

# Molybdenum in Drinking-water

Background document for development of  
WHO *Guidelines for Drinking-water Quality*

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## Preface

One of the primary goals of WHO and its member states is that “all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water.” A major WHO function to achieve such goals is the responsibility “to propose regulations, and to make recommendations with respect to international health matters ....”

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as International Standards for Drinking-Water. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO Guidelines for drinking-water quality (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared/updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A “final task force” meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO internet site and in the current edition of the GDWQ.

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*Headquarters:*

- H. Galal-Gorchev, International Programme on Chemical Safety
- R. Helmer, Division of Environmental Health

*Regional Office for Europe:*

- X. Bonnefoy, Environment and Health
- O. Espinoza, Environment and Health

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## GENERAL DESCRIPTION

### *Physicochemical properties*

Physicochemical properties (1,2)

<i>Property</i>	<i>Value</i>
Melting point	2610 °C
Boiling point	5560 °C
Density	10.2 g/cm <sup>3</sup>
Vapour pressure	0.133 kPa at 3102 °C
Water solubility	insoluble

### *Organoleptic properties*

Ammonium molybdate imparts a slightly astringent taste to water at concentrations above about 10 mg of molybdenum per litre (2).

### *Major uses*

Molybdenum is used in the manufacture of special steels, in electrical contacts, spark plugs, X-ray tubes, filaments, screens, and grids for radio valves, and in the production of tungsten, glass-to-metal seals, nonferrous alloys, and pigments. Molybdenum disulfide has unique properties as a lubricant additive. Molybdenum compounds are used in agriculture either for the direct treatment of seeds or in the formulation of fertilizers to prevent molybdenum deficiency (1,3,4).

### *Environmental fate*

Molybdenum disulfide is sparingly soluble in water but is readily oxidized to give more soluble molybdates, which are stable in water in the absence of a reducing agent (2).

## ANALYTICAL METHODS

Molybdenum can be determined by graphite furnace atomic absorption spectroscopy with a detection limit of 0.25 µg/litre. Inductively coupled plasma atomic emission spectroscopy has a detection limit of 2 µg/litre (5).

## ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

### *Air*

Human intake of airborne molybdenum is not likely to be a major exposure pathway (6).

### *Water*

Molybdenum was present in 32.7% of surface water samples from 15 major river basins in the USA at concentrations ranging from 2 to 1500 µg/litre (mean 60 µg/litre) (7,8). Levels in groundwater ranged from undetectable to 270 µg/litre in another survey in the USA (9).

In a survey of finished water supplies in the USA, concentrations ranged from undetectable to 68 µg/litre (median 1.4 µg/litre) (10). In another survey of 380 finished water samples from across the USA, 29.9% contained measurable concentrations of molybdenum, with a mean of 85.9 µg/litre and a range of 3–1024 µg/litre (8).

Levels of molybdenum in drinking-water do not usually exceed 10 µg/litre (11). However, in areas near molybdenum mining operations, the molybdenum concentration in finished water can be as high as 200 µg/litre. Tapwater concentrations as high as 580 µg/litre have been reported in Colorado (6).

### ***Food***

Legumes, grains, and organ meats are good food sources of molybdenum; fruits, root and stem vegetables, and muscle meats are relatively poor ones (12,13).

### ***Estimated total exposure and relative contribution of drinking-water***

Molybdenum intakes in the USA range from 240 µg/day for adult men to 100 µg/day for women. Average intake is higher in those on low incomes (13,14). In most areas, molybdenum intake via drinking-water will not exceed 20 µg/day (11).

## **KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS**

The rate of gastrointestinal absorption of molybdenum is influenced by its chemical form and the animal species. Hexavalent molybdenum is readily absorbed following oral administration, the amount absorbed being higher in nonruminants than in ruminants (15–17). Tetravalent molybdenum is not readily absorbed (15). In humans, 30–70% of dietary molybdenum is absorbed from the gastrointestinal tract (18,19).

Following gastrointestinal absorption, molybdenum rapidly appears in the blood and most organs. Highest concentrations are found in the liver, kidneys, and bones (15,16,20). Molybdenum crosses the placental barrier (21). There is no apparent bioaccumulation of molybdenum in human tissues (20).

In rodents, molybdenum compounds are excreted largely in the urine, and only to a small extent in faeces (15,16). In ponies, cattle, and sheep, molybdenum excretion is generally divided between faeces and urine, owing to less complete gastrointestinal absorption (17,22,23). Molybdenum intake and excretion are balanced in most nonruminant species, including humans (20).

## **EFFECTS ON LABORATORY ANIMALS AND *IN VITRO* TEST SYSTEMS**

### ***Short-term exposure***

Oral subchronic LD<sub>50</sub>s for molybdenum(VI) oxide, calcium molybdate, and ammonium molybdate in rats were 125, 101, and 330 mg of molybdenum per kg of body weight per day, respectively (15). Death occurred over a period of 8–232 days.

In animals, molybdenum interacts in a complex manner with copper and sulfate by a mechanism which is as yet unknown. Animals on copper-deficient diets are generally more susceptible to molybdenum toxicity than those on copper-adequate diets. Dietary sulfate protects nonruminants against the symptoms of poisoning; if the animals are copper-deficient, however, it can intensify them (24,26).

In a study in which Holtzman rats (4 per dose) were fed diets containing hydrogen molybdate at 75 or 300 mg/kg (7.5 or 30 mg of molybdenum per kg of body weight per day), molybdenum significantly inhibited growth and increased copper and molybdenum concentrations in liver. These effects were reduced or reversed by the addition of sulfate (25). An enlargement of the femorotibial joint and a thickening of the epiphysis of the femur and

tibia were observed at both doses. This study suggests a LOAEL of 7.5 mg of molybdenum per kg of body weight per day, based on body weight loss and bone deformities.

Three weanling guinea-pigs were fed a low-copper basal diet with dietary additions of 0, 200, 500, 1000, or 2000 mg of molybdenum (8, 20, 40, or 80 mg/kg of body weight per day) for 8 weeks (27). An increase in molybdenum in the blood, liver, and kidneys was observed with increasing dietary molybdenum levels. An increase in copper was observed in the blood and kidneys with increasing molybdenum intake; at the two highest doses, there was a decrease in liver copper concentrations.

Weanling Long-Evans rats receiving dietary sodium molybdate (50 or 80 mg of molybdenum per kg of body weight per day) over 5–8 weeks developed diarrhoea, while weight gain decreased and copper levels in the liver increased (28).

In ruminants, sulfate tends to increase the toxicity of molybdenum even in the absence of copper deficiency (26,29,30). Molybdenum concentrations of 10 mg/kg of body weight in the ruminant diet resulted in tissue copper depletion, potentiated by dietary sulfate (31).

A total of 12 male Holstein calves (3 per group) received ammonium molybdate at 0, 1, 10, or 50 mg of molybdenum per litre (average daily doses of <0.01, 0.07, 0.7, or 3.7 mg of molybdenum per kg of body weight per day) in drinking-water for 21 days (32). No effects on growth were observed, but nonceruloplasmin copper was significantly elevated and copper uptake from plasma into liver was less than the endogenous loss in calves receiving the highest dose. The author suggested that the minimum toxic concentration of molybdenum is between 10 and 50 mg/litre, so that the NOAEL would be 0.07 mg/kg of body weight per day.

The effects of dietary molybdenum (1.7 g/day) were tested in four Holstein cows that were on low copper intake (30). None of the animals showed overt signs of toxicity after 6 months. After the molybdenum intake was increased to 3.4 g/day (7 mg/kg of body weight per day), one cow developed severe diarrhoea and exhibited signs of lethargy, cessation of milk synthesis, and general emaciation. When the molybdenum dose was increased to 5.1 g/day (10 mg/kg of body weight per day), two of three cows exhibited diarrhoea and emaciation. The addition of 0.26% sulfate greatly increased the severity of molybdenum toxicity. Dietary molybdenum increased the content of copper in the kidney and brain but decreased it in the liver. The kidney and spleen concentrated molybdenum to a greater degree than the liver or other organs.

### ***Reproductive toxicity, embryotoxicity, and teratogenicity***

Five pairs of Charles River CD mice received 10 mg of molybdenum per litre (as molybdate) (about 1.5 mg of molybdenum per kg of body weight per day) in deionized drinking-water for up to 6 months (33). Excess fetal mortality was observed; there were 15 (of 238) dead pups in the F<sub>1</sub> generation and 7 (of 242) dead pups, five dead litters, and one maternal death in the F<sub>2</sub> generation. The experiment was discontinued after the F<sub>3</sub> generation because of the elevated incidence of deaths of offspring and parents and infertility.

Four pregnant Cheviot ewes given diets supplemented with 50 mg of molybdenum per day (as ammonium molybdate) gave birth to four lambs, three of which exhibited ataxia (34). Histological examination revealed degenerative changes in the cytoarchitecture of the cerebral cortex and demyelination of the cortex and spinal cord, lesions similar to those described by other investigators as "swayback."

The effects of dietary molybdenum on reproductive ability and pup growth during lactation were studied in Long-Evans rats fed diets containing 0.1, 2, 8, or 14 mg of molybdenum per kg of body weight per day and either 5 or 20 mg of copper per kg for 13 weeks (35). The reduced number of litters at the two highest molybdenum concentrations was attributed to the



apparent infertility of males in the groups concerned as a result of varying degrees of degeneration of the seminiferous tubules. Lactating mothers at the two highest doses lost less weight during lactation than females in the lower-dose groups, and there were indications that pups from mothers exposed to the highest dose of molybdenum gained less weight at weaning than other pups; these effects were probably due to reductions in milk production associated with high maternal dietary intake of molybdenum. The NOAEL was 2 mg/kg of body weight per day.

Molybdenum administered orally by capsule for 129 days to two male Holstein calves at doses between 4.1 and 7.8 mg/kg of body weight per day caused a gradual disappearance of the spermatogenic and interstitial tissue. The LOAEL was 4.1 mg/kg of body weight per day (36). Female sheep fed a diet low in copper (1 mg/kg) and high in both molybdenum (25 mg/kg) and sulfate (0.53%) exhibited signs of reproductive failure (37).

### ***Mutagenicity and related end-points***

Ammonium molybdate was mutagenic in two of three *Escherichia coli* strains. Molybdenum(V) chloride was negative and ammonium molybdate strongly positive in the *Bacillus subtilis* rec-assay using DNA repair-competent H17 and repair-deficient M45 strains (38). Ammonium and sodium molybdates were neither mutagenic nor recombinogenic in the *Saccharomyces cerevisiae* reverse mutation and gene conversion assays (39).

### ***Carcinogenicity***

Although a significantly increased incidence of lung adenomas was observed in strain A mice injected intraperitoneally with molybdenum(VI) oxide (40), this study has no direct relevance to molybdenum intake via drinking-water. Recent studies suggest that molybdenum may act to prevent certain forms of cancer induced by N-nitroso compounds, e.g. oesophageal, forestomach, and mammary gland cancer, in laboratory animals (41,42).

## **EFFECTS ON HUMANS**

Molybdenum is considered to be an essential trace element in both animals and humans. Safe and adequate intake levels have been suggested for various segments of the population, namely 0.015–0.04 mg/day for infants, 0.025–0.15 mg/day for children aged 1–10, and 0.075–0.25 mg/day for all individuals above the age of 10 (43).

An infant with inborn deficiency of the molybdoenzymes sulfite oxidase and xanthine dehydrogenase exhibited abnormal distribution of urinary metabolites, neurological disorders, dislocated ocular lenses, and failure to thrive (44). A Crohn disease patient receiving total parenteral nutrition developed tachycardia, tachypnoea, severe headaches, night blindness, nausea, vomiting, central scotomas, generalized oedema, lethargy, disorientation, and coma; these symptoms were attributed to dietary molybdenum deficiency resulting in impaired function of the two molybdoenzymes (45).

Urinary levels of molybdenum and copper and serum levels of uric acid and ceruloplasmin appeared to be affected by molybdenum levels in drinking-water over a 2-year period (12). The low-molybdenum group consisted of 42 individuals from Denver, Colorado (USA), where the molybdenum concentration in drinking-water ranged from 1 to 50 µg/litre. The high-molybdenum group consisted of 13 college students from Golden, Colorado, where the drinking-water molybdenum concentrations were equal to or greater than 200 µg/litre. Plasma molybdenum levels were within the normal range among subjects in the low-molybdenum group, and no adverse health effects were observed in these subjects. Higher daily urinary molybdenum was associated with higher molybdenum intake: the mean urinary molybdenum for the Denver subjects was 87 µg/day compared with 187 µg/day for those from Golden.

Higher mean serum ceruloplasmin (401 v. 30 mg per 100 ml) and lower mean serum uric acid (4.4 v. 5.3 mg per 100 ml) were also associated with the higher molybdenum intake. Because no adverse effects were seen in either group, this study suggested a NOAEL for molybdenum in drinking-water of 200 µg/litre.

Evidence to support the suggestion that the molybdenum may have influenced serum ceruloplasmin was provided by a follow-up study of 13 students in Golden, Colorado, 2 years after the initial study. During this time, the average concentration of molybdenum in the Golden water supply decreased to 40 µg/litre (12). At this lower level of molybdenum in the drinking-water, serum molybdenum was nearly identical to the mean for the Denver residents. Serum ceruloplasmin was within the normal range of 20–35 µg/dl. Although serum uric acid values increased, this was believed to be the result of alcohol consumption. There were no significant differences in urinary copper values.

An epidemiological study involving 557 subjects in India indicated that a form of lower-limb osteoporosis may be associated with the high molybdenum content of the cereals consumed by the population (46).

The results from a cross-sectional study of 400 persons in two settlements of a molybdenum-rich province of the former Soviet Union suggested that the high incidence (18–31%) of a gout-like disease was associated with high intake of molybdenum (10–15 mg/day). The disease was characterized by joint pains of the legs and hands, enlargement of the liver, disorders of the gastrointestinal tract, liver, and kidney, increased blood levels of molybdenum and uric acid, increased xanthine oxidase activity, decreased blood levels of copper, and increased urinary copper. An increased synthesis of the molybdoenzyme xanthine oxidase resulting from high dietary molybdenum levels was proposed as the mechanism for this disorder (47).

A cross-sectional study was conducted with 25 workers at a molybdenum smelter in Denver, Colorado, exposed to molybdenum in dust (predominantly molybdenum(VI) oxide and other soluble oxides). The calculated minimum daily body burden was 0.15 mg/kg of body weight per day. High levels of molybdenum were present in the blood of 15 workers (up to 300 µg/litre) and in the urine of 12 of 14 workers (up to 11 mg/litre) (48). Mean serum ceruloplasmin and uric acid were higher for workers than controls. According to answers to medical questionnaires, six workers had upper respiratory infections in the 2 weeks prior to the questionnaire, and 15 reported joint pains, back pains, headaches, or skin or hair changes.

## **GUIDELINE VALUE**

No data are available on the carcinogenicity of molybdenum by the oral route. In a 2-year study of humans exposed via drinking-water, the NOAEL was found to be 0.2 mg/litre (12), but there are some concerns about the quality of this study. Although an uncertainty factor of 10 would normally be applied to reflect intraspecies variation, it is recognized that molybdenum is an essential element, and a factor of 3 is therefore considered to be adequate. This gives a guideline value of 0.07 mg/litre (rounded figure), which is in the same range as that derived on the basis of the results of toxicological studies in animals and is consistent with the essential daily requirement for molybdenum.

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