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MOLYBDENUM HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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SUMMARY

The element molybdenum (Mo) is found in all living organisms and is considered to be an essential or beneficial micronutrient. However, the molybdenum poisoning of ruminants has been reported in at least 15 States and 8 foreign countries.

Molybdenum is used primarily in the manufacture of steel alloys. Its residues tend to be elevated in plants and soils near Mo mining and reclamation sites, fossil-fuel power plants, and Mo disposal areas. Concentrations of Mo are usually lower in fish and wildlife than in terrestrial macrophytes.

Aquatic organisms are comparatively resistant to Mo salts: adverse effects on growth and survival usually appeared only at water concentrations >50 mg Mo/l. But in one study, 50% of newly fertilized eggs of rainbow trout (*Oncorhynchus mykiss*) died in 28 days at only 0.79 mg Mo/l. High bioconcentration of Mo by certain species of aquatic algae and invertebrates--up to 20 grams of Mo/kg dry weight--has been recorded without apparent harm to the accumulator; however, hazard potential to upper trophic organisms (such as waterfowl) that may feed on bioconcentrators is not clear. Data on Mo effects are missing for avian wildlife and are inadequate for mammalian wildlife. In domestic birds, adverse effects on growth have been reported at dietary Mo concentrations of 200 mg Mo/kg, on reproduction at 500 mg/kg, and on survival at 6,000 mg/kg.

Molybdenum chemistry is complex and inadequately known. Its toxicological properties in mammals are governed to a remarkable extent through interaction with copper and sulfur; residues of Mo alone are not sufficient to diagnose Mo poisoning. Domestic ruminants, especially cattle, are especially sensitive to Mo poisoning when copper and inorganic sulfate are deficient. Cattle are adversely affected--and die if not removed--when grazing on pastures where the ratio of copper to Mo is <3, or if they are fed low copper diets containing Mo at 2 to 20 mg/kg diet; death usually occurs when tissue residues exceed 10 mg/kg body weight. The resistance of other species of mammals tested, including domestic livestock, small laboratory animals, and wildlife, was at least 10X that of cattle. Mule deer (*Odocoileus hemionus*), for example, showed no adverse effects at dietary levels of 1,000 mg/kg.

Additional research is needed in several fields: the role of Mo on inhibition of carcinomas and dental caries; the establishment of minimum, optimal, and upper daily requirements of Mo in aquatic and wildlife species of concern; the improvement in diagnostic abilities to distinguish molybdenum poisoning from copper deficiency; and the determination of sensitivity of early developmental stages of fishes to Mo insult.

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INTRODUCTION

Molybdenum (Mo) is present in all plant, human, and animal tissues, and is considered an essential micronutrient for most life forms (Schroeder et al. 1970; Underwood 1971; Goyer 1986). The first indication of an essential role for Mo in animal nutrition came in 1953 when it was discovered that a flavoprotein enzyme, xanthine oxidase, was dependent on Mo for its activity (Underwood 1971). It was later determined that Mo is essential in the diet of lambs, chicks, and turkey poults (Underwood 1971). Molybdenum compounds are now routinely added to soils, plants, and waters to achieve various enrichment or balance effects (Friberg et al. 1975; Friberg and Lener 1986).

There are certain locations where plants will not grow optimally because of a deficiency in Mo, and other places where the levels of Mo in plants are toxic to livestock grazing on the plants (Chappell and Peterson 1976). Molybdenum poisoning in cattle was first diagnosed in England in 1938; molybdenosis was shown to be associated with consumption of herbage containing large amounts of this element, and to be controllable by treatment with copper sulfate (Underwood 1971). Molybdenum poisoning of ruminants, especially cattle, has been reported in at least 15 States, and in Canada, England, Australia, New Zealand, Ireland, the Netherlands, Japan, and Hungary. Molybdenosis was most pronounced in areas where soils were alkaline, high in Mo and low in copper, or near industrial point sources such as coal, aluminum, uranium, or molybdenum mines; steel alloy mills; or oil refineries (Dollahite et al. 1972; Alloway 1973; Kubota 1975; Buck 1978; Ward 1978; Chappell et al. 1979; Kincaid 1980; King et al. 1984; Kume et al. 1984; Sas 1987). All cattle are susceptible to molybdenosis, milking cows and young stock being the most sensitive (Underwood 1971). Industrial molybdenosis in domestic cattle and sheep, which usually involved a single farm or pasture, has been widely documented: in Colorado in 1958 from contaminated river waters used in irrigation, in Alabama in 1960 from mine spoil erosion, in North Dakota in 1968 from fly ash from a lignite burning plant, in Missouri in 1970-1972 from clay pit erosion, in Pennsylvania in 1971 from aerial contamination by a molybdenum smelter, in South Dakota in 1975 from Mo-contaminated magnesium oxide, and in Texas in 1965-1972 from uranium mine waste leachate (Ward 1978). In humans, a gout-like disease in two villages in Armenia was attributed to the ingestion of local foods high in Mo and grown in high Mo soils (Friberg and Lener 1986). Esophageal cancer was prevalent in various parts of southern Africa where food was grown in low Mo soils; it was reported in China in a low frequency rate that was significantly correlated with increasing Mo concentrations in cereals and drinking water (Luo et al. 1983). Additional and more extensive data on ecological and toxicological aspects of Mo in the environment were reviewed by Schroeder et al. (1970), Underwood (1971), Friberg et al. (1975), Chappell and Peterson (1976, 1977), Ward (1978), Chappell et al. (1979), Gupta and Lipsett (1981), and Friberg and Lener (1986).

This report was prepared in response to an informational request from environmental specialists of the U. S. Fish and Wildlife Service. It is part of a continuing series of brief reviews on chemical contaminants and natural resources.

ENVIRONMENTAL CHEMISTRY

GENERAL

Molybdenum is a comparatively rare element that is used primarily in the manufacture of steel alloys for the aircraft and weapons industries. Most of the recent global production of about 100,000 tons annually comes from the United States--primarily Colorado. Anthropogenic activities that have contributed to environmental Mo contamination include combustion of fossil fuels, and smelting, mining, and milling operations for steel, copper, and uranium, as well as for molybdenum. In general, the chemistry of Mo is complex and inadequately known. Its toxicological properties are governed to a remarkable extent by interactions with copper and sulfur, although other metals and compounds may confound this interrelation.

SOURCES AND USES

Molybdenum, discovered about 200 years ago, entered the commercial market in the 1920's as a result of extensive metallurgical research into its alloying properties and to the finding at Climax, Colorado, of the largest proven reserves of Mo worldwide (King et al. 1973). Molybdenum does not occur free in nature and is found only in combination with sulfur, oxygen, tungsten, lead, uranium, iron, magnesium, cobalt, vanadium, bismuth, or calcium. The most economically important ores are molybdenite (MoS_2), jordisite (amorphous MoS_2), and

ferrimolybdate ($FeMoO_3$ - H_20); less important are wulfenite ($PbMoO_4$) powellite ($CaMoO_4$), and ilsemannite (Mo_3O_8) (Friberg et al. 1975; Chappell et al. 1979; Friberg and Lener 1986; Goyer 1986).

World Mo production has increased from about 90 metric tons in 1900--half from Australia and Norway, half from the United States--to 136 tons in 1906, 1,364 in 1932 (an order of magnitude increase in 26 years), 10,909 in 1946, and 91,000 tons in 1973. Through the years, Mo has been produced in about 30 countries; in 1973, about 60% of the worldwide production was from the United States, 15% from Canada, 15% from the USSR and China combined, and 10% from other nations--Chile, Japan, Korea, Norway, and Mexico (King et al. 1973). By 1979, the United States produced about 62% of the world production of 103,000 metric tons, and exported about half, chiefly to western Europe and Japan; other major producers in 1979 were Canada, Chile, and the USSR (Kummer 1980). In the United States, only three mines in Colorado account for almost 70% of domestic production. Other active Mo mining sites in North America are in Arizona, Nevada, New Mexico, Utah, and California; Mo reserves have also been proven in Idaho, Alaska, Pennsylvania, and British Columbia (Kummer 1980). About 65% of domestic Mo is recovered from ores rich in Mo; the rest is a by-product from ores of copper, tungsten, and uranium (Chappell et al. 1979).

As a result of various human activities, Mo enters the environment from many sources (King et al. 1973; Friberg et al. 1975; Chappell et al. 1979). Coal combustion is the largest atmospheric source of Mo, contributing about 550 metric tons annually, or 61% of all atmospheric Mo worldwide that comes from anthropogenic sources. In Sweden alone, about 2.5 tons of Mo are emitted into the atmosphere yearly from oil combustion (Friberg et al. 1975). Molybdenum mining and milling are the source of about 100 metric tons annually to aquatic systems. At the world's largest Mo mine in Climax, Colorado, where about 36,000 tons of tailings are generated daily, the operation releases up to 100 tons of Mo annually as aqueous effluent. Other sources are Mo smelting, uranium mining and milling, steel and copper milling, oil refining, shale oil production, and claypit mining.

Molybdenum is used in the manufacture of high-strength low-alloy steels and other steel alloys in the aircraft and weapons industries, and in the production of spark plugs, X-ray tubes and electrodes, catalysts, pigments, and chemical reagents (Friberg et al. 1975; Kummer 1980; Goyer 1986). The most important industrial compound is the trioxide, MoO_3, which is resistant to most acids, and is oxidized in air at >500 °C (Shamberger 1979).

CHEMICAL PROPERTIES

Molybdenum, which can function both as a metal and metalloid, is an essential component in a large number of biochemical systems--including xanthine oxidase. At least four metalloenzymes are known that are Mo-dependent, and all are molybdoflavoproteins (Schroeder et al. 1970). Molybdenum is characterized by the following physical and chemical properties: atomic number 42; atomic weight 95.94; density 10.2; melting point 2,617 C; boiling point 4,612 °C; oxidation states 0, +2, +3, +4, +5, and +6; crystalline forms as gray-black powder, or silver-white metal; mass numbers (percent contribution of naturally occurring Mo) of 92 (15.86%), 94 (9.12%), 95 (15.7%), 96 (16.5%), 97 (9.45%), 98 (23.75%), and 100 (9.62%.); and radioactive isotopes of mass number 90, 91, 93, 99 (Tb 1/2 of 67 hours, frequently used as a tracer), 101, 102, and 105 (Busev 1969; Schroeder et al. 1970; Shamberger 1979; Friberg and Lener 1986). In water at pH >7, Mo exists primarily as the molybdate ion, MoO_4^{2-};at pH <7, various polymeric compounds are formed, including the paramolybdate ion, $Mo_7O_{24}^{6-}$ (Busev 1969). In soils, molybdate was sorbed most readily to alkaline, high calcium, high chloride soils; retention was least in low pH, low sulfate soils (Smith et al. 1987). There is general agreement that molybdenum chemistry is complex and inadequately known. Additional and more extensive information on its properties were summarized in major reviews by Busev (1969), Boschke (1978), Brewer (1980), Coughlan (1980), Newton and Otsuka (1980), Parker (1983), and Mitchell and Sykes (1986).

MODE OF ACTION

Interactions among some trace metals are so pervading and so biologically influential that the results of nutritional and toxicological studies conducted with a single element can be misleading unless the dietary and body tissue levels of interacting elements are clearly defined (Underwood 1979). For molybdenum, interactions are so dominant--especially in ruminant species--that a particular level of intake in the diet can lead to Mo

deficiency or to Mo toxicity in the animal, depending on the relative intakes of copper and inorganic sulfate (Schroeder et al. 1970; Underwood 1971, 1979; Clawson et al. 1972; Suttle 1973, 1983a; Friberg et al. 1975; Buck 1978; Ward 1978; Chappell et al. 1979; Shamberger 1979; Van Ryssen and Stielau 1980; Gupta and Lipsett 1981; Ivan and Veira 1985; Friberg and Lener 1986; Goyer 1986; Kincaid et al. 1986).

The first indications of interaction between copper and Mo came more than 40 years ago from studies of grazing cattle in certain areas of England. Afflicted animals lost weight, developed severe diarrhea, and (in extreme cases) died. The disease is sometimes called teart (rhymes with heart) or molybdenosis, and is caused by eating herbage rich in Mo--i.e., 20 to 100 mg/kg dry weight diet compared to <5 mg/kg in nearby healthy pastures--and low or deficient in copper and inorganic sulfate (Underwood 1979). Molybdenosis is a copper-deficiency disease that occurs particularly in cattle and sheep and is usually caused by the depressing effect of Mo on the physiological availability of copper (Clawson et al. 1972; Dollahite et al. 1972; Alloway 1973; Erdman et al. 1978; Mills and Breamer 1980; Van Ryssen and Stielau 1980; Nederbragt 1982; Suttle 1983a; Goyer 1986). The disease was treated successfully with copper sulfate at 1 to 2 grams daily in the diet, or 200 to 300 mg daily by intravenous injection (Buck 1978; Underwood 1979; Ivan and Veira 1985). When ruminant diets contained copper at 8 to 11 mg/kg weight--a normal range--cattle were poisoned at Mo levels of 5 to 6 mg/kg and sheep at 10 to 12 mg/kg. When dietary copper was low (i.e., <8 mg/kg) or sulfate ion level was high, Mo at 1 to 2 mg/kg ration was sometimes toxic to cattle. Increasing the copper in diets to 13 to 16 mg/kg protected cattle against concentrations up to 150 mg/kg of dietary Mo (Buck 1978). Studies of Mo metabolism are of limited value unless one knows the status in the diet of inorganic sulfate, which alleviates Mo toxicity in all known species by increasing urinary Mo excretion (Underwood 1971, 1979).

Copper prevents the accumulation of Mo in the liver and may antagonize the absorption of Mo from food. The antagonism of copper to Mo depends on sulfate, which may displace molybdate (Goyer 1986). In certain sheep pastures, for example, the herbage may contain up to 15 mg copper/kg dry weight and <0.2 mg Mo/kg dry weight--conditions favoring the development of a high copper status that may lead to copper poisoning. Treatment consists of providing molybdate salt licks, which are highly effective in reducing copper levels in grazing sheep (Buck 1978; Underwood 1979).

A low copper:molybdenum ratio (i.e., <2), rather than the absolute dietary concentration of Mo, is the primary determinant of susceptibility to molybdenum poisoning; molybdenosis is not expected when this ratio is near 5 (Buck 1978; Ward 1978; Mills and Breamer 1980). Ratios of copper to molybdenum in sweet clover (*Melilotus* spp., a known Mo accumulator plant) growing in coal mine spoils in the Dakotas, Montana, and Wyoming ranged from 0.4 to 5, suggesting that molybdenosis can be expected to occur in cattle and sheep grazing in low Cu:Mo areas (Erdman et al. 1978). A similar situation existed in British Columbia, where 19% of all fodders and grains had a Cu:Mo ratio <2 (Underwood 1979).

There are several explanations for the high sensitivity of ruminants to increased dietary Mo and sulfur, the most plausible being the role of thiomolybdates (Penumarthy and Oehme 1978; Lamand et al. 1980; Nederbragt 1980, 1982; Suttle 1980, 1983b; Mills et al. 1981; Suttle and Field 1983; Weber et al. 1983; Hynes et al. 1985; Friberg and Lener 1986; Allen and Gawthorne 1987; Sas 1987; Strickland et al. 1987). Thiomolybdates are compounds formed by the progressive substitution for sulfur and oxygen in the molybdate (MoO_4^{2-}) anion when hydrogen sulfide and MoO_4^{2-} interact in vitro at neutral pH. Di-, tri-, and tetra-thiomolybdates are formed, but only the last of these effectively impairs copper absorption. When sufficient tetrathiomolybdate (MoS_4) is formed in the rumen, it and copper in the gut combine and the resultant complex is bound strongly to proteins of high molecular weight. The molybdoproteins so formed are strong chelators of copper, and may be the agents responsible for copper deficiency through formation of biologically unavailable copper complexes in gut, blood, and tissues of animals that consume diets containing high concentrations of Mo. To confound matters, the complex molybdenum-copper-sulfur interrelation can be modified, or disrupted entirely, by many compounds or mixtures. These include the salts of tungsten (Schroeder et al. 1970; Underwood 1971; Mills and Breamer 1980; Luo et al. 1983; Goyer 1986), zinc (Penumarthy and Oehme 1978; Parada 1981; Alary et al. 1983), lead and manganese (Underwood 1971), iron (Phillippo et al. 1987b), vanadium (Vaishampayan 1983), chromium (Vaishampayan 1983; Chung et al. 1985), phosphorus (Underwood 1971; Baldwin et al. 1981), cystine and methionine (Underwood 1971, 1979), fluoride (Goyer 1986), and proteins (Underwood 1971, 1979; Friberg and Lener 1986; Kincaid et al. 1986).

BACKGROUND CONCENTRATIONS

GENERAL

Molybdenum levels tend to be elevated in nonbiological materials and in terrestrial flora in the vicinity of Mo mining and reclamation activities, fossil-fuel power plants, and disposal areas for Mo-contaminated sewage sludge, fly ash, and irrigation waters. Concentrations of Mo in fish, wildlife, and invertebrates were low when compared to those in terrestrial plants, although certain aquatic invertebrates were capable of high bioconcentration. Concentrations of Mo alone, however, were not sufficient to diagnose Mo deficiency or toxicosis.

NONBIOLOGICAL SAMPLES

Elevated levels of Mo in nonbiological materials have been reported near certain mines, power plants, and oil shale deposits, as well as in various sewage sludges, fertilizers, and agricultural drainwaters (Table 1).

Molybdenum is concentrated in coal and petroleum, and the burning of these fuels contributes heavily to atmospheric Mo (King et al. 1973). Combustion of fossil fuels contributes about 5,000 metric tons of Mo annually to the atmosphere; atmospheric particulates contain about 0.001 ug Mo/m^3 air (Goyer 1986).

Natural Mo concentrations in ground and surface waters rarely exceed 20 ug/l; significantly higher concentrations are probably due to industrial contamination. Existing wastewater and water treatment facilities remove less than 20% of the Mo; accordingly, drinking water concentrations are near those of the untreated source (Chappell et al. 1979). Molybdenum concentrations in saline waters appear to be directly related to salinity (Prange and Kremling 1985; Sloot et al. 1985). In the Wadden Sea, for example, Mo concentrations were 0.08, 0.4, and 1.0 ug/l at salinities of 0.07, 1.2, and 3.3%, respectively (Sloot et al. 1985).

Table 1. Molybdenum concentrations in selected nonbiological materials.

Material, unit and location	Concentration [a]	Reference [b]
Seawater (ug/L)		
Worldwide	<1 to 10	1, 2, 3
Worldwide	4 to 12	4
Pacific Ocean, all depths	10.3 + 0.2	5
Drinking water (ug/L)		
USSR		
Winter	0.03 to 0.06	1
Summer	0.11 to 0.15	1
USA	0.1 to 6.2	2
USA	Usually <5, sometimes	
up to 500	1, 4	
Switzerland	Usually <1, Max. 29	1
Surface water (ug/L)		
North American rivers	0.4	4
California lakes	0.4 (<3 to 100)	1
USA rivers	1.2 to 4.1	1
Mineral waters	2 to 3	2
Near Mo mine and mill, Colorado	100 to 10,000	4

Ash pond effluent from coal fired power plant, New Mexico	170	4
Power station effluent, Vicotria Australia	330	6
Near Mo tailings pile, New Mexico	600	4
Evaporation ponds, California, 1985–86	1,100 (630 to 2,600)	7
Leachate from oil shale retort, Colorado	4,100 (2,500 to 8,300)	4
Irrigation water from Mo mining and reclamation	5,000 to 100,000	8
Groundwater (ug/L)		
USA	Usually <1	4
USSR	3	4
California, agricultural drain-water, 1985–86	1,200 to 5,500	7
Colorado		
Mining areas	Max. 25,000	1
Near urnaium mill	50,000	4
Sediments (mg/kg, dry weight)		
USA rivers	5 to 57	1
Evaporation ponds, California	18 (<2 to 22)	7
Near Mo tailings pile, Colorado	21	4
Baltic Sea	80	3
Near Mo mine and mill, Colorado	530, Max. 1,800	1, 4
Soils (mg/kg, dry weight)		
Natural soils		
Worldwide	0.1 to 10, usually 0.2 to 0.7	1, 2
Worldwide	1 to 2 (0.6 to 3.5)	4
USA	1.2 (0.1 to 40)	4
Molybdenosis areas	2 to >6	9
Elevated Mo	12 to 76 (2 to 190)	4
Economic Mo deposits	>200	4
Impacted soils		
In upper 5 cm at 0.3 or 3 km from Mo ore processing plant in 1982 and 1983		
0.3 km		
1982, Total	28	10
1982, Extractable	5	10
1983, Total	73	10
1983, Extractable	3	10

3 km		
1982, Total	3	10
1982, Extractable	0.4	10
1983, Total	8	10
1983, Extractable	0.8	10
Near Mo mine and mill, Colorado		
Irrigated with Mo-contaminated		
effluent from uranium mill	61 (49 to 72)	4
Ireland, highly mineralized	170 (11 to 4,000)	17
Sewage sludge (mg/kg, dry weight)		
Iowa	<1 to 75	11
USA	2 to 30	2
Most states, USA	5 to 39	11
North America	<10 (2 to 100)	1, 12
Michigan	32 (6 to 3,700)	11
Air (ugm^3)		
Rural, USA	0.0001 to 0.003	1, 2
Urban, USA	0.01 to 0.03	1, 2
Worldwide	<0.0005	13
Fertilizers (mg/kg, dry weight)		
Domestic	3 to 6	1, 14
Oil, oil shale, coal, and waste products		
(liquids, mg/L; solids, mg/kg dry weight)		
Coal conversion process waters	0.001 to 0.5	15
Oil shale retort water	0.06 to 0.3	15
Light oil	<0.1	1
Heavy oil	Max. 0.5	1
Spent oil shale	0.6	15
Coal	1 to 73	15
Coal	3 (0.3 to 15)	1, 16
Oil shale	5 to 87	15
Coal ash	7 to 160	16
Fly ash from power stations	Usually 10 to 40, Max 180	1

[b]References: 1, Friberg et al. 1975; 2, Friberg and Lener 1986; 3, Prange and Kremling 1985; 4, Chappell et al. 1979; 5, Collier 1985; 6, Ahsanullah 1982; 7, Fujii 1988; 8, Smith et al. 1987; 9, Kubota et al. 1967; 10, Schalscha et al. 1987; 11, Pierzynski and Jacobs 1986; 12, Lahann 1976; 13, Schroeder et al. 1970; 14, Goyer 1986; 15, Birge et al. 1980; 16, Elseewi and Page 1984; 17, Talbot and Ryan 1988.

The Mo content of soil may vary by more than an order of magnitude, causing both deficient and excessive concentrations for plants and ruminants in some parts of the world (Friberg et al. 1975). Native soils may contain enough Mo to cause molybdenosis in range livestock in some areas of the United States, particularly in Oregon, Nevada, and California (Kubota et al. 1967; Erdman et al. 1978). Elevated soil Mo levels can result from both natural and industrial sources. Usually when soil Mo levels exceed 5 mg/kg dry weight, a geological anomaly or industrial contamination is the likely explanation (Chappell et al. 1979). Molybdenum is more

available biologically to herbage plants in alkaline soils than in neutral or acidic soils (Underwood 1971; Friberg et al. 1975; Shacklette et al. 1978; Wright and Hossner 1984). Liming of acidic soils or treatment with Mo-containing fertilizers can effectively raise the Mo content of herbage (Underwood 1971; Pierzynski and Jacobs 1986).

The disposal of sewage sludge, fly ash from coal combustion, and Mo-contaminated irrigation waters to agricultural fields may result in the production of Mo-rich herbage. Sewage sludges rich in Mo and applied to agricultural soils resulted in elevated Mo content in corn and soybeans in a dose-dependent pattern (Pierzynski and Jacobs 1986). Similarly, fly ash from coal combustion applied to pasture and croplands at rates sufficient to provide Mo at concentrations of 40 g/kg and higher resulted in potentially hazardous levels in vegetation to ruminant grazers. Molybdenum in fly ash applied to soils remained biologically available for extended periods, especially in calcareous soils (Elseewi and Page 1984). Irrigation has also been proposed as a possible disposal method for large quantities of water having Mo concentrations of 5 to 100 mg/l that result from mining and reclamation activities. This method of disposal is not recommended unless all animals are kept off irrigated sites and the vegetation can be harvested and destroyed until Mo levels in the plants remain below 10 mg/kg dry weight (Smith et al. 1987).

BIOLOGICAL SAMPLES

All plants contain Mo and it is essential for the growth of all terrestrial flora (Schroeder et al. 1970). Molybdenum concentrations were elevated in terrestrial plants, especially in those collected from soils amended with fly ash, liquid sludge, or Mo-contaminated irrigation waters, in naturally occurring teart pastures, and in the vicinity of Mo mining and ore processing activities, steelworks, and other metal processors; Mo concentrations greater than 20 mg/kg dry weight were frequently documented in plants from contaminated areas (Table 2). Legumes, especially trefoil clovers (*Lotus* sp.) selectively accumulated Mo; concentrations of 5 to 30 mg/kg dry weight were common in Mo-contaminated areas (Friberg et al. 1975; Shacklette et al. 1978). The Mo levels were sometimes high and potentially toxic in legumes from poorly drained acidic soils (Kubota et al. 1967; Underwood 1971). Some terrestrial grasses displayed copper:Mo ratios between 0.5 and 3.7. Since ratios greater than 2 were within the range where molybdenosis is likely, and since most of the Mo concentrations were greater than the maximum tolerable level of 6 mg/kg dry weight, hypocuprosis (molybdenosis) in cattle was expected (Schalscha et al. 1987). Major sources of Mo overload in fodder were in plants grown on high-Mo alkaline soils and from industrial contamination by coal and uranium mines and alloy mills (Sas 1987). Variations in Mo content of pasture species ranged from 0.1 to 200 mg/kg dry weight, and most variations were due to soil and species differences (Underwood 1971). Pasture plants collected from mountainous areas of southern Norway were usually deficient in copper, and low to partly deficient in Mo. As a result, the copper:Mo ratios were generally high and may explain the occurrence of chronic copper poisoning in grazing sheep in that region (Garmo et al. 1986).

Except in terrestrial plants, Mo concentrations were low in all groups examined; maximum concentrations reported from all sampling locales were about 6 mg/kg dry weight in aquatic plants, about 4 mg/kg fresh weight in aquatic invertebrates, 2 mg/kg fresh weight in fishes (except for rainbow trout liver and kidney--26 to 43 mg/kg fresh weight--from fish collected near a Mo tailings outfall), 4 mg/kg dry weight in birds, 30 mg/kg dry weight in domestic ruminant liver, 85 mg/kg dry weight in the horse, and <4 mg/kg dry weight in mammalian wildlife and man (Table 2).

There are large interspecies differences among aquatic organisms in their ability to accumulate Mo from the medium. Marine bivalve molluscs usually contained 30X to 90X more Mo than the ambient seawater; however, some species from Greek waters had bioconcentration factors up to 1,300X (Eisler 1981). Marine plankton accumulated Mo from seawater by factors up to 25X (Goyer 1986). But growth in aquatic phytoplankton populations was inhibited under conditions of low or missing Mo, nitrogen, and organic matter concentrations; the role of Mo in this process requires clarification (Paerl et al. 1987). In rainbow trout (*Oncorhynchus mykiss*), residues of Mo in tissues were affected only slightly by the concentrations in water; tissue residues ranged from 5 to 118 ug/kg fresh weight in water containing trace (<6 ug/1) concentrations, 10, to 146 ug/kg in water containing low (6 ug/1) concentrations, and from 13 to 322 ug/kg in water containing high (300 ug/1) concentrations (Ward 1973). A similar pattern was reported for kokanee salmon, *Oncorhynchus nerka* (Ward 1973). Rainbow trout held for 2 weeks in live traps 1.6 km downstream from a Mo mine tailings outfall survived, but liver and kidney had significantly elevated levels of Mo, calcium, manganese, iron, zinc, strontium, and

zirconium, and 10% less potassium; the observed mineral changes may have been due to outfalls from nonmolybdenum mines discharged into the river system (Kienholz 1977).

Molybdenum mining operations are not detrimental to mammalian wildlife, as judged by normal appearance and low Mo levels in liver and kidney of nine species--including deer, squirrel, chipmunk, badger, beaver, marmot, and pika--collected from areas of high environmental Mo levels (Kienholz 1977). I must emphasize, however, that Mo levels in animal tissues give little indication of the dietary Mo status, and are of little diagnostic value for this purpose unless the sulfate, protein, and copper status of the diet are also known. This point is discussed in greater detail later.

Table 2. Molybdenum concentration in field collections of selected species of animals and plants. Values shown are Mo in mg/kg (ppm Mo) fresh weight (FW), dry weight (DW), or ash weight (AW).

Taxonomic group, organism tissue, and other variables	Concentration, [a] in mg/kg	Reference [b]
Terrestrial plants		
Bermuda grass, *Cynodon dactylon*		
Soil amended with Mo-contaminated irrigation water		
Control	5 DW	1
6 mg Mo/kg soil	225 DW	1
13 mg Mo/kg soil	309 DW	1
26 mg Mo/kg soil	447 DW	1
Herbage (forage)		
Normal	1 to 3 DW	2
Teart pastures	20 to 100 DW	2
Barley, *Hordeum vulgare*		
Soil amended with fly ash		
40 g/kg soil	6 DW	3
80 g/kg soil	11 DW	3
Moss, *Hypnum cupressiforme*,		
Sweden		
Normal	1 DW	4
Near waste disposal plant	8 DW	4
Near metal processor	400 DW	4
Near steelworks	560 DW	4
Legumes		
From molybdenosis areas	17 to 125 DW	5
From nonmolybdenosis areas	6 to 28 DW	5
Black medic, *Medicago lupulina*		
Carson Valley, Nevada	Max. 372 DW	6
Alfalfa, *Medicago sativa*		
Soil amended with fly ash		
40 g/kg soil	10 DW	3
80 g/kg soil	12 DW	3
Pasture plants, southern Norway	0.3 (0.01 to 4) DW	7

Peas, *Pisum sativum*		
Canada	0.2 FW	4
USA	0.3 to 5 FW	4
India	0.7 to 2 FW	4
Romania, Germany	1 FW	4
USSR	6 FW	4
Ballica grass, *Lolium perenne*		
Distance from Mo ore processing plant		
1982		
0.3 km	29 to 40 DW	8
1.0 km	8 to 10 DW	8
1983		
0.3 km	6 to 10 DW	8
1.0 km	7 to 10 DW	8
9.0 km	4 to 5 DW	8
In soil amended with liquid sludge to contain 410 mg Mo/ha	20 DW	9
White clover, *Trifolium repens*		
Soil amended with fly ash		
40 g/kg soil	27 DW	3
80 g/kg soil	36 DW	3
Soil amended with liquid sludge		
17 mg Mo/ha	31 DW	9
410 mg Mo/ha	90 DW	9
Wheat, *Triticum aestivum*		
Germany, Romania, USSR	0.2 to 0.8 FW	4
India	0.5 FW	4
USA	0.6 to 6 FW	4
Vegetables		
Mo symptoms in man	11 to 82 DW	4
Control site	3 to 5 DW	4
Vegetation		
Near Mo mine	Max. 5,400 AW	6
Normal	<2 to 500 AW	6
Aquatic plants		
Algae, whole		
Marine	0.03 to 0.2 FW; 0.1 to 1.3 DW	4
Canada, 11 species	0.2 to 1.4 DW	10
Marine plants	0.5 FW	11
Marsh plants, whole		
Texas, 14 species	0.4 to 2.5 DW	10
Seaweeds, whole		
UK, 5 species	0.2 to 1.3 DW; 0.04 to 0.2 FW	10

Norway, 11 species	0.3 to 6 DW	4
Aquatic invertebrates		
Aquatic insects, 4 species		
Near low Mo waters (<1.0 ug Mo/L)	0.3 to 1.4 DW	12
Upstream	Max. 0.2 DW	12
Downstream	Max. 0.3 DW	12
Corals, marine,		
34 species	<2 DW	13
Crustaceans, marine		
Tissues sold for human consumption,		
16 species	0.1 to 0.4 FW	14
Molluscs, marine		
Soft parts		
15 species	<0.1 to 0.6 FW	14
3 species	0.7 to 4 FW	14
Mussel, *Mytilus edulis aoteanus*		
Soft parts	0.6 DW	15
Gill	0.6 DW	15
Visceral mass	2 DW	15
Shell	11 DW	15
Other tissues	<0.1 DW	15
Scallop, *Pecten novae-zelandiae*		
Soft parts	0.9 DW	15
Mantle	2 DW	15
Gill	3 DW	15
Intestine	4 DW	15
Kidney	3 DW	15
Foot	0.4 DW	15
Plankton, Baltic Sea	2 DW	16
Fish		
Fishes, marine		
Liver		
43 species	0.1 to 0.3 FW	14
29 species	0.4 to 2.0 FW	14
2 species	0.4 to 1.0 DW	17
Muscle		
130 species	0.1 to 0.3 FW	14
29 species	0.4 to 0.6 FW	14
2 species	0.3 to 0.12 DW	17
Various	Max. 0.04 FW	11
Whole		
17 species	0.1 to 0.6 FW	14
8 species	0.012 to 0.15 FW	18

Rainbow trout, *Oncorhynchus mykiss*		
From low Mo waters (<6 ug/L)		
Liver	0.04 to 0.1 FW	19
Spleen	0.05 to 0.9 FW	19
Kidney	0.1 FW	19
Skin	0.07 FW	19
Bone	0.1 to 0.15 FW	19
Muscle	0.01 FW	19
Intestine	0.01 to 0.07 FW	19
Stomach	0.04 FW	19
Brain	0.02 FW	19
From high Mo waters (300 ug/L)		
Liver	0.2 FW	19
Spleen	0.2 FW	19
Kidney	0.15 FW	19
Skin	0.1 FW	19
Bone	0.2 FW	19
Muscle	0.01 FW	19
Intestine	0.1 FW	19
Stomach	0.3 FW	19
Brain	0.09 FW	19
Held 2 weeks in live traps 1.6 km downstream from Mo tailings outfall		
Liver	43 DW	20
Kidney	26 DW	20
Control location		
Liver	1 DW	20
Kidney	<2 DW	20
Birds		
Chicken, *Gallus* sp.		
Liver	3.6 DW	21
Kidney	4.4 DW	21
Muscle	0.1 DW	21
Robin, *Turdus migratorius*		
From Mo mine site		
Liver	1.6 DW	20
Kidney	1.9 DW	20
Mammals		
Alaskan moose, *Alces alces gigas*		
Hair	0.1 to 0.6 DW	22
Cattle, cows, *Bos* spp.		
Normal		
Blood	0.06 FW	2

Milk	0.07 (0.02 to 0.2) FW	2, 4
Liver	0.7 to 2 FW; 2.9 to 5.4 DW	2, 11, 23
Kidney	0.3 FW; 1.3 to 2.7 DW	2, 23
Muscle	0.1 FW; 0.5 DW	2, 23
Feces	1.1 to 2.1 DW	23
Elevated or poisoned		
Blood	0.6 to 0.8 FW	2
Kidney	21 FW	11
Rumen contents	21 to 28 DW	24
Horse, *Equus caballus*		
Liver	3 to 85 DW	21
Man, *Homo sapiens*		
Liver	0.5 to 1.0 FW; 3.2 DW	21, 25
Liver cortex	0.9 FW	21, 25
Kidney	0.2 to 0.3 FW; 1.6 DW	21, 25
Kidney cortex	0.2 FW	21, 25
Adrenal	0.7 FW	21, 25
Amnion	3.5 FW	21, 25
Chorion	0.6 FW	21, 25
Spleen	0.2 DW	21, 25
Lung	0.15 DW	21, 25
Brain	0.14 DW	21, 25
Muscle	0.14 DW	21, 25
Hair	0.06 (0.02 to 0.13) DW	21, 25
Blood	<0.005 to 0.1 FW	21, 25
Mule deer, *Odocoileus hemionus*		
Liver		
Mo mining area	1.0 FW	26
Control site	0.6 FW	26
Healthy	1.3 FW	26
Sheep, *Ovis aries*		
Wool	0.2 (0.03 to 0.6) DW	21
Liver		
Normal diet		
Adults	2 to 4 DW	21
Newborn lambs	2 to 4 DW	21
High Mo diet		
Adults	25 to 30 DW	21
Newborns	12 to 20 DW	21
Milk		
Grazing on low Mo		
(<1 mg/kg) pasture	<0.01 FW	21
Grazing on high Mo (13 mg/kg) pasture	>1 FW	21

Grazing on high Mo (25 mg/kg) pasture, and given high sulfate (23 g/daily) for 3 days	0.1 FW	21
As above, without sulfate administration	1 FW	21
Rat, *Rattus* sp.		
Liver	2 DW	21
Kidney	1 DW	21
Spleen	0.5 DW	21
Lung	0.4 DW	21
Brain	0.2 DW	21
Muscle	0.06 DW	21
Wildlife, 9 species		
From areas of high environmental Mo levels		
Liver	0.1 to 4 DW	20
Kidney	0.3 to 3 DW	20

[a]Concentrations are listed as means, minimum-maximum (in parentheses), and maximum (Max.).

[b]References: 1, Smith et al. 1987; 2, Penumarthy and Oehme 1978; 3, Elseewi and Page 1984; 4, Friberg et al. 1975; 5, Kubota et al. 1967; 6, Shacklette et al. 1978; 7, Garmo et al. 1986; 8, Schalscha et al. 1987; 9, Pierzynski and Jacobs 1986; 10, Eisler 1981; 11, Schroeder et al. 1970; 12, Colborn 1982; 13, Livingston and Thompson 1971; 14, Hall et al. 1978; 15, Brooks and Rumsby 1965; 16, Prange and Kremling 1985; 17, Papadopoulu et al. 1981; 18, Rao 1984; 19, Ward 1973; 20, Kienholz 1977; 21, Underwood 1971; 22, Flynn et al. 1976; 23, Kume et al. 1984; 24, Sas 1987; 25, Friberg and Lener 1986; 26, King et al. 1984.

EFFECTS

GENERAL

Trace quantities of Mo are beneficial and perhaps essential for normal growth and development of plants and animals. In mammals, Mo can protect against poisoning by copper, mercury, and probably other metals, and may have anticarcinogenic properties. For all organisms, the interpretation of Mo residues depends on knowledge of Mo, copper, and inorganic sulfate concentrations in diet and in tissues.

Some Mo compounds have insecticidal properties at low concentrations and have been proposed as selective termite control agents.

Aquatic flora and fauna seem to be comparatively resistant to Mo salts; adverse effects on growth and survival were usually noted only at water concentrations of 50 mg Mo/l, and higher. However, one study with newly fertilized eggs of rainbow trout produced an LC-50 (28 day) value of 0.79 mg Mo/l compared to an LC-50 (96 hour) value of 500 mg/l for adults. Also, bioconcentration of Mo by selected species of algae and invertebrates (up to 20 g/kg dry weight) poses questions on risk to higher trophic level organisms.

In birds, adverse effects of Mo have been reported on growth at dietary concentrations of 200 to 300 mg/kg, on reproduction at 500 mg/kg, and on survival at 6,000 mg/kg.

In mammals, cattle are especially sensitive to Mo poisoning, followed by sheep, under conditions of copper and inorganic sulfate deficiency. Cattle were adversely affected when grazing pastures with a copper:Mo ratio <3, or when fed low copper diets containing 2 to 20 mg Mo/kg diet, or when total daily intake approaches 141 mg Mo; cattle usually die at doses of 10 mg Mo/kg body weight. Other mammals, including horses, pigs,

rodents, and ruminant and nonruminant wildlife are comparatively tolerant to Mo. Deer, for example, are at least 10 times more resistant than domestic ruminants to Mo; no adverse effects in deer were noted at dietary levels of 1,000 mg/kg after 8 days, slight effects at 2,500 mg/kg after 25 days, and reduction in food intake and diarrhea at 5,000 mg/kg diet after 15 days.

TERRESTRIAL PLANTS

In a major literature review, Gupta and Lipsett (1981) concluded that Mo was essential for plant growth due to its role in the fixation of nitrogen by bacteria using the enzymes nitrogenase and nitrate reductase, and that plants readily accumulated MoO_4^{2-} except under conditions of low pH, high sulfate, and low phosphate, and in some highly organic soils. Molybdenum deficiency has been recorded in a variety of crops worldwide, but there is an extremely narrow range between adequacy and deficiency. In lettuce (*Lactuca sativa*), for example, adverse effects were noted at 0.06 mg/kg (dry weight) in plants, but sufficiency was attained at 0.08 to 0.14 mg/kg; a similar case is made for *Brassica* spp., i.e., Brussels sprouts, cabbage, and cauliflower (Gupta and Lipsett 1981). In certain species, such as beets (*Beta vulgaris*) and corn (*Zea mays*), the ratio between deficiency and sufficiency may differ by more than 10X (Gupta and Lipsett 1981).

Okra (*Abelmoschus esculentus*), grown in soils supplemented with Mo at 1, 2, or 3 mg/kg, as sodium molybdate, showed increasing growth and yields when compared to nonsupplemented soils; fruiting occurred earlier and persisted longer with increasing Mo concentration (Singh and Mourya 1983). The cashew (*Anacardium occidentale*)--one of the most valuable plantation crops in India--developed yellow- leaf spots accompanied by low Mo levels and excess manganese in low pH soils; in extreme cases the tree was defoliated (Subbaiah et al. 1986). The disorder was corrected by foliar spraying of Mo salts or by liming the soil. A similar case was reported for Florida citrus in the 1950's, which was shown to be due to Mo deficiency (Subbaiah et al. 1986).

Soils amended with sewage sludge containing 12 to 39 mg Mo/kg dry weight (soil contained 2 mg Mo/kg dry weight at start and 4.8 to 6 mg/kg after treatment) were planted with corn and bromegrass (*Bromus inermis*). A lime-treated sludge increased Mo concentrations in plant tissues after several years of sludge application; maximum values recorded were 1.9 mg Mo/kg dry weight in bromegrass and 3.7 in corn (Soon and Bates 1985). No toxicity of Mo has yet been observed in field-grown crops, although forages containing 10 to 20 mg/kg dry weight are considered toxic to cattle and sheep (Soon and Bates 1985).

TERRESTRIAL INVERTEBRATES

Sodium molybdate and other molybdenum compounds in toxic baits have potential for termite control (Brill et al. 1987). Baits containing 1,000 mg Mo/kg were fatal to 99% of the termite *Reticulitermes flavipes* in 48 days. After 8 to 10 days, termites became steel-gray in color, but appeared otherwise normal; mortality began only after day 16. Termites did not avoid the poisoned bait, even at concentrations of 5,000 mg Mo/kg. Yoshimura et al. (1987) reported similar results with another species of termite; sodium molybdate killed 100% of the workers in a colony of *Coptotermes formosanus* within 24 hours after they ate filter paper treated with a 5% solution. Some other species of insects--including fire ants (*Solenopsis* sp.) and various species of beetles and cockroaches--were not -affected when exposed to baits containing 5,000 mg Mo/ kg for 48 days (Brill et al. 1987).

AQUATIC ORGANISMS

Aquatic plants are comparatively resistant to Mo; in sensitive species adverse effects were evident on growth at 50 mg/l, and on development at 108 mg/l (Table 3). Bioconcentration of Mo from the medium by certain freshwater algae can result in residues up to 20 grams/kg dry weight without apparent damage (Table 3); the implications of this phenomenon to waterfowl and to other species that consume Mo-laden algae need to be explored.

Molybdenum is considered essential for aquatic plant growth, but the concentrations required are not known with certainty and are considered lower than those for any other essential element (Schroeder et al. 1970; Henry and Tundusi 1982). Molybdenum starvation restricts nitrogen fixation in algae, thereby limiting photosynthetic production during depleted conditions. Blue-green alga (*Anabaena oscillaroides*) cultured in Mo-deficient media containing 0.004 to 0.005 ug Mo/l rapidly depleted Mo in the medium; this ability was lost at higher

concentrations of added Mo, when *Anabaena* began to accumulate the element (Steeg et al. 1986). The addition of tungstate to Mo-deficient media enhances dinitrogenase inactivation, resulting in inhibited algal growth; this process is reversed at Mo levels of 0.005 to 0.04 ug/l (Steeg et al. 1986). On the other hand, algal growth was significantly enhanced when vanadium (V) was present at 12.5 ug/l, although higher concentrations of V were growth inhibitory in 7 days (Vaishampayan 1983).

Algal uptake of Mo is rapid during the first 2 hours, and slower thereafter; the sequential biological reduction of hexavalent to pentavalent to trivalent Mo occurs intracellularly in green algae (Sakaguchi et al. 1981). Uptake is greater in freshwater than in seawater, greater at increased doses, and greater at reduced algal densities (Sakaguchi et al. 1981); it is also greater at elevated temperatures (Penot and Videau 1975).

Molybdenum occurs naturally in seawater as molybdate ion, MoO_4^{2-}, at about 10 ug/l (Abbott 1977). Despite the high concentrations of dissolved Mo in offshore seawater, phytoplankton from offshore locales contain extremely low Mo residues, almost typical of Mo-deficient terrestrial plants (Howarth and Cole 1985). This phenomenon is attributed to the high concentrations of sulfate in seawater; sulfate inhibits molybdate assimilation by phytoplankton, making it less available in seawater than in freshwater. As one result, nitrogen fixation and nitrate assimilation--processes that require Mo--may require greater energy expenditure in marine than in fresh waters and may explain, in part, why marine ecosystems are usually nitrogen-limited and lakes are not (Howarth and Cole 1985). Experimentally increasing the ratio of sulfate to molybdate inhibits molybdate uptake by marine algae, slows nitrogen fixation rates, and slows the growth of organisms that use nitrate as a nitrogen source (Howarth and Cole 1985).

Table 3. Molybdenum effects on selected species of aquatic organisms.

Organism, Mo concentration, and other variables	Effect	Reference [a]
Aquatic plants		
Blue-green alga, *Anabaena oscillaroides*		
0.005 ug/L	Bioconcentration factor (BCF) of 3,300 in 60 min	1
0.073 ug/L	BCF of 550 in 60 min	1
25 ug/L	BCF of 7 to 24 in 60 min	1
Green alga, *Chlorella vulgaris*		
10 mg/L	Residues of about 20,000 mg/kg dry weight in 20 h. Dead (heat-killed) *Chlorella* contained 4,902 mg/kg dry weight in 1 h versus 3,264 mg/kg in live cells	2
20 mg/L	Normal growth in 96 h	2
50 mg/L	Reduced growth in 96 h	2
Euglena, *Euglena gracilis*		
5.4 mg/L	Normal growth and reproduction	3
96 mg/L	No abnormal cells in 48 h	3
108 mg/L	Abnormal development, cells forming clusters. Culture is photosensitive with blue color	3
>960 mg/L	No growth	3
Freshwater alga, *Nitella flexilis*		
0.014 ug/L	BCF of 628 in 25 days	4
3.3 mg/L	BCF of 39 in 24 days; elevated	

	residues of 130 mg/kg fresh weight	4
Blue-green alga, *Nostoc muscorum*		
17.7 ug/L	Required for growth	5
Invertebrates		
Amphipod, *Allorchestes compressa*		
60 mg/L	No deaths (=LC-0) in 96 h	6
247 mg/L	50% dead (=LC-50) in 96 h	6
450 mg/L	97% dead (=LC-97) in 96 h	6
Starfish, *Asterias rubens*		
127 mg/L	LC-50 (24 h) at pH 5.8	7
254 mg/L	LC-50 (24 h) at pH 8.2	7
Copepod, *Calanus marshallae*		
20 mg/L	Minor increase in oxygen consumption in 24 h	8
100 mg/L	Decreased oxygen consumption in 24 h	8
560 mg/L	LC-50 (19 days)	8
Green crab, *Carcinus maenus*		
1,018 mg/L	LC-50 (48 h)	7
American oyster, *Crassostrea virginica*		
1,375 mg/L	Reduction of 50% in shell growth in 96 h	9
Hermit crab, *Eupagurus bernhardus*		
100 mg/L	LC-0 (50 days)	7
222 mg/L	LC-50 (48 h)	7
Euphausiid, *Euphausia pacifica*		
560 mg/L	LC-50 (112 h)	8
Amphipod, *Gammarus* sp.		
3.3 mg/L	BCF of 4.8 in whole animal in 24 days	4
Lake periphyton		
0.014 ug/L	BCF of 3,570 in 24 days	4
Clam, *Margaretifera margaretifera*		
3.3 mg/L	Maximum BCF values in 15 to 24 days were 1.8 in shell, 0.9 in soft parts, and 0.3 in muscle	4
Mysid shrimp, *Mysidopsis bahia*		
1,205 mg/L	LC-50 (96 h)	9
Mussel, *Mytilus edulis*, larvae		
147 mg/L	Development reduced 50% in 48 h, based on survival and abnormalities	10
Crayfish, *Pacifiastacus leniusculus*		
3.3 mg/L	BCF in 24 days of 5.7 for muscle and 9.8 for carapace	4
Pink shrimp, *Penaeus duorarum*		
1,909 mg/L	LC-50 (96 h)	9
Pullet-shell (clam), *Venerupis pallustra*		
381 mg/L	LC-50 (24 h)	7

Fish		
Sheepshead minnow, *Cyprinodon variegatus*		
3,057 mg/L	LC-50 (96 h)	9
Bluegill, *Lepomis macrochirus*		
1,320 mg/L	LC-50 (96 h)	11
Fathead minnow, *Pimephales promelas*		
70 mg/L	LC-50 (96 h), soft water	11
360 mg/L	LC-50 (96 h), hard water	11
Steelhead trout, *Oncorhynchus mykiss*		
0.014 ug/L	Max. BCF of 1,143 in liver and gastrointestinal tract after chronic exposure	4
3.3 mg/L	Max. BCF in 24 days of 5.4 in spleen 4.5 in liver, 2.3 in muscle, 1.8 in gill, and 0.6 in gastrointestinal tract	4
Rainbow trout, *O. mykiss*		
Embryo and larval stages exposed for a total of 28 days starting at fertilization through 4 days posthatch		
28 ug/L	LC-1 (28 days)	12
125 ug/L	LC-10 (28 days)	12
790 (610 to 990) ug/L	LC-50 (28 days)	12
17.0 to 18.5 mg/L, exposed continuously for one year from eyed eggs to juvenile stage	No significant effect on survival, growth, or blood hematocrit	10, 11
500 mg/L	LC-25 (96 h), mean length 20 mm	11
800 mg/L	LC-50 (96 h), mean length 20 mm	11
1,320 mg/L	LC-50 (96 h), mean length 55 mm	11

[a]References: 1, Steeg et al. 1986; 2, Sakaguchi et al. 1981; 3, Colmano 1973; 4, Short et al. 1971; 5, Vaishampayan 1983; 6, Ahsanullah 1982; 7, Abbott 1977; 8, Anderson and Mackas 1986; 9, Knothe and Van Riper 1988; 10, Morgan et al. 1986; 11, McConnell 1977; 12, Birge et al. 1980.

Limited data suggested that aquatic invertebrates were very resistant to Mo; adverse effects were observed on survival at >60 mg Mo/l and on growth at >1,000 mg Mo/l (Table 3). Bioconcentration factors were low, but depending on initial dose, measured residues (mg/kg fresh weight) were as high as 16 in amphipods, and were 3 in clams, 18 in crayfish muscle, and 32 in crayfish carapace (Short et al. 1971). The host organisms seemed unaffected under these Mo burdens, but effects on upper trophic level consumers were not clear. Tailings from a pilot molybdenum mine on the North American Pacific coast were acutely lethal at concentrations of >61,000 mg tailings solids/1 seawater to larvae of the mussel *Mytilus edulis*, and to adults of the amphipod *Rhepoxynius abronius* and the euphausiid *Euphausia pacifica*; acute sublethal effects were observed at >277,000 mg/l (Mitchell et al. 1986). All species of invertebrates tested in this preliminary study were more sensitive than juvenile coho salmon, *Oncorhynchus kisutch* (Mitchell et al. 1986). In another study, zooplankton exposed to Mo mine tailings <8 um in diameter at high sublethal concentrations ingested and excreted these particles (-

Anderson and Mackas 1986). The lowest tailing concentration tested at which a deleterious effect was observed was 100 mg/l for depression of respiration in the copepod *Calanus marshallae*, and 560 mg/l for increased mortality in copepods and the euphausiid *Euphausia pacifica*; concentrations of Mo mine tailings were always <15 mg/l at 0.5 km downstream from a Mo tailings outfall (Anderson and Mackas 1986).

Freshwater and marine fishes were--with one exception--extremely resistant to Mo; LC-50 (96 hour) values ranged between 70 mg/l and <3,000 mg/l (Table 3). The exception was newly fertilized eggs of rainbow trout exposed for 28 days through day 4 posthatch; the LC-50 (28 day) value was only 0.79 mg/l (Birge et al. 1980), and suggested that additional research is needed on the sensitivity of early life stages to Mo. In general, Mo was more toxic to teleosts in fresh water than in seawater and more toxic to younger fish than to older fish; in rainbow trout it bioconcentrated up to 16 mg/kg fresh weight in liver, 18 in spleen, 7 in muscle, 6 in gill, and 2 in gastrointestinal tract (Table 3; Short et al. 1971). Environmental levels of molybdenum as molybdate measured in the Mo mining areas of Colorado were not considered harmful to rainbow trout (McConnell 1977). Molybdenum enrichment of Castle Lake, California (a high mountain lake in which Mo was determined to be the limiting micronutrient), coupled with favorable environmental conditions, led to record high yields of trout. The addition of 16 kg of sodium molybdate, or 6.4 kg Mo, to Castle Lake in July 1963 was followed by larger standing crops of zooplankton and bottom fauna, which probably promoted survival of the 1965 year class and resulted in record yields to the angler of rainbow trout and brook trout (*Salvelinus fontinalis*) in 1967 (Cordone and Nicola 1970). Enrichment of Mo-deficient waters to improve angler success merits additional research.

BIRDS

Data are missing on the effects of Mo on avian wildlife under controlled conditions. All studies conducted with birds have been restricted to domestic poultry.

Signs of Mo deficiency in domestic chickens included loss of feathers, lowered tissue Mo concentrations, reduced xanthine dehydrogenase activity in various organs, decreased uric acid excretion, disorders in ossification of long bones, and changes in joint cartilage that led to complete immobility; signs were eliminated when diets were supplemented with Mo at concentrations of 0.2 to 2.5 mg/kg (Reid et al. 1956; Friberg and Lener 1986). Efforts to produce a Mo deficiency syndrome in birds and mammals by feeding diets low in Mo have been unsuccessful (Friberg et al. 1975). Thus, it has been necessary to introduce a compound with a known property of inhibiting Mo, namely wolframate (Na_2WO_4), a tungsten compound. Wolframate increases Mo excretion, leading to Mo deficiency in rats and chickens. With this technique it has been possible to produce an assumed Mo deficiency in chicks consisting of reduced weight gain and sometimes death (Friberg et al. 1975). Dietary requirements to maintain normal growth in rats and chicks were probably less than 1 mg Mo/kg food, and thus substantially less than that of any other trace element recognized as essential (Mills and Breamer 1980). In fact, birds may require Mo at concentrations up to 6 mg/kg in their diets for optimal growth (Kienholz 1977). Dietary Mo counteracts adverse effects in chicks on growth and survival induced by hexavalent chromium. Chicks fed 900 mg chromium/kg ration for 4 weeks showed significantly depressed growth, 25% mortality, and elevated liver chromium; however, diet supplementation to 150 mg Mo/kg resulted in normal growth and liver chromium values, and no deaths (Chung et al. 1985).

Early studies with chicks and turkey poults showed that the addition of only 13 to 25 ug Mo per kg--as molybdate or molybdic acid--to basal diets containing 1.0 to 1.5 mg Mo/kg resulted in a growth advantage of 14% to 19% in 4 weeks over that in unsupplemented groups (Reid et al. 1956, 1957). Roosters given dietary supplements of 100 or 400 ug Mo per bird daily for 4 weeks to basal diets containing 0.51 mg Mo/kg had reduced serum uric acid values when compared to those of controls; the significance of this finding is not clear (Karring et al. 1981). Birds are relatively resistant to Mo. For example, day-old chicks fed diets containing 20% Mo mine tailings for 23 days were unaffected, and those fed diets containing 40% Mo mine tailings showed only a slight reduction in body weight during the same period (Kienholz 1977). Dietary levels of 200 mg Mo/kg ration results in minor growth inhibition of chicks; and at 300 mg/kg feed, the growth of turkey poults was reduced (Underwood 1971). Dietary supplements of 500 mg Mo/kg ration produced a slight decrease in growth rate of chicks after 4 weeks; hens, however, laid 15% fewer eggs than controls, and all eggs contained embryolethal concentrations of 16 to 20 mg Mo/kg (Friberg et al. 1975). At dietary supplements of 1,000 mg Mo/kg, egg production was reduced 50% in domestic chickens (Friberg et al. 1975). Dietary loadings of 2,000 mg/kg induced severe growth depression and a 100X increase in Mo content in tibia (Underwood 1971), and an 80% reduction in egg production (Friberg et al. 1975). At 4,000 mg/kg diet, severe anemia was reported in chickens

(Underwood 1971). Mortality of chicks fed 6,000 mg Mo/kg diet for 4 weeks was 33%; at 8,000 mg Mo/kg diet for 4 weeks, 61% of the chicks died and survivors weighed only 16% as much as the controls (Friberg et al. 1975). Chicks, unlike mammals, did not experience Mo reduction in tissues after sulfate administration--although sulfate markedly reduced the signs of No toxicity (Underwood 1971).

MAMMALS

Data on the effects of Mo on mammalian wildlife are scarce. Almost all studies conducted to date on Mo effects under controlled conditions have been on livestock, especially cattle and sheep.

Molybdenum is beneficial and perhaps essential to adequate mammalian nutrition; moreover, it can protect against poisoning by copper or mercury, and may be useful in controlling cancer. Evidence of functional roles for Mo in the enzymes xanthine oxidase, aldehyde oxidase, and sulphite oxidase suggests that Mo is an essential trace nutrient for animals (Underwood 1971; Earl and Vish 1979; Mills and Breamer 1980). Signs of Mo deficiency include decreased intestinal and liver xanthine oxidase activity (Mills and Breamer 1980). Molybdenum prevents damage to the liver in sheep recieving excess copper; accumulations of copper and Mo in kidney were present in a biologically unavailable form and of negligible physiological significance (Van Ryssen et al. 1982). Dietary supplements of 70 mg Mo per day for a restricted period is recommended for reduction of liver Cu in sheep, provided dietary Cu levels are simultaneously reduced (Van Ryssen et al. 1986). Molybdenum, as sodium molybdate, protects against acute inorganic mercury toxicity in rats by altering the metabolism of cysteine-containing proteins in the cytoplasm of liver and kidney, resulting in lowered mercury content in these organs (Yamane and Koizumi 1982; Koizumi and Yamane 1984). Anticarcinogenic properties of Mo in rats have been reported, although the mechanisms of action are unknown. In one study, 2 or 20 mg Mo/1 in drinking water significantly inhibited cancer of the esophagus and forestomach experimentally induced by N-nitrososarcosine ethyl ester (Luo et al. 1983). In another study with virgin female rats, 10 mg Mo/l in drinking water reduced by half the number of mammary carcinomas experimentally induced by N-nitroso-N-methylurea (Wei et al. 1985). Additional research seems warranted on the role of Mo in cancer inhibition.

Molybdenosis has been produced experimentally in many species of mammals, including cattle, sheep, rabbits, and guinea pigs (Friberg et al. 1975). Signs of Mo poisoning vary greatly among species, but generally include the following: copper deficiency, especially in serum; reduced food intake and growth rate; liver and kidney pathology; diarrhea and dark-colored feces; anemia; dull, wiry, and depigmented hair; reproductive impairment, including delayed puberty, female infertility, testicular degeneration, and abnormal or delayed oestrus cycle; decreased milk production; joint and connective tissue lesions; bone abnormalities; and loosening and loss of teeth (Underwood 1971; Dollahite et al. 1972; Friberg et al. 1975; Erdman et al. 1978; Penumarthy and Oehme 1978; Ward 1978; Chappell et al. 1979; Mills and Breamer 1980; Alary et al. 1981; Baldwin et al. 1981; Friberg and Lener 1986; Van Ryssen et al. 1986; Phillippo et al. 1987a). These authorities also agree on three additional points: first, early signs of molybdenosis are often irreversible, especially in young animals; second, the severity of the signs depends on the level of Mo intake relative to that of copper and inorganic sulfate; and third, if afflicted animals are not removed promptly from Mo-contaminated diets and given copper sulfate therapy, death may result.

Molybdenum poisoning in ruminants, or teart disease, has been known since the mid 1800's and affects only ruminants of special pastures. Degree of teartness varies from field to field and season to season, and is usually proportional to the Mo content in herbage. Molybdenum levels in typical teart pastures range from 10 to 100 mg/kg dry weight compared to normal levels of 3 to 5 mg/kg (Friberg and Lener 1986). If herbage contains more than 12 mg Mo/kg dry weight, problems should be expected in cattle, and to a lesser extent in sheep (Friberg et al. 1975). In situations where cattle are accidentally exposed to high Mo levels, the administration of copper sulphate should result in Mo excretion, up to 50% in 10 days (Penumarthy and Oehme 1978). Aside from cattle and sheep, all evidence indicates that other mammals are comparatively tolerant of high dietary intakes of Mo, including horses, pigs, small laboratory animals, and mammalian wildlife (Underwood 1971; Buck 1978; Chappell et al. 1979; Friberg and Lener 1986; Table 4). Cattle excrete Mo primarily through feces, but other (more tolerant) species such as pigs, rats, and man, rapidly excrete Mo through urine and this may account, in part, for the comparative sensitivity of cattle to Mo (Underwood 1971). Cattle normally excrete about 67% of all administered MoO_3 in feces and urine in 7 days; guinea pigs excreted 100% in urine in 8 days; and swine excreted 75% in urine in 5 days (Penumarthy and Oehme 1978). Cattle are adversely affected when they graze copper-deficient pastures containing 2 to 20 mg/kg Mo, and the copper to Mo ratio is less than 3; or when

they are fed low copper diets containing 5 mg (or more) Mo/kg dry weight; or when total daily intake approaches 141 mg Mo; or when body weight residues exceed (a fatal) 10 mg Mo/kg (Table 4). It is clear that both the form of Mo administered and the route of exposure affect Mo metabolism and survival (Table 4). By comparison, adverse effects (some deaths) were noted at 250 mg Mo/kg body weight (BW) (in guinea pigs), at 50 mg/kg BW in domestic cats (central nervous system impairment), at 10 mg/l drinking water in mice (survival), at 10 to 15 mg total daily intake in man (high incidence of gout-like disease), and at to 3 mg/m^3 air in man for 5 years (respiratory difficulties), or 6 to 19 mg/m^3 in man for 4 years (Table 4).

Table 4. Molybdenum effects on selected species of mammals.

Species, dose, and other variables	Effects (reference) [a]
Cattle, cows, *Bos* spp.	
Near steelworks, 20 kg Mo as MoO_3 emitted daily in gaseous form; fallout deposits ~2 mg/m^2 monthly, corresponding to a pasture Mo content from 2 to 20 mg Mo/kg dry weight. Pasture had slight copper deficiency of natural origin, with copper:Mo ratio in pastures <3	About 40% of 5,000 grazing cows with signs of molybdenosis. No signs of poisoning before steelworks began operations. Signs evident almost immediately in first grazing season; most pronounced in younger animals closest to source. Remedial actions included copper glycine administration to cattle, and installation of additional emission filters at the steelworks (1)
Low dietary Mo (<5 mg Mo/kg), adequate copper	Growth normal; fertilization rate 100% liver copper >70 mg/kg dry weight; 63% of embryos developed normally (2)
Fed diets containing 5 mg Mo/kg dry weight and 4 mg copper/kg dry weight for 84 weeks	Decreased food intake, reduced efficiency of food use, altered iron metabolism, clinical signs of copper deficiency. Onset of puberty delayed 10 weeks, decreased conception rate (fertility 12% to 33% vs. 57% to 80% in controls), disrupted oestrus cycle (67% were anoestrus vs. 7% in controls), and other signs consistent with decreased releases of luteinizing hormones associated with altered ovarian secretion (3, 4)
High dietary Mo (15 to 20 mg Mo/kg), copper-deficient	Growth and fertilization normal; liver copper 10 mg/kg dry weight; only 16% of embryos developed normally (2)

Fed diets of normal copper, and high Mo (30 mg/kg feed)	Blood Mo level of 0.6 to 0.8 mg/L (5)
Diets containing 40 mg Mo/kg and 6 mg copper/kg fed to lactating cows for 9 weeks	Reduction of 30% in milk yield; rapid decline in plasma copper; milk Mo levels of 1.6 mg/L; growth reduction in nursing calves (6)
Fed diets of 60 mg Mo/kg dry weight	Low liver copper, intestinal disturbances, brittle bones prone to fracture (7)
Dairy herd fed pelleted feed containing 140 mg Mo/kg fresh weight and up to 10 mg copper/kg fresh weight	Molybdenosis. Contaminated magnesium oxide (12,200 mg Mo/kg) added to ration at 1% was the source of the excess Mo (8)
Drinking water with Mo as ammonium molybdate. Basal diet with 13 mg copper/kg and 2,900 mg sulfur/kg	
1 or 10 mg Mo/L in drinking water for 21 days	In 5-week-old calves, there was no effect on liver or plasma copper levels (9)
50 mg Mo/L in drinking water for 21 days	Copper liver concentration reduced to 201 mg/kg dry weight vs. 346 in controls; copper in plasma elevated to 1,100 ug/L vs. 690 in controls. No effect on growth, or food and water consumption (9)
Total daily intake of 100 mg Mo	Normal milk Mo level of 0.06 mg/L (5)
Total daily intake of 141 mg Mo	Anorexia, darrhea, and weight loss in Swiss beef cattle (10)
Total daily intake of 500 mg Mo	Milk Mo level of 0.37 mg/L (5)
Total daily intake of 1,360 mg Mo daily as soluble molybdate	Signs of molybdenum poisoning (7)
10 mg Mo/kg body weight	Lethal dose (11)
Guinea pig, *Cavia* sp.	
Chronic exposure, daily dose in mg Mo/animal	

25, as MoO_3	75% mortality (12)
200, as calcium molybdate	25% mortality (12)
Air concentrations of 28 to 285 mg Mo/m^3	Hexavalent Mo compounds absorbed appreciably, but not disulfide compounds (10, 13)
Dose, in mg Mo/kg body weight, various administration routes	
80	LD-0 (12)
250	Some deaths (11)
400	LD-75 (4 days) (12)
800	LD-100 (4 months) (12)
Domestic ruminants	
Pastures containing 10 to 20 mg Mo/kg dry weight	"Risk" zone for molybdenosis (12)
Pastures containing 20 to 100 mg Mo/kg dry weight	"Teart" disease characterized by anemia, poor growth, diarrhea; prolonged exposure resulted in joint deformities, and death (13)
Horses, ponies, *Equus* sp.	
Feeding on teart pastures with elevated Mo content	No effect (5)
Given single oral dose of radio Mo-99, as molybdate, or about 20 to 28 mg	Mo appeared rapidly in plasma as Mo-99 molybdate, but quickly cleared with Tb 1/2 of 7 to 10 h (14)
Fed diets containing 20 mg Mo/kg dry weight for 4.5 months; diet supplemented with sulfur for one month at 1.2 g/kg feed	Animals remained healthy. No decline in total plasma copper or increase in plasma insoluble copper (14)
Fed diets containing up to 107 mg Mo/kg for 14 days	Increasing dietary Mo resulted in decreasing copper retention due to increasing excretion of copper in feces; up to 1.45 g Mo/kg body weight absorbed and retained with no obvious adverse effects (15)
Cat, *Felis domesticus*	
Intravenous injection, in mg Mo/kg body weight	
25	Increased arterial blood pressure (12)
50	Central nervous system impairment (12)

Man, *Homo sapiens*	
Drinking water, in ug/L	
50	No effect (11)
200	Increased urinary excretion, normal serum Mo levels, no change in copper metabolism (11)
Total intake, in mg Mo daily	
0.18	Average intake in United States (11)
0.5 to 1	Increased urinary copper excretion (11)
10	Increase in blood and urine Mo levels, increases in serum ceruloplasmin, increased xanthine oxidase activity (11)
10 to 15	Increased uric acid, decreased copper excretion, high incidence of gout-like disease (11)
Atmospheric concentrations, in mg Mo/m^3 air	
1 to 3; 5-year exposure	Respiratory difficulties (12)
6 to 19; 4-year exposure	Respiratory difficulties (12)
Mouse, *Mus* spp.	
10 mg Mo/L in drinking water of breeding mice	Decrease in survival of F_2 and F_3 generations (16)
Sheep, *Ovis* spp.	
Mo deficient diets of 0.03 mg/kg	High incidence of renal xanthine calculi (5)
Mo adequate diet of 0.4 mg/kg, due to resowing of pasture and lime treatment	Zero incidence of renal calculi (5)
Mo content of pasture 0.4 to 1.5 mg/kg dry weight	Mo concentrations, in mg/kg fresh weight, were 0.0 to 0.03 in plasma, 2.0 to 2.4 in liver, and 0.4 to 0.5 in kidney. No lameness or connective tissue lesions (17)

2.4 mg Mo/kg diet in lambs	Significantly enhanced growth when compared to sheep fed 0.36 mg Mo/kg diet; growth associated with increased cellulose digestibility by rumen biota (5)
Grazing pastures treated 3x with 420 g Mo/ha: at start, week 45 and week 72. Mo content of pasture usually 5.5 to 12.5 mg/kg dry weight	Mo concentrations, in mg/kg fresh weight, were 1.7 to 2.4 in plasma, 6.0 to 6.4 in liver, and 6.9 to 8.1 in kidney. Lameness and connective tissue lesions in most sheep (17)
Given diets of high copper (82 mg/kg) and sulfur (3.8 g/kg), and Mo at 20, 40, or 60 mg/kg for 193 days	Liver damage due to copper at low Mo (20 mg/kg) diets; at 40 and 60 mg Mo/kg, both metals accumulated in kidney cortex but no evidence of liver histopathology or kidney damage (18)
Breeding ewes fed diets of normal copper, high Mo (30 mg/kg feed)	Blood Mo level of 2.4 to 3.4 mg/L (5)
Diets of 50 mg Mo/kg	Avoidance by lambs; may be learned olfactory recognition (19)
Lambs grazing on soils where copper:Mo ratio is <0.4	Swayback observed in 15% to 39% (10)
Ram lambs fed diets of adequate sulfate and copper (7.7 mg/kg dry weight). Copper to Mo ratios of 5.5, 5.3, 1.1, or 0.7 for 105 days	No significant measurable effects at ratios of 5.5 and 5.3. Secondary copper deficiency (molybdenosis) at 1.1 ratio evident in blood and plasma, and in liver at 0.7 (20)
Lambs fed daily intake of 8 mg Mo, 36.3 mg copper, and 3.7 g sulfur for 125 days	No effect on growth of food intake; significant increases in levels of kidney cortex copper, liver Mo, and plasma copper; major differences in responses among breeds tested (21)
Total intake raised from 0.4 daily mg daily to 96 mg daily	Blood Mo level of 4.95 mg/L (5)
Fed 75 mg copper daily for 50 days,	Molybdenosis within 8 days (22)

followed by 140 mg Mo and 4 g sulfur daily for 13 days with no added copper	
As above, but 70 mg Mo daily at day 13 for 34 days	40% reduction in liver copper (22)
White rabbit, *Oryctolagus* sp.	
Dietary Mo concentrations, in mg Mo/kg ration	
100; lifetime exposure	Reduced growth, hair loss, dermatosis, anemia, skeletal and joint deformities, decreased thyroxin (11)
500; 12 weeks	No obvious effects (12)
1,000; 12 weeks	Some growth retardation (12)
2,000 to 4,000	Many deaths of weanlings in about 37 days, and of adults in 53 days. Survivors were anorexic, diarrhetic, anemic, and had front-leg abnormalities; successful recovery after copper therapy (12)
5,000	Thyroid dysfunction (11)
Rat, *Rattus* spp.	
Drinking water, in mg Mo/L	
10; chronic exposure of 3 years	Disrupted calcium metabolism (29). Increased sensitivity to cold stress, elevated tissue residues of 50 to 60 mg Mo/kg dry weight (23, 24)
20; 30 weeks exposure	No effect on growth or organ histology (25)
50; lifetime exposure	Some growth retardation (11)
1,000; lifetime exposure	No severe signs observed in breeding adults. Resultant pups, however, maintained on this regimen were stunted, rough haired, sterile (males), and hyperactive (11)
Dose, in mg Mo per animal daily for up to 232 days	
10	LD-25 to LD-50 for hexavalent Mo compounds (12)

100	LD-50 for calcium molybdate (12)
125	LD-50 for MoO_3 (12)
333	LD-50 for ammonium molybdate (12)
Atmospheric concentrations, in mg Mo/m^3	
64; 2 h	Outwardly normal, some microscopic damage due to MoO_3 exposure (12)
Up to 5,000 ammonium paramolybdate, 12,000 molybdenum dioxide, 15,000 molybdenum trioxide, or 30,000 metallic Mo; one-hour exposure	Four weeks postexposure, there were no adverse effects except for some irritation of upper respiratory passage (12)
Feeding levels, in mg Mo/kg diet	
50	Diet avoidance (19). In low sulfate diets and 5 weeks exposure, rats had reduced growth and mandibular exostoses (10)
80; copper-deficient	Inhibited growth and reduced survival (5, 26)
80; 35 mg $CuSO_4$/kg	No measurable effects (5, 26)
100; lifetime exposure	Appetite loss, weight loss, reduced growth, anemia, mandibular exostoses, bone deformities, liver and kidney histopathology, increased liver copper residues, male sterility (11)
400	After 5 weeks, growth depression, anemia, mandibular exostoses; some deaths at lifetime exposures (12)
500 or 800	No deaths in 6 weeks; growth retardation and anemia (12)
500 or 1,000; 77 mg copper/kg	Poor growth (5, 26)
500 or 1,000;	Normal growth (5, 26)
5,000	Lethal in 2 weeks (11, 12)
Dose, in mg Mo/kg body weight	

0.00002 to 0.001	50% excretion (Tb 1/2) in 60 h to 113 h for kidney, liver, spleen, small intestine, and skin (10)
0.003	Tb 1/2 in 47 h (10)
>0.003	Tb 1/2 in 3 h when administered subcutaneously, 6 h for intragastric application (10)
4.5, intravenous injection	Biliary excretion of Mo^{+6} compounds was more rapid than Mo^{+5} compounds (27)
100	When inhaled as MoO_3, irritating to eyes and mucous membranes and eventually lethal. Repeated oral administration leads to histopathology of liver and kidney (13)
100 to 150	Lethal (11)
114	All recovered after intraperitoneal injection of sodium molybdate (12)
117	All dead within a few hours after intraperitoneal injection of sodium molybdate (12)
500; daily	Tolerated when given as disulfide (13)
500; 28 days	Reduced growth, disrupted blood and enzyme chemistry, histopathology of liver and kidney; partly reversed by 20% protein diet (28)
Domestic pig, *Sus* sp.	
Fed diets containing 1,000 mg Mo/kg for 3 months	No effect (5)

[a]References: 1, Alary et al. 1981; 2, O'Gorman et al. 1987; 3, Phillippo et al. 1987a; 4, Phillippo et al. 1987b; 5, Underwood 1971; 6, Wittenberg and Devlin 1987; 7, Penumarthy and Oehme 1978; 8, Lloyd et al. 1976; 9, Kincaid 1980; 10, Friberg and Lener 1986; 11, Chappell et al. 1979; 12, Friberg et al. 1975; 13, Goyer 1986; 14, Strickland et al. 1987; 15, Cymbaluk et al. 1981; 16, Earl and Vish 1979; 17, Pitt et al. 1980; 18, Van Ryssen et al. 1982; 19, White et al. 1984; 20, Robinson et al. 1987; 21, Harrison et al. 1987; 22, Van Ryssen et al. 1986; 23, Winston et al. 1973; 24, Winston et al. 1976; 25, Luo et al. 1983; 26, Underwood 1979; 27, Lener and Bibr 1979; 28, Bandyopadhyay et al. 1981; 29, Solomons et al. 1973.

In newborn lambs from ewes that consumed high-Mo diets during pregnancy, demyelinization of the central nervous system was severe, accompanied by low copper contents in the liver (Earl and Vish 1979). Sheep are

more tolerant than cattle to Mo poisoning due, in part, to a lower turnover of ceruloplasmin, a copper-transporting enzyme that is inhibited by Mo; however, this characteristic makes sheep more sensitive than cattle to copper poisoning (Ward 1978). For example, chronic copper poisoning in sheep in several districts in Norway is probably due to Mo-deficient forages rather than to excess copper intake (Froslie et al. 1983). Swayback is a spastic paralysis in lambs born of ewes that were copper deficient during pregnancy Todd 1976). In northern Ireland, where cases have been reported, pastures were not copper deficient and swayback was due to an imbalance of copper, Mo, and sulfur. Very severely affected lambs were paralyzed in all limbs and died shortly after birth because they were unable to stand and suckle. Lambs less severely affected developed signs in about 2 weeks, but usually only the hind limbs were affected. Brain and spinal cord lesions were present, resulting in demyelination of spinal cord and cavitation of brain tissues; lesions were irreversible, but death might have been avoided with adequate copper therapy (Todd 1976).

Horses are generally considered to be tolerant of dietary copper deficiencies and of copper and Mo excesses that affected ruminants. Yet Mo accumulated in equine liver and has been implicated as a possible contributory factor in bone disorders in foals and yearlings grazing pastures containing 5 to 25 mg Mo/kg (Cymbaluk et al. 1981; Strickland et al. 1987). Cattle and horses are highly susceptible to pyrrolizidine alkaloids, an ingredient in certain poisonous plants such as tansy ragwort (*Senecio jacobaea*). Signs of poisoning included elevated copper levels in liver followed by fatal hemolytic crisis. Sheep are more resistant to alkaloids than equines or bovines, and sheep grazing has been recommended as a means of controlling tansy ragwort. However, dietary supplements of 10 mg Mo/kg increased the susceptibility of sheep to tansy ragwort intoxication, despite the observed increase in copper excretion (White et al. 1984).

In rodents, Mo is neither teratogenic nor embryocidal to golden hamsters at doses up to 100 mg/kg body weight, and has no measurable effect on fertility or gestation of female rats given similar high doses (Earl and Vish 1979). Voluntary rejection of high-Mo diets by rats results in anorexia. This phenomenon implies sensory, probably olfactory, recognition of molybdate in combination with other dietary constituents to form compounds with a characteristic odor detectable by rats (Underwood 1971). The ability to reject high-Mo diets requires a learning or conditioning period because it is lacking or weak with freshly prepared diets and extends to a discrimination between a toxic (high Mo) and nontoxic (high Mo plus sulfate) diet. Rats may associate a gastrointestinal disturbance with a sensory attribute of diets containing toxic levels of Mo (Underwood 1971).

Data on Mo effects to mammalian wildlife are scarce, although those available strongly suggest that domestic livestock are at far greater risk (Table 4). Studies with mule deer (*Odocoileus hemionus*) showed that this species was at least an order of magnitude more tolerant to high levels of dietary Mo than were domestic ruminants, and at least as resistant as swine, horses, and rabbits (Ward and Nagy 1976; Ward 1978; Chappell et al. 1979). Female mule deer showed no visible effects after 33 days on diets containing up to 200 mg Mo/kg feed, or after 8 days at 1,000 mg/kg. Only slight effects--some reduction in food intake and some animals with diarrhea--were observed at diets of 2,500 mg/kg for 25 days. At feeding levels of 5,000 and 7,000 mg/kg for periods of 3 to 15 days, signs were more pronounced; however, recovery began almost immediately after transfer to uncontaminated feed. Signs of copper deficiency and of molybdenosis are very similar, and careful diagnosis is necessary to ensure use of the correct remedial action. For example, some populations of Alaskan moose (*Alces alces gigas*) showed faulty hoof keratinization and decreased reproductive rates, but this was attributed to copper-deficient browse growing on low copper soils, and not to increased Mo levels in herbage (Flynn et al. 1977). In another case, a high proportion of white-tailed deer (*Odocoileus virginianus*) feeding near uranium-mine spoil deposits in several Texas counties--areas in which extreme molybdenosis has been documented in grazing cattle--had antlers that were stunted, twisted, and broadened or knobby at the tips (King et al. 1984). However, the copper levels in liver of these deer were similar to those of deer in a control area--16.7 mg/kg fresh weight vs. 18.0--and only 1 of 19 deer examined from the mining district had a detectable Mo concentration in liver (0.7 mg/kg fresh weight) vs. none in any control sample. On the basis of low contents of copper in soils and vegetation, it was concluded that white-tailed deer examined were experiencing copper deficiency (hypocuprosis), with signs similar to molybdenosis (King et al. 1984).

In humans, Mo is low at birth, increases until age 20 years, and declines thereafter (Goyer 1986). Although conclusive evidence that Mo is required by humans is lacking, there is general agreement that it should be considered as one of the essential trace elements. The absence of any documented deficiencies in man indicates that the required level is much less than the average daily intake of 180 ug Mo in the United States (Chappell et al. 1979). Human discomfort has been reported in workers from copper-Mo mines, and in those

eating food products containing 10 to 15 mg Mo/kg and <10 mg copper/kg and grown on soils containing elevated Mo of 77 mg/kg and 39 mg copper/kg. Symptoms included general weakness, fatigue, headache, irritability, lack of appetite, epigastric pain, pain in joints and muscles, weight loss, red and moist skin, tremors of the hands, sweating, dizziness (Friberg et al. 1975), renal xanthine calculi, uric acid disturbances (Schroeder et al. 1970), and increased serum ceruloplasmin (Friberg and Lener 1986). The typical human adult contains only 9 mg of Mo, primarily in liver, kidney, adrenal, and omentum (Goyer 1986). Most of the ingested Mo is easily absorbed from the GI tract and excreted within hours or days in urine, mostly as molybdate; excesses may be excreted also by the bile, particularly as hexavalent Mo (Friberg et al. 1975; Goyer 1986; Friberg and Lener 1986). At high dietary levels Mo reportedly prevents dental caries (Schroeder et al. 1970), but this requires verification.

RECOMMENDATIONS

Although Mo is generally recognized as an essential trace metal for plants and animals, and may reduce the incidence and severity of carcinomas in rats (Luo et al. 1983; Wei et al. 1985) and dental caries in humans (Shamberger 1979), there is no direct evidence of Mo deficiency being detrimental to animal health. The minimum daily Mo requirements in diets are not yet established due to problems in preparing Mo-free rations (Chappell et al. 1979). As a consequence, no regulatory agency recognizes Mo as safe and necessary, and Mo can not be legally incorporated into animal feeds (Penumarthy and Oehme 1978).

The richest natural sources of Mo (i.e., 1.1 to 4.7 mg Mo/kg fresh weight) are plants unusually high in purines such as legumes and whole grains (Schroeder et al. 1970), followed by leafy vegetables, liver, and kidney (Shamberger 1979); the poorest sources are fruits, sugars, oils, and fat (Schroeder et al. 1970).

The greatest economic importance of molybdenosis is associated with subclinical manifestations of copper deficiency resulting from forages containing a low copper:Mo ratio. Unfortunately, these conditions are often difficult to diagnose accurately, and animal response to copper may be difficult to demonstrate (Ward 1978). One recommended treatment for afflicted cattle is 2 grams daily of copper sulfate to cows and 1 gram daily to young stock, or intravenous injection of 200 to 300 mg of copper sulfate daily for several days (Underwood 1971).

The animals most sensitive to Mo insult are domestic ruminants, especially cattle. Diets containing >15 mg Mo/kg dry weight and with a low copper to Mo ratio, or drinking water levels >10 mg Mo/l were frequently associated with molybdenosis in cattle (Table 5). By contrast, adverse effects were documented in birds at dietary levels >200 mg Mo/kg ration, in ruminant wildlife at dietary levels >2,500 mg Mo/kg, and in aquatic organisms--with one exception--at >50 mg Mo/l (Table 5). The exception was newly fertilized eggs of rainbow trout, which were about 21X more sensitive to Mo than were zygotes about 1/3 through embryonic development, and about 90X more sensitive than adult fish (Table 5).

Table 5. Proposed Mo criteria for the protection of living resources and human health.

Resource, criterion, and other variables	Concentration	Reference [a]
Terrestrial plants		
Okra, *Abelmoschus esculentus*		
Increased growth	3 mg/kg soil	1
Lettuce, *Lactuca sativa*		
Mo deficiency	~0.06 mg/kg dry weight (DW) plant	2
Mo sufficiency	>0.08 mg/kg DW	2
Corn, *Zea mays*		

No adverse effect	3.7 mg/kg DW plant	3
Terrestrial invertebrates		
Toxic baits		
Termites	~1,000 mg/kg	4
Other insect species	>5,000 mg/kg	4
Aquatic life		
Algae		
Deficiency levels	<0.005 to 17.7 ug/L	5, 6
High bioconcentration	>0.014 ug/L	7
Growth reduction	>50 mg/L	8
Invertebrates		
Reduced survival	>60 mg/L	9
Fish		
Adults		
High bioconcentration	>0.014 ug/L	7
Reduced survival	>70 mg/L	10
Eggs		
Newly fertilized		
Reduced survival	>0.79 mg/L	11
No adverse effects	<28 ug/L	11
Eyed		
Adverse effects	>17.0 mg/L	10, 12
Birds		
Mo deficiency	13 to 200 ug/kg diet	13, 14, 15
Normal growth	~1.0 mg/kg diet	16
Optimal growth	6.0 mg/kg diet	17
Growth reduction	200 to 300 mg/kg diet	18
Reproductive impairment	500 mg/kg diet	19
Reduced survival	6,000 mg/kg diet	19
Mammals		
Cattle, Cows (*Bos* spp.)		
Forage		
Healthy pasture	3 to 5 mg/kg DW	18
Possibility of molybdenosis	10 to 20 mg/kg DW	19
Probability of molybdenosis	20 to 100 mg/kg DW	18, 19
Toxic	15 to 30 mg/kg DW	20
Maximum tolerable level	6 mg/kg DW	20, 21
Recommended	0.1 to 0.5 mg/kg DW	22
Ratio of Copper to Mo in diet		
Mo in diet		
Molybdenosis probable	<0.4	23

Critical	<2.0	20
Critical	>20.0	22
Optimal for growth and reproduction	6.1 to 10.1	22, 23
Drinking Water		
Safe level	<10 mg/L	24
Minimum toxic concentration for calves	10 to 50 mg/L	24
Guinea pig, *Cavia* sp.		
No effect on survival	80 mg/kg body weight	19
Cat, *Felis domesticus*		
Adverse nonlethal effects	25 to 50 mg/kg body weight	19
Mule deer, *Odocoileus hemionus*		
No effect	200 to 1,000 mg/kg diet	25, 26, 27, 28
Reduction in food intake	2,500 mg/kg diet	25, 26, 27, 28
Nonlethal adverse effects	5,000 to 7,000 mg/kg diet	25, 26, 27, 28
Sheep, *Ovis* sp.		
Forage, recommended	<0.5 mg/kg dry weight	22
Rat, *Rattus* sp.		
Minimum daily need	0.5 ug	29
Disrupted calcium metabolism, elevated tissue residues	10 mg/L drinking water	30, 31, 32
Cancer inhibition	2 to 20 mg/L drinking water	33, 34
Food avoidance	50 mg/kg diet	35
Human health		
Total daily intake, 70 kg adult		
Minimal need	120 ug	29
Average range	100 to 500 ug	18, 19, 28, 29, 36, 37
Maximum	10 to 15 mg	28
In Mo mining areas	>1 mg	19
From food		
USA	170 ug	28
USA	335 (210 to 460) ug	19
USSR		
Children	159 ug	19
Adults	353 ug	19
UK	128 (110 to 1,000) ug	19
From drinking water	<5 ug	28
No effect level	<500 ug	28
Adverse effects		
Biochemical	0.5 to 10 mg	28
Clinical	10 to 15 mg	28

Drinking water		
Safe level	<50 ug/L	28
Irrigation water		
Safe level	<10 ug/L	28
Air		
Maximum permissible concentration		
USSR	6 mg/m^3	3
USA, 8 h daily, 5 days weekly	9.5 to 10 mg/m^3	15, 36
Blood		
"Normal"	14.7 ug/L	

[a]References: 1, Singh and Mourya 1983; 2, Gupta and Lipsett 1981; 3, Soon and Bates 1985; 4, Brill et al. 1987; 5, Vaishampayan 1983; 6, Steeg et al. 1986; 7, Short et al. 1971; 8, Sakaguchi et al. 1981; 9, Ahsanullah 1982; 10, McConnell 1977; 11, Birge et al. 1980; 12, Morgan et al. 1986; 13, Reid et al. 1956; 14, Reid et al. 1957; 15, Friberg and Lener 1986; 16, Mills and Bremner 1980; 17, Kienholz 1977; 18, Underwood 1971; 19, Friberg et al. 1975; 20, Schalscha et al. 1987; 21, Kume et al. 1984; 22, Garmo et al. 1986; 23, Baldwin et al. 1981; 24, Kincaid 1980; 25, Nagy et al. 1975; 26, Ward and Nagy 1976; 27, Ward 1978; 28, chappell et al. 1979; 29, Schroeder et al. 1970; 30, Solomons et al. 1973; 31, Winston et al. 1973; 32, Winston et al. 1976; 33, Luo et al. 1983; 34, Wei et al. 1985; 35, White et al. 1984; 36, Goyer 1986; 37, Shamberger 1979.

Proposed criteria for human health protection include drinking water concentrations <50 ug Mo/l, and daily dietary intakes <7 ug Mo/kg food--based on a 70 kg adult (Table 5). Molybdenum concentrations in blood of "healthy" people averaged 14.7 ug Mo/l, distributed between the plasma and erythrocytes. Anemic people had significantly lower blood Mo levels; in leukemia patients Mo levels increased significantly in whole blood and erythrocytes but not in plasma (Shamberger 1979). Additional work is recommended on the use of blood in fish and wildlife as an indicator of Mo stress and metabolism.

Increasing problems associated with marginal mineral deficiencies and unfavorable mineral interaction--as has been the case in the older agricultural areas of northern Europe--can be anticipated as pasture and forage production becomes more intensive (Ward 1978). Research has been recommended in areas having a high Mo content in soils and vegetation, and also in non-contaminated areas where consumption habits favor a high Mo intake and an imbalance in relation to other dietary constituents of importance, such as copper (Friberg et al. 1975). In some parts of the world where Mo has been substituted for lime, the soils have become more acidic, thus making them difficult to farm. Liming under these conditions may elevate soil Mo from levels, previously considered safe to levels potentially hazardous to grazing animals through high Mo herbage (Gupta and Lipsett 1981). The addition of Mo fertilizers to sheep pastures resulted in small increments in Mo content with negligible risk of induced copper deficiency. But it would be unwise to apply Mo fertilizers to temperate grasslands grazed by animals of low initial copper status, as judged by growth retardation of lambs from pastures supplemented with Mo (Suttle 1983a).

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