

# **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 the World Health Organization (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 third edition of the GDWQ was published in 2004, the first addendum to the third edition was published in 2006 and the second addendum to the third edition was published in 2008. The fourth edition will be published in 2011.

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 and 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 of potential health concern in drinking-water. In the first and second editions, these constituted Volume 2 of the GDWQ. Since publication of the third edition, they comprise a series of free-standing monographs, including this one.

For each chemical contaminant or substance considered, a lead institution prepared a background document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Japan, the United Kingdom and the United States of America (USA) prepared the documents for the fourth edition.

Under the oversight of a group of coordinators, each of whom was responsible for a group of chemicals considered in the GDWQ, 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. The draft documents were also released to the public domain for comment and submitted for final evaluation by expert meetings.

During the preparation of background documents and at expert 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.

## Acknowledgements

The current version of Molybdenum in Drinking-water, Background document for development of WHO *Guidelines for Drinking-water Quality*, is a revision of the background document prepared for the second edition of the Guidelines.

The work of the following working group coordinators was crucial in the development of this document and others contributing to the fourth edition:

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The draft text was discussed at the Expert Consultation for the fourth edition of the GDWQ, held in December 2011. The final version of the document takes into consideration comments from both peer reviewers and the public. The input of those who provided comments and of participants at the meeting is gratefully acknowledged.

The WHO coordinators were Mr R. Bos and Mr B. Gordon, WHO Headquarters. Ms C. Vickers provided a liaison with the International Programme on Chemical Safety, WHO Headquarters. Mr M. Zaim, Public Health and the Environment Programme, WHO Headquarters, provided input on pesticides added to drinking-water for public health purposes.

Ms P. Ward provided invaluable administrative support at the Expert Consultation and throughout the review and publication process. Ms M. Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

Many individuals from various countries contributed to the development of the GDWQ. The efforts of all who contributed to the preparation of this document and in particular those who provided peer or public domain review comments are greatly appreciated.



### **Acronyms and abbreviations used in the text**

|                  |                                      |
|------------------|--------------------------------------|
| DNA              | deoxyribonucleic acid                |
| LD <sub>50</sub> | median lethal dose                   |
| LOAEL            | lowest-observed-adverse-effect level |
| NOAEL            | no-observed-adverse-effect level     |
| USA              | United States of America             |

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## ***1. GENERAL DESCRIPTION***

### ***1.1 Physicochemical properties***

The physicochemical properties of molybdenum are summarized below (Asmanguljan, 1965; Weast, 1986).

| <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              |

### ***1.2 Organoleptic properties***

Ammonium molybdate imparts a slightly astringent taste to water at concentrations above about 10 mg of molybdenum per litre (Asmanguljan, 1965).

### ***1.3 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, non-ferrous 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 (Climax Molybdenum Co., 1973; Stokinger, 1983; Weast, 1986).

### ***1.4 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 (Asmanguljan, 1965).

## ***2. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE***

### ***2.1 Air***

Human intake of airborne molybdenum is not likely to be a major exposure pathway (Chappell, 1973).

### ***2.2 Water***

Molybdenum was present in 32.7% of surface water samples from 15 major river basins in the United States of America (USA) at concentrations ranging from 2 to 1500 µg/l (mean 60 µg/l) (Kopp & Kroner, 1967; National Academy of Sciences, 1977). Levels in groundwater ranged from undetectable to 270 µg/l in another survey in the USA (Kehoe, Chalak & Largent, 1944).

In a survey of finished water supplies in the USA, concentrations ranged from undetectable to 68 µg/l (median 1.4 µg/l) (Durfor & Becker, 1964). 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/l and a range of 3–1024 µg/l (Kopp & Kroner, 1967). Similar results were obtained in the United Kingdom in an unpublished study that supported the Regional Heart Study (J. Fawell, personal communication, 2011). Subsequent, more modern studies, however, indicated much lower concentrations in tap water in the United Kingdom (Smedley et al., 2008) and in wells in Wisconsin, USA (Wisconsin Department of Health Services, 2010).

Levels of molybdenum in drinking-water do not usually exceed 10 µg/l (Greathouse & Osborne, 1980). However, in areas near molybdenum mining operations, the molybdenum concentration in finished water was reported to be as high as 200 µg/l. Tapwater concentrations as high as 580 µg/l have been reported in Colorado (Chappell, 1973). However, there appear to be no recent data to confirm these findings.

### ***2.3 Food***

Legumes, grains and organ meats are good food sources of molybdenum; fruits, root and stem vegetables, and muscle meats are relatively poor ones (Chappell et al., 1979; Tsongas et al., 1980).

### ***2.4 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 (Tsongas et al., 1980; Pennington, Young & Wilson, 1989). In most areas, molybdenum intake via drinking-water will not exceed 20 µg/day (Greathouse & Osborne, 1980).

## ***3. 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 non-ruminants than in ruminants (Fairhall et al., 1945; Miller et al., 1972; Kosarek, 1976). Tetravalent molybdenum is not readily absorbed (Fairhall et al., 1945). In humans, 30–70% of dietary molybdenum is absorbed from the gastrointestinal tract (Engel, Price & Miller, 1967; Robinson et al., 1973).

Following gastrointestinal absorption, molybdenum rapidly appears in the blood and most organs. Highest concentrations are found in the liver, kidneys and bones (Fairhall et al., 1945; Schroeder, Balassa & Tipton, 1970; Kosarek, 1976). Molybdenum crosses the placental barrier (Meinel et al., 1979). There is no apparent bioaccumulation of molybdenum in human tissues (Schroeder, Balassa & Tipton, 1970).

In rodents, molybdenum compounds are excreted largely in the urine and only to a small extent in faeces (Fairhall et al., 1945; Kosarek, 1976). In ponies, cattle and

sheep, molybdenum excretion is generally divided between faeces and urine, owing to less complete gastrointestinal absorption (Miller et al., 1972; Cymbaluk et al., 1981; Kelleher et al., 1983). Molybdenum intake and excretion are balanced in most non-ruminant species, including humans (Schroeder, Balassa & Tipton, 1970).

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

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

Oral subchronic median lethal doses (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 kilogram of body weight per day, respectively (Fairhall et al., 1945). Death occurred over a period of 8–232 days.

In animals, molybdenum interacts in a complex manner with copper and sulfate by a mechanism that 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 non-ruminants against the symptoms of poisoning; if the animals are copper-deficient, however, it can intensify them (Gray & Daniel, 1964; Suttle, 1974).

In a study in which Holtzman rats (four per dose) were fed diets containing hydrogen molybdate at 75 or 300 mg/kg (7.5 or 30 mg of molybdenum per kilogram 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. An enlargement of the femorotibial joint and a thickening of the epiphysis of the femur and tibia were observed at both doses (Miller, Price & Engel, 1956). This study suggests a lowest-observed-adverse-effect level (LOAEL) of 7.5 mg of molybdenum per kilogram 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 (Arthur, 1965). 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 kilogram of body weight per day) over 5–8 weeks developed diarrhoea, whereas weight gain decreased and copper levels in the liver increased (Cox et al., 1960).

In ruminants, sulfate tends to increase the toxicity of molybdenum even in the absence of copper deficiency (Huber, Price & Engel, 1971; Suttle, 1974; Campbell et al., 1976). Molybdenum concentrations of 10 mg/kg of body weight in the ruminant diet resulted in tissue copper depletion, potentiated by dietary sulfate (Suttle, 1980).

A total of 12 male Holstein calves (three 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 kilogram of body weight per day) in drinking-water for 21 days (Kincaid, 1980). No effects on growth were observed, but non-ceruloplasmin 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 was between 10 and 50 mg/l, so that the no-observed-adverse-effect level (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 (Huber, Price & Engel, 1971). 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.

#### ***4.2 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 kilogram of body weight per day) in deionized drinking-water for up to 6 months (Schroeder & Mitchener, 1971). 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 (Mills & Fell, 1960). 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 kilogram of body weight per day and either 5 or 20 mg of copper per kilogram for 13 weeks (Jeter & Davis, 1954). 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 (Thomas & Moss, 1951). 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 (Suttle & Field, 1969).

#### **4.3 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 deoxyribonucleic acid (DNA) repair-competent H17 and repair-deficient M45 strains (Nishioka, 1975). Ammonium and sodium molybdates were neither mutagenic nor recombinogenic in the *Saccharomyces cerevisiae* reverse mutation and gene conversion assays (Singh, 1983).

#### **4.4 Carcinogenicity**

Although a significantly increased incidence of lung adenomas was observed in strain A mice injected intraperitoneally with molybdenum(VI) oxide (Stoner et al., 1976), this study has no direct relevance to molybdenum intake via drinking-water. 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 (Luo, Wei & Yang, 1983; Wei, Luo & Yang, 1985).

### **5. 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 (National Academy of Sciences, 1989).

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 (Johnson et al., 1980). 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 (Abumrad et al., 1981).

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 (Chappell et al., 1979). 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/l. 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/l. 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  $\mu\text{g}/\text{day}$  compared with 187  $\mu\text{g}/\text{day}$  for those from Golden. Higher mean serum ceruloplasmin (401 vs 30  $\text{mg}/100\text{ ml}$ ) and lower mean serum uric acid (4.4 vs 5.3  $\text{mg}/100\text{ ml}$ ) 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  $\mu\text{g}/\text{l}$ .

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  $\mu\text{g}/\text{l}$  (Chappell et al., 1979). 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  $\mu\text{g}/\text{dl}$ . 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 (Krishnamachari & Krishnaswamy, 1974).

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  $\text{mg}/\text{day}$ ). 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 (Koval/skij, Jarovaja & Šmavonjan, 1961).

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  $\text{mg}/\text{kg}$  of body weight per day. High levels of molybdenum were present in the blood of 15 workers (up to 300  $\mu\text{g}/\text{l}$ ) and in the urine of 12 of 14 workers (up to 11  $\text{mg}/\text{l}$ ) (Walravens et al., 1979). 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.

## ***6. PRACTICAL CONSIDERATIONS***

### ***6.1 Analytical methods***

Molybdenum can be determined by graphite furnace atomic absorption spectroscopy with a detection limit of 0.25  $\mu\text{g}/\text{l}$ . Inductively coupled plasma atomic emission

spectroscopy has a detection limit of 2 µg/l (American Public Health Association, 1989).

### **6.2 Treatment methods and performance**

Molybdenum is not removed from drinking-water by normal treatment processes and appears to require specialist treatment such as ion exchange.

## **7. CONCLUSION**

Molybdenum generally occurs at very low concentrations in drinking-water, and it is therefore not considered necessary to set a formal guideline value. For guidance purposes, a health-based value can be derived.

In a 2-year study of humans exposed via drinking-water, the NOAEL was found to be 0.2 mg/l (Chappell et al., 1979), but there are some concerns about the quality of this study. As molybdenum is an essential element, a factor of 3 was considered to be adequate to reflect intraspecies variation. This gives a health-based value of 0.07 mg/l (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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