: 2–40 ppm, beans and maize; 2–60 ppm, buckwheat; 3–300 ppm, oats; 2–15 ppm, barley and beans; and 15–30 ppm, oats and sugar beets. These authors also reported from their own studies that nickel toxicity was observed in oats that had nutrient solutions ranging from 1 to 1.5 ppm (slight leaf mottling) to 10–30 ppm (chlorosis and necrosis). Soybean plants were killed in a soil of pH 6 when 1,000 ppm nickel was added to the soil. Higher pH decreased the toxicity of nickel (Wallace, Romney, Cha, Soufi, and Chaudhry, 1977).

Animals

Under natural dietary conditions—Mitchell (1945) recorded levels of 0.5–4 ppm (dry weight) nickel in pasture herbage; Allaway (1968) reported that toxicity to animals was moderate to low. In view of these relationships, toxicity to grazing animals seems unlikely.

Under man-induced dietary conditions—Compared to many other trace metals, the concentration of nickel in foods is high. A mechanism in mammals, however appears to limit its intestinal absorption. The toxicity of nickel to mammals is low (ranking with the essential trace elements) except in astringent doses. Nickel salts irritate the mucosal lining of the gut more than they cause inherent poisoning (Schroeder,

Balassa, and Tipton, 1962b). Underwood (1971) reported that 700 ppm nickel in the diet of chicks depressed growth, 1,600 ppm depressed growth of young mice, and 1,000 ppm had no apparent toxic effect on rats or monkeys. The National Research Council (1975) reported the following from the literature: 1–3 g/kg body weight of nickel metal, non toxic to dogs; 250, 500, and 1,000 ppm each of nickel carbonate, nickel soaps, and nickel catalyst (Raney nickel) in the diet of young rats for 8 weeks, non toxic; 5 ppm nickel acetate in the drinking water of mice over their lifetime, non toxic; 5 mg/kg nickel metal orally administered to guinea pigs, lethal; and 500 mg/kg nickel fluoborate orally administered to rats, lethal. Bowen (1966) gave as toxic to rats a ration of 50 mg Ni^{2+} in a 10-g/day dry-weight diet (5,000 ppm), duration not given. He listed the element as potentially carcinogenic.

Man

The toxicity of nickel to humans is low. Workers in nickel refineries have developed "nickel dermatitis" and respiratory-tract neoplasia; a direct link between the elemental form and these pathologies, however, has not been made (Schroeder, Balassa, and Tipton, 1962b). Nickel carbonyl, used in nickel refining, nickel plating, and the petroleum industry, is the only organic nickel compound that has been recognized as a cause of human systemic toxicity (National Research Council, 1975). The toxicity of this compound was summarized (Blomberg and others, 1977, p. 22) as follows: "Nickel carbonyl poisoning is considered mild if the urinary nickel concentration is less than 100 μg/L, moderately severe if the concentration is 100-500 μg/L and severe if the concentration is more than 500 μg/L." Nickel carbonyl, if inhaled, is extremely toxic and can be lethal.

SELENIUM

[Considered nonessential for most plants; essential trace element for most animals]

Plants

Under natural conditions—"There are no recorded instances of naturally occurring selenium causing damage to plants in the field" (National Research Council, 1976, p. 70). The occurrence and ecological relationships of selenium-indicating plants have been documented by many investigators. Two groups of native species may be recognized: the primary indicator plants are those found growing only on seleniferous soils and which absorb large amounts of selenium; the second group is composed of those species that are also capable of absorbing selenium in concentrations toxic to animals if growing on seleniferous soils but which may also grow elsewhere. Excess soil selenium may prevent plants not in these two groups from growing on seleniferous soils by causing toxicity symptoms to develop. These

symptoms are never seen in crop plants grown on naturally seleniferous soils, but under experimental conditions the symptoms in grasses ordinarily are snow-white chlorosis of the leaves and pink root tissue; symptoms in other plants vary, but commonly include stunting, yellow chlorosis, and pink leaf veins (Ganje, 1966). Lakin (1972) stated that certain species of *Astragalus* utilize selenium in an amino acid peculiar to these species and that they absorb many times as much selenium as do other plants growing in the same soil. These plants convert selenium into forms absorbed by non seleniferous species through foliage decomposition and oxidation to inorganic salts or soluble organic compounds (Ganje, 1966). The plants, both fresh and dried, often have a strong garlicky odor. Byers and Lakin (1939) analyzed about 300 samples of shales, soils, and plants collected in seleniferous areas of Canada and found the selenium of shales to vary from 0.3 to 3.0 ppm; of soils, from 0.1 to 6 ppm; and of vegetation, from 3 to 4,190 ppm in dry matter. The organic compounds of selenium in soil are derived from partially decayed seleniferous vegetation. Inorganic selenium exists as the element; as ion selenide; as a substitute in sulfide minerals, particularly pyrites; as selenite, particularly basic ferric selenite; and as selenate, particularly calcium selenate. Basic ferric selenite seems to be the most common form in soils, while very little is present in the elemental form. Selenate and organic selenium are the forms most available to plants (Ganje, 1966).

Under man-induced conditions—Soils producing seleniferous vegetation have been found only in arid or semiarid regions, at least in the United States, where the mean annual rainfall is less than 20 inches. For crop plants, only the soil content of water-soluble selenium is important, because elemental selenium and selenite are of only slight availability to these plants, as was demonstrated by Hurd-Karrer (1934). Corn grown in culture solutions containing 5 ppm of selenite or organic selenium accumulated 200 and 1,000 ppm selenium, respectively. At the 10-ppm level, corn accumulated 300 ppm from the selenite form and more than 1,500 ppm from the organic selenium form (Ganje, 1966). Resistance to selenium toxicity among plant species ranges so widely that a general toxicity level cannot be reliably estimated.

Animals

Under natural dietary conditions—There are numerous accounts in the literature of grazing animals being poisoned after consuming certain types of vegetation. Some of the very early descriptions of the disease symptoms (such as sloughing hooves) leave little doubt that many of these cases were the result of selenium intoxication (Trelease and Beath, 1949). These authors presented an interesting history of selenium poisoning, the documentation of which goes back to 1857 (in

an area of western South Dakota) and perhaps to 1295 (in an area of western China as described by Marco Polo). Lakin (1972, p. 49-50) reported, "Acute poisoning of cattle and sheep occurs when herds or bands are being driven from one pasturage to another. Animals, under these conditions, eat forage indiscriminately and many may die overnight. Both seleniferous *Astragalus* species and alkaloids are probably involved. . . . Subacute poisoning, known locally as blind staggers, loco disease, or pushing disease, has been reported in western United States and in South Africa. This is probably another instance of the confusion between selenium toxicity and naturally toxic vegetation. The actual cause of these diseases is in debate. Chronic poisoning is caused by daily ingestion of cereals and grasses containing 5 to 20 ppm selenium. . . . Subchronic poisoning has been reported by Brown and de Wet (1962) in South Africa. They suggest '. . . the moderate selenium content of the diets of these animals has weakened them and made them more susceptible to disease.' " Byers (1935) suggested 4 ppm (dry weight) of selenium in plants as a tolerance limit for animals that consume them and considered 5 ppm to be potentially dangerous. Forage with selenium concentrations of 10–30 ppm (dry weight) was considered capable of producing toxicity symptoms in cattle (Church and others, 1971). Water containing 0.5 ppm selenium may also be dangerous (Ganje, 1966).

Under man-induced dietary conditions—Because of the dual role of selenium as a micronutrient and a toxin to animals, the selenium concentrations in feeds can be critical factors in the health of the animals. Underwood (1971, p. 346) stated, "A dietary intake of 0.1 ppm [in dry feeds] provides a satisfactory margin of safety against any dietary variables or environmental stresses likely to be encountered by grazing sheep and cattle." Many factors determine the level of selenium in feeds that are toxic to animals, but amounts of 5 ppm (dry weight) or more may generally be considered toxic. Underwood (1971, p. 356) stated, "Signs of chronic poisoning are common in rats and dogs given diets containing 5–10 ppm selenium, and, at 20 ppm, there is complete refusal of food and death in a short time. Young pigs fed seleniferous diets containing 10–15 ppm selenium develop signs of selenosis within 2 to 3 weeks. Diets containing lower levels than these would induce similar effects if fed for a longer period. The minimum toxic levels for grazing stock are more difficult to determine. They probably lie close to 5 ppm selenium. Edible herbage in seleniferous areas commonly contain 5–20 ppm selenium." Church and others (1971) summarized numerous studies and reported that acute selenium toxicosis can be produced in cattle and sheep by feeding them plants that supply about 2 mg of selenium per kg of body weight. He also stated that selenium was lethal to cattle, under feedlot conditions, at a

rate of about 1.1 mg/kg of live weight (dosed 3 times weekly). Overt expressions of poisoning were observed when selenium in the blood exceeded 3 ppm. In work with sheep he reported that a dose of sodium selenate equivalent to about 1.8 mg/kg of live weight was lethal and that consumption of plant material containing 400–800 ppm selenium may be fatal when fed in quantities of 8–16 g/kg of body weight. Sauchelli (1969) reported that wheat can grow in soil containing 1 ppm selenium. If grain containing 8–10 ppm selenium was fed to rats, it retarded growth and killed them after a few weeks. Selenium may be introduced into the environment through the burning of coal, in phosphate fertilizers, and in selenium-containing insecticides, but the latter source has very little application to toxicity problems at present because the use of these insecticides on food plants is prohibited, except in special circumstances.

Man

Under dietary conditions in seleniferous regions—Lakin (1972, p. 51) wrote, "Soils producing crop plants that are toxic because of selenium are confined to small areas but occur throughout the world. Such soils are confined to semiarid regions or areas of impeded drainage. They contribute no significant hazard to human health and only locally to animal health." The selenium content of certain foods may reach levels that could affect human health. For example, the selenium content of tuna was reported (Kifer and others, 1969) to average 4.63 ppm (dry weight) and to range from 3.4 to 6.2. A daily intake of 1 mg selenium in food is probably not harmful to an adult person. Allaway (1975) reported that a concentration of selenium of 3–4 ppm in human and animal diets is considered safe. Robinson (1936) found that seleniferous wheat may be blended with sufficient selenium-free grain to drop the selenium concentration to the tolerance level. Hadjimarkos (1970) reported that the incidence of dental caries appears to be greater in areas naturally high in selenium. This is particularly true among children who consume small amounts of dietary selenium in addition to the naturally high amounts in the local water. Selenium concentrations of 0.08 ppm in the urine identified those individuals with increased incidence of dental caries. The situation regarding selenium poisoning in man was summarized by Lakin (1972, p. 50) as follows: "Fortunately, the human diet is rarely restricted solely to the products of a highly seleniferous area. Thus, selenium poisoning through the food-plant cycle is rare in the world human population and tends to be restricted to combinations of highly seleniferous areas whose populations depend largely on local agricultural produce." Chronic selenium poisoning in humans was reported to occur in Mexico by Byers (1937) and in Columbia by Ancizar-Sordo (1947) and Leonidas and Hernán (1970).

Under the effects of environmental pollution by selenium—Sauchelli (1969) reported that the maximum amount of selenium that man can consume in the whole diet without ill effects is believed to be 3 ppm. Bowen (1966) listed as toxic 5 mg $Se^{(IV)}$ (selenite) in a 750-g/day dry-weight diet (7 ppm), duration not given. Pollution-related selenium intoxication is most commonly associated with industrial exposure (Cooper, 1967), because selenium compounds are absorbed through the lungs in dust or fumes and through the skin. Whether ingested or inhaled, Cooper (1967, p. 186) stated, " . . . there is marked variations between and within animal species in their response to selenium at what may be considered toxic levels."

SILVER

[Considered nonessential for organisms]

Plants

Under natural conditions—We have no reports of silver toxicity in plants growing under natural conditions. The concentration of silver in plants is usually less than 0.5 ppm in ash; higher values, however, have been found in a wide variety of plant groups including mosses (as much as 7 ppm, Shacklette, 1965a); club moss (5 ppm); ferns (bracken, 3 ppm); coniferous trees (highest value reported was 200 ppm in dwarf juniper); deciduous trees (bur oak, 15 ppm); and vegetables (tomato fruits, 7 ppm, Connor and Shacklette, 1975). These levels of silver produced no observed toxicity in the plants. Warren and Delavault (1950) reported finding 0.1–1.4 ppm silver in ash of trees and herbs that grew in areas containing no known silver mineralization. The silver concentration in "normal" soils seldom exceeds 0.5 ppm, but a concentration of 5 ppm has been reported (Shacklette and others, 1971); concentrations may be much higher at mineralized sites.

Under man-induced conditions—Brooks (1972) reported that the toxicity of silver to plants was severe, but he did not give the conditions under which this toxicity occurred. Clark (1899) found that silver in a concentration of 0.0098 μg/ml was fatal to corn and that 0.0049 μg/ml was fatal to lupines. Wallace, Alexander, and Chaudhry (1977) wrote the following concerning silver toxicity to bush beans: "Silver was very lethal at 10^{-4} *M* $AgNO_3$; at 10^{-5} *M* yields were greatly decreased, but plants were grown without symptoms. Leaf, stem, and root concentrations of Ag for this treatment, respectively, were 5.8, 5.1, and 1,760 μg/g dry weight." Silver readily forms insoluble compounds, which reduces the risk of environmental contamination; but insoluble compounds or metallic silver can form minute amounts of ionic silver, either through chemical reactions or electrolysis, that can be toxic to microorganisms (Goetz and others, 1940). Its toxicity to a variety of microbes has been

utilized in the preservation of medicines and in water purification (Smith and Carson, 1977). Romans (1968) reported that water containing 15 g/L silver sand killed the bacterium *Escherichia coli* in 2–24 hours, depending on numbers of bacteria. Concern with the effects of silver iodide fallout from cloud seeding for increasing precipitation centers around the sensitivity of soil microorganisms to the silver ion. In a report of experiments on these organisms, Sokol and Klein (1975, p. 211) concluded, "These results suggest that high concentrations of seeding agent potentially can alter the soil microbial environment by inhibiting organic matter decomposition, but that the silver levels which accumulate in seeded target areas should have no overt effects on the soil environment."

Animals

Under natural environmental conditions—We have no report of silver toxicity in animals under these conditions. Silver apparently is not bioconcentrated, and silver in natural deposits is largely in an un-ionized state, therefore is unlikely to cause animal toxicity.

Under man-induced conditions—Cooper and Jolly (1970, p. 89) stated, in regard to silver, ". . . it is relatively harmless to higher animals, including man." Bowen (1966) gave the silver content of mammalian tissue as ranging from 0.01 ppm (in dry matter) in bones to 0.04 ppm in certain soft tissues. Younger and Crookshank (1978) reported that silver iodide (AgI) used in weather modification may be deposited on forages in the seeded target area and enter the food chain of livestock. In their experiments with sheep, 15 ewes were given oral doses of AgI at 0.1, 1.0, or 10 mg per kg per day without developing clinical signs of toxicity. Silver was absorbed from the gastrointestinal tract and deposited in soft and hard tissues of the ewes; the maximum concentration found was 17 ppm in the liver of a ewe that received the highest dose rate. In administering the AgI doses, the gelatin capsules were inadvertently broken in the mouths of two ewes; these ewes died from acute hemorrhagic pharyngitis within 72 hours after the accidental breakage. These investigators concluded that AgI from cloud-seeding operations is not likely to induce overt toxic effects on livestock.

Silver in minute amounts in water is very toxic to fish, probably by interference with gas exchange by the gills. Doudoroff and Katz (1953) found that solutions containing 0.0001 μg/ml were harmful to freshwater fish. Schmähl and Steinhoff (1960) reported that colloidal silver produced tumors in experimental animals.

Man

Smith and Carson (1977, p. 355) stated, "No systemic pathology has been directly attributable to silver among workers occupationally exposed to it." Cooper and Jolly (1970, p. 90) reported, "Silver, even in

highly soluble form, is only moderately harmful to mammals. Because of caustic action on the intestinal tract, 10 g of $AgNO_3$ is usually fatal to man, but 3 g can be taken safely." Continued intake of silver salts causes an irremedial discoloration of the skin and mucous membranes (argyria) from the deposition of organo-silver compounds that are reduced to dark-colored forms by sunlight. There are no other ill effects from this silver deposition (Sax, 1975). Because of the extremely low silver concentrations in food, dietary intake of silver by man probably is of no consequence.

SULFUR

[Essential for plants and animals]

Elemental sulfur is of little concern as a toxic substance. In this form it has long been used as a pesticide, particularly for the control of certain plant diseases. Compounds of sulfur, in contrast, may constitute important environmental pollutants that have adverse or frankly toxic effects on plant and animal tissues. Practically all sulfur toxicity problems of environmental concern in plants, animals, and man are caused by the mining of ores and fuels and the processing and use of these materials.

Plants

Under natural conditions—Areas barren of vegetation may exist at locations where natural deposits of sulfur-rich materials occur at or near the surface. "Oxidation of sulfur in soils can occur either as non-biological (auto-oxidation) or as biological oxidation, the latter being more important" (Blair, 1971, p. 117). The resulting SO_3^{-2} and SO_4^{-2} in the presence of water can form H_2SO_4 (sulfuric acid). The localized "acid spots" that are formed bear little or no vegetation more because of low soil pH than of toxicity to the sulfite or sulfate ions. Although sulfur dioxide (SO_2) is formed naturally by oxidation of hydrogen sulfide that occurs in bogs and swamps, we have no reports of damage to plants from this source.

Under man-induced conditions—Sulfur dioxide is increasing in importance as a plant toxicant. By far the largest amount of airborne sulfur compounds (principally SO_2) in areas affected by industrial operations is of man-made origin, although in certain areas sulfur from natural sources is significant. Grey and Jensen (1972, p. 1099) noted, "On a yearly basis, the major source of atmospheric sulfur compounds in and near Salt Lake City, Utah, is industrial. Isotopic studies suggest that the next most important source is bacteriogenic sulfur released by anaerobes from muds. On a seasonal basis, the bacteriogenic source of sulfur compounds may rival the industrial source in importance." Background levels of sulfur dioxide in air pro-

bably range from 0.28 to 2.8 $\mu g/m^3$ (1–10 ppb), whereas phytotoxic effects begin to appear between 27 and 224 $\mu g/m^3$ (10–80 ppb) mean annual local concentration (Nash, 1973). Lichens, bryophytes, and fungi are the most sensitive plants to sulfur dioxide insult and generally are damaged at concentrations of less than 100 $\mu g/m^3$ (<35 ppb) (Saunders and Wood, 1973). Species of coniferous trees probably are the most sensitive of the higher plants. Nash (1973) reported the following toxicity thresholds from the literature: Douglas-fir seedlings, 812 $\mu g/m^3$ (290 ppb) after 44 hours; and eastern white pine, 280 $\mu g/m^3$ after 8 hours. He also reported that the most sensitive non-coniferous higher plant species generally exhibit chronic toxicity symptoms at concentrations of 280–840 $\mu g/m^3$ (300–500 ppb).

The symptoms of acute injury to plants were summarized by Daines (1969, p. 1) as follows: "Acute injury, resulting from the rapid absorption of a toxic dose of SO_2, manifests itself as marginal or intercostal necrotic areas which at first have a dull dark green water-soaked appearance and on drying and bleaching become an ivory color in most plant species, but in some browns and reds predominate. The necrotic areas extend through the leaf and are visible on both surfaces. The areas immediately bordering the veins are seldom injured. These areas are characterized by few stomata and very limited intercellular spaces. The younger fully expanded leaves are most susceptible to SO_2, followed by the older leaves, while the enlarging leaves are the last to show acute injury."

Acid rain, produced when sulfur dioxide is oxidized to sulfuric acid, is a potentially serious toxicant to plants. Carrier (1977, p. 8-1) summarized as follows, "Acid rain, largely the result of sulfur dioxide pollution, may damage plants at distances of up to 1000 km from the pollution source. Indirect evidence suggests that it has caused a reduction in forest productivity in certain areas, and it has been shown to cause plant abnormalities and to have detrimental effects on plant nutrition. Acid rain also can destroy the protective waxy layers of foliage, cause enhancement of disease, and decrease plant productivity. Because the uptake of heavy metals is increased by lower than normal pH values, a synergistic effect can reasonably be expected where fallout of heavy metals and acid rain appear simultaneously."

Animals

Under normal dietary conditions—Sulfur toxicity in animals caused by feed or water that is normally consumed is unlikely because of the low sulfur content of these materials. Postgate (1968, p. 267) wrote, "Though certain animals, notably ruminants, can tolerate appreciable free sulphide in their intestines, H_2S is normally toxic to aerobic plant and animal tissues." He stated further (p. 278), "The rumina of sheep contain sulphate-reducing bacteria and the sulphide they form has

been shown, by isotope experiments, to contribute to the sulphur nutrition of sheep. Sulphate-reducing bacteria are found in the guts of insects where they may have a similar function."

The toxicity of sulfur to grazing animals is indirect. Copper deficiency diseases have been reported in lambs (Underwood, 1971) following the consumption of herbage naturally high in sulfate or from grazing in mining areas rich in sulfur minerals. These imbalances result from a synergistic metabolic relationship between copper, molybdenum, and sulfate. Underwood (1971, p. 133) wrote, "... chronic copper poisoning can occur in sheep with moderate copper intake and very low levels of molybdenum and sulfate, and, conversely, the depletion of the animal's copper reserves, to the extent of clinical signs of copper deficiency, can arise on normal copper and high-molybdenum and -sulfur intakes."

Under man-induced environmental conditions—The effects on aquatic organisms of mine drainage waters that are highly acidic because of the oxidation of pyrite (FeS_2) are well known and have been extensively reported. Again, the toxic effects are largely related to pH, not to the sulfur content, of the water. Toxic effects on animals of sulfur dioxide presumably are similar to those on humans that are discussed below, although we have no reports of these effects on domestic or other animals. Acid rain has been reported to have caused the death of fish by lowering the pH of water in lakes that naturally have a low buffering capacity. Attempts have been made to correct this toxicity by adding lime to the water.

Man

Under the effects of atmospheric pollution—Saunders and Wood (1973) stated that sulfur dioxide concentrations of 500–3,000 $\mu g/m^3$ (0.18–1.07 ppm) have a detectable odor and that concentrations greater than 8,500 $\mu g/m^3$ (3.04 ppm) are very pungent and irritating. Lillington (1974, p. 316) reported, "Sulfur dioxide primarily affects the upper respiratory tract and larger bronchi. Ciliary action and mucous flow are inhibited at concentrations of 10 to 15 ppm. Increased airway resistance (due to reflex broncho-constriction) occurs regularly with exposures of 4 to 6 ppm breathed by mouth for ten minutes, and some unusually sensitive individuals respond to concentrations of 1 ppm. ... Asthmatic and bronchitic subjects are clearly more sensitive in inhaled SO_2. ... Sulfur dioxide in the atmosphere can be oxidized to sulfur trioxide which forms sulfuric acid when dissolved in water. Sulfuric acid is considerably more toxic than SO_2, but the magnitude of this conversion in smog is not well established."

THALLIUM

[Considered nonessential for organisms]

Plants

Under natural conditions—Very few data are available on the normal concentrations of thallium in vegetation growing in the United States. The following parts per million in ash were reported (Shacklette and others, 1978) for trees and shrubs in the Rocky Mountain region: subalpine fir needles 2–100, stems 2–70; limber pine needles 2–5, stems 3–5; lodgepole pine needles 2–5, stems 3–7; Engelmann spruce needles 2–10, stems 15; myrtle blueberry stems and leaves 2–7; phyllodoce stems, 2; and ponderosa pine stems 15. Carson and Smith (1977) gave unpublished data from the U.S. Geological Survey on thallium concentrations in U.S. plants; 2–7 ppm in ash was considered anomalous and 10 ppm, highly anomalous. Most values, if detected (in about 1 percent of the samples), ranged from 0.4 to 0.5 ppm in ash. Berg (1925) reported 0.1 mg thallium in 100 g (1 ppm) of spinach and rye samples. Thallium in some European food and feed plants, given by Geilmann and others (1960) as parts per million in dry weight, follow: head lettuce, 0.021; red cabbage, 0.040; green cabbage, 0.125; leek, 0.075; endive, 0.080; clover, 0.008–0.010; and meadow hay, 0.020–0.025. Reports of thallium in ash of native plants in the Soviet Union (Dvornikov and others, 1976) gave a range of about 0.01 to 1.0 g/ton (0.01 to 1.0 ppm). The average for soils that they analyzed was 0.023 g/ton (0.023 ppm). The plant concentration factor, based on 14 species of plants and 38 soil samples, was calculated to be 1.74. Toxic levels of thallium were reported by Zýka (1972) in native vegetation of a region in Yugoslavia that was remarkable for high levels of thallium in the substrate. He found thallium in ash of herbaceous plants to range from 10 ppm in *Eryngium* to 17,000 ppm in bedstraw. Toadflax contents ranged from 3,000 to 3,800 ppm. He also noted zonation of species related to soil concentrations of thallium, which indicated the degree of toxicity of the element to certain plant species.

Under man-induced conditions—McMurtrey and Robinson (1938, p. 827) stated, "When artificially applied it [thallium] is probably taken up by the plant, and as little as 35 ppm in sandy soils has practically prevented the growth of plants." A bait for poisoning predators consisting of 7 g ground meat and 0.5 g thallium that was placed on stakes a few centimeters above the ground in Hawaii produced bare patches on the ground 3–6 dm long and about one-third as wide; no plants grew in these patches for at least two years (Brooks, 1932). Carson and Smith (1977) noted, "Many crop plants are injured by concentrations of about 7 ppm in the soil. Gramineae and seeds are more resistant. Tobacco plants are especially sensitive, showing toxic effects at 1 ppm in the

soil, or 4 ppm in water." Twelve-day-old crop plants in loam soil are completely inhibited by 1,100 ppm in irrigation water (Horn and others, 1936). Concentrations of 0.04 ppm in water induced chlorosis in tobacco seedlings (Carson and Smith, 1977). Wheat in sandy loam soil was injured with a concentration of 1.4 ppm in soil and killed at 28 ppm; toxic effects on buckwheat, alfalfa, and perennial ryegrass at near the same thallium concentrations were similar (McCool, 1933). Thallium levels of 2–3 ppm in freshwater reduces the photosynthetic activity of algae (Overnell, 1975), and at 7 ppm, definite toxicity occurs (DiGaudio, 1975, as cited in Carson and Smith, 1977, p. 315).

Animals

Under natural dietary conditions—Zýka (1972, p. 94) reported, regarding the high thallium region of Yugoslavia cited above, "The high thallium contents in the plants explain their toxic effects on cattle." Marsh (1958) and MacPherson and Hemingway (1969) reported natural poisoning by thallium of sheep in Israel. We have no other reports of thallium poisoning resulting from the consumption of vegetation by animals. Case (1974) stated that there was no effective antidote for thallium poisoning in sheep.

Under man-induced dietary conditions—Carson and Smith (1977, p 315) reported, " 'Non-effect levels' for mammals have not been established experimentally, but levels of near 3 ppb thallium in an animal's diet are likely to produce toxic effects detrimental to the individual's survival." The lethal dose for dogs was found to range from 35 to 253 mg/kg body weight; thallium in the blood of poisoned animals ranged from 5.8 to 3,470 ppm; and the range in other tissues was from 0.0 to 1,540, the latter concentration occurring in the spleen. Bowen (1966) reported that 7.5 mg Tl^+ in a 10-g/day dry-weight diet (750 ppm) was lethal to rats. Fatal doses of thallium for guinea pigs was found to be 20–80 mg/kg (Weinig and Walz, 1971). Meat from all marine seafood from Minamata Bay in Japan (where polluted water caused a serious disease in humans who ate seafood) averaged 0.08 ± 0.02 ppm thallium (Hamaguchi and others, 1960). Where algae containing as much as 0.85 ppm thallium serve as the principal food of aquatic animals, bioconcentration and direct toxicity to the consumer may occur (Carson and Smith, 1977).

Man

Thallium compounds are highly toxic to humans. McMurtrey and Robinson (1938, p. 827) wrote, "Thallium compounds have been used as depilatories with disastrous effects." Thomas (1973) gave 5–50 mg/kg body weight as the probable lethal dose of thallium salts for an adult, whereas Bowen (1966) listed 600 mg/day in a 750-g dry-weight diet (800 ppm) as lethal. Zitko and Carson (1975) concluded that there is no danger of thallium accumulating in aquatic mollusks to levels that are

toxic to humans. The concentration factor (stomach contents/tissue) for elk and deer was found to range from 0.04 to 0.14 in muscle and from 14 to 22.7 in bone (Carson and Smith, 1977), and they concluded that there seems to be little danger of human toxicity caused by eating meat of these animals. In regard to thallium toxicity from eating plants, Carson and Smith (1977, p. 164) wrote, "The highest value seen for U.S. soils is 5 ppm. If plants grown on such soils served as an entire human diet and had a concentration factor of 10, then this 'worst case imaginable' would provide a daily dietary intake of 80 mg or [about] 1 mg/kg." There has been much concern about the use of thallium poisons to control predatory animals resulting in the poisoning of other animals and birds. Shaw (1933) found that the minimal lethal dose of thallium on coated grain was 15 mg/kg body weight for mallard ducks and wild white geese, and for starved quail, 12 mg/kg. Tissues of these birds still retained 33 to 71 percent of the original dosage after 13 days. He concluded that secondary thallium poisoning in humans from eating a game bird was improbable. Carson and Smith (1977, p. 218), however, in commenting on Shaw's data, stated, "Continual eating of such birds might have been slightly more hazardous. . . . A man eating from 0.25 to 0.5 lb of such fowl per day would ingest about 2 to 4 mg thallium daily. This value is certainly much lower than the recommended *single* therapeutic epilant dose for an adult human of 300 mg but about 1,000 times more than the estimated normal dietary intake of thallium."

TIN

[Considered nonessential for plants and most animals, but recently found essential for rats]

Plants

Under natural conditions—Measurable amounts of tin are only rarely found in native plant species and, when found, are usually in concentrations of 20–30 ppm in ash (Connor and Shacklette, 1975). Harbaugh (1950) reported 17 ppm tin in ash of oak twigs, and Millman (1957) found 0.36 ppm (dry weight basis) in twigs of birch. Wallihan (1966, p. 476) stated, "Unlike lead, tin is generally a biologically innocuous element, as shown by its use for storing and conducting distilled water and in tin-coated food containers. Plants absorb it only to a very slight extent . . . ." We have no reports of tin toxicity to plants under natural conditions that are substantiated by data, although Brooks (1972, p. 225) stated, "Toxicity to plants, severe."

Under man-induced conditions—Prince (1957a, 1957b) reported 2.94 ppm (dry weight) tin in corn grains, Duke (1970) reported 0.10 ppm tin in dry material of avocado, and tin was reported in ash of one sample each of carrot root (20 ppm), corn grains (30 ppm), and beet root (20 ppm) (Connor and Shacklette, 1975). We have no other reports of tin in food plants. Forty ppm $Sn2^{+}$ (stannous ion) solution in sand cultures

has been reported as not affecting the growth of sugar beets (Schroeder, Balassa, and Tipton, 1964). Wallihan (1966, p. 476) wrote, "There appears to be no substantial evidence that tin is essential or beneficial to plants in any way, and it has not been shown to be detrimental under field conditions."

Animals

Under natural dietary conditions—We have no reports of animal toxicity attributed to naturally occurring tin in the environment.

Under man-induced dietary conditions—Schroeder, Balassa, and Tipton (1964) reported from the literature that relatively large doses of tin in the diet of laboratory animals are needed to produce toxicity. For cats and rats, 30–50 mg/kg body weight, and for dogs, 850–1,000 mg/kg, were toxic. They noted that a diet of 1 g every 6–10 days caused death in rabbits after 1 to 2 months. Further, 1,000 ppm tin in a tank of goldfish was fatal after 1.5 hours; at a concentration of 626 ppm, after 4.5 hours.

Man

Bowen (1966) reported 2,000 mg tin as toxic in a dry weight diet of 750 g/day (2,700 ppm), duration not given. Schroeder, Balassa, and Tipton (1964) reported from the literature that severe gastrointestinal symptoms in man have resulted from consumption of fruit punch (pH 3) that contained 2 g/L tin. Tin-plated containers for canned foods can be a source of large amounts of tin, particularly for foods that are acidic. However, the double-lacquered metal cans currently in use greatly reduce the tin in canned foods.

URANIUM[1]

[Considered nonessential for organisms]

Plants

Under natural conditions—Carnotite and minerals of the uranite-pitchblende series are the two kinds of uranium minerals likely to occur most abundantly in significant outcrops or near-surface deposits. Minerals of the pitchblende series seem to have no physiological effects on native plant species. Extensive examination by one author (Shacklette) of natural outcrops of this material and of tailings resulting from milling at Great Bear Lake, N.W.T., Canada, revealed no apparent effect on plant occupancy of these sites, or of decreased vigor of growth other than that which could be attributed to deficiencies of the major nutritive elements in the substrate. Tailings settling basins were rapidly colonized by species having highly efficient seed dispersal (especially, plants of the Compositae Family); the progress of plant succession on these tailings, however, was not determined by succes-

[1]This discussion examines only the chemical toxicity of uranium, not the effects of associated radiation, on organisms.

sive visits to the site. The prospecting methods for sedimentary uranium deposits in Western United States was studied extensively by Cannon (1957, 1959, 1960a, 1960b, and 1964) and by Cannon and Kleinhampl (1956), on the basis of both the analysis of plant tissue for uranium concentration and the presence of indicator plants that accumulate selenium (an element commonly associated with uraniferous deposits in western United States). Cannon (1959, p. 235) stated, "Trees rooted in ore commonly contain one to two parts per million in the ash compared to an average of 0.5 ppm in trees rooted in barren ground." The natural concentration of uranium minerals (principally carnotite) at these sites was not reported to have toxic effects on the native vegetation that was present. Connor and Shacklette (1975) reported uranium concentrations in 48 samples of soil from Wyoming and Montana to range from 1.7 to 7.0 ppm.

Under man-induced conditions—Brooks (1972) stated that the toxicity to plants was moderate; however, the evidence for this statement was not presented. Cultivated soils from 11 areas of commercial production of fruits and vegetables in the United States, although containing as much as 4 ppm uranium, caused no obvious damage to the produce growing thereon (Shacklette, unpub. data, 1978). Doubtless some uranium compounds of industrial origin are chemically toxic to plants under experimental conditions, but we have no evidence that uraniferous ores or mine tailings are chemically toxic.

Animals

Under natural dietary conditions—We have no reports of uranium toxicity caused by animals grazing on plant material from natural uraniferous locations, and we believe that toxicity from this kind of diet is unlikely. The uranium levels in vegetation are low (Brooks, 1972, gave the concentration in plant ash as 0.6 ppm); the areas of uraniferous deposits are usually small; and animals generally are not confined to grazing on these areas.

Under man-induced conditions—Wills (1949, p. 237) quoted from the literature that ". . . uranium is the most poisonous of all metals when injected intravenously or subcutaneously."He stated further, "When uranium compounds are given by mouth, the water-soluble salts are the most toxic forms of the metal, but such water-insoluble compounds as uranium oxide and sodium uranate are appreciably toxic because they are fairly soluble in dilute hydrochloric acid. When uranium compounds are applied to other surfaces of the body, the water-soluble salts appear to be the most toxic. The salts containing anions of strong acids tend to be corrosive." The primary effect of injection of uranyl salts is kidney failure; this starts a train of secondary effects leading to deterioration of other organs and blood, terminating in muscular apathy, progressive emaciation, excretory disfunction, coma, and de-

ath. These salts can also produce similar symptoms if inhaled as a dust concentration of 1.0 mg/m^3 of air (Wills, 1949). Uranium compounds in the air may be ingested by the animal swallowing mucus from the bronchial passages, licking its body, or eating feed contaminated with the compounds. Maynard and Hodge (1949) conducted extensive experimental studies of laboratory animals that were fed uranium compounds and provided the following classification (p. 310):

Non toxic (little effect was observed when the diet contained 20 percent) — UO_2, U_3O_8, UF_4.

Toxic (growth diminished when the diet contained 1 percent or less) — UO_3, UO_2Ac_2, UO_4, UCl_4, UO_2F_2, $UO_2(NO_3)_2 \cdot 6H_2O$.

They reported further that feeding the toxic compounds at levels of 20 percent of the diet frequently killed all the animals in a few days, and that for the more toxic compounds 2 percent killed most of the animals within a week. In general, non lethal doses of these compounds caused reduction in growth. The naturally occurring uranium compounds included in the above experiments can be summarized as follows:

Non toxic — The oxidation series from UO_2 (uraninite) to U_3O_8, sometimes referred to as pitchblende. The other compounds listed do not occur as natural minerals.

Toxic — The UO_3 radical occurs in the minerals becquerelite, masuyite, schoepite, and ianthinite. The other compounds listed do not occur in nature.

We have no reports of studies of ingested dosages of carnotite, $K_2(UO_2)_2(VO_4)_2 \cdot 1-3\,H_2O$, a common uranium ore, but the oxidation state of the uranium suggests that it may be non-toxic. Although the experiments reported above identified some of the uranium compounds as relatively non-toxic to rats, rabbits, and dogs, ingestion of these compounds by other species of animals (particularly, the ruminants) having high concentrations of hydrochloric acid in their gastric secretions (pH as low as 2) may present a toxicity hazard. These compounds could possibly be ingested with the soil on vegetation, or as water-suspended particles by animals grazing over uraniferous deposits or spoil, although we have no references to this condition having actually occurred.

Man

With the reference material we have at hand, it is best only to suggest toxic effects on man of naturally occurring uranium minerals as extrapolated from research data on animals. In this respect, the comments by Hodge (1949, p. 7–8) seem appropriate, "The not too satisfactory, but widely accepted, basis for expressing the toxicity is the collection of as much information about as many species as seems practical. Man would be expected to form a part of the series if all species were arranged from the most susceptible to the most resistant.

There are no rules of order about the susceptibility of a given species. . . . Nevertheless, these generalities can be offered:

1. Somewhere in the line-up of susceptibility, man must stand.
2. It has generally been the case that a given compound is highly toxic to many species, moderately toxic to many species or relatively non-toxic to many species.

Thus, by obtaining the relative toxicity of one of the uranium compounds in several species, the order of magnitude of the toxicity in man may be postulated. . . . In general, no better procedure has been suggested than obtaining the limits of toxic effect as related to dosage on a number of species and judging that the effects on man would be produced by doses of the same order of magnitude."

VANADIUM

[Considered nonessential for higher plants and most higher animals, although reported to be beneficial for some; essential trace element for some algae and other microorganisms, and for chicks and rats]

Plants

Under natural conditions—Pratt (1966b) stated that he had found no reports indicating either deficiency or toxicity of vanadium to plants under field conditions. Some food plants may accumulate high levels of vanadium without exhibiting toxicity symptoms, as is shown by the following data (maximum parts per million found in ash): snap bean, 700; cabbage, 50; tomato fruits, 30; and asparagus, 20 (Connor and Shacklette, 1975).

Under man-induced conditions—Literature reports indicate that vanadium is toxic to germinating seeds, but even more toxic at later stages of growth. Chiu (1953) reported that 500 ppm of vanadium oxide in culture solutions produced toxicity in rice seedlings and that 1,000 ppm killed all the seedlings. Cannon (1963) conducted plot experiments in a desert environment by adding sodium vanadate to the soil. After a few weeks' time, 840 ppm of vanadium was found in the soil-water solution, and no planted species was able to grow in the plots during the first season. By the second year after a winter of leaching, four species were grown and harvested from the plots which contained 140–560 ppm vanadium in the soil solution. The toxicity symptoms exhibited by these plants were extreme dwarfing and chlorosis. Pratt (1966b) suggested that vanadium toxicity may be indicated by two or more ppm vanadium (dry weight basis) in the tops of pea or soybean plants. He concluded that concentrations of 0.5 ppm or greater in nutrient solutions are toxic to plants, and that additions of the element to soils have produced toxicity for a variety of crop plants. As soluble vanadium, the following solution concentrations were reported as slightly toxic by Schroeder, Balassa, and Tipton (1963): 10–20 ppm for

soybeans, 26 ppm for beets, 40 ppm for barley, 20 ppm for wheat, and 22 ppm for oats.

Animals

Under normal dietary conditions—We have no reports of toxicity attributed to vanadium in feedstuffs.

Under man-induced dietary conditions—Vanadium is a relatively non toxic metal to animals. Schroeder, Balassa, and Tipton (1963) reported that 160 ppm in the diet of rats over an unspecified period of time was lethal owing to gastrointestinal irritation. Similar values were given by Bowen (1966), who listed 0.5 mg $V^{(V)}$ (vanadate) as toxic to rats in 10-g/day dry-weight diet (50 ppm) and 1.5 mg (150 ppm) as lethal, duration not given. The LD_{50} for mice given vanadium trioxide orally is reported from the literature as 130 mg/kg (National Research Council, 1974b), and for vanadium pentoxide and trichloride, as 23 mg/kg. Underwood (1971) gave the following concentrations and manifestations: >20–25 ppm vanadium in the diet resulted in growth depression in chicks; and 25 ppm was toxic to rats, whereas 50 ppm caused diarrhea and mortality. He listed the following relative toxicities for five elements to rats when fed 25-ppm diets: arsenic < molybdenum < tellurium < vanadium < selenium.

Man

Vanadium is not a particularly toxic metal to man. Not only is vanadium poorly absorbed by the gastrointestinal tract, but it results in minimal irritation to the lungs when inhaled. Curran and Burch (1967, p. 98) stated, "A specific toxicity from naturally occurring vanadium salts or those contacted in industry would seem to be minimal." There are in the literature, however, reports of workers who were exposed to vanadium oxide dust or aerosols for several days developing vanadium toxicosis (National Research Council, 1974b). Vanadium irritates the mucousal membranes and, therefore, symptoms of vanadium poisoning include eye, nose, and throat irritations; cough; wheezing; productive sputum; and "green tongue." Schroeder, Balassa, and Tipton (1963) fed patients 4.5 mg/day as the oxytartarovanadate for 16 months with no apparent toxicity. Underwood (1971) reported that experiments with ammonium vanadyl tartrate given orally to six subjects for 6–10 weeks produced no toxic effects other than some cramps and diarrhea at the larger dose levels.

ZINC

[Essential for plants and animals]

Plants

Under natural conditions—Zinc concentrations of 0.43–10.16 percent in New York peat soils were reported by Staker and Cummings

(1941) to be severely toxic to vegetable crops such as spinach, lettuce, and carrots. The effects of the several metals that commonly are associated with zinc in areas of natural enrichment cannot be distinguished with certainty by visual inspection; therefore, visible toxicity effects are often attributed to the zinc. A translation of a statement by Ernst (1974, p. 3) follows: "In nature soils rich in heavy metals are found over ore bodies that crop out at the surface or extend in the near-surface zone to within 30 m of the surface. . . . Because of the diversity of bedrock types, the distribution of the quality and quantity of heavy metals in the naturally metal-rich soil is quite variable. . . . At a concentration of at least 0.1 percent, all heavy metals have a strongly selective effect on vegetation. As a rule, zinc-rich soils (0.1–10 percent zinc) are distinguished also by having a high content of lead (0.1–3.2 percent), but not of cadmium." The following concentrations (ppm, dry weight) of zinc in leaf tissue are considered excessive: >300, Navel and Valencia oranges (Embleton and others, 1976); >300, avocado (Jones and Embleton, 1976); and 200, soybean (Ohki, 1977). Many reports in the literature of zinc toxicity to vegetation in natural environments are based on circumstantial evidence, in that the exact cause of the symptoms was not determined.

Under man-induced conditions—Contamination of vegetation with zinc, as well as with other heavy metals, is common in the vicinity of metal-working industries. For example, Ernst (1974) reported the concentrations in soil (mg/kg, dry soil) with distance from the sources as follows: zinc smelter, at 250 m, 4,500; at 400 m, 1,300; and at 4,000 m, 210; brass foundry, at 250 m, 4,520; at 400 m, 350; at 4,000 m, 173, and at 10,000 m, 91. These zinc values contrast with the geometric mean zinc concentration in U.S. soils of 44 ppm (Shacklette and others, 1971). Fields and orchards fertilized over a long period of time have been reported to have accumulated toxic levels of zinc when the soil has been acid. Keisling and others (1977) reported (from studies of peanuts in acid fields) tentative toxicity critical values of 12 mg/kg zinc for soil (acid extractable) and 220 mg/kg for plant tissue. Thorne, Laws, and Wallace (1942) found that total zinc differentiated zinc-deficient soils from those not deficient in zinc as well as did Hibbard's extraction procedure, but researchers prefer to judge deficiencies and toxicities on the basis of soluble zinc in soils. The zinc content of plant leaves as indicative of nutritional sufficiency and toxic excesses was summarized by Chapman (1966) as follows: deficiency levels are characterized by less than 20–25 ppm in dry matter; ample but not excessive levels are 25–150 ppm; and amounts greater than 400 ppm may indicate zinc excess. However, there is a wide range in content of zinc among plant species; likewise, tolerance to high levels of soil zinc ranges widely. For example, Robinson, Lakin, and Reichen (1947)

reported that a good indication of zinc-mineral outcrops is the presence of luxuriantly growing ragweed when other vegetation is stunted. At the location which they studied, total zinc in the soil was 12.5 percent.

Animals

Under normal dietary conditions—We have no reports of zinc toxicity to animals resulting from the consumption of ordinary feedstuffs. Underwood (1971, p. 242–243) stated, "Zinc is relatively nontoxic to birds and mammals and a wide margin of safety exists between normal intakes and those likely to produce deleterious effects. Rats, pigs, poultry, sheep, and cattle exhibit considerable tolerance to high intakes of zinc, the extent of the tolerance depending upon the composition of the basal diet, particularly its content of minerals known to affect zinc absorption and utilization, such as copper, iron, and cadmium."

Under man-induced dietary conditions—Zinc compounds are relatively non toxic to organisms, particularly to mammals. Schroeder and others (1967) reported that a diet for laboratory animals containing 0.25 percent (2,500 ppm) zinc salts is apparently non toxic. The following summary was obtained from Underwood (1971): rats—dietary intake of 2,500 ppm zinc, no discernible effects; 5,000 ppm, severe growth depression and severe anemia; and 10,000 ppm, heavy mortality; weanling pigs—dietary intake of 1,000 ppm zinc, no ill effects; higher levels, growth depression; and 4,000–8,000 ppm, high mortality; lambs—dietary levels of 1,000–1,500 ppm zinc, depressed feed consumption, reduced gains, and increased mineral consumption; steers—dietary levels of zinc of 500 ppm or less, no detrimental effects; 900 ppm, reduced gains and lower feed efficiency; and 1,700 ppm, in addition, a depraved appetite with excessive salt and mineral consumption and wood chewing. Bowen (1966) listed 50 mg Zn^{2+} in a 10-g/day dry-weight diet (5,000 ppm) as toxic to rats and 150 mg (15,000 ppm) as lethal, duration not given. A daily dose of 30–40 mg/kg produced severe chronic poisoning in young calves within 1 month (Clarke and Clarke, 1967, p. 68–69). The lethal dose for mature cattle is about 20 times this amount.

Man

Unlike the aberrant syndromes for most of the essential trace elements, no primary disorder of zinc metabolism has been described for humans. Moderate zinc toxicity was reported by Van Reen (1966) in individuals who breathed zinc oxide for as much as 12 minutes at a calculated zinc concentration in air of 600 mg/m^3. Underwood (1971) noted that a wide margin of safety existed between normal zinc intakes and those likely to produce deleterious effects. Acute zinc poisoning is commonly reported, however, when acidic foods stored in galvanized iron containers have been ingested. Schroeder and others (1967) specu-

lated that cadmium intoxication may also be associated with these cases. Thomas (1973) listed 50–500 mg/kg body weight of zinc salts (chloride, sulfate, acetate, and others) as probably lethal to adults. Bergqvist and others (1977) reported that 6 g of zinc chloride and 45 g of zinc sulfate taken orally are considered lethal doses; however, 1 g or more of most zinc salts results in vomiting and therefore expulsion of the intoxicant.

OTHER ELEMENTS THAT POSSIBLY PRESENT A TOXICITY HAZARD

The toxicity to organisms of some elements at levels that might be expected to occur in natural or polluted environments has been indicated by reports in the literature, but the data are too meager for adequately appraising the possible hazard. Brief notes on these elements follow.

BARIUM

[Considered nonessential for organisms]

We have only one report (Chaudhry and others, 1977) that gives toxicity levels of barium in plants. These authors stated, "Barium is ubiquitous in soils and all plants contain a small quantitity of it usually in levels of around 4 to 50 μg/g dry weight of plants. It is a constituent of geothermal brines and, hence, there is a possibility that it may become spilled into the environment in case of accidental release. . . . Barium levels of about 2% in bush bean leaves and 1% in barley leaves decreased yields considerably. The levels were obtained after application of 2,000 μg Ba per g of soil as $Ba(NO_3)_2$ with equivalent nitrate added in controls." Brooks (1972) stated that barium toxicity to plants was moderate.

We have no reports of barium toxicity to animals or man.

GERMANIUM

[Considered nonessential for organisms]

Hörmann (1970, p. 32-L-1) reported, "The Ge content of plants never exceeds 10 ppm Ge in ash. Experimental investigation on the Ge content of plants (Kudelašek, 1960) gave the results that at 10 ppm the toxic properties of Ge cause death of plants. The following plants were used in the experiments: *Equisetum arvense, Equisetum hiemale, Picea excelsa, Secale cereale.*" Bidwell (1974, p. 246) reported, "Germanium inhibits silicon metabolism and may be toxic to organisms which require or use silicon, such as diatoms, rice, and tobacco." Shacklette

and Connor (1973) reported 15 ppm in ash of 1 sample of Spanish moss, the only 1 of 123 samples having detectable concentrations of germanium. This sample probably was affected by urban and industrial pollution. Dry material of alpine fir needles was found to contain 5 ppm germanium (Gary C. Curtin, written commun., 1976). Mr. P. F. M. Paul (written commun., 1953), in discussing the germanium research of Dr. Hans Brauchli, stated that Dr. Brauchli had found 5 percent germanium in ash of lettuce, the highest value in any plant material he had examined, but that more frequently values of 2 percent to hundredths of a percent were found. On the bases of our experience, these concentrations seem much too high, and we question the accuracy of his analyses. Brooks (1972) gave 5 ppm as the concentration of germanium in plant ash and said the toxicity to plants was slight. Some organic compounds of germanium are toxic to animals (Sax, 1975), but poisoning from naturally occurring compounds seems unlikely.

MAGNESIUM

[Major essential element for plants and animals]

True magnesium toxicity to plants is apparently unknown. Kelley (1948) cited an example of a soil near Napa, Calif. in which more than 90 percent of the cation exchange capacity was saturated with magnesium and stated that this soil was almost completely unproductive. In this case, the inhibition of vegetation was due to deprivation of other elements induced by excess magnesium, not to the magnesium itself. Sievers (1924) reported that magnesium oxide dust from a magnesite processing plant in the State of Washington was deposited on the surface of adjacent soil in quantities great enough to be toxic to plants. In sand culture experiments, Carolus (1935) showed that magnesium toxicity in potatoes resulted when magnesium was extremely high and potassium was extremely low.

We have no reports of magnesium toxicity in animals. High levels of available soil potassium reduce magnesium uptake by plants, especially in low magnesium soils, and this condition may produce pastures (especially grasses) that are extremely low in magnesium (<0.2 percent, dry weight basis). Animals grazing on this grass may develop a nutritional disease called hypomagnesaemia or grass tetany (Mortvedt and Cunningham, 1971). Whereas the inhalation of fumes of freshly sublimed magnesium oxide may cause "metal fume fever" in man there is no evidence that magnesium itself produces true systemic poisoning (Sax, 1975).

RUBIDIUM

[Considered nonessential for organisms]

Steinbach (1962) stated that rubidium appears not to be an essential component of living matter, but rather a toxic agent that may partly substitute for potassium. He reported rubidium in non-woody plants to range from 11.3 to 36.7 ppm (dry weight basis). Smales and Salmon (1955) found rubidium concentration in seven species of marine algae to range from 0.61 to 2.4 ppm of wet plants. Connor and Shacklette (1975) reported rubidium concentration in ash of hickory and oak stems to range from 18–40 ppm. Although Sax (1975) reported rubidium chromate, rubidium chromium sulfate, and rubidium dichromate as recognized carcinogens and rubidium hydroxide as highly caustic and toxic, the kinds and concentrations of rubidium compounds in plants and other natural materials most likely are not hazardous. We have no reports that poisoning of animals or man has occurred from natural occurrences of rubidium.

TELLURIUM

[Considered nonessential for organisms]

The following information was obtained mainly from the excellent review of the literature on tellurium toxicity by Cerwenka and Cooper (1961). Apparently, elemental tellurium (like so many of the metals) is biologically inert whereas salts of tellurium yielding telluride and tellurate ions are somewhat toxic. These authors reported no serious cases of industrial tellurium intoxication but warned against exposure to tellurium vapors or tellurium oxide. Ingested tellurium salts are reduced to elemental tellurium in the gut by bacteria. Much of the absorbed tellurium is converted to dimethyl tellurdie, which is volatile and readily lost via exhaled air. This compound has a distinctive garliclike odor which, along with other symptoms such as nausea, giddiness, and metallic taste, can indicate tellurium poisoning in humans. These authors reported that as little as 0.5 μg of tellurium dioxide given orally produced "garlic breath" for 30 hours and 15 mg for 237 days. Rats fed 750 ppm tellurium showed no change in growth rates, whereas rats fed 1,500 ppm showed some reduction in growth rate but no pathological or anatomical abnormalities.

TITANIUM

[Considered nonessential for organisms]

We have only one report (Wallace, Alexander, and Chaudhry, 1977) that gives quantitative data on titanium toxicity to plants. Titanium was reported to be somewhat more mobile than vanadium and to cause

considerable yield decrease in bush bean at 10^{-4} *M*; leaf, stem, and root titanium concentrations, respectively, were 202, 48, and 2,420 μg/g (dry weight). Symptoms were chlorosis, necrotic spots on leaves, and stunting.

We have no reports of titanium toxicity to animals and man.

TUNGSTEN

[Considered nonessential for organisms]

Krauskopf (1970, p. 74-L-1) stated, "Like other heavy metals, tungsten in more than traces is poisonous to most organisms. An important biogeochemical role for the element is therefore hardly likely, but minor concentrations in organic processes is certainly possible and is strongly suggested by the fragmentary data on carbonaceous metamorphic rocks." The toxicity of tungsten to plants was said by Brooks (1972) to be moderate, and the concentration in plant ash, 0.5 ppm. Dekate (1967) observed that plants growing in a mineralized zone contained 2–18 times the concentration of tungsten that was considered to be the background value (2.7 ppm). Tungsten in the ash of trees was reported as follows: Douglas-fir stems, 50 ppm; dwarf juniper leaves, 50 ppm, stems, 100 ppm; and alpine fir stems, 5–50 ppm (Gary C. Curtin, written commun., 1976). Connor and Shacklette (1975) reported tungsten concentrations in ash of single samples of the following trees: sassafras leaves, 50 ppm; and sweet gum stems, 30 ppm, leaves, 70 ppm. They found 1,000 ppm tungsten in one of 492 samples of soil from western United States.

We have only one reference giving quantitative data on the effects of feeding tungsten to animals. Sax (1975, p. 1221) stated, "Tungsten compounds are considered somewhat more toxic than those of molybdenum. However, industrially, this element does not constitute an important health hazard. Exposure is related chiefly to the dust arising from the crushing and milling of the two chief ores of tungsten, namely, scheelite and wolframite. There is very little published with reference to its toxicity. The feeding of 2, 5, and 10 percent of diet as tungsten metal over a period of 70 days has been shown to be without marked effect upon the growth of rats, as measured in terms of gain in weight. . . . Recent studies have failed to indicate any serious toxic effect following the inhalation or ingestion of various tungsten compounds, although heavy exposure to the dust or the ingestion of large amounts of the soluble compounds produces a certain rate of mortality in experimental animals." Judged from this report, tungsten probably does not constitute an important environmental hazard to animals and man.

ZIRCONIUM

[Considered nonessential for organisms]

Zirconium is a common constituent in ash of some kinds of plants and is found less frequently in others. A part of the reported concentrations in plant samples may be due to soil contamination, as indicated by the fact that soil values nearly always are greater than plant values. Parts of some plants that are protected from surface contamination by soil, however, have been found to contain measurable concentrations of zirconium. For example, Connor and Shacklette (1975) reported pea and lima bean seeds that had been removed from the pods to contain 20–70 ppm zirconium in ash. They also reported stems of trees and shrubs that contained 20–500 ppm zirconium in ash, part of which may have been the result of contamination by soil.

Sax (1975, p. 1257) stated, "Zirconium is not an important industrial poison. Deaths in rabbits have been caused by intravenous injection of 150 mg/kg of body weight. Most zirconium compounds in common use are insoluble and considered inert. Pulmonary granuloma in zirconium workers has been reported and sodium zirconium lactate has been held responsible for skin granulomas. Avoid inhalation of Zr containing aerosols which can cause lung granulomas."

Zirconium selenate was listed by Sax (1975) as a recognized carcinogen, and sodium zirconium lactate, in inhalation experiments with rabbits, produced bronchiolar abcesses, lobar pneumonia, and peribronchial granulomas.

Because of the very low solubility of naturally occurring zirconium compounds and the low concentrations of zirconium in plants, this element seems unlikely to constitute an environmental hazard under most conditions.

RARE EARTH ELEMENTS (LANTHANIDES)

[Considered nonessential for organisms]

Certain of the rare earth elements have been found toxic to experimental animals if ingested, or to cause illness in humans on inhaling dust containing the element or its compounds. Because of their infrequent occurrence in high concentrations in natural materials, they are not expected to be of great environmental concern, and the greatest hazard probably is occupational exposure or reactions from pharmaceutical applications. However, experimental studies on the effects of these elements on organisms seem to be few and to have largely excluded plants and humans; therefore, an adequate appraisal of potential hazards cannot be made. We have no reports of chemical toxicity, either naturally occurring or experimentally produced, in plants.

Brief summaries of known toxicities of these elements, based largely on the reports of Sax (1975), follow. Radiation effects are omitted.

CERIUM

The insoluble salts of cerium are said to be non toxic, even in large doses. Doses of 0.05–0.5 g are used to prevent vomiting in pregnancy. The toxicity of cerium compounds may be taken to be that of elemental cerium, except when the anion has a toxicity of its own. Cerium tartrate has a direct injurious action on the hearts of small animals. The effects on the nervous system of humans through inhalation may preclude the use of welding rods or compounds that contain the rare earth elements. Salts of cerium increase the blood coagulation rate.

Cerium in rocks and soils commonly ranges from <150 to 300 ppm; it is seldom reported above 150 ppm in plant ash, but extreme values of 300 ppm in tomatoes and 700 ppm in hickory trees were found by Connor and Shacklette (1975). Duke (1970) reported the following concentrations in dry material of food plants: breadfruit, 0.1 ppm; taro, 0.5 ppm; and cassava, 0.05 ppm.

LANTHANUM

This element is reported to delay blood clotting, leading to hemorrhages, and has caused liver injury in experimental animals. Inhalation and ingestion cause readily reversible symptoms which disappear after the end of exposure. Compounds of lanthanum cause somewhat more severe, but not lethal, reactions.

Concentrations in rocks and soils are frequently less than 30 ppm but, when determinable, generally range from about 30 to 200 ppm. The lanthanum content of trees and shrubs generally range from <30 to 300 ppm in ash, except for hickory trees which have concentrations of as much as 2,000 ppm (Connor and Shacklette, 1975).

DYSPROSIUM, GADOLINIUM, HOLMIUM, NEODYMIUM, PRASEODYMIUM, SAMARIUM, TERBIUM, AND THULIUM

As lanthanides, these elements are suspected of having at least a moderate degree of toxicity to animals and humans, but experimental evidence is very limited. Some are known to depress blood coagulation, leading to hemorrhage, and the others are suspected of causing this effect. We have no reports of natural exposures or experimental treatments causing toxicity in plants.

These elements are seldom detected in soils and plants by routine analytical methods; and, when detected, concentrations are generally low.

ERBIUM, EUROPIUM, AND LUTETIUM

We have no reports of toxicity in plants, animals, or man attributable to these elements.

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APPENDIX

[Common plant names with corresponding scientific names]

Common names, as used in the text, are listed alphabetically. Generally, only common names were given in the publications consulted in preparing this report. For most of these names there is no doubt as to their proper corresponding scientific names; for others, only the genus name is given here. If several cultivars of a species are listed, they are so identified. The common names used are those generally applied in the United States.

Alfalfa, *Medicago sativa* L.
Almond, *Prunus amygdalus* Stokes
Alpine fir, *Abies lasiocarpa* Nutt.
Anemone, *Pulsatilla patens* Mill.
Apple, *Pyrus malus* L.
Apricot, *Prunus armeniaca* L.
Asparagus, *Asparagus officinalis* L.
Aspen, *Populus tremuloides* Michx.
Avocado, *Persea gratissima* Gaertn. f.

Barley, *Hordeum vulgare* L.
Bean, *Phaseolus vulgaris* L.
Bedstraw, *Galium* sp.
Beet, *Beta vulgaris* L.
Birch, *Betula* sp.
Black gum, *Nyssa* sp.
Black medic, *Medicago lupulina* L.
Bracken, *Pteridium aquilinum* (L). Kuhn
Breadfruit, *Artocarpus altilis* (Parkinson) Fosberg
Broadbean, *Vicia faba* L.
Buckwheat, *Fagopyrum sagittatum* Gilib.
Bur oak, *Quercus macrocarpa* Michx.
Bush bean, *Phaseolus vulgaris* L.

Cabbage, *Brassica oleracea* var. *capitata* L.
Camellia, *Camellia japonica* L.
Carnation, *Dianthus caryophyllos* L.
Carrot, *Daucus carota* var. *sativa* DC.
Cassava, *Manihot esculenta* Crantz
Cauliflower, *Brassica oleracea* var. *botrytis* L.
Celery, *Apium graveolens* var. *dulce DC.*
Clover, Trifolium pratense L.
Club moss, *Lycopodium obscurum* var. *dendroideum* (Michx.) D.C. Eat.
Corn, *Zea mays* L.
Cotton, *Gossypium* sp.
Cucumber, *Cucumis sativus* L.

Douglas-fir, *Pseudotsuga menziesii* (Mirb.) Franco
Dwarf juniper, *Juniperus communis* subsp. *nana* Syme

Eastern white pine, *Pinus strobus* L.
Endive, *Cichorium endivia* L.
Engelmann spruce, *Picea engelmannii* Parry
Eryngium, *Eryngium* sp.
Flax, *Linum usitatissimum* L.
Fig, *Ficus carica* L.
French bean, *Phaseolus vulgaris* L. cultivar

Grape, *Vitis vinifera* L.
Grapefruit, *Citrus paradisi* Sw.

Hickory, *Carya* sp.

Kale, *Brassica oleracea* var. *acephala* DC.
Kidney bean, *Phaseolus vulgaris* L. cultivar

Labrador tea, *Ledum decumbens* (Ait.) Lodds
Lactic acid bacteria, *Lactobacillus* sp.
Leek, *Allium porrum* L.
Lemon, *Citrus limonia* Osbeck
Lespedeza, *Lespedeza* sp.
Lettuce, *Lactuca sativa* L.
Lima bean, *Phaseolus limensis* Macf.
Limber pine, *Pinus flexilis* James
Lodgepole pine, *Pinus contorta* var. *latifolia* Engelm.
Low juniper, *Juniperus communis* subsp. *nana* (Willd.) Syme
Lupine, *Lupinus* sp.

Mandarin, *Citrus nobilis* var. *deliciosa* Swingle
Myrtle blueberry, *Vaccinium myrtillus* L.

Navy bean, *Phaseolus vulgaris* L. cultivar

Oak, *Quercus* sp.
Oats, *Avena sativa* L.
Olive, *Olea europea* L.

Onion, *Allium cepa* L.
Orange, *Citrus sinensis* Osbeck

Pea, *Pisum sativum* L.
Peach, *Prunus persica* L.
Peanut, *Arachis hypogaea* L.
Pear, *Pyrus communis* L.
Pecan, *Carya illinoensis* Koch
Pepper, *Capsicum frutescens* var. *grossum* Bailey
Perennial ryegrass, *Lolium perenne* L.
Persimmon, *Diospyros virginiana* L.
Phyllodoce, *Phyllodoce empetriformis* (Smith) D. Don
Plum, *Prunus* sp.
Ponderosa pine, *Pinus ponderosa* Laws.
Potato, *Solanum tuberosum* L.
Prince's plume, *Stanleya pinnata* (Pursh) Britt.
Prune, *Prunus* sp.

Radish, *Raphanus sativus* L.
Ragweed, *Ambrosia artemisiifolia* L.
Red cedar, *Juniperus virginiana* L.
Redtop, *Agrostis alba* L.
Rice, *Oryza sativa* L.
Rose, *Rosa* sp.
Rutabaga, *Brassica napobrassica* Mill.
Rye, *Secale cereale* L.

Sassafras, *Sassafras albidum* (Nutt.) Nees
Sheep fescue, *Festuca ovina* L.
Snap bean, *Phaseolus vulgaris* L. cultivar
Sorghum, *Sorghum vulgare* Pers.
Soybean, *Glycine max* Merr.
Spanish moss, *Tillandsia usneoides* L.
Spinach, *Spinacia oleracea* L.
Subalpine fir, *Abies lasiocarpa* Nutt.
Sudan grass, *Sorghum halepense* (L.) Pers.
Sugar beet, *Beta vulgaris* L. cultivar
Sweetclover, *Melilotus* sp.
Sweet gum, *Liquidambar styraciflua* L.
Sweetleaf, *Symplocos tinctoria* (L.) L'Her.
Sweet potato, *Ipomea batatas* L.

Taro, *Colocasia esculenta* Schott.
Tea, *Thea sinensis* L.
Timothy, *Phleum pratense* L.
Tobacco, *Nicotiana tabacum* L.
Tomato, *Lycopersicum esculentum* Mill.
Toadflax, *Linaria triphylla* Jacq.
Turnip, *Brassica napus* L.
Walnut, *Juglans regia* L.
Wheat, *Triticum aestivum* L.
White birch, *Betula papyrifera* Marsh.

Yeast, *Saccharomyces* sp.

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