TRACE ELEMENT TOXICITY RELATIONSHIPS TO CROP PRODUCTION AND LIVESTOCK AND HUMAN HEALTH: IMPLICATIONS FOR MANAGEMENT

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ABSTRACT

In nature, trace element toxicities occur in all living organisms. The consequences of these toxicities have been described in crops, livestock and humans. In some instances, the toxicities are a direct consequence of the organism’s position in the food chain and their environment, while in others, they are based upon genetic abnormalities resulting in physiological impairment. Nutrient toxicities in crops are more frequent for manganese (Mn) and boron (B) than for other nutrients. Manganese toxicity is found on acid soils in many parts of the world. Boron toxicities occur in irrigated regions where the well or irrigation waters are exceptionally high in B. Most other nutrient toxicities occur when large amounts of nutrients in question have been added in waste, e.g., sewage sludge. Crops grown near mines and smelters are prone to nutrient toxicities. Generally, the symptoms of toxicity in crops occur as burning, chlorosis and yellowing of leaves. Toxicities can result in decreased yield and/or impaired crop quality. Toxicity levels of trace elements range from 20 to 50 μg g⁻¹ for copper (Cu) and B to several hundred μg g⁻¹ for Mn, molybdenum (Mo) and zinc (Zn). With the exception of Mo, toxicity of other nutrients can be reduced by liming. Following recommended rates of fertilizers and the safe and controlled use of waste materials, such as sewage sludge and coal fly ash, should reduce metal loading and nutrient toxicity in crops. Use of crop species and genotypes less susceptible to toxicity are recommended where toxicity is suspected. Toxicities of trace elements in animals are caused by the consumption of either feeds or grazing on pastures with high contents of the element in question. Accidental excess applications of minerals in grain mixes or oral ingestions of elements have been described as causing toxicity. Some toxicities, e.g., of Mo result in deficiency of other elements such as Cu. Some of the most toxic elements for livestock include Cu, lead (Pb), mercury (Hg), Mo, and selenium (Se). Under certain conditions, toxicities of arsenic (As), cobalt (Co), fluoride (F), iodine (I), iron (Fe), Mn, and Zn have also been reported. Symptoms of toxicity have been described in detail in the animal section. Trace elements, when in excess quantities, accumulate chiefly in the blood, liver, and kidneys. Measures of control for various trace element toxicities include removal of animals from affected areas or removal of the source of toxicity; gastric lavage and the specific use of oral doses of salts and chelates.
depending upon the element in question. Trace element/metal toxicities in humans are not common under normal conditions. Most toxicities are caused by environmental and/or genetic abnormalities, from excessive intake, by deliberate or accidental overdose, or from induced deficiencies (e.g. excess Zn causing Fe deficiency). Among the elements causing relatively frequent cases of toxicity are Pb, cadmium (Cd), Hg, Cu, Zn, and Fe. Selenium toxicity is generally limited to those areas/regions of the world, e.g. in certain parts of China, where soils with abnormally high Se content produce food crops containing highly toxic Se concentrations. Effective measures to control metal toxicities include gastric lavage, resuscitation, and the use of chelating agents in the acute phase. Protective legislation against the use of metal alloy utensils used for cooking is the long-term control strategy.

INTRODUCTION

Toxicity in crops may be defined as a situation in which a potentially harmful substance accumulates in the plant tissue to a level affecting its optimal growth and development. Increased industrial growth has led to the introduction of large quantities of nickel (Ni), for example, in the environment which causes potential hazards to animals and human health through their relative positions in the food chain (Singh et al., 1990). Certain essential plant nutrients as well as non-essential elements can be absorbed by plants in sufficient quantities to be toxic. Crops with toxic nutrient content when consumed by animals and humans caused toxicities in them. Essential trace elements that are known to occasionally produce toxicity in plants are Mn, Cu, B, Mo and Cl (Bennett, 1993). Among the non-essential elements, toxicity of Ni is the most prevalent (Singh et al., 1990), but symptoms of most non-essential elements are difficult to define or describe.

If the early symptoms of toxicity (phytotoxicity) in crops persist through maturity, yield reduction is the most important measure of phytotoxicity. Some toxicities are related to natural factors such as regional soils high in certain nutrient concentrations (Severson and Gouch, 1983) or irrigation water containing excess quantities of a nutrient, for example, B (Neilsen et al., 1991). Soils developed over serpentine deposits are high in chromium (Cr) and Ni (Proctor and Woodell, 1975), and cinnar deposits (and mine tailings) contribute to high Hg background levels (Harsh and Doner, 1981). However, anthropogenic factors are more common and can include: additions of metal sludge, one of the main sources of heavy metal contaminants (de Villarroel et al., 1993), non-ferrous metal smelters in Bulgaria and other parts of the world (Zheljazkov and Nielsen, 1996), coal fly ash (Page et al., 1979) and accidental applications of high levels of nutrients/metals in addition to contribution from mineral weathering of parent material.

Whenever land becomes contaminated with trace metals, there are usually two or more of these in excess (Wallace and Wallace, 1992). Therefore, data must be obtained for many elements and their distribution in different parts of the plants to understand the effect of various interactions on toxicity. The nature of interactions varies with soil pH, soil texture, presence of salinity, presence of metals in toxic quantities and soil organic matter levels (Wallace and Wallace, 1992). Nickel concentrations as low as 1-2 μg g⁻¹ have been shown to be toxic to some species of plants (Mishra and Kar, 1974). High concentrations of Ni have been found in plants and soils near Ni mines and smelters and toxic effects on vegetation have been recorded (Temple and Bisessar, 1981).

Crops containing excess amounts of trace elements, when consumed by livestock and humans can result in toxicity. The most toxic effects in livestock appear to be dependent upon different factors (Fraser, 1986). Consumption of feeds high in Mo
Air pollutants from cigarette smoke, industrialization, house paints, house dust, vehicle emissions, and population growth are some of the factors responsible for Cd and Pb toxicity in humans (Stewart-Pinkham, 1989). Metal toxicity cases in humans have also been attributed to cases of overdose or therapeutic use, e.g. high doses of Zn (van Campen, 1991). Toxicities of Cu to humans are rare and are usually associated with consumption of acidic food or drink that had a prolonged contact with Cu containers or with ingestion of gram quantities of Cu salts in suicide attempts (van Campen, 1991). Iron toxicity in humans occurs primarily from ingestion of mineral supplements containing the divalent form of Fe (Olson and Becker, 1990).

This review includes discussion on trace element toxicities, toxic levels, toxicity symptoms and implications for management practices of crops, livestock, and humans.

**DISCUSSION**

**Crops**

Trace elements or heavy metal toxicity can cause excessive accumulation or depletion of nutrient elements that may be deleterious to the plants in addition to other primary damage caused by the heavy metal ions (Brune and Dietz, 1995). For example, Ni, Cu, and Cd can induce Fe chlorosis in different plant species (Alcantara et al., 1994). Vascular-arbuscular mycorrhizal (VAM) colonization in acid weathered coal fly ash suppressed the growth of cucumbers which was related to toxic levels of Zn (Dosskey and Adriano, 1993).

The term phytotoxicity is normally associated with the accumulation of potentially harmful substances or toxic nutrients in the plant tissue to a level affecting its optimal growth and development. In order to positively confirm an incidence of metal/nutrient toxicity, one must make certain that plants have sustained injuries, that a potentially phytotoxic element has accumulated in the plant tissue, and that the observed abnormalities are not due to other disorders of plant growth (Chang et al., 1992).

Often the polluted soils are the source of toxic elements in crops. Toxicities in crops due to B, Cd, Cu, chromium (Cr), Fe Mn, Mo, Ni, and Zn, their possible causes, symptoms, and means to alleviate such symptoms are discussed in this section.

**Boron**

Boron toxicity chiefly occurs under two conditions: its natural occurrence in irrigation water or due to accidental applications of too much B in treating B deficiency. Boron toxicity in arid and semi-arid regions is frequently associated with saline soils, but it most often results from the use of high B irrigation waters. In the United States, the main areas of high B waters are along the west side of the San Joaquin and Sacramento Valleys in California (Branson, 1976). The high B content of water in wells results in a high B content in the soil, especially under conditions of poor drainage.

Large additions of materials high in B - for example, compost - can also result in B toxicity in crops (Gupta et al., 1973; Purves and MacKenzie, 1973). Soil type and texture have a significant effect on the status of B in plants. In a study on alfalfa (Medicago sativa L.), yields were significantly reduced with increased B application rate in sandy loam and loam soils whereas no yield reductions were observed in the...
silt loam soil (Gestring and Soltanpour, 1987). These yield reductions were associated with very high plant B levels of 800 to 900 mg kg\(^{-1}\).

**Toxicity Symptoms**

The B toxicity symptoms are similar for most plants. Because of the slow transport of B in the plant, symptoms generally appear on the older leaves and consist of marginal and tip chlorosis, which is quickly followed by necrosis (Shorrocks, 1974). Detailed toxicity symptoms for various crops are described by Gupta (1993a). Toxicity B levels range from 10-50 \(\mu g\) g\(^{-1}\) in the vegetative tissue of sensitive crops such as cereals and snapbeans (*Phaseolus vulgaris* L.) to over 200 \(\mu g\) g\(^{-1}\) in tolerant crops such as sugar beets (*Beta vulgaris* L.), rutabaga (*Brassica napobrassica* Mill.), and cucumber (*Cucumis sativus* L.) as summarized by Gupta (1993b).

**Possible Toxicity Control Measures**

Recommended rate of B applications at 1 to 4 kg ha\(^{-1}\) depending upon the crop, method of application and soil type can eliminate B deficiency without causing B toxicity under most conditions (Mortvedt and Woodruff, 1993). Under certain conditions leaching, liming, and applications of nitrogen (N) can reduce B toxicity (Gupta, 1993c).

**Cadmium**

Cadmium is a heavy metal of significant concern because its accumulation in plants may be harmful to human health (Mortvedt, 1996). Cadmium is not toxic to plants but it can accumulate to levels toxic to humans and livestock. High rates of Cd have been found to impede absorption and/or translocation of Fe, Mn, and Zn in the root and Mn in the fruit (Moral et al., 1994).

Results of a study by Dudka et al. (1994) showed that the Cd concentration in the vegetative parts of wheat plants (25 times higher than the control level) were not harmful to the plants. Soil applied Zn has been found to decrease the Cd concentration in wheat grain but foliar applied Cu can cause Zn loading in the leaf without major effect on plant Cd concentration (Choudhary et al., 1995). On the other hand, in the presence of very high Zn concentrations in soil, Cd level in wheat (*Triticum aestivum* L.) plants was significantly increased (Dudka et al., 1994).

Cadmium application rates as high as 50 \(\mu g\) g\(^{-1}\) did not cause any decrease in spring wheat yield (Dudka et al., 1994). However, the resulting grain containing 4 \(\mu g\) Cd g\(^{-1}\), if used as a feed source, would cause toxicity in livestock. Liming of soils decreases the availability of Cd to plants and may be useful to a certain extent in reducing Cd toxicity.

**Copper**

Data on Cu toxicity in crops are limited. Baker (1974) reported that Cu toxicity to some plants, on some soils, can be expected when the amount added over a period of time exceeds 150 to 400 \(\mu g\) Cu kg\(^{-1}\). A potential for Cu phytotoxicity from excess Cu application exists because only a small proportion of applied Cu is lost by leaching, and because reversion of applied Cu to unavailable forms is relatively slow in soils (Vitosh et al., 1980; Varvel et al., 1983). In certain cases, continued long-term application of Cu as a fungicide has resulted in Cu toxicity and extreme care should be taken to prevent further Cu application.

According to Wallingford and Simkins (1977), plant tissue Cu contents of 21 mg kg\(^{-1}\) and above could indicate excessive or toxic accumulation of the element. Rao
and Deshpande (1971) reported onion (Allium cepa L.) yield decreases when the rate of applied Cu exceeded 13.4 kg Cu ha$^{-1}$. Toxicity levels of Cu in crops vary greatly and range from 10 μg g$^{-1}$ in cucumber to 70 in corn as summarized by Gupta (1997a). Copper rates as high as 54 kg ha$^{-1}$ on a soil with pH 6.7 were not toxic to snapbeans while rates of 486 kg Cu ha$^{-1}$ decreased yields (Walsh et al., 1972).

The metabolic role of Cu in plants appears to limit the plant Cu concentration that aerial plant tissues can tolerate before the plant roots are killed. This serves to limit the maximum Cu concentration found in plant leaves and stems to about 20 to 30 μg g$^{-1}$ with few exceptions (Dragun, 1976).

**Toxicity Symptoms**

Copper toxicity causes reduced branching, thickening, and abnormally dark coloration in the rootlets of many plants (Reuther and Labanauskas, 1966). Copper toxicity can result in poor growth of head and reduced yield in lettuce (Lactucasativa L.) (MacKay et al., 1966); in wilting of plants and darkened root tips in cotton (Sowell et al., 1957); in chlorotic leaves and stunted root development in beans (Phaseolus spp.) and in inhibition of root growth in corn (Zea mays L.) (Forbes, 1917).

**Possible Toxicity Control Measures**

The recommended cumulative limits for Cu application to crop land are 140, 280, and 560 kg Cu ha$^{-1}$ for soils with cation exchange capacity (CEC) of <5, 5-15, and >15 cmol$_c$ kg$^{-1}$. A pH of 6.5 or above must be maintained where sewage sludge is incorporated into soil to decrease Cu availability (Martens and Westermann, 1991).

**Chromium**

Chromium can be toxic to plants in its common oxidation states, Cr(III) and Cr(VI) (Bartlett and James, 1988). However Cr(III), the form normally found in plants, is essential for human nutrition (Huffman and Allaway, 1973). Toxic levels of Cr may accumulate in soils amended by Cr-rich sewage sludge or wastes from the dyeing and tanning industry (Srivastava and Gupta, 1996).

Average values of Cr in most soils range from 20 to 220 mg kg$^{-1}$ and are present in lowest quantities in podzols and highest in fluvisols and chernozems (Kabata-Pendias et al., 1992). Translocation of Cr from soil to crops is generally insignificant, and lowest Cr levels are found in grain especially in corn grain at 70 to 1700 μg kg$^{-1}$ (Juste and Mench, 1992). Likewise, Sauerbec (1991) reported that the Cr content in grain, chaff and straw are extremely low and not related to soil content.

**Toxicity Symptoms**

Toxicity symptoms of Cr include restriction in the growth of roots and shoots and chlorosis in leaves (Pratt, 1966). In monocots, leaves become narrow and appear brownish-red with small necrotic spots in oats and purple-green in maize (Zea mays L.). Chromium toxicity produces chlorosis due to a decrease in the chlorophyll and carotenoid content (Barcelo et al., 1986).

**Possible Toxicity Control Measures**

Application of lime and single superphates to soils can be helpful in correcting Cr toxicity.
Iron

A direct toxicity from a general soil application of Fe fertilizers would not be expected because of the relatively rapid conversion of soluble Fe to insoluble Fe compounds in soil systems (Martens and Westermann, 1991). Nutrient culture studies showed that some varieties of soybean [*Glycine max* (L.) Merr.] show Fe toxicity while others do not with the same amount of Fe in the solution (Brown and Jones, 1977).

Iron Toxicity Symptoms

Iron toxicity can be a problem for sorghum (*Sorghum bicolor* L. Moench) grown on some acid soils. Excess Fe causes leaves to turn light and blackish with straw-colored lesions at the margin (Clark, 1993). Older leaves are affected first followed by younger leaves.

Iron toxicity symptoms in soybean leaves are very similar to those of Mn except the Fe-toxic leaves are less crinkled than Mn-toxic leaves (Brown and Jones, 1977).

Possible Toxicity Control Measures

Liming of soils results in decreased Fe uptake by crops, for example, chlorosis caused by Fe deficiency appeared in lupins (*Lupinus* spp.) on calcareous soils when the pH of the soil solution was close to pH 7.5 (Bertoni et al., 1992). Soils with a pH range of 5.1 to 7.0 showed no effect on Fe concentration in several plant species (Gupta, 1992).

Manganese

Manganese toxicity and high levels of Mn are normally found in plants growing on strongly acid soils (Gupta, 1972; Vitosh et al., 1981) and on soils with high levels of water-soluble or salt-extractable Mn (Moraghan, 1979). Manganese availability and plant absorption are enhanced by reducing conditions in poorly aerated or submerged soils. Consequently, crops like barley (*Hordeum vulgare* L.) adapted to a well-drained soil environment suffer intensely due to excess Mn more than e.g. rice grown under submerged conditions (Vlamis and Williams, 1964). Manganese toxicity symptoms in white lupins have been reported when grown without added P (Moraghan, 1992). Plants that are sensitive to Mn toxicity include alfalfa, cabbage (*Brassica oleracea* L.), cauliflower (*Brassica oleracea* L. spp. Botrytis), clovers (*Trifolium* spp.), cotton (*Gossypium hirsutum* L.), small grain, and sugarbeet (Vitosh et al., 1981).

Toxicity Symptoms

Severe Mn toxicity symptoms in barley and rice (*Oryza sativa* L.) were related to Mn concentrations of 1,200 and 7,000 μg g⁻¹, respectively, and 380 μg Mn g⁻¹ for wheat (Keisling et al., 1984) and 475 μg Mn g⁻¹ for wheat shoot (Fales and Ohki, 1982). Manganese toxicity in flax (*Linum usitatissimum* L.) plants has been attributed to poor Fe availability (Moraghan, 1979). Accumulation of Mn in flax was eliminated by application of Fe chelate to problem soils.

Genotypic differences in soybeans can affect severe Mn toxicity with some soybean varieties suffering from more severe symptoms than others (Brown and Jones, 1977).

Visible symptoms of Mn toxicity in wheat include stunting, general chlorosis, necrotic leaf spots, white flaking, purpling and leaf tip burn (Keisling et al., 1984). In flax, symptoms of Mn included development of brown spots at the distal end of
TRACE ELEMENT TOXICITY

older leaves (Moraghan, 1979). Older leaves of barley showed a severe incidence of brown spotting (Vlamis and Williams, 1964).

Possible Toxicity Control Measures

Applications of lime or lime plus phosphate have been found to increase soil pH and decrease Mn toxicity in orchard sub-soils (Baugher and Singh, 1989). Liming a soil to pH 6.0 generally reduces Mn availability to a normal level and alleviates Mn toxicity. Studies in Atlantic Canada showed that on a strongly acid soil, the Mn levels in timothy (Phleum pratense L.) decreased from 175 µg g\(^{-1}\) at soil pH 4.5 to 50 µg g\(^{-1}\) at pH 6.7, and in bromegrass (Bromus inermis Leyss) from 225 to 65, respectively (Umesh C. Gupta, Charlottetown Research Centre, unpublished results).

Molybdenum

Molybdenum toxicity to plants seldom occurs, and therefore data on the toxic Mo concentrations in plant tissue are extremely limited. Toxic plant Mo concentrations differ according to the crop species. Plants belonging to the dicotyledonous species generally are less tolerant of excess plant Mo than are the monocotyledonous species (Graminae) (Kluge, 1983). Sometimes there may be slight toxicity symptoms on the foliage even though the yields may not be affected.

Toxicity Symptoms

The most striking symptom of Mo toxicity is a yellow or orange-yellow chlorosis, with some brownish tints that start in the youngest leaves (Bergmann, 1992). Specific symptoms of toxicity include purpling of young seedlings and delayed maturity in cauliflower (Vitosh et al., 1981); intense yellow-orange colour seeds in kidney beans (Phaseolus vulgaris L.) (Gartel, 1993); golden yellow to bronze colour leaves (Falke, 1983); seedling injury and detrimental effect to Rhizobia in soybeans (Sedberry et al., 1973) and dark violet coloration of lamina in sorghum [Sorghum bicolor (L.) Moench] (Clark, 1993).

Toxicity Levels

Toxicity Mo levels in crops range from 100 mg kg\(^{-1}\) in sugar beets, peas (Pisum sativum L.) and lettuce to 1,000 mg kg\(^{-1}\) in cucumber and winter wheat as summarized by Gupta (1997b). Feed crops, containing in excess of 5 to 10 µg Mo g\(^{-1}\), could cause Mo toxicity in livestock and this is described in detail in the livestock section below.

Nickel

Nickel toxicity occurs on soils to which large quantities of Ni have been added in wastes, e.g. in the form of sewage and sewage sludge (Uren, 1992) and in crops growing near Ni mines and smelters (Temple and Bisessar, 1981). Soils developed on ultrabasic rocks are rich in Ni (Anderson et al., 1973). Studies conducted on these soils showed that oat (Avena sativa L.) plant leaves with very severe chlorosis due to Ni toxicity contained 308 mg Ni kg\(^{-1}\) (Anderson et al., 1973). Nickel toxicity in wheat tops was associated with 92 and 112 µg Ni g\(^{-1}\) at N rates of 60 and 120 µg g\(^{-1}\), respectively (Singh et al., 1990).

Toxicity Symptoms and Levels

The phytotoxic effects of Ni have been known for a long time. Apart from a decrease in growth, the symptoms of Ni toxicity include chlorosis, stunted root...
growth, and sometimes brown interveinal chlorosis and symptoms specific to plant species (Uren, 1992). Nickel levels in plants related to toxicity symptoms vary widely. For example, Patterson (1971) reported Ni toxicity symptoms in spring wheat at 8 $\mu$g g$^{-1}$, but no yield loss in oats at 90 $\mu$g Ni g$^{-1}$ and Bolton (1975) reported no yield loss in oats at 147 $\mu$g Ni g$^{-1}$. Nickel concentrations of 12 and 14 $\mu$g g$^{-1}$ in barley and ryegrass were considered the minimum to cause Ni toxicity (Davis and Beckett, 1978).

Possible Toxicity Control Measures

Of all soil properties, soil pH is the most important factor in reducing the availability of Ni to plants. For example, symptoms of Ni toxicity in wheat were observed where greater than 20 $\mu$g Ni g$^{-1}$ had been added to the soil at pH 5.1. However, as summarized by Uren (1992), no symptoms of Ni toxicity appeared where 160 $\mu$g Ni g$^{-1}$ had been added at soil pH 7.5, as evident by a decrease in plant Ni concentration from 8 to 3 $\mu$g g$^{-1}$.

Zinc

Crops are highly tolerant to Zn. For example, Zn applications of up to 300 $\mu$g g$^{-1}$ did not cause reduction in spring wheat yield, although the grain contained 98 $\mu$g Zn g$^{-1}$ (Dudka et al., 1994).

Potential Zn phytotoxicity from excess Zn applications exists because only small amounts of Zn leach, and because reversion of applied Zn to unavailable forms is relatively slow in soils (Payne et al., 1988). Application of weathered fly ash has been reported to depress the growth of cucumber due to toxic levels of Zn in the plant (Dosskey and Adriano, 1993).

Toxic Soil and Crop Levels

Even at very high Zn applications of 1,000 $\mu$g g$^{-1}$, wheat yield was reduced only by 40% (Dudka et al., 1994). In soybeans, Zn phytotoxicity resulting in 10% growth reduction was associated with 95 $\mu$g Zn g$^{-1}$ in mature primary and trifoliate leaves (Ruano et al., 1987). Zinc application rates as high as 800 lb acre$^{-1}$ (896 kg ha$^{-1}$) were toxic to only two out of 12 leafy vegetables studied (Boawn, 1971). The two affected crops were Swiss orchard [Beta vulgaris subs. cicla (L.) Koch] and spinach (Spinaciaoleracea L.) and contained 150 and 340 $\mu$g Zn g$^{-1}$, respectively.

In studies conducted in India, plant Zn levels of 60 and 81 $\mu$g g$^{-1}$ in wheat and corn, respectively (Takker and Mann, 1978) and 190 $\mu$g g$^{-1}$ in rice were toxic (Rattan and Shukla, 1984). Minimum concentration of Zn required to cause toxicity in crops are relatively independent of growing conditions at 210 $\mu$g Zn g$^{-1}$ in barley and 221 $\mu$g Zn g$^{-1}$ in ryegrass (Lolium multiflorum L.).

Plants differ in their sensitivity to Zn toxicity. For example, normal growth of corn, a tolerant crop, may occur on a soil where Zn phytotoxicity limits growth of sensitive crops such as red kidney beans (Vitosh et al., 1981). Seven to 11 kg Zn ha$^{-1}$ applications caused severe leaf scorching and reduced yields of irrigated bean plants (Boawn, 1973). However, Zn application rates of 40 kg ha$^{-1}$ were not toxic to corn (LeBlanc et al., 1997).

Possible Toxicity Control Measures

In addition to decreasing yields, Zn toxicity has also been found to decrease the net assimilation rates and the growth of all organs in beans (Ruano et al., 1988). Based on guidelines prepared by USEPA (1983), the recommended cumulative limits
for Zn application to cropland are 280, 560, and 1,120 kg Zn ha\(^{-1}\) for soils with CEC levels of <5, 5-15, and >15 cmol\(_c\) kg\(^{-1}\), respectively. A pH of 6.5 or above must be maintained where sewage sludge is incorporated into soil in order to decrease Zn availability.

**Summary**

Summary of toxicity symptoms for trace elements in plants and a range of toxic levels in crops are given in Table 1. Possible trace element toxicity control measures in crops include using recommended rates of fertilizers and waste materials, liming and use of leaching for B.

**LIVESTOCK**

Most toxic effects of trace elements in livestock are dose dependent. Different doses may cause undetectable, therapeutic, toxic, or lethal effects (Fraser, 1986). Although the dose is a primary concern, the amount of toxic nutrient ingested by the animal is seldom known. Trace elements, Se, Cu, and Zn, often become toxic as the dose and exposure increase. Biological factors such as the age and size of animal also affect toxicity: the amount of a toxicant is correlated to body weight. Chemical factors, for example solubility of an element can affect toxicity by influencing the absorption of the nutrient in the animal body. Delayed absorption generally decreases toxicity. The toxicity of elements arsenic (As), Co, Cu, F, I, Fe, Pb, Mn, Hg, Mo, Se, and Zn will be discussed in this section. Cadmium (Cd) is one of the more toxic metals, however, poisoning due to Cd has not been observed in domestic animals and is thus excluded from discussion here.

**Arsenic**

Arsenic toxicity is now relatively infrequent because of the diminishing use of inorganic arsenals applied as rodenticides, weed killers, baits and insecticides, (Fraser, 1986). Inorganic As compound toxicity can cause dehydration, weakness, and lethargy.

**Toxicant Concentrations**

Toxicity is related with As concentrations of >3 µg g\(^{-1}\) in the liver and kidneys of animals. Sodium thiosulfate can be used to treat poisoning due to inorganic As.

**Toxicity Symptoms**

Organic arsenicals such as phenylarsonic acid derivatives are used for swine and poultry to improve production and/or dysentery (Fraser, 1986). Toxicity occurs from the use of excessive amounts of As-containing additives in the diet of swine and poultry resulting in reduced weight gain and blindness. There is no specific treatment of demonstrated value and the toxic effects of these compounds are often irreversible if competition for food has been eliminated.

**Cobalt**

Cobalt toxicosis is less likely to occur than its deficiency, and toxic levels appear to be at least 3,000 times greater than the requirement in most species (NRC, 1980). Cases of toxicity are often the result of accidental over-supplementation to prevent Co deficiency. Dietary concentrations of 10 mg Co kg\(^{-1}\) are considered safe.
TABLE 1. Trace element toxicity levels and symptoms in plants.

<table>
<thead>
<tr>
<th>Element</th>
<th>Toxic Mo Levels* in plants, Range, µg g⁻¹</th>
<th>Toxicity Symptoms in Plants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boron</td>
<td>10-50</td>
<td>Marginal and tip chlorosis of older leaves</td>
</tr>
<tr>
<td></td>
<td>&gt;200 for tolerant crops</td>
<td></td>
</tr>
<tr>
<td>Copper</td>
<td>10-70</td>
<td>Chlorotic leaves and reduced branching, thickening and dark coloration in the rootlets</td>
</tr>
<tr>
<td>Manganese</td>
<td>400-7000</td>
<td>Stunting, general chlorosis and necrotic leaf spots and brown spotting of older leaves</td>
</tr>
<tr>
<td>Molybdenum</td>
<td>100-1000</td>
<td>Yellow or orange-yellow chlorosis, seedling injury and delayed maturity</td>
</tr>
<tr>
<td>Nickel</td>
<td>8-147</td>
<td>Chlorosis, stunted root growth</td>
</tr>
<tr>
<td>Zinc</td>
<td>95-340</td>
<td>Severe leaf scorching, reduced yield, and decreased net assimilation rate</td>
</tr>
</tbody>
</table>

*Associated with toxicity symptoms and/or reduced yield

Toxicity Symptoms
Severe toxicosis in chicks was observed at 50 mg Co kg⁻¹ (Turk and Kratzer, 1960) causing polycythemia in simple stomached animals, and reduced feed intake and body weight, emaciation, anemia, and debility in ruminants (NRC, 1980). Large doses of Co are toxic and produce a depressed appetite and body weight loss in sheep (Becker and Smith, 1951) and calves (Ely et al., 1948). Clinical signs of poisoning in calves include loss of appetite, decreased water consumption, salivation, and lack of muscular coordination. The lethal intravenous dose of Co is >9 mg kg⁻¹ body weight in the dairy calf (Ely et al., 1948) and 75 mg d⁻¹ for sheep (Becker and Smith, 1951). Dickson and Bond (1974) found that calves and cows suspected to have suffered from Co toxicity had 58 to 69 mg Co kg⁻¹ in the liver on dry weight basis (normal range 0.08-0.12 mg kg⁻¹).

Possible Toxicity Control Measures
Injection of methionine prior to Co administration is said to prevent or greatly reduce the severity of its toxic effects (Humphreys, 1988).

Copper
Copper poisoning is encountered in most parts of the world. Sheep are affected most often but other animals such as swine and cattle are also susceptible (Fraser, 1986). Acute poisoning is usually observed after accidental administration of excessive amounts of Cu salts which may be present in anthelmintic drenches, mineral mixes and improperly formulated rations. Factors such as low levels of Mo
or sulfate in the diet enhance the absorption or retention of Cu, resulting in Cu poisoning.

Copper salts, such as copper sulfate, have been widely used in agriculture and veterinary medicine. Compounds of Cu have been used as sprays against fungal diseases, added to pig rations as a growth promoter and in seed dressings (Humphreys, 1988). Losses in lambs due to Cu toxicity have been recorded after contamination of foot rot, involving either contamination of the ewe’s udder and of forage with copper sulfate or drinking foot-rot treatment-bath contents (Muth, 1952).

Copper toxicity affects ovines, bovines (young calves, rarely adult) and canines (Murphy, 1996). In most species, it causes marked reduction of red cell glutathione and increased methemoglobin ensues; generally, intravascular hemolysis occurs with subsequent anemia (Murphy, 1996). Liver Cu concentrations in sheep often exceed 150 μg g⁻¹ wet weight.

### Toxicant Concentrations in Body Organs and Symptoms

- **Acute Cu poisoning** may follow intakes of 20-100 mg Cu kg⁻¹ body weight in sheep and young calves and 200-800 mg Cu kg⁻¹ in mature cattle (Fraser, 1986). Symptoms of Cu toxicity include kidney Cu levels of >15 μg g⁻¹ wet weight and blood Cu levels of 5-20 μg mL⁻¹ (1 μg mL⁻¹ considered normal). Liver Cu concentrations of >150 μg g⁻¹ (wt. weight) are significant in sheep.

- Copper in calf’s liver in excess of 200 mg kg⁻¹ (800 mg kg⁻¹ dry weight) indicates a chronic process of Cu poisoning of the animal (Hadrich, 1996). The liver in calves on some big farms in Germany contained up to 600 mg Cu kg⁻¹ (2,400 mg kg⁻¹ dry weight). Species vary widely in susceptibility to toxicity, in part due to differences in S metabolism as well as to dietary levels of S, Mo, Zn, and Fe (Miller et al., 1991). Adult animals can tolerate higher levels of Cu than the young. In some animals, Cu toxicity is less dramatic and includes growth inhibition, anemia, muscular dystrophy, impaired reproduction, and decreased longevity (NRC, 1980). Specific symptoms of Cu toxicity include gastroenteritis with erosions and ulcerations in the abomasum of ruminants. The liver is enlarged and friable. Swollen gums, metal-colored kidneys, port-wine-colored urine, and splenic enlargement are manifestations of hemolytic crisis.

- Signs of toxicity in swine include reduced hemoglobin levels, and generalized jaundice, resulting from excessive accumulation of Cu in the liver and breakdown of normal liver function (Cromwell, 1997). Copper to Mo ratio of greater than 10:1 results in hemolytic crisis. In animals suffering from Cu toxicity, the Cu levels of 8,000-10,000 μg g⁻¹ in feces, >15 μg g⁻¹ in kidneys and 5-20 μg Cu L⁻¹ in blood are found; Cu level in blood of 1 μg L⁻¹ is considered normal.

### Possible Toxicity Control Measures

- Often treatment for Cu toxicity is not successful. Measures to control the toxicity include administration of 100 mg ammonium molybdate and 1 g sodium sulfate which would reduce losses in affected lambs (Fraser, 1991). Top dressing of pastures with 2.5 oz Mo acre⁻¹ (70 g Mo ha⁻¹) in the form of molybdenized superphosphate or by Mo supplements or by restriction of Cu intake minimizes the risk of Cu toxicity in livestock. Treatment includes supplementation of ration with 2-16 μg Mo g⁻¹ ammonium molybdate 50 mg head⁻¹ d⁻¹ with 0.3 to 1 g thiosulfate daily for three weeks (Murphy, 1996). Molybdenized superphosphate is used to increase Mo content of forages; and the proper ratio of Cu to Mo in sheep is 6:1 for molybdenized salt licks.
**Fluoride**

Toxic quantities of fluorides occur in some products, e.g. certain raw rock phosphates, the superphosphates produced from them and the phosphatic limestones that are used in feeding animals (Fraser, 1986). Sodium fluoride is the most toxic, and calcium fluoride the least toxic of the common fluorides; the latter has low solubility.

**Toxicant Concentrations in Bones and Symptoms**

Advanced stages of F toxicity are marked by abnormalities of the skeletal system as the bones become chalky white, soft and thickened and degenerative changes occur in the kidney and liver. In dairy cattle, the toxicosis is associated with >5,500 μg F g⁻¹ F and >7,000 μg F g⁻¹ in compact bone and cancellous bones, respectively.

**Possible Toxicity Control Measures**

Control other than removal of animals from affected areas, is difficult. The feeding of calcium carbonate, aluminum oxide or aluminum sulfate reduces absorption of F by about a third and thus could offer some control of chronic fluorosis under certain conditions.

**Iodine**

Newton et al. (1974) showed that 50 mg I kg⁻¹ significantly reduced growth rate and feed intake of calves weighing approximately 100 kg. Excessive dietary I or treatment of foot rot with organic I for an extended period can cause I toxicity (Miller et al., 1991).

**Toxicity Symptoms**

Signs of I toxicity in cattle include excessive lacrimation and salivation, a watery nasal discharge and tracheal congestion that causes coughing (Newton et al., 1974; NRC, 1988). Toxic effects of I in the poultry include reduced egg production and hatchability (NRC, 1980). Large doses of iodides cause nervousness and cardiac depression (Humphreys, 1988).

The minimum toxic level of I was <50 μg g⁻¹ in calves given I in their diets for long periods (Newton, 1973). Morrow and Edwards (1981) reported that feeding 170 mg I cow⁻¹ to high yielding cows for 30 days resulted in abortions within a 68-day period following treatment.

**Iron**

Probably the best known type of Fe poisoning in veterinary toxicology is that seen in young piglets injected with an Fe dextran preparation for the prevention of post-natal anemia (Humphreys, 1988). However, Fe intoxication can also occur in other animal species. Sources of Fe toxicity include injectable and oral Fe preparations, Fe supplements and overzealous use of Fe preparations (Murphy, 1996). Many factors that can affect Fe absorption may in turn affect Fe toxicity in animals because the toxicity is governed by its absorption (NRC, 1980).

**Toxicity Symptoms**

Characteristic signs of chronic Fe toxicosis for most species are reduced growth rate, and reduced feed intake and efficiency (NRC, 1980). The clinical signs of acute toxicosis include anorexia, diarrhea, oliguria, hypothermia, metabolic acidosis, and
death (Boyd and Shanas, 1963). An excessively high level of supplemental Fe may elevate serum unbound Fe which encourages bacterial growth and results in increased susceptibility to infection and diarrhea (Weinberg, 1978; Knight et al., 1983; Kadis et al., 1984).

Symptoms of Fe toxicity comprise severe depression, followed by coma and death (Murphy, 1996). Acute clinical symptoms include shock, collapse, vomiting, edema at the site of injection, “anaphylactic” death within 1-3 hours of injection. Iron toxicity results in excess of 2,000 mg Fe kg\(^{-1}\) in 1-3 day-old pigs, if given 200 mg Fe.

**Possible Toxicity Control Measures**

Treatment of Fe toxicity consists of removal of animal from the source, emesis, and gastric lavage. Magnesium hydroxide (Milk of Magnesia) can be used to precipitate Fe in gastro-intestinal (GI) tract. Desferroxamine and ascorbic acid are used to enhance excretion of excess Fe (Murphy, 1996).

**Lead**

Lead poisoning is the most frequently diagnosed toxicological condition in veterinary medicine (Fraser, 1986). Its occurrence has been reported in all domestic species and most commonly in cattle and dogs because of their indiscriminate eating habits and relative susceptibility to Pb. Wild ducks are frequently poisoned by ingested Pb pellets.

**Toxicity Symptoms**

Clinical signs of Pb toxicity entail loss of appetite, weight loss, depression, muscular weakness, stiffness of the joints, diarrhea, and often anemia. The concentration of Pb in the kidney cortex, liver or in whole blood provides definitive confirmation of Pb toxicity. Lead values of >4, 4, and 0.2 \(\mu g \text{ g}^{-1}\) (wet weight) for these tissues, respectively, indicate abnormal Pb accumulation. Levels of Pb at least twice these are found in fatal cases.

**Possible Toxicity Control Measures**

Extensive and prolonged injury to animals makes treatment of little value. Intestinal lavage, such as magnesium sulfate orally may be applied to remove Pb remaining in the digestive tract. Administration of edetate calcium disodium intravenously is also effective.

**Manganese**

Manganese poisoning has not been recorded in domestic animals (Humphreys, 1988). In general, adverse effects have not been seen in most species with dietary Mn concentrations of up to 1,000 mg kg\(^{-1}\) (Miller et al., 1991). The maximum tolerable level of Mn is 1,000 mg kg\(^{-1}\) for cattle and sheep, 2,000 mg kg\(^{-1}\) for poultry and 400 mg kg\(^{-1}\) for swine (NRC, 1980).

**Toxicity Symptoms**

Signs of Mn toxicosis include growth retardation, anemia, gastrointestinal lesions and sometimes neurological signs (NRC, 1980). A Mn-Fe antagonism has been observed in baby pigs (Humphreys, 1988). A level of 50-125 \(\mu g \text{ Mn} \text{ g}^{-1}\) in the diet interfered with hemoglobin formation. Supplementation of the diet with 1,250-2,000 \(\mu g \text{ Mn} \text{ g}^{-1}\) caused growth depression (Matrone et al., 1959).
Mercury

Mercury poisoning due to inorganic compounds in animals is usually caused by accidental ingestion of mercuric chloride or its solution, and by organic compounds resulting from their inadvertent exposure to treated seed used for livestock feed (Fraser, 1986).

Contaminated fish are a concern where Hg pollutes the waters; the resulting methyl mercury accumulates in the fish. In animals, the kidneys selectively accumulate Hg.

Toxicity Symptoms
Symptoms of Hg poisoning entail vomiting, bloody diarrhea, and necrosis of the alimentary mucosa.

Possible Toxicity Control Measures
As a Hg toxicity antidote, oral use of sodium thiosulfate may be beneficial (Fraser, 1986). Gastric lavage with sodium formaldehyde sulfoxalate is also useful, as it serves to reduce the divalent Hg to the less toxic monovalent form. Dimercaprol is an effective antidote for mercurial diuretics.

Molybdenum

When Mo is present in excessive quantities in soil, it may be taken up by plants in sufficient quantities to produce intoxication in grazing animals (Humphreys, 1988). In the U.K., herbage grown on soils in parts of Somerset, Warwickshire, and Gloucestershire have been found to contain 20-100 \(\mu g\) Mo g\(^{-1}\) and cattle grazing on these pastures suffer from Mo toxicity (Ferguson et al., 1943). The toxic syndrome of Mo poisoning or molybdenosis locally known as “teart” also occurs in parts of Florida, California, Canada, and Australia. The severity of disease is related to the water soluble Mo content of the herbage.

Toxicity Symptoms
Intake of excess Mo was found to result in histological damage in the pancreas of guinea pigs and this was thought to result from Cu deficiency rather than Mo toxicity (Howell et al., 1993). The Mo content of typical teart pastures may range from 20 to 100 mg Mo kg\(^{-1}\) (dry basis) (Underwood, 1977). Molybdenosis is essentially a secondary Cu deficiency manifested by diarrhea, anorexia, depigmentation of hair and wool, neurological disturbance and premature death. The maximum tolerable level of Mo can be increased substantially by increased amounts of Cu as inorganic sulfate (NRC, 1980).

In ruminants, the dietary intake of excessive Mo causes, in part a secondary hypocuprosis. Ruminants are much more susceptible to Mo toxicity than non-ruminants (Fraser, 1991; Fraser, 1986). Tolerance to Mo falls as the content and intake of Cu falls. Low dietary sulfate causes high blood Mo because of its decreased excretion. Molybdenosis associated with Cu deficiency has been observed in areas with peat or muck soils, where plants grow in alkaline sloughs, e.g. in western United States as a result of industrial contamination where excess Mo-containing fertilizers have been applied and where lime applications appeared to increase plant Mo uptake (Fraser, 1991). In the diet, Cu:Mo ratios of 6:1 are considered ideal; 2:1 - 3:1 are borderline and <2:1 are toxic. Although 10 \(\mu g\) Mo g\(^{-1}\) is toxic, 1 \(\mu g\) Mo g\(^{-1}\) can be toxic if Cu content is less than 5 \(\mu g\) g\(^{-1}\) on a dry weight basis (Fraser, 1986).
Symptoms of Mo toxicity are characterized by persistent severe scouring with passage of liquid feces riddled with gas bubbles (peat scours or teart). Non-specific signs of Mo toxicity comprise - poor body condition, anemia, emaciation, lameness, osteoporosis, and fading of the coat color. Depigmentation is most noticeable in black animals, especially around the eyes (Fraser, 1991) which gives a spectacled appearance. Clinical signs appear within 1-2 weeks of grazing on an affected pasture.

Possible Toxicity Control Measures

Use of one percent copper sulfate in salt has provided satisfactory control of molybdenosis if Mo is <5 µg g⁻¹. With higher Mo levels, use of 2% copper sulfate and up to 5% where the Mo levels are very high is recommended. In areas where cattle do not consume mineral supplements, the required Cu may be supplied as a drench given weekly or as a top dressing to the pasture (Fraser, 1986).

Selenium

Soils bearing vegetation containing higher than 5 mg Se kg⁻¹ are designated as seleniferous and are associated with livestock and wild life poisoning (Dhillon and Dhillon, 1991). Selenium is highly toxic at high doses, but is required in trace amounts to prevent deficiency diseases in cattle, sheep, swine, and chicks (Fraser, 1986). Maximum tolerable levels of Se for all species has been established as 2 µg g⁻¹.

Evidence of Se toxicity in grazing animals was first identified in the great plains of the United States. Localized seleniferous areas have also been identified in Ireland, Israel, Canada, Australia, the former Soviet Union, China, and South Africa (Fraser, 1986). Toxicity arises from consuming plants with relatively high Se content. Selenium levels as low as 4-5 µg g⁻¹ will inhibit growth. Selenium content in hair in excess of 3 µg g⁻¹ indicates toxicity (Fraser, 1986). Chronic Se poisoning, “alkali disease”, results when animals consume naturally-produced seleniferous forages and grains that contain 5-40 µg Se g⁻¹ (Fraser, 1986). Vegetation containing >50 µg Se g⁻¹ has been found growing in states of the great plains of the Northwestern United States (Colorado, Nebraska, South Dakota, and Wyoming) west of the Mississippi river except those adjoining the river and the State of Washington. Such vegetation has also been found in the prairie provinces of Canada and Mexico. Areas producing highly seleniferous vegetation are spotty and localized.

Toxicity Symptoms

The most pronounced symptoms of chronic Se poisoning are atrophy of the heart, and atrophy and cirrhosis of the liver (Humphreys, 1988). Animals showing these symptoms contain Se at 2.2 to 5.3 µg g⁻¹ in blood, 12.6 to 55.2 µg g⁻¹ in hairs and 21.2 to 39.9 µg g⁻¹ in hooves (Dhillon and Dhillon, 1991).

With alkali disease, the Se levels in blood range from 1-2 µg g⁻¹ and with “blind staggers” it ranges from 1.5-4 µg g⁻¹. Selenium poisoning in cattle, horses, and swine causes cracking of the hooves, lameness, stiffness of joints, emaciation, and loss of hair. Sows have a lowered conception rate and an increase in the number of pigs born dead. This “alkali disease” type is not common in sheep. Neurological dysfunction occurs only among ruminants.

Embryocidal deformities occur in birds feeding on Se-enriched feedstuffs. “Blind staggers” type Se toxicity develops in animals consuming highly seleniferous plants or grains (>30 µg Se g⁻¹) over a period of weeks or months (Fraser, 1986). Symptoms include wandering of animals into objects in their path, impairment of vision, and poor appetite, and subsequently death follows from respiratory failure.
Acute Se poisoning occurs mainly in cattle and sheep and results from ingestion of enough seleniferous plants, usually in a single feed, to produce very severe signs of toxicity. Death occurs in a matter of a few hours to a few days (Humphreys, 1988). Acute Se toxicity is characterized by abnormal movement and posture, breathing difficulties, diarrhea and rapid death (NRC, 1988; Keen and Graham, 1989). Consumption of limited amounts of Se accumulator plants results in alkali disease where the symptoms include lameness, loss of vitality, loss of hair, liver cirrhosis and anemia (NRC, 1980).

Selenium toxicity symptoms include development of cracks in hoofs followed by their gradual detachment, peeling off of horns and loss of hair from the body, necrosis of tail and death in severe cases (Dhillon and Dhillon, 1991).

Possible Toxicity Control Measures
There is no known way to counteract Se toxicity. The most effective method is to remove the source of Se. A high protein ration will help to control chronic Se poisoning. The use of salt containing As at 0.00375% may reduce the incidence of chronic Se poisoning in cattle grazing on seleniferous range (Fraser, 1986). There is no known treatment for acute Se poisoning.

Zinc
Acute Zn toxicity in animals is uncommon and is first observed at Zn intake of several grams (Sandstrom, 1995). Zinc is a divalent cation which reacts with red cells and hepatocytes when in excess (Murphy, 1996). More subtle negative effects of Zn intakes of 50-150 mg d\(^{-1}\) are lowered activity of Cu, Zn-superoxide dismutase, Fe deficiency, and lowered levels of HDL (high density lipoprotein) cholesterol.

Toxicity Symptoms
Zinc toxicosis is manifested as gastrointestinal distress, decreased food consumption, pica, decreased growth, anemia, and poor bone mineralization and arthritis (NRC, 1980). Sows fed a 5,000 mg Zn kg\(^{-1}\) diet (Zn from ZnO) have small litters, lighter weight pigs at weaning, and a higher incidence of osteochondrosis. The offspring of these sows have reduced tissue levels of Cu (Hill and Miller, 1983; Hill et al., 1983).

Clinical signs of Zn toxicity include sudden onset of weakness, trembling, anorexia, hyperventilation, hemoglobinuria, and icterus. It mostly affects canine and porcine species (Murphy, 1996). The Zn concentration of 40-100 \(\mu\)g g\(^{-1}\) are considered normal in the liver.

Possible Toxicity Control Measures
Treatment of Zn toxicity include 500-1,000 mg ascorbic acid d\(^{-1}\) to enhance Cu elimination and cimetidine to decrease gastric acidity; calcium EDTA should be given at 100 mg kg\(^{-1}\) body weight every 6 h (Murphy, 1996) to treat acute toxicity.

Summary
A general summary of toxicity symptoms for trace elements in livestock and toxic element levels in body organs are given in Table 2. Possible trace element toxicity control measures in livestock include using balanced rations; removal of animals from the source or affected areas, gastric and intestinal lavage and use of chelates.
TABLE 2. Trace element toxicity levels in body organs and symptoms in livestock.

<table>
<thead>
<tr>
<th>Element</th>
<th>Toxic Mo Level in body organs, μg g⁻¹</th>
<th>Toxicity Symptoms in Livestock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>&gt;3 in liver, kidney</td>
<td>Reduced weight gain, blindness, dehydration and depression</td>
</tr>
<tr>
<td>Cobalt</td>
<td>58-69 in liver</td>
<td>Reduced feed intake, emaciation, anemia and lack of muscular coordination</td>
</tr>
<tr>
<td>Copper</td>
<td>&gt;15 in kidney, 5-20 in blood, &gt;150 in liver</td>
<td>Gastroenteritis, swollen gums, reduced hemoglobin levels, jaundice, metal-colored kidneys and splenic enlargement</td>
</tr>
<tr>
<td>Fluoride</td>
<td>&gt;5500 in compact bone, &gt;7000 in cancellous bone</td>
<td>Bones chalky white, soft and thickened, and degenerative changes occur in liver and kidney</td>
</tr>
<tr>
<td>Iron</td>
<td>&gt;2000 in pigs blood</td>
<td>Reduced growth rate and feed intake, anorexia, diarrhea, metabolic acidosis and death</td>
</tr>
<tr>
<td>Lead</td>
<td>&gt;4 in kidney cortex&amp; liver, &gt;0.2 in whole blood</td>
<td>Loss of appetite, weight loss, depression, stiffness of joints and anemia</td>
</tr>
<tr>
<td>Molybdenum</td>
<td>Variable</td>
<td>Diarrhea, anorexia, depigmentation of hair and wool, neurological disturbance</td>
</tr>
<tr>
<td>Selenium</td>
<td>&gt;3 in hair, 2.2 - 3.3 in blood, 21.2-39.3 in hoofs</td>
<td>Atrophy of heart, cirrhosis of liver, cracking of hoofs, lameness, loss of hair, stiffness of joints and diarrhea</td>
</tr>
<tr>
<td>Zinc</td>
<td>Variable</td>
<td>Gastrointestinal distress, anorexia, anemia and arthritis</td>
</tr>
</tbody>
</table>

**HUMANS**

Although many trace elements are required by humans for normal metabolism, most may also be toxic in levels above the very low body requirements. The pathologic conditions related to metal toxicity result from either biochemical (genetic) or physiological (environmental) abnormalities. The excesses are due to one of the following: (i) excessive dietary intake, (ii) excessive absorption, (iii) decreased loss from the body, and (iv) decreased metabolism of the nutrient due to antagonism or metabolic block.
In this section, the epidemiology, pathophysiology, and recommended therapies for Cd, Cu, Fe, Pb, Mn, Hg, Mo, Se, and Zn toxicities will be presented.

**Cadmium**

Cadmium is of concern because it can be taken up by crops in amounts potentially harmful to human beings (Page and Chang, 1994). Cadmium is not toxic to plants but its accumulation in plants in large quantities is toxic to humans and livestock who consume the laden plants. Plant Cd uptake is increased by phosphate. The major source of Cd pollution in the air is emission from iron and steel works. However, Cd toxicity is not of major concern because the threshold of Cd absorption in the body above which kidney failure would occur after 50 years continuous exposure is approximately 10 \( \mu g \) d\(^{-1}\) (Anonymous, 1982). Most of the worst Cd hotspots are close to old Pb and Zn mines—both of which bring up Cd with the other metals (Anonymous, 1982).

**Toxicity Symptoms**

Cadmium, an air pollutant from cigarette smoke, industrialization, and population growth is known to be an infrequent cause of hypertension, cancer, and immune disorders (Stewart-Pinkham, 1989). Other symptoms of Cd toxicity include severe gastric cramps, vomiting, diarrhea, cough, headache, brown urine, and renal failure (Berkow, 1992). Cadmium toxicity effects can be confused with Pb effects because free Cd causes Zn deficiency which increases Pb absorption and also causes bone resorption which releases Pb from stores (Stewart-Pinkham, 1989). While free Cd is very toxic, Cd has also been found to greatly increase the toxicity of other agents. Therefore, if one can reduce the toxicities of such substances, e.g. chlorides, by reducing the exposure and release of Cd, it may be more cost effective.

**Recommended Therapies**

Cadmium toxicity can be treated by gastric lavage with milk or albumin, use of respiratory support, or hydration by giving acetate calcium disodium (Berkow, 1992). There is no defence against absorption of Cd from air.

**Copper**

Copper toxicity can be associated with cooking in an unlined Cu utensil and a single dose of 0.1 to 0.2 mg Cu kg\(^{-1}\) body weight can elicit gastrointestinal disturbances in sensitive persons (Bosshard and Zimmerli, 1994). Acute Cu toxicity in humans is rare and is associated with consumption of acidic foods or drinks that had prolonged contact with Cu containers, or with ingestion of gram quantities of Cu (van Campen, 1991).

**Toxicity Symptoms**

Wilson’s disease is an autosomal recessive disorder in which the inherited metabolic defect is associated with the gradual and progressive accumulation of Cu in the liver (Randolph and Rotter, 1989). Nearly all patients with Wilson’s disease progress to cirrhosis if the Cu retention is not treated. Excess Cu in the liver could be caused by chronic cholestasis (or a severe block in the flow of bile from the liver). Salivary gland swelling can be caused by excessive Cu ingestion (Glenn and Waymuller, 1989). Toxicity symptoms of Cu include hemolysis, hepatic necrosis, and renal damage (van Campen, 1991).
**Trace Element Toxicity**

**Recommended Therapies**

Chelating agents are useful in treating poisoning by many metals and other toxic substances. A list of such agents can be found in Table 288-3 by Berkow (1992). Utensils made with Cu or Cu alloys in contact with food should be covered by protective legislation (Bosshard and Zimmerli, 1994).

**Iron**

Iron poisoning results primarily from ingestion of mineral supplements containing divalent iron: ferrous sulfate (20% Fe), ferrous fumarate (33% Fe) and ferrous gluconate (12% Fe) (Olson and Becker, 1990). Iron poisoning in adults is related to unusual circumstances such as *Bantu siderosis*, which results from consumption of beer that is home brewed in Fe kettles and is very high in Fe (Bothwell et. al., 1979).

**Toxicity Concentrations and Symptoms**

More than 20-30 mg elemental Fe kg\(^{-1}\) causes toxicity and amounts over 60 mg kg\(^{-1}\) are potentially lethal. A peak Fe concentration in serum often occurs 4-5 hours after ingestion.

Serum Fe concentrations over 350-400 \(\mu g\) dL\(^{-1}\) are serious (Olson and Becker, 1990). Iron toxicity causes vomiting, upper abdominal pain, pallor, diarrhea, drowsiness, and shock (Berkow, 1992). The mechanism of toxicity is through vasodilation and disruption of the cellular electron transport mechanism resulting in metabolic acidosis.

**Recommended Therapies**

Treatments to control Fe toxicity include emptying the stomach by induced emesis with syrup of ipecac or gastric lavage (Olson and Becker, 1990). For serious or massive ingestion, enhance removal of Fe from the stomach by gastric lavage with sodium bicarbonate or a saline laxative combining sodium phosphate and sodium biphosphate (Fleet's phosphosoda) diluted 1:4 to form insoluble Fe salts. For shock, give deferoxamine 1 g intravenous (iv) (maximum rate 15 mg kg\(^{-1}\) h\(^{-1}\)) exchange transfusion (Berkow, 1992). Intravenous chelation with deferoxamine is the treatment of choice when symptoms of Fe poisoning are evident or when the serum Fe level is over 350-400 \(\mu g\) dL\(^{-1}\) (Olson and Becker, 1990). The Fe-deferoxamine complex is excreted in the urine and has a pink color.

**Lead**

Lead is widely distributed in the environment and industrial workers in smelters, storage battery and paint factories are at great risk for exposure (Langston and Irwin, 1989). Children are much more susceptible than adults and childhood cases result from the ingestion of lead-based paints which were commonly used prior to World War II. Ingestion and inhalation represent the primary routes of absorption of Pb. Elevation of blood Pb occurs when there is excessive absorption of Pb or excessive mobilization of Pb from bone stores. Feeding Pb in low doses to animals increases Cd uptake into the brain (Stewart-Pinkham, 1989).

**Toxicity Concentrations and Symptoms**

In adults, excessive exposure to Pb (blood concentrations greater than 80 to 100 \(\mu g\) dL\(^{-1}\)) is accompanied by pallor, gingival lead line, anemia, and a variety of neurological symptoms (Stewart-Pinkham, 1989; Langston and Irwin, 1989). Blood Pb concentrations greater than 25 \(\mu g\) dL\(^{-1}\) would alert suspicion of toxic exposure.
Urinary Pb in excess of 120 μg 24 h⁻¹ also indicates recent exposure (Langston and Irwin, 1989).

In Bangkok, Thailand, a study conducted in 1990 showed that by age 7 the average child had lost six points in IQ tests because of Pb poisoning from the air (Toronto Globe and Mail, April 8, 1996). Average blood Pb levels in Thailand were 40-45 μg dL⁻¹ which is 10 times the U.S. standard. This is attributed to a large number of cars per capita and due to thermal plants.

**Recommended Therapies**

Treatments for Pb toxicity must address the prevention of continued exposure. Chelation therapy, to reduce the body burden of Pb should be initiated as rapidly as possible, using edetate calcium disodium (CaEDTA) and dimercaprol (BAL), followed by oral penicillamine.

**Manganese**

Manganese is present in coal and crude oil. Intoxication of Mn was first observed in miners and millers of Mn-containing ores (Langston and Irwin, 1989; Berkow, 1992). Manganese toxicity generally results from chronic inhalation of airborne Mn in mines and factories (Ulrich et al., 1979); orally ingested Mn is relatively non-toxic. Manganese toxicity has also developed from drinking contaminated water. After inhalation or ingestion, Mn rapidly accumulates in the kidneys, liver, and brain.

**Toxicity Symptoms**

Toxicity of Mn can result in psychiatric disorders resembling schizophrenia, followed by permanent neurological disorders resembling Parkinson's Disease (van Campen, 1991). Symptoms also include hallucinations, memory impairment, disorientation and emotional instability.

**Recommended Therapies**

For treating Mn toxicity, chelating agents such as CaEDTA may have benefit if used early (Langston and Irwin, 1989).

**Mercury**

Mercury poisoning results from all Hg compounds, calomel, diuretics, mercuric chloride, Hg vapor, and methiolate (Berkow, 1992). Elemental Hg, when ingested orally, is poorly absorbed and therefore causes little toxicity (Langston and Irwin, 1989). Occupational exposure to Hg typically occurs in the manufacture of paint and fungicides or in the electrical, chemical, mining, and agricultural industries (Langston and Irwin, 1989). The inadvertent consumption of seed grain containing methyl mercury as a fungicide has produced epidemics of Hg poisoning. In Japan, Hg waste disposed in Minamata Bay was converted to methyl Hg by the action of microorganisms, and thus gained access to the food chain, poisoning hundreds of humans who consumed contaminated fish. The central nervous systems (CNS) toxicity due to Hg results primarily from exposure to the elemental and organic forms.

**Toxicity Symptoms**

Characteristic signs of chronic, low level exposure to elemental or organic Hg include depression, irritability, confusion, and tremor (Langston and Irwin, 1989). Methyl Hg is particularly damaging to the CNS causing sensory disturbances, including visual defects, hearing loss, ataxia, and cognitive disturbances. Effects of
methyl Hg on the fetus include mental retardation and neuro-muscular deficits, and toxicity may occur even when mothers are asymptomatic (Langston and Irwin, 1989). Other symptoms of Hg poisoning are severe gastroenteritis, burning mouth pain, salivation, abdominal pain, vomiting, colitis, chronic gingivitis, mental disturbances, and neurological deficits (Berkow, 1992).

**Recommended Therapies**

Elemental Hg poisoning is treated by chelation. Dimercaprol is the chelator of choice for symptomatic patients or those with high blood Hg levels. For low exposure or asymptomatic patients, penicillamine is recommended (Langston and Irwin, 1989). Other means of treating Hg toxicity include using gastric lavage, activated charcoal, penicillamine, and hemodialysis to maintain fluid and electrolyte balance. Skin-scrub with soap and water, and lung-supportive care can also provide some control (Berkow, 1992).

**Molybdenum**

Studies on the effect of excess Mo on human health are very limited and direct cause/effect relations have not been established. A Soviet population exposed to a high Mo environment had increased blood xanthine oxidase, increased concentrations of uric acid in blood and urine, and a high incidence of gout (Kovalskiy et al., 1961 as cited in Nielsen and Mertz, 1984).

**Selenium**

Selenosis in humans is not as well defined as in animals (van Campen, 1991). Early studies in the high Se areas of the United States did not reveal any problems that could be attributed specifically to high Se intakes (Levander, 1986).

**Toxicity Symptoms and Concentrations**

An outbreak of Se toxicity apparently occurred in China from 1961 to 1964 with the most common indicators being loss of hair and nail deformities (Yang, 1987; Berkow, 1992).

After visiting the affected areas, Whanger (1989) attributed high Se intakes, primarily the result of the use of high Se-coal. Burnt coal was used as a fertilizer, which greatly increased soil Se and the Se content of plant foods grown on the contaminated soil. Selenium content of corn grain was as high as 40 mg kg⁻¹ and dietary intakes of up to 38 mg d⁻¹. In China in endemic selenosis patients, dietary Se d⁻¹ was as high as 4990 µg d⁻¹ and blood Se was 3180 µg L⁻¹. Consumption of 50-300 µg Se d⁻¹ is considered in the normal recommended dose.

**Zinc**

Zinc toxicity is generally limited to cases of accidental overdose or therapeutic use of high doses of Zn.

**Toxicity Symptoms and Concentrations**

Ingestion of Zn in large amounts, usually from an acid food or drink from a galvanized container, has caused vomiting and diarrhea. Metal fume fever is an industrial hazard caused by inhalation of Zn oxide fumes and results in neurologic damage. It is also called brass founder’s ague or Zn shakes. Zinc/copper ratios of 10:1 may depress Cu utilization (Solomons, 1983). Sickle cell anemia patients...
<table>
<thead>
<tr>
<th>Element</th>
<th>Toxic Doses</th>
<th>Symptoms of Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadmium</td>
<td>$10 \mu g \text{d}^{-1}$ continuous exposure for 50 years</td>
<td>Acute: gastric cramps, vomiting, diarrhea, cough, headache, brown urine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic: hypertension, malignancy, immune disorders, renal failure</td>
</tr>
<tr>
<td>Copper</td>
<td>$0.1-0.2 \text{mg kg}^{-1}$ body weight</td>
<td>Wilson's disease and cirrhosis, haemolysis, hepatic necrosis, renal damage and salivary</td>
</tr>
<tr>
<td>Iron</td>
<td>$20-30 \text{mg kg}^{-1}$ body weight (&gt;$60 \text{mg kg}^{-1}$ is lethal)</td>
<td>Mild-moderate: vomiting, upper abdominal pain, pallor, diarrhea, drowsiness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Severe: metabolic acidosis and shock (disruption of cellular electron transport)</td>
</tr>
<tr>
<td>Lead</td>
<td>$80-100 \mu g \text{dL}^{-1}$ (blood) $&gt;$120 $\mu g \text{24h}^{-1}$ (urine)</td>
<td>Pallor, gingival lead line, anemia, multiple neurological symptoms (cognitive)</td>
</tr>
<tr>
<td>Manganese</td>
<td>variable in different organs</td>
<td>Psychiatric disorders followed by neurological disorders resembling Parkinson's Disease</td>
</tr>
<tr>
<td>Mercury</td>
<td>highly variable for both elemental and organic forms</td>
<td>Acute: gastrointestinal tract inflammation (oral, gastric, and colonic), abdominal pain,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>neurologic deficits</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic: depression, irritability, confusion, tremor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CNS damage: visual and auditory defects, ataxia and cognitive disturbances</td>
</tr>
<tr>
<td>Molybdenum</td>
<td>unknown</td>
<td>Epidemiological evidence of increased blood xanthine oxidase and incidence of gout</td>
</tr>
<tr>
<td>Selenium</td>
<td>average of $3180 \mu g \text{L}^{-1}$ (blood) average of $4990 \mu g \text{d}^{-1}$ (diet)</td>
<td>Hair loss, nail deformities</td>
</tr>
<tr>
<td>Zinc</td>
<td>several grams required for acute toxicity $160 \text{mg d}^{-1}$ for Cu deficiency</td>
<td>Acute: vomiting, diarrhea, neurological damage (&quot;Zn shakes&quot;)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic: depressed Cu utilization, Fe deficiency, lowered levels of HDL cholesterol</td>
</tr>
</tbody>
</table>
receiving 150 mg Zn d⁻¹ (10 times the RDA) developed signs of Cu deficiency and young men receiving 160 mg Zn d⁻¹ experienced substantial declines in plasma high density lipoproteins (HDL) (Hooper et al., 1980). The acute toxicity of Zn is low and is first observed at intakes of several grams. The negative effects include lowered activity of a Cu containing enzyme (Cu, Zn-superoxide dismutase), Fe deficiency and lowered levels of HDL cholesterol at Zn supplementation of 50-150 mg d⁻¹.

Summary

A summary of toxic doses and symptoms of trace element toxicity in humans is presented in Table 3. The epidemiology of trace element toxicities in humans are variable, as are the therapeutic modalities used in their treatments. Some of these measures include gastric lavage, hemodynamic support, chelating agents, and specific antidote medications.

CONCLUSION

Toxicity of trace elements in crops is less common than deficiencies. Toxicities occur in nature where crops are grown on soils naturally high in nutrients or near mining areas or where the irrigation water contains higher than desired content of nutrients/metals. Careful management practices can reduce toxicities in many instances. Consumption of feed crops high in nutrients/metals or excess application of minerals in grain mixes can result in toxicity to animals. The chief measure of control of trace element toxicity includes removal of animals from affected areas or source of toxicity. Trace element toxicities in humans are chiefly dose dependent, and are the result of metabolic abnormalities or are caused by high accidental or suicidal intakes. Most foods generally contain small quantities of trace elements. Gastric lavage and use of oral doses of salts and chelates are helpful measures in lessening metal toxicity in animals and humans.

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REFERENCES:


TRACE ELEMENT TOXICITY


